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This Issue in the Journal

How low can it go? Projecting ischaemic heart disease mortality in New Zealand to 2015
M Tobias, K Sexton, S Mann, N Sharpe

Ischaemic heart disease mortality has been falling steadily in New Zealand for over three decades, yet this disease still accounts for almost one-quarter of all deaths and large ethnic inequalities persist. Age, period, and cohort effects shaping ischaemic heart disease mortality were analysed and used to project rates and counts to 2015. Rates are projected to continue to fall, but more slowly than in the past. Counts (absolute numbers of deaths) are projected to decline even more slowly, because of demographic trends. For Māori, the absolute burden of ischaemic heart disease mortality is projected to increase.

Nutritional information about sodium: is it worth its salt?
A Gilbey, S Fifield

This study examined whether members of the general public in New Zealand were able to accurately identify the nutritional information about salt on the packaging of food products. We found that most consumers were unaware of the "optimal" amount of salt in the diet, and were unable to accurately interpret the amount of salt in a product from the nutritional information given on the label. More than half assumed that salt and sodium are interchangeable terms, leading to an underestimation of the amount of salt in the product. The findings suggest a need to review the content of nutritional information panels.

Secondhand tobacco smoke exposure in New Zealand bars: results prior to implementation of the bar smoking ban
J Fowles, A Christophersen, D Fernando, R Lea, A Woodward, S Dickson, M Hosking, R Berezowski

The study involved measuring salivary cotinine levels in non-smoker volunteers before and after a 3-hour visit to randomly selected bars from Auckland, Wellington, and Invercargill (prior to smokefree legislation enacted on 10 December 2004). In all bars, and in all volunteers, exposure to secondhand smoke was clearly established with mean cotinine increasing by about 8-fold after visiting the bars. Study results also indicated exposure to secondhand smoke, with regional and seasonal variation, prior to the introduction of smokefree legislation.
Early recognition and early access for acute coronary syndromes in New Zealand: key links in the chain of survival
H Tanner, P Larsen, N Lever, D Galletly

The median time 100 patients took to present to hospital following the onset of symptoms of acute coronary syndromes was 228 minutes (range 33–9633 minutes). We found that multiple factors influenced this time, including type of health professional contacted initially, level of initial anxiety, and age, but not the belief that the problem was cardiac in origin. The factors associated with increased delays in presentation to hospital need to be addressed if we are to reduce mortality from acute coronary syndromes.

New Zealand health professionals do not agree about what defines appropriate attendance at an emergency department
S Richardson, M Ardagh, P Hider

Emergency Departments (EDs) worldwide are facing increasing pressure from high numbers of presenting patients and are often described as ‘overcrowded’. Several possible causes for this overcrowding have been suggested, including the possibility that some people who use the ED do not really need to do so—i.e. that there are other possible, more ‘appropriate’ places to receive care. These patients are sometimes referred to as ‘inappropriate’, and it has been suggested that if this group of people used alternative services, some of the overcrowding in EDs would be reduced. This study looks at this idea in more detail, asking whether a group of health professionals who are involved in policy making, referral, treatment, or transport of patients to the Christchurch Hospital ED agree about whether this is a problem, and whether they agree with what ‘inappropriateness’ means in this context. The study found that while there was some agreement between the different groups, there was no clear consensus about the definition of ‘inappropriateness’ in relation to ED patients.

Cardiovascular risk factors and their associations with alcohol consumption: are there differences between Māori and non-Māori in Aotearoa (New Zealand)?
D Bramley, J Broad, R Jackson, P Reid, R Harris, S Ameratunga, J Connor

This study describes the relationship between alcohol consumption and a number of cardiovascular risk factors, and then tests whether these relationships are same for non-Māori as for Māori. For most of the cardiovascular risk factors examined, there are clear associations with alcohol consumption. These associations are similar for Māori and non-Māori except for blood pressure and cigarette smoking. These differences need to be explored further as they may impact on inequalities related to cardiovascular disease.
Estimated prevalence of cardiovascular disease and distribution of cardiovascular risk in New Zealanders: data for healthcare planners, funders, and providers
S Wells, J Broad, R Jackson

New Zealand cardiovascular (CVD) risk management guidelines advocate risk assessment targeted mainly according to a patient’s age, gender, and ethnicity and recommend drug management for people at high risk of a CVD event in the next 5 years (CVD risk greater than 15%). To support plans around service delivery, this paper provides the data needed on the size of the issue for New Zealand and for individual district health boards (DHBs). We estimate that 7 out of 10 New Zealanders over 35 years of age should have a baseline risk assessment; and of those risk-assessed people, 1 out of 5 would meet criteria for drug treatment.
Heart health has an adverse future forecast in New Zealand: an alarm call to action across the continuum

Norman Sharpe

Coronary heart disease death rates peaked for both men and women in all age groups in New Zealand in the late 1960s and have since fallen by about 60%.\(^1\) This steep decline appears to have resulted entirely from a steadily increasing period effect, with incidence reduction (primary and secondary prevention) and case fatality reduction (medical and surgical treatment) contributing equally.

Despite this favourable change, cardiovascular disease (coronary heart disease, other heart disease, and stroke) still accounts for 40% of all deaths in New Zealand\(^1\) and many of these deaths are premature and preventable. Furthermore, large and intolerable inequalities in cardiovascular risk, outcomes, and access to services still remain within our small country. These inequalities are socioeconomically and ethnically determined and are also, in part, geographical. Steep risk factor gradients are plainly evident, particularly in relation to obesity, diabetes, and smoking.

The most socioeconomically deprived men and women in New Zealand have a cardiovascular death rate two to three times that of the least deprived (NZ Deprivation Index 9 and 10 vs 1 and 2).\(^2\) Middle-aged Māori men have a coronary heart disease death rate almost four times greater than non-Māori, non-Pacific men; Pacific men are intermediate.\(^3\) Coronary revascularisation rates as a measure of cardiovascular service access (or “dis-service”) vary two to three-fold across District Health Boards, being lowest in Northland, the Midland region (Tairawhiti, Bay of Plenty, and Taranaki), and Mid-Central.\(^4\)

Now, in this issue of the Journal, the lead article outlines a new and disturbing vision of the heart health future which can be added to the mix.\(^5\) Future projections for coronary heart disease mortality are provided which are of serious concern. These projections mandate that we urgently refocus and intensify our efforts across the heart health continuum for the benefit of all New Zealanders and to counter inequalities.

For the first time ever, the possible emergence of a substantive and adverse cohort effect is identified from the 1951 birth cohort onward. Put simply, coronary heart disease death rates for young and middle-aged adults for these recent birth cohorts are higher than previously. For young and middle-aged Māori men, the rates which have previously been very high, remain high, and those for non-Māori men and women and also Māori women are increasing.

Combining projected rates (based on extrapolation of the recent cohort and period effects) with projected demographic trends to 2015, the mortality burden is projected to decrease for non-Māori (albeit more slowly than before)—but it is projected to actually increase for Māori. Thus present inequalities may potentially increase during the next decade. Effective preventive measures and clinical care may be working differentially to reinforce the inverse care law—those most in need are least likely to receive these benefits.
The cohort data are even more concerning when coupled with recently published retrospective hospital admission data from 1989 to 2002/03 which testify to the increasing burden now placed on acute cardiac care services. During this period, hospitalisations for acute myocardial infarction (heart attack) in New Zealand doubled. While this increase could be attributed, in part, to changes in diagnostic criteria and coding, hospitalisations for acute coronary syndromes (acute myocardial infarction and unstable angina) increased by two-thirds—from 15,000 in 1989 to 24,000 in 2002/03. Hospitalisations for acute coronary syndromes doubled for Māori and Pacific men and women. These increases in admissions were evident across all age groups but particularly among younger adults.

The emerging cohort effect as well as (to some extent) the increasing hospital admissions related to coronary heart disease can speculatively but plausibly be related to the increased prevalence of obesity and diabetes. An epidemic of obesity and diabetes is leading to a new wave of coronary heart and vascular disease in ethnically predisposed populations and relatively younger people in both developed and developing countries around the World. This epidemic has been described as resulting from “a collision between ancient genes and the environment—Coca-colonisation” and as “the tsunami of metabolic syndrome, diabetes, and cardiovascular disease.” This tsunami could indeed inundate the clinical service arena—the long-term human and financial costs are scarcely calculable.

To achieve quality standards and equity of heart health outcomes for all New Zealanders in the foreseeable future, more urgent, intensive, and balanced actions are required nationally and regionally across the heart health continuum. The New Zealand Health Strategy (2000) lists 13 priority objectives, 6 of which are directly related to diabetes and heart health. This Strategy has been guiding providers in producing strategic heart health and diabetes action plans. It is urgent that these plans are now more vigorously prioritised, adequately resourced, and implemented.

Actions for improvement and priority recommendations can be referenced to the continuum which spans public and population health and clinical care and which also encompasses a life-course approach:

- A high-level strategic “task force” approach (environmental and regulatory) to obesity and diabetes action. The forthcoming Health Select Committee on Obesity and Diabetes provides a timely opportunity for this to be considered.
- Continuation and intensification of tobacco control measures coupled with widespread availability of effective smoking cessation support to achieve the vision of a truly smokefree New Zealand.
- Implementation of the Healthy Eating, Healthy Action (Oranga Kai, Oranga Pumau) Plan which has already gained widespread support. This is a collaborative multi-sector and multi-agency task which will require ongoing commitment and adequate resources from the respective Ministries and providers.
- Implementation of the Cardiovascular Risk Assessment and Management Guideline (2003), the keystone between population health and clinical care, which should be a topmost priority for all providers. Systematic implementation through primary care can offer large benefits in a relatively short period of time. Appropriate resources with a particular focus on disadvantaged people and an
appropriate cultural fit are crucial if we are to see a reduction of current and future disparities.

- Acute coronary syndromes management. Modern management is potentially extremely effective, but current access to the investigative and intervention facilities in the five main centres in New Zealand is very uneven and often delayed or limited by transportation, staffing, and capacity issues. Coordinated forward planning nationally and regionally is urgently needed to ensure equitable management of patients to agreed national guideline standards.12,13 This requires a realistic review of regional service support and capability, workforce, and facilities, and provision of adequate national and equitable regional resources.

- Elective coronary revascularisation. Increasing demands for acute revascularisation have displaced more stable patients who have a high need, leaving these patients at risk and increasing overall long-term healthcare costs. Recent revision of national access and urgency priority scoring systems can now provide more reliable assessment of unmet needs, but waiting times will not change without an increase in funding of procedures. In some areas, unused private capacity has provided a temporary solution. Regional planning and solutions are required in tandem with improved management of acute services.

- Chronic disease management and cardiac rehabilitation. Guideline-based integrated care approaches to chronic disease management have led to improvements in the management of heart failure in some regions and should be extended. Cardiac rehabilitation guidelines likewise are being implemented and gradually extended to improve access and uptake of Phase 2 (post-discharge) programmes. A home-based approach to cardiac rehabilitation is also being developed and the role of Phase 3 long-term programmes currently evaluated. Service delivery models that meet the needs of Māori and Pacific people as well as low-income groups are urgently needed.

Cardiovascular disease, and coronary heart disease in particular, will continue to place increasing demands on clinical health and social support services in the decades ahead. Without a more concerted and balanced investment in both preventive and clinical care, present inequalities in heart disease risk, outcomes, and service delivery are likely to worsen. The task of turning back the oncoming new wave of heart disease will require leadership as well as sustained, coordinated, and effective action across a wide range of governmental and non-governmental agencies and providers. The need is urgent.

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References:


4. NMDS public hospital data. NZ Health Information Service; 2003.


Reducing salt intake: action beyond the label

Alex Chisholm, Jim Mann

Nutrition labelling, which applies to pre-packaged food (but not to foods purchased unpacked, or pre-prepared food from food outlets), is one example of a population approach aimed at helping consumers to make healthy choices based on the nutrient content of a food. The approach assumes that consumers recognise the nutrients, their units of measurement, and the relationship to health in the context of their eating pattern—a challenge when consumers have been reported not to understand the terms fat, calories, sugar, vitamins, and salt. Indeed, the relationship between sodium and salt appears to be one of the least understood concepts.⁰

The findings in the study by Gilbey and Fifield in this issue of the Journal (Nutritional information about sodium: is it worth its salt? URL: http://www.nzma.org.nz/journal/119-1232/1934) confirm the difficulty experienced by some people in interpreting such information regarding salt.

In Western countries, processed food contributes 60–85% of the non-discretionary salt intake. Thus, achieving meaningful reductions in consumption of salt requires consumers to make optimal food choices based on the salt/sodium content of pre-prepared and processed foods.

There is a dose response to salt reduction within the range of 3 to 12 grams (g)/day—the lower the salt intake, the lower the blood pressure; and reduction in salt intake for 4 or more weeks has a significant effect in individuals with normal as well as those with elevated blood pressure.² An even lower blood pressure can be achieved by following an appropriate dietary pattern together with reduction in sodium intake.

The randomised controlled feeding study, the Dietary Approaches to Stop Hypertension (DASH)-Sodium Trial, lowered blood pressure at all levels of sodium consumption. However the combination of low sodium (50 mmol sodium or 2.9 g salt per day) and the DASH diet was more effective than either alone in people with stage 1 hypertension or blood pressure in the high normal range.³

The prime features of the DASH diet were regular low fat dairy products, fruit and vegetables, legumes, fish, and nuts. One year after initiating the DASH sodium diet, participants were still eating more fruit and vegetables and had sustained reductions in blood pressure despite an increased sodium intake.⁴ Despite the clearly established blood pressure lowering benefits of the combination diet, there is additional evidence for the beneficial effect of sodium restriction per se.

A recent prospective study involving a random sample of the Finnish population and measuring 24 hour urinary sodium excretion (the only accurate means of establishing intake) reported that a high salt intake predicted morbidity and mortality from coronary heart disease independent of other risk factors including blood pressure.⁶

In New Zealand, the daily intake of salt (sodium) for adults is approximately 9 g (150 mmol),⁷ which is appreciably higher than the recommended intake of 2.3–5.9 g per day (40–100 mmol), and the New Zealand Ministry of Health has endorsed the
recommendations made by many national and international organisations that suggest a drop of 50 mmol per day in sodium intake in New Zealand could substantially reduce systolic blood pressure in the population.8

Cardiovascular disease and its associated comorbidities including hypertension are increasingly associated with disadvantaged groups in society5. Unfortunately as the price of foods decreases, the sodium content often increases markedly. Breads, margarines, extruded cereal, and some canned products (at the lower end of the price range) have been found to contain more sodium9 than more expensive products.

Bread provides 11% of the total energy and accounts for one-quarter of the sodium in the New Zealand diet.10 There is evidence that the salt level in bread may be reduced by up to 25% without any difference in taste being noticeable,11 suggesting a possible simple means of reducing salt intake. The same probably applies to breakfast foods.

Easily understood information, which permits informed choices, is important for consumers faced with an extensive range of products of the same type such as bread or breakfast cereals. There is clearly a need for understandable consumer friendly nutrient panel labelling that assists healthy food choice, but Gilbey and Fifield’s findings suggest that this may be some way off with regard to sodium (salt).

Given that at the point of purchase the information panel on the product label is usually the only nutrient information available, signposting such as Pick the Tick offers assistance to consumers and it has been reported that 59% of shoppers use the tick to assist them in making healthy food choices. The reduction of salt through reformulation of breads, breakfast cereals, margarine, and vegetable oil based spreads to conform to the requirements of the Pick the Tick programme removed 33 tonnes of salt from the New Zealand food supply over a 1-year period.12

While such a reduction spread over 4 million New Zealanders may have a limited impact on blood pressure13 the potential long-term benefit of such a programme may be considerable especially for some population groups (e.g. young males) who have a high intake of such foods. The Food Industry Accord, established as part of the implementation of Healthy Eating, Healthy Action, provides another opportunity for reduction in non-discretionary sodium intake.

Legislative measures such as removing goods and services tax (GST) from healthy fresh food, and appropriate packaged and pre-prepared food, are other possibilities. Decreasing the use of discretionary salt intake will be facilitated in the long-term by education of children in nutrition and food skills. The Healthy Schools initiative, cooking in schools, and school gardens, are a move in this direction. The re-emergence of mild iodine deficiency serves as a reminder that all salt used should be iodised.

