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This Issue in the Journal

Differences in cardiovascular mortality between Australia and New Zealand according to socioeconomic status: findings from the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study
Ralph A H Stewart, Fiona M North, Katrina J Sharples, R John Simes, Andrew M Tonkin, Harvey D White; for the Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Investigators

This paper analysed deaths from cardiovascular disease from a large study of patients who had stable coronary artery disease undertaken in both Australia and New Zealand during the 1990s. The study found a modest increase in the risk of death from cardiovascular disease with decrease in income in both countries. In addition, New Zealanders were, on average, about 40% more likely to die from cardiovascular disease (than Australians) across all the income groups. These differences could not be explained by any single factor, but differences in both lifestyle and medical care played a role.

Gaps in primary care documentation of cardiovascular risk factors
Natasha Rafter, Susan Wells, Alistair Stewart, Vanessa Selak, Robyn Whittaker, Dale Bramley, Paul Roseman, Sue Furness, Rod T Jackson

New Zealand has high rates of heart disease and stroke. It is possible to estimate a person’s risk of these diseases by collecting information about smoking, previous heart disease or stroke, any family history of these problems, and testing for diabetes and measuring blood pressure and cholesterol. We did an audit of GP electronic patient notes to see whether this information was being recorded. Our results from 2001–2003 showed that recording was incomplete, especially for diabetes and smoking status. Whilst a GP may know this information for their patient, it is important to have good documentation so the risk for the whole practice population can be assessed and tracked.

Survey of clinical echocardiography in New Zealand (SCANZ)
Paul G Bridgman, Akbar N Ashrafi, Stewart Mann, Gillian A Whalley; on behalf of the SCANZ collaborators

Echocardiograms are ultrasound scans of the heart. This study captured every echo performed in New Zealand in 1 week. There are large differences in service provision on a population basis across the country, but particularly between the 5 major centres. Overall service provision is well short of accepted standards.
Comparison of different markers of socioeconomic status with cardiovascular disease and diabetes risk factors in the Diabetes, Heart and Health Survey
Patricia A Metcalf, Robert R K Scragg, David Schaaf, Lorna Dyall, Peter N Black, Rod T Jackson

Heart disease and diabetes risk factors were more strongly associated with living in more deprived areas of Auckland and household income than with the socioeconomic index based on an individual’s occupation or with level of education. In general, the strongest associations were observed with living in more deprived areas of Auckland. These findings provide support for the application of area-based measure of deprivation in health policy development in New Zealand when other measures of socioeconomic status (SES) are not available.

Informed consent for vascular intervention: completing one audit loop
Katie Carter, Justin A Roake, Timothy Buckenham, Christopher M Frampton, David R Lewis

The process of informed consent for an operation or medical intervention is complex and spans many interactions between patients and medical staff during “the patient journey”. The aim is to provide adequate and appropriate information to patients to help them understand and choose between treatment (or indeed no treatment) options. Gaining a patient signature on a consent form does not equate to obtaining informed consent and some forward-thinking institutions have done away with generic consent forms. Divulgence of information to patients and patient understanding of this information is extremely difficult to assess. Documentation of the consent process, provision of patient information sheets, and use of procedure-specific consent forms may simplify as well as improve the consent process. Endorsement of these “aids to consent” by surgical institutions and national legislative bodies is desirable if not essential.
Cardiovascular health in New Zealand: areas of concern and targets for improvement in 2008 and beyond

Chris J Ellis, Andrew W Hamer

Cardiovascular (CVS) disease accounts for 39% of mortality in New Zealand, the commonest cause of death. Ischaemic heart disease (IHD) which accounts for 22% of all deaths and cerebrovascular disease (10% of deaths) are essentially the result of an ageing process of the arterial system: atherosclerosis.

Although ‘age-standardised’ mortality has fallen, atherosclerosis still affects large numbers of younger individuals who suffer from decades of ‘life years’ lost as a result. Further, there has been more than a doubling of hospital discharges for a heart attack from 1989 to 2002/2003, indicating that a larger number of older New Zealanders have been diagnosed with IHD and have been hospitalised.2

A comprehensive strategy of prevention, treatment, and ongoing management of affected patients is clearly required to limit the effect of this epidemic.

How well do we achieve this goal?

A unique opportunity to assess how well we are doing as a nation comes from a paper by Stewart et al (http://www.nzma.org.nz/journal/121-1269/2931) published in this issue of the Journal. It uses data from the Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) trial, which randomised 9014 stable IHD patients from Australia and New Zealand to either pravastatin 40 mg daily (which is approximately as potent as simvastatin [Lipex] 20 mg3) or a placebo medicine.

Overall, in the LIPID study published in 1998, there was a 25% reduction in cardiovascular (CVS) mortality over 6.1 years. The current analysis is from the extended follow up LIPID study and compares CVS risk factors, medical treatments, CVS mortality, and socioeconomic differences between our two countries.

Patients from both New Zealand and Australia, with remarkably similar baseline characteristics, were enrolled; the importance of this study is obvious as, of all countries, Australia has the closest mix of peoples, lifestyle, and diseases to New Zealand.

The findings are dramatic: there is a 35% greater chance of a New Zealander dying of CVS disease in the median follow-up period of 7.8 years, compared to an Australian. The 2784 New Zealanders were uncannily similar, although not identical, in baseline characteristics—including age, gender, risk factors (except total:HDL cholesterol ratio), and socioeconomic status—to the 5949 Australians enrolled in the study.

The two major differences seen in the studies analyses were that more Australians used ‘extra’ statin medication, outside of the trial, and that more Australian patients received revascularisation by coronary artery bypass graft (CABG) surgery or percutaneous coronary intervention (PCI).
Other unknown factors may also be affecting these figures, but the clear implication from this important study is very clear: we are not managing heart disease as well as the Australians!

**What are the major areas of concern for CVS patient management in New Zealand?**

**PHARMAC’s 15-year involvement in the New Zealand health care environment has been a difficult and sometimes dangerous experience for patients,** although there has been a resultant, general awareness of the need to ration expensive medicines.

Access to a number of CVS medications in recent years has been delayed or remains limited. The benefits from such medications, which are generally well proven in robust clinical trials, are large but have often not been readily available to New Zealanders.

In terms of statin availability and use, PHARMAC’s delays in making available appropriate medications has probably caused more unnecessary death and major morbidity to New Zealanders than any other of their policies.

In the late 1990s, most New Zealand patients with heart disease simply could not access these life-saving medicines. The extra use of statins outside of the trial in Stewart et al’s study is likely to account for some of the mortality advantage for those Australian patients. The additional lost opportunity for hundreds of thousands of other New Zealanders denied access to a statin during this time has simply been ignored by PHARMAC.

From 1993 to 2007 there was a 35% increase in funding for PHARMAC. However, the compound inflation rate over these 14 years was 33%, the population grew by 17%, and hence per person in New Zealand, there was a 15% decrease in drug funding over these 14 years for every man, woman, and child.

New Zealand currently spends less than half of the amount that Australia spends on pharmaceuticals, per capita. See Figure 1.

PHARMAC’s pride in this achievement is misguided: spending less money on medicines may not be a good idea if the cost in patients’ health is too great. It is anomalous that expenditure is capped in this area of health care but not others. The increasing imbalance has inevitably led to cost-shifting and set us aside from most other developed countries. The effects on patient risk, health outcomes, and also the medical workforce have been negative.

The importance of cost containment and achieving best value is well understood and the New Zealand taxpayer needs to rely on such an organisation to achieve this. However, there is wide agreement across the sector that the current model and funding policy should be reviewed for the future benefit of New Zealand. What is now desperately needed is a resetting of funding for pharmaceuticals in New Zealand, to allow a rapid change from one of rationing to one of facilitating access to medicines.
There is a limited availability of cardiac surgery and percutaneous coronary intervention in New Zealand. However, the fact is that there are only a limited number of patients who would benefit from these life-saving and symptom-limiting procedures, each year. This is not a ‘bottomless pit’ of expense.

If resource were made available and the operations undertaken, there would be an ongoing benefit for the individuals concerned. There would also be financial savings to be made, as patients are returned to the (taxpaying) workforce, and are able to lead productive lives with their families. Further, there would also be cost savings in hospitals.

Patients currently have a prolonged wait in a hospital bed to access heart surgery at 1 of our 5 Public Cardiothoracic Centres, without which treatment they are unable to be discharged home. At an approximate cost of $2000 per day per patient for staying in a coronary care unit, if 20 patients are always waiting for 2 weeks in each region, a scandalous $40,000 daily is wasted, or $15 million dollars per year, or approximately 500 cardiac operations per year (at $30,000 per operation) per Centre, on unnecessary hospital in-patient charges.
Appropriate investment in Health, where it is needed, will result in remarkable cost efficiencies as well as improved health care. People requiring heart-preserving surgery should no longer be ignored.

**Clinicians as a group, have a very real understanding of patient management structures, health costs and rationing, and societal and individual needs—and yet they are underutilised in decision-making.** They spend their life in the health care environment and develop a unique understanding of this complex workplace.

The current management structure pays scant attention to this group of health professionals, instead relying on well intentioned, but transient managers, to make the majority of important decisions which control the structure, and hence outcomes of health care. That clinicians do not play a major role in the structural planning of the New Zealand health services is a key problem.

The Health Ministry must actively seek out busy clinicians, who are representative of the majority of clinicians across New Zealand, to positions of influence. These clinicians should be found by asking the doctors’ medical colleges and professional organisations, such as the Cardiac Society, to nominate such individuals.

**Due to its small population New Zealand has a unique opportunity to develop a comprehensive and caring health service in CVS medicine and throughout all aspects of health care.** With only 4.2 million people, it is small enough for a progressive, efficient management team to plan services.

At most, probably five regions (centred on Auckland, Hamilton, Wellington, Christchurch, and Dunedin) should efficiently and smoothly organise all CVS and other services in New Zealand. This would not, and must not, result in a ‘centralisation’ of service, but a structure with real ability to direct funding out to the regional centres, where it is clearly needed—and also into key central services (e.g CABG operations), when needed.

Until this change occurs, we will continue to have extraordinary examples of health care waste. For example, in Auckland, which is divided into three health boards, a patient who is admitted to one hospital (e.g. Middlemore Hospital) close to where they may work, will have initial investigations in the emergency department to ensure that they are fit enough to be sent by ambulance to their ‘own’ hospital (e.g. North Shore Hospital) where they live! In the reverse direction, is another ambulance, transferring a ‘Middlemore’ patient back ‘home’.

**Health inequalities.** The second conclusion of the current paper reconfirms the well known fact that those in the lower socioeconomic groups suffer worse health, both CVS and in other areas. This is the crux of the matter. The challenge for New Zealand is to provide a comprehensive health service for all, because if we cannot adequately organise the health service, then the most vulnerable in our community will suffer most, a true indictment on the current state of play.
Conclusion

With the publication of Stewart et al’s important paper there is a unique opportunity for us to reassess our CVS health priorities. It represents a chance to change direction in the listed four key areas, whilst pursuing the already promoted and vital lifestyle campaigns.

All individuals working in CVS health care in New Zealand should refocus, following this report, and aim to improve how we use our limited health, and population resource. We have a new health minister and a new chief clinical advisor to the Health Ministry, a real chance for a new beginning.

Competing interests: None known.

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References:

Differences in cardiovascular mortality between Australia and New Zealand according to socioeconomic status: findings from the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study

Ralph A H Stewart, Fiona M North, Katrina J Sharples, R John Simes, Andrew M Tonkin, Harvey D White; for the Long-term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Investigators

Abstract

Background Cardiovascular mortality is higher in New Zealand compared to Australia, but reasons for this difference are uncertain. This study describes differences in cardiovascular risk factors and cardiovascular mortality in Australians and New Zealanders with stable coronary artery disease stratified by socioeconomic status.

Methods Socioeconomic status was estimated from the residential area of 5949 Australians and 2784 New Zealanders with a history of myocardial infarction or unstable angina who participated in the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study. Socioeconomic and international differences in cardiovascular risk factors, medical treatments, and cardiovascular mortality during a median follow-up period of 7.8 years were evaluated.

Results Cardiovascular mortality increased as the median residential-area income decreased in both Australia (hazard ratio [HR]/income tertile 1.20, 95% confidence interval [CI] 1.08–1.32) and New Zealand (HR 1.16, 95%CI 1.02–1.31), but was higher in New Zealand across all socioeconomic groups (HR 1.42, 95%CI 1.25–1.61). Obesity, smoking, and a high white blood cell count at baseline were associated with higher cardiovascular mortality and were more common in lower-income areas in both countries.

The total:HDL cholesterol ratio was higher in New Zealand, but similar across all socioeconomic groups. In both countries there were socioeconomic gradients in open-label usage of cholesterol-lowering medication, percutaneous coronary intervention, and coronary artery bypass surgery. However, Australians in all socioeconomic groups were more likely than New Zealanders to receive these treatments.

Conclusions Although there is an important socioeconomic gradient in cardiovascular mortality in both Australia and New Zealand, cardiovascular mortality is higher in New Zealanders than Australians with stable coronary disease from all socioeconomic groups.

International differences in cardiovascular morbidity and mortality appear to be strongly influenced by social, economic, and political factors. Cardiovascular mortality is falling in the established market economies of North America, Western Europe, Australia, and New Zealand—but differs substantially.
between countries and between different socioeconomic groups within these countries. It has been estimated that approximately half of the decline in mortality is due to improvements in the treatment and secondary prevention of coronary artery disease.

Besides differences in medical care, differences in diet, lifestyle, psychosocial stress, and other socioeconomic factors may contribute to mortality differences between populations.

It is possible that international differences in cardiovascular disease among the established market economies are driven by the same mechanisms that are responsible for socioeconomic differences in cardiovascular disease within these countries. Further, it has been proposed that strategies to reduce socioeconomic gradients within a country may also reduce overall mortality.

Australia and New Zealand have broadly similar political systems, cultures, and socioeconomic gradients in wealth, but mortality from ischaemic heart disease is about 25% higher in New Zealand than in Australia. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study was a cholesterol-lowering clinical trial comparing the 3-hydroxy-3-methylglutaryl coenzyme-A (HMGCoA) reductase inhibitor, pravastatin, with placebo treatment in 9014 stable patients with a history of myocardial infarction or hospitalisation for unstable angina, randomised throughout Australia and New Zealand between 1990 and 1992.

The aim of this analysis was to determine whether differences in cardiovascular mortality between Australian and New Zealand LIPID participants were mediated by the same mechanisms that influenced socioeconomic mortality differences within each country, or by different mechanisms.

Methods

Study population—The LIPID Study enrolled Australian and New Zealand men and women aged 31 to 75 years with a history of acute myocardial infarction or hospitalisation for unstable angina within the previous 3 months to 3 years. The exclusion criteria included significant illness during the preceding 3 months, unavailability for long-term follow-up, significant cardiac failure (New York Heart Association [NYHA] class III or IV), and treatment with cholesterol-lowering medication. After a run-in phase, participants with a fasting total serum cholesterol level in the range of 4.0 to 7.0 mmol/L and a serum triglyceride level of ≤5.0 mmol/L were randomly assigned to receive either pravastatin 40 mg/daily or a matching placebo. All participants received dietary and general lifestyle advice. The patients’ care was otherwise under the direction of their usual doctors, including the option for commencement of open-label cholesterol-lowering medication if deemed necessary.

Baseline assessment—The following cardiovascular risk factors were documented at the baseline assessment: current smoking, height, body mass index, measured systolic and diastolic blood pressure, reported diagnosis of hypertension, fasting blood glucose level, reported diagnosis of diabetes, urine protein level, urine sugar level, white blood cell count, serum total and high-density lipoprotein (HDL) cholesterol levels, and the triglyceride level.

The following clinical measures of disease severity were documented: history of myocardial infarction, myocardial infarction prior to the qualifying event, angina duration, Canadian Cardiovascular Society (CCS) angina classification, and NYHA symptom classification. Usage of drug treatments including aspirin, beta-blockers, angiotensin-converting-enzyme (ACE) inhibitors, and calcium antagonists was also documented, as was percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) before the qualifying event or between the qualifying event and randomisation.

Follow-up—Randomisation for the LIPID Study took place between June 1990 and December 1992. Routine visits were scheduled every 6 months thereafter. Drop-in to active cholesterol-lowering
treatment during the study was documented. Discontinuation of the study medication for longer than 2 weeks was classified as temporary discontinuation. All acute cardiovascular events and hospital admissions were documented, including admissions for PCI or CABG.

Information on deaths was obtained from hospital records, death certificates, autopsy reports, and physicians’ notes—and an Outcome Assessment Committee reviewed the documentation on all deaths from coronary heart disease, including fatal myocardial infarction, sudden death, death in hospital after possible myocardial infarction, and death related to heart failure.

At the final study follow-up visit in 1997, all patients were offered open-label pravastatin treatment and invited to participate in the LIPID Cohort Study. Patients unable or unwilling to attend further clinic visits were asked to give consent for follow-up of their health status through their usual doctor and/or medical records. The vital status of all but two individuals in the LIPID Study and LIPID Cohort Study was documented to December 1999.

**Measurement of socioeconomic status**—A residential-area-based measure of socioeconomic status was obtained by linking the street address at randomisation to the corresponding census collection districts of 5949 (99.8%) Australian and 2784 (91.2%) New Zealand LIPID participants. The population census used for the analysis were undertaken on 5 March 1991 in New Zealand and 6 August 1991 in Australia. The median income of the general population aged >15 years in each census collection district (averaging approximately 200 households) was then obtained from the Australian Bureau of Statistics or Statistics New Zealand.

For comparison of the LIPID Study participants with the general population of each country, subjects were classified into tertiles based on the median income of the general population in their residential area, matched for age (in 5-year bands) and gender in each country.

**Statistical analysis**—The dataset used for analysis included all LIPID participants to whom we were able to assign a residential-area income value. Income was examined separately for each country as a continuous variable and also as a categorical variable stratified into deciles and tertiles. Other variables were also examined as both continuous and categorical variables. For binary versions, the median value of the LIPID population was used where an accepted cut-off level had not been prespecified (height, baseline white blood cell count, serum lipids). For ease of presentation, univariate results are shown for income tertiles and the binary versions of other variables.

Chi-squared ($\chi^2$) tests were used to compare proportions, and the Chi-squared test for trends was used to evaluate linear trends in proportions. For age, the Student’s t-test was used to compare means, and linear regression was used to evaluate linear trends. Standard survival analysis methods were used to compare cardiovascular mortality by country and by income, including Kaplan-Meier survival curves and Cox regression methods. PCI and CABG during follow-up were treated as time-dependent covariates. A modified backwards-stepwise procedure was used to select the models.

Variables were included in the final model if they were significantly associated with a risk of cardiovascular mortality, or if they confounded the associations between cardiovascular mortality and country or residential-area income. Residential-area income was included in the final model as a linear term, centred at decile 6. This enhanced the comparability of income classifications between Australia and New Zealand, and there was no evidence of any departure from a linear decrease in the log hazard with a decrease in the income decile. The proportion of the country and income effects explained by measured prognostic variables was estimated using the landmark method:

Bootstrap confidence intervals were constructed with 500 replications using Stata Statistical Software (Release 7.0, Stata Corporation, College Station, Texas, USA).

**Results**

**Residential-area income by country**

The median residential-area incomes of LIPID participants were broadly representative of the general population in both Australia and New Zealand.

Australian participants had a median residential-area income of AU$13,538 (interquartile range [IQR] AU$11,408 to $16,049), while New Zealand participants had a median residential-area income of NZ$14,487 (IQR NZ$12,501 to $17,502).
The median residential area income of Australian participants in $NZ was $17,813 (IQR $15,013 to $21,120) using an estimated exchange rate of AU$0.76=NZ$1.00 for 1991. Thirty-three percent of Australian participants lived in the lowest, 35% in the middle, and 31% in the highest residential-area income tertile of the general population matched for age and gender, while 29% of New Zealand participants lived in the lowest, 36% in the middle, and 35% in the highest income tertile.

Both countries had a similar distribution of income. In Australia, the median income of the middle tertile was 1.3 times that of the lowest tertile, while the median income of the highest tertile was 1.6 times that of the lowest tertile. The corresponding figures in New Zealand were 1.2 and 1.6 times higher, respectively.

**Cardiovascular risk factors**

The distributions of age and sex were similar in both countries. Participants who lived in higher-income areas were more likely to be younger and male, but this trend was small (Table 1). A higher percentage of Australians than New Zealanders had a reported diagnosis of hypertension, although the percentages of participants with a measured blood pressure of ≥140/90 mmHg at baseline were similar in both countries.

Australians were also more likely to have a diagnosis of diabetes, although blood glucose levels were similar in both countries. New Zealanders had a higher total:HDL cholesterol ratio than Australians. Similar percentages of the participants in both countries were current smokers, obese (body mass index >30 kg/m²), of shorter stature, had a high baseline white blood cell count, or had a high serum triglyceride level.

Socioeconomic gradients in risk factors differed from those between countries. Neither country had a clear socioeconomic gradient in hypertension, measured blood pressure ≥140/90 mmHg, elevated blood glucose, or serum total:HDL cholesterol ratio. Lower-income participants in both countries were more likely to be current smokers, to be obese, and to be of shorter stature than higher-income participants. In Australia, lower-income participants were more likely to have a high white blood cell count than higher-income participants.

