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# This Issue in the Journal

**The impact of New Zealand CVD risk chart adjustments for family history and ethnicity on eligibility for treatment (PREDICT CVD-5)**
*Susan Wells, Andrew Kerr, Joanna Broad, Tania Riddell, Tim Kenealy, Rod Jackson*

Established New Zealand best-practice guidelines advocate an assessment of the risk of a heart attack or stroke in the next 5 years. By doing this, doctors and nurses can identify adults who have moderate or high risk and offer them effective treatments whilst those at low risk would receive general heart health promoting advice. For some population groups such as people with a family history of early cardiovascular disease and people of Māori, Pacific, or Indian ethnicity, the assessment test (developed in USA many years ago) is thought to underestimate their actual risk. So the guidelines recommended a small upward risk adjustment to their calculated risk. We investigated the impact of this adjustment and found that a relatively small change in risk assessment had a substantial potential impact on health care resources. We recommend an economic review of this adjustment but more pressing is the need for New Zealand-specific risk assessment tools to be developed.

**Issues in the assessment of cardiovascular risk in selected general practices in Canterbury, New Zealand**
*Ian Sheerin, Greg Hamilton, Alistair Humphrey, Alf Scragg*

Patient records were audited in three selected general practices to assess what is currently happening compared with recommended guidelines for assessment of risk of heart disease. There were significant differences in the type and coverage of information that was recorded, even for patients with diagnosed heart disease. Some practices relied heavily on paper medical record systems that made it difficult to find key information. Only one of the three general practices had a fully computerised medical record system that was capable of retrieving information and systematically inviting patients for follow-up. It is necessary to improve information in primary care, before it is practical to undertake systematic screening for risk of heart disease. Such systematic screening has the potential to significantly reduce risk of coronary events such as heart attacks, angina, and stroke.

**Accuracy of the Wells Rule in diagnosing deep vein thrombosis in primary health care**
*Maelen Tagelagi, C Raina Elley*

Deep vein thromboses (DVTs) are difficult to diagnose because the symptoms, like pain and swelling of the calf, can be caused by a number of other less serious conditions. While most people with these symptoms do not have DVT, ultrasound investigation is often required to rule out DVT. Ultrasound is expensive and inconvenient to patients, most of whom do not have DVT. Therefore, a systematic

clinical scoring system (the Wells Rule) was developed to identify which patients are very unlikely to have DVT, so that fewer patients would need unnecessary ultrasound investigations. While the Wells Rule has been found to be predictive and useful in secondary care settings, this study found that the Wells Rule, alone, was not good at ruling out DVT when used in general practice.

**Survival over 5 years in the initial hospital survivors with acute coronary syndrome: a comparison between a community hospital and a tertiary hospital in New Zealand**

*Cheuk-Kit Wong, Eng Wei Tang, Peter Herbison*

There was a difference in long-term outcome between survivors with a heart attack from a regional hospital and from a tertiary hospital in Otago / Southland in the years 2000 to 2002. Patients discharged from Invercargill Hospital fare worse over the first 2 years and tended to fare worse over the first 5 years. This was due both to their higher baseline risk and the under-use of statins at discharge. Of note, PHARMAC rules for statins only changed around the end of the study period allowing more liberal use of statins.

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# Reducing the lifetime cardiovascular risk of New Zealanders

Ralph A Stewart, Harvey D White

New Zealand guidelines recommend screening of cardiovascular risk factors for men older than 45 and women older than 55 years, and then targeting preventive treatments to those with a greater than 15–20% 5-year risk of a cardiovascular event.[1] Lifestyle advice is usually recommended for individuals with risk factors who have a lower 5-year risk.

However a recently published analysis from the Framingham study[2]—which estimates the lifetime risk of cardiovascular disease according to the risk factor burden at age 50—raises questions about this approach. In this study, men with ≥2 risk factors had a lifetime cardiovascular risk of almost 70% and median survival was 11 years shorter than for men with no risk factors. Equivalent figures for women with ≥2 risk factors were a 50% lifetime risk and a decrease in survival of 8 years.

A striking observation was that many individuals with a high lifetime risk had a low 5-year cardiovascular risk in middle age. For example a sedentary 45-year-old man with abdominal obesity, impaired glucose tolerance, a total to HDL cholesterol ratio of 7.0, and blood pressure of 160/95 mmHg would have a 5-year cardiovascular risk from the New Zealand risk charts of <5%,[1] but a lifetime risk from Framingham data of ~50%.

Based on current guidelines, most health professionals would offer lifestyle advice alone. However lifestyle advice which targets conventional cardiovascular risk factors has little impact in many patients. In a meta-analysis of clinical trials of multiple risk factor interventions for primary prevention, the pooled odds ratio for total mortality was 0.96 (95% confidence interval: 0.92–1.01). The mean reduction in systolic blood pressure was only 3.6 mmHg, and total cholesterol decreased by only 0.07 mmol/L.[3]

These small benefits compare poorly with those of statins, antihypertensive medication, and aspirin which have been proven to reduce cardiovascular mortality in large randomised clinical trials, and in combination more than halve cardiovascular risk.[4]

While lifestyle changes often have little impact on serum cholesterol and blood pressure levels, they reduce cardiovascular risk in other ways. For instance, epidemiological studies consistently show strong associations between regular exercise and reduced cardiovascular mortality.[5,6] Furthermore, improved diet and increased exercise has been shown in clinical trials to decrease the risk of diabetes.[7] These lifestyle changes if maintained over time are likely to translate to substantial reductions in cardiovascular mortality.

However, successful lifestyle changes require considerable motivation. It is likely that indicating to the average 45-year-old with multiple risk factors that the lifetime risk of a heart attack or stroke is more than 50% and life expectancy may be decreased by 10 years is more persuasive than indicating that the chance of a heart attack or stroke in the next 5 years is 5%.

Estimates of lifetime cardiovascular risk are also relevant to population-based strategies which aim to reduce the burden of cardiovascular disease. A striking observation from the Framingham report was that individuals with no risk factors at age 50 had a lifetime risk of cardiovascular disease of only ~5%.[2] However less than 5% of the study population were in this low-risk group.

Similar observations have been made in other large cohort studies[8] and are likely to apply in New Zealand. The public health message is that reducing the risk factor burden by age 50 is likely to translate to reductions in lifetime cardiovascular risk and greater life expectancy. Once acquired, adverse lifestyle risk factors are hard to change so prevention needs to start early and be maintained throughout adulthood.

Successful population-based strategies have significantly reduced smoking rates in New Zealand, but changing the diet and reducing obesity among the population remain huge challenges.

Town planners could give greater priority to environmental changes which encourage walking and cycling rather than those which encourage people to use their car as the sole mode of transport.

Legislation to eliminate industrially produced trans fatty acids from food supplies would, in one US estimate, decrease cardiovascular mortality by 10 to 20%. Indeed, such legislation has already been implemented in Denmark.[9]

Encouraging healthy eating, regular exercise, and avoidance of smoking in schools (if translated to less obesity and a healthier lifestyle during adult life) may also substantially improve the life expectancy of many New Zealanders.

Another strategy, discussed in a systematic literature review by Novak and colleagues in this issue of the *NZMJ*,[10] is to promote cardiovascular health in 'blue collar' workplaces such as factories. This strategy is potentially attractive because lifestyle changes are encouraged in lower socioeconomic groups and in Māori and Pacific peoples who often have a high lifetime risk of cardiovascular disease.

Limited evidence suggests that workplace interventions are more effective than community-based attempts to change individual behaviour. However few studies of cardiovascular prevention in workplaces have been conducted in New Zealand, and Novak and colleagues argue that more research is needed.

The use of risk scores to target preventive treatments to individuals at greatest risk was pioneered in New Zealand, and has been an important advance when compared to treatment of individual risk factors irrespective of the absolute risk of disease.[11]

However a focus on 5-year cardiovascular risk underestimates the cumulative burden of cardiovascular disease which occurs with increasing age. This risk is greater in Māori, Pacific peoples, and lower socioeconomic groups, who have a higher prevalence of lifestyle cardiovascular risk factors throughout life.

We believe it is time to move toward lifetime risk assessment.

**Competing interests:** None.

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# Trading PREDICTions

Brian Cox

Disease progression, host response, and the effects of ageing vary among individuals. When these individual variations are small, such as when the disease has almost completely overcome the body's defences, empirical observation of only a few cases allows an accurate prediction of the outcome. In other circumstances, the prediction of outcome is likely to be less accurate. However, some comparison, with and without treatment, of the predicted outcome of disease does guide clinical decisions.

Models such as the SCORE,[1] DECODE,[2] or PREDICT[3] risk equations enable some overall assessment of the chance of cardiovascular disease (CVD) events by incorporating various risk factors and their interactions. As the human mind cannot easily do all of these calculations, the models are valuable and accepted tools that assist clinical decision-making.[4] These models are most useful for individuals who have multiple risk factors, rather than a single risk factor, for CVD.[4]

The PREDICT model attempts to provide the probability of a CVD event for an individual. It uses the magnitude of the association of known risk factors and fatal CVD identified in the Framingham study population to aid decisions about a person's suitability for treatment. Risk equations for CVD from the Framingham study have been found in some populations to be reasonably accurate, but in some population groups they do not perform as well.[4] Coefficients for risk equations are derived by maximising their capacity to explain the observed outcomes in the particular population analysed, but the accuracy of the same coefficients to predict outcomes in other datasets, including further data from the same population, is almost always reduced.

To assess this reduction in accuracy requires comparing observed outcomes in a new population group or data set with prior predictions. As the modellers indicate, the PREDICT model requires such a validation. Similar prediction scores overseas have had mixed success[4–7] and the Framingham study has not provided reliable risk equations for the risk of CVD from Type 2 diabetes,[8] possibly because there were relatively small numbers of Type 2 diabetics in the cohort studied.

This has prompted the development of guidelines for the computer modelling of diabetes and its complications.[9] While these models tend not to use modern advanced statistical techniques for prediction, such techniques[10] may improve their predictive capability.

Some risk scores overestimate the chance of fatal cardiovascular disease. In a United Kingdom cohort of people with Type 2 diabetes, such scores were found to be erratic in predicting the 10-year likelihood of fatal events. Different scoring systems were better for male versus female, Caucasian versus non-Caucasian, and diabetic versus non-diabetic patients.[8]

The relatively poor performance of risk equations for Type 2 diabetics is thought to be partly due to its inclusion as a dichotomous variable. It has been suggested that the

inclusion of graded measures of glycaemic control or severity may provide more accurate risk scores.

The imprecision of applying to individuals estimates of risk that are derived from populations has been well described.[4,11] The blunt approach of adding 5% because of Māori, Pacific (mostly Samoan, Tongan, Niuean, or Cook Islands origin), or Indian ethnicity; the presence of a family history of premature CVD; or a high risk of diabetes appears to acknowledge this fundamental problem, but may not be the wisest solution.

There are major resource implications from this adaptation of the use of the PREDICT model, with an increase of about 20% in drug treatment, intensive lifestyle management (referral to a dietician as well as individualised assessment and counselling), and diabetic referral and an increase of 50% for individualised lifestyle treatment and counselling in primary care.[3]

So, whose treatment should be deferred for this increase in the management of CVD? The answer is not clear. The use of the PREDICT risk equation, to estimate the likely change in case load for the health service from an adaptation in the use of the risk score, is entirely compatible with its epidemiological derivation and the case load estimates are not as imprecise as the risk estimated by the model for an individual.

One of the aims of treatment is to minimise the chance of adverse events, but trying to predict the outcome for individuals is often fraught. Some of the benefits of medical care, including some of the reduction in mortality from CVD, result from the treatment of individuals at relatively low risk of such events. Treatment of individuals at low risk is sometimes labelled “over treatment”.

When the prediction of the outcomes of disease is poor, all patients need to be treated as if they are at significant risk, including those patients that are initially thought to be at low risk of an adverse event, providing the potential harm from treatment is even lower. This underpins many of the beneficial effects of medical practice and while resources do not always enable the treatment of all patients, it is also important that professional standards are not compromised to the point that patients who have a reasonable chance of benefiting from treatment are denied it.

The use of scores, such as PREDICT, to determine eligibility for treatment, as opposed to guidance in clinical decision-making, requires rigorous validation. Accurate measurement of the sensitivity and specificity of such screening tests are required. A randomised controlled trial of the outcomes from the use of the PREDICT risk assessment in the New Zealand health service setting appears to be warranted, especially if some patients were having treatment withheld due to a low risk score.

The difference between the recording of ethnicity in national and general practice databases, with the National Health Index database tending to overestimate the prevalence of Type 2 diabetes in Māori, may also need to be addressed to ensure accurate follow-up information is obtained.[12]

As usual, it is the relative merit of benefits, harms, and available resources that is under debate. Despite the need for further refinements, the PREDICT risk assessment tool and its adjustments are very helpful in the pursuit of a rational, socially acceptable, and professionally acceptable response to cardiovascular disease. However, these can on occasion be forces that are difficult to reconcile.

**Competing interests:** None.

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# The impact of New Zealand CVD risk chart adjustments for family history and ethnicity on eligibility for treatment (PREDICT CVD-5)

Susan Wells, Andrew Kerr, Joanna Broad, Tania Riddell, Tim Kenealy, Rod Jackson

**Abstract**

**Aims** Current New Zealand cardiovascular (CVD) risk management guidelines advocate targeting treatment to patients with a high 5-year CVD risk assessed using a calculator derived from the Framingham Heart Study. For some high-risk population subgroups, a 5% upward adjustment to their calculated 5-year CVD risk is recommended. We estimated the impact of these adjustments on eligibility for treatment in a primary care setting.

**Methods** Between 2002 and 2006, 23,709 patients visting their primary care provider in Auckland, New Zealand had CVD risk assessments as part of an opportunistic screening programme using PREDICT, a web-based clinical decision support system. We calculated their baseline CVD risk with and without the 5% upward adjustment for family history of premature ischaemic CVD or for being of Māori, Pacific or Indian subcontinent ethnicity.

**Results** A baseline CVD risk could be calculated for 23,693 (99.9%) patients of whom 90% were between ages 35 and 74 years. Unadjusted risk scores classified the majority (70%) below the 10% 5-year risk threshold for specific individualised treatment. A further 11% were between 10 to 15% risk (recommended to receive individualised lifestyle counselling in general practice) and 19% had a greater than 15% risk ( recommended for drug treatment and referral to a dietician in addition to individualised lifestyle counselling). Over a quarter of patients were recorded as having a premature family history of CVD; 21% were Māori, Pacific, or Indian subcontinent and thus met the criteria for a single 5% upward adjustment. This increased the number of people eligible for drug treatment, intensive lifestyle management, and dietician referral by approximately 20% and individualised lifestyle assessment and counselling by 50%.

**Conclusions** The upward adjustments to the calculated CVD risk recommended by the New Zealand CVD risk management guidelines has the potential to substantially increase resource requirements for CVD preventive services in primary care. Moreover the true impact is likely to be underestimated given other adjustment factors related to diabetes risk that were not available in this dataset. Given the impact of these relatively small changes to the CVD risk calculator, locally developed and validated risk equations are urgently needed.

Current New Zealand CVD risk management guidelines (2003) recommend a quantitative CVD risk assessment for all men over the age of 45 years and women over 55 years; 10 years earlier for people of Māori, Pacific or Indian subcontinent ethnicity or if patients have known CVD risk factors or are at high risk of developing diabetes.[1]

Absolute 5-year CVD risk or the likelihood of a fatal or non-fatal cardiovascular event (including new onset angina, myocardial infarction, stroke, transient ischaemic attack, peripheral vascular disease, and left ventricular failure) in the next 5 years is estimated using a Framingham Heart Study prediction equation.[2] This equation takes into account the synergistic effect of major risk factors and includes age, gender, blood pressure, total and HDL cholesterol, smoking, and diabetes status.[2]

For some population groups, a once only 5% upward addition to their calculated 5-year CVD risk is recommended as the guideline developers considered that their calculated absolute risk was likely to be underestimated by the Framingham equation.[1] These high-risk groups include; people with a premature family history of coronary heart disease or ischaemic stroke; Māori and Pacific people; people from the Indian subcontinent; and patients with diabetes who have microabuminuria; Type 2 diabetes for at least 10 years or who have an HbA1c consistently over 8%; or people with metabolic syndrome.

The rationale for adjusting risk was based on evidence suggesting an increased risk of cardiovascular disease in these subpopulations compared with the general population, over and above their calculated CVD risk.[1]The adjustment was set at 5% for pragmatic reasons because at the time the guidelines were developed, the risk charts that most general practitioners used for CVD risk assessment, displayed estimated risk in 5% increments (Personal Communication, Rod Jackson, 2006).

Management recommendations from the New Zealand CVD guidelines are based primarily on the patient's calculated (and adjusted) absolute 5-year CVD risk and involve general lifestyle advice for those at less than 10% risk, individualised lifestyle assessment and counselling in primary care for those between 10–15% risk and drug treatment together with intensive lifestyle management (referral to a dietician as well as individualised assessment and counselling) for those at high risk (calculated risk ≥15%; or patients with a prior history of a CVD event who are assumed to be at >20% risk).

Whilst intensive treatment has the potential to more than halve future CVD events[4] and will be more cost-effective if targeted to the highest risk patients, direct costs to the health service due to additional drug treatment and other preventive services will accrue earlier and must be planned for. We therefore estimated the potential impact of upward risk adjustment for ethnicity and family history on the number of people eligible for drug treatment and specific lifestyle interventions using data from people risk assessed via an opportunistic CVD screening programme conducted in routine primary care.

## Methods

From August 2002, ProCare Health Ltd, a large Auckland primary care organisation, implemented an opportunistic CVD risk assessment and management programme.[5,6] This programme used PREDICT, a web-based real-time decision support programme, that was integrated with the most commonly used practice management system software (MedTech). The programme is delivered as a window within a patient medical record in the same manner as other templates within the patient management system (PMS).

The integration allowed coded CVD risk data to be automatically extracted from a patient's electronic medical record and put into the PREDICT web template. Other data that were not available in the medical record were entered by the general practitioner or practice nurse. These risk profiles were automatically sent via a secure internet connection to a central server. Within seconds the clinician

 

received the patient's calculated 5-year CVD risk as well as evidence-based risk management recommendations derived from New Zealand CVD guidelines. Meanwhile, the central server stored the CVD risk factor profile for each patient and with permission from ProCare were able to be extracted anonymously.

From these patient profiles, we derived a first recorded (baseline) risk estimate for each patient using the New Zealand CVD risk assessment criteria. Those who had had a previous CVD event were classified as being clinically at high risk without using the risk calculator. For all other patients we estimated their risk of a cardiovascular event in the next 5 years according to the Framingham equation.[3] The model includes gender, age, systolic blood pressure, smoking, total cholesterol:HDL ratio, and diabetes.

Valid ranges for the physiological parameters were determined *a priori* according to population based data from New Zealand epidemiological surveys and Diagnostic Medlab, New Zealand's largest community based pathology laboratory (S Wells and J Broad, unpublished report, 2005). Records containing values outside of the valid ranges were removed from the analyses. We then applied the recommended 5% upward adjustment (once only) if the patient's ethnicity was Māori, Pacific, or Indian subcontinent or the patient had a family history of premature ischaemic coronary heart disease or stroke (a first-degree male relative before the age of 55 years or a first-degree female relative before the age of 65 years). Pacific peoples were defined according to New Zealand Health Information Service ethnicity data protocols[7] as having Level 2 codes 31 to 37 and Indian subcontinent defined as Level 2 codes 43 and 44 excluding Japanese and Korean.

As the PREDICT module used in ProCare until 2006 was developed prior to the publication of the 2003 New Zealand Guidelines for the assessment and management of cardiovascular risk,[1] data on the additional risk adjustment groups (related to metabolic syndrome and other diabetes risk) were not available for these analyses.

All analyses were conducted using SAS (version 9.1) software.

The PREDICT research project was approved by the Auckland Ethics Committee (AKY/03/12/314).

## Results

Between 2002 and 2006, 41,451 CVD risk assessments were undertaken for 23,709 patients. These assessments were undertaken by 407 GPs and 89 practice nurses. A baseline (unadjusted) CVD risk could be derived for 23,693 (99.9%) patients with 16 patients having data that was outside valid ranges. A profile of those risk assessed by age, gender and ethnicity is shown in Table 1.

Of those risk assessed, 90% were between ages 35 and 74 years. The majority (70%) were below 10% 5-year risk, 11% were between 10 to 15% risk, and 8% had a calculated CVD risk greater than 15%. A further 11% had a prior history of a cardiovascular event (assumed 5-year risk >20%). Three-quarters of those assessed were European & Other (e.g. Middle Eastern, African) ethnicity, 7% were Māori, 11% were Pacific, 3% were Indian subcontinent, and 4% were Asian.

Compared to European & Other, Māori (odds ratio [OR] 1.33; 95% confidence interval [CI] 1.22–1.51) and Pacific (OR 1.33; CI 1.22–1.46) patients were more likely to have a calculated risk of 15% or more or had had a prior cardiovascular event. The risk distribution for Indian was not significantly different to that of European and Other but those of Other Asian ethnicity were less likely to be in high risk groups than European and Other (OR 0.71; CI 0.58–0.87).

Table 2 shows the proportion of patients assessed who met one, both, or either adjustment criteria. Over one-quarter (28%) were recorded as having a family history of a premature ischaemic CVD event, with the highest reported proportions in those between 45 and 64 years. There was a marked decline in the proportion of reported family history in those aged over 65 years.