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References:


How low can it go? Projecting ischaemic heart disease mortality in New Zealand to 2015

Martin Tobias, Kerry Sexton, Stewart Mann, Norman Sharpe

Abstract

Aims This study aims to identify how ischaemic heart disease (IHD) mortality rates in New Zealand have varied between successive cohorts and time periods. This information is then used to project IHD mortality rates and counts (burdens) out to year 2011–15.

Methods Age / period / cohort models were constructed (5-year periods and 5-year age groups, generating 10-year overlapping cohorts) using both frequentist and Bayesian methods. Data were available from 1956 for the total population and from 1981 for Māori. The projection period was 2001-5 to 2011-15. Uncertainty was quantified as the Bayesian 90% credible interval.

Results IHD mortality rates for all age by gender groups increased from 1956–60 to peak in 1966–70, then declined by more than 60% to current (1996–2000) levels. However, the decline has been much shallower for Māori.

This decline has resulted from increasingly favourable period effects since 1971-75 (less marked for Māori). However, no substantive cohort effects have been seen, at least from the 1891 to the 1951 cohort. Our model suggests that, for the first time, a substantive and unfavourable cohort effect may be emerging among recent birth cohorts.

Conclusions IHD mortality rates are projected to continue to fall from 2001-05 to 2011-15, albeit more slowly than in the past as the increasing (favourable) period effect is partly offset by an emerging (unfavourable) cohort effect. The result is a relatively small projected decline in absolute IHD mortality burden overall, but an actual increase among Māori.

Ischaemic heart disease (IHD, coronary heart disease) mortality has been falling steadily in New Zealand for at least the past three decades yet this disease still accounts for almost one-quarter of all deaths. Also, large ethnic inequalities continue to exist, with Māori IHD mortality rates at least twice those of non-Māori. Projections of future trends in IHD mortality (overall and by ethnicity) are therefore of continuing interest to national health policy advisors and funders, District Health Board planners, and health service providers.

The aim of this study is to identify how IHD mortality rates have varied in the past between successive birth cohorts and time periods, and to use this analysis of cohort and period effects to project these rates (and counts) into the future.

Methods Data sources—Unit record mortality data from 1956 to 2000 was made available by the New Zealand Health Information Service. To minimise diagnostic and coding bias over the study period, a broad
definition of IHD was used, corresponding to ICD-9 codes 410–414. These codes may still omit IHD deaths attributed to codes for ‘sudden death’, ‘heart failure’, ‘cardiovascular disease not otherwise specified’ and ‘(type 2) diabetes mellitus’. All deaths so coded over the study period were assessed using multiple cause of death coding, and (where possible) were also linked to hospital discharges (for mention of IHD). This process yielded relatively few additional IHD deaths, however (approximately an additional 10%).

Māori IHD deaths were adjusted for undercounting of Māori ethnicity on death certificates using the New Zealand Census – Mortality Study (NZCMS) adjustors. These adjustors were derived by linking census to mortality records for the 3 years following each census from 1981 onwards. So for the ethnic (Māori – non-Māori) analysis, the study was restricted to 1981–2000 (rather than 1956–2000).

Midyear population estimates for 1956–2000 and projections (series 4) for 2001–2015 were obtained from Statistics New Zealand. Māori populations were interpolated intercensally, with the 1996 census population being re-estimated from the 1991 and 2001 censal populations (as the ethnicity item varied in 1996 from earlier and later censuses).

Rates were age standardised for summarisation by the direct method, with the World Health Organization (WHO) World population as the standard.

Age/period/cohort (APC) modelling—For readers unfamiliar with APC modelling, the following brief explanation is provided. Mortality rates can be thought of as realisations of three dimensions of time: age (at death), period (calendar year of death) and cohort (year of birth). Age, period and cohort are proxies for the real drivers of IHD mortality (e.g. ‘age’ captures the cumulative process of atherosclerosis over the life course, ‘period’ captures developments in treatment and prevention, and ‘cohort’ captures risk exposures related to birth cohorts such as tobacco use or diet). Given a sufficiently long time series of IHD mortality data, regression models can be constructed to project rates based on the historical trends in age, period and cohort effects.

APC models were fit to the available data, using 5-year age groups and 5-year calendar periods, so defining 10-year overlapping birth cohorts, using the statistical package S Plus. However, the entire dataset could not be modelled because of poor fit. Instead, the data had to be truncated to the 35–74 age range to obtain good fits, omitting the substantial proportion of IHD deaths that occur in very old age. Both classical (frequentist) and Bayesian models were constructed. For the frequentist models, the assumption was made that the underlying risk of IHD mortality increases exponentially with age, allowing period-cohort models (rather than full APC models) with identifiable (i.e. unique) period and cohort effects to be fit. Projections were then obtained by linear regression of future period and cohort effects on the most recent three observed effects.

For the Bayesian models, the ‘random walk 2’ full APC models were fit. These models have unidentifiable effects but identifiable projections, and were used:

- To test the validity of the linearity assumption used in the frequentist projections; and
- To quantify uncertainty around the projected rates (presented here as the 90% credible interval, the Bayesian equivalent of the frequentist confidence interval).

Ex-post tests were carried out by fitting both sets of models to a reduced dataset that omitted the most recent observed period (1996–2000). Mortality for this period was then projected and the projections compared to the observed values.

Results

Descriptive

Period—For the total population, rates for almost all age by sex groups increased from 1956–61 to peak in 1966–70 and then declined steadily to 1996–2000 (Figure 1).

Rates are now well below those seen at the beginning of the study period, having fallen on average about 60% from the peak in 1966–70 (slightly more for females and younger age groups). This corresponds to an average annual percentage change of approximately –3.5% over the observation period.
Figure 1. Ischaemic heart disease (IHD) mortality rates by period (1956–2000) and age (35–54), total population

### Males

**Period**
- 1956–1960
- 1961–1965
- 1966–1970
- 1971–1975
- 1976–1980
- 1981–1985
- 1986–1990
- 1996–2000

**Age-specific rate per 100,000**
- 35-39
- 40-44
- 45-49
- 50-54

### Females

**Period**
- 1956–1960
- 1961–1965
- 1966–1970
- 1971–1975
- 1976–1980
- 1981–1985
- 1986–1990
- 1996–2000

**Age-specific rate per 100,000**
- 35-39
- 40-44
- 45-49
- 50-54

**Note:** Due to differences of scale, only rates for younger age groups are shown as these are of most interest (rates for older age groups are similar in pattern, though higher in level, to those for the 50–54 age group; these rates are available from the authors).

For Māori, age-specific rates have declined more slowly over the 1981–2000 period (both sexes). Only in the older age groups have substantial falls been seen, with rates in younger age groups declining relatively little over the observation period (data not shown).

**Cohort**—For the total population, each cohort experienced lower IHD mortality rates at corresponding ages than preceding cohorts (Figure 2).

For Māori, the pattern was essentially similar (for the included cohorts), although with less change in the age specific rates of successive cohorts (data not shown).
Figure 2. IHD mortality rates by cohort (1891–1961) and age (35–74), total population

Note: Log scale is used to allow all ages to be represented (focus is on parallelism of the age specific curves).

Modelling

Period effects—Period effects derived from the frequentist model are presented in Figure 3. The effects are expressed as relative risks ie an effect <1 is protective (lowers the IHD mortality rate) while an effect >1 is adverse (increases the IHD mortality rate).

For the total population, period effects become progressively less adverse from the 1971–75 period onwards and by the most recently observed period are protective and large This steady trend in the period effect over three or more decades provides support for our assumption of linear projection over the 2001–2015 period.

For Māori, there has also been an improving (ie downward) trend in period effects from 1981–85 onwards, although the trend is shallower than that for non-Māori, especially among males. The period effect reaches 1 in the most recent observed period (1996–2000) or shortly before this, and is thereafter projected to become protective, more so for females than males.
Figure 3. Period effects, 1961–2015 (projected), ages 35–74

Males:

Exp (period effect)

Females:

Exp (period effect)
Figure 4. Cohort effects, 1891 to 1976 (projected), ages 35–74

Males:

![Graph showing Exp (Cohort effect) for males across different periods from 1891 to 1976.]

Females:

![Graph showing Exp (Cohort effect) for females across different periods from 1891 to 1976.]

**Cohort effects**—Cohort effects derived from the frequentist model are presented in Figure 4. The effects are again expressed as relative risks ie an effect <1 is protective while an effect >1 is adverse.
For the total population (both sexes), the striking finding is the absence of any strong cohort effects (at least from the 1891 cohort onwards). Nevertheless, there are interesting albeit minor variations in cohort effects to be seen. From the 1891 cohort (females) or 1896 cohort (males), the cohort effects become increasingly adverse (greater than 1), although still small, up to the 1916 cohort, whereafter the trend reverses and the cohort effect becomes increasingly protective (although still small) up to the 1946 cohort.

The most recently observed cohorts (i.e. the 1951, 1956 and 1961 cohorts) reverse the trend once more, and become increasingly adverse—although only the 1956 and 1961 effects (males) or 1961 effect (females) actually exceed 1.

Although the recent trend is far less clear than that for the period effects (see Figure 3), linear projection over recent cohorts produces a substantive adverse cohort trend, at least for the next few cohorts (both sexes). Thus our projection indicates the possible emergence, for the first time ever, of a substantive (and adverse) cohort effect (as shown by the dotted line in Figure 4).

For Māori, the pattern is essentially similar, with no strong cohort effects being detected. Nevertheless, the projection is for an upward trend in cohort effects over the next fifteen years, although this is of much smaller magnitude for males than females.

**Projections**—Projections were done using both the frequentist model (which required the assumption that recent trends in period and cohort effects would continue linearly into the future) and the Bayesian model (which required no such assumption). In fact, both models gave almost identical projections across all age by sex by ethnicity groups, supporting the linearity assumption (data available from the authors). Further validation of both models was provided by the post test: using the reduced dataset, both models produced near identical projections for the 1996–2000 period, which agreed closely with the observed rates (data available from the authors). Only the Bayesian projections are reported here.

For the total population (both sexes), and for both ethnic groups (Māori and non-Māori), age-specific and age-standardised IHD mortality rates are projected to continue to decrease until 2015. However, the rate of decrease will progressively slow (Figure 5). This reflects the interaction of increasingly protective period effects with increasingly adverse cohort effects.

For both total and ethnic populations, age standardised within the 35–74 age range, IHD mortality rates are projected to continue their long-term downward trend for both sexes. It should be noted that the 90% credible interval is wide for Māori (reflecting small numbers), but does not encompass an actual increase in rates.

**IHD burden**—Projections of the IHD burden were done by applying the projected age-specific IHD mortality rates to the projected population (within the 35–74 age range); see Figure 6.

For the total population (both sexes), the count of IHD deaths (within the age range 35–74) is projected to continue to decrease, albeit slightly more slowly than in previous decades. The projected average annualised count in 2001–05 is 1447 (males) and 507 (females), decreasing to 1103 and 345 in 2011–15 respectively (reductions of 24% and 32% respectively).
Figure 5A. Age-standardised (35–74) IHD mortality rates and projections, 1956–2015, by sex, total population
Figure 5B. Age-standardised (35–74) IHD mortality rates and projections, 1981–2015, by sex, Maori and non-Maori population

**Males**

**Females**

![Graph showing age-standardised IHD mortality rates for males and females, differentiated by Maori and non-Maori populations, from 1981 to 2015.](image-url)
Figure 6A. Average annualised IHD mortality count, ages 35–74, by sex, total population, 1956–2015 (projected)

![Graph showing average annualised IHD mortality count, ages 35–74, by sex, total population, 1956–2015 (projected)]

Figure 6B. Average annualised IHD mortality count, ages 35 – 74, by sex, Maori population, 1981 – 2015 (projected)

![Graph showing average annualised IHD mortality count, ages 35 – 74, by sex, Maori population, 1981 – 2015 (projected)]
This lesser reduction in burden than risk of IHD mortality results because the declining risk is partially offset by increasing population size together with a small contribution from population ageing (within the 35–74 age range) as the large ‘baby boom’ cohorts reach late middle and early old age.

For Māori, the decline in IHD mortality risk is (relatively) smaller and the growth (and ageing) of the population is (relatively) greater. As a result, the number of Māori deaths (both sexes) is projected to actually increase over the next ten to fifteen years. By 2011–15, the average annualised count of IHD deaths among Māori is projected to reach approximately 560, a 15% increase from the 480 (average annualised) deaths estimated for 2000–05.

Discussion

Although the analysis had to be limited to the 35–74 age group, this study nevertheless provides new and valuable information. It confirms that the peak of the IHD epidemic occurred in New Zealand in the late 1960s, as was already known from earlier research.\(^ \text{8} \) Since then, rates have fallen substantially (by approximately 60%) at all ages, although much less steeply for Māori. However, our models project that the rate at which IHD mortality declines in the next decade will progressively slow among both sexes and both major ethnic groups. To avoid this outcome, improvements in the coverage, quality, and effectiveness of prevention and treatment interventions will be required over and above those anticipated by projecting the historical trend.

This projected slower decline in IHD mortality risks, coupled with a growing and ageing population, leads us to forecast that the burden of IHD mortality (i.e. counts as opposed to rates) will decrease by only 25–30% (approximately) over the next decade. Indeed, the burden (and corresponding need for preventive and therapeutic coronary care services) is projected to increase for Māori. This finding has major policy implications, not least the need to urgently improve access for Māori to and through coronary care, if worsening of inequality in heart health between Māori and non-Māori is to be avoided.

Our projections relate only to the burden of IHD mortality. Trajectories for non-fatal burdens (including need for acute coronary care and management of people with heart failure) may be very different. Furthermore, trends in the 35–74 age group may differ from those in the 75+ age group.

Our study reveals an interesting pattern with regard to cohort effects. The absence of any strong cohort effects from the 1891 to the 1951 cohorts contradicts the hypothesis advanced by Barker\(^ \text{9} \) (which states that the risk of IHD is largely predetermined in utero), at least at the population level. Under the fetal origins hypothesis, dramatic increases followed by decreases in cohort effects should have been detected—yet no strong cohort effects were found at all. This finding does not mean that the hypothesised relationship does not exist at the individual level, merely that it is unlikely to have had a substantive impact on the IHD epidemic at the population level.

Our model does, however, suggest the possible emergence of a rising cohort effect among those born since the early 1950s. If confirmed, this would provide an explanation for the projected slowing in the secular trend of IHD mortality over the
next 10 to 15 years. That is, recent cohorts are projected to experience higher underlying risks of IHD mortality than their preceding cohorts—so partially offsetting the benefits that would otherwise accrue to them from the projected continuing (protective) trend in period effects.

What might explain these projected trends in period and cohort effects? Continuing improvement in period effects is likely to reflect better coverage and quality of preventive and therapeutic interventions for IHD. Our model is unable to disaggregate the period effect into incidence reduction and case fatality reduction components. However, analysis of Auckland MONICA data suggests that approximately half may be attributable to downshifts in population risk factor distributions and half to more effective and accessible treatments (including secondary prevention and thrombolysis in particular).10

The emergence of a substantive adverse cohort effect is harder to explain. Firstly, it may simply be an artefact of our frequentist model, specifically the linear regression of future cohort effects. However, the Bayesian model, which requires no such assumption, gave almost identical projections. Secondly, it could reflect changing proportions of different ethnic groups in the population. This explanation is unlikely as the same pattern is seen in the ethnic specific analyses (although less convincingly so for Māori males). Thirdly, it could reflect the emergence of a ‘core’ of people who are less responsive to health promotion messages such as not smoking cigarettes—although this would be expected to produce stabilisation of cohort effects rather than actual reversal of the prior trend.

A more likely explanation relates to the emergence, since the 1970s, of the epidemic of obesity (and consequential type 2 diabetes) in New Zealand and indeed throughout the developed world.11 In fact, similar slowing in IHD mortality declines has been observed recently in some other developed countries, and a rising cohort effect has been detected in both Australia12 and Sweden.13 If this explanation is confirmed, our study will have provided the first signal of an impact of the obesity epidemic on IHD mortality rates and burdens in New Zealand.

Regardless of the explanation, our projections for the next decade have clear implications for policy. At the very least, these projections imply that there is no room for complacency in regard to the prevention and treatment of IHD—especially if we are concerned about reducing inequalities in health between Māori and non-Māori.

Note: This paper is published with the permission of the Deputy Director General of Health (Public Health). However, opinions are the authors’ own and do not necessarily reflect Ministry of Health policy advice.

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Acknowledgements: The authors gratefully acknowledge statistical assistance from Sue Paul and Craig Wright (Ministry of Health).