**Treatments**

At baseline, Australians were more likely to be taking a calcium antagonist, and New Zealanders were more likely to be taking a beta-blocker or ACE inhibitor. The usage of aspirin was similar in both countries. Australians were more likely to be treated with PCI or CABG than New Zealanders. During follow-up in the double-blind phase of the trial, New Zealanders were less likely to discontinue the study medication, while Australians were more likely to receive off-study open-label cholesterol-lowering medication.

In both countries, lower-income participants were less likely to be taking aspirin, but more likely to be taking calcium antagonists. The percentages of participants who discontinued the study medication were similar across all socioeconomic groups. Lower-income participants were less likely to be treated with off-study cholesterol-lowering medication, PCI, or CABG than higher-income participants.
### Table 1. Age, gender, and cardiovascular risk factors stratified by country and by socioeconomic group in Australia and New Zealand

<table>
<thead>
<tr>
<th>Cardiovascular risk factors at baseline</th>
<th>Country of residence</th>
<th>All (%)*</th>
<th>P value</th>
<th>Residential-area income tertile (%)*</th>
<th>P value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lower</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Middle</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Higher</td>
<td></td>
</tr>
<tr>
<td>Mean age (years)*</td>
<td>Australia</td>
<td>61</td>
<td>0.9</td>
<td>61</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>62</td>
<td>0.9</td>
<td>62</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male gender</td>
<td>Australia</td>
<td>84</td>
<td>0.01</td>
<td>81</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>82</td>
<td>0.01</td>
<td>82</td>
<td>0.0002</td>
</tr>
<tr>
<td>History of hypertension</td>
<td>Australia</td>
<td>44</td>
<td>0.9</td>
<td>44</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>37</td>
<td>&lt;0.0001</td>
<td>38</td>
<td>0.3</td>
</tr>
<tr>
<td>Blood pressure ≥140/90 mmHg</td>
<td>Australia</td>
<td>48</td>
<td>0.6</td>
<td>49</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>47</td>
<td>0.6</td>
<td>49</td>
<td>0.2</td>
</tr>
<tr>
<td>Diagnosed diabetes</td>
<td>Australia</td>
<td>9</td>
<td>0.003</td>
<td>10</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>7</td>
<td>0.003</td>
<td>8</td>
<td>0.02</td>
</tr>
<tr>
<td>Fasting glucose &gt;7 mmol/L</td>
<td>Australia</td>
<td>9</td>
<td>0.3</td>
<td>9</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>8</td>
<td>0.5</td>
<td>8</td>
<td>0.2</td>
</tr>
<tr>
<td>Urine protein present</td>
<td>Australia</td>
<td>5</td>
<td>0.5</td>
<td>6</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>5</td>
<td>0.5</td>
<td>6</td>
<td>0.2</td>
</tr>
<tr>
<td>Total:HDL cholesterol ratio &gt;6.1</td>
<td>Australia</td>
<td>48</td>
<td>&lt;0.0001</td>
<td>50</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>53</td>
<td>&lt;0.0001</td>
<td>52</td>
<td>0.9</td>
</tr>
<tr>
<td>HDL cholesterol &lt;1.0 mmol/L</td>
<td>Australia</td>
<td>62</td>
<td>0.01</td>
<td>62</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>62</td>
<td>0.01</td>
<td>62</td>
<td>0.7</td>
</tr>
<tr>
<td>Triglycerides ≥1.6 mmol/L</td>
<td>Australia</td>
<td>51</td>
<td>0.07</td>
<td>52</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>49</td>
<td>0.07</td>
<td>52</td>
<td>0.7</td>
</tr>
<tr>
<td>White blood cell count ≥8.2 × 10⁹/L</td>
<td>Australia</td>
<td>26</td>
<td>0.4</td>
<td>30</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>27</td>
<td>0.4</td>
<td>28</td>
<td>0.4</td>
</tr>
<tr>
<td>Current smoker</td>
<td>Australia</td>
<td>9</td>
<td>0.4</td>
<td>11</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>10</td>
<td>0.4</td>
<td>12</td>
<td>0.001</td>
</tr>
<tr>
<td>Body mass index &gt;30</td>
<td>Australia</td>
<td>18</td>
<td>0.6</td>
<td>20</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>18</td>
<td>0.6</td>
<td>21</td>
<td>0.0005</td>
</tr>
<tr>
<td>Height &lt;166 cm</td>
<td>Australia</td>
<td>24</td>
<td>0.5</td>
<td>27</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>25</td>
<td>0.5</td>
<td>30</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Table shows the percentage of participants (%) with each risk factor, except for age, where the mean age is shown. HDL = high-density lipoprotein.
Table 2. Measures of cardiovascular disease severity, cardiovascular symptoms, and treatments stratified by country and by socioeconomic group in Australia and New Zealand

<table>
<thead>
<tr>
<th>Country of residence</th>
<th>Residential-area income tertile (%)</th>
<th>P value for trend</th>
<th>P value for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Lower</td>
<td>Middle</td>
</tr>
<tr>
<td>Cardiovascular disease at baseline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>Australia</td>
<td>71</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>74</td>
<td>72</td>
</tr>
<tr>
<td>Angina duration &gt;5 years</td>
<td>Australia</td>
<td>13</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>14</td>
<td>16</td>
</tr>
<tr>
<td>Dyspnoea, NYHA class ≥2</td>
<td>Australia</td>
<td>50</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>49</td>
<td>52</td>
</tr>
<tr>
<td>Angina, CCS class ≥2</td>
<td>Australia</td>
<td>35</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>41</td>
<td>43</td>
</tr>
<tr>
<td>Treatments at baseline assessment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>Australia</td>
<td>82</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>83</td>
<td>80</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>Australia</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>17</td>
<td>20</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>Australia</td>
<td>45</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>50</td>
<td>49</td>
</tr>
<tr>
<td>Calcium antagonist</td>
<td>Australia</td>
<td>37</td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>29</td>
<td>30</td>
</tr>
<tr>
<td>Previous PCI*</td>
<td>Australia</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Previous CABG*</td>
<td>Australia</td>
<td>33</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>25</td>
<td>24</td>
</tr>
<tr>
<td>Treatments during follow-up</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Off-study cholesterol treatment</td>
<td>Australia</td>
<td>17</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Discontinued study treatment†</td>
<td>Australia</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>25</td>
<td>26</td>
</tr>
<tr>
<td>PCI during follow up</td>
<td>Australia</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>CABG during follow-up</td>
<td>Australia</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>New Zealand</td>
<td>11</td>
<td>9</td>
</tr>
</tbody>
</table>

*Includes PCI or CABG before or after the qualifying event; †Includes temporary and permanent discontinuation; ACE = angiotensin-converting enzyme; CABG = coronary artery bypass surgery; CCS = Canadian Cardiovascular Society; NYHA = New York Heart Association class; PCI = percutaneous coronary intervention.
Symptoms of cardiovascular disease

Exertional dyspnoea was more common among New Zealanders than among Australians, although dyspnoea classifications were similar in both countries (Table 2). Lower-income groups in both countries were more likely to report angina and dyspnoea, and to have a longer history of angina. There was a small but statistically significant difference between countries in the proportion of subjects with a history of myocardial infarction, but no such difference between socioeconomic groups.

Figure 1. (A) Cardiovascular mortality and (B) non-cardiovascular mortality of Australian and New Zealand LIPID participants in the lowest, middle, and highest tertiles of residential area-based income
Table 3. Multivariate risk factor model for cardiovascular death in all study participants

<table>
<thead>
<tr>
<th>Risk predictor</th>
<th>Hazard ratio</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiovascular risk factors at baseline</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of hypertension</td>
<td>1.14</td>
<td>(1.00–1.30)</td>
<td>0.047</td>
</tr>
<tr>
<td>Fasting glucose &gt;7.0 mmol/L</td>
<td>1.59</td>
<td>(1.33–1.90)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Total:HDL cholesterol ratio*</td>
<td>1.04</td>
<td>(1.02–1.06)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>HDL cholesterol &lt;1.0 mmol/L</td>
<td>1.17</td>
<td>(1.01–1.36)</td>
<td>0.03</td>
</tr>
<tr>
<td>Triglycerides ≥1.6 mmol/L</td>
<td>0.79</td>
<td>(0.69–0.91)</td>
<td>0.001</td>
</tr>
<tr>
<td>Urine protein present</td>
<td>1.50</td>
<td>(1.20–1.87)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>White blood cell count†</td>
<td>1.04</td>
<td>(1.03–1.06)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.71</td>
<td>(1.35–2.17)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Body mass index &gt;30</td>
<td>1.17</td>
<td>(0.99–1.38)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Cardiovascular disease severity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>1.67</td>
<td>(1.40–2.00)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Dyspnoea, NYHA class ≥2</td>
<td>1.24</td>
<td>(1.01–2.02)</td>
<td>0.04</td>
</tr>
<tr>
<td>Angina, CCS class 1</td>
<td>1.25</td>
<td>(1.08–1.44)</td>
<td>0.002</td>
</tr>
<tr>
<td>Angina, CCS class ≥2</td>
<td>1.35</td>
<td>(1.08–1.67)</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Treatments</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>0.85</td>
<td>(0.73–0.99)</td>
<td>0.04</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>1.78</td>
<td>(1.54–2.06)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Beta–blocker</td>
<td>0.84</td>
<td>(0.73–0.96)</td>
<td>0.01</td>
</tr>
<tr>
<td>Randomisation to pravastatin</td>
<td>0.72</td>
<td>(0.63–0.81)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Off-study cholesterol-lowering treatment</td>
<td>0.44</td>
<td>(0.34–0.57)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Temporarily discontinued study treatment</td>
<td>0.71</td>
<td>(0.56–0.92)</td>
<td>0.008</td>
</tr>
<tr>
<td>Permanently discontinued study treatment</td>
<td>1.06</td>
<td>(0.91–1.25)</td>
<td>0.4</td>
</tr>
<tr>
<td>PCI before qualifying event</td>
<td>0.72</td>
<td>(0.49–1.05)</td>
<td>0.09</td>
</tr>
<tr>
<td>PCI between qualifying event and randomisation</td>
<td>0.61</td>
<td>(0.45–0.83)</td>
<td>0.002</td>
</tr>
<tr>
<td>PCI during follow-up</td>
<td>1.66</td>
<td>(1.23–2.23)</td>
<td>0.001</td>
</tr>
<tr>
<td>CABG before qualifying event</td>
<td>1.53</td>
<td>(1.28–1.83)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>CABG between qualifying event and randomisation</td>
<td>0.77</td>
<td>(0.64–0.94)</td>
<td>0.01</td>
</tr>
<tr>
<td>CABG during follow-up</td>
<td>1.40</td>
<td>(1.13–1.72)</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>Country and residential-area income</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Country</td>
<td>1.34</td>
<td>(1.17–1.53)</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Residential-area income decile</td>
<td>1.04</td>
<td>(1.01–1.08)</td>
<td>0.003</td>
</tr>
<tr>
<td>Country-income decile interaction</td>
<td>0.99</td>
<td>(0.94–1.03)</td>
<td>0.5</td>
</tr>
</tbody>
</table>

The model also included terms for age (as a quadratic) and gender. Height, blood pressure, low-density-lipoprotein cholesterol and calcium antagonist treatment at baseline were not significant (P>0.05) in the multivariate model; *The hazard ratio and CI are for a 1-unit increase in the total:HDL cholesterol ratio; †The hazard ratio and CI are for a 1 x 10^9/L increase in the white blood cell count; ACE = angiotensin-converting-enzyme; CABG=coronary artery bypass surgery; CCS Canadian Cardiovascular Society; HDL=high-density-lipoprotein; NYHA = New York Heart Association; PCI = percutaneous coronary intervention.

**Cardiovascular mortality**

Cardiovascular mortality during the LIPID Study was higher in New Zealand than in Australia (age- and sex-adjusted hazard ratio [HR] 1.42, 95%CI 1.25–1.61). The difference in non-cardiovascular mortality between the two countries was smaller and not statistically significant (New Zealand versus Australia HR 1.08, 95%CI 0.90–1.29). There was a definite gradient in cardiovascular mortality according to socioeconomic status in both countries, with mortality being higher in participants from lower-income areas (Figure 1A).
The increase in cardiovascular mortality with a decrease in residential-area income of one tertile was similar in both countries (Australian HR 1.20, 95%CI 1.08–1.32; New Zealand HR 1.16, 95%CI 1.02–1.31). There was also a graded increase in non-cardiovascular mortality with a decrease in residential-area income of one tertile in Australia (HR 1.18, 95%CI 1.04–1.35), but this gradient was not statistically significant in New Zealand (HR 1.04, 95%CI 0.87–1.24; Figure 1B).

There was no evidence that the benefits of pravastatin differed between countries or between socioeconomic groups.

**Multivariate model**

Multivariate predictors of cardiovascular mortality are presented in Table 3. After adjustment for all prognostic variables, the excess risk of cardiovascular death in New Zealand compared with Australia was reduced from 42% (with the analysis adjusted only for age and sex) to 35%. The difference in cardiovascular mortality between the two countries was present at all income levels, with a slightly greater difference in the higher-income deciles.

In Australia, the risk of cardiovascular death increased by 58% from the highest to the lowest income decile after adjustment for age and sex, and by 46% after adjustment for all baseline prognostic factors. In New Zealand, the risk of cardiovascular death increased by 45% from the highest to the lowest income decile after adjustment for age and sex, and by 35% after adjustment for all baseline prognostic factors.

**Discussion**

In this study, which recruited representative coronary heart disease patients, mortality in both Australia and New Zealand increased progressively as the median residential-area income decreased. However, while non-cardiovascular mortality was similar in both countries, cardiovascular mortality was about 40% higher in New Zealand across all socioeconomic groups. This excess was not explained by coronary heart disease risk factors associated with socioeconomic status, or by greater socioeconomic disparities in measures of disease severity, treatments or income in New Zealand. There were treatment differences between countries across all socioeconomic groups, but the extent of their influence upon the differences in cardiovascular mortality observed in the study is uncertain.

Off-study open-label cholesterol-lowering drug treatments, which were relatively expensive during the 1990s, were more widely used by higher-income participants than lower-income participants during the double-blind phase of the trial, and more widely prescribed in Australia than in New Zealand. Government policy to restrict access to statins in New Zealand at this time may have contributed to these differences.

The rates of PCI and CABG were lower in New Zealand than in Australia, and lower in lower socioeconomic groups within both countries. This was despite a greater prevalence of angina in New Zealand and in lower-income participants. However the impact of different rates of revascularization on outcomes is difficult to evaluate in this study. In addition accurate information on indications for PCI/CABG and LV ejection fraction is not available.
Clinical trials suggest coronary revascularisation does not decrease mortality for most patients with stable coronary artery disease, but PCI early after an acute coronary syndrome decreases the risk of recurrent myocardial infarction.

The average serum total:HDL cholesterol ratio was higher in New Zealand than in Australia. The explanation for this is unclear, but may reflect differences in the national diet, genetic factors or selection of participants for the trial. There was no socioeconomic gradient in serum lipid levels.

Although Australians were more likely to have received a diagnosis of hypertension or diabetes, measured blood pressure and blood glucose levels were similar in both countries. This may suggest that the diagnoses of hypertension and diabetes were made earlier in Australia. There were no clear international differences in other cardiovascular risk factors.

In contrast to international differences, there was no socioeconomic gradient in serum lipid levels or hypertension, but obesity and smoking were more prevalent in lower socioeconomic groups. These lifestyle risk factors probably reflect health behaviours that are established by young adulthood and continue to influence disease rates for many years.

The socioeconomic gradient in the white blood cell count, which was more evident in Australia, suggests that inflammation may be more prevalent in individuals from lower-income areas. The association between the white blood cell count and cardiovascular mortality in this study is consistent with previous studies in which non-specific markers of systemic inflammation were associated with an increased risk of first or recurrent cardiovascular events.

Previous analyses suggest that multiple factors have contributed to the decline in coronary heart disease mortality both in New Zealand and internationally during the 1980s and 1990s. In a recent analysis from the US, about half the decline in coronary heart disease mortality, was attributable to changes in risk factors and about half to medical treatments.

In contrast, in the current study, measured variables appear to explain only a small proportion of the international and socioeconomic differences in cardiovascular event rates in the multivariate analysis. There are several possible explanations. A number of important risk factors were not measured including diet, exercise, psychosocial variables and many aspects of medical care. Categorisation of risk factors as “present” or “absent” will under estimate the graded association between most risk factors and events. Finally, multivariate analysis may not appropriately adjust for effects of treatments which are known from randomized clinical trails to improve outcomes.

The study population was selected by agreement to participate in a clinical trial. The socioeconomic distribution of study participants was similar to that of the general population in both Australia and New Zealand. However, certain groups may have been under-represented, and we have no data on ethnicity. It is also possible there were differences in selections of patients invited to participate in the study between countries. For example New Zealand participants were more likely on average to have a history of angina, suggesting they may have more severe coronary disease at baseline.
Participation in the LIPID Study may also have influenced standards of care, and it is possible that the magnitude of treatment differences was greater or smaller than in routine medical practice. The proportion of current smokers was lower than in the general population, but the proportion of ex-smokers was higher, reflecting the impact of the diagnosis of coronary artery disease on smoking behaviour.

Census-based measures of socioeconomic status have been consistently associated with health outcomes in many studies.\textsuperscript{4,22,23} However, because income is heterogeneous within areas, an aggregate measure is a crude estimate of individual income, which would underestimate any socioeconomic gradient related to personal income.

Residential-area-based measures do, however, include information on other health effects associated with the residential area.\textsuperscript{24,25} They also have the advantage of being determined by long-term socioeconomic status, and may therefore be more reliable than current income when assessing health over many years in a study involving participants who may be working, retired or unemployed at the time of assessment. It is difficult to adequately account for international differences in buying power. For this reason, relative differences between residential areas and between countries were used for the analysis.

In conclusion, this study suggests that there are differences in the risk factors that contribute to the international and socioeconomic gradients in cardiovascular mortality in Australia and New Zealand. This suggests that specific strategies are needed to reduce socioeconomic gradients in cardiovascular mortality in Australia and New Zealand, and these may differ from those designed to reduce national cardiovascular mortality rates.

\textbf{Competing interests:} None of the authors has any conflict of interest in connection with this work.

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4. Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia.

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analysis and interpretation of this substudy were conducted independently of the sponsor.

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References:

Gaps in primary care documentation of cardiovascular risk factors

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

Abstract

Background New Zealand guidelines recommend that cardiovascular risk management should be informed by the absolute risk of a cardiovascular event. This requires knowledge of a person’s age, sex, ethnicity, medical and family history, blood pressure, total and HDL cholesterol, diabetes, and smoking status.

Aim To establish the extent of primary care documentation of cardiovascular risk factors.

Methods An audit of electronic patient records was conducted in practices affiliated with an Auckland primary care organisation (ProCare Health Ltd). The audited population were patients eligible for risk assessment (all Māori and a random sample of non-Māori) who had a consultation with their general practitioner during a four week study period (1 year before the doctor first used cardiovascular electronic clinical decision support software). Audit nurses searched for risk factors documented prior to the study period.

Results The records of 1680 individuals from 84 doctors were audited. The study periods prior to which the records were inspected ranged from August 2001 to June 2003. The proportions of records with risk factors documented were: blood pressure 81.8%, cholesterol 62.4%, smoking status 41.5%, diabetes status 16.1%, all these risk factors 6.8%. Recording of blood pressure and of cholesterol was higher in those with cardiovascular disease or diabetes. Recording of blood pressure increased with increasing age, then levelled off at about age 60 years. Documentation of cholesterol was lowest in the oldest and youngest age groups, and in women (at all ages) compared to men.

Conclusions Primary care documentation of cardiovascular risk factors was incomplete. Whilst many doctors may know whether patients are smokers or have diabetes, systematic documentation of these factors in particular, is not occurring. In order to realise the large potential benefits associated with population-based cardiovascular risk assessment and management, a substantial investment by government, healthcare organisations, health professionals, and patients is required to collect and record this information.

In New Zealand, for over a decade, cardiovascular guidelines have recommended that risk management decisions be based primarily on a person's absolute risk of having a cardiovascular event. Determination of a person's cardiovascular risk requires knowledge of their age, sex, ethnicity, medical and family history, blood pressure, total and high density lipoprotein (HDL) cholesterol, diabetes, and smoking status.
Intensive lifestyle and pharmaceutical-based treatment is recommended if a person is identified as being at high risk (defined as a 15% or greater probability of having a fatal or nonfatal cardiovascular event in the next 5 years according to the Framingham risk prediction equation).  

Cardiovascular events could be reduced by up to 50% in high risk individuals who are managed appropriately.  

In light of these benefits, cardiovascular risk assessment and management criteria have been proposed for primary health organisation (PHO) performance indicators.  

The objective of this paper is to describe the baseline level of documentation of cardiovascular risk factors (blood pressure, cholesterol, diabetes, and smoking status) in electronic patient medical records in a sample of Auckland general practices prior to the implementation of cardiovascular electronic clinical decision support (ECDS) software.

**Methods**

This research is part of a larger ‘before-after’ study investigating the impact of cardiovascular ECDS software on cardiovascular risk assessment and documentation.  

General practitioners (GPs) invited to participate were members of ProCare Health Ltd, had used electronic patient records for at least a year, and had PREDICT<sup>TM</sup> ECDS integrated into their practice management system.  