## Table 1. Estimated (unadjusted) cardiovascular disease (CVD) risk of the assessed population by age, gender, and ethnicity

| Variables | n | Unadjusted Framingham CVD risk (%) | | | Prior history of CVD (%) |
|---|---|---|---|---|---|
| | | 0–10% | >10–15% | ≥15% | (≥20%)* |
| Total Assessed | 23693 | 70.0 | 10.8 | 8.0 | 11.2 |
| **Gender** | | | | | |
| Women | 10288 | 76.9 | 8.5 | 4.3 | 10.3 |
| Men | 13405 | 64.6 | 12.7 | 10.7 | 12.0 |
| **Age groups (years)** | | | | | |
| 15–34 | 804 | 97.9 | 0.2 | 0.0 | 1.9 |
| 35–44 | 3581 | 95.8 | 1.0 | 0.2 | 3.0 |
| 45–54 | 6926 | 86.4 | 5.6 | 2.0 | 5.9 |
| 55–64 | 7043 | 65.6 | 15.1 | 8.6 | 10.6 |
| 65–74 | 3864 | 36.1 | 21.3 | 20.4 | 22.2 |
| 75–84 | 1326 | 24.7 | 17.5 | 23.6 | 34.2 |
| 85 and over | 149 | 20.8 | 18.1 | 17.4 | 43.6 |
| **Ethnicity** | | | | | |
| European & Other** | 17912 | 70.7 | 10.9 | 7.7 | 10.7 |
| Māori | 1663 | 65.2 | 10.4 | 9.9 | 14.4 |
| Pacific† | 2506 | 63.4 | 12.8 | 9.4 | 14.4 |
| Indian‡ | 726 | 74.5 | 7.7 | 5.6 | 12.1 |
| Other Asian‡‡ | 886 | 78.6 | 7.9 | 6.5 | 7.0 |

*Those with a history of prior CVD are estimated clinically as being greater than or equal to 20% CVD risk; **Not European, Māori, Pacific, or Asian (e.g. Middle Eastern, African); †All Pacific Islands (mostly of Samoan, Tongan, Niuean, or Cook Islands origin); ‡Indian, Fijian Indian, Pakistani, Sri Lankan, Bangladeshi, Nepali, Afghani, Tibetan; ‡‡Asian but not Indian subcontinent (e.g. Chinese, Japanese, Korean).

## Table 2. Percentages of the assessed population meeting CVD risk adjustment criteria by age, gender, and ethnicity

| Variables | n | Risk adjustment criteria | | | |
|---|---|---|---|---|---|
| | | Premature family history CVD* | High-risk ethnic group** | Premature family history CVD AND high-risk ethnic group | Premature family history CVD OR high-risk ethnic group |
| **Total assessed** | 23693 | 27.5 | 20.7 | 5.3 | 42.8 |
| **Gender** | | | | | |
| Women | 10288 | 29.7 | 21.9 | 5.5 | 46.1 |
| Men | 13405 | 25.8 | 19.7 | 5.1 | 40.3 |
| **Age groups (years)** | | | | | |
| 15–34 | 804 | 3.9 | 25.9 | 9.2 | 48.3 |
| 35–44 | 3581 | 17.1 | 28.0 | 8.6 | 50.4 |
| 45–54 | 6926 | 32.8 | 23.6 | 6.6 | 47.9 |
| 55–64 | 7043 | 29.2 | 17.5 | 4.0 | 40.6 |
| 65–74 | 3864 | 13.0 | 17.7 | 2.9 | 36.6 |
| 75–84 | 1326 | 3.7 | 10.0 | 1.7 | 26.2 |
| 85 and over | 149 | 0.3 | 2.0 | 0.0 | 16.8 |
| **Ethnicity** | | | | | |
| European & Other | 17912 | 28.2 | 0.0 | 0.0 | 28.2 |
| Māori | 1663 | 33.7 | 100.0 | 33.7 | 100.0 |
| Pacific | 2506 | 17.7 | 100.0 | 17.7 | 100.0 |
| Indian | 726 | 34.6 | 100.0 | 34.6 | 100.0 |
| Other Asian | 886 | 23.5 | 0.0 | 0.0 | 23.5 |

*Includes all those with a family history of premature ischaemic cardiovascular disease; **Includes all those of Māori, Pacific, or Indian subcontinent ethnicity.

Table 3 shows the impact on classification and treatment of the recommended 5% risk adjustment for family history and ethnicity (only applied to patients with unadjusted

CVD risk <15% and those without previous CVD events as by definition those ≥15% risk or with a previous history already met drug treatment criteria). The adjustments increased the number of people classified as ≥15% calculated risk by 48% (2790 adjusted compared with 1885 unadjusted—not shown in Table). When combined with those assumed to be >20% risk based on a prior history of CVD, the overall relative increase in the proportion of patients meeting drug and intensive lifestyle treatment criteria was 20%. The increase in the proportion of patients classified as 10–15% risk and therefore requiring individualised lifestyle interventions after adjustment was 48% (3806 adjusted compared with 2569 unadjusted).

**Table 3. Classification of CVD risk (5-year CVD risk or personal history of CVD), recommended treatment, and impact on treatment eligibility with additional 5% CVD risk adjustment**

| Classification of CVD risk | Recommended treatment | Unadjusted CVD risk (N=23693) | Adjusted CVD risk (N=23693) | Relative change in treatment recommendations |
|---|---|---|---|---|
| <10% | General lifestyle advice | 16579 | 14437 | 13% decrease |
| 10–15% | Individualised lifestyle assessment & counselling in primary care | 2569 | 3806 | 48% increase |
| ≥15% or personal history of CVD | Drug treatment and referral to dietician in addition to individualised lifestyle assessment and counselling in primary care | 4545 | 5450 | 20% increase |

## Discussion

In this cohort of patients, the 5% upward adjustment of 5-year CVD risk (recommended for patients with a family history of premature CVD and/or being of Māori, Pacific, or Indian ethnicity) increased by approximately 20% the numbers of people eligible for drug treatment (aspirin, blood pressure-lowering medication and statins), intensive lifestyle management, and referral to a dietician.

The adjustment also increased (by almost 50%) the numbers recommended for individualised lifestyle assessment and counselling by the primary care team. These increases were mainly due to over a quarter of patients reported as having a premature family history of CVD.

The basis of the risk adjustment of 5% for people of Māori, Pacific, and Indian subcontinent ethnicity recommended by the New Zealand Guidelines Group appears to have been based on multiple indirect sources of evidence. Indeed, national mortality and morbidity data have shown an increased burden of diabetes and cardiovascular disease in these ethnic groups compared to other groups.[8–11] However, at the time of guideline development, there were no published studies that systematically compared CVD risk profiles by ethnic group.

Epidemiological surveys of CVD absolute risk for Māori and Pacific workers (Workforce Diabetes Survey 1988-1990,[12–15] Fletcher-Challenge/University of Auckland survey 1992[16]) compared to European people (Auckland Heart and Health

Study[17–19]) suggested that differences in CVD outcomes would not be adequately explained by the standard risk factors although the healthy worker effect would underestimate true risk profiles. The addition of 5% risk as opposed to 2% or 4% does not appear to be based on robust evidence. However, preliminary analyses from anonymised linkage of the PREDICT cohort to CVD outcomes suggest that the adjustment factor of 5% for ethnicity may be justified.

In terms of family history of premature ischaemic CVD, the single most common criticism by clinicians of the New Zealand risk charts prior to the development of the New Zealand CVD guidelines (2005) was the lack of inclusion of this risk factor and the extent to which this variable increases a patient's risk of a CVD event over and above the standard risk factors (personal communication, Rod Jackson, 2006).

A recent systematic review[20] found that (after adjustment for other known risk factors) a family history of premature CHD was associated with over 70% increased relative risk of a CHD event. Moreover, a family history of ischaemic stroke was associated with a 89% increased personal risk of ischaemic stroke in men.[21]

The cohort of patients described in this study were risk assessed oppportunistically within routine primary care. As these patients represent approximately 10% of the ProCare population aged over 35 years, they cannot be considered a representative sample of a resident population[5]. As this cohort is assembled over time, it is hoped that the data will be progressively more representative. However the pattern of their observed risk levels is very similar to expected New Zealand population estimates in 2005.[22] Not all the adjustment factors in the current New Zealand CVD risk management guidelines were able to be taken into account with this early version of PREDICT and so we have understimated the true impact of adjustment on drug treatment eligibility, lifestyle management, and referral to a dietician.

An updated PREDICT module (CVD-Diabetes), currently being implemented in ProCare practices and other PHOs within the three Auckland regional district health boards (DHB) and in Northland DHB, will be able to address this.

The New Zealand CVD risk guidelines thresholds for intervention were informed by an economic analysis[23] on the cost-effectiveness of screening and lipid-lowering treatment and the resource requirements and sustainability of CVD risk assessment.[1] However the guideline developers did not specifically investigate the potential impact of the recommended 5% upward adjustments in CVD risk on the numbers of New Zealanders who would be eligible for interventions.

The aim of explicit CVD risk assessment linked to graded management according to risk of future CVD events is to achieve a cost-effective and equitable reduction in adverse health outcomes across the New Zealand population.

The recommended upward risk adjustments were instituted to enhance the targeting of interventions to at-risk populations. However the substantial potential impact of these adjustments on resources required for lifestyle assessment and advice and drug therapy is of concern.

We believe comprehensive evaluations should be undertaken to investigate these adjustments as part of a review and update of the current guidelines. Firstly, by linking the PREDICT cohort risk assessments to observed hospital admission and fatal outcomes, we will be able to directly determine the appropriate adjustments. We

 

are currently waiting for the study to generate sufficient person-years of follow-up for robust estimations. Secondly the evaluation will require an economic analysis to determine if the impact of these adjustments on healthcare costs would be offset by the reduction in future hospital admissions and deaths.

Of note, there was a surprisingly high prevalence of reported premature family history of CVD, and the unexpected variation by age group (higher in younger patients) suggesting a difference in recall. The reliability of this variable needs to be investigated as it potentially compromises the validity of this recommended risk adjustment. We are unaware of any published information on the likelihood of over- or under-reporting of a premature family history of CVD in New Zealand, however.

Internationally, most investigators use dichotomous measures of family history of coronary disease that do not consider family size, number of affected relatives, or relatives' age and risk profile.[24] Reported reliability is around 67–83% senstitivity (true positive) and above 90% specificity (true negative).[25,26]

To enable systematic assessment and management of CVD risk for all eligible New Zealanders, the capacity of primary care to deliver these preventive services needs to be augmented, and the current costs of delivering this preventive service must be planned for. This includes the tools and resources to identify the target population, support patients to access this care, audit to ensure all groups are participating, and train/upskill primary care teams to deliver consistent and systematic care.

**Competing interests:** None.

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# Issues in the assessment of cardiovascular risk in selected general practices in Canterbury, New Zealand

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**Abstract**

**Aims** The aim was to audit patient records at selected general practices in Canterbury, New Zealand to assess the potential: (a) to improve identification and management of people with risk factors for cardiovascular disease (CVD); and (b) to develop a geographically distinct community database of CVD risk factor prevalence that could be used to plan public health programmes to improve cardiovascular health.

**Methods** Patient records were audited in three general practices in a Canterbury rural town and information on cardiovascular risk factors recommended for the screening and management of CVD by the New Zealand Guidelines Group was extracted and entered into an electronic database. The data was analysed to assess the extent of recording of information on recommended risk factors.

**Results** Most patient records contained information on smoking, blood pressure and lipid profiles. Low levels of information recording were found for physical activity, body mass index (BMI), and family history. There were statistically significant differences between general practices in the type and coverage of information recorded, even for patients with diagnosed cardiovascular disease. Because of deficiencies in information, it was *not* possible to calculate CVD risk using the guidelines for 43% of patients. Some practices remain reliant on paper records which make it extremely difficult to undertake a systematic programme of screening and management of CVD risk factors.

**Conclusions** Before it is practical to undertake a systematic screening programme for CVD risk factors in primary care, it is necessary to reduce reliance on paper records and to fully implement computerised patient management systems that allow for information storage and retrieval. In addition, it is essential to improve the systematic collection of key information in primary care that is used to assess risk of CVD.

Screening for cardiovascular disease (CVD) in primary care has been promoted since the early 1990s in the UK,[1–3] and this was formally endorsed in 2003 in New Zealand.[4] It received a boost when the Framingham equation was validated for use in New Zealand[5] and when the New Zealand screening guidelines were published.[4]

Because CVD is the leading cause of death for both Māori and non-Māori in New Zealand, a screening tool in primary care offers an apparently attractive opportunity for identifying and treating people who have higher risk.

Research published by New Zealand Guidelines Group (2003) indicates the existence of significant undiagnosed disease in the community (for example diabetes) where it is thought only about 50% of cases have actually been previously identified.[6] Also, it was found that only 20% of the reduction in coronary heart disease in the United

Kingdom due to prevention was due to early detection and management of CVD risk factors.[7]

Unfortunately, there have been several studies showing less than optimal treatment for people who have known CVD.[8,9] Other audits of general practice have indicated low levels of recording of some CVD risk factors[10] even following introduction of electronic screening tools such as PREDICT and CCM.[11,12]

For people who have a 5-year absolute CVD risk >15%, both medical treatments and lifestyle changes are recommended.[4] Because the majority of such individuals live in the community, with only a minority referred to secondary hospital services, the identification and management of CVD in primary health care has great potential for increasing reach and reducing CVD-related disease. However, there are recognised problems with compliance and uptake of recommended medications as well as lifestyle interventions such as smoking cessation, diet, and physical activity.

Therefore there is scope for improving the focus of primary healthcare services as well as wider health promotion programmes to educate the public and encourage uptake of both prescribed medicines and lifestyle changes.

In this study we aimed to audit selected general practices in Canterbury to assess the potential to improve identification and management of people with CVD risk factors and also to investigate the potential to develop a community database for a defined geographic region of CVD risk factors that could be used to design public health programmes to improve heart health.

## Methods

Patient records were audited in three practices in a rural town in Canterbury, which is undisclosed due to privacy issues. The GPs in these practices volunteered to participate after being approached by one of the investigators (AH). The audit was carried out in a 10-week period between November 2005 and February 2006 by three students funded through studentships offered by the Christchurch School of Medicine and Health Sciences, University of Otago, Christchuch.

The audit aimed to collate information on CVD risk factors in the general practice medical records of all patients aged 25 to 74 years of age, using protocols in the New Zealand guidelines for assessment and management of cardiovascular risk.[4] It was proposed to include patients aged 25 to 34 years, because some unpublished research in Canterbury indicated significant numbers of Māori in this age group with positive results for fasting plasma glucose (A Humphrey, Personal Communication, 2005). This corroborates an earlier study from Auckland which showed a diabetes prevalence of 5.7% in Māori in this age group.[13]

Where possible, information on CVD risk factors was obtained through the electronic patient information systems. Two of the participating practices (Practices A and C) used MedTech 32 (an electronic patient management system) and its query builder was used to extract the required information. In Practice A, patient information was all electronic and it was relatively easy and efficient to extract the required information using the query builder. Paper-based files from this practice were not explored.

Where information was not recorded electronically, paper files were searched to find information to supplement the electronic data. In Practice C, it was found that some doctors recorded information electronically, while others maintained mainly paper records with limited computerised data. Similarly, in Practice B, most of the medical records were contained in paper files, with very limited computerised information. Therefore, in Practices B and C, a large amount of the information was manually searched through paper files. This problem limited the number of patient records that could be audited within the 10-week timeframe, as described under “Results”.

Data was entered into a Microsoft Excel spreadsheet and analysed using SPSS (version 13) software. Differences between practices recording of patient information were explored using Chi-squared tests. Ethical approval was obtained from the Southern Region Ethics Committee.

## Results

These three practices used very different modes for storing and retrieving patient information and this made it extremely difficult to systematically extract important data on risk factors. Only one of the practices (Practice A) had a full electronic patient management system (Table 1). This fully computerised practice was the only one where information on CVD risk factors could be readily extracted and analysed; where every patient in the target age range was able to be identified (N=503); and absolute risk of CVD calculated. The data query functions in MedTech 32 were able to efficiently extract the data and identify CVD risk factor profiles.

In the other two practices, which relied on paper medical records, it was extremely difficult and time-consuming to manually search the paper files and identify individuals who were at elevated risk of a CVD event. Within the 10-week time period of the audit, only records of people aged 60 to 75 years were able to be audited in Practice B (N= 339), while in Practice C, only records for men aged 60 to 75 years were able to be audited (N=391).

**Table 1. Summary of patient records at three Canterbury general practices audited by age, gender, and type of practice**

| Variables | Practice A | Practice B | Practice C |
|---|---|---|---|
| Usual number of general practitioners | 1 | 1 | 6 |
| Number patient records audited | 503 | 339 | 391 |
| Age range in years | 25–75 | 60–75 | 60–75 |
| Gender of patients audited | Male and female | Male and female | Male only |
| Extent of electronic data recording | Fully electronic | Mainly paper records | Partially electronic |

The majority of patient records had information recorded on smoking, blood pressure (BP), and on the lipid profile (Table 2). Recording of information was low or extremely variable for body mass index (BMI), height, waist circumference, physical activity, and family history (Table 2).

Due to common non-compliance with fasting requirements, Practice A frequently referred patients for non-fasting blood tests. Statistically significant differences between practices were found in levels of recording of all risk-factor information except BP, weight, and height (Table 2).

We investigated if some of these differences between practices may have been explained by the differences in age ranges audited. However, these significant differences persisted for all variables, even when patients aged less than 45 years were excluded from Practice A. Thus, there was significant variation between GPs in both the type of information that is recorded as well as in the mode of data storage.

**Table 2. Percent of all audited patients at three Canterbury general practices with information recorded on cardiovascular disease (CVD) risk factors**

| Variables | Practice A (N=503) Patients aged 25–75 years % | Practice A (N=197) Patients aged 60–75 years % | Practice B (N=339) Patients aged 60–75 years % | Practice C (N=391) Patients aged 60–75 years % |
|---|---|---|---|---|
| Family history of CVD* | 26 | 19 | 81 | 52 |
| Family history of Type 2 diabetes* | 26 | 19 | 75 | 48 |
| Family history of genetic lipid disorder* | 26 | 19 | 12 | 42 |
| Smoking* | 74 | 64 | 89 | 74 |
| Physical activity** | 44 | 34 | 22 | 24 |
| Blood pressure [ns] | 89 | 91 | 88 | 88 |
| Pulse* | 0 | 0 | 37 | 41 |
| Weight [ns] | 61 | 60 | 54 | 58 |
| Height [ns] | 41 | 42 | 28 | 32 |
| BMI* | 14 | 9 | 10 | 30 |
| Waist circumference* | 6 | 8 | 8 | 15 |
| Fasting lipid profile* | 51 | 56 | 66 | 71 |
| Fasting plasma glucose* | 46 | 49 | 69 | 65 |

*Significant at $p<0.001$, using Pearson Chi-squared; **Significant at $p<0.01$, using Pearson Chi-squared;
[ns]Not statistically significant.

For patients with a *known history of CVD*, there was also significant variation in the type of information recorded (N=240) (Table 3). As anticipated, most patients in all practices had data recorded for smoking, BP and lipids. However, physical activity was noted for only approximately 50% of patients with a known history of CVD. Recording of data was significantly variable for family history, BMI, fasting plasma glucose, urine albumin:creatine ratio (ACR) and serum creatinine. A minority of patients in all practices had information recorded on HbA1c, waist circumference, or family history of genetic lipid disorder.

For patients who had previously been diagnosed with *diabetes, impaired glucose tolerance, or impaired fasting glucose* (N=146), there was a high level of information recorded for HbA1c, serum creatinine, lipids and fasting glucose profile (Table 4). Statistically significant differences between practices were observed in recording of data on family history, BMI, fasting lipid and plasma glucose, and ACR. As stated earlier, the different percentages for fasting lipids and plasma glucose were because Practice A referred patients for non-fasting tests to try to reduce problems of non-compliance with fasting tests.

Because of deficiencies in information, it was *not* possible to calculate CVD risk using the guidelines in 46% of patients for Practice A, 37% in Practice B and 46% in Practice C.

**Table 3. Percent of patients at three Canterbury general practices with a personal history of CVD and with information recorded on cardiovascular risk factors**

| Variables | Practice A (N=73) Patients aged 25–75 % | Practice A (N=57) Patients aged 60–75 % | Practice B (N=60) Patients aged 60–75 % | Practice C (N=107) Patients aged 60–75 % |
|---|---|---|---|---|
| Family history of CVD* | 16 | 10 | 83 | 52 |
| Family history of type 2 diabetes* | 16 | 11 | 80 | 40 |
| Family history of genetic lipid disorder** | 16 | 10 | 13 | 33 |
| Smoking [ns] | 72 | 79 | 95 | 80 |
| Physical activity [ns] | 57 | 58 | 45 | 48 |
| Blood Pressure [ns] | 100 | 100 | 98 | 99 |
| Pulse* | 0 | 0 | 62 | 52 |
| Weight [ns] | 81 | 82 | 79 | 75 |
| Height [ns] | 56 | 58 | 65 | 57 |
| BMI* | 3 | 3 | 12 | 55 |
| Waist circumference [ns] | 37 | 40 | 40 | 46 |
| Fasting lipid profile [ns] | 70 | 68 | 85 | 87 |
| Fasting plasma glucose** | 63 | 63 | 85 | 79 |
| Urine albumin: creatinine ratio (ACR)* | 34 | 39 | 22 | 10 |
| Serum creatinine* | 100 | 100 | 78 | 70 |

*Significant at p<0.001, using Pearson Chi-squared;**Significant at p<0.01, using Pearson Chi-squared; [ns]Not statistically significant.