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References:


Nutritional information about sodium: is it worth its salt?

Andrew Gilbey, Sarah Fifield

Abstract

This study examined whether members of the general public were able to accurately interpret the nutritional information regarding salt on the packaging of food products. 226 participants answered brief questions about their salt intake awareness and were asked to estimate the salt content of a food product, using the nutritional information panel on the packaging as required. Results indicated that most participants did not know how to interpret the nutritional information, and that many underestimated the salt content of the product by confusing it with sodium content. This finding suggests that the sodium information on packaged foods in New Zealand is not easily understood by the target population, and is therefore of little use in its current form.

It is generally accepted that a high level of salt intake is harmful to humans and that reducing the salt content in processed food will have large benefits in cardiovascular health for the wider population. Consistent with this view, the UK Food Standards Authority recommends reducing salt intake from an average of 11 grams (g) a day (for men) and 8.1 g a day (for women) to 6 g a day for both genders.

There are several reasons why people may exceed the recommended level of salt intake: salt is one of the primary determinants of taste in food; people are not concerned about their salt intake with regard to their health; and some food products, such as takeaway meals, are not labelled with nutritional information.

A further reason, and the one explored in this paper, is that information provided by food manufacturers about the salt content of their products is simply misunderstood by the target population. Such misunderstanding might reasonably be expected as researchers tend to recommend maximum levels of salt (NaCl) intake, whereas the nutritional information on most food packaging tends to be indicated by sodium (Na) content.

More than 25 years ago, it was found that approximately one-fifth of participants on limited sodium diets understood that sodium and salt are not directly interchangeable terms. If only one-fifth of those on a limited sodium diet understand the difference between salt and sodium, there is little reason to expect people not explicitly on a limited sodium diet will perform any better. Indeed, unless consumers are aware that 1000 mg of sodium is equivalent to approximately 2500 mg of salt—that is, that salt and sodium are not directly equivalent terms—then they would underestimate the amount of salt in any given food product by a factor of approximately 2.5.
Method

Participants were surveyed in multiple locations of a small city in the North Island of New Zealand, and asked:

- Whether they tried to monitor their salt intake;
- What they believed the recommended maximum level of salt intake to be; and
- How much salt would be consumed in one serving of Wattie’s Baked Beans (participants were handed this product and shown the panel containing nutritional information).

Wattie’s is a premium food brand identifiable by most New Zealanders. This product was chosen because it is readily identifiable and the sodium content was easy to convert to salt content (should a participant understand that this was required to state the salt content per serving). The nutritional information on this food product states it contains 1000 milligrams (mg) of sodium per 210 g serving. Thus, in a 210 g serving, there will be approximately 2500 mg of salt. Apart from the name, the packaging is virtually identical to the equivalent Heinz brand sold in the United Kingdom (although interestingly the sodium content is higher in Wattie’s Baked Beans).

Results

There were 226 participants (139 female; 87 male) comprising those aged less than 20 years (n=7); 21–30 years (n=47); 31–40 years (n=52); 41–50 years (n=54); 51–60 years (n=38); and more than 61 years (n=28).

Sixty-seven percent of participants (104 female, 48 male) claimed they took care over how much salt they consumed. Only 46 (20.4%) participants believed they knew the recommended daily consumption of salt, however, only 23 (10.2%) correctly answered that it was 6 g (the range of actual responses was 0.02–20 g per day).

159 participants (70.4%) believed they knew how much salt was contained in one serving of the food, of whom 132 (83%) stated there was exactly 1000 mg per serving. The actual range of estimates was 475 mg to 2500 g per serving. Four participants stated that the actual amount of salt in each serving was approximately 2500 mg and that they understood sodium and salt were not directly interchangeable terms.

Discussion

The findings indicate that the majority (67%) of the sample reported they cared about the amount of salt in their diet, although only 10% of the total sample was aware of the recommended daily maximum consumption of salt.

When asked to state the amount of salt in one serving, 132 participants answered using the exact figure stated for sodium content as being directly indicative of salt content. In other words, more than 58% of the sample appeared to believe that salt and sodium are interchangeable terms, and just over 98% were unable to identify the amount of salt present in the product.

Even if people are aware of the recommended maximum level of salt consumption, attempts to limit salt consumption using nutritional information on packaged foods would result in the underestimation (by a factor of 2.5) of the amount of salt consumed. In contrast to earlier findings, less than 2% of the surveyed participants appeared to understand that salt and sodium are not interchangeable terms (i.e. sodium content has to be multiplied by 2.5 to give the approximate pro rata salt content).
These findings suggest that research regarding the recommended level of salt intake is not disseminated in a way that is easily understood by the target population. To help increase compliance with recommendations about salt intake, the authors strongly support Sharp’s (2004) recommendation that “food labelling should include amount of salt (not sodium) per serving and per 100 g and at least a mention of the consensus maximum of, say, 6 g daily for adults” (p2080).4

As this study was conducted in New Zealand it is suggested that replications are carried out in countries where salt consumption is above the recommended maximum and nutritional information about salt content of food is reported in terms of sodium. Where applicable, the results of further research should be disseminated to relevant health agencies and food manufacturers, along with recommendations as to how such information could be conveyed more effectively.

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Secondhand tobacco smoke exposure in New Zealand bars: results prior to implementation of the bar smoking ban

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Abstract

Aims To measure secondhand smoke (SHS) levels in New Zealand bars prior to smokefree legislation enacted on 10 December 2004.

Methods Thirty bars were randomly selected from urban, suburban, and surrounding rural areas of Auckland, Wellington, and Invercargill. Bars were visited (on a Friday or Saturday night for a 3-hour stay between 1800 and 2400 hours) in July/August/September 2004 (winter) and again in October/November 2004 (spring). Each bar was visited by a group of 4 or 5 non-smokers participating in the study. All groups of participants spent a 3-hour block of continuous time in the bar. Saliva samples (approximately 0.5–2 mL) were provided immediately prior to entering the bar as well as 5–15 minutes after leaving the bar. Each group recorded the initial impression of air quality and ventilation, the number of observed lit cigarettes over three 10-minute intervals throughout the evening, and the number of patrons at each interval. In addition, any general comments about the venue (relevant to bar attendance or air quality on the evening) was recorded. Cotinine, the main metabolite of nicotine, was measured in saliva samples using Liquid Chromatography with tandem Mass Spectrometry (LC-MS-MS).

Results In all bars, and in all volunteers, exposure to SHS was evident. Saliva cotinine increased after 3 hours in the bar (mean increase=0.66 ng/mL, SE=0.03 ng/mL, p value of <0.0001). The 30 bars randomly selected provided a good spectrum of SHS exposures, with mean cotinine increasing by approximately 8-fold. Smaller population centres showed greater exposures to SHS. A north-south gradient of exposure was also seen (highest exposures were in Southland). Higher exposures were seen in the winter than in the spring. The objective measures of SHS exposure correlated strongly with the volunteers' subjective observation of ventilation, air quality, and counts of lit cigarettes. One exception was where objective salivary markers indicated that even “seemingly smokefree” venues with “good ventilation” produced discernable levels of SHS exposure.

Conclusions We have utilised an objective, non-invasive scientific approach to assess SHS smoke exposure in patrons of New Zealand bars. Our results clearly indicate exposure to SHS, with regional and seasonal variation, prior to the introduction of smokefree legislation.

The purpose of this study was to provide a quantifiable measurement of SHS exposure levels in New Zealand bars, before and after the smokefree law required bars to be totally smokefree in indoor areas. From 10 December 2004, the Smokefree Environments Amendment Act 2003 requires all workplaces, including restaurants
and bars, to be smokefree. This followed the examples of Ireland, Norway, and several US States who have enacted similar changes in recent years.

The smokefree law aims to protect workers and nonsmoking patrons from the significant health risks of exposure to SHS. Exposures to SHS in New Zealand contribute to approximately 400 premature deaths each year\(^1\) almost the same as the annual road toll. Until 10 December 2004, bars represented one of the most common public places in New Zealand for SHS exposures. This is the first national study of SHS exposure in bars in New Zealand and builds on a pilot study completed in Wellington in 2003 by Woodward and colleagues.\(^2\)

**Methods**

This paper reports on two baseline measurements of SHS levels in 30 randomly selected bars during winter and spring 2004, prior to the smokefree law change. A final report will compare the results with two proposed follow-up measurements in the same venues during winter and spring 2005, after the smokefree law change.

Ethical approval was sought from the three centres where this research was conducted. Conditional approval from all three sites, was granted by the Wellington Ethics Committee under reference No. 04/07/045. All volunteers were given information sheets describing the risks of exposure to SHS, and were required to sign consent forms.

**Selection of study regions**—SHS exposures were measured in Auckland, Wellington, and Invercargill bars. Auckland and Wellington include about 42% of the New Zealand population. Areas within those cities that have higher than average populations of Maori and Pacific Island residents were sampled to ensure sufficient representation from these ethnic groups. Invercargill was chosen to provide representation of a smaller population centre. Rural bars were also chosen from the three regions. The selected bars do not necessarily reflect all bars with in the study areas.

**Selection of bar venues**—Bars were selected randomly from the three regions using lists of licensed premises obtained from the local Public Health Units. The random selection was stratified to include one or more ‘rural’ bars from each area. All venues were visited by one of the investigators prior to their inclusion. To be included, venues had to be classified as clearly representing bars/taverns, rather than venues that were primarily restaurants with (for example) a side waiting area serving alcoholic beverages.

In addition, venues that had only recently opened (less than a year old), or had changed management in the past year were excluded from the study for consistency. Venues that had attendance that was primarily driven by the popularity of particular nightly events (such as music bands) were excluded. Finally, venues had to be deemed to be safe and acceptable places to send groups of volunteers.

**Selection of study participants**—Volunteer non-smokers living or working in a nonsmoking environment were recruited from within Institute of Environmental Science & Research Ltd (ESR) Wellington and Auckland offices, and from local Public Health Units in Auckland and Wellington. In Invercargill, participants from a local teacher’s college were recruited through help from Public Health South (the local Public Health Unit). The age range of all participants was 24–45 years old. Each group consisted of five individuals (three females and two males). Each group of volunteers visited five different venues on Friday or Saturday nights over an 8-week period; visits were spaced 1–2 weeks apart to ensure levels of salivary cotinine returned to baseline levels. Occasional substitutes in each group occurred due to various commitments for some participants.

**Study design**—Fifteen bars were chosen from Auckland, 10 from Wellington, and 5 from Invercargill. A power calculation was based on a pilot study conducted in Wellington.\(^2\) The results of this calculation were that a sample size of 5 individuals per venue would suffice to detect differences between pre- and post-cotinine levels (cotinine is the main metabolite of nicotine). In addition, the power calculation indicated that 30 venues would suffice to detect significant variations between pre- and post-legislation.

Bars were visited (on a Friday or Saturday night for a 3-hour stay between 1800 and 2400 hours) in July/August/September 2004 (winter) and again in October/November 2004 (spring). The average starting time in the study was 1919 hours.
During the visit, participants recorded the time of entry, the number of patrons present, and lit cigarettes in three 10-minute intervals evenly spaced throughout the 3-hour visit. Volunteers attempted to neither seek out, nor avoid, particularly smoky parts of the bar, but rather to try and capture a representative setting. All five volunteers stayed together throughout each 3-hour bar visit.

Saliva (0.5–2.0 mL) was provided into a Salivette plastic tube just prior to entering the venue, and 5–15 minutes after exiting the venue. Samples with pre-cotinine levels exceeding 4 ng/mL were excluded (4 out of 600 samples). The tubes were stored in a cooler until they could be placed in a –20°C freezer for longer-term storage. Studies in our laboratory show that cotinine in saliva is very stable, losing less than 10% after 1 week at room temperature (data not shown).

Two follow-up cycles are scheduled to be completed at the same times in 2005. The second half of the study will take place on the month, the night of the week, and, as much as possible, the time of day that the venue was sampled in 2004.

**Analytical methods**—Non-smokers carried out all extractions and extreme care was taken to ensure that contamination was eliminated. Each saliva sample (0.5 mL) was spiked with 50 µL of cotinine D3 internal standard solution, allowed to equilibrate for 5 minutes, basified, and then extracted with 3 mL ethyl acetate. The ethyl acetate was transferred to culture tubes, glacial acetic acid (30 µL) was added, and then the ethyl acetate was evaporated just to dryness in Savant evaporator. The dry residue was reconstituted in 100 µL 10:90 acetonitrile and deionised water.

Analyses were conducted using a Shimadzu 10AVP HPLC system attached to an Applied Biosystems API 300 Triple Quadrupole mass spectrometer equipped with a TurboIonSpray ion+ source. The HPLC column used was a Phenomenex Synergi™ 4 µm Polar, 75×2.0 mm ID. The mobile phase was a gradient of acetonitrile and 5-millimolar ammonium acetate. A nine-point standard curve over a concentration range of 0 to 40 ng/mL was created by spiking 0.5 ml of Barnstead H₂O with cotinine standards. The intraday reproducibility (five replicates) of the standard (0.3 ng/mL) had a CV of 9.4 %. The interday CV (3 days) was 14.5 %. The detection limit was at least as good as 0.1 ng/mL of cotinine in 0.5 mL of saliva.

**Statistical methods**—Initially, descriptive analyses were performed focusing on the increase in cotinine levels following bar visits. To investigate regional and seasonal variations for increase in cotinine analyses, and to assess the association between the increase in cotinine with ventilation and air quality ratings, two-tailed t-tests (or two-tailed t-tests with Satterthwaite correction for unequal variances), Spearman correlation, and ANOVA statistical analyses were performed. Assumptions and data checking were carried out prior to performing these analyses. Statistical analyses were performed using the Statistical Analysis Software (SAS) System version 9.1. A p value of <0.05 was considered statistically significant.

**Results**

After spending 3 hours in selected bars, the overall cotinine levels of the study participants increased by 0.66 ng/mL (SE=0.03 ng/mL) from a baseline cotinine average of 0.21 ng/mL. This increase was significant (p value of <0.0001). The overall cotinine levels increased by 0.76 ng/mL in winter and 0.54 ng/mL in spring (from a baseline cotinine average of 0.25 ng/mL in winter and 0.17 ng/mL in spring). The difference in cotinine increases between the two seasons was significant (p value of 0.0008); winter being greater.

In the winter round of testing, increases in saliva cotinine were found to be significantly correlated with the observed total number of lit cigarettes across three 10-minute counting intervals by total number of patrons during the bar visit (correlation coefficient (r) = 0.42, p value of <0.0001). In spring, this association was much weaker (r=0.13, p value of 0.1733). The correlations between saliva cotinine increase and cigarettes count by number of patrons at individual 10-minute counting intervals were 0.27, 0.30, and 0.32 in winter (p value of 0.0015, 0.0014, and 0.0010, respectively) and -0.07, 0.15, and 0.26 in spring (p value of 0.4205, 0.1102, and 0.0050, respectively).
Regional variations—The pattern of mean saliva cotinine increases across the three regions and two seasons is shown in Figure 1. Invercargill had the highest mean saliva cotinine increase of 1.15 ng/mL (SE=0.13 ng/mL) and both Auckland and Wellington had a significantly lower saliva cotinine increase compared to Invercargill. The mean increase in Auckland was 0.50 ng/mL (SE=0.03 ng/mL) and in Wellington was 0.59 ng/mL (SE=0.04 ng/mL). The difference in cotinine increases between Auckland and Wellington bars was not statistically significant (p value of 0.08). Both seasons showed similar regional variations where Invercargill had the highest mean saliva cotinine increase (1.26 ng/mL in winter and 1.03 ng/mL in spring) followed by Wellington (0.75 ng/mL and 0.47 ng/mL) and Auckland (0.60 ng/mL and 0.37 ng/mL). The results correspond to a north-south gradient of exposure.

Cotinine increases and ventilation—Overall there was a statistically significant difference in cotinine increases with ventilation ratings (p value of 0.0007). Figure 2 illustrates the association between the cotinine increases and ventilation ratings for both seasons. Spearman correlation suggests that for winter there is a significant negative trend in cotinine across poor, medium, and good ventilation (p value of <0.0001). However, the negative trend during spring was not significant (p value of 0.6667).