Electronic queries were run on the practice management system to find patients registered with the participating doctor who were eligible for cardiovascular risk assessment under the New Zealand cardiovascular risk guidelines’ criteria (Māori/Pacific/Indian subcontinent men aged 35 years and over; Māori/Pacific/Indian subcontinent women aged 45 years and over; and 10 years later respectively for all other ethnic groups), who had consulted their doctor during a four week study period 1 year prior to the doctor’s first use of the ECDS software.  

As the implementation of the ECDS was in several stages (starting in August 2002 and continuing to June 2004), the study periods ranged from August 2001 to June 2003.  

The final audit list consisted of all the eligible Māori patients and a randomly selected 15% sample of the eligible non-Māori patients. All Māori patients were included to enhance statistical power as obtaining adequate explanatory power for Māori was an objective of the main study.  

Patients whose ethnicity was documented as New Zealand Māori or equivalent (Māori, M, or New Zealand Health Information Service ethnicity level 2 code 21) in the electronic medical record were included as Māori. Where no ethnicity was recorded, patients were assumed to be non-Māori.  

For each patient, audit nurses manually inspected 2 years of electronic records prior to their entry into the study. The following cardiovascular risk factors were audited:

- Blood pressure (defined as documented if there was a recorded systolic and diastolic measurement anywhere in the patient medical record; otherwise not documented).
- Cholesterol (defined as documented if there was a recorded total/HDL cholesterol ratio, or total cholesterol if there was no ratio, anywhere in the patient medical record; otherwise not documented).
- Smoking status (defined as documented if there was a statement anywhere in the patient medical record or diagnostic code indicating the patient is a current smoker, non-smoker, or past smoker [quit smoking for over 12 months]; otherwise not documented).
- Diabetes status (defined as documented if there was a statement anywhere in the patient medical record or diagnostic code indicating the patient has diabetes or does not have diabetes; otherwise not documented).

In addition, the electronic records were searched for evidence of a history of cardiovascular disease or diabetes at any time prior to the audit date. Cardiovascular disease was defined as present if any of the following conditions were recorded—which include heart disease, myocardial infarction, angina, coronary artery bypass graft (CABG) surgery, angioplasty or other coronary revascularisation procedure,
ischaemic stroke (not haemorrhagic stroke), transient ischaemic attack (TIA), claudication, peripheral vascular disease—or if there was more than one prescription for oral or transdermal nitrates.

Diabetes was defined as present if there was a statement to that effect or evidence of prescriptions for oral hypoglycaemic agents, insulin or test strips, or a glycosylated haemoglobin (HbA1c) result above 6%.

To assess factors associated with the recording of blood pressure and cholesterol a mixed logistic regression model was used. In the model, practices and GPs were regarded as random effects and all other variables as fixed effects.

The following patient characteristics were included in the model: age group, sex, ethnicity (Māori or non-Māori), current smoker, the presence of existing cardiovascular disease or diabetes, and holding a High Use Health Card (government subsidy for those with medical conditions requiring frequent GP visits) or Community Services Card (government subsidy for lower income families).

Statistical analyses were conducted using SAS statistical software (version 9.1).

The PREDICT-CVD Evaluation Study was approved by the Auckland Regional Ethics Committee (AKY/04/07/185).

**Results**

Eighty-four out of 107 (78.5%) eligible doctors consented to be in the study. Consent was not obtained from 5 doctors who were unable to be contacted, and 18 declined to participate.

The electronic medical records of 1680 individuals were audited; their demographic characteristics are described in Table 1.

**Table 1. Patient demographics**

<table>
<thead>
<tr>
<th>Variable</th>
<th>N = 1680</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age group (years)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35–44</td>
<td></td>
<td>107</td>
<td>6.4</td>
</tr>
<tr>
<td>45–54</td>
<td></td>
<td>366</td>
<td>21.8</td>
</tr>
<tr>
<td>55–64</td>
<td></td>
<td>488</td>
<td>29.0</td>
</tr>
<tr>
<td>65–74</td>
<td></td>
<td>377</td>
<td>22.4</td>
</tr>
<tr>
<td>75–84</td>
<td></td>
<td>269</td>
<td>16.0</td>
</tr>
<tr>
<td>≥85</td>
<td></td>
<td>73</td>
<td>4.3</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>827</td>
<td>49.2</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>853</td>
<td>50.8</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Māori</td>
<td></td>
<td>474</td>
<td>28.2</td>
</tr>
<tr>
<td>Non-Māori</td>
<td></td>
<td>1206</td>
<td>71.8</td>
</tr>
<tr>
<td><strong>High Use Health Card status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No HUHC</td>
<td></td>
<td>1519</td>
<td>90.4</td>
</tr>
<tr>
<td>HUHC</td>
<td></td>
<td>161</td>
<td>9.6</td>
</tr>
<tr>
<td><strong>Community Services Card status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No CSC</td>
<td></td>
<td>919</td>
<td>54.7</td>
</tr>
<tr>
<td>CSC</td>
<td></td>
<td>761</td>
<td>45.3</td>
</tr>
</tbody>
</table>

Of those audited, one in five patients (19.5%) had evidence of cardiovascular disease and 14.7% had evidence of diabetes. Blood pressure was documented in 81.8% of
electronic notes, cholesterol in 62.4%, and diabetes status in 16.1% (this included where diabetes was documented as not present).

Smoking status was documented in 41.5%; the coding of smoking and factors that were associated with its documentation are described in more detail elsewhere. Of those without a cardiovascular history, only 6.8% (92/1353) had documentation of all the required cardiovascular risk factors necessary to calculate a 5-year cardiovascular risk score.

The proportion of patients with blood pressure and cholesterol recorded according to the presence or absence of cardiovascular disease and diabetes is shown in Figure 1. The 95% confidence intervals of these proportions are indicated.

**Figure 1. Influence of presence of cardiovascular disease (CVD) and diabetes on recording of blood pressure and cholesterol (proportions and 95% confidence levels; associated p values from the multivariate model are included)**

In the multivariate model, age, cardiovascular disease, and diabetes were significantly related to the documentation of blood pressure. Documentation increased steeply with increasing age up to about 60 years, but this increase diminished among the older age groups (Figure 2).

The recording of cholesterol varied significantly with age, cardiovascular disease, diabetes, and sex. A greater proportion of males than females had their cholesterol
levels recorded (69% of males compared to 60% of females overall). Documentation was highest in those aged between about 50 and 70 years, but fell off steeply in both older and younger people (Figure 2).

**Figure 2. Relationship of cholesterol and blood pressure recording with age and sex using the predicted values from the multivariate model**

There were no differences in the recording of blood pressure or cholesterol by recorded smoking status.

**Discussion**

In this sample of people eligible for cardiovascular risk assessment, who had visited their GP over a specified 1-month period between August 2001 and June 2003, the documentation of cardiovascular risk factors necessary for risk assessment ranged from 16% (diabetes status) to over 80% (blood pressure).

Whilst many GPs may know the smoking and diabetes status of their patients, this research demonstrates major gaps in the completeness of documentation of risk factors; although GPs are achieving high levels of documentation of blood pressure and cholesterol in patients with cardiovascular disease and diabetes.
Direct comparison with other studies examining documentation is difficult because recording of risk factors is likely to be affected by the population under study and the healthcare system environment, and the method of data extraction may also influence the accuracy of the assessment. However, audits in the United Kingdom have reported similar patterns of low levels of cholesterol documentation particularly in women and older patients.9–11

Several studies of cardiovascular risk factor documentation in New Zealand primary care medical records have been published. Lower rates than the current study were reported in 2000, when 64% of men aged at least 45 years and women at least 55 years had systolic blood pressure recorded and 28% had cholesterol recorded—based on electronic data extraction from 25,384 individuals whose medical records were included in the Dunedin Royal New Zealand College of General Practitioners Research Unit database.12

Similarly, slightly lower rates than ours were found by the Bold Promise Project audit in 2004 of 180 clinical records from three primary care practices in South Auckland and Hawke’s Bay. The Project authors estimated that among the enrolled primary care population who were eligible for cardiovascular screening, 52% had a lipid profile recorded, 72% had a blood pressure, and only 36% had smoking status documented.13

In 2003, diabetes audit nurses found 83% of 5917 primary care patients with diabetes in South and West Auckland had their cholesterol ratio documented, with systolic blood pressure recorded in 94% and smoking status in 81%; results which are very similar to our findings.14

As with any medical records-based research, there are limitations. General practices in this study are members of one large urban primary care organisation and were the ones who agreed to have the ECDS tool integrated in the practice, so may not be representative of all general practice. However the strengths of the study are the relatively large number of doctors included, good response rate, and the large number of patient medical records manually searched by experienced audit nurses.

This paper presents an estimate of the level of cardiovascular risk factor documentation in the electronic records of New Zealand primary care patients prior to the publication of The Assessment and Management of Cardiovascular Risk guideline by the New Zealand Guidelines Group in late 2003.3 The findings indicate there were major gaps and variability (by medical history, age and sex) in recording of cardiovascular risk factors.

New strategies are required to improve documentation and increase the ability of primary care to implement the cardiovascular risk guideline.3 Whilst the recording of risk factors or risk may not in itself always lead to improved clinical management of an individual, a controlled trial in patients with diabetes has demonstrated that documentation of cardiovascular risk increases appropriate prescription of preventive therapy and a Canadian study showed greater cholesterol reduction when patients were regularly informed of their cardiovascular risk profile.15,16
Lomas provides a framework which can be used to translate research into interventions to enhance systematic risk factor documentation. It focuses on four strategic areas of influence on clinical practice and these are presented below with examples of New Zealand cardiovascular risk initiatives:

- **Patient-centred approaches**—Social marketing to consumers to increase awareness about cardiovascular risk. The *One Heart, Many Lives* social marketing campaign undertaken by PHARMAC between 2003 and 2004 targeted Māori and Pacific men aged 35 and over and raised awareness of heart disease using a theme of positive roles within whānau (family).

- **Practitioner-centred approaches**—Education of primary care teams in the value of systematic risk factor documentation and provision of training in the use of computer software for risk factor documentation. Nurses in Northland and Auckland trained in the use of ECDS software now run cardiovascular risk clinics in a variety of urban and rural settings. The implementation of PREDICT™ ECDS in Auckland which included training of GPs through regular continuing medical education meetings, showed a four to five fold increase in cardiovascular risk documentation in Māori and non-Māori.

- **Administrative approaches**—The development of an information culture in primary care which acknowledges the time and effort necessary to change behaviour, and of systems which support systematic documentation, preferably within chronic care management programmes. For example, standardised and user friendly coding tools, automatic prompts, performance indicators, regular feedback, and audits to evaluate programme success.

Systems changes and audit were integral to the Bold Promise Project and included pop-up alerts, recalls, use of a standardised screening template, simplified Read coding, and reporting on clinical performance indicators. A Masterton primary care initiative incorporated similar system changes and information technology tools which tracked progress at the practice population level; within 3 years ninety percent of the population eligible for risk assessment had a calculated cardiovascular risk.

Such systematic processes are consistent with the Royal College of General Practitioners’ Cornerstone practice accreditation programme. Indeed their *Aiming for Excellence* framework recommends practices have systems to update smoking status and participate in population screening programmes.

- **Economic approaches**—Incentives that recognise the resources required to collect and record risk factor information. Cardiovascular risk assessment is one of the priority indicators in the New Zealand Ministry of Health’s *Diabetes and Cardiovascular Disease Quality Improvement Plan*; DHBNZ’s PHO performance indicator programme is likely to provide incentive payments for the completion of risk assessments in 2008.

In conclusion, we have demonstrated that significant gaps existed in the documentation of cardiovascular risk factors in primary care patients eligible for...
cardiovascular risk assessment prior to the publication of the most recent national cardiovascular risk assessment and management guidelines at the end of 2003. While several initiatives have been implemented since then to address the problem, these have not been introduced systematically or countrywide. It is likely that a range of strategies will be required to achieve systematic population-based risk factor recording and cardiovascular risk assessment in the eligible population.

It will be imperative that practices, PHOs, and district health boards (DHBs) develop the capability to accurately audit practice to ensure that any strategies introduced are effective.

Competing interests: None known.

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References:


2. The management of raised blood pressure in New Zealand. A consensus development conference report to the National Advisory Committee on Core Health and Disability Support


Survey of clinical echocardiography in New Zealand (SCANZ)

Paul G Bridgman, Akbar N Ashrafi, Stewart Mann, Gillian A Whalley; on behalf of the SCANZ collaborators

Abstract

Aims In New Zealand, we have neither guidelines nor data regarding the provision of echocardiography and disparities between regional echocardiography are believed to exist. The purpose of this study was to provide a cross-sectional snapshot of clinical use of echocardiography within New Zealand (NZ).

Methods Over a 1-week period (5/12/2005–11/12/2005) echocardiography laboratories around NZ (tertiary, secondary hospitals, and private practices) sent copies of their echo reports and referral forms (with patient identifiers removed) to a central site. Demographic information, clinical indication, measurements performed, and interpretation were collated, recorded, reviewed, tabulated, and entered into a Microsoft Access database.

Results 1498 echoes were performed, 92% were transthoracic examinations. Adult examinations comprised 83% of the echocardiograms performed: median age was 61.7 years (interquartile range 47.3 to 74.1) and 56% were male. The three most common primary clinical indications were: left ventricular (LV) function (43%), valve disease (14%), and murmur (7.5%). Seventy-five percent reported abnormal findings. There was wide disparity in the population adjusted rates of echoes performed across NZ’s district health boards.

Conclusion This prospective survey provides a contemporary overview of echocardiography in NZ and highlights the inherent geographical disparity in echocardiography utilisation throughout the country.

Echocardiography plays a pivotal role in modern cardiology and is an invaluable tool for the evaluation of cardiac structure, function, and haemodynamics. Its application extends beyond diagnosis to prognosis, monitoring, and management of patients with various heart conditions such as congestive heart failure, valvular heart disease, ischaemic heart disease, congenital heart disease, pulmonary disease and arrhythmias. Echocardiography is second only to electrocardiography (ECG) as the most frequently utilised cardiac investigative tool.¹

New Zealand is geographically small, yet there remains diversity in both hospital services and utilisation of cardiac services. A recent study of New Zealand clinical practice related to the management of acute coronary syndrome (ACS)² revealed wide disparities between centres in terms of management, available technologies, and utilisation of tests.

This study highlighted that only 30% of patients with ACS underwent echocardiography, despite its proven diagnostic and prognostic benefits in such
patients. However, there are no data available to determine how echocardiography is currently utilised in wider clinical practice in New Zealand.

Although echocardiography services are spread throughout the country, and available in all major and most smaller centres, there is a perception and some anecdotal evidence that there are differences in the provision of echocardiography between centres. While guidelines for the clinical application of echocardiography exist elsewhere, New Zealand and Australia have not developed any such strategies.

The aim of this study was to prospectively investigate (over a 1-week period) all echocardiograms performed within New Zealand, with particular emphasis on service provision, patient demographics, referral indication, and technology utilised, thus providing a snapshot of the overall clinical utilisation patterns of echocardiography within New Zealand. This will be an important and essential first step towards the development of New Zealand echocardiography services.

**Methods**

**Data collection**—All echocardiography laboratories around New Zealand were asked to record their echocardiograms over a designated one week period (05/12/2005–11/12/2005). As an aid to ensure full data capture each centre was provided with a single page table for the week on which they tabulated the number of echocardiograms performed at their site each day. They then provided a photocopy of each echocardiography report and the associated referral form with patient identifiers deleted. These forms were then reviewed, tabulated and coded for entry into a database at the Department of Medicine, The University of Auckland. The National Ethics Committee approved this as a clinical audit.

A protocol was set up prior to the study to review all echocardiography referrals and reports. Briefly, this protocol included date, location, demographics, type of echo, referral details, reporting characteristics, echocardiogram examination details, interpretations and measurements for various echocardiography parameters such as ejection fraction and left ventricular (LV) size.

A clinical record form was carefully created to incorporate all relevant data from the report review protocol. All centres were coded for easy identification and follow-up and were categorised according to their District Health Boards (DHBs). Private practices were coded as a single separate category and were not individually identified in the analysis.

**Statistical analysis**—A database was created to transfer the data on paper into an electronic format. During this time the data entry was rechecked and cross referenced. Each hospital’s data was kept separate and merged when required using the statistical analysis package SAS, which was also used for all analysis. Statistics where considered significant if p<0.05. Regional comparison between DHBs was population-standardised using DHB-based census statistics from Statistic New Zealand (accessed February 2006) expressed per 10,000 people served by that DHB.

**Results**

All public services were included, however three smaller centres in two DHBs performed no echocardiograms that week. Overall, 1498 echocardiograms were performed across 35 hospital sites, comprising 23 public hospitals and 14 private services (Table 1). Private echocardiography services performed 318 (21%) of the echoes.
Table 1. Number of echocardiograms by echocardiography centre

<table>
<thead>
<tr>
<th>Echocardiography centre</th>
<th>Echocardiograms number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All public centres</td>
<td>1180 (78.8%)</td>
</tr>
<tr>
<td>All private centres</td>
<td>318 (21.2%)</td>
</tr>
<tr>
<td>Total</td>
<td>1498</td>
</tr>
</tbody>
</table>

Transthoracic echocardiography (TTE) was the most common type of procedure (92.3%), stress echocardiography (dobutamine and exercise) accounted for 5.2% and transoesophageal echocardiography (TOE) accounted for 2.5%.

Demographics

A total of 1234 (83%) of echoes were performed on adult patients (defined as age over 18 years at the time of the echocardiogram). Age was not recorded in 11 patients. Fifty-six percent were male (gender was unknown in 63 patients) and the median age was 61.7 (interquartile range 47.3–74.1) years. Age distribution was similar for males and females, with the majority being 50–90 years (Figure 1). There were more males than females in all age groups except for the elderly (>80 years).

Figure 1. Age distribution of patients undergoing echocardiograms by gender

Age Distribution by Gender

Black bars = male; White bars = female.
District Health Board comparison

The average number of procedures adjusted for population was 2.28±1.62 echoes per 10,000 people served by the DHB. Significant differences across the DHBs were observed, but no differences were seen when comparing tertiary surgical centres with other DHBs (tertiary 2.24±1.57, other 2.28±1.30) (Figure 2). There was a 2–3 fold difference in the population-based numbers of echoes performed overall and within each category of centres.

Figure 2. Number of echocardiograms performed per week per 10,000 population served by District Health Boards

Echocardiography referral patterns

Outpatients comprised 46% of echocardiograms performed, compared with inpatients, who only made up 5.2% (this was unknown in 29.6% patients). Thirty-six percent were clearly identified as first echocardiograms, 32% as follow-up or repeat studies (32% unknown). Most of the referrals were made by cardiologists (53%) or other specialist physicians (37%) with a relatively small proportion of echocardiography referrals coming from general practitioners (GPs) (7.5%).

Amongst the adult patients, the most common primary indication for echocardiography was assessment of left ventricular function (43%), followed by valve disease (13.5%) and evaluation of murmur (7.5%) (Table 2).
Table 2. Primary referral indication for adult patients

<table>
<thead>
<tr>
<th>Primary indication</th>
<th>Number of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessment of LV function</td>
<td>532 (43.1%)</td>
</tr>
<tr>
<td>Valve disease</td>
<td>167 (13.5%)</td>
</tr>
<tr>
<td>Evaluation or murmur</td>
<td>100 (7.5%)</td>
</tr>
<tr>
<td>Arrhythmias:</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>68 (5.5%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>42 (3.4%)</td>
</tr>
<tr>
<td>Other</td>
<td>26 (2.1%)</td>
</tr>
<tr>
<td>Preoperative assessment</td>
<td>55 (4.5%)</td>
</tr>
<tr>
<td>Source of emboli</td>
<td>52 (4.2%)</td>
</tr>
<tr>
<td>Suspected endocarditis</td>
<td>38 (3.1%)</td>
</tr>
<tr>
<td>Palpitations</td>
<td>21 (1.7%)</td>
</tr>
<tr>
<td>Pericardial or pleural effusion</td>
<td>20 (1.5%)</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>14 (1.1%)</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>14 (1.1%)</td>
</tr>
</tbody>
</table>

Implementation of echocardiography

Sonographers or technologists performed the majority of examinations (88.2%); most of these were transthoracic echoes (Table 3).

Table 3. Patterns in reporting and performing of the echocardiogram examination in New Zealand

<table>
<thead>
<tr>
<th>Health professional</th>
<th>Performed by (%)</th>
<th>Reported by (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiologist</td>
<td>8.2</td>
<td>57.5</td>
</tr>
<tr>
<td>Physician</td>
<td>1.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Registrar</td>
<td>1.2</td>
<td>0.8</td>
</tr>
<tr>
<td>Technician/Sonographer</td>
<td>88.2</td>
<td>39.1</td>
</tr>
<tr>
<td>Not Known</td>
<td>1.1</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Of the echoes performed by cardiologists (8.2%), most were dobutamine stress echoes or transoesophageal echoes. The reporting of echoes was not as clear cut. Evaluating the echo reports that had been released to the referring physician or placed in patient notes, cardiologists reported a little over half of all the echoes performed and sonographers reported about 40%.

Only 26% of the final echo reports (65% of those reported by sonographers) were marked “preliminary—not reviewed by a cardiologist”, thus of the 74% echo reports that could be considered final, a significant number were generated by the sonographer. In some centres, all or nearly all reports were sonographer generated, whereas in other centres there was 100% physician reporting.