**Table 4. Percent of patients at three Canterbury general practices with diabetes, impaired glucose tolerance, and/or impaired fasting glucose with information recorded on cardiovascular risk factors**

| | Practice A (N=57) Patients aged 25–75 % | Practice A (N=38) Patients aged 60–75 % | Practice B (N=33) Patients aged 60–75 % | Practice C (N=56) Patients aged 60–75 % |
|---|---|---|---|---|
| Family history of CVD* | 10 | 5 | 88 | 52 |
| Family history of type 2 diabetes* | 10 | 5 | 82 | 41 |
| Family history of genetic lipid disorder* | 10 | 5 | 6 | 37 |
| Personal history of CVD [ns] | 95 | 97 | 97 | 86 |
| Smoking [ns] | 84 | 72 | 100 | 91 |
| Physical activity [ns] | 58 | 55 | 58 | 39 |
| Blood pressure [ns] | 98 | 100 | 100 | 100 |
| Pulse* | 0 | 0 | 42 | 48 |
| Weight [ns] | 88 | 87 | 91 | 95 |
| Height [ns] | 67 | 63 | 85 | 79 |
| BMI* | 14 | 13 | 24 | 77 |
| Waist circumference [ns] | 18 | 26 | 21 | 30 |
| Fasting lipid profile* | 54 | 53 | 97 | 91 |
| Fasting glucose** | 67 | 55 | 97 | 80 |
| Urine albumin: creatinine ratio (ACR)** | 88 | 95 | 73 | 61 |
| Serum creatinine | 93 | 92 | 79 | 82 |
| HbA1c [ns] | 95 | 95 | 88 | 84 |
| Date of first diagnosis of diabetes | 98 | 100 | 97 | 73 |

*Significant at p<0.001, using Pearson Chi-Squared; **Significant at p<0.01, using Pearson Chi-Squared;

[ns] not statistically significant.

## Discussion

Variation in modes of information storage and retrieval is critical because we found that it was not practical to undertake systematic screening for CVD risk factors in general practices that rely on paper records. Indeed, it was not feasible to efficiently retrieve the key information, identify higher risk patients, and provide ongoing patient management in practices that do not have fully electronic patient records.

In such practices, staff are faced with manually hunting through piles of paper. In addition to adding an untenable workload for general practices, such an approach would be unlikely to provide screening information required for timely intervention. Electronic records, however, provide opportunities for instantaneous calculation of absolute CVD risk profiles.[11,12]

Our findings were consistent with those of other studies that found high rates of recording of information on BP and lipids, but low rates of recording of information on lifestyle factors, particularly BMI and physical activity.[10]

The lack of some of the necessary information has meant that CVD risk could *not* be calculated for 43% of patients whose records were audited in this study. Rafter et al (2005) also found that in a general practice database covering 25,384 individuals, there was sufficient information to calculate CVD risk using the New Zealand guidelines in only one-third of cases. These findings indicate that there is currently a need to improve consistency in the type and coverage of information that is used to assess risk and in the computerised record systems used for storage and retrieval of information. Improvement in these basic building blocks is necessary before it is feasible to roll out an organised programme to calculate and manage cardiovascular risk.

Our results showed there was statistically significant variation between practices in the type of CVD risk factor information recorded, even for patients with a known history of CVD. This significant variation in assessment practice may help to explain findings from other studies that many people with higher risk of CVD are not receiving currently recommended treatments.[9] This suggests that at least part of the problem may be due to under-identification of moderate to higher risk patients.

When trying to identify individuals at higher risk, it was found that strict adherence to the guidelines [4] underestimated numbers of patients with at least moderate risk, because 27% of patients who were audited were already on medication to lower cholesterol (N=331), and 42% were on medication to lower BP (N=523).

In total, 23% of audited patients were on medication for lowering *both* BP and cholesterol. It was necessary to identify such patients separately, in order to account for people who were on medication to control risk factors prior to screening. The guidelines should be applied to those not on medication and clarification sought as to how the guidelines should be applied for patients using medication.

Some other issues that emerged during the study include:

- It may not be feasible to obtain fasting lipid and plasma glucose tests for some patients. This was also noted by the New Zealand Society for the Study of Diabetes[14] who commented that a random glucose test fulfils the need in spite of a less than ideal predictive value.
- The New Zealand guidelines may need to be adapted for patients who have previously been identified as moderate to higher risk and who are taking medications for lowering BP and/or cholesterol prior to the time of re-assessment. These patients could have controlled BP and/or cholesterol, but still be at moderate risk. The guidelines don't appear to provide guidance on how to assess risk for such people.
- The results indicate there may be a need for greater investment in educating primary care staff about the guidelines and how they should be applied. Some general practitioners stated that there has been only limited education and effort to implement screening for CVD risk factors, compared with other policies. For example, the Accident Compensation Commission guidelines on the treatment of shoulder injuries, were followed up with education sessions for general practitioners.
- There is a lack of New Zealand research on the costs of collecting and storing information on CVD risk factors compared with the health outcomes that may be achieved from identifying undiagnosed disease and providing improved care. Some GPs feel that the additional costs need to be recovered. A 2001 editorial in the *British Medical Journal* queried whether the extra costs of CVD screening are justified in terms of improved health outcomes.[15] In the UK, additional contracts were offered to GPs to cover the costs of CVD assessment. However, there was considerable debate about the adequacy of payments and the health gains in relation to the costs.[16]
- Some GPs consider that their patient record systems are designed for patient care, rather than as databases for research or planning of public health initiatives. This issue was also discussed in the *One Heart Many Lives* seminars, where it was noted that there is a wealth of information in primary care databases, but a lack of systems to analyse it in ways that help to inform decisions about improving services.
- There is evidence that improving the electronic functions of data systems has a flow-on effect of improving coverage and collection of information on risk factors.[12] At the time of this study, there was a financial barrier to using the PREDICT tool. It is possible that if PREDICT or a similar tool could be made available at an acceptable cost, it would improve the feasibility and coverage of a CVD screening programme.
- Consistency between measuring tools for CVD risk factors is variable and this may be one reason why some GPs record certain information less frequently than others. For example, blood pressure is well understood and easy to record. In contrast, physical activity not only lacks a universally understood short and easy measure, but also lacks a field in most patient management software. There is some evidence that a short, reliable measure of physical activity can be used in general practice, but such a measure has not been included in any patient management software in New Zealand.[17]

- Similarly, there were substantial and perhaps surprising differences between practices in the recording of family history, which may reflect confusion about its importance and how to define it.

Public health authorities currently operate independently from general practice. There appears to be potential to improve heart health outcomes by planning public health promotions in co-ordination with primary health care, in a way that is informed by up to date information on community prevalence of CVD risk factors. Examples may include targeting and promoting improved prescription and compliance with medicines and lifestyle changes such as physical activity and diet.

In recent times, primary prevention (targeting those *without* diagnosed CVD) has resulted in the vast majority of reduced coronary heart disease from prevention.[7] This study suggests there may be significant potential for systematic screening, identification and secondary prevention to contribute to reducing the burden of disease from CVD.

This research indicates that several factors should be reviewed including: current assessment protocols of CVD risk in general practice; patient record systems; some of the indicators in the guidelines; the costs and benefits of screening for CVD risk factors.

Although considerable deficits in assessment information were identified, this audit was a constructive co-operative effort by a group of primary care practitioners to review current systems and the way forward.

At this stage, the results indicate that priority needs to be given to:

- Improving assessment protocols to ensure consistency in the type and coverage of information that is collected on CVD risk factors: and
- Improving electronic information systems for storage and retrieval.

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# Accuracy of the Wells Rule in diagnosing deep vein thrombosis in primary health care

Maelen Tagelagi, C Raina Elley

**Abstract**

**Aim** A clinical decision rule to aid in the diagnosis of deep vein thrombosis (DVT) was derived by Wells and has been validated in several secondary care settings. It is now used to reduce unnecessary radiological examinations. The aim of this study was to assess the accuracy of the Wells Rule in New Zealand primary health care.

**Method** Validation study of the Wells clinical decision rule for DVT in primary health care, using ultrasound as the comparator. Participants included patients with symptoms of DVT from 410 general practitioners in Auckland between 1 October 2004 and 30 September 2005. Sensitivity, specificity, and likelihood ratios were calculated.

**Results** 432 patients were assessed over 12 months, of whom 327 were eligible to participate. Mean age was 65.5 years (SD 16.1) and 203 (62%) were women. DVT was confirmed in 12% (39/327). Sensitivity was 82% (95% confidence interval [CI]: 67.3–91.0) and specificity 22.5% (CI: 18.1–27.7). Likelihood ratio for a positive test was 1.06 (CI: 0.90–1.24) and for a negative test 0.80 (CI: 0.39–1.61).

**Conclusions** The Wells Rule used alone had only moderate sensitivity and poor specificity and likelihood ratios, so has limited use in the diagnosis of DVT in primary care.

Deep vein thrombosis (DVT) can occur in any location within the venous circulation. The large majority of venous thrombi occur in the deep veins of the leg but are also relatively common within the veins of the pelvis.[1] DVTs in the proximal leg veins frequently give rise to symptoms including pain, swelling, oedema, venous distension, or discolouration of the skin.[2] However, none of these symptoms are exclusive to DVT.

Hence, when patients present with symptoms that might be interpreted as being due to DVT there is a range of potential alternative diagnoses, such as infection, trauma, cardiac failure, congenital malformations, thyroid disease, arthritis, malignancy, or the effects of various medications. This creates something of a diagnostic dilemma for the physician who has to decide which patients need investigation to exclude DVT and which patients can be managed without unnecessary testing.

Venography has been a reference standard test for DVT since 1963.[3] However, it has now been superseded by venous duplex ultrasonography—as ultrasound has similar levels of accuracy, is less invasive, requires less operator skill to perform, and is associated with fewer side effects.[4] The presence of D-dimer fragments within the circulation has also been considered as a hallmark of thrombosis. Tests for D-dimer are highly sensitive but lack specificity because D-dimer production is seen in association with a variety of other conditions. Malignancy, advancing age, trauma,

infection, haemorrhage, surgery, pregnancy, and smoking are all associated with the production of D-dimer. Circulating levels of D-dimer may also be elevated in certain ethnic groups.[5] The value of tests for D-dimer is in their high negative predictive value making them a useful exclusion test for DVT.

To reduce the frequency of unnecessary tests Wells and co-workers developed a clinical diagnostic tool widely referred to as the Wells Rule.[6] The rule has good sensitivity and specificity for DVT in secondary care settings and is now used in many secondary care settings to reduce the cost of unnecessary radiological investigations.[6] By combining various components of the history, examination, and investigations, the Wells Rule simplifies diagnostic assessment, improves clinical accuracy and reduces the overall cost of investigation needed for DVT.[7] The validity of the Wells Rule in primary health care was untested until recent studies in the Netherlands suggested that it may not be as valid in that setting.[8]

The Waitemata District Health Board (DHB) was keen to reduce the secondary care costs of unnecessary hospital investigation of suspected DVTs. A DHB initiative called Primary Options, proposed using the Wells Rule in primary health care to identify those at very low risk who did not require referral to secondary care for radiological investigation. However, an initial evaluation was necessary to test the accuracy of the rule in this context. Therefore, the aim of this study was to test the validity of the Wells Rule in a New Zealand primary health care setting.

## Methods

**Design—**This study was a prospective diagnostic validation study of the Wells Rule for DVT in primary health care, using ultrasound as the gold standard comparison.

**Participants—**All 410 general practitioners (GPs) in the Waitemata DHB region of Auckland, New Zealand, were invited to participate in a study to streamline the diagnostic management of DVT through the Primary Options programme. Patients were included if, in the opinion of the GP, they presented with symptoms that could be consistent with DVT.

Patients who were pregnant, receiving hormonal therapy, had experienced DVT previously, were anticoagulated for any reason, or who had undertaken long-haul air travel within 6 weeks of presentation were excluded from the study and were referred immediately for further investigation. Patients were enrolled from 1 October 2004 to 30 September 2005 following written informed consent. The Auckland Regional Ethics Committee considered the study to be audit and deemed formal ethics approval unnecessary.

**Diagnostic test—**The Wells Rule (Table 1) was used by the examining GP to assess likelihood of DVT. Patients with a high likelihood (Wells score >1) were referred straight for ultrasound. All other patients underwent serum D-dimer testing but also underwent ultrasound examination. Ultrasound was carried out in a number of community-based radiology facilities in the Auckland region.

Prior to the study, assessment of patients using the Wells Rule was incorporated into a DVT diagnostic algorithm. This was presented to GPs at a series of continuing medical education evenings. GPs who attended the meetings were provided with documented guidelines outlining the Wells Rule and given clear instruction regarding its use when investigating patients with suspected DVT. In addition, two project coordinators visited all GP medical practices within the Waitemata DHB region to discuss the use of the algorithm.

**Statistical analysis—**Sensitivity, specificity, negative and positive likelihood ratios, and negative and positive predictive values with 95% confidence intervals were calculated comparing the results of the Wells Rule with ultrasound results. The statistical calculator on the Centre for Evidence-Based Medicine (CEBM) website was used for all calculations.[9] Two sensitivity analyses were conducted for the data missing on ultrasound results.

**Table 1. The Wells clinical decision rule criteria for deep vein thrombosis (DVT)**
(**Source:** Wells P, Anderson R, Bormanis J, et al. Value of assessment of pretest probability of deep-vein thrombosis in clinical management. Lancet. 1997;350:1795–801.)

| Clinical feature (Wells) | Score |
|---|---|
| Active cancer (treatment ongoing or within previous 6 months, or palliative) | 1 |
| Paralysis, paresis or recent plaster immobilisation of lower extremities | 1 |
| Recently bedridden >3 days or major surgery within 4 weeks | 1 |
| Localised tenderness along the distribution of the deep venous system | 1 |
| Entire leg swollen | 1 |
| Calf swelling 3 cm greater than the asymptomatic side (measured 10 cm below tibial tuberosity) | 1 |
| Pitting oedema confined to the symptomatic leg | 1 |
| Collateral superficial veins (not varicose) | 1 |
| Alternative diagnosis as likely as or greater than that of DVT—i.e. cellulitis, phlebitis, Baker's cyst, oedema | -2 |

## Results

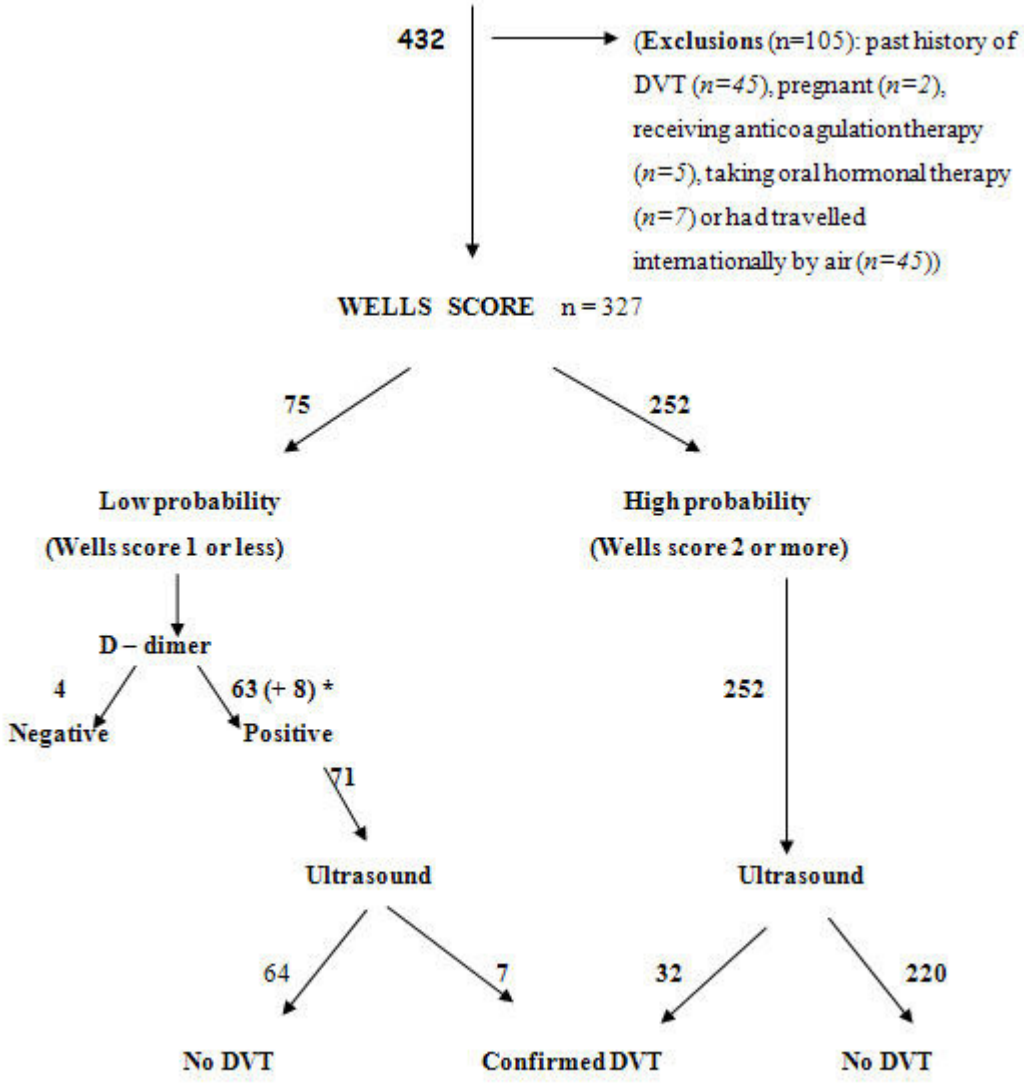
Over the 12 months of the study, 432 patients were assessed including 269 (62.2%) women and 163 (37.8%) men. The age range was 11 to 99 years with a mean age of 64 years.

The flow diagram of participants through the study is depicted in Figure 1. Of the 432 patients assessed, 105 patients were not included on the basis of exclusion criteria. Of the remaining 327 patients (mean age 65.5 years (SD 16.1); 62% (203/327) women), 11.9% (39/327) had DVT confirmed. In contrast, 19% (20/105) of patients excluded from the main analysis subsequently had DVT confirmed. Four patients were excluded from the main analysis on the basis that their final diagnosis, DVT very unlikely (Wells score ≤ 1 and D-dimer test negative), was not confirmed with ultrasound. This left 323 patients in the main analysis.

Sensitivity, specificity, and positive and negative likelihood ratios of the Wells rule, and prevalence of DVT in the study population, are depicted in Table 2. Sensitivity was 82% (95% confidence interval [CI]: 67.3–91.0) and specificity 22.5% (CI: 18.1–27.7). Likelihood ratio for a positive test was 1.06 (CI: 0.90–1.24) and for a negative test 0.80 (CI: 0.39–1.61). The prevalence of DVT in the study population was 12% (CI: 9–16).

Two sensitivity analyses were carried out and are also presented in Table 2, firstly assuming the four excluded patients who did not undergo ultrasound had a DVT and secondly assuming they did not have DVT. If all four patients had DVT, sensitivity falls from 82% to 74% and the likelihood ratio for a positive test falls from 1.06 to 0.96. If all four did not have DVT, then there are very few differences to the results found in the main analysis; sensitivity remains at 82% and specificity may improve from 22.5% to 24%. The likelihood ratio for a positive test is virtually the same (improves marginally from 1.06 to 1.07 but with large overlapping 95% confidence intervals).

**Figure 1. Flow diagram of assessment of patients with suspected DVT in general practice using the Wells Rule, D-dimer, and ultrasound**




*8 patients in the DVT unlikely group underwent ultrasound without prior D-dimer.

To determine the post test likelihood of DVT using the Wells Rule in primary care, a Fagan nomogram was used for both positive and negative likelihood ratios (Figures 2 and 3). Knowing that the prevalence or pre-test likelihood of DVT in symptomatic patients was 12% (as it was in this population), if the Wells assessment was positive, then the post-test likelihood of having DVT for that individual was only 13% (Figure 2). If the Wells assessment was negative, then the post-test likelihood was still 10%.

**Table 2. Validation of Wells Rule for diagnosing DVT in primary health care with sensitivity analyses[*]**

| Variables | | Main analysis (95% confidence intervals) | | Sensitivity analysis 1 [4 patients included & assumed ultrasound positive] (95% confidence intervals) | | Sensitivity analysis 2 [4 patients included & assumed ultrasound negative] (95% confidence intervals) | |
|---|---|---|---|---|---|---|---|
| | | ultrasound | | ultrasound | | ultrasound | |
| | | DVT | No DVT | DVT | No DVT | DVT | No DVT |
| Wells Rule | DVT likely ≥2 | 32 | 220 | 32 | 220 | 32 | 220 |
| | DVT unlikely ≤1 | 7 | 64 | 11 | 64 | 7 | 68 |
| Sensitivity (%) | | 82 (67–91) | | 74 (60–85) | | 82 (67–91) | |
| Specificity (%) | | 22.5 (18–28) | | 22.5 (18–28) | | 24 (19–29) | |
| Positive predictive value (%) | | 0.13 (0.09–0.17) | | 0.13 (0.09–0.17) | | 0.13 (0.09–0.17) | |
| Negative predictive value (%) | | 0.90 (0.81–0.95) | | 0.85 (0.76–0.92) | | 0.90 (0.82–0.95) | |
| Positive likelihood ratio | | 1.06 (0.90–1.24) | | 0.96 (0.80-0.96) | | 1.07 (0.92–1.26) | |
| Negative likelihood ratio | | 0.80 (0.39–1.61) | | 1.14 (0.65–1.98) | | 0.76 (0.38–1.54) | |
| Prevalence (%) | | 12.0 (9–16) | | 13.2 (9–17) | | 11.9 (8–15) | |

[*]Including four patients who did not undergo ultrasound, firstly assuming they had DVT, then assuming they did not.