During the winter round, increase in cotinine and ventilation from ‘poor’ to ‘medium’ to ‘good’ was observed with a significant negative correlation. Winter mean cotinine increase was 1.07 ng/mL (SE=0.14 ng/mL) for poor ventilation, 0.61 ng/mL (SE=0.06 ng/mL) for medium ventilation, and 0.36 ng/mL (SE=0.06 ng/mL) for good ventilation.
Figure 1. Mean saliva cotinine increases across the three regions and two seasons (bars represent standard errors)

Figure 2. Mean saliva cotinine increases versus perceived ventilation quality (bars represent standard errors and ‘n’ represents number of bar visits in a given category)

During spring mean cotinine increase was similar between poor and medium ventilation (0.60 ng/mL with SE=0.13 ng/mL and 0.64 ng/mL with SE=0.10 ng/mL, respectively). However, the observed cotinine increase was much lower for good...
ventilation (0.38 ng/mL with SE=0.41 ng/mL). Figure 2 shows that ‘poor’ ventilation resulted in considerably greater SHS exposures in the winter months, whereas ‘medium’ and ‘good’ ventilation venues did not exhibit a seasonal difference in SHS exposures.

**Cotinine increases and perceived air quality**—Study subjects filled in a questionnaire rating the perception of smokiness in the venue, on a scale of 1 to 4. These values were grouped: 1=seemingly smokefree, 2=mildly smoky, 3=moderately smoky, and 4=severely smoky (blue). Figure 3 shows that these groups’ subjective ratings of air quality predictably corresponded with the degree of increase in saliva cotinine. Overall there was a statistically significant difference in cotinine increases with air quality ratings (p value of 0.0017).

Figure 3. Mean saliva cotinine increases versus perceived air quality
(bars represent standard errors and ‘n’ represents number of bar visits in a given category)

Figure 3 illustrates a significant association between increase in cotinine was observed with the smokefree environment changing to severely smoky environment for both seasons. Spearman correlations suggest that the trends across seemingly smokefree, mildly smoky, moderately smoky, and severely smoky bars for both winter and spring seasons are positive and significant (p values of <0.0001).

The winter mean cotinine increase was 0.34 ng/mL (SE=0.04 ng/mL) for seemingly smokefree bars, 0.73 ng/mL (SE=0.13 ng/mL) for mildly smoky bars, 0.96 ng/mL (SE=0.15 ng/mL) for moderately smoky bars, and 1.11 ng/mL (SE=0.24 ng/mL) for severely smoky bars (blue).
The Spring mean cotinine increase was 0.29 ng/mL (SE=0.07 ng/mL) for seemingly smokefree bars, 0.52 ng/mL (SE=0.14 ng/mL) for mildly smoky bars, 0.55 ng/mL (SE=0.14 ng/mL) for moderately smoky bars, and 0.69 ng/mL (SE=0.19 ng/mL) for severely smoky bars (blue).

**Discussion**

The saliva cotinine analysis provides a quantitative and objective measurement that correlates with subjective measures of air quality in the study. However, it was evident that this test detected SHS exposures even when individuals perceived venues to be ‘seemingly smokefree,’ and to have “good” ventilation. Thus, cotinine in saliva, measured by this analytical method provides a highly sensitive identifier of SHS exposures.

This finding is significant as it shows that improved ventilation is unlikely to remove patron exposure to SHS in bars that allow smoking, even if nonsmoking bar patrons fail to notice their own exposures. Subjective ratings of ‘seemingly smokefree’ corresponded to about 30–40% of the SHS exposure in a bar that was ‘severely smoky’ (blue).

**Regional variation**—The study covered urban, suburban, and surrounding rural areas in three centres of New Zealand and found similar relationships between subjective measures of air quality and salivary cotinine at each site.

It is noteworthy that the less densely populated areas showed greater exposures to SHS. The 30 bars randomly selected in this study provided a good spectrum of SHS exposures, with mean cotinine increasing by approximately 8-fold, and also provided a corresponding range of subjective ratings of air quality and ventilation.

**Seasonal variation**—Higher exposures were seen in the winter than in the spring. This might be due to patron behaviour changing as the deadline for the ban approached, or to seasonal effect (whereby we would have expected higher exposures to SHS in winter than in spring—as more people are likely to smoke outside in warmer weather and bars might have more windows open thus allowing better air circulation).

**Underestimate of SHS exposure due to delay in peak cotinine**—The half-life of cotinine in various body fluids has been reported to range from 16–19 hours. On the other hand, nicotine has a half-life of roughly 2 hours depending on ethnicity and some physiological states such as pregnancy. This means that the patrons in the current study would have had peak salivary cotinine levels approximately 3 to 4 hours after leaving the bar venue—it was not practical to collect samples at this time, however, as many bar visits were late at night.

Therefore the increases in cotinine reported in this study underestimate the actual exposures, and primarily reflect the exposures from the first 60–90 minutes in the bar. The correlations between saliva cotinine increase and observed total number of lit cigarettes across 10-minute counting intervals by total number of patrons during 60–90 minutes in the bar, were 0.44 (p value of <0.0001) in winter and 0.01 (p value of 0.8838) in spring.

The increase in salivary cotinine averaged 0.66 ng/mL at 3 hours, and it is unlikely that this represents the peak salivary level. Jarvis and colleagues found that bar staff
exhibited salivary cotinine levels on average of 9.28 ng/mL, which corresponded to an 
estimated nicotine intake of 0.6 mg.\textsuperscript{5} The exposures in the current study would 
suggest that a single 3-hour visit to a pub by a non-smoker, results in an exposure that 
is substantially less than that experienced by bar staff. Given that the purpose of the 
Smoke-free Environments Amendment Act 2003 is to protect workers in particular 
from exposure to SHS, this study confirms that workers who spend even 3 hours in a 
smoky workplace face considerable levels of SHS exposure with possible associated 
adverse health effects.

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**Acknowledgements:** The New Zealand Ministry of Health funded this study. We also 
thank the study participants, and Clare Bear from the Ministry of Health, for their 
contributions.

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**References:**

1. Woodward A, Laugesen M. How many deaths are caused by second hand cigarette smoke? 
2. Woodward A, Fowles J, Dickson S, et al. Increase in saliva cotinine after three hours’ 
   fluids: Implications for non-invasive measurement of tobacco smoke exposure. Am J Pub 
4. Dempsey D, Jacob P, Benowitz N. Accelerated metabolism of nicotine and cotinine in 
Early recognition and early access for acute coronary syndromes in New Zealand: key links in the chain of survival

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Abstract

Aims We studied the behaviour of patients prior to admission to hospital with symptoms of acute coronary syndromes, and what barriers may exist to early recognition of these symptoms and to early activation of emergency medical services.

Methods Over a 7-week period, we interviewed 100 patients admitted to the Cardiac Care Unit in Wellington Hospital with suspected acute coronary syndromes.

Results Within 5 minutes of symptom onset, 46 of 100 patients believed they were having a heart attack. Sixty-two of the patients believed that they had a cardiac-related problem. Patients took a median time of 90 minutes (range 0–9600 minutes) to contact a health professional from the time of symptom onset; and a median time of 228 minutes (range 33-9633 minutes) to arrive at hospital. We observed significant differences in both these end-points according to which health professional was contacted first. Patients who presented directly to hospital arrived significantly faster (median 72 minutes) than those who first called an ambulance (180 minutes) or contacted a general practitioner (485 minutes) (p=0.001, Kruskal Wallis test).

Conclusions Considerable delays exist in the presentation of patients with symptoms of ACS to hospital. These delays are multifactorial, but the psychological intimidation of the 111 telephone system and delays incurred by inappropriate out-of-hospital management of patients with chest pain are probably significant. It is likely that these delays contribute to mortality from acute coronary syndromes.

The 1992 American Heart Association Guidelines described early access to emergency medical services (EMS), early basic cardiopulmonary resuscitation, early defibrillation, and early advanced cardiac life support as four independent links in the chain of survival required to optimise survival from out of hospital cardiac arrest. Since the publication of that guideline, the concept of the chain of survival has been widely promoted by resuscitation councils throughout the World. Following the publication of the 2000 Consensus on Science document, which reiterated the importance of the four links, a supplement in the Annals of Emergency Medicine discussed the 1999 Evidence Evaluation Conference which recommended the addition of up to three links in the chain of survival; “early prevention” and “early recognition” of cardiac symptomatology added prior to early activation of EMS and “rehabilitation” added at the end of the chain of survival.

There has been widespread recognition that the time taken to access EMS is a key predictor of mortality for patients suffering acute coronary syndromes (ACS), with delays as short as 30 minutes significantly increasing mortality rates at 1 year.
addition, it has been estimated that 50% of those who die from ACS do so within 1 hour of symptom onset.\(^8\)

It is therefore essential that those suffering ACS are in close proximity to a defibrillator as soon as possible and, in the event of an acute myocardial infarction, angioplasty or thrombolysis is commenced as soon as possible. The reduction in arrhythmic death by availability of defibrillation, and infarct size by early thrombolysis, are substantial benefits of early access to advanced care. With these thoughts in mind, the addition of the early recognition link in the chain of survival was suggested: “The early access link in the current chain of survival implies but does not specifically address early recognition. Adding early recognition to the chain of survival would place greater emphasis on the early signs of heart attack … which would provide victims with a greater chance of survival”\(^3\).

To strengthen the chain of survival in the community, it is necessary to understand how patients think and act when experiencing symptoms of ACS. This present study was conducted to identify what patients do prior to admission to hospital with symptoms of ACS, and what barriers they describe to early recognition and to early activation of the EMS.

**Methods**

**Study design**—The study was conducted over a 7-week period on all patients admitted to the Cardiac Care Unit in Wellington Hospital with suspected acute coronary syndromes. Patients transferred from other centres were excluded from the study. Patients were interviewed using a structured questionnaire.

**Definitions and criteria used for the analysis**—

- **Time of initial symptoms** was taken as the self reported time of onset of symptoms. For patients who reported symptoms that were initially intermittent and subsequently constant, the onset time was defined as the time symptoms changed from intermittent to constant.
- **Time to contact a health professional** was defined as the time from symptom onset to the time at which a health professional was first contacted, either by telephone or in person.
- **Arrival at hospital** was the time from symptom onset to the time of physical arrival at the emergency department.

Patients were asked to score pain and anxiety at the onset of symptoms using a scale from 1 to 10, where “1” represented no pain or anxiety and “10” represented the worst possible pain or anxiety.

In the presented data, patients were classified as having had a previous myocardial infarction (MI) if they stated that they believed they had suffered a previous heart attack and classified as having previous angina if they believed that they had a history of angina.

Symptoms were defined as *typical* if they included central chest pain or left chest pain, and *atypical* if they did not.\(^9\)

The criteria used for final diagnosis were:\(^9,10\)

- **STEMI** (ST elevation myocardial infarction): Positive troponin-T assay and evidence of ST segment elevation on the electrocardiogram (ECG).
- **NONSTEMI** (Non-ST elevation myocardial infarction). Positive troponin-T assay with no evidence of ST segment elevation on the ECG.
- **Unstable angina**: Negative troponin-T assay but the ECG showed evidence of ischaemic changes.
- **Non cardiac diagnosis**: Negative troponin-T assay and no evidence of ECG changes.

To maintain consistency in interpretation of ECG changes, all ECGs were reviewed by one cardiologist (NAL).

**Statistical analysis**—Relationships between continuous variables were examined using Spearman rank correlation test, between group comparisons were performed using Mann Whitney U test (2 groups).
and Kruskal Wallis test (3 or more groups). All tests were performed using Statview 5 (SAS, Cary, NC, USA).

**Results**

Over a 7-week period, 108 patients were admitted to the Cardiac Care Unit with an suspected acute coronary syndrome. Eight patients were excluded from the study, six because of language difficulties, one was discharged prior to being interviewed, and one died prior to interview. Patient characteristics are given in Table 1.

**Table 1. Demographic information of the patients in the study**

<table>
<thead>
<tr>
<th>Age [mean (range) in years]</th>
<th>65 (32–88)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (male/female)</td>
<td>69/31</td>
</tr>
<tr>
<td>Final diagnosis</td>
<td></td>
</tr>
<tr>
<td>STEMI</td>
<td>16</td>
</tr>
<tr>
<td>Non- STEMI</td>
<td>43</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>26</td>
</tr>
<tr>
<td>Non cardiac</td>
<td>15</td>
</tr>
<tr>
<td>Previous history</td>
<td></td>
</tr>
<tr>
<td>Acute MI</td>
<td>31</td>
</tr>
<tr>
<td>Angina</td>
<td>34</td>
</tr>
<tr>
<td>Hypertension</td>
<td>46</td>
</tr>
<tr>
<td>CABG</td>
<td>9</td>
</tr>
</tbody>
</table>

STEMI=ST elevation myocardial infarction; Non- STEMI=Non-ST elevation myocardial infarction; MI=Myocardial infarction; CABG=Coronary artery bypass grafting.

Symptomatology and “early recognition”—Initial symptoms experienced by patients are presented in Table 2. Symptoms were classified as “typical” in 76 patients and “atypical” in 24 patients, respectively.

When asked “where did you initially think your symptoms were coming from,” 62 subjects identified the heart. This response was not statistically related to whether symptoms were typical or atypical.

When asked “did you think within 5 minutes of symptom onset you were having a heart attack?,” 46 patients answered “yes”. Again, this was unrelated to whether symptoms were typical or atypical.

Of the 38 patients who did not believe that their symptoms were related to the heart:

- 14 believed symptoms were indigestion (most commonly because of previous episodes of “indigestion”);
- 9 believed symptoms were musculoskeletal (most commonly because of increased exercise or exertion prior to the onset of symptoms); and
- 8 reported that they did not know what was causing their symptoms.

When asked to describe symptoms they believed were associated with a heart attack, patient’s responses are given in Table 3. The most common misconception, in relation to the symptoms actually experienced, was the expectation that chest pain would be very severe. Although 56 patients described likely symptoms similar to those they
initially experienced, these patients were not more likely to have believed that they were having a heart attack within 5 minutes of symptom onset.

In an attempt to relieve initial symptoms, 33 patients took glyceryl trinitrate (GTN), 18 rested, 10 took an antacid, 7 exercised (4 of whom believed they had a cardiac-related problem), and 32 did nothing. Only 3 patients took aspirin.

Table 2. Initial patient symptoms

<table>
<thead>
<tr>
<th>Pain</th>
<th>91</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central chest pain only</td>
<td>31</td>
</tr>
<tr>
<td>Central chest pain with radiation</td>
<td>25</td>
</tr>
<tr>
<td>Left chest pain only</td>
<td>10</td>
</tr>
<tr>
<td>Left chest pain with radiation</td>
<td>9</td>
</tr>
<tr>
<td>Right chest pain only</td>
<td>3</td>
</tr>
<tr>
<td>Right chest pain with radiation</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>10</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>2</td>
</tr>
<tr>
<td>Tiredness</td>
<td>3</td>
</tr>
<tr>
<td>Other</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pain quality</th>
<th>16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tightness</td>
<td>14</td>
</tr>
<tr>
<td>Burning</td>
<td>15</td>
</tr>
<tr>
<td>Sharp</td>
<td>12</td>
</tr>
<tr>
<td>Pressure</td>
<td>11</td>
</tr>
<tr>
<td>Ache</td>
<td>7</td>
</tr>
<tr>
<td>Dull</td>
<td>7</td>
</tr>
<tr>
<td>Heaviness</td>
<td>9</td>
</tr>
<tr>
<td>Other</td>
<td>9</td>
</tr>
</tbody>
</table>

Table 3. Patients’ belief about the symptoms associated with heart attack

<table>
<thead>
<tr>
<th>Symptom</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain plus at least one other symptom</td>
<td>36</td>
</tr>
<tr>
<td>Chest pain only (note that 25 patients believed chest pain needed to be severe)</td>
<td>38</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>13</td>
</tr>
<tr>
<td>Did not know</td>
<td>8</td>
</tr>
<tr>
<td>Collapse</td>
<td>2</td>
</tr>
<tr>
<td>Paralysis</td>
<td>2</td>
</tr>
<tr>
<td>Arm pain</td>
<td>1</td>
</tr>
</tbody>
</table>

Contacting a health professional: “early activation of EMS”—We used time to contact a health professional, and time to arrive at hospital, as the two primary measures of adequacy of activation of the emergency medical services. This data is presented in Table 4. We observed significant differences in both time to contact a health professional (p=0.01, Kruskal Wallis test) and time to arrive at hospital (p=0.001, Kruskal Wallis Test) according to which health professional was contacted first. Those who telephoned a GP made contact with a health professional most rapidly; those who presented directly to hospital arrived at hospital most rapidly.
Table 4. Activation of emergency medical services (EMS)

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Time to contact health professional</th>
<th>Time to arrive at hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>100</td>
<td>90 minutes (0–9600)</td>
<td>228 minutes (33–9630)</td>
</tr>
<tr>
<td>Telephoned hospital</td>
<td>4</td>
<td>81 minutes (5–150)</td>
<td>170 minutes (90–248)</td>
</tr>
<tr>
<td>Present directly to hospital</td>
<td>8</td>
<td>72 minutes (5–2385)</td>
<td>72 minutes (5–2385)</td>
</tr>
<tr>
<td>Telephoned GP</td>
<td>14</td>
<td>15 minutes (0–1740)</td>
<td>305 minutes (34–2505)</td>
</tr>
<tr>
<td>Presented to GP</td>
<td>33</td>
<td>300 minutes (0–4140)</td>
<td>485 minutes (115–4340)</td>
</tr>
<tr>
<td>Telephoned ambulance</td>
<td></td>
<td>39 minutes</td>
<td>112 minutes (0–1072)</td>
</tr>
</tbody>
</table>

Median time taken in minutes (range) for patients to contact a health professional, or to arrive at hospital from the time of symptom onset. Values are given for all patients, and by mode of contacting a health professional. Note that 2 patients contacted a health professional through private consultations and arrived at hospital 155 and 9630 minutes after symptom onset.