Discussion

This prospective survey provides an important snapshot of the provision of echocardiography services around New Zealand. The high participation rate and the number of echo reports captured provides a near complete sample from which we can
draw some robust conclusions about the utilisation of clinical echocardiography in New Zealand.

Not surprisingly, most of the patients were between 40 and 80 years old. There were more males than females in every age group up until 80 years, after which females outnumbered males (in the 80–90 and >90 age groups). This is attributed to longer life expectancy of females in New Zealand. A larger proportion of males was observed in the younger age groups and may reflect the higher incidence of some congenital heart abnormalities in males.

Echocardiography has been practiced in New Zealand for over 30 years. In other Western countries, the echocardiographic examination has traditionally been performed collaboratively by both physicians and sonographers or technologists. The sonographer has been responsible for the technical aspect of the examination (the production of images with some interpretation), while the physician (typically a cardiologist) has taken the role of providing a final review of the data before generating a definitive final report.

The American Society of Echocardiography supports this division of responsibility “Although the echocardiographer (cardiac sonographer) must be knowledgeable about the various cardiac abnormalities in order to produce diagnostic echocardiograms, he/she should not be placed in the position of rendering clinical interpretation of results.”

The majority of reports in this audit were reviewed and signed off by cardiologists. Interestingly, of the 39% that were interpreted and reported solely by a sonographer, only 65% included a proviso to indicate that this was a preliminary report (i.e. not reviewed by a cardiologist). Sonographers are thus interpreting images and preparing “final” reports. This may be partly due to the lack of specialist personnel and is not unique to New Zealand.

Acceptance of sonographers reporting echocardiographic examinations and the implementation of such reports warrants consideration as we further develop the clinical echocardiography service in New Zealand. The scarcity of resources, especially in rural areas, may have a role in influencing the role of different personnel. This ‘non-traditional’ approach to ultrasonic interpretation has been suggested in the UK and is being considered in the USA with the development of higher level ultrasound practitioner roles.

Deficiencies in physician ultrasound training and latency in physician interpretation are important shortcomings of the traditional echocardiography practice. However, some GPs believe that physician reporting is important to maintain the highest standards of quality and may be increasingly important if echocardiography were to be broadened to encompass GP referral.

Under current fiscal conditions it may not be possible to achieve 100% physician reporting. However, the ready availability of digital archiving and remote review should facilitate cardiologist support and reviews for even the most geographically isolated cardiac sonographers.

Very few referrals currently arise from general practice and this may reflect the fact that GPs do not have open access to publicly-funded echocardiography services in
New Zealand. In the majority of cases, a cardiologist acts as the ‘gate-keeper’ to echo—patients are generally referred to a cardiologist who then requests an echocardiogram to be performed. Referrals for echocardiography have risen dramatically over the last two decades in other countries. In this modern era of medical cost-effectiveness, and given echocardiography is not an inexpensive utility, this gate-keeping strategy is considered important to keep echo referrals in check. It also allows prioritisation of patients according to clinical need.

In contrast, recent data from the USA reveals a very different referral pattern with only 29% of echocardiography referrals generated by cardiologists and 20% from general practice. Many patients may not need a specialist referral and open access echocardiography has been suggested as a cost-saving exercise in these patients with the additional benefit of expediting the process for patients. Open-access echocardiography may play an expanding role in the future and may be particularly important for the timely diagnosis of heart failure.

Perhaps the most surprising finding from this survey is the degree of disparity between service utilisation around the country. The data suggest that significant regional disparity exists in terms of the rate of utilisation of echocardiography across District Health Boards (DHBs) on a population basis. Interestingly, there was almost a three-fold difference between the DHBs performing the most echocardiograms and those performing the least number of echocardiograms.

We have not identified individual Health Boards, but have separated the tertiary referral centres from the others for two reasons. Firstly, these centres are the surgical referral centres for their regions and thus manage patients from outside of their DHB catchment area. Secondly, these centres might be anticipated to have a more complex and demanding caseload, which might necessitate more examinations. In the recent ACS audit, echocardiography was more commonly performed in the larger intervention centre.

Similarly, it might be expected that these centres undertake more echocardiograms, but it is apparent that there is considerable disparity between the five surgical centres. Importantly, the mean number of echoes performed did not vary between the tertiary and non-tertiary DHBs and the variation observed within each group of DHBs was larger than between them as a group.

This survey suggests that the provision of service may be different across the different DHBs. These differences are large and cannot be driven by clinical patterns alone. It may be that in some DHB catchment areas there is higher volume access to private services and thus the public utilisation is lower. This trend could not be assessed in this survey as the private providers take patients from different DHB catchment areas and this cross-flow could not be evaluated.

The causes of these overt differences are difficult to speculate but it is likely that a number of factors are involved. These factors may include the availability of resources, the scarcity of personnel, physical accessibility, cost to the hospital, and compliance issues. In a recently published study of echocardiographic utilisation within the Medicare system in the USA, a 3-fold difference was observed between states. This difference was related to both the number of available cardiologists and the prevalence of heart failure. The magnitude of disparity was similar to that
observed in this NZ audit, but perhaps more surprisingly the regional differences in New Zealand were also observed between the tertiary hospitals.

Regional differences in the management of patients with acute coronary syndromes (ACS) have already been reported in a national audit.\(^2\) Furthermore, that audit demonstrated that echocardiography may be underutilised in ACS patients.\(^2\)

In this survey, 75% of the echo reports demonstrated significant abnormalities suggesting that patient referrals are likely to be clinically appropriate. Whether this truly reflects appropriate referral to the service remains unknown, as most certainly some of these abnormal echoes will be repeat examinations and determining the clinical appropriateness of these echoes was beyond the scope of this survey. However, the number of echoes with no abnormalities detected may be seen as low since one of the main indications of clinical echocardiography is to assess the origin of murmurs.

In many cases these are benign flow murmurs and the echoes are essentially normal and thus if these patients comprised a large proportion of the caseload, a higher rate of normal echo reports might be anticipated.

New Zealand is a small country with limited health resources. These resources are subject to many pressures from different areas of medicine and healthcare. Cardiovascular disease remains the leading cause of death for New Zealand adults, accounting for 40% of all deaths. This is unlikely to change due to the growing number of older people, and incidence of risk factors such as diabetes and this needs to well managed. Careful consideration of how clinically proven tests are utilised will form an important step towards optimising service provision.

**Limitations**

The approach to data collection was minimalist in order to maximise the inclusion of centres, by minimising staff time commitment. As a result, the data collected is limited but the near complete nationwide participation has produced a robust dataset. Whilst it may not be entirely accurate to extrapolate from a single week, we believed it was crucial to have all centres recording the same time period and to minimise the impact on their workflow. This may have led to some minor differences such as centres performing fewer or more examinations that week due to external reasons (e.g., staff leave, visiting specialists). However, such anomalies may not have been avoided by extending the survey and further sampling would have proven logistically difficult. We may not have achieved complete data collection at all sites.

Although the reporting of echocardiograms was a two-stage process, we did not perform further checks with the sites. Each site was provided with a single page table to record total numbers on for cross-checking with final returns. We did not separately audit the completeness of data from each site.

This survey does not address is the clinical consequences of the current application of echocardiography services in New Zealand. Future studies could be undertaken to evaluate the impact of echocardiography—such as changing clinician’s management based on the echocardiogram report, whether it be physician or sonographer based.
Conclusions

This first-ever national survey of current clinical echocardiography practice provides a snapshot of the service. It provides important information of the age-specific utilisation rates, the main clinical indications and referral patterns. Importantly, it has highlighted significant regional differences in the utilisation rates of echocardiography around New Zealand. Echocardiography is widely applied in the management and investigation of cardiac patients. This survey will provide a platform for further development of the service.

Competing interests: None known.

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*See appendix

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* indicates principal collaborator
Comparison of different markers of socioeconomic status with cardiovascular disease and diabetes risk factors in the Diabetes, Heart and Health Survey

Patricia A Metcalf, Robert R K Scragg, David Schaaf, Lorna Dyall, Peter N Black, Rod T Jackson

Abstract

Aim To compare different markers of socioeconomic status (SES) with cardiovascular disease (CVD) and diabetes risk factors.

Methods Data were from 4020 participants aged 35–74 years from the Diabetes, Heart and Health Survey that was carried out in 2002 and 2003. Measures of SES were the occupation-based NZ Socioeconomic Index (NZSEI), combined household income, education, and the area-based deprivation measure NZDep2001.

Results After adjusting for all other SES measures, there were relatively few independent risk factor associations with NZSEI or education. Both low income and being more deprived as measured by NZDep2001 were independently associated with higher 2-hour glucose tolerance concentrations, HbA1c levels, waist-to-hip ratio, urinary albumin concentrations, 5-year CVD risk, current cigarette smoking, lower HDL-cholesterol, and less time spent exercising compared to the highest SES strata. Low income was independently associated with a higher prevalence of total and previously diagnosed diabetes mellitus, and lower stature.

More deprivation was independently associated with higher diastolic blood pressure levels, fasting glucose concentrations and BMI. Associations with height, and 2-hour glucose levels, and prevalence of total and previously diagnosed diabetes were greater with income, whereas NZDep2001 showed stronger associations with diastolic blood pressure, raised blood pressure, HDL-cholesterol, fasting glucose, BMI, waist-to-hip ratio, exercise levels, urinary albumin concentrations, 5-year risk of CVD and prevalence of smoking compared to the highest SES groups. Associations of income and NZDep2001 with HbA1c were similar.

Conclusions Cardiovascular disease and diabetes risk factors were more strongly associated with the area-based NZDep2001 and household income than with the individual’s occupation-based NZSEI or education. In general, the strongest associations were observed for NZDep. These findings provide support for the application of NZDep in health policy development in New Zealand, when other measures of SES are not available, and we recommend that this very accessible indicator of socioeconomic and health status continue to be updated.
simultaneously adjust for the other SES measures. Furthermore, mortality from CVD has been reported to be higher in lower SES groups, particularly with the area-based deprivation indicator NZDep91.5

We have previously reported a trend to a more adverse pattern of CVD risk factor levels in the lower SES groups.6 The strongest associations were related to income and education rather than New Zealand Socioeconomic Index (NZSEI).7 However, raised blood pressure was associated with low education, and prevalence of diabetes mellitus with income.6

A study carried out in Sweden reported that education, income, and occupational class could not be used interchangeably, as although they are correlated, they each measure a different phenomena and tap into different causal mechanisms.8 The latter study found that education was the strongest predictor of diabetes, income was the strongest predictor of all cause mortality, but myocardial infarction morbidity and mortality showed a more mixed picture.8

Occupational class is a measure of the physical work environment and how workplace itself is organised.8 Income provides material or immaterial resources for health, such as better housing, clothing, food and resources for dealing with stressful and demanding situations.8 On the other hand, educational attainment relates to the ability to turn information into practical measures and behaviours.8 There appear to have been few previous reports where the NZSEI, income, education, and NZDep2001 have been adjusted for simultaneously.

The aim of this study was to compare CVD and diabetes risk factors across New Zealand Socioeconomic Index (NZSEI) classes, income, levels of tertiary education groups, and NZDep2001 classes to determine whether there were important independent gradient differentials after adjusting for other measures of socioeconomic status.

Methods

The Auckland Diabetes, Heart and Health Survey was carried out between December 2001 and November 2003. Adults aged 35 to 74 years were recruited from two sampling frames: one was a cluster sample where random starting point addresses were obtained from Statistics New Zealand and the probability of selection was proportional to the number of people living in that mesh block (response rate 61.3%); and the other was a random sample taken from the November 2000 Auckland electoral rolls stratified into 5-year age bands and included all people living in the Auckland area, but excluding Franklin and Rodney (response rate 60%).

Out of the 4049 participants interviewed, 1408 were from the cluster sample, and 2641 were from the electoral roll. Twenty-nine people were excluded as they were outside the age range thus leaving 4020. These participants comprised 47.8% males and 52.2% females, 50.3% Europeans and Others, 25.0% Maori, and 24.7% Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin). Ethical Committee approval was obtained from the Auckland Ethics Committees.

Interviews were carried out in halls or clinics close to participant’s homes. Personnel were trained in the administration of the questionnaires and in taking blood pressure and other measurements. Participants filled in a questionnaire which included questions on ethnicity, education level attained, smoking history, occupation, gross combined household income, and past medical history. Ethnicity was defined according to the current NZ census.9

Occupations were first coded using the New Zealand classification of occupations.10 Occupational class was then assigned as the highest of the participant or their spouse, or for retired people using their main lifetime occupation for the New Zealand Socioeconomic Index (NZSEI).7 NZSEI was then transformed into discrete ‘occupational classes’ as proposed by Davis et al.7
These classes are comprised of: Class 1 – legislators and administrators; Class 2 – various professionals; Class 3 – corporate managers, associate professionals, and the armed forces; Class 4 – trade workers, plant operators and office clerks; Class 5 – other trade workers, machine operators and labourers; and Class 6 – market-orientated agricultural and fishery workers. Classes 1 and 2 and Classes 5 and 6 were combined due to their small numbers.

Education was classified as no tertiary education, Certificate (e.g. Trade or Technicians, apprenticeship or typing), Diploma (e.g. Teacher, Nurse, or Business Management), or Degree (e.g MA, PhD, BA, BSc, or Medicine). Combined yearly household income categories were “missing” and <$30,000, $30,001 to <$50,000, $50,001 to $70,000, and >$70,000. After geocoding the address of each participant, the 10-category NZDep2001 was assigned according to Salmond and Crampton.

Participants fasted from 10pm the evening before the interview and collected a first morning urine sample which they brought along. A 75-gram oral glucose tolerance test was carried out in participants who had not been previously diagnosed with diabetes, and a fasting and 2-hour post Glucaid drink blood samples were collected for glucose measurement. Plasma glucose was measured using an enzymatic method [Roche Products (NZ)]. Participants were classified as having newly diagnosed diabetes mellitus using 1998 WHO criteria using fasting glucose ≥7.0 mmol/L or 2-hour post glucose load of ≥11.1 mmol/L for diabetes.

Serum cholesterol was measured using an enzymatic method and HDL-cholesterol was measured using a combination of a polyanion and a divalent cation (Roche). Serum triglycerides were measured enzymatically. Urinary albumin was measured using an immunoturbidimetric method. Haemoglobin A1c was measured by High Performance Liquid Chromatography on a Biorad Variant II instrument. An Omron-Hem-706 oscillometric blood pressure pulse monitor was used to measure blood pressure two times after the participant had been seated for at least 5 minutes. A person was classified as having raised blood pressure if the mean of the two measured blood pressures was ≥140 mmHg systolic or ≥90 mmHg diastolic, or if they reported taking medication for raised blood pressure.

Weight and height were measured to the nearest 0.1 kg and 0.5 cm, respectively. Body mass index (BMI) was calculated as weight (in kg) divided by the square of height (in m). Obesity was defined as a body mass index >30 kg/m², and overweight as a body mass index between >25 and 30 kg/m². Waist and hips were measured to the nearest 0.5 cm. The 5-year cardiovascular risk was calculated using the Framingham equation. Moderate and vigorous leisure exercise was assessed using a 3-month physical activity recall questionnaire that has been validated.

Participant data were weighted according to the sampling frame that they were obtained from and means, standard errors, prevalences and odds ratios calculated using dual frame sampling methodology using SAS survey procedures. Odds ratios and means were first estimated after adjusting for age, ethnicity and gender; and in the second step, NZSEI, income, education, and NZDep2001 were entered to estimate their independent effects.

Because of the positively skewed frequency distribution of urinary albumin and exercise times, these were converted to log values for calculations; the results are presented as geometric means (the exponential of the mean of the logged data) and associated 95% tolerance factor. The strength of the associations of CVD and diabetes risk factors with SES measures were assessed using partial correlation coefficients adjusted for age, gender, and ethnicity.

**Results**

Income was similarly correlated with NZSEI and NZDep2001 (both 0.37). The correlation between the NZSEI and NZDep2001 was -0.34 (NZSEI has 10 = low and 90 = high, whereas NZDep has 1 = least deprived and 10 = most deprived), and between NZSEI and education was 0.24. The correlations between income and education (0.16) and NZDep2001 and education (-0.18) were lowest.

Means and odds ratios for CVD and diabetes risk factors are shown in Table 1 by NZSEI occupational classes after adjusting for age, gender and ethnicity. Compared to NZSEI class 1, 2, & 3, the prevalence of total and previously diagnosed diabetes and current smoking were significantly higher in NZSEI class 5 & 6, and mean...
Mean exercise levels, HDL-cholesterol concentrations, and stature were significantly lower. However, after further adjusting for income, education and NZDep2001, the only significant difference between NZSEI class 1, 2, & 3 and NZSEI class 5 & 6 was the higher waist-to-hip ratio in the latter group.

Table 1. Mean (SE) or odds ratio (95% confidence interval) for cardiovascular disease and diabetes risk factor levels by NZSEI occupational class (1, 2, &3 = highest, 5 & 6=lowest) adjusted for age, gender, and ethnicity

<table>
<thead>
<tr>
<th>NZSEI occupational class</th>
<th>1, 2, &amp; 3</th>
<th>4</th>
<th>5</th>
<th>5 &amp; 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>828</td>
<td>714</td>
<td>931</td>
<td>1547</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>125 (0.81)</td>
<td>124.3 (0.81)</td>
<td>126.0 (0.85)</td>
<td>126.5 (0.57)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>77.4 (0.48)</td>
<td>76.9 (0.48)</td>
<td>77.4 (0.47)</td>
<td>78.0 (0.41)</td>
</tr>
<tr>
<td>Raised blood pressure†</td>
<td>1.00</td>
<td>1.02 (0.75, 1.38)</td>
<td>1.10 (0.84, 1.45)</td>
<td>1.27 (0.97, 1.66)</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>5.6 (0.05)</td>
<td>5.5 (0.05)</td>
<td>5.5 (0.04)</td>
<td>5.5 (0.04)</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.42 (0.02)</td>
<td>1.41 (0.02)</td>
<td>1.40 (0.02)</td>
<td>1.37 (0.01)††</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.52 (0.06)</td>
<td>1.48 (0.04)</td>
<td>1.56 (0.07)</td>
<td>1.57 (0.04)</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>5.31 (0.66)</td>
<td>5.47 (0.07)</td>
<td>5.46 (0.06)</td>
<td>5.63 (0.06)</td>
</tr>
<tr>
<td>2hr glucose (mmol/L)</td>
<td>6.15 (0.15)</td>
<td>5.90 (0.12)</td>
<td>6.32 (0.15)</td>
<td>6.48 (0.12)</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.8 (0.03)</td>
<td>5.8 (0.03)</td>
<td>5.8 (0.04)</td>
<td>6.0 (0.03)***††</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.00</td>
<td>0.88 (0.55, 1.42)</td>
<td>1.16 (0.76, 1.77)</td>
<td>1.56 (1.07, 2.28)††</td>
</tr>
<tr>
<td>New diabetes</td>
<td>1.00</td>
<td>0.85 (0.28, 1.50)</td>
<td>1.02 (0.51, 2.07)</td>
<td>1.24 (0.61, 2.50)</td>
</tr>
<tr>
<td>Known diabetes</td>
<td>1.00</td>
<td>1.03 (0.58, 1.82)</td>
<td>1.24 (0.76, 2.05)</td>
<td>1.70 (1.11, 2.62)††</td>
</tr>
<tr>
<td>5-year CVD risk (%)</td>
<td>7.6 (0.20)</td>
<td>7.4 (0.20)</td>
<td>7.8 (0.19)</td>
<td>8.2 (0.17)††</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.4 (0.22)</td>
<td>29.3 (0.27)</td>
<td>29.4 (0.22)</td>
<td>30.3 (0.21)***††</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.9 (0.31)</td>
<td>169.1 (0.33)</td>
<td>167.9 (0.22)††</td>
<td>167.3 (0.25)***††</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.83 (0.003)</td>
<td>0.88 (0.003)</td>
<td>0.88 (0.003)</td>
<td>0.89 (0.002)***††</td>
</tr>
<tr>
<td>Urinary albumin (mg/L)²</td>
<td>4.5 (1.05)</td>
<td>4.0 (1.08)</td>
<td>4.6 (1.10)</td>
<td>5.0 (1.07)</td>
</tr>
<tr>
<td>Exercise (min/week)³</td>
<td>55.9 (1.27)</td>
<td>61.5 (1.21)</td>
<td>45.3 (1.23)††</td>
<td>34.9 (1.20)***††</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.00</td>
<td>1.06 (0.73, 1.54)</td>
<td>1.41 (1.02, 1.94)††</td>
<td>1.60 (1.16, 2.20)***††</td>
</tr>
</tbody>
</table>

*P < 0.05; **P < 0.01; ***P < 0.001 compared to NZSEI Classes 1, 2 & 3 (highest). BMI = body mass index, CVD = cardiovascular disease, †No longer significant after adjusting for education, income and NZDep2001.  
1. Rising blood pressure was defined as systolic ≥140 mm Hg or diastolic ≥90 mm Hg or current blood pressure-lowering treatment.  
2. Geometric mean (tolerance factor).

The higher prevalence of total diabetes mellitus and previously diagnosed diabetes in the lowest NZSEI group were no longer significant after further adjusting for income, and the higher BMI level was no longer significant after further adjusting for education. The lower HDL-cholesterol levels were no longer significant after further adjusting for income or NZDep2001, the higher 5-year CVD risk was no longer significant after adjusting for income, education or NZDep2001, the higher HbA1c
levels by income and education, the lower stature was no longer significant after further adjusting for income and NZDep2001, the lower exercise levels were explained by education and NZDep2001 or income and education, and the higher smoking levels by education or NZDep2001.