**Figure 2. Fagan nomogram to calculate post-test likelihood of a DVT following a positive Wells criteria test in primary care patients with symptoms suggestive of DVT. Likelihood ratio 1.06 (Pretest probability 12%)**

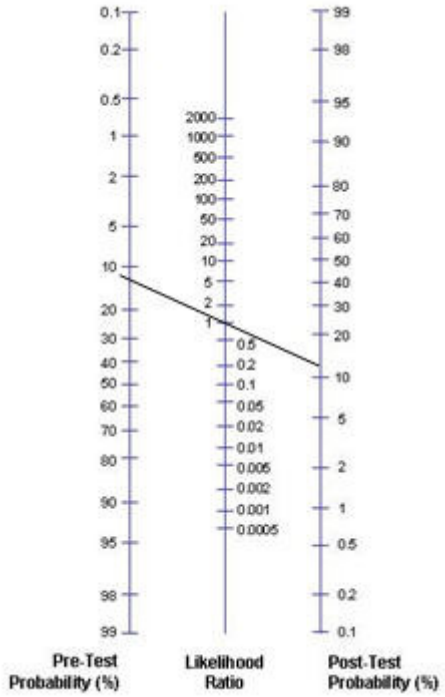
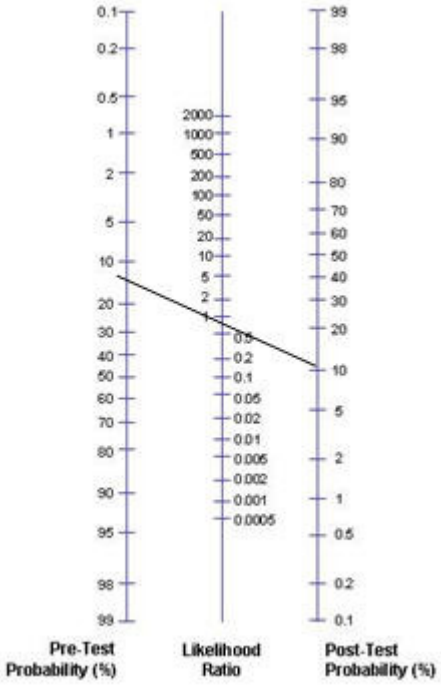


**Figure 3. Fagan nomogram to calculate post-test likelihood of DVT following a negative test using the Wells criteria in primary care patients with symptoms suggestive of DVT. Likelihood ratio 0.8 (Pretest probability 12%)**




## Discussion

This study found that the Wells Rule as it was used in the Primary Options programme in primary health care had moderate to low sensitivity (82%) and poor specificity (23%). Designed to function as a "rule out" test, the Wells Rule in this study had a likelihood ratio for a negative test of 0.8 (CI: 0.4–1.6), which is poor.

While the pretest probability of DVT was 12%, the post-test probability of DVT after a negative test was still high (10%) thus suggesting the rule (used by GPs in the Primary Options programme) did not effectively exclude DVT. The likelihood ratio of a positive test was also poor (1.06). Likelihood ratios measure the strength of a test to predict the correct outcome.[10] For a diagnostic test to be clinically useful, the likelihood ratio for a negative test should be less than 0.1 and a likelihood ratio for a positive test should be greater than 5.[11]

Performance of the rule remained poor even when the four low-risk patients who did not undergo ultrasound (i.e. they had a Wells score ≤1 and negative D-dimer test) were included in sensitivity analyses and assumed not to have DVT. Although it would be very unlikely, if all 4 patients were assumed to have had DVT, sensitivity fell further to 74% (CI: 60–85) and the negative likelihood ratio increased to 1.14 (CI: 0.7–2.0), which would imply that DVT was more likely after a negative Wells test result.

This study was conducted in a real life community primary care setting involving a large number of general practitioners. This improves the generalisability of results

when compared with previous studies where less diverse settings were used.[6,8] However, this study did have some methodological problems.

Patients with suspected DVT were enrolled after presenting to their GP. This may not have represented all patients in the community as some may have self referred directly to hospital. In addition, while GPs were encouraged to enrol patients into the Primary Options DVT study, they were not compelled to do so. If for some reason, a GP felt it was preferable, they could refer a patient directly to hospital or to a specialist haematologist working in the community. Details of those who were not enrolled and were managed elsewhere were not recorded. As a result, the prevalence of DVT in the validation study may not be truly representative of all symptomatic community-based patients.

A significant effort was made to canvas all GPs in the Waitemata DHB region to inform them of the change in clinical practice surrounding the diagnosis of those with suspected DVT. However, there was no record in place to assess whether GPs fully understood the Wells Rule and because of the large number of GPs it was likely that different interpretations of the Rule and how to apply it was a source of bias. Combined with the knowledge that most GPs would only see only one or two patients per year with suspected DVT, it is probable that their use of the rule was sub-optimal and variable.

Adding serum D-dimer to the Wells rule may have improved diagnostic accuracy. However, this was not tested in this study because only those with a low Wells score received a D-dimer test. In addition, the few patients that had a negative D-dimer did not have the gold standard ultrasound, so sensitivity and specificity could not be calculated.

This study confirms the findings of Oudega and co-workers.[8,12] In particular, sensitivity and specificity of the Wells Rule was lower in primary care than in secondary care.[13,14] Negative predictive values and negative likelihood ratios were also weaker than in the original benchmark study by Wells in secondary care.[6]

Recently Oudega and co-workers were able to achieve a higher level of accuracy using a diagnostic tool adapted from the Wells Rule in primary health care in the Netherlands.[12] While this appeared to be successful, it may suffer the same diagnostic problems as the Wells Rule when applied to a completely new primary care population.[15] The implication for New Zealand is that, in its present form, the Wells rule alone is not useful in the diagnosis of DVT in primary health care.

**Competing interests:** None.

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# Survival over 5 years in the initial hospital survivors with acute coronary syndrome: a comparison between a community hospital and a tertiary hospital in New Zealand

Cheuk-Kit Wong, Eng Wei Tang, Peter Herbison

**Abstract**

**Aims** To compare the outcome of hospital survivors with acute coronary syndrome (ACS) discharged from a community hospital (Invercargill hospital) versus from a tertiary teaching (Dunedin) hospital followed for up to 5 years.

**Methods** All ACS survivors discharged from Dunedin and Invercargill coronary care units between the years 2000–2002 were included. We previously found higher 1-year mortality for Invercargill patients but the explanation was unclear.

**Results** Of the 844 patients admitted to Dunedin and 299 admitted to Invercargill hospital, 1057 survived the index ACS episode and formed the cohort for the current study. At 2 years, the mortality of these initial survivors was 8.5% higher for Invercargill patients (18.4% vs 9.9%, p<0.001). Over up to 5 years of follow-up, comparing Invercargill patients to Dunedin patients, the unadjusted hazard ratio for mortality was 1.26 (95%CI: 0.90–1.75). After adjusting to the hospital discharge GRACE score (119±40 for Dunedin patients and 130±40 for Invercargill patients, p=0.001), this dropped to 1.12 (95%CI: 0.80-1.57). After further adjusting to the discharge medications aspirin (97% vs 98%) and ACE-inhibitors (53% vs 49%), this was 1.14 (95%CI 0.81–1.59). After further adjusting to the use of beta-blockers (78% vs 71%), this was 1.07 (95%CI: 0.76–1.50). After final adjustment for the use of statins (65% vs 42%), this was 0.96 (95%CI: 0.68–1.36).

**Conclusion** Patients discharged from Invercargill hospital fare worse over the first 2-years and tended to fare worse over the first 5-years. This was due both to their higher baseline risk at discharge and the under-use of statins. Of note, PHARMAC rules for statins only changed around the end of the study period allowing more liberal use of statins.

Our previous study[1] revealed a worse outcome for acute coronary syndrome (ACS) patients managed in a community hospital staffed by general physicians (Invercargill Hospital) compared to those managed in a tertiary hospital with cardiologists and catheterisation laboratory (Dunedin Hospital). Dunedin Hospital served as the referral centre for Invercargill patients (a 3-hour ambulance journey or 1-hour helicopter journey).

In-hospital mortality was higher for Invercargill patients (10.7%) than for Dunedin patients (6.4%, p=0.02) but this was not statistically different when adjustments were made for baseline differences (odds ratio for death 1.45, 95% confidence interval [CI]: 0.97–2.17). However, a large and statistically significant difference in survival was seen at 6-months (19.1% vs 9.6%, p<0.0005) and 1-year (22.1% vs 12.1%,

p<0.0005), and this was significant after the same adjustments for baseline characteristics.[1]

The current study focussed on the hospital survivors examining survival over a follow-up period that extended up to 5 years for some patients, and possible explanations for the difference. We used the GRACE hospital discharge risk score[2] to measure the risk for all hospital survivors.

Risk parameters in this GRACE score (Appendix 1) are increasing age, history of congestive heart failure, history of myocardial infarction, higher resting heart rate and lower systolic blood pressure on arrival, ST segment depression, elevated initial serum creatinine, elevated cardiac enzymes, and not having in-hospital percutaneous coronary intervention (PCI). Each parameter is scored and the summed GRACE score corresponds to an estimated probability of all-cause mortality from hospital discharge to 6 months, in this original derivation in the GRACE cohort[2] and up to 4 years in the current cohort of patients.[3] Of note, the last parameter, PCI, is a therapy.

The 2006 update of the AHA/ACC guidelines for secondary prevention for coronary disease includes a class 1 recommendation for aspirin, statins, beta-blockers, and ACE-inhibitors,[4] and the benefit from incremental use of these evidence-based medications (EBM) has been reported.[5,6]

We have previously shown comparable rates in the use of EBM in Dunedin to international registry data,[7] but the use of statins was lower in Invercargill.[1] Our secondary aim was to evaluate whether the difference in the use of EBM might explain the discrepant outcome between the two centres.

## Methods

**Background—**Details of this study have previously been reported.[1] In brief, we retrospectively studied consecutive patients with ACS admitted into the coronary care units (CCU) of two related centres in New Zealand (Dunedin Hospital and Invercargill Hospital) in the years 2000–2002. Dunedin Hospital served as the referral centre for Invercargill Hospital. Patients initially admitted to Invercargill Hospital and transferred later on to Dunedin were categorised as Invercargill patients.

All patients had ACS as their primary discharge diagnosis and were above 18 years of age. The first admission with ACS was used for analysis if readmissions with ACS were present. This study protocol was in accordance with local hospital research guidelines.

**Study design—**Clinical data collected include, among others,[1] baseline characteristics (such as age, sex, cardiac risk factors, history of ischaemic heart disease); presenting clinical features (heart rate, blood pressure, Killip class, episodes of cardiac arrest on arrival and cardiogenic shock); ECG characteristics (degree of ST-deviation & T-waves changes); biochemistry (initial and maximum troponin rise, initial creatinine level); left ventricular function on echocardiography; and treatment.

We recorded both the use of in-hospital medications in the first 24-hour, reperfusion and revascularisation therapy, and the use of different medications at discharge.

Death was defined as all-cause mortality. Information on the deaths (until 1 April 2005) was obtained from medical records and the national death registry. This represented a 5-year follow-up period for some patients.

Patients with ACS were classified into ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina, defined as ischaemic chest pain lasting more than 30 minutes with no evidence of myonecrosis or ST elevation.

**Statistical analysis**—Statistical analysis was carried out in STATA v9 software. Data are presented as mean ± standard deviation, median and interquartile range (IQR), or actual numbers with percentages as appropriate. Chi-squared tests were used to compare proportions. The test was double-sided and considered statistically significant at $\alpha<0.05$. Multivariable analysis was performed using Cox's

proportional hazards regression for hospital survival up to 5-years to examine the association between admission to Invercargill Hospital vs Dunedin Hospital and mortality.

These models were adjusted for the GRACE hospital discharge risk score (see Appendix 1 at the end of this paper[2,3]). For the GRACE score calculation, we used in-hospital congestive cardiac failure instead of history of congestive heart failure and history of ischaemic heart disease instead of history of myocardial infarction because these data were likely to be more accurate in the clinical records. Also, elevated troponin levels were read as a positive score for cardiac enzyme in the GRACE score calculation.[3]

Finally, we adjusted the model for the use of EBM at discharge (aspirin, ACE-inhibitors [angiotensin II antagonists were also recorded in this category], beta-blockers, and statins). The model was performed both for the whole period until 1 April 2005 and for the first 2-year period.

## Results

From 1 January 2000 to 31 December 2002, 843 patients and 299 patients were admitted into Dunedin Hospital and Invercargill Hospital CCUs respectively. The current study focussed on the 1057 hospital survivors. Table 1 detailed their baseline characteristics and the use of EBM at discharge (aspirin, ACE-inhibitors, beta-blockers, and statins).

The GRACE hospital discharge risk score was 119±40 (median 116, IQR 90–147) in the Dunedin patients and 130±40 (median 129, IQR 99–161) in the Invercargill patients ($p<0.001$). If the parameter of in-hospital PCI was not scored, the score was 111±38 and 118±39 respectively ($p=0.03$).

Comparing the two centres, the use of aspirin and ACE-inhibitors was not different, but beta-blockers use was 7% lower and statins use 24% lower for Invercargill patients in absolute terms.

**Long-term outcome—**Proportions of patients reaching the follow up time points of 2 years, 3 years, 4 years, and 5 years were 100%, 82%, 54%, and 11% respectively. Mortality at 2 years was 18.4% for Invercargill patients and 9.9% for Dunedin patients ($p<0.001$).

**Table 1. Baseline demographics and use of medications in the ACS hospital survivors (n=1057)**

| | Dunedin Hospital | Invercargill Hospital | P value |
|---|---|---|---|
| **Characteristics** | | | |
| No of patients | 790 | 267 | |
| % of Men | 61.5% | 67.8% | 0.06 |
| Age, mean (SD) | 65.9±12.8 | 64.7±13 | 0.07 |
| Medical history | | | |
| Hypertension | 50.6% | 43.1% | 0.03 |
| Diabetes | 16.6% | 15.5% | 0.97 |
| Smoking history | 54.9% | 54.3% | 0.86 |
| Dyslipidaemia | 63.1% | 67.4% | 0.21 |
| History of IHD | 48.9% | 40.1% | 0.01 |
| Previous CABG | 10.4% | 2.3% | <0.001 |
| CVA/TIA | 12.2% | 7.1% | 0.02 |
| PVD | 6.9% | 6.0% | 0.62 |
| Presenting characteristics | | | |
| Heart rate, median (IQR) beats/min | 74 (62-88) | 74 (64-90) | 0.37 |
| SBP, median (IQR), mmHg | 137 (120-158) | 140 (120-155) | 0.47 |
| DBP, median (IQR), mmHg | 76 (65-88) | 80 (70-90) | <0.001 |
| Killip class | | | 0.07 |
| I | 84.4% | 80.9% | |
| II | 12.3% | 17.2% | |
| III+IV | 3.4% | 1.9% | |
| **ACS subgroup** | | | |
| STEMI | 31.7% | 55.8% | <0.001 |
| NSTEMI | 41.1% | 34.1% | |
| Unstable angina | 27.2% | 10.1% | |
| Cardiac arrest on admission | 6.5% | 10.1% | 0.05 |
| Renal impairment (Cr>0.12mmol/L) | 21.9% | 12.8% | <0.001 |
| Initial creatinine level, µmol/L mean (SD) | 107±43 | 99±37 | 0.01 |
| **LV dysfunction** | | | |
| Good function | 42.1% | 35.9% | 0.15 |
| Mild | 26.1% | 31.7% | |
| Moderate | 20.6% | 19.8% | |
| Severe | 10.8% | 12.6% | |
| **Medications On discharge** | | | |
| Aspirin | 97.3% | 97.7% | 0.72 |
| ACE-I | 53.4% | 49.1% | 0.22 |
| Beta-blocker | 78.3% | 71.3% | 0.02 |
| Statins | 65.1% | 41.5% | <0.001 |

ACE-I: ACE inhibitor, CVA: cerebrovascular accident, TIA: Transient ischemic attack, PVD: Peripheral vascular disease

Table 2 shows the hazard ratio (95% confidence intervals) for mortality, with stepwise adjustments.

**Table 2. Hazard ratio (95% confidence intervals) for mortality**

| Period | Unadjusted | Adjusted (1) | Adjusted (2) | Adjusted (3) | Adjusted (4) |
|---|---|---|---|---|---|
| 2 years | 1.69 (1.15–2.48) | 1.43 (0.97–2.10) | 1.42 (0.97–2.09) | 1.33 (0.90–1.97) | 1.22 (0.82–1.81) |
| Whole period* | 1.26 (0.90–1.75) | 1.12 (0.80–1.57) | 1.14 (0.81–1.59) | 1.07 ( 0.76–1.50) | 0.96 (0.68–1.36) |

**Adjusted (1):** Adjusted for GRACE score

**Adjusted (2):** Adjustment (1) + Adjusted for the use of aspirin, and the use of ACE-inhibitors

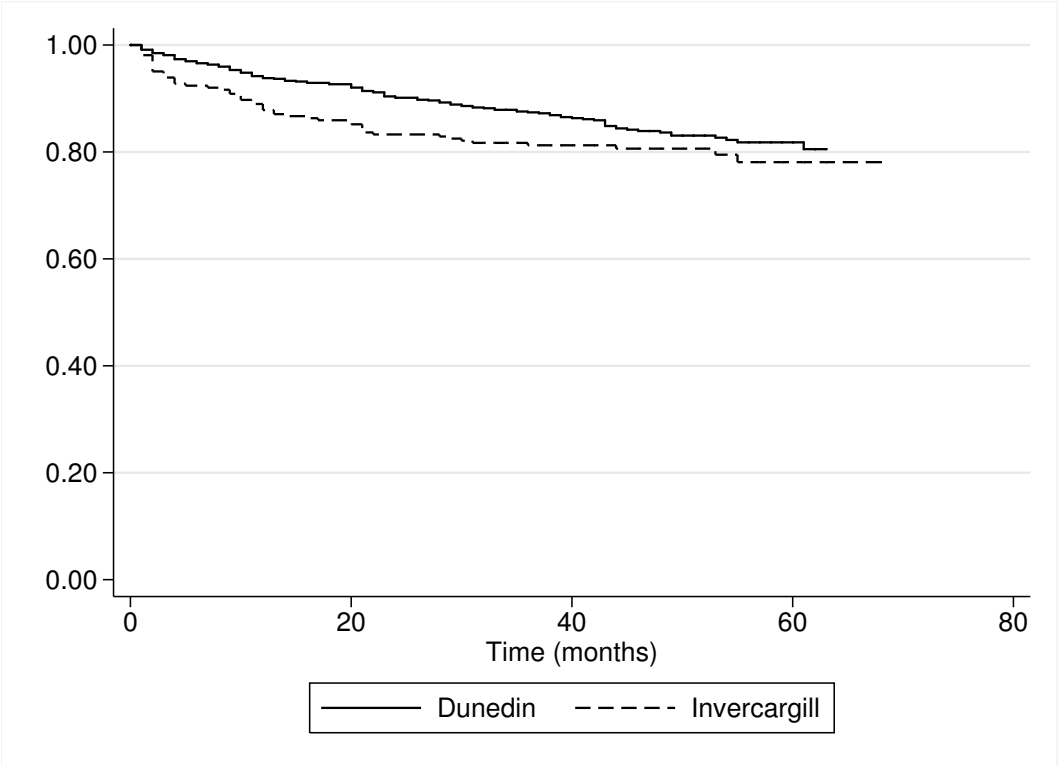
**Adjusted (3):** Adjustment (2) + Adjusted for the use of beta blockers

**Adjusted (4):** Adjustment (3) + Adjusted for the use of statins

*5 years

The Kaplan-Meir survival curves of 2 cohorts of initial hospital survivors with ACS from the 2 hospitals are shown in Figure 1.

**Figure 1. Kaplan-Meir survival curves of two cohorts of initial hospital survivors**



## Discussion

A major impetus for the current study is the fact that the mortality difference of 3.3% at hospital discharge between the two centres widened to about 10% at 6-months and 1 year.[1] The current study confirmed an absolute 8.5% mortality difference at 2 years among the initial hospital survivors and a trend of difference up to 5 years. More importantly, the under-use of statins for Invercargill patients was an important factor for this association.

The GRACE hospital discharge risk score summarising nine relevant prognostic factors was originally derived from the GRACE cohort using 6-month total mortality as the outcome measure with a c-index of 0.81 (i.e. a 81% chance of correctly identifying the survival status using the GRACE score in a pair of randomly selected patients including one survivor and one non-survivor).[2]

We validated this score in the current cohort of patients with ACS[3] with a C-index of 0.81 at 6 months, and importantly, the C-index staying ≥0.80 at all time points up to 4 years. The GRACE score as a single individual parameter providing accurate long-term prognostic information for the hospital survivors of ACS allowed us to model on this moderate-size cohort with higher statistical power the impact of different EBM on survival.

The GRACE score was higher for Invercargill patients and this was at least partly due to a lower rate of in-hospital PCI for Invercargill patients. While this has been discussed along with other issues concerning patient transferral from Invercargill to Dunedin,[1] it is noteworthy that the in-hospital angiography (26%) and revascularisation rate (22.4% PCI or CABG) for Invercargill patients was actually quite similar to the findings from the New Zealand Acute Coronary Syndrome audit (25% angiography, 11.2% PCI or CABG) of 2002.[8]

The transferred patients from Invercargill generally had lower risk (younger, clinically stable, fewer pre-morbidities) than those not transferred,[1] but obviously there were numerous practical issues in transferring high-risk ACS patients.[1] Overall, it would be fair to consider the decision to transfer as a joint decision between doctors of the two hospitals.