Time to arrive at hospital correlated inversely with the degree of initial anxiety (r=-0.29, p=0.003, Spearman rank correlation) and correlated positively with age (r=0.26, p=0.009, Spearman rank correlation) but did not differ according to gender, belief that the problem was cardiac related, previous MI, or previous angina.

When asked why they chose to contact a health professional, 34 said it was because they were not getting better, 4 because they were getting worse, and 10 because they had had similar episodes previously. For 22 patients, someone else had made the decision to contact a health professional; in 14 of these patients, a member of the immediate family made contact.

Fifty-seven patients said that they hesitated before contacting the health professional of their choice. When asked why they hesitated, 17 said they did not want to bother the health services, 15 thought their condition was not serious enough, 15 thought their symptoms would go away, and 7 reported a dislike for hospitals.

When asked why they chose to contact a specific health professional, those who contacted the ambulance (39) cited past medical history (12), previous education (11) and seriousness of their condition (8).

Those who contacted the hospital (12) cited seriousness of their condition (6), and those who contacted a GP (47) did not believe their condition was serious (27) or thought that the ambulance and hospital system was too busy (6).

Four patients telephoned a hospital, and all were told to call an ambulance immediately. Fourteen patients phoned a GP initially, and 5 of these were told to call an ambulance. Seven patients were told to come to see the GP immediately, one was told to ‘wait an hour and call back if symptoms had not improved’, and one was told to ‘get a blood test the following morning, and then see the GP’.

Forty-two patients presented to a GP prior to admission to hospital; 31 were referred to hospital following an examination, a median time of 31 minutes (range 4–180 minutes) after initially being seen by the GP. Eleven patients were sent for blood tests (1 prior to seeing the GP, 10 following a GP visit) and were only referred to hospital following a positive troponin-T test.
The 10 patients sent for blood tests following a GP visit were advised to go to hospital a median time of 450 minutes (range 60-1480 minutes) after initially seeing the GP. Nine of the 11 patients sent for blood tests had typical ACS symptoms, 2 had a history of a previous MI, and 2 had previous angina. Two of these patients had STEMI events, and they arrived at hospital 376 and 1137 minutes after initially being seen by their GP.

When asked “what should you do when having a heart attack?”, 80 said ‘one should take an ambulance to hospital’. However, only 17 of the 46 patients who thought they were having a heart attack within 5 minutes of symptom-onset, took an ambulance to hospital.

Twenty-nine patients reported that the symptoms which lead to their hospital presentation were not preceded by any similar episodes, but 71 patients reported that they had experienced similar symptoms ranging from 2 days to 6 months prior to that which lead to hospital admission. These previous episodes appeared to resolve spontaneously and did not lead to the patient to present to a health professional.

**Discussion**

In the current study, we have found significant problems associated with two of the early links in the chain of survival, “early recognition” and “early access to EMS”. Patients are slow to recognise symptoms associated with ACS, and are reluctant to rapidly access emergency medical services.

We found that only 46 of 100 patients in the study believed that they were having a heart attack within 5 minutes of symptom-onset. When asked where they thought their symptoms were coming from, 62 replied from the heart. This belief did not result in any difference in subsequent behaviour.

Patients in the current study did not gain rapid access to the security of the hospital or emergency medical services. The median arrival time at hospital was 3 hours 41 minutes, with only 25% of patients arriving within 2 hours.

Previous studies in the UK, Australia, and USA have reported median times of 2 hours from symptom onset to hospital presentation. We found that patients who had higher levels of initial anxiety, and were younger, presented to hospital faster—but choosing to contact a GP resulted in marked delays in arrival to hospital. The delay caused by choosing to contact a GP was unrelated to the delay in initial contact since these patients made initial contact more rapidly than those who chose to activate the EMS.

Several previous studies have found that choosing to visit a doctor outside of hospital introduces significant delays in the time taken to present to hospital. Although there are many factors that could cause delay at a general practitioner’s office, these were outside the scope of the current study.

Several patients chose to see a GP initially because they were concerned that the hospital and ambulance services were overworked. Over half of the GPs contacted by telephone did not suggest that the patient with chest pain should call an ambulance and be transported to hospital immediately. The GP’s decision to send 11 of 41 patients for blood tests prior to referral to hospital suggests a failure to understand the risks of delay in treatment for patients with AMI.
Nine of these 11 patients had typical symptoms of an acute MI and (in two patients) a previous history of acute MI. The median delay to hospital admission, associated with the request for a blood test, was 9 hours 30 minutes. These results suggest that clear guidelines for EMS activation by GPs (and for situations in which the use of troponin-T assay for out-of-hospital identification of AMI in patients with chest pain may be inappropriate) are needed.

There are several limitations to our present study. We have relied upon recall of symptoms and times by patients and, in doing so, will have introduced an undetermined degree of unavoidable error. It is also possible that the information given to GPs by patients in the study was not the same as they subsequently reported to us, and GP management of some patients may have been significantly influenced by this.

By limiting the study to those patients who have been admitted to a Cardiac Care Unit, we have also introduced a selection bias in favour of patients most likely to have had genuine acute coronary events. If all individuals presenting to any medical service with ACS symptoms were studied, then it is probable that a higher proportion would have a final diagnosis of a non-cardiac condition. It is unclear whether patients with similar ACS symptoms (but who are not admitted to a CCU) act in any way differently from the patients studied here.

Results from this current study suggest that the problems of early access and early recognition are not related entirely to inadequate patient eduction; the majority knew that they should call an ambulance if having a heart attack, and most were able to describe heart attack symptoms. Despite this knowledge, however, patients were unwilling to admit that they could be having a heart attack, and even if they believed this, were unwilling to activate ambulance transport.

The psychological barriers that prevent patients from acknowledging that they could be having a heart attack (and are sufficiently unwell to warrant calling for an ambulance) may be considerable. Media publicity of overworked emergency departments, overstretched ambulance services, and the psychological image of the ambulance service as a “lights and sirens, Rescue 111” service are unlikely to help matters.

Our results suggest that although patients find a call to a general practitioner less threatening than a call to ambulance, the delays associated with contacting a GP are often considerable.

Our results suggest that overall it would substantially reduce the hospital admission delay if ambulatory patients with unrelieved chest pain were simply transported to hospital by a bystander or relative. However the safety of such a proposal could be questioned and it is unlikely that this strategy would find favour with any of the present health professional groups involved with transport and care of these patients.
Alternative strategies to overcome the barriers of recognition and access are therefore needed. These could include:

- General practitioners recognising that their primary role in the management of chest pain of possible cardiac origin is to refer the patient on to the emergency services.

- Encourage patients (who are unwilling to access the 111 system) to telephone a general practitioner or (if unavailable) the hospital emergency department. Both of these sources should then activate the emergency services if the symptomatology is appropriate.

- Promoting a national chest pain telephone line which may be far less threatening to a patient than a 111 call, but could still result in a rapid activation of emergency medical services where described symptoms are at all suggestive of ACS.

- Promoting an ambulance-based mobile assessment system whereby an appropriately trained health professional could assess a patient’s symptomatology and ECG in their home or workplace and then decide whether formal ambulance transport on-site thrombolysis is warranted.

- Laboratories could justifiably query out-of-hospital requests for acute troponin-T assays.

- Ongoing audit, by coronary care units, of the symptom onset to hospital arrival times.

In summary, considerable delays in the arrival of patients with acute MI to hospital are occurring. These delays are multifactorial, but the psychological intimidation of the 111 telephone system and delays incurred by inappropriate use of troponin-T as a diagnostic tool for out-of-hospital chest pain are probably significant.

It is likely that these delays contribute to arrhythmic mortality from AMI and infarct size through delays in defibrillation, angioplasty, and thrombolysis. Given that chain of survival in a community is only as strong as its weakest link, we may well need to look at ways of promoting early recognition and early access to emergency medical services as being every bit as important as early CPR and early defibrillation.

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**Acknowledgement:** This study was supported by the Wellington Surgical Research Trust.

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References:


New Zealand health professionals do not agree about what defines appropriate attendance at an emergency department

Sandra Richardson, Michael Ardagh, Philip Hider

Abstract:

Aims Emergency Departments (EDs) worldwide are facing a crisis from overcrowding—a common perception exists that inappropriate use of the ED is the major contributing factor. This study aims to examine the concept of ‘inappropriate’ ED attendances in relation to the Emergency Department at New Zealand’s Christchurch Hospital. It specifically seeks to determine whether there is a consensus opinion among healthcare providers regarding a definition of ‘inappropriate’.

Methods An exploratory survey of health professionals involved with the referral, assessment, transport, and treatment of ED patients in Christchurch was carried out. A range of health professionals, including ambulance personnel, general practitioners, emergency department physicians, emergency nurses, and hospital managers were approached. A series of questions relating to definition and response to ‘inappropriate’ patients was asked, with an additional open-ended question relating to the definition of ‘appropriateness’.

Results There are significant differences in the attitudes and perceptions of key health professionals involved in the referral, treatment, and admission of patients to the ED.

Conclusions While there are some areas of general agreement, there is no clear consensus between the professionals surveyed regarding the concept of ‘appropriateness.’ This has implications for any interventions aimed at addressing ED ‘overcrowding’ that assume the presence of a consensus understanding of this concept.

Over the previous two decades, concerns have been raised in a number of Western countries about increasing numbers of patients attending Emergency Departments (EDs), with particular attention given to the concept of ‘inappropriate’ ED use.1–7 It is perceived that some patient groups ‘inappropriately’ seek primary health care from the ED.8–11 Several writers have suggested limiting the number of patients presenting by tightening (or at least defining) the criteria by which ‘appropriate’ patient conditions are identified.11,14–20 The main purpose of these criteria is to permit the development of targeted interventions designed to change behaviour, and thus reduce the number of ‘inappropriate’ attendances.

The impetus to develop and disseminate an ED appropriateness survey arose following increased awareness of overcrowding within the ED of Christchurch Hospital. This ED is one of the busiest in Australasia, seeing an average of 65,000 patients per annum, and with an admission rate of approximately 48%.24 Initial planning to respond to overcrowding at the ED raised the question of whether there was a problem with ‘inappropriate’ attendees. It became apparent that to answer this question, a consensus definition of ‘appropriateness’ would be needed. From the limited available research conducted overseas there is little evidence to support the
belief that a consensus opinion amongst health professionals exists.21 Moreover, there is no research specific to this in New Zealand.

Several guidelines and tools for assessing the prevalence of ‘inappropriate attenders’ have been developed.14–16,18 Attempts to categorise ‘appropriate’ patients have typically been derived following a retrospective audit of patient charts.7 Cases are then assessed by medical ‘experts’, usually focusing on patients whose triage codes indicate a low degree of urgency. The ‘gold standard’ against which cases are measured is the opinion of a group of emergency medicine specialists, or general/primary care practitioners. However, these groups may not in fact present a clear or united perspective.

The aims of this project were to examine attitudes and perceptions amongst health professionals regarding the concept of ‘inappropriate attenders’ and to provide the basis for possible interventions to reduce ED overcrowding and plan further research related to patient flow and ED attendance.

Methods

Sample—A purposive sample of health professionals was sought, including ambulance staff, ED doctors (the survey was disseminated to all medical staff working in the area and included house surgeons, registrars, and consultants), ED nurses, general practitioners, and hospital managers. Inclusion criteria were that the participant was:

- A member of one of the designated professional groups;
- Involved in the referral, transport, assessment, or treatment of Christchurch ED patients; and
- Willing to consent to participate.

Potential ED and management participants were identified from staff lists, GPs were contacted through the largest local GP-contracting organisation, and ambulance staff were approached when delivering or uplifting patients from the ED. Consultation occurred with ED and hospital managers, GP liaison officers, and the ambulance authority. 210 survey forms were distributed; 120 were returned complete thus giving an overall response rate of 57%.

Survey—Following an initial series of demographic questions, respondents were asked to estimate the percentage of local ED patients who could be treated appropriately in other settings, and to define the concept of ‘an inappropriate attender’.

A three point scale (agree, unsure, disagree) was used to gauge response to a range of patient scenarios, indicating whether these represented an appropriate primary reason for seeking ED care. The third section posed a range of potential responses for dealing with identified ‘inappropriate patients’ and respondents were again asked to indicate whether they agreed, disagreed, or were uncertain about these potential responses. The survey tool was piloted among a group of ED nurses, GP liaison officers, and ED doctors.

Thematic analysis—A single free-text question was included in the survey, asking respondents to define the concept of an ‘inappropriate ED attender.’ Responses were categorised by professional group, and within each group subject to thematic analysis. This involved the identification of central themes or ‘codes’. The themes were determined deductively on the basis of pre-existing understanding of the issues (from existing literature) and validated by an inductive review of the material. Content analysis of the participant’s responses occurred where specific instances of narrative data relating to the themes were identified.22,23

Results

Responses to survey statements—Overall, 120 responses were received from the survey, 27 from 40 distributed to general practitioners (68% response rate), 35 from 70 ED nurses (54% response rate), 14 from 30 ED physicians (47% response rate), 12
from 20 management personnel (60% response rate), and 32 from 50 surveys
distributed to ambulance staff (64% response rate).

Respondents were evenly distributed between genders (47% female and 53% male)
however there was some variation in age as shown in Figure 1.

**Figure 1. ED Attendance Study’s respondent groups by age and occupation**

The first question asked respondents to indicate whether “some patients attending the
ED could be more appropriately treated elsewhere (e.g. GP or After Hours Service).”
All ED doctors and management agreed with this statement, as did most (97%)
ambulance staff, ED nurses (97%), and GPs (93%).

The second question asked, whether “patients have the right to choose care from the
ED, rather than elsewhere.” The majority of ED doctors, ED nurses and ambulance
staff agreed with this statement (64%, 60%, and 56% respectively), however
relatively few GPs and managers agreed (18% and 15%; see Figure 2).

Most respondents, regardless of professional group, agreed that it was appropriate to
present to the ED if the patient believed their condition to be ‘serious’ (see Figure 3).
Indeed, four of the five occupational groups (except ambulance staff) agreed that the
patient’s perception of urgency was an appropriate reason, and four of the five (all
except GPs) agreed that it was appropriate to present with an acute psychiatric
problem.

There was over 50% agreement within each of the groups that it was inappropriate for
a patient to attend ED because they did not have a regular GP, or needed to have
blood tests taken. All groups except ED nurses agreed that it was inappropriate to present to ED for a second opinion.

Figure 2. Agreement to survey question, ‘patients should have the right to choose care from the ED rather than elsewhere’

Over 50% of groups (except ambulance staff) felt it was inappropriate to present for care if the patient’s primary reason for attending was that they:

- Required X-rays;
- Could access the ED more easily than elsewhere; and
- Had a chronic psychiatric problem.

There were clear divisions regarding several other statements. These included whether it was appropriate to attend the ED for social issues. Over 50% of ambulance, GPs, and management respondents disagreed with this statement, over 50% of ED doctors were in agreement with it, and ED nurses were conflicted. Ambulance staff, ED doctors, and ED nurses agreed that subsequent admission to hospital was indicative of an appropriate presentation, but GPs and managers disagreed. Ambulance staff and ED nurses did not agree that arrival by ambulance signified an appropriate
presentation, while GPs, ED doctors, and managers felt it was an appropriate indicator (see Table 1).