Table 2. Mean (SE) or odds ratio (95% confidence interval) for cardiovascular disease and diabetes risk factor levels by income adjusted for age, gender, and ethnicity

<table>
<thead>
<tr>
<th>Household Income</th>
<th>≥ $70,001</th>
<th>$50–$70,000</th>
<th>$30–$50,000</th>
<th>&lt; $30,000</th>
<th>missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>982</td>
<td>636</td>
<td>752</td>
<td>1211</td>
<td>339</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>126.7 (1.73)</td>
<td>125.6 (0.87)</td>
<td>125.2 (0.81)</td>
<td>126.0 (0.81)</td>
<td>124.4 (1.54)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>77.9 (0.42)</td>
<td>77.6 (0.50)</td>
<td>77.1 (0.47)</td>
<td>77.8 (0.48)</td>
<td>77.6 (1.03)</td>
</tr>
<tr>
<td>Raised blood pressure†</td>
<td>1.00</td>
<td>0.97 (0.72, 1.31)</td>
<td>0.84 (0.63, 1.13)</td>
<td>1.27 (0.97, 1.67)</td>
<td>1.03 (0.70, 1.59)</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>5.5 (0.94)</td>
<td>5.6 (0.95)</td>
<td>5.1 (0.95)</td>
<td>5.5 (0.95)</td>
<td>5.4 (0.89)</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.04 (0.01)</td>
<td>1.01 (0.02)</td>
<td>1.00 (0.02)†</td>
<td>1.00 (0.03)</td>
<td>1.00 (0.03)†</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.00 (0.06)</td>
<td>1.00 (0.05)</td>
<td>1.00 (0.05)</td>
<td>1.00 (0.05)</td>
<td>1.00 (0.05)</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>5.41 (0.06)</td>
<td>5.46 (0.07)</td>
<td>5.44 (0.08)</td>
<td>5.63 (0.07)†</td>
<td>5.69 (0.11)†</td>
</tr>
<tr>
<td>2 hr glucose (mmol/L)</td>
<td>7.06 (0.12)</td>
<td>6.13 (0.12)</td>
<td>6.00 (0.13)</td>
<td>6.73 (0.15)†</td>
<td>6.96 (0.16)</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.7 (0.12)</td>
<td>5.8 (0.14)</td>
<td>5.9 (0.15)</td>
<td>6.3 (0.14)***</td>
<td>6.0 (0.09)***</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.00</td>
<td>1.21 (0.72, 2.02)</td>
<td>1.50 (0.90, 2.51)</td>
<td>2.17 (1.46, 3.51)***</td>
<td>2.64 (1.55, 4.41)***</td>
</tr>
<tr>
<td>New diabetes</td>
<td>1.00</td>
<td>1.79 (0.31, 3.86)</td>
<td>0.86 (0.38, 2.04)</td>
<td>2.20 (1.05, 4.61)†</td>
<td>2.37 (1.09, 5.56)</td>
</tr>
<tr>
<td>Known diabetes</td>
<td>1.00</td>
<td>0.94 (0.31, 3.14)</td>
<td>1.89 (1.01, 3.54)</td>
<td>2.09 (1.03, 4.20)**</td>
<td>2.49 (1.35, 4.67)***</td>
</tr>
<tr>
<td>5-year CVD risk (%)</td>
<td>7.4 (1.7)</td>
<td>7.2 (2.0)</td>
<td>7.7 (2.5)</td>
<td>8.5 (2.5)***</td>
<td>7.8 (1.6)</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>26.9 (0.20)</td>
<td>26.9 (0.29)</td>
<td>26.9 (0.25)</td>
<td>29.3 (0.25)</td>
<td>30.3 (0.40)</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>85.2 (0.20)</td>
<td>86.3 (0.24)***</td>
<td>86.4 (0.24)***</td>
<td>86.5 (0.24)***</td>
<td>86.5 (0.24)***</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.87 (0.02)</td>
<td>0.88 (0.03)**</td>
<td>0.88 (0.03)**</td>
<td>0.89 (0.03)**</td>
<td>0.89 (0.03)**</td>
</tr>
<tr>
<td>Urinary albumin (mg/dL)</td>
<td>3.9 (0.08)</td>
<td>4.3 (0.10)</td>
<td>4.5 (0.10)†</td>
<td>5.5 (0.11)***</td>
<td>4.6 (0.17)</td>
</tr>
<tr>
<td>Exercise (minutes/week)</td>
<td>69.1 (1.19)</td>
<td>47.3 (1.26)**</td>
<td>47.1 (1.26)**</td>
<td>31.1 (1.23)**</td>
<td>46.4 (1.45)**</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.00</td>
<td>1.49 (1.21, 2.80)**</td>
<td>2.17 (1.37, 3.06)**</td>
<td>1.94 (1.28, 2.73)**</td>
<td>2.00 (1.26, 3.37)**</td>
</tr>
</tbody>
</table>

*P < 0.05, **P < 0.01, ***P < 0.001 compared to income ≥ $70,001. BMI = body mass index, CVD = cardiovascular disease.
† No longer significant after adjusting for education, NZSEI occupational class and NZDep2001.
1 Raised blood pressure was defined as systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg or on current blood pressure lowering treatment.
2 Geometric mean (cigarettes per day).

Table 2 shows means and odds ratios for CVD and diabetes risk factors by combined household income groups, adjusted for age, gender, and ethnicity. Compared to people with incomes ≥$70,001, people on incomes <$30,000 had significantly lower HDL-cholesterol concentrations, lower stature, and lower time spent exercising per week, and significantly higher fasting and 2-hour glucose concentrations, HbA1c levels, 5-year CVD risk, urinary albumin concentrations and waist-to-hip ratios—plus higher prevalence of total, newly and previously diagnosed diabetes mellitus, and current cigarette smoking levels.

After further adjusting for the other SES measures, only fasting glucose and newly diagnosed cases of diabetes mellitus were no longer significant and were explained by NZDep2001.

The last column in Table 2 shows mean levels of CVD and diabetes risk factors in those who either did not know their combined household income or refused to record it. Based on the risk factor levels, it would appear that many belonged to the lowest income group, with the exception of exercise times, urinary albumin concentrations and 5 year CVD risk, that were intermediate between the 2 lowest income groups.
Means and odds ratios for CVD and diabetes risk factors by level of education are shown in Table 3. After adjusting for age, gender, and ethnicity, people with no tertiary education had higher fasting glucose levels, Hba1c levels, BMI, and waist-to-hip ratios and a higher prevalence of current cigarette smoking, and lower exercise times compared to those with a university degree. After further adjusting for the other SES measures, only the higher prevalence of smoking and higher BMI levels remained significant.

The higher waist-to-hip ratio was no longer significant after NZSEI was included in the model, the lower exercise time was no longer significant after NZSEI or income were included in the model, Hba1c was no longer significant after NZDep2001 was
Table 4. Mean (SE) or odds ratio (95% confidence interval) for cardiovascular disease and diabetes risk factor levels by NZDep2001 (1 & 2 = high, 9 & 10 = lowest) adjusted for age, gender, and ethnicity

Table 4 shows means and odds ratios for CVD and diabetes risk factor levels by NZDep2001 classes. Compared to NZDep2001 class 1 & 2 (least deprived), there were trends towards higher diastolic blood pressure, fasting and 2-hour glucose concentrations, HbA1c, 5-year CVD risk, BMI, waist-to-hip ratios, urinary albumin and higher smoking, total and newly diagnosed diabetes, and raised blood pressure prevalence, and lower exercise, stature, and HDL-cholesterol levels in the more deprived NZDep2001 classes. However, the initially significant higher raised blood pressure, total and newly diagnosed diabetes mellitus prevalence and lower stature in the most deprived NZDep2001 class were explained by household income.

Further adjustment for the number of adults and number of children in the household tended to attenuate the associations slightly for both income and NZDep2001.

After adjusting for age, gender, and ethnicity, partial correlation coefficients showed stronger associations between income and 2-hour glucose concentrations, height, total, and previously diagnosed diabetes. Similarly adjusted partial correlations were stronger with NZDep2001 for diastolic blood pressure, HDL-cholesterol, 5-year CVD risk, BMI, waist-to-hip ratio, exercise time, urinary albumin, raised blood pressure, and smoking. The partial correlations with HbA1c were similar for both NZDep2001 and income.
Discussion

**Main findings**—The current study has shown independent associations for low household income and more deprivation with 2-hour glucose concentrations, HbA$_{1c}$ levels, 5-year CVD risk, waist-to-hip ratios, urinary albumin concentrations, and cigarette smoking—and lower HDL-cholesterol levels and exercise time compared to the highest SES stratum.

Income also showed independent adverse associations with total and previously diagnosed diabetes, and height. More deprived NZDep2001 classes showed adverse independent associations with fasting glucose concentrations, diastolic blood pressure, and BMI. The occupation-based NZSEI only showed an independent association with waist-to-hip ratio, and education only showed independent associations with BMI and smoking habit.

Income showed stronger associations with 2-hour glucose, total, and previously diagnosed diabetes mellitus, whereas NZDep2001 showed stronger associations with diastolic blood pressure, raised blood pressure, HDL-cholesterol, fasting glucose, 5-year CVD risk, BMI, waist-to-hip ratio, urinary albumin concentrations, exercise levels, and prevalence of smoking. The strength of the association with HbA$_{1c}$ was similar for NZDep2001 and income.

**Blood pressure**—There was a trend towards a higher prevalence of raised blood pressure across the more deprived NZDep2001 groups, and diastolic blood pressure levels were significantly higher in the most deprived NZDep2001 class compared to the least deprived (Table 4). But raised blood pressure was not associated with any of the other SES measures. In contrast, the 1996–1997 Health Survey reported an inverse association between self-reported high blood pressure and income, level of education and NZDep96, as did the 2002–2003 NZ Health Survey using NZDep2001, but they did not adjust for the other SES measures simultaneously.

The 1988–1990 Workforce Diabetes Survey reported higher systolic blood pressure levels and higher prevalence of raised blood pressure in the lower education groups compared to those with a University education in a working population. These results, taken together, indicate a consistent SES determinant for blood pressure.

**Lipids**—Both low income and more deprived NZDep2001 classes showed independent associations for HDL-cholesterol with lower levels, but no significant differences for total cholesterol or triglycerides. HDL-cholesterol levels showed a similar association with the Elley-Irving SES measure and education in females in the 1989-90 LINZ survey. HDL-cholesterol concentrations were inversely associated with income and NZDep96 in the 1996-1997 NZ Health Survey, and with NZDep2001 in the 2002-2003 NZ Health Survey, but not with self-reported cholesterol lowering medications. The 1997 NNS also reported an inverse trend between HDL-cholesterol and NZDep96, and an inverse trend between cholesterol and NZDep96 in males.

**Diabetes**—The current study showed that both income and NZDep2001 had adverse associations with measures of glucose tolerance and prevalence of diabetes mellitus (Tables 2 and 4). However, NZDep2001 explained the initially elevated fasting
glucose concentrations and higher prevalence of newly diagnosed diabetes associated with income.

On the other hand, the initially significant associations between NZDep2001 and total and newly diagnosed diabetes were explained by income. This suggests that both income and level of deprivation are associated with newly diagnosed diabetes. However, only income was associated with previously diagnosed cases of diabetes, suggesting that the presence of diabetes may have an adverse impact on an individual’s earning power. The 1996–1997 NZ Health Survey reported a higher prevalence of self-reported diabetes in lower income and NZDep96 groups, but not education, and the 2002–2003 also reported a higher prevalence of self-reported diabetes in the lower NZDep2001 groups. The 1988–1990 Workforce Diabetes Survey also found inverse associations between diabetes prevalence, 2 hour glucose levels and income.

CVD risk—The 5-year risk of CVD was higher in the lowest income and more deprived NZDep2001 groups (Tables 2 and 4), and was stronger for NZDep2001. Although this finding does not appear to have been reported previously, the New Zealand census-mortality study found a strong gradient between death from CVD and NZDep2001.

Urinary albumin—The finding of increasing urinary albumin concentrations with more deprived NZDep2001 classes also does not appear to have been previously reported. We have noted that increased urinary albumin concentrations may be a marker of CVD risk.

BMI and waist-to-hip ratio—BMI showed an inverse relationship with NZDep2001, but not income (Tables 2 and 4), and the waist-to-hip ratio showed an inverse relationship with both NZDep2001 and income, that was stronger for NZDep2001. The 1989-1990 LINZ survey also reported an inverse relationship for BMI with the Elley-Irving SES measure, and a trend in females with education. Similarly, the Elley-Irving SES and education showed inverse trends with waist-to-hip ratio. The 1997 NNS also showed an inverse trend between BMI and NZDep96 and an inverse trend in females between NZDep96 and waist-to-hip ratio.

Height—Both income and NZDep2001 showed an inverse association with height, that was stronger for NZDep2001. Similar associations were observed with the Elley-Irving SES measure in the 1989-1990 LINZ survey, and the 1996–1997 NZ Health Survey. In females, there was an inverse trend between height and education in the LINZ survey, and an inverse trend in both males and females with NZDep2001 in the 1997 NNS.

Leisure-time exercise—Exercise times were lower in the lowest income groups and there was an inverse trend across NZDep2001 groups, which were stronger for NZDep2001 than for income (Tables 1 and 4). The 1989–1990 LINZ survey reported a similar trend in females with the Elley-Irving SES measure, but not with education. Whereas, the 1996–1997 NZ Health survey reported an inverse association between exercise levels and education, but not with income or NZDep96. However, the 2002–2003 NZ Health survey reported an inverse trend between exercise and NZDep2001 in females only.
Smoking—The finding of an increased prevalence of smoking in the lower SES strata, but particularly for NZDep2001 in the current study, has been consistently reported with all measures of SES.\textsuperscript{2,4,20,21}

Study limitations—NZSEI is an occupation-based measure that can be difficult to assign to a housewife or a person who has retired or is unemployed. This can be partly overcome by using a past occupation, or the occupation of an employed spouse. In the current survey we have assigned the NZSEI to the higher of the participant or spouse, or for those who had retired to their main life-time occupation. Another disadvantage, compared to income or education, is that the occupation(s) of an individual have to be coded and then mapped onto the NZSEI scale. It can also be difficult to code an occupation if insufficient information is given, such as ‘Engineer’.

A major disadvantage of income is that some people refuse to divulge the information and others do not know (Table 2 missing column), however it is easy to measure and code, as is education. In addition, poor health may actually lead to a drop in income.

A disadvantage of NZDep2001, aside from being an area-based rather than individual-based, is that the address of the participant must be first geocoded using a computer that requires matching addresses. In the current study, many people who lived on the borders of suburbs chose the next suburb as their domicile. Furthermore, NZDep is likely to have a higher misclassification error than the other SES measures as not all deprived people live in deprived small areas, and vice-versa. Despite these limitations, both household income and the area-based NZDep2001 have shown important associations with cardiovascular disease risk factor levels.

We note that when collecting data in surveys that it still important to collect information on income, education and occupation (and ethnicity) as they measure different aspects of the construct of SES, and may have varying associations with different risk factors due to different causal pathways. Including both the area-based NZDep and individual SES information in any model is required to fully adjust for confounding in analyses of the association of other exposures (e.g. diet) with CVD.\textsuperscript{27}

Study strengths—The major strengths of the current study are its size, and its community-based sample. Limitations to the current study include the collection of a single measure for lipids, glucose tolerance, and urinary albumin, the measurement of blood pressure on a single occasion, and that cigarette smoking and exercise information was based on self-report.

Conclusions

Cardiovascular disease and diabetes risk factor levels showed a more adverse pattern in the lower SES groups compared to the highest SES groups. In general, stronger associations were observed for NZDep2001 than for the other measures of SES. These findings endorse the use of NZDep as a tool for informing health-related policy development in New Zealand, where other measures of SES cannot be obtained. It will be important to continue to update this readily accessible tool in order to maintain its predictive validity.

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References:

Informed consent for vascular intervention: completing one audit loop
Katie Carter, Justin A Roake, Timothy Buckenham, Christopher M Frampton, David R Lewis

Abstract

Aim To reaudit documentation of the process of informed consent in patients undergoing vascular surgical and vascular radiological procedures.

Method A retrospective audit of randomly selected elective vascular radiological and surgical admissions from October 2005–2006 was undertaken to assess the impact of a previous audit on the documentation of the consent process carried out in 2005. Outpatient clinic letters, handwritten entries in the patients’ admission notes, and consent forms were scrutinised and data collated on which doctors took consent, when consent was obtained, what details of the consent process were documented, and whether additional information was made available to patients.

Results 99 sets of notes were reviewed (surgical n=50, radiological n=49). For patients undergoing vascular surgery, the consent form was signed by a consultant in 16 (32%) cases compared to 2 (4%) in the previous audit (p=0.013; \( \chi^2 \)). Significantly more vascular radiological consent forms were signed by a consultant (43) compared with surgical consent forms (16) (p<0.001; \( \chi^2 \)). Documentation that the risks of surgery had been discussed with the patient was present in 31 (62%) surgical notes and in 20 cases such discussions were documented in letters from clinics.

For radiological consent documentation, 34 (69.4%) patient notes recorded discussions regarding procedural risk. Twenty-two (44.9%) of the vascular radiological patients had such risks documented in their outpatient notes by a vascular surgeon compared with 1 (2%) (p <0.001; \( \chi^2 \)) in the previous audit. Additional written information was given to 7 (14%) of the vascular surgical patients which was similar to the previous audit. No additional information was given to patients who underwent vascular radiological procedures.

Conclusions Significant improvements have been made since the previous audit with more surgical consultants signing the consent forms and increased documentation of the nature of radiological procedures and risks discussed in outpatient clinics. From the current audit, provision of additional written information (patient information sheets) was an area identified for future improvement.

Informed consent is a process that leads to an understanding of the diagnosis, the planned procedure, the therapeutic alternatives, and the inherent procedural risks of an intervention by the patient. Under the New Zealand Health and Disability Commissioner’s Code of Health and Disability Services Consumers’ Rights, all patients have the legal right to be fully informed of potential risks and benefits, the right to make an informed choice of treatment options, and the right to give informed consent for treatment (Rights 6 and 7).
An integral part of the consent process is the written documentation of the patient’s understanding and agreement to proposed treatment. The concept of informed consent began as a legal doctrine to protect patients from physical battery. A common modern (mis)understanding is that clear documentation of the consent process, carefully transcribed to the patient’s notes, protects doctors from medicolegal retribution if complaints occur. As limited as this evidence may be, it is often the only evidence to resolve or settle complaints.\(^3\)

The rate of documentation reflecting patient-doctor discussions of procedural risk is reported to be between 33% and 53%\(^2\) with our previous audit comparing favourably to other published data.\(^3\)

The aim of the current study was to reassess the documentation of the process of informed consent for elective vascular surgical and radiological procedures.

**Methods**

A retrospective reaudit of the completeness of documentation of the consent process for elective vascular surgery and elective vascular radiological procedures was performed at Christchurch Hospital, New Zealand. The audit was performed on 99 randomly selected elective admissions under the care of the vascular team between October 2005 and October 2006. A computerised patient management system (PMS) was used to identify patients. Medical notes were reviewed and documentation of the consent process was evaluated.

Similar to our previous audit (which evaluated documentation of the consent process in 2005), the following data was collated from typed outpatient clinic letters, handwritten entries in the patient notes, and from the consent form:

- The type of operation or radiological procedure planned,
- Who obtained written consent (i.e. what grade of Doctor signed the consent form),
- When the consent was obtained,
- Documented evidence of an explanation of the procedure,
- Documentation of discussion of treatment options,
- Documentation of the risks of the procedure, and
- Documentation of whether any additional written information was made available to the patient.

Statistical analysis comparing surgical and radiological consent documentation as well as interaudit variation was performed on SPSS (v11.5) statistical software using the Chi-squared (\(\chi^2\)) or Fisher’s exact test for comparison of discontinuous data.

**Results**

Of the 99 sets of notes analysed, 50 were from patients who underwent a vascular surgical procedure and 49 from patients who underwent a vascular radiological procedure. The median age for both surgical and radiological patients was 72 years with a range of 49–92 years and 38–89 years respectively.

There was no statistically significant differences between these groups of patients and those in the previous audit with regard to age. In the surgical group of patients, the male to female ratio was 1.2:1 which was not significantly different to the previous audit. In the radiological group of patients, the male to female ratio was 3.5:1 compared with 1.2:1 in the previous audit (p=0.02; \(\chi^2\)).

The surgical interventions performed are as shown in Table 1 and were similar to the previous audit. The radiological procedures all involved intra-arterial angiography.
Table 1. Elective vascular surgical procedures

<table>
<thead>
<tr>
<th>Type of procedure</th>
<th>Number of procedures</th>
<th>Current audit</th>
<th>Number of procedures</th>
<th>Previous audit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid endarterectomy</td>
<td>13</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infrainguinal reconstruction</td>
<td>9</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Varicose vein surgery</td>
<td>7</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amputation—major and minor</td>
<td>7</td>
<td>3 (major only)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Open aortic surgery</td>
<td>6</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endovascular aneurysm repair</td>
<td>8</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td><strong>51</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Thirty-three of the 49 patients had angioplasty, 6 patients with stent insertion, and 6 with other radiological procedures. Documentation of the process of consent for surgical and radiological procedures as well as comparison with the previous audit is summarised in Table 2.