All patients reached the 2-years follow-up time point. Survival discrepancy of an absolute 8.5% at 2 years was partly related to the higher baseline risk for Invercargill patients as captured by the GRACE discharge risk score. After adjusting for this score, there was still a strong trend of worse survival for Invercargill patients with 43% relative increase in 2-year mortality (Table 2).

This relative increase in risk dropped towards unity when adjustments were made for beta-blockers and statins. The survival analysis for the whole follow-up period of up to 5 years followed the same pattern as the 2-year analysis, but the hazard ratio (relative increase in risk) for Invercargill patients was smaller.

Our previous report[1] aimed to provide some local data comparing the treatment and outcome of ACS patients admitted into a community hospital versus a tertiary hospital, as we believed this information would be crucial to both the public and health professionals/policymakers for an equitable healthcare service to all New

Zealanders. However, it deserves re-emphasis that community hospitals and tertiary hospitals are actually partners (rather than competitors) in the delivery of care.

Therefore, the critical step was to tease out the explanations, particularly the modifiable factors, for better survival. In this cohort of 1057 hospital survivors of ACS, we have examined the relationship of EBM and survival up to 5years. We found that among the 1025 patients taking aspirin, different EBMs (beta-blockers, ACE-inhibitors, and statins) conferred different survival benefit with statins likely being the most important. Furthermore, significant mortality reduction with statins was observed early on from 6 months.[9]

In the current report, the disparity in outcome between the two hospitals, as reflected by the hazard ratios, both at 2 years and for whole follow-up period was much reduced when the discrepant use of EBM particularly the statins at the time of initial discharge was adjusted for. Of note, the PHARMAC rule for statins only changed in April 2002 allowing more liberal use of this class of medications.

Even among the Invercargill patients, the use of statins was higher in those transferred to Dunedin than those not transferred (75.0% vs 27.9% for non-ST elevation ACS patients).[1] The different practice of the two hospitals with regard to the threshold on starting statins had contributed to the disparity in survival.

We previously reported an improving hospital outcome with ACS over the last 3 decades in Dunedin despite an increasing proportion of older patients with the condition.[10] As in all fields of medicine, improved outcomes follow scientific advances. The example of statins therapy serves as a major milestone,[11] and could possibly be the most lifesaving invention in medicine.

As the field evolved based on very many large-scale randomised clinical trials,[11] lower and lower targets of LDL levels are set. In parallel with these advancements, PHARMAC rules on lipid lowering therapy have also been changing over the years.

**Study limitations—**Multivariable regression incorporating GRACE score cannot adjust for factors, such as angiographic variables, not contained in the score but we previously showed a good C-index of this score in predicting mortality.[3]

This study included only patients admitted into the CCUs of the twp hospitals, and would have been affected by issues like admission policies. The use of medications (including statins and other EBM) and revascularisation after initial hospital discharge could have affected outcome. Indeed, the less dramatic difference between the two centres after the first 2 years, as shown in the Kaplan-Meir survival curve, might possibly be due to increased prescriptions of statins to Invercargill patients at subsequent follow-up visits following the change in PHARMAC funding on statins.

## Conclusion

There was a disparity in survival between ACS patients managed in a community hospital and a tertiary hospital during an up to 5-years follow-up period but this was due to many factors including a difference of baseline patient risk. The use of EBM particularly statins must be encouraged even if revascularisation is not performed.

**Competing interests:** None.

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## Appendix 1. The GRACE risk calculator for 6-month postdischarge mortality after hospitalisation for acute coronary syndrome

| **Medical History** | |
|---|---|
| Age in Years | Points |
| ≤29 | 0 |
| 30-39 | 0 |
| 40-49 | 18 |
| 50-59 | 36 |
| 60-69 | 55 |
| 70-79 | 73 |
| 80-89 | 91 |
| ≥90 | 100 |
| History of Congestive Heart Faihre | 24 |
| History of Myocardial Infarction | 12 |
| **Findings at Initial Hospital Presentation** | |
| Resting Heart Rate, beats/min | |
| ≤49.9 | 0 |
| 50-69.9 | 3 |
| 70-89.9 | 9 |
| 90-109.9 | 14 |
| 110-149.9 | 23 |
| 150-199.9 | 35 |
| ≥200 | 43 |
| Systolic Blood Pressure, mmHg | |
| ≤79.9 | 24 |
| 80-99.9 | 22 |
| 100-119.9 | 18 |
| 120-139.9 | 14 |
| 140-159.9 | 10 |
| 160-199.9 | 4 |
| ≥200 | 0 |
| ST-Segment Depression | 11 |
| **Findings During Hospitalisation** | |
| Initial Serum Creatinine, mg/dL | |
| 0-0.39 | 1 |
| 0.4-0.79 | 3 |
| 0.8-1.19 | 5 |
| 1.2-1.59 | 7 |
| 1.6-1.99 | 9 |
| 2-3.99 | 15 |
| ≥4 | 20 |
| Elevated Cardiac Enzymes | 15 |
| No In-hospital Percutaneous Coronary Intervention | 14 |

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# Blue-collar workplaces: a setting for reducing heart health inequalities in New Zealand?

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**Abstract**

**Aim** To review the evidence for the effectiveness of workplaces as settings for cardiovascular health promotion and reduction of heart health inequalities in New Zealand.

**Methods** Literature review and structured appraisal of 154 articles meeting inclusion criteria, of which one review and three trials addressed cardiovascular interventions specifically, and four systematic reviews addressed the effectiveness of workplace health promotion programmes generally.

**Results** The reviewed studies showed that workplaces have good potential as settings for health promotion. We found mixed but largely supportive evidence that workplace interventions can lead to improvements in health outcomes, workplace environments, lifestyles, and productivity. Workplace programmes that ranked highest in both clinical and cost-effectiveness targeted industries employing large numbers of blue-collar workers, tackled multiple risk factors, intervened at both individual and environmental levels and incorporated occupational safety components. Such programmes appear to offer a substantial return on investment for employers in other countries, but local evidence is lacking.

**Conclusions** Employers and workers in blue-collar industries should be encouraged to participate in comprehensive heart health promotion programmes as a strategy for reducing existing socioeconomic and ethnic disparities in health. However, high-quality evidence of improved employee health and productivity is needed from well-designed New Zealand-based research to ensure that these programmes are optimally configured for effectiveness and attractive to employers and employees alike.

Cardiovascular disease (CVD), New Zealand's leading modifiable cause of death,[1–4] is associated with widening socioeconomic and ethnic inequalities in risk factors and outcomes.[5–7] The CVD burden experienced by Māori and Pacific (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) people in New Zealand is high, and the burden for Māori in particular is projected to increase.[2,8] Indeed, innovative interventions in a range of settings are urgently needed to reduce the level and impact of CVD risk factors amongst Māori and Pacific people.[8–10]

The Ministry of Health's *Healthy Eating Healthy Action* strategy has again thrown light on workplaces as settings with potential for large-scale health gain.[11,12]

Workplaces have much to offer in promoting health and reducing inequalities:[4,13–15]

- People of working age can be reached before disease develops;[16]
- There is significant time spent at work (22% of employees work at least 50 hours/week);[17,18]
- The working population is large and relatively stable (over two-thirds of adult New Zealanders are in paid work);[17,19–21]
- Barriers to participation such as cost, time, and travel are low;[19,20]
- Established channels of communication exist;[22]
- The setting is familiar and offers flexibility;[19]
- There is scope for peer support;[21] and
- The ability to influence the workplace environment can be significant.[20,23]

So-called 'blue-collar' workplaces (with a workforce comprising mainly unskilled or semi-skilled manual labourers on an hourly wage) in New Zealand typically have large numbers of Māori and Pacific employees and therefore have potential as settings for health improvement interventions.[24]

However, while the number of 'white-collar' workplaces with established programmes for improved worker health and wellbeing (such as subsidised gym membership and group walking programmes) is growing,[25] relatively few programmes target blue-collar workers in New Zealand.

The National Heart Foundation's *Heartbeat Challenge* (HBC) programme, delivered by Auckland Regional Public Health Services, is the largest of these programmes for blue-collar workers; it operates in a range of workplaces throughout the greater Auckland region.[26] However, the lack of local data on the return to employers on investment of time and resources in such programmes is a significant stumbling block to employer participation.[27] Furthermore, whilst scaling-up current workplace health initiatives such as HBC is possible, the uptake by employers and the health and economic gains that might be expected from such an investment are uncertain.

In this paper we review the evidence for the effectiveness of workplace cardiovascular health promotion programmes to improve heart health and reduce inequalities, and consider the scope for health gain and return on investment from such interventions in the New Zealand setting.

## Methods

We undertook a structured literature review between December 2005 and January 2006, first looking for evidence for the effectiveness of workplace *cardiovascular* health promotion programmes, specifically focusing on risk factor reduction and productivity indicators in multi-factorial randomised controlled trials (RCTs).

We then retrieved articles that considered the effectiveness of workplace health promotion programmes in general, focusing on robust systematic reviews. Next, we searched for evidence of the cost-effectiveness of interventions identified in the first two searches. Table 1 gives details of the search strategy.

The review was confined to English language articles and studies based solely in workplaces. One reviewer (BN) reviewed the titles and abstracts of articles and excluded those that did not meet our

 

criteria. Two reviewers screened articles that met the criteria or that were unable to be excluded from the abstract alone. Disagreements were resolved by a third reviewer.

Studies were excluded if they did not use measures of effectiveness, were not conducted solely in the workplace, did not randomly allocate participants or worksites to the intervention, or did not include a control group. From 154 studies reviewed we identified one systematic review and three primary studies that addressed cardiovascular interventions specifically, and four systematic reviews that examined the effectiveness of workplace health promotion programmes generally. These were appraised using the GATE tool[28] and are summarised in Table 2. A meta-analysis was not attempted due to the wide heterogeneity in methods, interventions and outcomes reported in the different studies.

### Table 1. Search strategy

| Databases searched | Search terms | Hand searches (journals and websites) |
|---|---|---|
| Medline (1966-June 2006), Pubmed, CINAHL (1982-January 2006), Current Contents (1995-January 2006), Health and Medical Complete, Embase (1980-January 2006), Google Scholar, Index New Zealand (1987-January 2006), Proquest, Science Direct, Cochrane Controlled Trials Register, Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews and Effects, NHS Economic Evaluation Database, and Health Economic Evaluation Database. | workplace, work place, job site, work location, work-site, worksite; wellness, health promotion; effectiveness; programme*, program*; random*; low income, blue collar, white collar, social class, caste, socioeconomic status, middle class; New Zealand; cardiovascular (including diabetes, hypertension, hypercholesterolaemia, smoking, nutrition, physical activity, obesity, or similar synonyms) | American Journal of Health Promotion (1988-December 2005) and American Journal of Public Health, and articles in the bibliographies of literature retrieved. New Zealand Guidelines Group, New Zealand Ministry of Health, United States Centers for Disease Control, Health Canada, Department of Health & Ageing Australia, and UK Department of Health. |

## Results

**Workplace cardiovascular health promotion programmes—**In a critical review of workplace cardiovascular health promotion interventions, Pelletier found (from 12 randomised placebo controlled trials [RCTs]) strong evidence of effectiveness in reducing total mortality, coronary heart disease (CHD) incidence, sick leave, and absenteeism in the medium term—and smoking cessation in the long term, [20] A variety of interventions aimed at the individual, group and environmental levels were included in the trials (e.g. health education, personal counselling; individual goal setting; behaviour change incentives; group exercises; skills-based risk reduction modules; workplace environment modifications and policy changes; and employee steering committees to tailor activities).[20]

Outcomes included smoking, blood pressure, weight, blood cholesterol, physiological measures of fitness, dietary fat intake, self-reported health and wellbeing, absenteeism, and morbidity and mortality.[20] The majority of studies reported reduced risk behaviours in the intervention groups in the short-term. Long-term effects were less impressive, with the exception of smoking cessation, which was maintained at both 5 and 12-year follow-up periods.[20]

We found three additional RCTs that met our inclusion criteria.[29–31] These reported modest CVD risk factor improvements when measured between 3 and 18 months after the study intervention (Table 2).

Sample sizes ranged from 299–431; the studies targeted such factors as weight, blood pressure, cholesterol, smoking, and physical activity. None showed improvements in all the targeted risk factors.

## Table 2. Cardiovascular disease intervention studies

| Authors | Study type | Outcomes | Intervention description | Duration of follow-up |
|---|---|---|---|---|
| Pelletier KR (1997)[20] | Systematic review (12 RCTs) | Total mortality 17.5% lower in the intervention group than control (p=0.038). Coronary heart disease incidence reduced by 24.5% (p=0.031). Non-fatal myocardial infarction reduced by 26.1% (p=0.030);[32]<br>Sick leave reduction of 22% in participant group and 16% in 'all employee' group (no p-value stated);[33]<br>1.25 days less absenteeism in intervention group (p<0.0001).[34] | Medical screening and health risk assessment.<br>Comprehensive worksite-based programmes with a variety of interventions including combinations of:<br>• Educational interventions on diet, smoking and exercise;<br>• Personal counselling to high-risk groups;<br>• Individual goal setting;<br>• Behaviour change incentives;<br>• Group exercises;<br>• Skills-based risk reduction modules;<br>• Environmental modifications and policy changes;<br>• Employee steering committees to tailor activities. | 3 months to 12 years |
| Muto T, Yamauchi K[30] | RCT | BMI reduction of 0.5 (p=0.001).<br>Systolic BP reduction of 3.4 mmHg (p=0.041).<br>Total Cholesterol reduction of 10.9 mg/dl (p=0.002). | • Health promotion seminars.<br>• Multiple-components - education on lifestyle and CVD risk factors through lectures, practical training, individual counselling, group discussion, and self-education.<br>• Instructors - physician, a dietician, an exercise trainer, and program coordinators.<br>• Follow up programme - self-evaluation of goals. | 18 months |
| Gomel M et al[29] | RCT | 4% Less BMI increase (p=0.04).<br>Smoking cessation rates increased by 18% at 3 months, 9% at 6 months, and 7% at 12 months (p=0.004, 0.05, and 0.05 respectively). | 4 groups:<br>• Health Risk Factor Assessment (HRFA). CVD risk factors. 30 minute session. Referral to GP if abnormality.<br>• HRFA+Risk Factor Education (RFE) - general/unpersonalised life-style change advice. 50 minutes duration.<br>• HRFA+RFE+Behavioural Counselling (BC). As above but also up to 6 life-style counselling sessions over 10 weeks, and a life-style change manual.<br>• HRFA+RFE+BC+incentives. As above plus goal setting and follow up counselling session and incentives such as lottery draws and prizes. | 12 months |
| Proper KI et al[31] | RCT | Cholesterol reduction of 0.18 mmol/l (p=0.04).<br>Percent body fat reduction of 0.79 (p=0.015). | • Individual counselling intervention by a physiotherapist trained to use the PACE materials.<br>• Focused on physical activity and healthy eating.<br>• Option for stress and smoking counselling. | 9 months |

The range of approaches utilised in the three primary studies focused largely on behaviour change interventions in a variety of different workplace settings.

Muto et al[30] reported that (when compared to an annual health assessment) an intervention comprising individual risk factor counselling, group lifestyle education and/or skill-development sessions, health promotion seminars, self-directed education, and goal-setting resulted in improvements in surrogates of CVD risk in a predominantly blue-collar Japanese workforce (Table 2).

Gomel et al[29] found that behavioural counselling was more effective (than health risk assessment and risk factor education interventions) for minimising body mass index (BMI) increases and improving smoking cessation rates amongst ambulance officers in Sydney, Australia.

Proper et al[31] reported that individual risk factor counselling was more effective than written health information (controls) to reduce total cholesterol and body fat percentage amongst Dutch civil servants.

Interventions that target multiple CVD risks are likely to be more effective than single risk factor programmes,[20] as employees who engage in a single high-risk behaviour are more likely to have other risk behaviours (e.g. 88% of manufacturing workers who were smokers in one intervention had other CVD risk factors);[35] there is a greater opportunity to gain participation from 'high-risk' employees through offering multiple points of access;[20] employees have a choice as to which risk factor they choose to modify first;[36] and after they have successfully changed one risk factor they may be more motivated to try to change others.[37]

**Key design factors in workplace health promotion programmes—**Measures of effectiveness addressed in reviews included process factors (e.g. participation and employee acceptability), health parameters (both behavioural and biological), economic indicators, and environmental changes captured by audit (Table 3).

Factors that improve participation and acceptability (identified largely from process evaluations of workplace health interventions) included: senior management involvement; promoting employee health as a shared employee and employer responsibility; engaging workers in programme planning and implementation; providing convenient programme delivery (e.g. on-site); utilising organisational and peer support; providing incentives (e.g. free or discounted activities, recognition, competitions, good scheduling and/or using work time); tailoring the programme to the workplace environment (including occupational safety and health); providing choice across a variety of alternatives; and process evaluation.[19,22,38–41]

Two recent reviews highlighted the importance of modifying the workplace environment; they conclude that interventions with environmental components ranked highly in both clinical and cost-effectiveness compared to single component interventions that only focused on lifestyle behaviour change.[38,40] Such interventions resulted in improvement across the range of effectiveness indicators.

**Table 3. Success factors and effectiveness indicators for workplace health promotion programmes**

| Variables | Management | Workers | Workplace/programme design |
|---|---|---|---|
| Design factors | Involvement of senior staff<br>Organisational support<br>Return on investment/ cost-benefit<br>Shared responsibility for employee health | Participation in planning and implementation<br>Peer support<br>Inclusion of families<br>Occupational safety and health useful for low-income workers | Use work time<br>On site programme<br>Tailored programme to workplace<br>Environmental change (cf. lifestyle change only)<br>Address multiple risks (cf. single component)<br>Thorough evaluation<br>Incentives (discounted activities, competitions)<br>Increased choice in programme design |
| Effectiveness indicators | Productivity<br>Absenteeism<br>Presenteeism<br>Staff turnover | Participation<br>Knowledge<br>Behaviour<br>Skill development<br>Smoking cessation<br>CVD risk measures*<br>CVD mortality/morbidity<br>Job satisfaction | Workplace environment modifications |

*BMI, lipids, BP, diabetes, fruit & vegetable intake, physical activity

**Economic evaluation findings—**Internationally, companies have benefited from workplace programmes through: reducing sick leave, absenteeism and staff turnover; improving productivity, company profile, corporate image, staff morale, employee satisfaction, and customer loyalty; and producing a more receptive climate for (and better ability for employees to cope with) workplace changes.[11,41–43]

Review articles demonstrate that well-conducted health promotion programmes can save employers money.[19,43–45] Favourable economic returns have been delivered through targeting high-risk blue-collar employees, often through reducing medical costs, absenteeism, and staff turnover.[19,40]

Returns on investment (ROI) between US$1.42–$8.81 per dollar spent have been found.[11,41–43] Despite workplace cardiovascular health promotion research often lacking formal economic evaluation,[43,45] Pelletier's 1997 review[20] identified reduction in sick leave and absenteeism amongst participants in the intervention groups of a number of RCTs of workplace interventions.[33] However, the applicability of such evidence to the New Zealand setting is difficult to determine. For example, US businesses incur more direct costs due to poor employee health because employers pay for their health insurance.[46] New Zealand-based research on cost- and productivity-related outcomes is critical to support an evidence-based business case for workplace health promotion here.

## Discussion

Our review indicates that there is good evidence from a small number of well-conducted RCTs in overseas contexts that workplace health promotion interventions can achieve improvements in objective biological parameters of CVD risk and provide a positive return on investment for employers.[20,29–31]

We identified a mismatch between the qualitative design factors that increase employee participation in workplace programmes (Table 3) and the health promotion

intervention element of the RCTs in our review. Those trials that showed evidence of efficacy largely used individual health education to promote behaviour change. However, this approach has limitations: workers who volunteer for programmes requiring active participation tend to be those who are already motivated with the knowledge and resources to change.[25,47]

Environmental changes are likely to be more effective for achieving behaviour change and reducing CVD risk—especially for blue-collar workers.[48] A Cochrane review of randomised trials of *community* interventions targeting multiple risk factors using a strategy of individual behaviour change showed disappointing efficacy.[49]

Features of a workplace (such as the stable environment, social support, and number of hours spent at work) may account for the apparent superiority of workplaces over community settings for improving parameters of CVD risk. Furthermore, health promotion strategies (such as incorporating capacity building and empowerment dimensions into workplace interventions) align with the interests of unions and may lead to greater sustainability.[50] Moreover, occupational safety messages integrated with health promotion activities appear to be effective at increasing engagement.[51]

The evidence for effectiveness of workplace CVD health promotion interventions in New Zealand is sparse. The only published account we identified was that by Cook et al,[52] a non-randomised, controlled field study of key worker planning, nutrition cafeteria displays, and monthly workshops for 6 months among South Auckland male blue-collar workers that found that self-reported lifestyle behaviours (increased vegetable/reduced fat intake and increased physical activity) were significantly improved at 12 months in the intervention group, although objective biological measures showed variable results. Study strengths included a high retention rate (94% and 89% at 6 and 12 months respectively) and high participation levels at workshops (77%).

Our review suggests that blue-collar workplaces are feasible settings for achieving health gain. The National Heart Foundation's *Heartbeat Challenge* programme; focusing on healthy food choices, regular physical activity and being smokefree; has been successful in creating and sustaining supportive health-promoting workplace environments, including structural changes such as to cafeteria food policy.[53] However, there is no evidence of its effectiveness in CVD risk reduction, nor on what outcomes are important to workers, companies, and stakeholders in the New Zealand setting.