Table 1: Percentage of agreement with given statement (is this an appropriate reason to present to ED?)

<table>
<thead>
<tr>
<th>Statements: The patient…</th>
<th>Ambulance n=32</th>
<th>ED drs n=14</th>
<th>ED nurses n=35</th>
<th>GPs n=27</th>
<th>Management n=12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Believes their condition to be urgent</td>
<td>44</td>
<td>86</td>
<td>69</td>
<td>65</td>
<td>64</td>
</tr>
<tr>
<td>Wished to be seen in ED</td>
<td>38</td>
<td>36</td>
<td>31</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Does not have a regular GP</td>
<td>0</td>
<td>0</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Is unable to see their usual GP</td>
<td>22</td>
<td>43</td>
<td>23</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Requires blood tests</td>
<td>22</td>
<td>14</td>
<td>9</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Requires X-rays</td>
<td>50</td>
<td>29</td>
<td>23</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>States they cannot afford to visit a GP or AHS</td>
<td>28</td>
<td>43</td>
<td>54</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Believes their condition to be serious</td>
<td>50</td>
<td>79</td>
<td>86</td>
<td>56</td>
<td>58</td>
</tr>
<tr>
<td>(or their family) have social issues they that they believe can best be dealt with in the ED</td>
<td>16</td>
<td>58</td>
<td>29</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Can access the ED more easily than elsewhere</td>
<td>50</td>
<td>0</td>
<td>14</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Is subsequently admitted to hospital</td>
<td>78</td>
<td>65</td>
<td>63</td>
<td>39</td>
<td>43</td>
</tr>
<tr>
<td>Has a psychiatric problem (chronic)</td>
<td>44</td>
<td>7</td>
<td>26</td>
<td>13</td>
<td>14</td>
</tr>
<tr>
<td>Has a psychiatric problem (acute)</td>
<td>66</td>
<td>79</td>
<td>63</td>
<td>43</td>
<td>72</td>
</tr>
<tr>
<td>Wishes (to have) a second opinion</td>
<td>19</td>
<td>7</td>
<td>40</td>
<td>17</td>
<td>0</td>
</tr>
<tr>
<td>Arrives by ambulance</td>
<td>44</td>
<td>57</td>
<td>26</td>
<td>69</td>
<td>50</td>
</tr>
</tbody>
</table>

AHS=After Hours Service; ED=Emergency Department; drs=doctors; GPs=general practitioners.

The third section of the survey presented a range of statements offering various potential responses to the issue of overcrowding. Respondents were again asked to indicate whether they were in agreement, unsure, or disagreed with the potential effectiveness and acceptability of each of the interventions. Within and between members of all the professional groups there was a higher level of agreement with statements in this section. More than 50% of the ambulance respondents agreed with all statements, ED doctors agreed with 10 of the 12 statements, and ED nurses agreed with 11 of the 12 statements. The management group agreed with 9 out of 12 and the GP group with 7 of the 12 statements, respectively.

There was general agreement that ‘increased public education about the role of the ED and greater public awareness of social supports would lessen the number of inappropriate ED presentations.’ There was also agreement that ambulance staff should make the decision whether to transport a patient to the ED, GP, or After Hours Surgery. The management group disagreed that faster access to outpatient services would reduce ED presentations. Ninety-six percent of GPs surveyed agreed that inappropriate patients should be referred away from the ED; 43% of ED doctors agreed; and over 50% agreement was reached by ED nurses, ambulance, and management staff.

When asked whether inappropriate patients should be charged a fee for service, ED doctors, ambulance and GPs agreed (ranging from 57-81% support), while ED nurses and management disagreed. GPs also disagreed with the suggestion that there would
be fewer ED presentations if GP fees were lower, (22% agreement), while the remaining groups agreed with this (from 57-71%).

**Table 2. Percentage of agreement with given statement (responses to overcrowding)**

<table>
<thead>
<tr>
<th>Statement</th>
<th>Ambulance n=32</th>
<th>ED drs n=14</th>
<th>ED nurses n=35</th>
<th>GPs n=27</th>
<th>Management n=12</th>
</tr>
</thead>
<tbody>
<tr>
<td>'Inappropriate' patients should be referred away from the ED</td>
<td>84</td>
<td>43</td>
<td>66</td>
<td>96</td>
<td>71</td>
</tr>
<tr>
<td>'Inappropriate' patients should be charged a fee for service</td>
<td>81</td>
<td>57</td>
<td>29</td>
<td>61</td>
<td>29</td>
</tr>
<tr>
<td>There would be fewer ED presentations if GP fees were lower</td>
<td>66</td>
<td>57</td>
<td>71</td>
<td>22</td>
<td>57</td>
</tr>
<tr>
<td>There would be fewer ED presentations if patients had faster access to OP services</td>
<td>65</td>
<td>79</td>
<td>89</td>
<td>65</td>
<td>43</td>
</tr>
<tr>
<td>Increased education would decrease inappropriate use</td>
<td>81</td>
<td>93</td>
<td>71</td>
<td>65</td>
<td>78</td>
</tr>
<tr>
<td>Access to GP services within the ED would decrease pt waiting times</td>
<td>75</td>
<td>57</td>
<td>69</td>
<td>39</td>
<td>71</td>
</tr>
<tr>
<td>Access to GP services within the ED would increase pt satisfaction</td>
<td>59</td>
<td>43</td>
<td>80</td>
<td>43</td>
<td>64</td>
</tr>
<tr>
<td>Access to a Nurse Practitioner would decrease ED waiting times</td>
<td>75</td>
<td>64</td>
<td>86</td>
<td>22</td>
<td>57</td>
</tr>
<tr>
<td>Access to a Nurse Practitioner would increase pt satisfaction</td>
<td>56</td>
<td>57</td>
<td>74</td>
<td>17</td>
<td>57</td>
</tr>
<tr>
<td>Greater awareness of support available in the community would decrease inappropriate ED presentations</td>
<td>84</td>
<td>71</td>
<td>77</td>
<td>61</td>
<td>86</td>
</tr>
<tr>
<td>Ambulance staff should have a choice of whether to deliver pt to the waiting room or resus(citation) area</td>
<td>81</td>
<td>57</td>
<td>71</td>
<td>65</td>
<td>29</td>
</tr>
<tr>
<td>Ambulance staff should have a choice of whether to transport a pt to the ED or an AHS</td>
<td>97</td>
<td>79</td>
<td>97</td>
<td>78</td>
<td>57</td>
</tr>
</tbody>
</table>

AHS=After Hours Service; ED=Emergency Department; drs=doctors; GPs=general practitioners; OP=orthopaedic; pt=patient;

Questions were asked regarding the potential impact of GP and Nurse Practitioner services offered within the ED environment. All groups except GPs agreed that access to GP services within the ED would decrease waiting times, but both GPs and ED doctors had only 43% agreement that this would increase patient satisfaction.

GPs also disagreed with the suggestion that a Nurse Practitioner service could either decrease waiting times in ED (22% agreement) or increase patient satisfaction (17% agreement). However, all other groups agreed with these two statements; ranging from 57–86% support (see Table 2).

**Free-text discussion (a thematic analysis)**—Respondents’ comments were grouped within professions, and then within each group, subjected to thematic analysis. The following broad themes were identified and used to categorise responses: service issues, generic condition description, specific condition identification, time
dependent, financial costing, personal characteristics, and comments which re-framed the question on the basis that no patient was ‘inappropriate’.

There was strong agreement that ‘inappropriateness’ was related to an inability or unwillingness by some patients to use sources of care other than the ED, in particular GP services.

One example of this is the definition of inappropriate ED attenders given by an ED nurse, all those whose care can be given at a facility other than the only Emergency Department in the city.

Many comments related to patients’ failure to seek initial GP assessment prior to presenting to ED. This can be seen in one GP’s description of inappropriate patients as those people who haven’t first attended their regular GP or 24hrs surgery. ED stands for Emergency Department! GP stands for General Practice!

The second theme identified was that of Generic Description, where the respondents described the concept of inappropriateness in terms of the general nature of the presenting complaint. This was typically coined in terms of acuity rather than by disease or injury, and included such concepts as ‘non urgent’ and ‘minor.’. Again, this theme was common to all groups with definitions given including a condition that is non-acute, non life-threatening (ED Nurse) and attending for minor or non-urgent medical problems (GP).

While there appears to be consensus that ‘non-acute’ conditions do not require ED care, there was little clarification of what made a condition acute or non-acute. An additional concern is whether members of the public are well enough informed to make this determination themselves. This was identified by one GP who noted that ‘inappropriate attenders’ might include those who conditions could be managed elsewhere but didn’t understand this at the time and a manager who suggested inappropriate attenders were those whose medical problems will definitely not lead to admission and not life and death situations, problem here is that as a patient, how would I know?

The role of the GPs as ‘gatekeeper’ to the hospital was emphasised in a number of responses. Examples include identification of an ‘inappropriate’ attender as one who makes no contact with own GP (ED Nurse), has not attended GP prior to presentation (Manager) and who presents without first seeking advice from primary care/GP/After Hours Service (GP).

One GP noted If they (patients) self refer they should be sent back to their GP or 24hr surgery unless it is an emergency. This begs the question, however, of who defines the ‘emergency’—the patient or the doctor? Comments made suggest that there is a range of opinion, with some clearly seeing this as a purely medical function, while others question whether the patient’s perception should play a part. This can be seen in the comment by an ED nurse, that: I believe patients by definition should be entitled to see their illness as acute or emergent and therefore attend the emergency service.’

A further theme was the attempt to specify particular injuries or illnesses, which might meet the criteria for ‘inappropriateness.’. Specific identification involved the listing or description of conditions or circumstances that were seen as inappropriate for ED care. These included flu-like symptoms/colds/upper respiratory tract infections.
URTIs), minor musculoskeletal problems, including sprains/strains/bruising, requests for prescriptions and patients presenting with problems that were primarily related to alcohol intoxication.

Two further themes were linked with specific descriptions; these were time-dependant criteria and financial matters. Some respondents attempted to clarify the concept of ‘minor’ or the listing of specific conditions by adding in a time-related dimension.

Examples of this view include:

- Problems more than 2 days without major deterioration, or those prepared to wait greater than 4 hours to be seen (ED Doctor);
- Patients who are in the area and ‘pop’ in with their minor problems (ED Nurse); and
- Patients presenting with problems or signs and symptoms that have been occurring over a long period ie 4–5 hours and could have been seen by GP (Ambulance Personnel).

Issues relating to finance were identified by participants from all groups except management as precipitating factors leading to ED presentations. Descriptions given included the phrases:

- Using the ED as GP service because of financial reasons and uses ED as a free service rather than paying for their GP (Ambulance Staff);
- Some lower socioeconomic people do not have the finances to see a GP especially after hours; availability of GP and finances is acceptable [as a reason for presenting to ED] but still inappropriate (ED Nurses); and
- People who could get safe effective treatment elsewhere but who attend due to cost reasons; and a person who attends for financial and other reasons… than for the perception that their need is an emergency (GPs).

A further theme associated with inappropriateness relates to the individual patient, and focuses on perceived characteristics and attitudes. These personal characteristics are generally portrayed in the negative, and include reference to non-medical reasons for presentation, as well as personality/behaviour traits. The most common groups identified here were simply referred to as regulars, social problems and those wanting a second opinion. The reference to regular attenders was particularly strong from the ambulance respondents, and there was an additional suggestion that many people were ‘using the system’ in an attempt to be seen earlier than their condition warranted.

Comments made included:

- Regulars or time-wasters;
- Repetitive attention seekers;
- Someone who believes that ED maybe an easy way of getting care/drugs;
- Patients who call an ambulance with the perception they will be seen quicker [given a] greater triage, and
• Hypochondriac-type individuals who do not require ED attention, but demand to be taken to hospital (Ambulance Staff).

The concept of ‘using the system’ appeared in other group responses. ED nurses comments included: patients who turn up to ED to get access to tests/investigations that are taking too long (i.e. Outpatient appointments/CT/MRIs etc), and patients who feel they are going to fast track the system or waiting lists by presenting in ED.

The concept of ‘social admissions’ was noted by respondents and often grouped together with reference to ‘inappropriate’ transfers from rest homes or private hospitals. Examples include patients who are experiencing ‘social’ difficulties (e.g. heating); patients from resthomes whose condition has deteriorated and staff have called ambulance rather than doctor to attend (Management).

ED nurses also saw social issues as significant, with the comments, social disposition ie no fixed abode, requires a warm bed for the night and food; people who live alone.

Finally, the underlying concept of ‘inappropriateness’ was questioned by a number of ED doctors and nurses.

ED physicians presented a number of statements challenging this concept, including:

• All ED presentations are appropriate. There are levels of appropriateness, however. An inappropriate attender may be someone who would choose treatment elsewhere given the means—educational—transport—financial—social; and

• Inappropriate is completely different to ‘more appropriately treated elsewhere’. Inappropriate attenders are those patients who seem to have nothing better to do with their lives than attend ED.

ED nurses also questioned the validity of the underlying concept, with one suggesting that:

• I would not define an attender as inappropriate. Some attenders may receive better long-term care by attending a regular medical service. Thus the regular medical service would provide more appropriate (continuity) of care; and

• The phrase [inappropriate attender] is loaded with value judgments. It seems to be a way of negatively defining the purpose of ED. Appropriateness may equally apply to the service we offer. We have, currently, ample evidence of the uncertainty that health professionals may have about clinical judgments (e.g. meningitis management). How much more uncertain may a lay person be when presented with uncertain signs and symptoms.

Discussion

The survey results present a range of professional perceptions, based on a purposive survey carried out at a major New Zealand ED. This survey was limited in only sampling the opinions of health professionals. Consumer opinions are the subject of further study. The main finding from this survey is the recognition that there is a range of opinions both within and across professional groups.

While there is some clear consensus on general principles, for example some ED patients could be treated appropriately elsewhere, there is little evidence of clear agreement on how these individuals should be defined. Given the increasingly team-
based nature of healthcare and the multiple entry points to the ED, lack of agreement on this basic concept may undermine any efforts to limit or re-direct ‘inappropriate’ patients.

While it was not possible to show consensus across the groups surveyed, there were indications of identifiable foci within professions. For example, ambulance staff were more likely to see patient admission as a clear indicator of appropriateness, whereas ED doctors and nurses were more likely to see patient perception of urgency or seriousness as a reliable indicator. This apparent support for the parameters of arrival method (ambulance) and outcome (admission) provides a starting point for planned further applications of the Delphi technique in a bid to determine whether consensus is a realistic outcome.

The findings from this study suggest that the existing reliance on a ‘gold standard’ that assumes consensus may be misplaced. Indeed, there appears to be little evidence to suggest that there is agreement regarding this concept within individual professions, much less between professions. Failure to acknowledge this fact thus limits the ability to accurately identify/forecast likely percentages of ‘inappropriateness’ amongst presenting patients. The assumption that such inappropriateness does exist and can be quantified may be based on a fundamental inaccuracy—that those involved are working from the same principles. If there is misconception, it follows that specific interventions that lead to the re-direction of patients away from the ED may themselves be flawed.

The underlying purpose of identifying ‘inappropriate’ patients seems to be to quantify a group of individuals whose care could be provided in an alternative venue or by an alternative service provider. There is no doubt that EDs are under pressure to refer patients with non-urgent conditions to other settings. What is debated, however, is whether the removal of this group would significantly impact on overcrowding. The first issue here is how such patients are quantified—is there a consensus agreement and means of determining the size of the presumed ‘problem’? This article has sought to raise awareness of the underlying assumptions associated with this process.

Further issues are apparent when considering the use to which such information (percentage of ‘inappropriate’ patients) is put. There is an assumption that these ‘inappropriate’ patients are necessarily ‘non-urgent’, and that therefore it is acceptable and appropriate to defer their care. Little specific follow-up has been carried out to determine the accuracy of this assumption—in terms of whether such patients do seek the alternative care pathway they are directed to, and what the ultimate outcome is. Washington et al carried out a randomised controlled trial with the aim of ‘evaluating the safety and acceptability of deferring emergency department care’ (p707). Their findings suggested that 36% of the screened patients (n=1176) met the explicit deferred care guidelines. Study participants in both groups (usual care and deferred care) showed improvement in health status, and no patient in either group was hospitalised or died. The authors did acknowledge a number of limitations, in particular lack of generalisability to centres where next-day care could not be guaranteed. In addition, several patients declined to participate in the study, so that the acceptability of this option can not be clearly determined. A potential factor if such a system were introduced into NZ EDs is the impact of additional cost—patients presenting to ED are not charged a fee for service, and although alternative care
venues are often available, the necessity to pay for care may be a limiting factor in the uptake of such services.