Table 2. Information documented in patient medical records by consenting doctor for surgical procedures and radiological procedures in the previous audit compared to the current audit

<table>
<thead>
<tr>
<th>Information documented</th>
<th>Previous audit</th>
<th>Current audit</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consultant signature on consent form</td>
<td>2 (0.04%)</td>
<td>16 (32%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Registrar signature on consent form</td>
<td>46 (93.9%)</td>
<td>43 (87.8%)</td>
<td>0.487 (Fisher’s )</td>
</tr>
<tr>
<td>House officer signature on consent form</td>
<td>20 (39.2%)</td>
<td>32 (62%)</td>
<td>0.013</td>
</tr>
<tr>
<td>House officer signature on consent form</td>
<td>3 (6.1%)</td>
<td>5 (10.2%)</td>
<td>0.715 (Fisher’s )</td>
</tr>
<tr>
<td>House officer signature on consent form</td>
<td>29 (56.9%)</td>
<td>2 (4%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>House officer signature on consent form</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
<td>1.000 (Fisher’s )</td>
</tr>
<tr>
<td>Procedure explained in outpatient clinic letter</td>
<td>20 (39.2%)</td>
<td>13 (26%)</td>
<td>0.157</td>
</tr>
<tr>
<td>Procedure explained in outpatient clinic letter</td>
<td>6 (12.2%)</td>
<td>20 (40.8%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Procedure explained in inpatient notes</td>
<td>12 (23.5%)</td>
<td>6 (12.0%)</td>
<td>0.130</td>
</tr>
<tr>
<td>Procedure explained in inpatient notes</td>
<td>0 (0%)</td>
<td>5 (10.2%)</td>
<td>0.062</td>
</tr>
<tr>
<td>Treatment options discussed in outpatient clinic letter</td>
<td>25 (49.0%)</td>
<td>11 (22%)</td>
<td>0.005</td>
</tr>
<tr>
<td>Treatment options discussed in outpatient clinic letter</td>
<td>5 (10.2%)</td>
<td>5 (10.2%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Risks outlined in detail in outpatient clinic letter</td>
<td>20 (39.2%)</td>
<td>20 (40%)</td>
<td>0.936</td>
</tr>
<tr>
<td>Risks outlined in detail in outpatient clinic letter</td>
<td>1 (2%)</td>
<td>22 (44.9%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Risks outlined in detail on consent form</td>
<td>1 (2.0%)</td>
<td>12 (24.5%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Risks outlined in detail on consent form</td>
<td>18 (36.7%)</td>
<td>5 (10%)</td>
<td>0.002</td>
</tr>
<tr>
<td>Additional written information given</td>
<td>6 (11.8%)</td>
<td>7 (14%)</td>
<td>0.737</td>
</tr>
<tr>
<td>Additional written information given</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Grey cells = surgical patients; White cells = radiological patients.

Forty-six (93.9%) of the radiological consent forms were signed on the day of the procedure, with two being signed 1 day before and one 2 days before intervention. An equal number of surgical consent forms were signed on the day of surgery (23 [46%]) and the day before surgery (23 [46%]). All patients signed the official District Health Board consent form.
There has been significant improvement in documentation of the consent process for radiological procedure in our unit between audits. Documented evidence of an explanation of the risk of angiography/angioplasty appeared in the outpatient clinic letters from vascular surgeons in 20 (40.8% current audit) compared to 6 (12.2%) (p=0.001; \chi^2), and 22 (44.9% current audit) compared to 1 (2%) (p<0.001; \chi^2) for respective audits.

The proportion of consent forms signed by a consultant surgeon has improved (p<0.001) for surgical patients; junior staff only signed 4% of the consent forms in the current study compared with 56.7% in the previous audit (p<0.001).

Documentation that risks were discussed in detail either in outpatient clinic letters, in patient notes or on the consent form (or in more than one of the above) was present in 32 (64%) for surgical patients and 27 (55%) for radiology patients.

There has been no improvement in the documentation that additional written information regarding the proposed procedure was made available to patients but surgical patients still receive significantly more written material than patients undergoing radiological procedures (p=0.012; \chi^2).

**Discussion**

There has been a significant improvement in the number of consent forms signed by surgical consultants between the two audits. Documentation of radiological procedures and risks of angiography/angioplasty being discussed in outpatient clinics by the vascular surgeons has also increased significantly.

Written documentation of the procedural risks has decreased since the previous audit but still compares favourably with other published data. This observed difference may be explained by the more stringent inclusion criteria of the current audit. This audit focused on the documentation of specific procedural risks while the previous audit counted documentation of a more general discussion of risks (e.g. “…the risks of surgery have been discussed…”).

Data from this audit have been presented locally as well as at national and international meetings. The subject of informed consent never fails to stimulate debate and such debate, whatever an individual’s views on the consent process, can only increase awareness of this subject.

Another area that we have identified for potential improvement include the documentation of the provision of additional written information, which still appears to remain under used, as reported in the previous audit.

The study may be criticised for being retrospective in design and containing a relatively small sample size. In order to assess differences between the two studies the study designs were similar. The current results are comparable to published prospective studies and we believe they are a true reflection of our current practice.\(^2\)

This study focuses on the documentation of the consent process and it is important to understand that consent is not an event or a signature on a form, but an ongoing process of communication during perioperative care.\(^3\) Documentation of informed consent should be viewed as a dynamic process that stimulates discussion and assures
comprehension rather than a static historical record of the discussion between surgeon and patient.

A signed consent form does not shelter the surgeon from litigations and it is important to convert the process from a legalistic exercise into a substantive conversation that accomplishes the education and partnership that are the hallmarks of shared decision-making.

To keep this process patient-centred, it is necessary to consider what information patients themselves identify as important. A survey to determine preference of patients regarding information provided preoperatively found that patients considered meeting the surgeon prior to surgery and discussing alternative treatment options and complications of surgery as most important to them.

It has been documented that patients do not lay down memory when counselled regarding the risks of surgery but the same authors suggest a patient’s position can be “the reason why I have the right to decide what should happen to my body is because I will have to live with the consequences. It is not because I have remembered having decided.”

The lack of recall emphasises the need for doctors to make detailed notes especially since the surgeon is arguably less likely to remember the details of what they have said on a specific occasion than the patient. The significant improvement in the number of surgical consent forms signed by consultants was encouraging, and implies that the patients in our study had a final opportunity to comprehensively discuss aspects of their procedure and have any questions answered by a senior doctor.

As a direct result the number of house surgeons signing consent forms has fallen significantly. This indicates that the responsibility taken by consultants in our unit is in accordance with the view of the New Zealand Medical Council which has stated: “obtaining informed consent is a skill best learnt by the house surgeon observing consultants and experienced registrars in the clinical setting. Doctors on probationary registration should not take informed consent where they do not feel competent to do so.”

We did not document whether junior medical staff were in attendance to learn about the consent process in this study.

The majority of patients were consented either on the day of surgery or the day before surgery. Since informed consent is a process rather than a signature it can be assumed that prior discussions took place in outpatient clinics. A limited time between outpatient consultations and signing of consent forms prior to surgery is arguably not only necessary to allow patient’s the opportunity to comprehend their diagnosis and treatment options but also to ensure they have had any questions adequately.

Published literature shows that different methods of delivery of the consent process have a significant impact on patient understanding, recall, and satisfaction. Additional written information has been found to be a useful adjunct in the consent process—as documented by two independent groups of orthopaedic surgeons in the UK.
In light of this evidence it is disappointing that the use of written material appears not to have improved between our audits, however it is important to note that (as of May 2006) patient information sheets have been made available on abdominal aortic aneurysm (AAA) and femoral popliteal bypass procedures in addition to the existing documents on carotid and varicose vein surgery. Hopefully future audits will find increased use of this new resource and further patient information sheets will be produced.

Furthermore, the use of video has been shown to reduce time spent informing patients and improve patient satisfaction. Indeed, a systematic review of video as part of informed consent is currently underway.

As a result of the previous audit, a consulting room is now available in the Department of Radiology at Christchurch Hospital. This audit did not study the setting in which consent was obtained and we therefore cannot comment on whether the new consulting room has improved this process, although we suspect it has.

A procedure-specific consent form for common vascular procedures may be part of the solution to improving documentation and disclosure around consent for intervention. This type of form was shown to be successful in a study performed in a Pittsburgh neurosurgical outpatient clinic.

The official District Health Board consent form is currently under review. A draft has been prepared which includes tick boxes for what method(s) were used to gain informed consent (diagrams, anatomical models, consultant letter) as well as additional space for documentation of the specific risks of intervention.

Informed consent is a controversial and complex process that requires significant time, knowledge, and communication skills. Documentation of this process should be used as a method of directing discussion, assuring understanding, and providing an historic record.

This study has demonstrated significant improvement in the number of surgical consent forms signed by senior medical staff. There has also been improvement in the documentation of discussion of radiological procedure and risks in the outpatient setting. This outpatient consent and documentation was generally performed by vascular surgeons which reflect our team approach to the management of patients with vascular disease. There is still, however, room for improvement with the current consenting process.

It is hoped that consent form improvements, a greater range of written patient information sheets, and continued reaudit will help achieve this goal.

**Competing interests:** None known.

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Endovascular repair of mycotic aortic aneurysms

David Yong, Justin A Roake, Timothy Buckenham, David R Lewis

The management of mycotic aneurysms is difficult, with high morbidity and mortality. Traditional open operative approaches include removal of infected material with either extra-anatomic reconstruction or in situ graft repair.\textsuperscript{1,2} Since the advent of endovascular aneurysm repair (EVAR) in the early 1990s, its use in the treatment of aneurysm disease has increased due to proven decrease in 30-day mortality. There have been few case studies reporting the use of EVAR for infected aneurysms.\textsuperscript{9,10}

We describe two cases of mycotic aneurysm of the abdominal aorta that were successfully managed with EVAR.

Case 1

A 45-year-old lady (who presented to her general practitioner with a 3 week history of abdominal pain and fever) was referred to the vascular surgical service following an ultrasound scan that showed a 10 cm abdominal aortic aneurysm.

On arrival at hospital, she was stable haemodynamically but was found to have significant comorbidities including a renal transplant 14 years previously, systemic lupus erythematosis, ischaemic heart disease, osteoporosis, hypertension, asthma, and hyperlipidaemia. Computed tomography (CT) confirmed a 12 cm abdominal aortic pseudo-aneurysm at the level of superior mesenteric artery, most likely mycotic in origin (Figure 1).

She went on to have a hybrid procedure that included laparotomy, bifurcated bypass from right iliac artery to splenic artery and superior mesenteric artery, followed by EVAR of the abdominal pseudo-aneurysm. The endovascular graft covered the ostio of the celiac and superior mesenteric arteries.

Her postoperative hospital stay was uneventful except for an episode of hypoxia due to a cardiac event on fourth postoperative day. Bacterial cultures including blood were negative. She received intravenous ceftriaxone for 6 weeks before commencing oral ciprofloxacin and augmentin for life.

She represented to hospital 2 months after discharge with further abdominal pain and fever. She was commenced on intravenous meropenem. No focus of infection was found on extensive investigations including a radio-labelled white blood cell scan.

The patient remained well on intravenous antibiotic for the next 6 weeks via a peripheral intravenous central catheter (PICC) line. She successfully came off intravenous antibiotic and on her last review, 14 months post surgery, she remained well with stable renal function on oral antibiotics.
Case 2

A previously well 63-year-old lady presented with 3 weeks history of gastrointestinal illness, nausea, vomiting, and diarrhoea with fevers; and 6 hours history of more acute onset of blurred vision and mild confusion.

Physical examination did not reveal any persisting neurological defect but investigations revealed raised inflammatory markers and impaired renal function. She was started on intravenous cefuroxime and metronidazole and rehydrated with a working diagnosis of sepsis and acute renal impairment.

Computed tomography showed a large penetrating ulcer in the infrarenal abdominal aorta with significant surrounding inflammation consistent with aortitis. She went on to have an endovascular repair and made an uncomplicated post-procedure recovery. All her blood cultures were negative.

She received 6 weeks of intravenous cefuroxime and metronidazole before changing to life-long oral ciprofloxacin and augmentin. She remained well at the last outpatient follow-up at 7 months post operation.

Discussion

Mycotic aneurysm represents a challenging part of vascular surgical practice. When Osler initially used the term ‘mycotic’ in 1885, the most common cause was direct extension of florid infective endocarditis leading to endarteritis and aneurysm formation.\(^3\)
Due to improved hygiene and antibiotics, the majority of cases of mycotic aneurysms in modern practice result from systemic sepsis, bacteremia, and arterial seeding with a resulting microbial arteritis in an artery already weakened by disease, most commonly atherosclerosis.

From large autopsy series and studies of surgical practice, the reported incidence of mycotic aneurysms is between 1.3 and 3.3% of all aortic aneurysms. Major risk factors for mycotic aneurysm formation include immunosuppression (such as HIV infection, administration of systemic chemotherapy, or immunosuppression agents), alcohol abuse, radiotherapy to the affected region, and chronic renal failure.

To avoid missing the diagnosis, a high index of suspicion is required. The majority of the patients present with a fever of unexplained origin or unexplained sepsis. This is commonly associated with an elevation in the circulating white blood cell count and increased C-reactive protein, both of which have a high sensitivity but poor specificity.

Patients commonly report severe back, abdominal, or thoracic pain which may be attributed to other pathology. Complications of mycotic aneurysms including arteriovenous fistulae, respiratory failure, and renal failure can also feature in the presentation.

From a review of the microbiological culture of the blood or aneurysm wall, this was positive in 98% of patients with 47% of cases due to Gram-negative bacilli (Salmonella species have a particular predilection for diseased arteries), 33% due to Gram-positive cocci, and 18% due to rare organisms such as Candida, Bacteroides, Mycobacteria, and Clostridia spp.

In our cases, no tissue could be sent for microbiological investigation since the pathology was treated through an endovascular approach.

The traditional surgical management of a mycotic aneurysm of abdominal aorta involves resection of aneurysm, debridement of infected tissues, and oversewing the aortic stump with extra-anatomic bypass through uncontaminated areas.

Resection of infected aneurysm and in situ repair with or without antibiotic bonded/soaked graft has also been reported. Allowing for small reported numbers, the morbidity and mortality associated with mycotic aneurysm repair is significantly higher than that associated with the repair of non-infected aneurysms.

In some unselected series, the in-hospital mortality exceeded 50% and in cases of rupture, the prognosis is grave.

More recently, EVAR has been developed to treat aneurysmal disease. A few case series have reported the successful management of mycotic aneurysms involving a variety of arterial segments including the abdominal aorta using this approach. This is typically combined with lifelong antibiotic therapy.

Most authors only reported short-term outcome. This is expected since the long-term results of EVAR in the treatment of aneurysmal disease are not yet established. The less invasive nature of EVAR in the treatment of mycotic aneurysm of the abdominal aorta, and the apparent low perioperative morbidity and mortality, makes this an attractive approach.
Given the rarity of mycotic aneurysm, it is unlikely that the use of EVAR in this setting will ever be tested in randomised trials.

The use of EVAR in the first case enabled us to protect the transplanted kidney (anastomosed to left internal iliac artery) by performing a bypass from right common iliac artery without interrupting the blood supply to the transplanted kidney. In both cases, EVAR may have avoided the high morbidity and mortality often seen with open aortic surgery.

In conclusion, the treatment of abdominal aorta mycotic aneurysm is difficult, with high mortality. EVAR appears an alternative to open surgery.

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Deep inguinal infection after percutaneous coronary intervention

Ibrahim Sari, Vedat Davutoglu, Serdar Soydinc, Mehmet Aksoy

Abstract

A 62-year-old female underwent percutaneous coronary stent implantation of the left anterior descending artery without any complications except for a small haematoma in the right inguinal region where femoral artery puncture was performed. Forty days after discharge, she presented to the emergency department with a deep ulcerative wound in the femoral artery puncture site. The wound-site culture revealed methicillin-sensitive \textit{Staphylococcus aureus}. After appropriate antibiotic treatment and wound care, the deep inguinal infection healed with proper epithelisation and without any complications. This case underscores the importance of prevention and management of access site haematoma during percutaneous procedures.

Case report

A 62-year-old female underwent percutaneous coronary stent implantation of the left anterior descending artery without any complication except a small hematoma in the right inguinal region where femoral artery puncture was performed.

Forty days after discharge, she presented to the emergency department with a deep ulcerative wound in the femoral artery puncture site. There was an approximately 3.0 × 1.5 cm open, ulcerative purulent area in which cutaneous and subcutaneous tissues were destructed and surrounded by a hyperaemic area (Figure 1).

Figure 1. Deep ulcerative wound in the femoral artery puncture site
(Note the presence of an ulcerative purulent area in which cutaneous and subcutaneous tissues are destructed and surrounded by a hyperaemic area)
On physical examination she was completely normal. Right femoral arterial and venous Doppler examination was normal. Wound-site culture revealed methicillin-sensitive *Staphylococcus aureus* (MSSA). The plastic surgery department recommended medical treatment for primary healing of the wound site, and antibiotic treatment was arranged according to the antibiogram.

After appropriate antibiotic treatment and wound care, the deep inguinal infection healed with proper epithelisation and without any complications (Figure 2).

**Figure 2. Right inguinal region of the same patient after appropriate antibiotic treatment and wound care**

![Image of right inguinal region after treatment](image)

**Discussion**

Hospital-acquired infections are a major cause of morbidity and mortality throughout the world. Most hospital-acquired infections are associated with medical devices such as urinary catheters, central venous catheters, and/or diagnostic/therapeutic interventions.

Cardiac catheterisations are common medical procedures and are widely used for both diagnosis and treatment. Among possible complications of cardiac catheterisation, those related to the access site are relatively common. The majority of the complications related to access site in cardiac catheterisation are bleeding, haematoma, and pseudoaneurysm formation from punctured femoral arteries.\(^1\) However, groin infection after femoral artery catheterisation is unusual, occurring with an estimated frequency of less than 1%.\(^2\)

The causative agent of inguinal infection complicating cardiac catheterisation is usually *Staphylococcus aureus*.\(^3-6\) Early reuse of the initial puncture site, prolonged retention of the femoral sheath, bleeding or haematoma at the femoral sheath insertion site, pseudoaneurysm formation, and use of percutaneous suture mediated closure devices are mentioned to be as risk factors of development of infectious complications after cardiac catheterisation.\(^3-6\)
Proper antibiotic therapy will sometimes suffice as in our case, but surgery may also be required for treatment of this complication. The contralateral site is suggested when repeat catheterisation is indicated in a short time period. If the ipsilateral site is used, some authors recommend antibiotic prophylaxis.

In our case, development of the haematoma after percutaneous coronary intervention and a repeat procedure in the same site soon after the first were predisposing factors for infection development. Indeed, our patient stated that her right inguinal site remained purple and swollen and then a sinus formed in the centre of the swollen region thus suggesting abscess formation.

In conclusion, this case underscores the importance of prevention and management of access site haematoma during percutaneous procedures which can cause numerous complications.

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Wolff-Parkinson-White syndrome in association with tuberous sclerosis

Pazhanivel Mohan, Kunchithapatham Raghavan, Haritha Sagili

Abstract

Wolff-Parkinson-White syndrome occurring in association with tuberous sclerosis is rare in adults. Electrocardiography and echocardiography should be routinely performed in the assessment of patients with tuberous sclerosis as sudden cardiac death may be the first manifestation of Wolff–Parkinson–White syndrome in previously asymptomatic individuals. We report a case of Wolff-Parkinson-White syndrome in a 20-year-old female with tuberous sclerosis.

Tuberous sclerosis is an autosomal dominant disorder with genetic heterogeneity; it has a wide clinical spectrum of disease. Wolff-Parkinson-White syndrome occurring in association with tuberous sclerosis is rare in adults although it has been described in children with or without rhabdomyoma.\(^1\)

We present a case of Wolff-Parkinson-White syndrome in a 20-year-old female with tuberous sclerosis.

Case report

A 20-year-old girl presented with two episodes of acute onset generalised tonic clonic convulsions. There was no past history of similar episodes. Her younger sister was a known case of tuberous sclerosis on antiepileptics since childhood, but the rest of her family had not been screened at the time of diagnosis.

On examination, she had facial angiofibromas. The ultrasonogram of the right kidney showed multiple small echogenic areas in upper, mid, and lower poles—the largest measuring 0.7 × 0.8 cm in the posterior aspect of mid region consistent with angiomyolipoma. The left kidney also showed tiny echogenicities in the cortex (largest measuring 0.5 cm). A small cyst was seen in the left kidney measuring 0.5 cm (cortical cyst).

An electrocardiogram (ECG) carried out showed evidence of ventricular pre-excitation (Figure 1) and an echocardiogram did not reveal any intracardiac masses. CT scan of the brain showed subependymal calcifications. On confirmation of the diagnosis of tuberous sclerosis, she was started on regular anticonvulsants.
Discussion

Tuberous sclerosis is a genetic disorder affecting cellular differentiation and proliferation, resulting in hamartoma formation in many organs. The revised diagnostic criteria for tuberous sclerosis consist of major and minor features. Definite tuberous sclerosis complex is diagnosed by the presence of two major features or one major feature plus two minor features.