Calls have been made for more robust research to understand the specifics of the New Zealand workplace and how programmes might affect employer and employee behaviour.[25] Indeed, workplace health promotion research has often lacked rigour: most studies are non-randomised and/or uncontrolled;[21] with poor description of the interventions;[40] using uncorroborated self-reports; high losses to follow-up; inadequate sample sizes for statistical power;[19] and inconsistency between the unit of randomisation and analysis.[20,38,51] Process evaluations are rarely conducted, with inadequate documentation of: the degree of workplace 'readiness' and how this interacts with implementation and outcome successes or failures; the level of involvement of the workforce in programme planning, implementation and evaluation; the multiple components of complex programmes; and the aspects of

programme implementation that help explain positive or negative findings.[16,19,22] Furthermore, most workplace trials have involved middle-income people.[54]

A study incorporating the design elements associated with improved participation and economic measures of relevance to New Zealand would add to the evidence base for the workplace as a setting to address heart health inequalities in the local context. Innovative features such as text messaging to improve participation and outcomes could be incorporated.[55]

Local research to establish a business case for such programmes is vital to improve employer interest and enhance the likelihood of sustainability. However, the expected magnitude of improvement in employee CVD risk from any intervention may be small; and the economic climate and low rate of employer provision of health care insurance in New Zealand will also affect the return on investment.

While workplaces with 100 or more employees comprise only 0.5% of the total number of workplaces in New Zealand they employ almost half (47.2%) of all employees,[24] and many Māori and Pacific people. Therefore, to reduce heart health inequalities, large blue-collar workplaces (e.g. factories) should be the initial focus for any such interventions.[40]

## Conclusions

Sufficient evidence exists to proceed with comprehensive workplace heart health promotion programmes, especially those involving large blue-collar worksites. In addition, evaluations of existing interventions plus research which explores acceptability, effectiveness, changes to productivity, and return on investment for employers should be encouraged, to build the local evidence base and business case.

**Competing interests:** None.

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# Sudden cardiac death in the young: don't forget abnormal coronary arteries!

Ali Khan, Jim Stewart, Warren Smith

## Case report

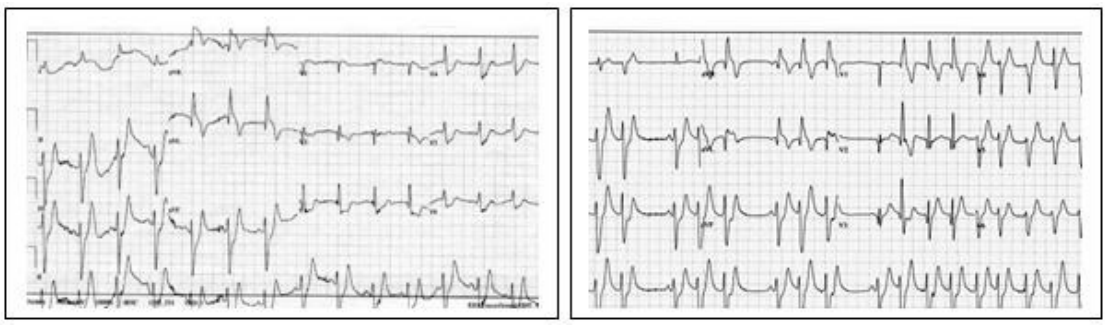
A 16-year-old boy was found collapsed on the ground following an outdoor concert. He was conscious but shocked, had severe respiratory distress with frothing secretions from his mouth, and was incontinent of urine. A cardiac rhythm strip from the ambulance showed junctional tachycardia with ST-depression.

He developed severe chest pain and breathlessness before he collapsed, as he was running to catch up with friends for a ride home. He later described one prior episode of severe breathlessness after a run at age 13 years. He had avoided heavy exertion since then. He was well built and had no history of syncope or any other medical problem. He denied any recreational drug use. His father had drowned while fishing, in circumstances that were unclear.

On arrival in ED he was in florid heart failure with central cyanosis and cardiogenic shock. The chest X-ray (CXR) showed pulmonary oedema with a normal-sized heart. His cardiac rhythm was very unstable, alternating between junctional tachycardia and ventricular tachycardia (VT). A 12-lead ECG showed 2 mm anterior ST-depression (Figure 1).

He had metabolic acidosis with high lactate (5.9 mmol/L). His haemoglobin (Hb) was normal, white blood cell (WBC) count was mildly elevated, and serum creatinine was raised to 150 mmol/L. A bedside transthoracic echocardiogram showed severe hypokinesis and poor thickening of the anterior left ventricular wall, consistent with significant ischaemia/evolving infarction.

**Figure 1. Admission ECG showing anterior ST-depression and subsequently junctional tachycardia**

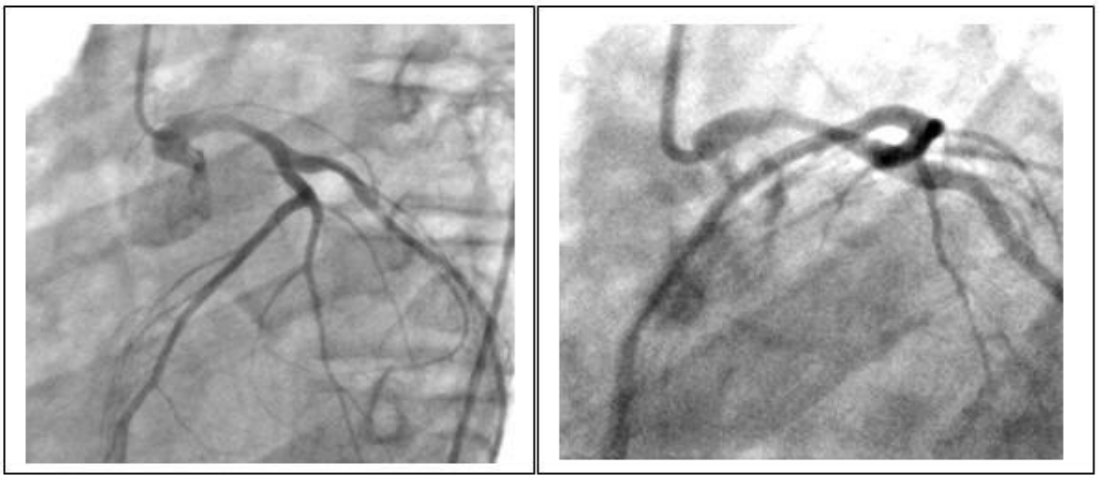


He was admitted to the Cardiac Intensive Care Unit on non-invasive ventilation support and inotrope infusion. A 4-hour troponin-T level was raised at 9.9 mcg/L.

Urgent coronary angiography showed anomalous origin of the left coronary artery from right aortic sinus, coursing between the aorta and the pulmonary trunk. A balloon pump was inserted. A few hours later he had a cardiac arrest. Following successful resuscitation he underwent emergency coronary artery bypass graft (CABG) surgery with left internal mammary artery (LIMA) to the left anterior descending (LAD) artery and a vein graft to the circumflex artery. He needed a external left ventricular assist device (LVAD) to support weaning from cardiopulmonary bypass.

The LVAD was removed on the third postoperative day. He remained on ventilator and circulatory support for a week. His condition gradually improved and he was discharged after a lengthy hospital stay. At 4-month follow-up, coronary angiography showed patent native coronaries and grafts.

**Figure 2. LAO 38 and cranial 43 views showing left coronary arising from right and coursing upward, posteriorly and to the left between aorta and pulmonary trunk**



## Discussion

Anomalous coronary artery origin is a rare but important cause of sudden cardiac death in young people. The estimated incidence from large series' of angiographic studies is ~1% in the general population.[1] It is the cause of 13% of sudden death in athletes in the United States.[2]

Anomalous left coronary artery arising from right aortic sinus is quite rare, but frequently associated with sudden death during exertion. The condition is often diagnosed retrospectively at autopsy or investigating a case of aborted sudden death. Chest pain, effort dyspnoea, or syncope in a young person should raise suspicion. Resting and stress electrocardiogram are usually unhelpful.[3] Transthoracic

echocardiography can be useful but transoesophageal echo has higher sensitivity in detecting anomalous coronary origin and defining the proximal course.[4]

Coronary angiography remains the definitive investigation, but the origin and proximal course of the anomalous vessel may be difficult to define.[5] Cardiac MR compares favourably with angiography.[6]

Anomalous origin of the left coronary artery from the right sinus may have one of four different proximal courses: inter-arterial, anterior free wall, retro-aortic, and septal course.

The inter-arterial variety is more frequently associated with exertion related sudden death, the possible mechanisms of which may include acute angle of take-off and kinking of left coronary artery (LCA), compression between aorta and pulmonary artery during exertion or obstruction in the aortic intramural course, spasm, and ventricular arrhythmia due to myocardial fibrosis from recurrent ischemia.[7,8]

There is no specific management guideline but surgery is accepted as the definitive treatment in such cases of aborted sudden death, or in young (<35 years) patients with evidence of ischemia or ventricular arrhythmias. Management in older patients is controversial as they have lower risk of sudden death; the same is probably true for young patients without evidence of ischaemia.

The standard surgical approach has been grafting the LAD and circumflex.[9,10] Newer techniques like 'unroofing' of the aortic intramural course of the vessel with reconstruction of the ostium has been described with promising results but carries risk of aortic valve damage.[11] Emergency bypass surgery usually has higher complication rates. Moreover, young patients will outlive their grafts and may require multiple operations in their lifetime. Competitive flow in the native coronary artery may predispose to early graft occlusion.[12] Although percutaneous left main stenting is feasible, its role in management of anomalous coronary arteries has not been tested. It is not currently recommended as an alternative to CABG.

This case highlights some important aspects in management of such cases of aborted sudden death. Our patient is well on follow-up, but some issues regarding his ongoing care and prognosis remains unclear. He is young and his grafts are vulnerable due to competitive flow; he is likely to need multiple future interventions. We are unsure of the ideal way of monitoring his graft function; a symptom-directed approach may prove to be hazardous and risk his life.

It is unclear how much physical activity can be safely recommended for him; consensus is for restriction of vigorous physical activity. Available data is scarce on long-term follow-up of such cases due to the rarity of ante-mortem diagnosis. Maintaining an International Registry may provide data to guide future management and follow-up strategies.

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# A 40-year-old woman with breathlessness

Ami Kamdar, Andrew F Muller, Stamatis Kapatenakis, Louise Thompson, David A Lythall

A 40-year-old Caucasian lady, with known aortic regurgitation, attended our outpatient clinic with a 4-week history of progressively worsening shortness of breath both on exertion and at rest. She admitted to symptoms of malaise, loss of appetite, and night sweats.

Her past medical history included mild mitral regurgitation; moderate to severe aortic regurgitation; a right-sided Wilm's tumour at 3 years of age (successfully treated with nephrectomy); followed by chemotherapy and radiotherapy for a lung metastasis; an Arnold-Chiari malformation treated with decompression; primary hyperparathyroidism; and scoliosis. Her current medications included daily ramipril 10 mg, atenolol 25 mg, bendroflumethiazide 2.5 mg, and aspirin 75 mg.

On examination, the patient was found to have a grade 3/4 diastolic murmur of aortic regurgitation at the left sternal edge and a grade 2/6 apical systolic murmur of mitral regurgitation. There were no stigmata of endocarditis or evidence of cardiac failure. There were surgical scars. Blood pressure was 160/40 mmHg.

Blood tests were normal (full blood count, electrolytes, troponin Ts, blood cultures, and inflammatory markers). The chest X-ray was unremarkable as was the ventilation-perfusion scan. Her electrocardiogram fulfilled the criteria for left ventricular hypertrophy.

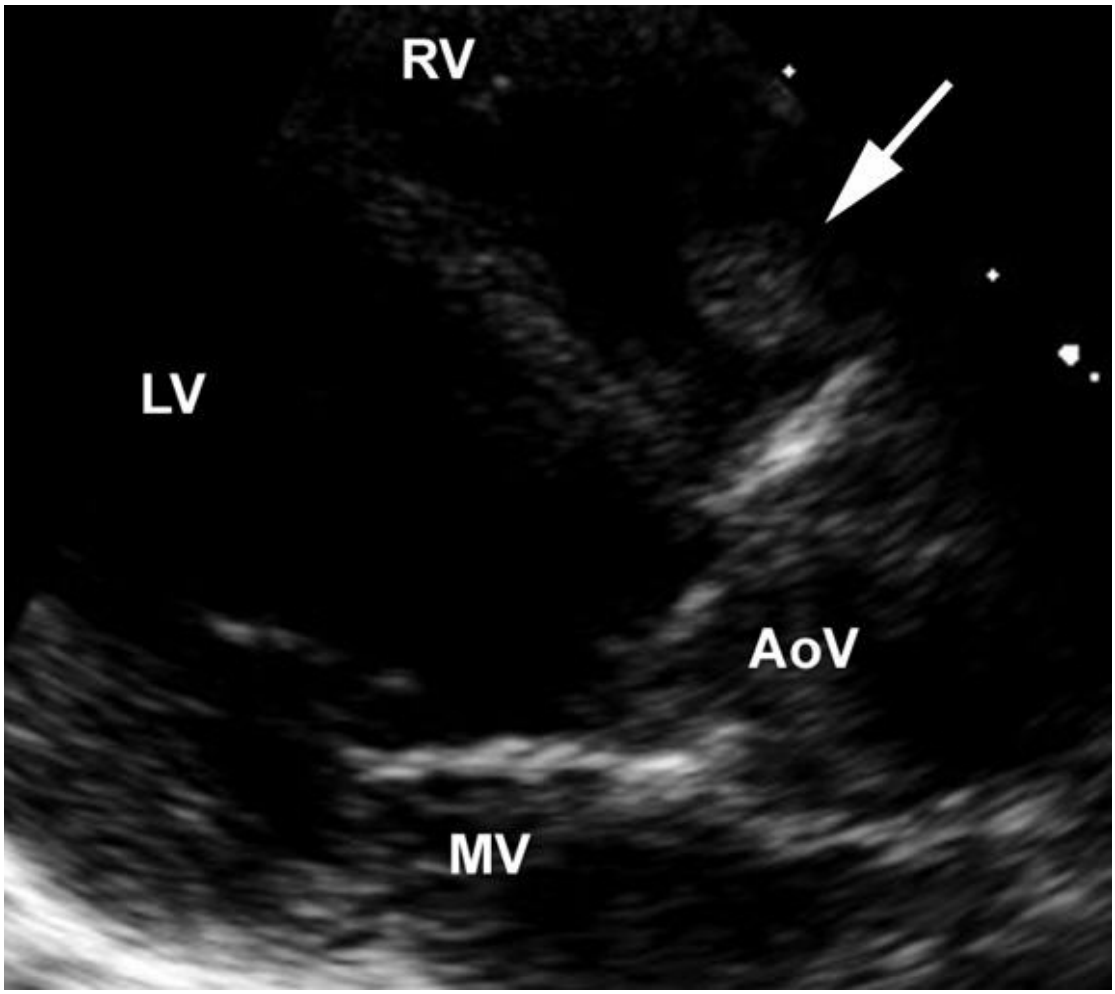
An urgent transthoracic echocardiogram was performed to re-evaluate left ventricular dimensions, the degree of aortic regurgitation, and to rule out echocardiographic evidence of subacute bacterial endocarditis. This confirmed the previous findings of moderate-severe aortic and mild mitral regurgitation, with no visible vegetations. However a 4×2 cm mass was noted in the right ventricular outflow tract (Figures 1 and 2).

A subsequent transthoracic echocardiogram indicated that the mass was not attached to either the tricuspid or pulmonary valves but was associated with the posterior aspect of the mid-right ventricular outflow tract wall. This was suggestive of either a tumour or a thrombus.

The patient proceeded to a surgical excision of the mass and aortic valve replacement. Histology revealed a papillary fibroelastoma; a postoperative echocardiogram showed no evidence of residual tumour, but there was some abnormal septal wall motion. The ejection fraction was approximately 72%.

She made a good recovery postoperatively with good symptomatic improvement of her symptoms.

**Figure 1. Oblique long axis parasternal view demonstrating a large mass near the right ventricular outflow tract (arrow)**

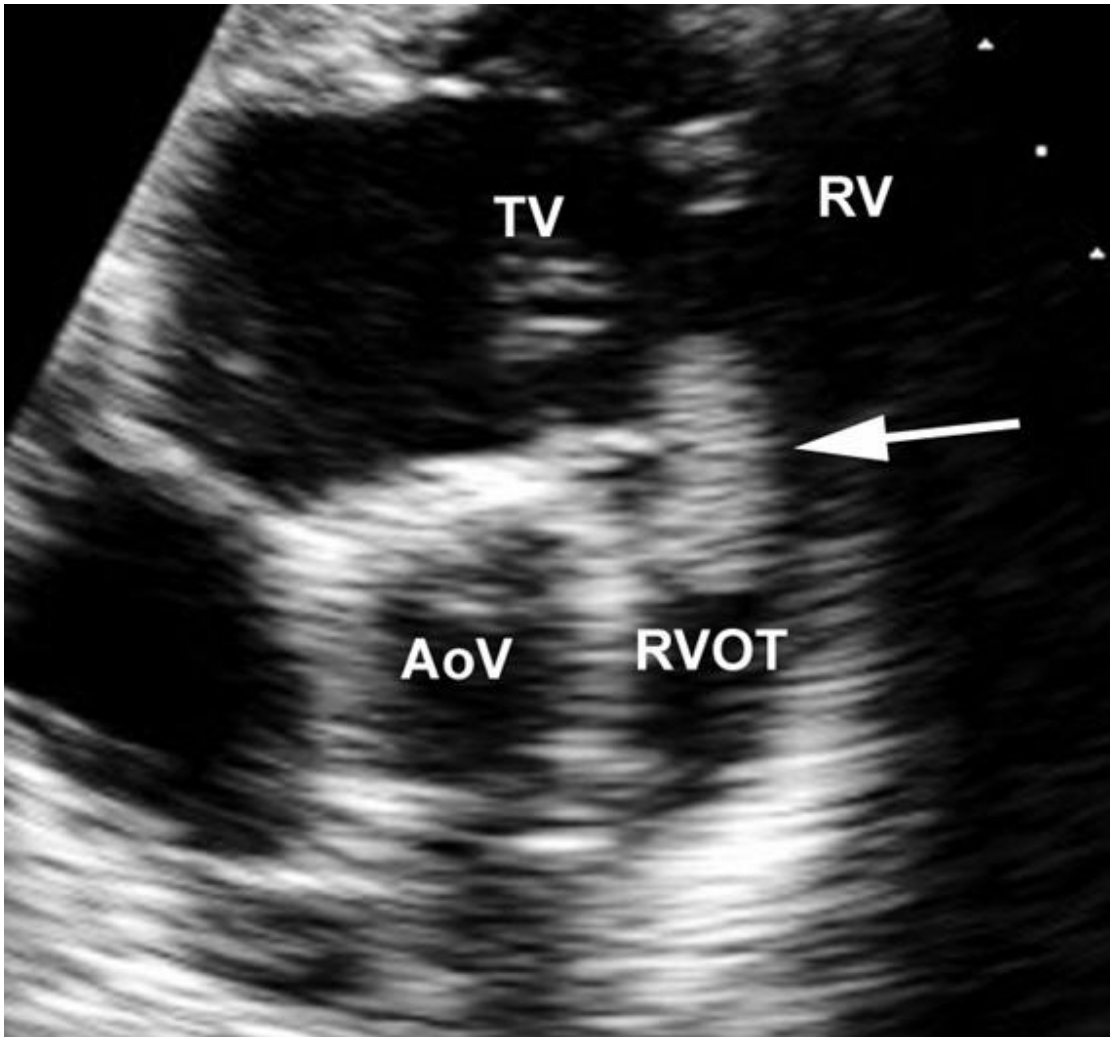



LV: Left ventricle; RV: Right ventricle; MV: Mitral valve; AoV: Aortic valve.

Cardiac papillary fibroelastomas (CPF) are the second most common primary cardiac tumours. They are histologically benign and are generally slow growing tumours. They were first described in 1931 by Yater,[1] though the term 'papillary fibroelastoma' was first coined by Cheitlin et al[2] in 1975, and Fishbein et al[3] described the microscopic characteristics of the tumour.

About 75% of CPFs tend to be valvular tumours. Gowda et al,[4] in a retrospective review of cases with histological confirmation of CPF, found only 9 out of 621 patients to have the tumour originating from the right ventricle. They described the common clinical presentations of CPF were embolic disease (transient ischaemic attacks, angina, myocardial infarction, pulmonary embolism, blindness, mesenteric ischaemia, peripheral emboli, and renal infarction), sudden death, heart failure, presyncope, or syncope.

**Figure 2. Short axis view at the aortic valve level (AoV) from the subcostal approach. A large mass (arrow) is seen filling most of the right ventricular outflow tract (RVOT). The right ventricle (RV) inflow tract and tricuspid valve (TV) are also seen**




The most frequent age of presentation of CPF tends to be between the fourth and eighth decades of life. Most cases are probably acquired and can be iatrogenic[5] such as from previous cardiac surgery or radiation treatment. A surgical opinion regarding resectability is warranted even with small CPFs because of their potential for embolisation.[6] The aetiology of CPF is not yet known.

Echocardiography is the most useful noninvasive mode of diagnosis. A transthoracic echocardiogram[7] may confirm the presence of cardiac tumour in terms of location and attachment to underlying structures. Surgery is a curative procedure.[4] Symptomatic patients who are not surgical candidates should be offered long-term anticoagulation, but there are no randomised, controlled trials that have assessed the efficacy of this treatment.

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# Is it time to stop treating dyslipidaemia with fibrates?

Jocelyne R Benatar, Ralph A Stewart

**Abstract**

**Aim** To determine whether evidence from randomised clinical trials supports the use of fibrates to reduce non-fatal and fatal cardiovascular events in patients with dyslipidaemia.