Vertesi (2004) also examined the possibility of deferring non-urgent patients away from the ED as a means of reducing overcrowding. This study identified that the greatest access problem and longest delays were associated with patients waiting for placement in the acute care cubicles. It was found that non-urgent patients take up only a small number of urgent ‘ED stretchers and acute care resources’ (p337), and as such their diversion is unlikely to improve access for more acute patients. Not only was little benefit seen to attach from a deferral system, but it was felt that this was ‘measurably unsafe’ and would lead to the inappropriate refusal of care to patients who required hospital treatment. This retrospective study revealed that of those patients who would have been identified as ‘non-urgent’ (using the Canadian ED triage and acuity scale), 7.3% still required admission.

Most of the literature around patient deferral from the ED-setting aims to identify the impact of this on the ED (in terms of waiting times, patient flow, and overcrowding). The Society for Academic Medicine (SAEM) takes a more explicit look at the ethical issues associated with policies that triage patients out of the ED prior to complete evaluation and treatment. It is suggested that decisions around the availability of emergency care and rationing of this service (whether implicit or explicit) should be a societal decision, a position supported by other authors. These authors suggest that the ethical and legal issues are influenced by whether the practice of deferring patients involves ‘triage away’ or ‘triage to’, and conclude that there is no system whereby a brief ‘triage’ exam can identify all patients with potentially serious emergencies.

It is apparent that the data (generated in attempts to quantify ‘inappropriate’ reasons for ED presentation) can be used in several ways, with significant implication for patient care. It is essential, therefore, that in developing a valid and reliable method of determining ‘appropriateness’ recognition be given to the range of assumptions and interpretations underlying this concept.

While the data gathered in this study may not be generalised to a wider group (it is neither a random sample nor statistically representative), it does appear indicative of significant variation of attitude and opinion, and calls into dispute the concept of a general consensus which would allow the formation of criteria defining ‘appropriateness’. Without a core definition, it is hard to say what contribution ‘inappropriate attenders’ make to the problem of ED overcrowding.

While this study has not given evidence to support a consensus definition of ‘appropriateness,’ it has highlighted the need for further research into this area. Further use of the Delphi technique is planned to develop a wider data base of health professionals’s views. This would allow for further analysis of the sub groups represented and identification of areas of specific significance both within professions as well as between. Similarly, there is a clear need to examine the perspective of lay people and to determine whether there are points of agreement between health professionals and ‘others.’

Once such research has been undertaken, then it is possible to consider future directions should a consensus definition be developed. This would include the potential to develop a measurement tool to be applied within the NZ setting, which
would allow the quantification of ‘inappropriate’ use of EDs. This is a necessary first step to developing possible guidelines or recommendations related to the attendance process.

Overcrowding has many contributors, and ‘inappropriate attenders’ may (or may not) be one of these. Many respondents suggested that inappropriate attendance occurred because of barriers to accessing more appropriate care. If this is a contributor, then solutions could involve increasing access to alternative care sources, rather than raising barriers to ED care. Recognition of ‘appropriateness’ and the best response to this is only one of the issues associated with overcrowding, and on its own is unlikely to provide significant benefits. What is important, however, is the need to ensure a sound evidential base for any interventions, and to develop a sustainable response. For this to happen, research relevant to the issues needs to be undertaken, with context specific features acknowledged.

**Conclusion**

While there are some areas of general agreement, there is no clear consensus between the professionals surveyed regarding the concept of ‘appropriateness’. This has implications for any interventions aimed at addressing ED ‘overcrowding’ that assume the presence of a consensus understanding of this concept.

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**References:**


Cardiovascular risk factors and their associations with alcohol consumption: are there differences between Māori and non-Māori in Aotearoa (New Zealand)?

Dale Bramley, Joanna Broad, Rod Jackson, Papaarangi Reid, Ricci Harris, Shanthi Ameratunga, Jennie Connor

Abstract

Aims To describe the relationship between indicators of alcohol consumption and major known cardiovascular risk factors, and to test whether these relationships are different for Māori and non-Māori.

Methods Data from five New Zealand studies (national and population specific) conducted since 1988 were made available to the investigators and were re-analysed by Māori and non-Māori classification using multivariate modelling adjusting for sex and age. Three indicators of alcohol consumption were used: frequency of drinking, volume drunk on a typical or usual occasion, and average daily consumption. Interaction terms were used to test for differences between Māori and non-Māori in the associations between alcohol consumption and cardiovascular risk factors (tobacco smoking, systolic and diastolic blood pressure, high density lipoprotein (HDL), the ratio of total cholesterol to HDL, serum glucose, reported diagnosis of diabetes, and body mass index).

Results There were a total of 44,830 people in the combined study populations of whom 6926 (15.4%) were Māori. For the risk factors examined, in general Māori had higher levels of risk compared to non-Māori. The pattern of associations between each of the three indicators of alcohol consumption and lipid factors, diabetes, serum glucose level, and obesity were not shown to be different in Māori and non-Māori. However for systolic blood pressure and tobacco smoking, the patterns of association were different.

Conclusion There are clear associations for most of the cardiovascular risk factors examined and alcohol consumption. These associations are consistent for Māori and non-Māori, except for blood pressure and cigarette smoking. As the study is hypothesis-generating, further investigation is required for confirmation.

The relationship between alcohol consumption and cardiovascular disease mortality has been investigated in numerous studies, including several case control studies in non-Māori New Zealanders. Most studies show that low to moderate alcohol consumption is associated with a reduced risk of coronary disease in middle-aged and older people.1–3 However little is known about this relationship in Māori.

We have previously reported the differences in drinking patterns of Māori and non-Māori, and showed that (although average daily consumption was similar) compared to non-Māori, Māori drink alcohol less often but in greater volume.4 In the absence of direct evidence from case-control or cohort studies on the relationship between alcohol and cardiovascular disease (CVD) in Māori, we have
attempted to examine the relationship indirectly by describing the relationship between alcohol consumption and CVD risk factors in Māori and non-Māori.

The aims of this paper are to:

- Describe the relationship between indicators of alcohol consumption and major cardiovascular risk factors: tobacco smoking; systolic blood pressure (SBP) and diastolic blood pressure (DBP); high-density lipoprotein (HDL) and the ratio of total cholesterol to HDL; serum glucose; and body mass index (BMI).
- Test whether the relationships found are different for Māori and non-Māori.

**Methods**

**Data collection**—We identified five large New Zealand studies that gathered information about cardiovascular risk factors and alcohol consumption, had large Māori representation, and for which data were available to the authors. Included were two national cross-sectional surveys (New Zealand Health Survey 1997 and the Sleep Survey 1999) and baseline data from three cohort studies (Fletcher Challenge /University of Auckland Survey 1992, NZ Blood Donors Health Study 1998-1999, and the Workforce Diabetes Survey 1988-1990). Details of sampling, inclusion criteria, and data collection procedures are available in the literature. Of the five studies, two were based on randomly selected population samples: the NZ Health Survey (merged with its subset the NZ Nutrition Survey), and the Sleep Survey. All studies included information about age, sex, ethnicity, and drinker/non-drinker status and tobacco smoking status. Only drinker/non-drinker status and average daily volume could be assessed in The Workforce Diabetes Study, whereas all other studies were also able to assess frequency of drinking and volume consumed per occasion.

Only adults aged 18 to 74 years were included in the study, and they were classified in three age-groups (18–34, 35–54, and 55–74 years). Participants selected the ethnic group(s) with which they identified, and for this purpose were classified as Māori if there was any mention of Māori ethnicity, or otherwise as non-Māori.

Three indicators of alcohol consumption were used:

- Frequency of alcohol consumption was categorised in a slightly different manner in each survey. To obtain a comparable measure across the surveys, the mid-point of the interval selected in each study questionnaire was used to estimate the number of days on which alcohol was consumed each year.

- Volume of alcohol consumed on a typical or usual occasion was calculated for each participant according to the type of alcoholic drink typically consumed, and the number of drinks consumed.

- Estimates of average daily consumption were calculated from the number of days on which alcohol was consumed and the volume consumed on a typical or usual occasion.

Frequency of drinking, volume drunk on a typical occasion, and average daily consumption were categorised into five ordinal groups roughly approximating quintiles. The cut-points of each group were selected to ensure that reasonable numbers of participants in each study were classified in each group.

In all, eight cardiovascular risk variables were classified if available in the survey data. Six cardiovascular risk factors were continuous measures: systolic blood pressure (SBP) and diastolic blood pressure (DBP) in mmHg, high density lipoprotein (HDL) in mmol/L, the ratio of total cholesterol to HDL, serum glucose in mmol/L, and body mass index (BMI) calculated as weight in kilograms divided by height in metres squared.

In addition, participants were classified as either ‘yes’ or ‘no’ for ‘being a current smoker’ and ‘having had diabetes diagnosed by a doctor’. Weighting to represent the total population was not undertaken since three of the surveys used were convenience samples of particular groups.

**Statistical analyses**—In all analyses, generalised estimating equations were used to assess the association of one measure of alcohol consumption with each cardiovascular risk factor variable in turn being the dependent variable. Each model adjusted for survey, sex and age group, age and age-squared (in years), ethnicity (Māori or non-Māori), and two interaction terms. The first interaction term, survey
by measure of alcohol, was used to adjust for the differing questionnaires and definitions employed in the surveys. A logistic link was used in those models where the dependent variable was binary. Participants for whom either consumption data or cardiovascular risk factor data were missing were dropped from the models for that analysis, but do appear in other models.

Since the question addressed in this paper relates to the differences between Māori and non-Māori in the association of the indicators of alcohol consumption and individual cardiovascular risk factors, the test for difference was the statistical significance of the second interaction term—between ethnicity and the alcohol consumption indicators. Estimates obtained from the models are reported, with their 95% confidence intervals (using exponential transforms for the logistic models) to describe the associations. Lack of significance of the second interaction term was interpreted as lack of a significant difference by ethnicity and/or lack of statistical power to detect a difference.

Generalised estimating equations were also constructed to test whether BMI and tobacco smoking accounted for some of the association of the alcohol variables with SBP, given the known relationships between these variables. Similar models as before were created, each with three additional predictors: BMI, BMI squared, and tobacco smoking. The predictors of interest in these final models were the interaction of the terms for ethnicity and alcohol consumption.

Results

Data for a total of 44,830 participants were reviewed. The five studies and demographic characteristics are described elsewhere, but briefly, 45.4% were women, 29.4% were aged over 50 years, and 29.4% were aged under 35 years; 15.5% identified themselves as Māori.

Table 1. Indicators of alcohol consumption in five New Zealand studies

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total</th>
<th>Blood Donor</th>
<th>Fletcher Chal/UoA</th>
<th>NZHNS</th>
<th>Sleep</th>
<th>Workforce Diabetes</th>
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<tbody>
<tr>
<td>N</td>
<td>44,830</td>
<td>17,437</td>
<td>7,936</td>
<td>6,909</td>
<td>6,928</td>
<td>5,620</td>
</tr>
<tr>
<td>Drinker (%)</td>
<td>83.6</td>
<td>81.0</td>
<td>89.1</td>
<td>80.7</td>
<td>83.3</td>
<td>87.4</td>
</tr>
<tr>
<td>Non-drinker (%)</td>
<td>16.4</td>
<td>19.0</td>
<td>10.9</td>
<td>19.3</td>
<td>16.7</td>
<td>12.6</td>
</tr>
<tr>
<td>Frequency of drinking (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–9 days per year)</td>
<td>19.4</td>
<td>19.2</td>
<td>19.6</td>
<td>19.7</td>
<td>16.8</td>
<td>–</td>
</tr>
<tr>
<td>Q2 (10–34)</td>
<td>21.2</td>
<td>17.2</td>
<td>10.6</td>
<td>25.6</td>
<td>39.2</td>
<td>–</td>
</tr>
<tr>
<td>Q3 (35–74)</td>
<td>21.0</td>
<td>17.9</td>
<td>10.6</td>
<td>25.6</td>
<td>39.2</td>
<td>–</td>
</tr>
<tr>
<td>Q4 (75–184)</td>
<td>21.3</td>
<td>23.7</td>
<td>28.4</td>
<td>19.9</td>
<td>8.5</td>
<td>–</td>
</tr>
<tr>
<td>Q5 (185+)</td>
<td>17.6</td>
<td>22.0</td>
<td>20.3</td>
<td>11.9</td>
<td>8.9</td>
<td>–</td>
</tr>
<tr>
<td>Volume pure alcohol drunk on typical occasion (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–&lt;5 grams)</td>
<td>19.4</td>
<td>11.4</td>
<td>19.6</td>
<td>16.9</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Q2 (5–&lt;20)</td>
<td>24.3</td>
<td>21.1</td>
<td>14.3</td>
<td>44.6</td>
<td>23.8</td>
<td>–</td>
</tr>
<tr>
<td>Q3 (20–&lt;40)</td>
<td>34.2</td>
<td>38.0</td>
<td>40.7</td>
<td>17.1</td>
<td>34.2</td>
<td>–</td>
</tr>
<tr>
<td>Q4 (40–&lt;60)</td>
<td>9.2</td>
<td>8.9</td>
<td>8.3</td>
<td>9.5</td>
<td>10.6</td>
<td>–</td>
</tr>
<tr>
<td>Q5 (60+)</td>
<td>14.9</td>
<td>12.7</td>
<td>25.3</td>
<td>9.2</td>
<td>14.5</td>
<td>–</td>
</tr>
<tr>
<td>Average daily volume pure alcohol drunk (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–&lt;0.2 grams)</td>
<td>20.3</td>
<td>19.4</td>
<td>17.4</td>
<td>19.6</td>
<td>30.4</td>
<td>15.5</td>
</tr>
<tr>
<td>Q2 (0.2–&lt;2.0)</td>
<td>18.5</td>
<td>15.1</td>
<td>9.7</td>
<td>33.4</td>
<td>23.8</td>
<td>16.5</td>
</tr>
<tr>
<td>Q3 (2.0–&lt;6.0)</td>
<td>18.2</td>
<td>19.9</td>
<td>18.4</td>
<td>21.5</td>
<td>11.9</td>
<td>16.9</td>
</tr>
<tr>
<td>Q4 (6.0–&lt;15.0)</td>
<td>23.7</td>
<td>28.1</td>
<td>29.1</td>
<td>15.7</td>
<td>15.8</td>
<td>22.1</td>
</tr>
<tr>
<td>Q5 (15+)</td>
<td>19.3</td>
<td>17.5</td>
<td>25.5</td>
<td>9.9</td>
<td>18.1</td>
<td>29.0</td>
</tr>
</tbody>
</table>

A comparison of alcohol consumption data from the five studies is described in Table 1. Most participants reported that they did consume alcohol (overall 83.6% of participants), this varied from 80.7% in the New Zealand Health and Nutrition Survey to 89.1% in the Fletcher Challenge/University of Auckland Study. Considerable variation was seen in the proportions of people classified at different levels of alcohol consumption, as described by frequency of drinking and volume consumed on a typical occasion.

Table 2 shows that Māori and non-Māori participants had similar distributions by sex and age, but there are differences in indicators of alcohol consumption. Unadjusted comparisons indicate that Māori tend to drink alcohol less often (albeit more volume per occasion) than non-Māori.