Skin lesions are found in 70–80% of cases. They include angiofibromas, periungual fibromas, shagreen patches, and ash leaf–shaped macules. The neurologic manifestations include epilepsy, cognitive disability, and neurobehavioural abnormalities. The renal lesions comprise of benign angiomyolipomas, malignant angiomyolipomas, cysts, oncocytomas, and renal cell carcinoma.

The most frequent cardiac manifestation is rhabdomyoma, which is also the commonest primary cardiac tumour of infancy and childhood. Cardiac examination may reveal rhabdomyomas in nearly 50 to 70% of infants with tuberous sclerosis. The tumours regress with increasing age.

Rhabdomyoma may present with cardiac failure in infancy and in association with various types of cardiac dysrhythmias such as atrial tachycardia, ventricular tachycardia, complete heart block, and Wolff–Parkinson–White syndrome. Wolff–Parkinson–White syndrome can be diagnosed during sinus rhythm from an ECG: there is a shortened PR interval and the QRS complex is deformed and widened in its initial portion by a slow rising slurred deflection called a delta wave.

However the association of Wolff–Parkinson–White syndrome and tuberous sclerosis in adults is rare and only two cases have been reported in literature. Ijaola et al
described it in a 24-year-old female who had palpitations and moderate cardiomegaly. Minquez et al\(^5\) reported the case of a 68-year-old patient with tuberous sclerosis whose first cardiac manifestation was a pre-excited atrial fibrillation. Our patient did not have any cardiac symptoms at presentation.

Wolff-Parkinson-White syndrome is relatively common, with a prevalence of 1 in 800 to 1 in 1000 unselected adult populations. As such it is unclear whether the association in this patient and the other two reports in adults are of a causal or coincidental nature.

The intermittent nature of the abnormalities makes it difficult to estimate the prevalence precisely. It has been shown to occur in 0.15% of the pediatric general population and in 0.5% of children with cardiac disease.\(^6\) Callaghan et al\(^1\) highlighted the association between Wolff-Parkinson-White syndrome and tuberous sclerosis in a series of 10 children. It has been presumed that rhabdomyomatous tissue traversing the atrioventricular junction acts as the accessory pathway bypassing the atrioventricular node.\(^2\)

All but one of their patients had echocardiographic evidence of cardiac rhabdomyomata. The higher rate of resolution of Wolff-Parkinson-White syndrome in tuberous sclerosis patients could be explained by the fact that rhabdomyomata are known to regress over time and therefore the accessory pathway may disappear or cease to conduct.\(^1\) However caution should be exercised about assuming causation, as the true incidence of Wolff-Parkinson-White syndrome is not well described in the general population of infants.

The splitting up of conduction tract by infiltrating tuberous sclerosis tissues may possibly result in accessory pathways in patients without rhabdomyoma.\(^4\) Although some patients may remain asymptomatic throughout their lives, others are prone to tachyarrhythmias that may be life-threatening.

Sudden cardiac death may be the first manifestation of Wolff–Parkinson–White syndrome in previously asymptomatic individuals.\(^8\) Hence, a high index of suspicion is required in patients with tuberous sclerosis. Electrocardiography and echocardiography should be routinely performed in the assessment of patients with tuberous sclerosis.

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Miss K., nearly 16. Parnell; typewriter, B—Building s, Queen Street. First seen on Friday afternoon, May 10th. A pale, anaemic-looking, ill-developed girl, who had always been in poor health.

The evening before had been to a dentist and had had a tooth extracted (a lower left molar). Came home feeling very much shaken and out of sorts, was restless during night, had stiffness in legs, and vomited once towards morning. When seen was slightly feverish, pulse normal. Said she felt out of sorts, but had no pain nor could give me any other symptoms. No abnormal physical signs in chest or abdomen. Site of extraction of tooth quite clean—no slough, no swelling of gums, cheek, or submaxillary glands.

Patient was apparently better on Saturday, according to mother, so I was not called in to see her again till Sunday afternoon between 5 and 6 o’clock. T. 102.5; P. 150. Patient looking very ill indeed, semi-delirious and had been delirious since morning, had vomited once. Urine dark, highly concentrated. Bowels had been opened by purgation. Patient complained of slight soreness in throat, but chiefly of pain in right groin.

On examination enlargement of right femoral gland—the other inguinal glands could with difficulty be felt owing to effusion beneath skin over Scarpa’s Triangle. There was no vaginal discharge. The site of extraction of tooth was again examined—no sloughing; no swelling of gums, no pain on pressure, no swelling of left cheek, nor in submaxillary region. There was slight injection of soft palate and fauces. I could get no history of rigors.

The mother was then informed that the patient was suffering from Septicaemia of some kind, and that the possibility of Bubonic Plague had to be considered. The patient was again visited about two hours later, and found to be in a dying condition. Immediately after death the skin was bluish and mottled, and later haemorrhage per vaginam was noticed. Dr. Lindsay arrived about 10 minutes after death, and agreed with regard to the suspicions of Bubonic Plague, and on his advice the matter was reported to Dr. Purdy.

Miss M., about 27, Parnell; worked with her elder sister, who had a millinery establishment in B—Buildings, Queen Street, City.

First seen on Tuesday evening, May 14th., 1907, at 9.30 o’clock. Had been attended by Dr. Marsack for Asthma; had lately returned from Cambridge much benefitted in health. T. 102°; P. 120. Dated her illness from Saturday, May 11th., 1907.
Had fainted on getting out of car in Parnell, but thought it was due to excitement of seeing a man attempt to get through the window of the car. Was in a drowsy condition on Saturday and Sunday. Vomiting commenced on Monday. Getting gradually worse until later on Tuesday could keep nothing on her stomach except lemon drinks. Felt feverish on Sunday—fever increasing each day. Had been restless on Monday and Tuesday, with pains in back and legs. Complained chiefly when seen of pain in right iliac region and in right groin.

On examination, enlargement of right femoral gland and of inguinal glands, with pain on pressure. Pressure on gland also seemed to increase pain in right iliac region. On percussing over the latter deep dulness could be obtained. There was no history of rigors. The remainder of abdomen normal. Heart normal. Lungs, breath sounds slightly harsh. No vaginal discharge.
An unusual case of leg ischaemia and congestive heart failure

Andrei M Beliaev

A 57-year-old woman presented with bilateral leg pain at rest, and orthopnoea. Five years ago she underwent coronary artery bypass grafting and intra-aortic balloon pump insertion via the right groin. The patient underwent a magnetic resonance angiography (MRA) procedure. See Figure 1.

Figure 1

What is the diagnosis, and what is the most appropriate treatment for this patient’s condition?
**Diagnosis**

Contrast-enhanced MRA revealed a diffuse narrowing of infra-renal aorta, high grade stenoses of both common iliac arteries (long arrow), and an early venous filling (short arrow) via the right profunda femoral arteriovenous fistula (AVF) (Figure 1). The diagnosis is *infra-renal aorto-iliac occlusive disease with the right groin AVF*.

**Most appropriate treatment**

The patient’s aorto-iliac occlusive disease was amenable to endoluminal stenting (Figure 2). An ultrasound-guided external compression as a method of AVF closure was not applicable because of the patient’s leg pain and the method’s high failure rate. A covered stent was contraindicated because it would obstruct the right superficial femoral artery. A catheter-directed embolisation was not feasible in this patient because the fistula was wide-based and short. Thus, the right femoral AVF was best dealt with surgically.

Following endoluminal repair of the aorto-iliacs and surgical repair of the fistula, the patient’s chronic limb ischaemia and congestive heart failure markedly improved.

**Figure 2**
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Anterior mediastinal mass
Turgay Celik, Atila Iyisoy, Hurkan Kursaklioglu, Ersoy Isik

Clinical

A 75-year-old female patient underwent diagnostic coronary angiography because of severe typical angina pectoris. Figure 1 was obtained during fluoroscopy.

Figure 1

What is the diagnosis, and what are the management problems?
Answer

*Porcelain ascending aorta*

Figure 1 shows heavy calcification encircling the aortic root and ascending aorta with dilatation of the aorta. On coronary angiography, multiple severe stenoses were found in all coronary arteries. Left ventriculography revealed global hypokinesis.

The porcelain ascending aorta makes coronary artery bypass or aortic surgery difficult due to problems with clamping and cannulation.

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Medical student loans

Student debt arising from their loans has been much in the news recently. Is this just a New Zealand problem? Apparently not.

The American Medical Association (AMA) recently asked the US Department of Education to extend medical student loan deferment based on economic hardship criteria. The AMA estimated the average debt burden for a medical school graduate is $130 571, while the average first-year stipend for a medical resident is $43 266. Bear in mind that these are US dollars. In New Zealand terms the debt is some $166 000.

JAMA 2007;298:2611

Occupational therapy after stroke

The authors of this paper point out that stroke is the second leading cause of death in the world and leading cause of serious, long term disability in adults; about half of those who survive are dependent on others for assistance with personal activities of daily living 6 months after the stroke.

Most clinicians strongly recommend occupational therapy (OT) for stroke victims. But does it work? This report concerns a systematic review of OT trials that focused on the improvement of the activities of daily living. The authors found 9 randomised controlled trials including 1258 participants which met the inclusion criteria.

They concluded that such OT techniques produced significantly better results. In terms of numbers they found that around 11 (95% confidence interval 7 to 30) people with stroke would need to be treated to avoid a poor outcome in one person.

BMJ 2007;335:922–5

Direct to consumer genome testing?

As of November 2007, two American companies have made available direct-to-consumer “personal genome services”.

In this paper, the authors discuss what (might) happen next. A patient turns up with a test report that states his genomic profile is consistent with an increased risk of both heart disease and diabetes. What are you going to do about it? Very difficult. You would have to tell him that most of the diseases listed in the direct-to-consumer testing companies (e.g. diabetes, various cancers, and heart disease) are so-called complex diseases thought to be caused by multiple gene variants, interactions among these variants, and interactions between variants and environmental factors.

So a few more tests and a discussion about lifestyle—avoid obesity and smoking and exercise more. We hope this technological advance is confined to North America.

Medical staff and the hospital

Medical staff and other health professionals have been progressively side-lined in the management of hospitals. These are now mostly micromanaged by top-down structures more attuned to political whims than public wishes. It should be no surprise that this dysfunctional type of management has coincided with a time when our public hospitals have lurched from crisis to crisis.

This from editor of the Medical Journal of Australia in commenting about and emergency department debacle in a hospital in New South Wales. He concludes by saying that health professionals’ views about and contributions to the management of their hospitals must be acknowledged and actively sought.

Sounds good.


Incidence and complication rates of herpes zoster (HZ)

Stimulated by the fact that in 2006 a vaccine to prevent herpes zoster (HZ) was licensed for use in older immunocompetent adults in the USA, this group decided to research the incidence (prevaccination) of HZ. The records of 125,000 residents of Olmsted County, Minnesota were scrutinised and the incidence of HZ was 3.6/1000 person-years. 1669 patients in all. 60% were women and 68% were 50 years of age or older. Only 8% were rated as immunocompromised. Postherpetic neuralgia occurred in 18% of adult patients with HZ and in 33% of those aged 79 years and older. Overall, 10% of all patients with HZ experienced 1 or more nonpain complications—such as ocular palsy or disseminated HZ.


Treatment of acute maxillary sinusitis

Acute sinusitis is a common clinical problem that usually results in a prescription for antibiotics, but the role of antibiotics is debated. Anti-inflammatory drugs such as topical steroids may also be beneficial.

Clinical doubt—hence this trial. 240 adult patients were randomised to 1 of 4 treatment groups; antibiotic and nasal steroid; placebo antibiotic and nasal steroid; antibiotic and placebo nasal steroid; placebo antibiotic, and placebo nasal steroid.

The active medications were 500 mg of amoxicillin 3 times per days for 7 days and 200 µg of budesonide in each nostril once per day for 10 days.

And as you suspected neither an antibiotic nor a topical steroid alone or in combination was effective as a treatment for acute sinusitis in the primary care setting. The paper was from Southampton, UK and an editorial commentator from Norway agreed with the results with some quibbles. The most significant of these being that obviously very sick subjects would need antibiotics.

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White-Tail Tales

The Australian spiders commonly known as white-tail spiders comprise two species in New Zealand: *Lampona cylindrata* (L. Koch) and *L. murina* (L. Koch), which have been present in the country for over 100 years.\(^1\,^2\) White-tail spiders are common household spiders and can be found throughout much of New Zealand, including areas as remote as the Kermadecs\(^1\) and the Chatham Islands (PJS, pers. obs).

Banks et al have previously discussed the media attention given to these spiders in New Zealand,\(^2\) with regard to their alleged effects on human health.\(^3\,^4\,^5\) Claims are frequently made of necrotic wounds caused by white-tail spider bites. However, evidence such as the collection of the organism responsible for the bite is invariably lacking.\(^6\)

Nonetheless, these spiders have been given an ‘official’ reputation in New Zealand. Medical doctors appear to blame these creatures for a number of skin ailments of unknown origin, possibly so that patients are able to receive adequate cover from the Accident Compensation Corporation (ACC).\(^2\) We are sceptical that all of the 22,000 ACC payouts for spider bites in the 2005–6 financial year are genuine cases.\(^7\)

This issue is not unique to New Zealand, and white-tail spiders have a bad reputation in their Australian homeland as well, also without medical evidence to substantiate the claims.\(^6\)

Isbister and Gray’s review of 130 definite cases of bites by *Lampona* spp. in Australia provided the following information:\(^8\)

- 95% of the bites occurred indoors [confirming the species’ synanthropic habits];
- Pain/discomfort occurred in all cases, and was severe in 27%;
- Other effects included puncture marks (17%), redness/red mark (83%) and itchiness (44%), with systemic effects occurring in 9% of victims;
- There were no cases of necrotic ulcers or confirmed infections;
- Median duration of effects was 24 hours;
- There were three distinct clinical patterns: pain only (21%), pain and red mark for < 24 hours (35%), and a persistent painful or irritating red lesion (44%).

These observations led the authors to conclude that “bites by *Lampona* spp. cause minor effects in most cases, or a persistent painful red lesion in almost half the cases. White-tail spider bites are very unlikely to cause necrotic ulcers.”\(^8\)

The authors’ conclusions are shared by Associate Professor Julian White (Head of Toxinology, Adelaide Women’s and Children’s Hospital), who vehemently criticised the “spurious diagnosis of white-tail spider bite necrosis”.\(^6\) White calls the unwarranted diagnosis of necrotising arachnidism and its attribution to white-tail spiders as “a prolonged and sad medical fable in Australia”, pointing out that this
problem had been “regrettably now exported beyond our [Australian] shores”, in
reference to similar claims in New Zealand.\textsuperscript{6}

Despite the publication of Banks et al’s article in 2004,\textsuperscript{2} necrotic wounds of unknown aetiology continue to be attributed to white-tail spider bites in New Zealand. The media also continues to spread the impression that skin ulcers of unknown origin are caused by these spiders,\textsuperscript{9} perpetuating the creatures’ undeserved reputation.

We regularly hear from people claiming a relative or friend was bitten by a white-tail spider and consequently experienced severe reactions. Typically no spider was seen, let alone collected for identification. Interestingly, in almost all cases, the supposed victim did not feel the ‘bite’. This contrasts with the evidence of the Australian study where pain/discomfort occurred in all cases.\textsuperscript{8}

It is important therefore, to adequately substantiate claims of necrotising arachnidism and other dermatological lesions or systemic effects regularly attributed to white-tail spider bites in New Zealand. The only way this can be confirmed is if the biting organism is collected and accurately identified. Specimens can be identified by staff at any of the main centre museums (Otago, Auckland, Canterbury, Te Papa) as well as Landcare Research in Auckland. However, we request that those spiders confirmed as having bitten a person should be sent to one of us to help us compile a more comprehensive picture of spider bite effects.

Spider specimens should ideally be preserved in a solution of 70\% ethanol and 30\% distilled water. It would also be helpful if these are adequately labelled with the date, locality (city, suburb) and location (indoors, backyard, etc) of collection. However, since most households are unlikely to have ethanol on hand, specimens may be kept in a freezer or even preserved in methylated spirits. Keeping them frozen would also preserve DNA for molecular identification,\textsuperscript{10} in case morphological identification is not possible.

It should be stressed that while white-tail spider bites are over-diagnosed, we in no way wish to make light of the very real suffering experienced by the victims in alleged spider bite cases. However, we feel that everyone would be better served by more accurate diagnoses. As White observed, “when presented with skin damage of initially uncertain origin, medical practitioners must look for all the many and varied non-spider-bite causes for such damage, leaving necrotising arachnidism as a diagnosis of last resort and uncertain validity after all other possibilities are excluded”. \textsuperscript{6} In the meantime, as the available scientific evidence indicates that severe reactions to white-tail spider bites are very unlikely, we request that health professionals refrain from perpetuating the myth about these spiders in New Zealand.

In the absence of a culprit, one may as well blame the ‘vicious attack’ on the ‘killer nine-inch nail’.
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Transmission of HBV from patient to healthcare worker

In January 2007 a support worker at a Hawke’s Bay residential facility for intellectually handicapped adults (Hawke’s Bay, New Zealand) received an unintentional bite from a client who was a known hepatitis B carrier. Serology the next day showed the worker to be hepatitis B virus (HBV) naïve, with no evidence of protective immunity. Although she reported receiving three doses of hepatitis B vaccine in 1999, no record of immunisation could be found by the worker’s employer or by the three general practitioners whom the worker had consulted in the previous 10 years.

The worker was unintentionally bitten again by the same client in May. It is not known whether the client had any bleeding mouth lesions at the time of the injuries. On neither occasion was vaccine or hepatitis B immune globulin offered by the general practitioner. Eight weeks after the second injury, investigations confirmed acute hepatitis B infection (anti-HBcore IgM strongly positive). She developed acute liver failure with confusion, acute renal failure, and severe coagulopathy (INR >5) so was transferred to the New Zealand Liver Unit for further management. She successfully underwent emergency liver transplantation 3 days later and has made an uneventful recovery. Sequencing of HBV isolates obtained from both the healthcare worker and the source demonstrated identical HBV genotypes, consistent with cross-infection.

This patient-to-healthcare worker transmission is assumed to be secondary to the bite sustained 3 months earlier. Salivary transmission has been documented previously.\(^1\text{-}^8\) Advice in the Ministry of Health’s Communicable Disease Control Manual (1998) and in its Immunisation Handbook (2006) are not specific concerning the need for post-exposure prophylaxis (PEP) following inoculation with saliva (as opposed to blood) from a hepatitis B carrier.

In the month following this presentation, the New Zealand Liver Transplant Unit admitted two further cases of acute liver failure from acute hepatitis B in nonimmune European adults. One case underwent emergency transplantation whilst the second died before a donor liver became available.

Acute hepatitis B infection is the most common cause of acute liver failure in New Zealand, with 8–12 such cases per annum. Almost all cases are adult Europeans. Prior to the introduction of neonatal vaccination in 1987, less than 10% of New Zealand European adults had protective immunity to HBV. In comparison, >70% Maori, Pacific Island, and Asian adults have natural immunity from childhood exposure.

Therefore, New Zealand Europeans born prior to 1987 and not involved in catch-up vaccination programmes remain at lifetime risk for acute HBV infection. Although the risk of chronicity is low in adult infection, 1% will die from acute liver failure. The risk factors for adult HBV infection are well recognised—sexual or other invasive contact (including biting) with HBsAg+ individual and injecting drug use (sharing with HBsAg+ individual).
We recommend that New Zealand publications advise that PEP is offered following all such incidents. PEP comprises combination of hepatitis B immunoglobulin 400 iu IM within 72 hours of exposure and hepatitis B vaccine within 7 days, repeated after 1 and 6 months.

We also recommend that all New Zealand adults at risk for HBV exposure (healthcare worker, new sexual partner, injecting drug user) have their HBV status checked, and if nonimmune, receive vaccination.

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References:
Cost-benefit analysis when the conclusion drives the method: a review of “Report on tobacco taxation in New Zealand”

As I wrote in opposition to the anti-tobacco lobby’s work on tobacco taxation in 2007, and particularly chided the citing of work not publicly available, it’s only fair that I give some attention to the their report now that they’ve unveiled the creature behind the curtain.

The SmokeFree Coalition argues that tobacco taxes should be increased substantially, that future increases be indexed to inflation, and that a dedicated portion of the revenues collected be put to programmes assisting individuals to quit smoking.

Parts of the report are sound. If the goal is to stop people from smoking, increasing tobacco taxes are an effective means to that end. For now, let’s take the report’s desiridata as given and consider whether a package of increased cigarette taxes, coupled with spending on anti-smoking initiatives, is an effective way of improving the lot of current smokers, and especially that of current poor smokers.

The report notes that such a policy would make current poor smokers who quit better off while making worse off those who continue to smoke. If the goal is to reduce the amount of smoking while minimising the harm done to low-income people who continue to smoke, directing any additional cigarette taxes raised to reductions in the income tax rate facing low income earners would seem a more efficient means to that end than the proposed tax-and-programmes package. As cigarette taxes are highly regressive and disproportionately hurt poor people, poor smokers who continue smoking would most likely prefer to get some of the tax increase back via reduced income taxes rather than have it fund further browbeating about their consumption decisions.

The authors estimate that some 39,000 non-quitting decile 1 households would spend an extra NZ$348 per year with a 20% rise in prices and that 37,000 non-quitting decile 1 households would spend an extra $928 per year with a 50% rise. These are not insignificant figures for low-decile households.