**Method** Review of randomised clinical trials of fibrates that assess clinical outcomes.

**Result** In clinical trials which have which have included over 40,000 patients there was no difference in all cause mortality for patients randomised to a fibrate compared to placebo. Treatment with a fibrate was associated with a small reduction in the risk of non-fatal cardiovascular events.

**Discussion** Current evidence does not support the use of fibrates to reduce cardiovascular mortality. Other proven strategies including statins, aspirin, angiotension converting enzyme (ACE) inhibitors, good blood pressure control, and lifestyle interventions should be used to reduce cardiovascular risk.

In both epidemiological and clinical studies, individuals with lower high-density lipoprotein (HDL) cholesterol[1] and higher serum triglycerides[2,3] have a greater risk of cardiovascular disease during long-term follow-up. Fibrates increase plasma levels of HDL cholesterol and lower triglycerides, and are generally well tolerated. For these reasons they have been widely used to treat dyslipidaemia. However accumulating evidence suggests other treatments are much more effective for reducing cardiovascular risk, even in patients with diabetes.

The most recent large randomised clinical trial of fibrate treatment was the Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study[4] undertaken in Australia, New Zealand, and Finland. This study randomised to fenofibrate 200 mg daily or placebo nearly 10,000 people with Type 2 diabetes who had a total cholesterol to HDL ratio greater than 4.0 or plasma triglycerides of 1.0 to 5.0 mmol/L. Fenofibrate was well-tolerated and adverse events were rare. During over 5 years follow-up fenofibrate reduced cardiovascular events from 13.9% to 12.5% (p=0.035). However total mortality was not reduced by fenofibrate (placebo 6.6%, fenofibrate 7.3%).

The absence of any favourable effect on mortality is disappointing, but is consistent with outcomes in previous studies.[5–7] Prior to the FIELD study, about 30,000 persons had been randomised in clinical trials of fibrates, with almost 3000 deaths. The majority of studies included persons with a higher cardiovascular risk because of male gender, previous myocardial infarction, and a low HDL cholesterol.

Combining all results in a meta-analysis, total mortality was not influenced by fibrate treatment (relative risk 1.0; 95% confidence interval: 0.91–1.11). In several studies there appeared to be a small decrease in cardiac mortality, but this was balanced by an increase in non-cardiovascular mortality. For all studies combined, these trends were

not statistically significant, and there is no clear evidence for an excess in non-cardiovascular mortality from any specific cause with fibrates. However combined results from randomised trials indicate that fibrates do not reduce fatal cardiovascular events.[8]

Fibrates do, however, decrease the risk of non-fatal cardiovascular events, including coronary revascularisation, stroke, peripheral vascular disease related amputations, and non-fatal myocardial infarction.[5–7] Based on the FIELD study,[4] approximately 500 patient-years of treatment are needed to prevent 1 non fatal myocardial infarction and 350 patient-years to prevent any non-fatal vascular event. By comparison ~100 patient-years treatment with a statin will prevent a major vascular event or death, with a greater benefit in individuals at high risk.[18]

Outcomes from the fibrate trials are in striking contrast to clinical trials of statins which have consistently demonstrated reduction in cardiovascular events in many populations, including patients with a low HDL cholesterol level. Statin therapy can safely reduce major coronary events, coronary revascularisation, and stroke by about one-fifth per mmol/L reduction in low-density lipoprotein (LDL) cholesterol, largely irrespective of the initial lipid profile or other presenting characteristics.[18]

Recent studies suggest aggressive cholesterol reduction with high dose atorvastatin[17] results in a greater reduction in cardiovascular risk more than more modest LDL cholesterol reduction with less potent or lower dose statins. In addition, there is good evidence from clinical trials that other interventions including angiotensin converting enzyme (ACE) inhibitors[9,10] vigorous blood pressure-lowering,[11] anti-platelet drugs,[12,13] and some lifestyle interventions reduce cardiovascular risk.

These benefits are likely to be present in a broad range of patients with different cardiovascular risk factors, including patients with low HDL cholesterol and the metabolic syndrome. Given these alternatives are there situations where treatment with fibrates can be justified?

Current guidelines recommend fibrates for type III genetic dyslipidaemia which is rare, occuring in 0.01-0.02% of the general population.[20–22] Fibrates[23] are also recommended for severe hypertriglyceridaemia (levels above 20 mmol/L) which is associated with a high risk of pancreatitis.[14–16] However there are no clinical trials on treatment of marked hypertriglyceridaemia.

Of note the commonest cause of very high triglyceride levels is adverse lifestyle, and reducing alcohol and simple carbohydrate intake has dramatic effects on triglyceride levels. Ironically in the FIELD study there was a small but significant increase in pancreatitis (0.5 vs 0.8%) for patients taking fenofibrate.

Fibrates are also often used in combination with statins in high-risk patients who fail to reach a cholesterol treatment target with a statin alone. This treatment combination is currently being evaluated in large randomised clinical trials. However current evidence does not provide strong support for the use of fibrates in combination with statins.

The New Zealand Cardiovascular Guidelines recommend that preventive treatments are initiated in patients at high absolute risk of cardiovascular disease based on a combination of risk factors rather than serum lipids alone. For these patients medications with proven clinical benefit based on results of randomised clinical trials should be prescribed.

**Competing interests:** None.

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# Increasing inability of mothers to suckle their offspring

*Extract from article entitled 'Physiological Economy in the Nutrition of Infants' by Truby King. Published in N Z Med J 1907;6(24):71–102.*

The paramount importance to the mother of sound kidneys forced itself on our profession at any early date, on account of the relationship between uraemia and puerperal eclampsia, but other allied considerations possibly of equal importance have so far almost escaped attention.

Defects of liver and kidneys which can give rise to convulsions must be attended by imperfect digestion, assimilation and mammary secretion, and one can scarcely imagine it to be a mere accident that a very large proportion of women who cannot suckle their offspring are found on examination to be suffering from albuminuria.

The observations made by Dr. Emily Siedeberg at the St Helen's Maternity Home, Dunedin, last year in this connection (see New Zealand Medical Journal, Oct. 1906) are both interesting and significant. Half the mothers who could not suckle their babies were found to be suffering from albuminuria; some thirteen per cent of those who could suckle showed albumen in the urine; and, speaking from memory, Dr. Siedeberg's impression is that the albuminuria was for the most part slight and transitory in the women whose milk secretion was satisfactory.

Recent observations made in Germany and in America show that if a woman is unable to suckle her baby her daughter shows the same disability. Further, it is established that inability to suckle is on the increase. Holt actually estimates that 75 per cent of New York society women are unable not merely unwilling to suckle their offspring.

Taking such facts as the above into consideration, we are forced to the conclusion that our first duty to the babies who are to be the mothers of the future, is to ensure as far as possible that they shall be fed in accordance with their nature as young human beings, and in accordance with the indications furnished by the study of physiological economics—that they shall be suckled if possible, and, where breast-milk is not forthcoming, that they shall be given milk so modified as to approach as nearly as practicable, both quantitatively and qualitatively, to the natural food.

If this were done, one can see no reason why suckling power should not be restored in most cases to the progeny of persons supposed to have a hereditary tendency to fail as mothers.

It is absurd to assume that the failure of a normal function, brought about by artificial conditions—such as careless-bottle-feeding, lack of sunshine, fresh-air and physical exercise; alcoholism &c.,—dating back only one or two generations, must necessarily be perpetuated.

On the other hand, .there is every reason to believe that inability to suckle offspring will be carried on from one generation to another so long as our profession continues to tolerate, and even to advocate, the use of crude cow's milk, diluted or undiluted, as a food for babies.

**NZMJ Note:** Read about Sir F Truby King at
http://www.teara.govt.nz/1966/K/KingSirFredericTrubyCmg/KingSirFredericTrubyCmg/en

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# Radiologic evolution of pulmonary thromboembolism with infarct

Sheng-Hsiang Lin, Tsu-Tuan Wu

A previously healthy 55-year-old woman presented with right-side chest pain for 4 days. The initial chest radiograph (Figure 1) showed a peripheral infiltrate involving the right costophrenic angle (arrow) and an enlarged right descending pulmonary artery (Palla's sign, arrowhead) with abrupt tapering (knuckle sign).

**Figure 1**

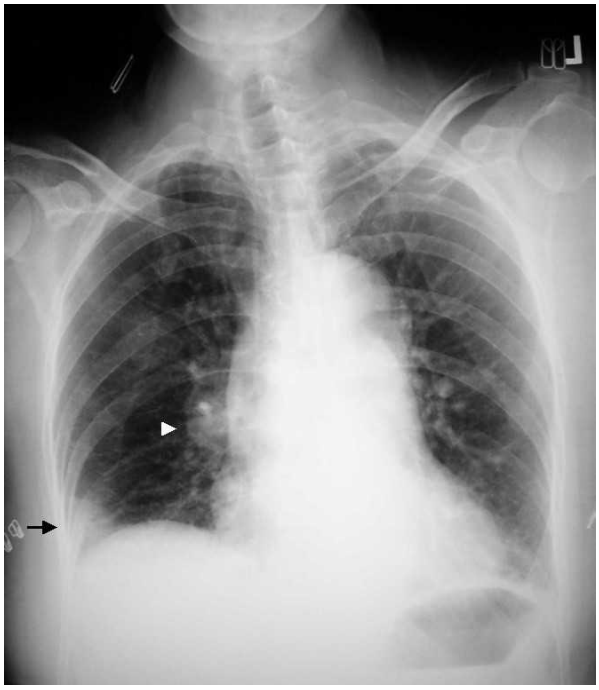
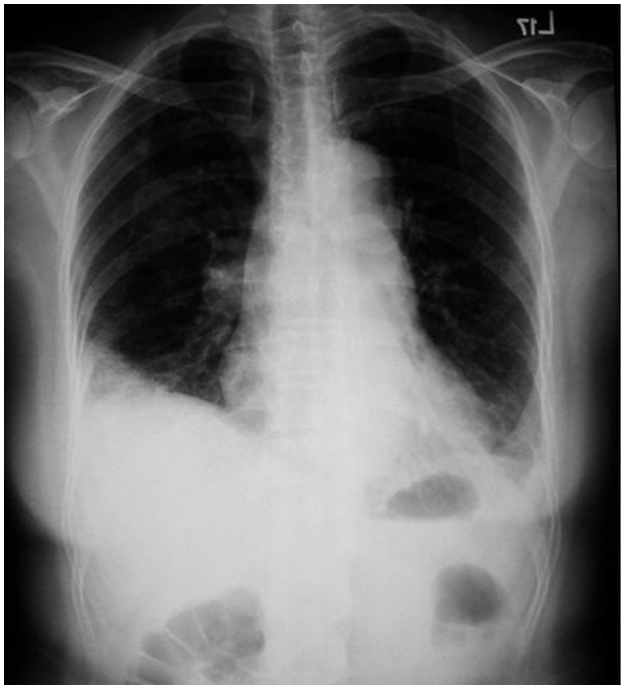


**Figure 2**



One day later (Figure 2), the infiltrate developed as a wedge-shaped consolidation at right lower lobe. Chest computed tomography with contrast (Figure 3) revealed a large filling defect in right descending pulmonary artery with extension to main pulmonary artery (Panel A) and lung consolidations in right lower lobe with a small amount of pleural effusion (Panel B). She received anticoagulation therapy.

On the 7th day (Figure 4), a pleural-based consolidation in the form of a truncated cone with the base against the pleural surface and the rounded convex apex directed toward the hilum (Hampton's hump) was noted.

On the 48th day (Figure 5), the sequelae were presented with an elevated right hemidiaphragm and the development of a localised pleural thickening along the lateral chest wall. She recovered completely and became asymptomatic.

**Figure 3**

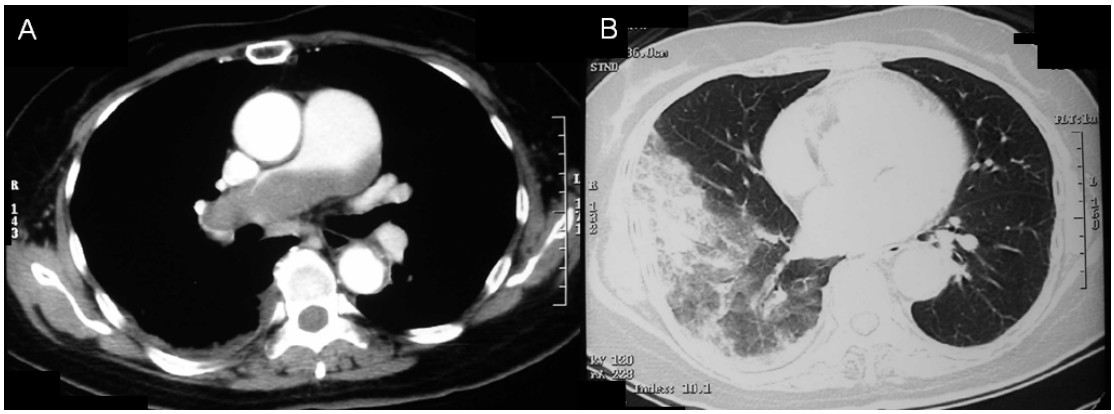



**Figure 4**

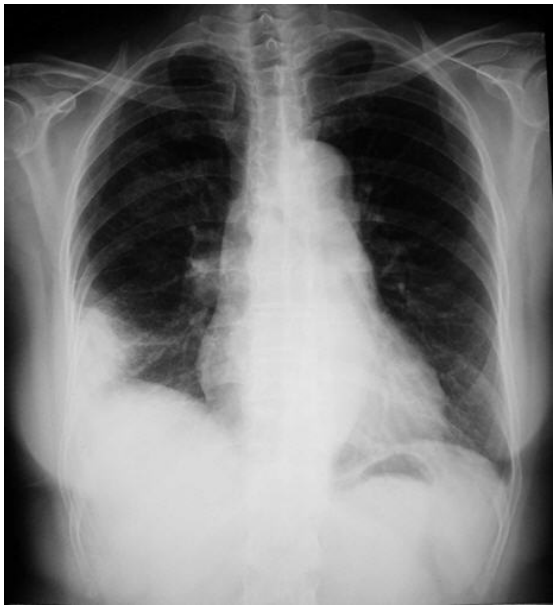
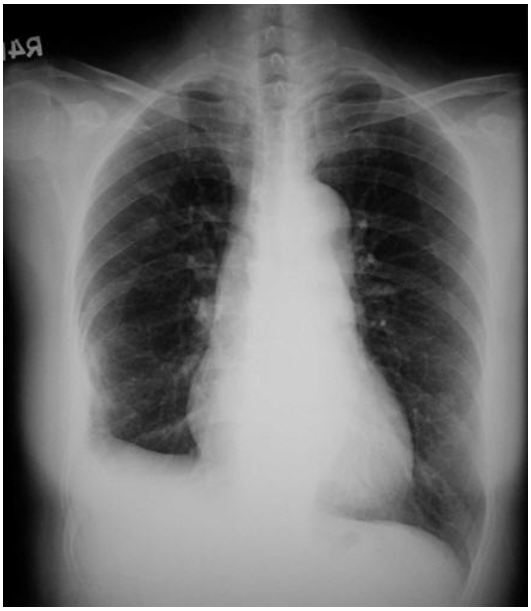


**Figure 5**



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## Medical journals and guns, again

As noted in an abstract in this journal on 29 June 2007, Reed Elsevier, the publisher of the *Lancet* and many other scientific journals, is also involved in arms fairs around the world.

In spite of much criticism they were reluctant to drop their weapon-related activities. But, there is a new development—the CEO of Reed Elsevier is quoted in the *Lancet* as saying "…it has become increasingly clear that growing numbers of important customers and authors have very real concerns about our involvement in the defence exhibitions business…We have listened closely to these concerns and this has led us to conclude the defence shows are no longer compatible with Reed Elsevier's position as a leading publisher of scientific, medical, legal, and business content."

Agreed.

Lancet 2007;369:1902

## Admission to hospital for nose bleeds

Epistaxis is the most common nasal emergency and if nasal packing is required this commonly results in admission.

In this paper, the topic is reviewed as the authors suspect that too many are admitted. They audited 116 patients with epistaxis and their findings were that (apart from cautery) 62 had nasal packing inserted. Only 17 required admission. Forty-six patients were discharged with nasal packing *in situ* and only 7 (16%) returned due to bleeding. The overall return rate was 11%. They believe that their protocol prevented 39 unnecessary hospital admissions. However, patients with significant anaemia and/or a bleeding disorder should be admitted.

The Journal of Laryngology & Otology 2007;121:222–7

## A breakthrough in the treatment of acute ischaemic stroke?

Currently, thrombolysis with alteplase (tissue plasminogen activator [rt-PA]) is the only widely approved treatment for acute stroke, and it is underused. But what about the use of neuroprotectants?

The free-radical-trapping agent NXY-059 showed promise as a neuroprotectant in animal models and in one human trial. The team who reported the earlier promising trial now report on a larger (over 3000 patients) randomised placebo controlled trial to confirm the efficacy of NXY-059. Sadly the results reveal that NXY-059 is ineffective for the treatment of acute ischaemic stroke within 6 hours after the onset of symptoms.

N Engl J Med 2007;357:562–71

## Preoperative haemoglobin and postsurgical outcome

Elderly patients are at high risk of both abnormal haematocrit values and cardiovascular complications of non-cardiac surgery. Despite nearly universal screening of patients for abnormal preoperative haematocrit levels, limited evidence demonstrates the adverse effects of preoperative anaemia or polycythemia.

These researchers reviewed postoperative outcome in 310,311 (mostly male) patients who were 65 years of age or older at the time of their non-cardiac surgery. They found that each percentage point increase or decrease in haematocrit was associated with a 1.6% increase in unfavourable outcome. And, more importantly, the adjusted risk of 30-day postoperative mortality and cardiac mortality begins to rise when haematocrit levels decrease to less than 39% or exceed 51%.

An editorial notes the results and cautions against the dangers of transfusion for moderate anaemia and mentions the possibility that in an observational study associations may be markers of adverse outcomes rather than predictors of outcome.

And, your scribe notes that 39% and 51% are practically normal haematocrit levels.

JAMA 2007;297:2481-8 & 2525–6

## Retail health clinics in the USA

What are they and where are they? They are walk-in health clinics staffed by nurse practitioners or physician assistants who diagnose and treat common illnesses, give immunisations, do physical examinations, and perform a limited number of procedures. Most are located in shopping malls or near pharmacies.

Apparently very popular with the patients but disliked by American doctors. Popular with patients because of convenient locations, long opening hours, low prices, and consistent (if not always the highest) quality. Unpopular with doctors for the obvious reasons. The question of patient safety arises and the clinics answer that they hire certified, experienced nurse practitioners who know what they are doing and when to get help.

And (perhaps), patients understand what these clinics can do and can’t do and triage themselves accordingly?

BMJ 2007;335:21

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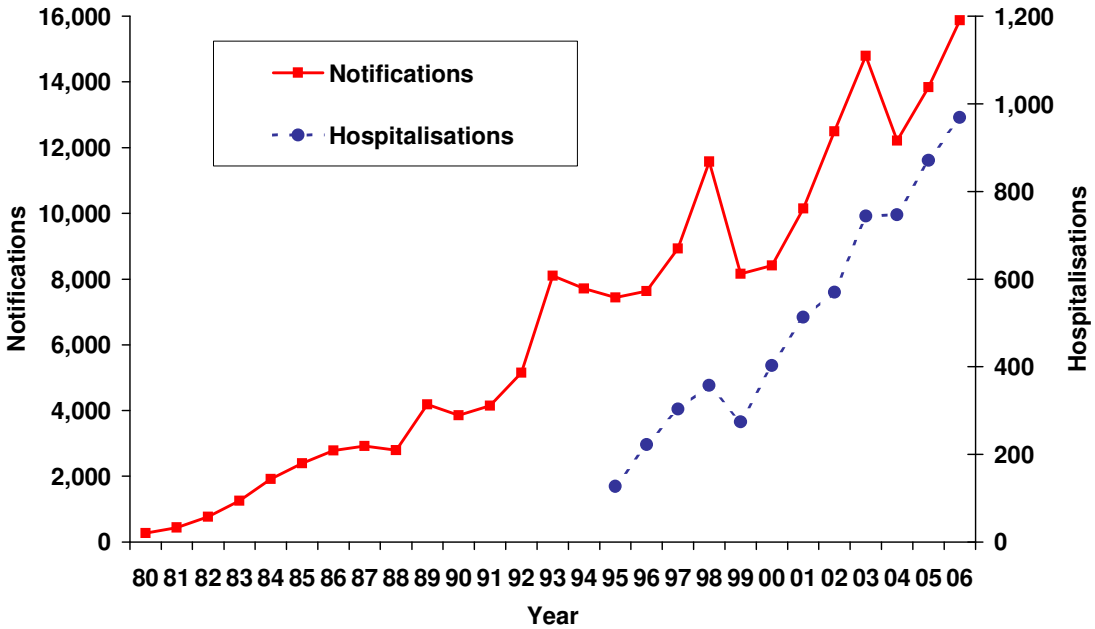
# *Campylobacter* infection and chicken: an update on New Zealand's largest 'common source outbreak'

This letter aims to provide an update on New Zealand's substantially foodborne epidemic of *Campylobacter* infection. It also seeks to put into perspective poultry industry claims that their "Campylobacter campaign shows encouraging signs".[1]

## Epidemic update

Our epidemic of *Campylobacter* infection reached a new peak in 2006 with 15,873 notifications and 969 hospitalisations, the highest totals ever reported in New Zealand[2] (Figure 1). The health impact of this epidemic now places it amongst New Zealand's most important infectious disease problems. The total number of *Campylobacter* infections in the community is conservatively estimated at 120,000 per annum (based on a widely used multiplier of 7.6 times the number of notified cases[3]).[4]

**Figure 1. Campylobacteriosis notifications (1980–2006) and hospitalisations (1995–2006) for New Zealand, by year**




**Sources:** Institute of Environmental Science and Research Limited (notifications) and New Zealand Health Information Service (hospitalisations, based on principal diagnosis).