It would seem somewhat perverse to me if tobacco excise taxes were indexed to inflation, as per the report’s recommendation, while income tax bracket thresholds were not, but that’s something of a value judgment.

The biggest problems in the report are in the cost-benefit analysis. In my prior editorial, I emphasised the importance of noting the benefits of smoking as experienced by smokers. I do not smoke but I have it on good authority from smokers that they enjoy smoking. A supporter of the report, in response to my prior editorial, argued that I ought to discuss the benefits of smoking with a cancer patient. That one may suffer adverse health consequences from smoking does not mean that the smoker didn’t enjoy smoking. It’s perhaps reasonable to argue that smokers might be myopic.
and weigh too-heavily current pleasures against future pains, but we can’t assume away those pleasures. We have to have some way of measuring them.

The report makes some accounting for the benefits enjoyed by smokers, but, following Easton\textsuperscript{4} and Collins and Lapsley,\textsuperscript{5} subject to massive discounting. Collins and Lapsley deem those smoking more than 10 cigarettes per day to be addicted and, based on Australian figures provided in a 1989 study, tally 89\% of tobacco to be consumed by addicts. They use this estimate to add up the costs of addictive consumption: the value of real resources used to produce tobacco destined for addictive use. Easton then adds that those in the addictive categories enjoy no utility from smoking; he counts 11\% of cigarette expenditures in New Zealand as the benefits of smoking to smokers.

There are more than a few problems with this approach. First, total spending on a product necessarily comprises a lower bound estimate of the gross utility enjoyed by consumers. The standard economic approach is to estimate consumer surplus—that is to say, the utility one receives from consumption over and above the price one pays for the good consumed - based on the elasticity of demand and total purchases. The report estimates demand elasticity at -0.45, which means that a 1\% increase in the price of cigarettes is associated with a 0.45\% drop in consumption.

If per capita consumption is 1000 cigarettes with 25\% of the population being smokers, we have average consumption among smokers being 4000 cigarettes per year. At a price of $0.21 per cigarette and assuming linear demand functions, consumer surplus then is about $933 per smoker: $1773 in total enjoyment less $840 spent on cigarettes. This seems to me to be a lower bound estimate of consumer surplus as I would expect demand to become more inelastic with price increases: the demand curve would then follow a hyperbola lying above the linear curve here estimated, with a correspondingly larger area beneath it.

If we have 750,000 smokers in New Zealand, consumer surplus from smoking then totals about $700 million, again as a lower bound estimate. This sort of measure of consumer surplus is far more consistent with the literature estimating the benefits of smoking.\textsuperscript{6–8}

Moreover, discounting the utility of “addictive consumption” is fundamentally at odds with our basic theory on the matter,\textsuperscript{9} which defines addictive goods as those for which the utility of consumption is increasing in our prior stock of consumption. It seems rather odd to set equal to zero utility that ought to be increasing with consumption. For those who disagree with the standard model, though, net surplus is the appropriate measure to discount because of addiction, not gross surplus. Drive the consumer surplus down to zero if you must, but arguing that the net surplus is negative (less than the amount spent on cigarettes) tortures the method too much.

Finally, even if it were legitimate to discount entirely the consumption benefits of smoking for those smoking more than 10 cigarettes per day, we really don’t know whether the proportions of such smokers in Australia in the late 1980s correspond at all to the proportions in New Zealand today. There’s no attempt made to check the cross-country validity of an estimate taken almost 20 years ago, or even any
acknowledgement that applying such a measure 20 years later in a different country might be somewhat problematic.

After massively and inappropriately discounting the benefits of smoking, the report goes on to inflate the costs. The worst error here is including as a cost of smoking the real resources that go into tobacco production. If the report used a gross measure of consumer surplus, such an accounting would be fine.

Let’s take a simple example. Suppose that it costs $1 to produce an apple and you derive $1.50 in enjoyment from eating the apple. You pay $1 for the apple. We can then say that you receive $0.50 in net surplus or $1.50 in gross surplus. If we do a total accounting of the costs and benefits of apple eating and wish to include the $1 in apple production activities as a cost, we’d better use the gross measure of consumer surplus; it would be ridiculous to conclude that we’re worse off for the apple having been grown and eaten based on comparing a $0.50 net benefit to the consumer and a $1 cost of production.

In the case of cigarettes, consumers enjoy roughly $700 million in enjoyment from smoking over and above the $1.6 billion they spend on cigarettes. If we include $650 million in tobacco production costs on the cost side of the ledger, we have to use the gross measure of surplus on the benefits side: about $2.3 billion. You simply cannot honestly have a net measure on the benefit side and then double-count by including the resource cost on the cost side. In fairness to the authors, they seem here to have been led astray by Easton\(^4\) who uses similar method.

Similarly, we cannot count the health care costs on the cost side if we do not simultaneously include the tax revenues collected on the benefits side. While the tax revenues are a transfer and the health costs a real resource cost, the taxes were imposed precisely to offset those health costs. Indeed, tobacco taxes collected, at $980 million, dwarf health care expenditures of $350 million. As the report notes, “it does seem reasonably apparent that the tax contribution of approximately $1 billion annually by smokers exceeds substantially the external costs of smoking which fall on non-smokers. If savings on pension costs from premature mortality were added as well the net fiscal contribution of smokers, to the fiscal gain of non-smokers, would be further increased.” (Vol 1., p. 46)

The remaining costs fall almost entirely on the smokers themselves. But, smokers already have weighed these costs against the benefits of smoking: that they smoke is evidence that they find the benefits to outweigh those costs. Mortality, morbidity, loss of production from each of those causes, smoking-induced fires—all of these are costs borne by the smoker. Again, if we wish to include these on the cost side, we’d need to weigh them against a more comprehensive measure of the gross benefits. Such a measure of gross benefits would be rather in excess of the $2.3 billion I highlighted above.

In sum, the cost-benefit analysis presented is fundamentally unsound: its methods seem to have been chosen with the aim of maximising the monetised costs of tobacco use and minimising the monetised value of the utility derived by smokers from tobacco use. And why? The report explicitly states that the best case for increased tobacco taxes is that it reduces tobacco use and improves the health of those quitting
(Vol 1., p. 46); it then argues that tax increases ought to be presented and justified as a public health measure (Vol 1., p. 76) rather than as a way of internalising externalities, recovering costs on the health system, or because of the prior cost-benefit analysis.

Why distort the numbers if the numbers aren’t the basis for the policy recommendation? All of the cost-benefit analysis could be excised from the report and replaced with the simple and honest, albeit contestable, assertion that the authors know better what’s good for smokers than do the smokers themselves.

**Competing interests:** None.

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**References:**

Mother Teresa

"Mr B was an air force pilot in the World War 2" was how the 'caretaker' introduced Mr B to me. I was sitting on padded chairs in the tea room of a Picton (Marlborough, New Zealand) retirement home with two other medical students. We were on a mission to find out about the health system in Picton.

Mr B raised his hand and explained to me how it ended up in a cast. He fell while working at the local museum where he was advising on the air craft model they were building. "So accurate, they are," he told me "they know more about the air craft than I do." He was pleased with the models, but the fall "was the most painful experience" in his life. "There is no doubt about it," he assured me.

The accident happened on a Saturday afternoon. Bad timing. The Picton medical centre is only open 9 am to 5 pm, Monday to Friday, meaning they had to drive 25 minutes to go to Blenheim Hospital. "I've had it easy," he says, "my son lives in Picton, so he drives me to the hospital whenever I need to go there." Unfortunately many of the other residents don't have it so easy.

When we asked others at the retirement home about the road trip, they told us that it's inconvenient. It's not just the time, but also the taxi fare and "The effort!" they said, "A lot of it!"

For naïve medical student like us, the solution seemed straightforward: why not have a GP on call after hours?

"Because," Mr F, the financial adviser at the Medical Centre, answered our question like this…"It is just not financially viable." In a dry manner which contradicted his warm personality, he told us that it's not just the doctor who has to be paid. It's the receptionist, the power, and other things, anything, that cost.

"You see, young men, the public health is not a charity. What we have here is a business that provides customers with a service. Patients are paying for a 15 minutes consultation and, hopefully, improved health. We do not change patients' lives, we do not rescue their souls, and we do not show them the way. Our job description does not include those things."

Walking out of Mr F's office, it made perfect sense not to have an after hour service in Picton. The 25 minutes drive is, after all, nothing compared with the length that some people have to travel in other parts of the country to get their medical treatment.

Then I remembered one of the residents at the home reminiscing about good old days when it was run by the Catholic Church. "We used to have 24-hour care by three lovely church nurses." But when a businessman bought out the home, the three nurses were replaced by one 'caretaker'—still lovely—and the night became a big problem for the residents. "Now, I'm trying to move out," the same lady told us in a quieter voice, "but nobody will buy my unit because it has a stairway." She also added that even if she did find somebody to buy the unit, 25% of the sale went back to the
resthome, despite paying in full for the unit herself. "It may not be in bold, but it's in the contract that you signed."

On the ferry to Wellington, I'm talking to two fellow medical students. We have just finished our placements and are all looking forward to the 2 weeks break. We start talking about why we wanted to become doctors. We all agree that we all wanted to help people. We still do.

But we have started to see our limitation. The limit to our ability and willingness to help others. The reality hits you in the face; you can only do as much as your budget allows you, money talks louder than the heart. We are starting to realise that we cannot dedicate our whole lives to medicine, or the idea of medicine, the idea of helping others.

One of the classmates says, "You still need to have time for yourself. Time to spend with family, or time to play golf." The other agrees, "We are not Mother Teresas, we have to live our own lives too." And shrugs her shoulders.

I can't point out exactly why, but her statement leaves a bitter feeling in my stomach. It may not be in bold, but it’s in the contract that you signed.

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Herbert John Hall Hiddlestone

(CMG, MD, FRACP, FRCPE, MCCM, FPS, FNZMA; 1925–2008)

John Hiddlestone died in Nelson on 27 January, aged 82. He was born in Auckland, the son of Rev John Hiddlestone (a distinguished chaplain in World War 2), and educated at Auckland Grammar School.

When he was in the 4th form, his father was reported missing (believed killed) on Crete, just when his mother was undergoing radiotherapy for cancer, and the young John was mentored by a close friend of his father’s, Douglas Robb, who remained a friend throughout his later career.

He went to Otago Medical School, graduating in 1948. While there, he served on the OUMSA Executive for 6 years, two of them as President, and in 1947 he was awarded the Fowler Scholarship.

After residencies at Nelson Hospital he went to the UK in 1952, initially to Edinburgh, where he gained an MRCPE and joined the staff of the professorial chest unit and commenced research on the biochemistry of serous pleural effusions—the basis of his MD thesis.

He was appointed to London’s Brompton Hospital, then to the Sully Cardiothoracic Hospital in south Wales.

John returned to New Zealand in 1954, working as full-time specialist and area chest physician at Nelson Hospital, where in 1963 he was appointed medical superintendent. Three years later he was appointed Superintendent-in-Chief at Invercargill’s Kew Hospital, continuing work begun by Sir Charles Burns that led to a rejuvenation of services there.

In 1969 he joined the Health Department, becoming director of the Hospitals Division and, following the death of DP Kennedy in 1973, Director General of Health. He was deeply involved in health service reorganisation and served seven Ministers of Health until 1983. John remained a member of the NZMA throughout this time.

Two ministers in particular paid John great compliments. One was the Hon George Gair who, when an ex-Treasury official had drafted a bill to remove the requirement that the Director General be medically qualified, stated he “always felt better if accompanied by so distinguished a doctor as John Hiddlestone when fronting up to the NZMA”, and the bill was dropped.
In later years, looking back on the time when they had worked together, the Hon Aussie Malcolm wrote to John, “I respect you as a person and as an official, as much as I do anyone I have ever worked with. Your wise understanding of the bureaucracy and my youthful willingness to take on political challenges may have constituted one of the most successful health administrations. I regretted your departure and genuinely wish that we had both been around to tackle the challenges of the mid-1980s.”

When John retired after 10 years as Director General, two awards gave him particular pleasure. One was his election as a Fellow of the NZMA. Upon announcing this, the Chairman of Council Jeremy Hopkins wrote to him saying the award was in appreciation of John’s role as a bridge between the Association and the government. The other award was the first and only Fellowship of the Pharmaceutical Society. As Director General, John had valued strong relationships with many groups of health professionals, and the relationship with the Pharmaceutical Society was especially significant for him.

Like two of his predecessors, HB Turbott and DP Kennedy, John played a major role representing New Zealand in the World Health Organization (WHO). He was Vice-President of the World Health Assembly in 1975, elected to the Executive Board of WHO, and appointed Chairman in 1982.

While Chairman, he took a leading role in dealing with a problem concerning the Arab states and Israel, and was subsequently invited by Dr Halfdan Mahler, Director General of WHO, to become Director of Health and WHO Representative to the United Nations Relief and Works Agency (UNRWA), the agency caring for 2.4 million Palestinian refugees in Lebanon, Syria, Jordan, the West Bank, and the Gaza Strip. He served 5½ years in this post, instigating and seeing through considerable improvements to the provision of health services to refugees.

John was elected to the board of the International Hospitals Federation and, in 1983, invested at Buckingham Palace as Companion of The Most Distinguished Order of Saint Michael and Saint George.

In 1988 John retired and returned to New Zealand, where he took on a great deal of voluntary work. He became national president of NSAD, chairman of the board of the Home of Compassion, and a member of the board of the Te Omanga Hospice. A devoted freemason for over 50 years, he was appointed President of the Board of Benevolence, taking particular interest in the Freemasons Chair of Geriatric Medicine in Auckland, paediatric research based at Otago Medical School, and support of research by the Auckland Medical Research Foundation. At the time of his death, he was Past Deputy Grand Master of the Grand Lodge of New Zealand.

John was also a keen Rotarian for over 50 years and a member of two Probus clubs. He was president of the Nelson, Invercargill South, and Wellington Rotary clubs and received Rotary’s highest recognition as a Sapphire Paul Harris Fellow. He was president of Khandallah Probus and later the Trafalgar club in Nelson.

A son of the manse, John attended Presbyterian churches in Nelson, Invercargill, and Wellington. He later joined Nelson Cathedral, where he was an official guide, sidesperson, and reader.
John married Rosina Maclean, a nurse, in 1949 and their life together was a wonderful partnership. John is survived by Rosina, their 5 children, and 12 grandchildren. John Hiddlestone wrote this obituary prior to his death. The Hiddlestone family and John Ryder also provided assistance.
Bernard (Bernie) Vance Kyle

MB, ChB (NZ), DGO (Melb), FRCS, FRACS, FRCOG, FRNZCOG

Bernie Kyle died recently following a long and distressing illness. He was educated at Palmerston North Boys High School and Otago University, and was a resident at Knox College.

He was a successful all-round student, representing Otago University, Otago Province, and NZ Universities (captain) in hockey from 1944–46.

Bernie graduated MB, ChB in 1947, having topped his year in O&G (with distinction). He spent his two house surgeon years in Wellington and it was here that he met his wife Joan.

During his second year in Wellington he worked for Ken Pacey, New Zealand’s leading gynaecologist. At this time, he was contacted by the Otago Dean, Charles Hercus suggesting he should apply for the NZ Travelling O&G Scholarship—which he was subsequently awarded.

In 1950, Bernie and Joan moved to Melbourne and he took up his scholarship at the Royal Women’s Hospital. Bernie regarded his training in Melbourne as having the greatest influence on his future career. While there he witnessed the benefits of heroin in labour, and, on returning to Auckland, persuaded the obstetricians to introduce this.

In 1952, he was appointed assistant to Spence Smythe, the first professor in the newly established Postgraduate School in O&G housed at the American 39th General Hospital which later became Cornwall Hospital.

He then travelled to England as a ship’s doctor and spent 2 years gaining further experience and postgraduate qualifications, an FRCS (England) and MRCOG. They didn’t venture to Europe but were fortunate to have tickets to the NZ stand to watch Queen Elizabeth II’s coronation where they learnt of Hillary’s conquest of Mt Everest.

In 1955, Harvey Carey the second professor requested that he urgently return from England to be assistant medical director. Bernie flew home leaving Joan to return by ship. That year he performed the first vaginal hysterectomy and second radical vulvectomy in Auckland. The following year he was appointed to part-time University and Auckland Hospital Board positions.
Bernie was a conscientious practical obstetrician, working long hours and always being available for his patients. He was one of the first doctors to have a radiotelephone installed in his car.

Sir Douglas Robb, ever keen to promote local surgeons, encouraged Kyle to apply for the vacant postgraduate chair in 1963, but he declined having already established himself in private practice (subsequently John Taylor joined him). Had he been appointed to the Chair the history of National Women’s would almost certainly have taken a different course.

Within the hospital a working party of three local clinicians was set up to examine concerns raised regarding Associate Professor Green’s management of carcinoma in situ of the cervix. Kyle twice declined nomination, suggesting an independent committee “because of personalities and the fact that local staff have had a long time in which to prejudge the issue.”

Judge Cartwright observed that if Dr Kyle’s recommendations had been acted upon, the subsequent public inquiry would have been unnecessary. The National Women’s Hospital (NWH) Medical Committee in the 1960–1980s period was dominated by strong personalities, often based on sectional interests, in particular “town versus gown”. Kyle’s intellect, commonsense and ability to clearly express the wisest approach to contentious issues were often persuasive though not always heeded.

Kyle worked in the old “B Team” at NWH (mainly with Bruce Grieve, Ron Elvidge, and Ian Ronayne). He was an astute clinician and an excellent though conservative surgeon. His teaching ward rounds provided an ideal base for those preparing for higher examinations, and his knowledge and wealth of practical experience was widely sought. General practitioners commented on his thoughtful, well considered opinions and scholarly correspondence.

Bernie Kyle served on the RCOG (NZ) Council, numerous Auckland Hospital/Board committees, and on the Rawhiti Hospital Trust for many years.

Beyond medicine, Bernie’s family and home always came first. He rarely ventured far beyond the Akarana Golf Course and his bach at Hatfields Beach where he enjoyed boating and fishing.

Joan devoted unstinting attention to him during his last 15 years as his rare and relentless IBM (inclusion body myositis) progressed. Bernie’s declining health was matched by his increasing computer skills leading to increased contacts (with many a wry comment) to friends and colleagues. During this time he developed an interest in writing poetry.

Bernie will be remembered by many thousands of women whose babies he delivered and a generation of house surgeons and registrars at NWH. To Joan, his sons Cam (pathologist), John (general practice), and Alastair (optometrist), we extend our sincere sympathy.

Ron Jones and David Cole wrote this obituary.
Medical Self-Regulation: Crisis and Change

Mark Davies. Published by Ashgate (Hampshire, UK), 2007. ISBN 9780754644590. Contains 432 pages (hardback). Price £70.00

This easy-to-read and comprehensive book examines the “crisis” within medical self-regulation since the formation of the General Medical Council (GMC) in 1858. High-profile cases such as the Bristol Royal Infirmary Inquiry into paediatric cardiac surgery have lead to a failure in public confidence in the medical profession’s ability to safely (for the patients) self-regulate.

Written in three parts, Mark Davies sets the scene by describing the development and framework of medical regulation in the United Kingdom. Extensively covering the GMC and National Health Service (NHS) processes for complaints as well as how doctors as individuals and grouped together have responded.

The second part critically examines (in the form of case reports) failings in the system. These provide insightful and thought-provoking views from a non-medical perspective of where medical regulatory systems have broken down. In the final section the proposed changes to medical regulation are reviewed along with suggestions for alternative models discussed.

This book written by a senior lecturer in law is as one would expect meticulously referenced and footnoted. The clarity of purpose however is rare and, while a lengthy argument at 432 pages, its readability makes these pages seem less.

This book the first in a planned series of books on medical law and ethics bodes well for the rest of the series in terms of depth of review, academic robustness and ability to be read cover to cover. It should appeal to regulators, physicians, lawyers, and lay readers alike.

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Practical Paediatrics (6th edition)


Practical Paediatrics is one of the core paediatric text books for medical students and postgraduate students studying for Diploma of Child Health. This latest edition continues the tradition of offering a solid and broad knowledge base in paediatrics and child health delivered in an easy reading style.

There are Clinical Example boxes scattered within chapters that help keep the information clinically relevant, and the Practical Points boxes neatly summarise the chapter or section information.

This text has advantages over the alternatives (which are from the United Kingdom or the United States) by having local statistics—a lot of the statistics are actually Australian but still more relevant to New Zealand than European or North American figures.

The chapter on SIDS (Sudden Infant Death Syndrome) has New Zealand data and is very relevant locally. There is also a chapter on Indigenous Culture and Health which includes a section on Māori View of Child Health and Illness. There is an interesting chapter on Poisoning and Envenomation, and an excellent chapter on Electronic Learning Resources.

The chapters are well organised but it would have been easier to navigate the book if different colours had been used to delineate chapters. Indeed, the monochromic look is not as inviting as some other multicolour textbooks.

One of the best features is the online access which has the full text plus additional features. The additional features include questions in both review format, helpful for revision, and also questions in multi-choice test format with a timer—again a very useful feature for anyone sitting paediatric or child health examinations. There is also a drug database and an image library. This is very good value for money.

Overall, this is an excellent textbook that is very relevant to undergraduate and postgraduate students studying paediatrics and child health.

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