*Campylobacter* infection can also kill. There is about one recorded fatality a year from the acute effects of this disease.[2]

There are also delayed fatalities, such as the highly publicised death of Green Party co-leader Rod Donald from myocarditis following *Campylobacter* infection. Such complications are well documented, including in New Zealand.[5] More sophisticated cohort studies have found a 3-fold increase in the risk of death within the first month following infection.[6]

Despite the scale of this epidemic, in June 2007 the executive director of the Poultry Industry Association of New Zealand (PIANZ) claimed some success in efforts to control this problem: "The New Zealand Poultry industry says small drops in the number of reported cases of human Campylobacter, is an encouraging sign and indicates the industry's extensive science and research programme…is on the right track."[1] This industry representative also went on to claim on television that progress was being made and there was a 38% drop in human cases in May 2007 compared to the same month last year.[7]

We think it is far too soon to claim progress in reducing this epidemic. We are also concerned with what appears to be highly selective use of data that could mislead the public into thinking that this problem is coming under control.

- Firstly, the numbers quoted are potentially deceptive. As Table 1 shows, the number of *Campylobacter* infections has barely declined in 2007. The incidence for the year to May was about 6% less than at the same time in 2006.
- More importantly, it is hardly reasonable to choose the worst year on record (2006) as a baseline for comparison purposes. Even though cases in May 2007 were less than in May 2006 (as PIANZ correctly pointed out), the total for May 2007 was still markedly higher than for every other May in the preceding six years (Table 1 and Figure 2).
- The incidence of *Campylobacter* infection, and other notifiable diseases, may fluctuate considerably from year to year (Figure 1) and month to month (Figure 2). We therefore need to see a substantial and sustained downward trend for several years before it is reasonable to suggest that progress is being made with controlling this disease.
- Finally, we should be wary about choosing a goal based on New Zealand's recent past history of *Campylobacter* infections. Our current notification rate (383.5 per 100,000 in 2006)[2] is so much higher than other developed countries we should be looking overseas to find an appropriate public health target. A reasonable initial goal would be to achieve the incidence rate reported by Australia (121 per 100,000 in 2005[8]). Even that rate is still 10 times higher than the United States (12.7 per 100,000 in 2006[9]).

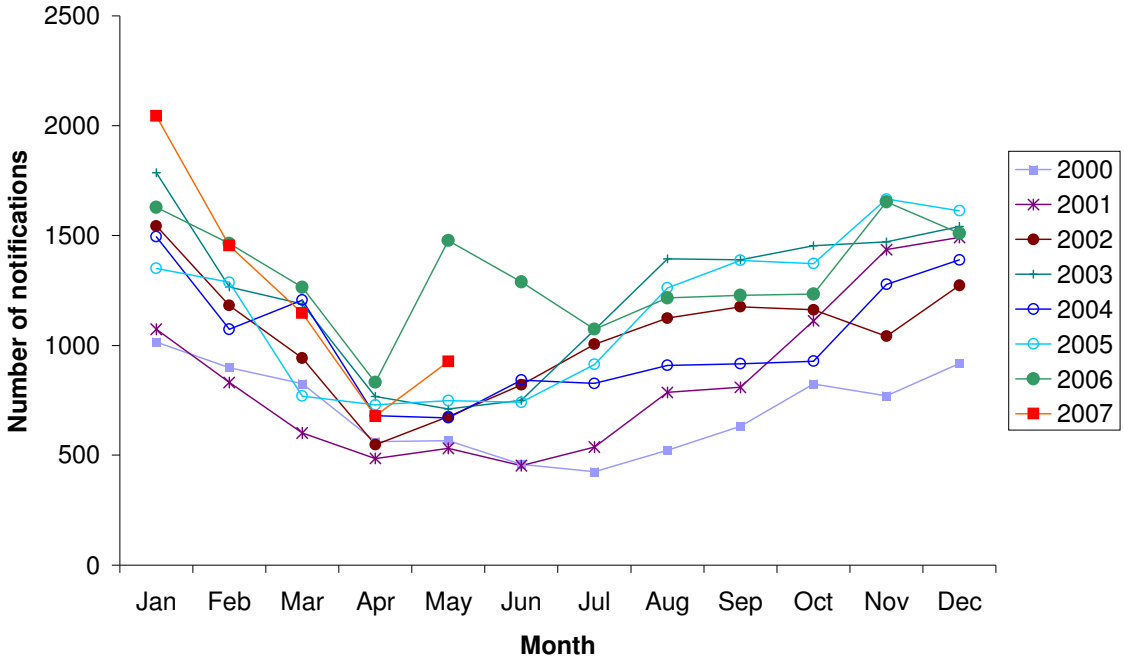
A piece of good news from all of this is that at least PIANZ is taking some ownership of this problem and agree that "…poultry is one of the main causes of human Campylobacter infection…"[1] However, if they want to gain the trust of New Zealand consumers and health professionals they need to adopt a very honest approach to how they present data about the epidemic.

**Table 1. Campylobacteriosis notifications by month, New Zealand, January 2000 to May 2007.**

| Month | 2000 | 2001 | 2002 | 2003 | 2004 | 2005 | 2006 | 2007 |
|---|---|---|---|---|---|---|---|---|
| Jan | 1016 | 1073 | 1544 | 1787 | 1495 | 1351 | 1629 | 2045 |
| Feb | 899 | 831 | 1182 | 1266 | 1073 | 1287 | 1464 | 1454 |
| Mar | 825 | 601 | 942 | 1189 | 1206 | 769 | 1265 | 1149 |
| Apr | 561 | 485 | 548 | 768 | 680 | 729 | 832 | 678 |
| May | 566 | 531 | 675 | 710 | 670 | 748 | 1478 | 926 |
| Jun | 458 | 453 | 820 | 750 | 842 | 741 | 1289 | |
| Jul | 424 | 537 | 1006 | 1070 | 827 | 914 | 1074 | |
| Aug | 523 | 786 | 1124 | 1394 | 909 | 1262 | 1216 | |
| Sep | 632 | 809 | 1176 | 1389 | 916 | 1387 | 1228 | |
| Oct | 824 | 1112 | 1162 | 1454 | 928 | 1372 | 1234 | |
| Nov | 771 | 1436 | 1042 | 1471 | 1278 | 1666 | 1654 | |
| Dec | 918 | 1492 | 1273 | 1542 | 1389 | 1613 | 1510 | |
| Jan-May total | 3867 | 3521 | 4891 | 5720 | 5124 | 4884 | 6668 | 6250 |

**Source:** Institute of Environmental Science and Research Limited. Monthly surveillance reports.

**Figure 2. Campylobacteriosis notifications by month, New Zealand, January 2000 to May 2007**




**Source:** Institute of Environmental Science and Research Limited. Monthly surveillance reports.

## What to do about the epidemic

The more certain good news is that there is now wide consensus on where New Zealand's *Campylobacter* epidemic is coming from. The evidence that fresh chicken is the dominant source is overwhelming[10] and well accepted by the New Zealand Food Safety Authority.[11]

Given that almost all chicken sold in New Zealand comes from just three large producers, this epidemic could reasonably be described as a 'common source outbreak'. However, the scale of this 'outbreak' dwarfs all of the reported outbreaks in this country combined. Over the 10-year period from 1997 to 2006, New Zealand had 116,426 notified campylobacteriosis cases. If we assume, conservatively, that 50% of them came from chicken, this gives a total burden of 58,213 notified cases from this one source. This is twice as many cases as came from all reported disease outbreaks combined for that 10-year period (29,162 cases from 3,301 outbreaks).

New Zealand's 'common source outbreak' of *Campylobacter* infection could be largely eliminated by decisive action taken to remove the source. As we have previously noted, available technology (such as freezing) is likely to be highly effective at controlling this source.[4, 12] We have not seen the New Zealand Food Safety Authority present a well reasoned argument against taking this kind of decisive action to quickly control this epidemic.

In the absence of effective Government action on New Zealand's *Campylobacter* infection epidemic, consumers may have to 'vote with their shopping trolleys' and switch from chicken to alternative protein foods. As we have shown, some of these foods are cheaper, safer and more nutritious than chicken.[13]

**Competing interests:** There was no external funding for this work. One of the authors (MB) has provided technical advice to the NZFSA and another (NW) has had two previous research contracts with the NZ Food Safety Authority (NZFSA) in 2005.

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# Regarding the 'Phytophotodermatitis caused by contact with a fig tree (*Ficus carica*)' case report

It is difficult to see how a phytophotodermatitis could be "pathopneumonic", given the inability of light to reach the lungs, and, indeed, there appears to be no other respiratory reference in the case report by Derraik and Rademaker on the perils of embracing fig branches.[1]

Could it be that the combination of an errant spell-check and inadequate proof-reading converted an intended "pathognomonic" to "pathopneumonic"?

Tony E J Fitchett
General Practitioner
Mornington Health Centre
Dunedin

## Reference:

1. Derraik GB, Rademaker M. Phytophotodermatitis caused by contact with a fig tree (*Ficus carica*). N Z Med J. 2007;120(1259). http://www.nzma.org.nz/journal/120-1259/2658

## Response

Indeed Dr Fitchett is quite correct. Although a number of medical articles incorrectly use 'pathopneumonic' or 'pathoneumonic' (as we have just done in the referred piece!), the proper spelling is indeed "pathognomonic", even though the etymology of the word is still unclear (as per the Oxford English Dictionary).

Our apologies for the mistake.

José G B Derraik
Senior Adviser (Human Health)
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# Regarding the 'Five-year experience of corneal scrapes at Wellington Eye Department, New Zealand' article

Bacterial cultures on 34 corneal scrapes at the Wellington Hospital were positive in 85% instances[1] while, in all probability, no fungi were encountered during Gram staining and culture.

The scenario with corneal scrapes in a private, tertiary care hospital in the Indian capital metropolis has been totally different—little bacterial growth but plenty of fungal infections.

Effective December 2002, corneal scrapes from outpatients with corneal ulceration are screened jointly by ophthalmology and clinical microbiology staff. A database is being maintained from January 2007. The majority of cases have had a history of trauma; use of several topical eye drops and therapeutic interventions elsewhere.

An ophthalmologist would scrape the corneal lesions with simultaneous smear-making and plating on the slit lamp itself. Scrapes are plated on blood agar, MacConkey agar, mannitol salt agar, and Sabauraud medium. The patient would have to wait in the hospital, on average for 1 hour, before the microbiology staff would communicate microscopy findings to the ophthalmologist and obtain a consequent prescription from the ophthalmologist.

During the interval January to August 2007, scrapes were drawn from 20 patients, 13 males, 7 females, age ranging 6–78 years, mean 47.7 years, SE ± 4.86 years. Gram/Leishman staining showed fungal hyphae in 15 cases, degenerated hyphae in 2 cases, intranuclear inclusions suggestive of herpes virus infection 9 cases, and pus cells in 7 cases. Microbial cultures feasible only in 17 cases were negative for bacteria.

On the contrary, fungi cultured included *Aspergillus* and *Candida* species in 4 and 1, respectively. The prescriptioin for fungal keratitis would be with hourly pimafuscin eye drops and 200 mgm fluconazole orally, while acyclovir eye ointment would be applied five times daily in cases of herpes keratitis. The clinical response continues to be an affirmative one, with few dropouts for the follow-up. The final outcome has been remarkable with very little opacity.

There has not been any notable isolation of bacteria from the local corneal scrapes.[2] The free over-the-counter sale of ophthalmic formulations of antibiotics including steroids has been a rule rather than exception in several countries. Patients would move ‘from pillar to post’ to select effective and rapidly acting medicines. Furthermore, like the 20 patients during 2007, self-mediation or inadequate doses for a short duration would hardly leave many cultivable bacteria. Indeed there were two cases with ghost fungal hyphae but an effective therapeutic response.

Conventionally, a specific diagnosis of herpes virus keratitis should have been attempted through viral culture and molecular investigations. Such tests are costly, time-consuming, and available at research or academic hospitals.

The slit lamp scrape and concurrent microscopy would be a reasonable alternative in hospitals lacking such facilities. Last but not least that would assist towards surveillance of microbes responsible for keratitis, including the local antimicrobial susceptibility profiles.[1]

**Acknowledgement:** The technical assistance of Ms Kamini Singh and Ms Nisha Mashi is acknowledged.

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# Geoffrey Buckland Orbell

*7 October 1908 – 15 August 2007 (ENT specialist, MBE, founder of the NZ Deerstalkers Association, rediscoverer of the takahe)*

Geoffrey Orbell, a doctor who was happiest in the outdoors where he found the "extinct" takahe in 1948, died in Dunedin Public Hospital recently just a few weeks short of his 99th birthday.

He was born at Pukeuri, Oamaru, and educated at Waitaki Boys' High School and Christ's College before attending Otago University.

He graduated MB ChB in 1934, then studied at Moorfields Eye Hospital, London, where he received a Diploma in Ophthalmic Medicine and Surgery.

He also worked at the Melbourne Eye and Ear Hospital before setting up as an ear, eye, nose and throat specialist in Invercargill. He maintained his practice for 46 years. He also found time to serve on Invercargill and Southland local authorities (city council, licensing trust, hospital, and school boards).

Orbell was a man of many talents—ophthalmologist, cabinet maker, boat and house builder, skilled shot (founder of the NZ Deerstalkers Association), tramper, fisherman, and local body politician. But above all he will be remembered for his rediscovery of the *takahe*, the unique and flightless blue-green, hen-like bird with the bright red bill in the depths of Fiordland.

The bird, last seen in 1898 and widely considered extinct, fascinated Orbell from school days. He read about possible sightings and reports of its calls and tracks.

"From hearsay and from stories told around camp fires...I picked up little bits of information...these and other stories added circumstantial evidence of the existence of *Notornis mantelli* (the bird's biological name)," he wrote later.

Orbell figured that if the bird did survive, it might be found in an area of the unexplored Murchison Mountains west of Lake Te Anau. And that's exactly where it was. Orbell and two companions heard strange bird calls and saw tracks on his first expedition into the area in April 1948.

They returned the following spring, climbed 4 hours to reach the open tops and eventually emerged above a large lake with a valley beyond. In an area of snow grass near the lake Orbell and his friends found several birds and photographed them at close quarters.

Their discovery caused a sensation in New Zealand and overseas. Although the birds had been rediscovered their hold on existence was precarious and it has taken years of effort by wildlife officers to increase their numbers.

Almost 300 birds survive today, in Fiordland and other sanctuaries to which they've been transferred.

Orbell made his last trip back to Takahe Valley just after he turned 90 in 1998—flown in by helicopter for a ceremony marking the 50th anniversary of his momentous find. The aircraft whirled in over the lake that's now officially Lake Orbell.

Orbell is survived by his wife Sheila, who is 96, one son and two daughters.

Read more about the takahe at http://www.mtbruce.org.nz/takahe_more.htm

This obituary has been slightly adapted from one written by the New Zealand Press Association. We thank its editor for allowing us to republish it.

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# Margaret Stuart Smith

*Anaesthetist, OBE; 13 April 1912 – 7 August 2007*

Christchurch's first fully trained anaesthetist honed her skills working with renowned New Zealand plastic surgeon Sir Harold Gillies on severely burned servicemen in World War 2.

Dr Margaret Smith returned to New Zealand after the war. She worked at Christchurch Hospital for 30 years and in private practice until she retired in 1982.

The first New Zealand woman to pass the Diploma in Anaesthetics examination, which she did at Guys Hospital, London, died in Christchurch. She was 95.

Born Margaret Riddell, she was born and raised in Wellington but made Christchurch her home after meeting local businessman Carl Smith, owner of Munns menswear shop. They married in 1948 and had three children.

Smith introduced major post-war initiatives in anaesthesia to Christchurch. She gained satisfaction from seeing its status elevated from a skill to "its rightful place as a science" and a recognised medical specialty. She also took pride in being a pioneer "working mum". Two of the couple's children became doctors.

Smith graduated from Otago University's medical school on the same day in 1936 as her brother, Claude. He became a doctor at Kaiapoi but died young.

Inspired by the lectures and demonstrations of Dr Marion Whyte at Otago, she chose to pursue a career in anaesthetics. Two years as a house surgeon at Wellington Hospital served to reinforce her desire and she applied to take the study course and examinations for the new Diploma in Anaesthetics at Guys Hospital.

To gain entry to the course and to sit the exam, Smith had to perform 1000 anaesthetic procedures, 500 of them for major operations, and have each one certified by a supervisor, at Wellington Hospital. Early in 1939 she travelled to London, with her mother as chaperone. After passing the examination, she worked as an anaesthetist at Leicester Royal Infirmary before becoming a specialist anaesthetist in emergency medical services at Bangour Hospital in Edinburgh, in 1941. She remained there until 1945, working with casualties of the war. Many were flyers who had suffered horrific burns.

Her next position, as anaesthetics registrar at London's Hospital for Sick Children, in 1946, confirmed Smith's intense interest in paediatric anaesthesia. She returned to New Zealand the following year and was a consultant specialist at Christchurch Hospital from 1947 to 1977. Her appointment followed a stage in which anaesthetists were largely self- trained and used a limited range of techniques and drugs. Her first decade in Christchurch coincided with dramatic developments worldwide, in anaesthesia, the implementation of which she led on the local scene.

Her work with children, particularly in association with correcting birth deformities of the mouth and palate, brought her prominence. The citation for the Australian and NZ College of Anaesthetists' medal, which she received in 2002, praised her "pioneering

contributions" to anaesthesia, especially in the paediatric area. She was elected a fellow of the college in 1992.

Smith was awarded the Queen's Silver Jubilee Medal in 1977 and the OBE in 1990. She took an active part in professional organisations, being president of the Medical Women's Association, the Canterbury branch of the Society of Anaesthetists and the NZ Society of Anaesthetists, and the Canterbury branch of the National Council of Women. She was a lector at Christ Church Cathedral and a member of the Social Development Council.

She is survived by husband Carl, sons Rodney and Tony, daughter Jill, and nine grandchildren.

This obituary entitled *Leading lady in anaesthetics* originally appeared in *The Press* newspaper (Christchurch) on August 18 and was written by Mike Crean. We are also grateful to Bruce Rennie of *The Press*.

 

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# Vincent John Murphy

Dr Murphy, a long-time Morrinsville GP, died on 17 July 2007 in Waikato Hospital's Intensive Care Unit following complications from a skull fracture caused when he fell in his kitchen on 8 July after returning home from attending a Sunday morning clinic. He was 55 years old.

Vince was an active member of his community, a medico-political leader, a great supporter of general practice, a champion of Māori health, a devoted family man, and a keen sportsman (especially rugby union).

Vince was born on 23 November 1951, the third of four children. His father, a returned service man, and his English war bride settled in Panmure. Vince attended Sacred Heart Boys College where he was head boy and captain of the first XV rugby team.

Vince loved all sports including tramping, but rugby was his passion.

He attended Otago University graduating MBCHB in 1975. He married classmate Anne Hodgson in their final year. After house surgeon years in Rotorua and Auckland they settled into general practice in Morrinsville, Waikato. Along with wife Anne they practised with 5 other doctors for 29 years at the Morrinsville Medical Centre.

As a Fellow of the RNZCGP Vince was involved with the Waikato Faculty Board for 8 years, including as Secretary in 1986. He also recognised the unique opportunity the 2001 *New Zealand Primary Health Care Strategy* offered general practice, and he quickly got involved with the Waikato Pinnacle PHO. He was on the board from 2001. He was Chair of both Pinnacle general practice organisations: the IPA and the MSO from 2003 until last year. He completed a Postgraduate Diploma in Strategic Leadership in 2005. General practice in the Waikato is much indebted to his tireless campaigning and his dedication to many late-night meetings.

Vince was very much the old-style rural town general practitioner. He was fully integrated into the town and practice. He ran the local tennis club for several seasons, giving up many years of Saturday mornings, offering coaching. In the winter time for years he would support the local rugby team, not only as a spectator but as unofficial team doctor. He served on the Board of Trustees for St John's College, Hamilton, for 3 years, one as Chairman. The community in turn were able to rally around Vince and his family after the tragic death of their 5-year-old daughter in a 1987 motor vehicle accident.

One of Vince's concerns was Māori health. A unique opportunity was offered him when he was invited to open a clinic on the Kaitimari Marae in 2002. He enjoyed the

opportunity of combining rongoa and general practice. He initially held weekly clinics on a voluntary basis. More recently he had directed his attention to children with disability and he and his wife Anne had provided respite care.

Vince was a hugely valued team member; he gave 100% whether as a tennis team member, committee member, or part of the practice team. He was known for his reliability, humour, wisdom, and vision.

Vince was not least known as a loving husband, father, and uncle. His deep Christian faith equipped him for all the tasks he involved himself with. He jokingly talked about the priestly role of the general practitioner, a role that so often GPs find themselves in.

Vince is survived by his wife, Anne, and three children: Nigel and his wife Katrina, Diana and her fiancé Jonathan, and Catherine aged 18.

Dr Fraser Hodgson, GP for 20 years in Te Awamutu and brother-in-law of Vince, wrote this obituary.

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# Erratum

Derraik JGB, Rademaker M. Phytophotodermatitis caused by contact with a fig tree (*Ficus carica*). N Z Med J. 2007;120(1259).
http://www.nzma.org.nz/journal/120-1259/2658

After being alerted by a reader, the authors advise that the word pathopneumonic (first line of Discussion) should have been **pathognomonic**, and apologise for the mistake.

Please refer to the above URL to view the corrected copy of the case report.