Table 2. Demographic characteristics and indicators of alcohol consumption in five New Zealand studies, unadjusted

<table>
<thead>
<tr>
<th>Variable</th>
<th>All N=44,830</th>
<th>Māori N=6,926</th>
<th>Non-Māori N=37,904</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex and age group (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men aged 15–34 years</td>
<td>6,404</td>
<td>14.6</td>
<td>14.2</td>
</tr>
<tr>
<td>Men aged 35–49 years</td>
<td>10,465</td>
<td>22.7</td>
<td>23.5</td>
</tr>
<tr>
<td>Men aged 50–74 years</td>
<td>7,615</td>
<td>13.8</td>
<td>17.6</td>
</tr>
<tr>
<td>Women aged 15–34 years</td>
<td>6,770</td>
<td>16.2</td>
<td>14.9</td>
</tr>
<tr>
<td>Women aged 35–49 years</td>
<td>8,013</td>
<td>20.3</td>
<td>17.4</td>
</tr>
<tr>
<td>Women aged 50–74 years</td>
<td>5,563</td>
<td>12.5</td>
<td>12.4</td>
</tr>
<tr>
<td>Non-drinker (%)</td>
<td>7,367</td>
<td>21.1</td>
<td>15.6</td>
</tr>
<tr>
<td>Drinker (%)</td>
<td>37,463</td>
<td>78.9</td>
<td>84.4</td>
</tr>
<tr>
<td>Frequency of drinking (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–9 days per year)</td>
<td>8,023</td>
<td>25.2</td>
<td>19.4</td>
</tr>
<tr>
<td>Q2 (10–34)</td>
<td>8,229</td>
<td>33.9</td>
<td>18.3</td>
</tr>
<tr>
<td>Q3 (35–74)</td>
<td>8,118</td>
<td>24.0</td>
<td>19.9</td>
</tr>
<tr>
<td>Q4 (75–184)</td>
<td>8,248</td>
<td>11.3</td>
<td>22.8</td>
</tr>
<tr>
<td>Q5 (185+)</td>
<td>6,811</td>
<td>5.7</td>
<td>19.6</td>
</tr>
<tr>
<td>Volume pure alcohol drunk on typical occasion (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–&lt;5 grams)</td>
<td>6,663</td>
<td>22.1</td>
<td>16.4</td>
</tr>
<tr>
<td>Q2 (5–&lt;20)</td>
<td>9,320</td>
<td>14.3</td>
<td>26.3</td>
</tr>
<tr>
<td>Q3 (20–&lt;40)</td>
<td>13,132</td>
<td>23.6</td>
<td>36.3</td>
</tr>
<tr>
<td>Q4 (40–&lt;60)</td>
<td>3,521</td>
<td>11.4</td>
<td>8.7</td>
</tr>
<tr>
<td>Q5 (60+)</td>
<td>5,730</td>
<td>28.5</td>
<td>12.2</td>
</tr>
<tr>
<td>Average daily volume pure alcohol drunk (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (0–&lt;0.2 grams)</td>
<td>8904</td>
<td>27.6</td>
<td>19.0</td>
</tr>
<tr>
<td>Q2 (0.2–&lt;2.0)</td>
<td>8104</td>
<td>21.7</td>
<td>17.9</td>
</tr>
<tr>
<td>Q3 (2.0–&lt;6.0)</td>
<td>8002</td>
<td>16.5</td>
<td>18.6</td>
</tr>
<tr>
<td>Q4 (6.0–&lt;15.0)</td>
<td>10,395</td>
<td>15.5</td>
<td>25.2</td>
</tr>
<tr>
<td>Q5 (15+)</td>
<td>8,475</td>
<td>18.7</td>
<td>19.4</td>
</tr>
</tbody>
</table>
Table 3 shows cardiovascular risk factors for Māori and non-Māori. The cardiovascular risk factor indicators were all worse for Māori than for non-Māori.

Table 3. Cardiovascular risk factors in five New Zealand studies, unadjusted

<table>
<thead>
<tr>
<th>Variable</th>
<th>Information available</th>
<th>Māori N=6,926</th>
<th>Non-Māori N=37,904</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lipid profile</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL (mmol/L), mean (SE)</td>
<td>8,473</td>
<td>1.21 (0.011)</td>
<td>1.29 (0.004)</td>
</tr>
<tr>
<td>Ratio total cholesterol: HDL, mean (SE)</td>
<td>8,471</td>
<td>5.14 (0.060)</td>
<td>5.04 (0.019)</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic (mmHg), mean (SE)</td>
<td>32,656</td>
<td>126.1 (0.33)</td>
<td>125.8 (0.10)</td>
</tr>
<tr>
<td>Diastolic (mmHg), mean (SE)</td>
<td>32,655</td>
<td>80.4 (0.22)</td>
<td>78.0 (0.06)</td>
</tr>
<tr>
<td><strong>Lifestyle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current cigarette smoker, % (SE)</td>
<td>44,827</td>
<td>44.1 (0.6)</td>
<td>21.4 (0.2)</td>
</tr>
<tr>
<td><strong>Body build</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Mass Index, mean (SE)</td>
<td>34,720</td>
<td>29.0 (0.10)</td>
<td>26.5 (0.03)</td>
</tr>
<tr>
<td><strong>Diabetes related</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum glucose (mmol/L), mean (SE)</td>
<td>13,510</td>
<td>5.16 (0.05)</td>
<td>4.92 (0.01)</td>
</tr>
<tr>
<td>Reported diagnosis by doctor, % (SE)</td>
<td>37,899</td>
<td>4.0 (0.3)</td>
<td>1.4 (0.1)</td>
</tr>
</tbody>
</table>

SE = Standard error; Crude means and percentages (unadjusted for sex, age or survey); Obese is defined as BMI over 30 if non-Māori and non-Pacific, over 32 if Māori or Pacific.

Results from the regression models are shown in Figures 1–3. Figure 1(a) and 1(b) show the association between lipid profile and alcohol consumption. There was no evidence of a different association for Māori compared to non-Māori. However, there are quite different associations for tobacco smoking (Figure 1[c]). For Māori, low and high levels of frequency of drinking are associated with a lower proportion of current smokers. For non-Māori, there is a consistent slight increase in smoking with frequency of drinking (p=0.0001).

Figure 2 shows that the associations between blood pressure and one of the measures of alcohol intake (volume usually consumed) are clearly different for Māori compared to non-Māori. For non-Māori, increasing volume usually consumed is associated with higher systolic, diastolic, and adjusted systolic blood pressure. In contrast, the pattern is more variable for Māori.

Although Māori have a higher proportion with a diabetes diagnosis, and higher serum glucose levels, no statistically significant differences between Māori and non-Māori were evident in the associations with alcohol consumption (Figure 3).

The associations of body build with alcohol consumption are also shown in Figure 3. Māori have a higher BMI than non-Māori on average; among Māori, the inverse association of BMI with frequency of drinking followed a similar pattern to non-Māori although the gradient was steeper (p<0.0001).
Figure 1. Relationship between lipid profile, tobacco smoking and alcohol consumption, by ethnicity, adjusting for age, sex and survey

a. HDL cholesterol

b. Ratio total cholesterol: HDL

c. Current tobacco smoker
Figure 2. Relationship between blood pressure and alcohol consumption, by ethnicity, adjusting for age, sex and survey

i) Frequency of drinking
(Q1 = none or low, Q5 = high)

ii) Volume usually consumed
(Q1 = none or low, Q5 = high)

iii) Average daily volume
(Q1 = none or low, Q5 = high)

a. Systolic blood pressure

b. Diastolic blood pressure

c. Systolic blood pressure adjusted for BMI and tobacco smoking
Figure 3. Relationship between diabetes-related factors and alcohol consumption, by ethnicity, adjusting for age, sex and survey

1) Frequency of drinking
(Q1 = none or low, Q5 = high)

a. Reported diagnosis of diabetes by doctor

b. Serum glucose

c. BMI
Discussion

This research has been undertaken using a kaupapa Māori framework whereby the study analysis was undertaken from a Māori perspective. This is distinct from other methodologies that may “minoritise” Māori with insufficient data quantity or quality to undertake analyses necessary to inform Māori health development. Where appropriate, this type of analysis enables disparities to be identified and their elimination prioritised. This is consistent with the Treaty of Waitangi.

The data from the five studies combined shows that:

- For all risk factors reported, Māori have higher cardiovascular risk compared to non-Māori. For many of the risk factors, there are clear associations between one or more indicators of alcohol consumption and the risk factor of interest, which do not differ between Māori and non-Māori.

- For the lipid measures used, reported diagnosis of diabetes, and serum glucose level there were no statistically significant differences in their associations with alcohol consumption between Māori and non-Māori. However, for systolic blood pressure there is a highly significant difference in volume usually consumed and a significant difference in frequency of alcohol consumption.

These differences remain after adjustment for BMI and tobacco smoking. For BMI, as frequency of drinking increases among Māori, the associated decrease in BMI is more pronounced than in non-Māori. However no differences are apparent for either measure of volume of alcohol consumed. For tobacco smoking, highly significant differences exist in the shape of relationships between ethnicity all three indicators of alcohol used.

In a previous paper, we showed that drinking patterns for Māori and non-Māori were different. Overall, Māori drink less often with higher volumes per occasion, resulting in similar average daily levels of alcohol consumption.\textsuperscript{4} Ethnic differences in drinking patterns have also been reported elsewhere. For example, African Americans appear to have higher frequency of heavy drinking occasions and higher proportions of non-drinkers.\textsuperscript{13,14}

Ethnic-specific differences in cardiovascular mortality may relate to alcohol consumption.\textsuperscript{15} Sempos found that after analysing the relationship between average volume of alcohol consumed and all cause mortality that no J-shaped relationship existed for African Americans whereas it did for whites (Caucasians).\textsuperscript{15}

Sempos has also explored the association between average volume of alcohol consumed and coronary artery disease mortality and morbidity in African Americans and whites. In general, average moderate alcohol consumption in African Americans was associated with higher levels of coronary artery disease risk with fewer apparent protective effects when compared to whites.\textsuperscript{16}

We found that for some cardiovascular risk factor associations (notably systolic and diastolic blood pressure by volume usually consumed, and tobacco smoking by any of the three measures of alcohol consumption), the associations with alcohol consumption do vary between Māori and non-Māori—results which may support a plausible biological pathway through which ethnic differences in cardiovascular mortality may arise.
Research has consistently found that high alcohol intake is associated with hypertension.\textsuperscript{17–19} However there is less certainty regarding the relationship of light to moderate alcohol consumption with blood pressure. Some studies report that low alcohol intake may be associated with decreased blood pressure\textsuperscript{18,20} whilst others demonstrate a gradual increase in blood pressure as alcohol intake increases.\textsuperscript{17,21} These discrepancies may reflect differences in investigational design, measurement methods and populations.\textsuperscript{22}

A previous study undertaken in New Zealand demonstrated a U-shaped relationship between average daily volume of alcohol consumed, and systolic and diastolic blood pressures, in both men and women, with light to moderate drinkers having lower blood pressure than either non-drinkers or heavy drinkers.\textsuperscript{23} Our study confirms that relationship for both Māori and non-Māori.

For non-Māori, but not Māori, a more linear relationship with blood pressure exists between both frequency of drinking and volume usually consumed. These findings suggest that ethnic variation in the relationship between usual volume of alcohol consumption and blood pressure may partially explain inconsistencies in the published international literature regarding the relationship of alcohol consumption and blood pressure. Ethnic differences have also recently been reported for black (African American) men in the US compared to whites regarding the association between low-to-moderate alcohol consumption and hypertension.\textsuperscript{22}

Several international studies have shown that alcohol consumption is strongly associated with tobacco use. In general, smokers are more likely to consume alcohol than non-smokers.\textsuperscript{24–26} Our study shows that among drinkers, as average daily volume and volume usually consumed on a typical occasion increases so does the likelihood of being current a smoker for both Māori and non-Māori. Again, however, a difference exists for Māori, in that (in the upper quintiles of frequency of drinking) the probability of being a smoker decreases, whilst for non-Māori it increases.

In regards to lipids, it has been estimated that approximately half the protective effect of alcohol on coronary heart disease is related to the beneficial effect of alcohol on HDL cholesterol.\textsuperscript{27} Therefore our finding (for the lipid measures used) that there were no statistically significant differences in the associations with alcohol consumption between Māori and non-Māori is of importance.

Concerning the other two risk factors used in this study the literature is consistent in demonstrating both a small protective association between light-to-moderate alcohol consumption and diabetes (type 2),\textsuperscript{28,29} and an inverse relationship between light-to-moderate alcohol consumption and BMI.\textsuperscript{30} These analyses provide indirect evidence that the protective effect of light-to-moderate alcohol consumption on CVD risk (previously demonstrated in non-Māori New Zealanders) may be similar in Māori, as the pattern of association between alcohol consumption measures and the metabolic-related cardiovascular risk factors is generally similar. However Māori/non Māori differences in blood pressure associated with usual volume of alcohol consumed could adversely impact on this protective association given the substantial differences in usual volume consumed per occasion between the two ethnic groups.
Moreover, while the relationship between cigarette smoking and alcohol consumption is behavioural rather than physiological, the strong association between them and the differences in this association by ethnicity may differentially influence the relationship between alcohol consumption and coronary disease in Māori and non-Māori. Until aetiologic studies are conducted among Māori, however, the balance of benefits and harms of alcohol consumption on cardiovascular risk remains uncertain.

Several associations between alcohol consumption and CVD risk factors are of interest that have potential to partially explain some differences in cardiovascular risk between Māori and non-Māori. However, it should be noted that the alcohol-related associations reported here are not necessarily causal, since cross-sectional studies can only describe relationships. Of note are those with tobacco smoking and blood pressure.

There are several potential biases that may occur with our study methodology. The studies we were able to include were conducted during different time periods (1988–2001), and drinking patterns may have changed over that period. The use of a mixed group of cross-sectional studies, only some of which were population-based, and their different instruments for measuring alcohol consumption make it possible that reported means and proportions maybe inaccurate.

We attempted to overcome these potential problems by adjusting for those variations in the models so the interaction terms of interest are less likely to be affected—since most study-specific differences would be absorbed within the study term in the models.

Measurement bias may be possible given that the indicators of alcohol consumption used were self-reported and these biases may differ by ethnicity. Although these results demonstrate comparisons between Māori and non-Māori (regarding the association of cardiovascular risk factors with alcohol consumption), they should not be interpreted as good indicators of the prevalence of risk factors since not all the studies contributing data were representative of the population. Indeed, for some of the variables examined, the proportion of missing data was significantly higher for Māori compared to non-Māori and this may account for some of the significant differences found, or absence of differences that may exist.

In any study comparing ethnic groups there is a possibility that selection biases may be different for one ethnic group than another. It is unlikely that language itself is an issue, as most Māori speak English as their first language. But it is known that response rates in New Zealand to questions are higher if interviewers and interviewees are of the same ethnicity. Unfortunately, we are unable to assess if this is the case for these studies.

Some confounders known to be associated with cardiovascular risk were not available for analysis—for example, socioeconomic status, salt intake, other dietary factors, appropriate levels of treatment, access to medical care, and levels of certain psychosocial stressors. Further, some associations may have arisen by chance alone (many associations are being described and there is considerable statistical power), so it is important that these results are confirmed.

This research is important in addressing issues related to inequalities. Questions about whether the interaction and association of cardiovascular risk factors with
cardiovascular disease differ by ethnicity are of high importance to the academic discourse on inequalities in this country (cardiovascular disease being the leading cause of death for Māori and non-Māori with significant inequalities existing for Māori).

This paper attempts to investigate ethnic differences in the association of alcohol consumption with cardiovascular risk factors and begins to address the current lack of research available regarding these important issues.

**Conclusion**

There are clear associations for most of the cardiovascular risk factors examined and alcohol consumption. These associations are consistent for Māori and non-Māori, except for blood pressure and cigarette smoking. As the study is hypothesis-generating, further investigation is required for confirmation.

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**Acknowledgements:** This paper is part of a series of papers commissioned by the Alcohol Advisory Council of New Zealand. The New Zealand Health Survey and the National Nutrition Survey were funded by the Ministry of Health and undertaken by Statistics New Zealand and the University of Otago. The Sleep Study was funded by the Health Research Council of New Zealand and undertaken by Te Rōpū Rangahau Hauora a Eru Pōmare, University of Otago and the Sleep/Wake Research Centre, Massey University. Fisher and Paykel Healthcare sponsored the incentive prize for participants. The Fletcher Challenge University of Auckland Heart and Health survey was supported in part by grants from the Fletcher Challenge Welfare Fund, the Health Research Council of New Zealand, and the National Heart Foundation of New Zealand. The New Zealand Blood Donors Health Study was funded by the Health Research Council of New Zealand and undertaken as a collaboration between the University of Auckland, the University of Otago, and the NZ Blood Service. Data management and blood collection were supported by the Clinical Trials Research Unit (University of Auckland) and Baxter Healthcare Ltd, respectively. The Workforce Diabetes Survey was funded by the Health Research Council of New Zealand and undertaken by the University of Auckland, Department of Community Health. Datasets for analysis were provided from Robert Scragg and Patricia Metcalf (Workforce Diabetes Survey); Stephen MacMahon, Robyn Norton, and Shanthi Ameratunga (New Zealand Blood Donors Health Study); Rod Jackson, Stephen MacMahon, and Robyn Norton (Fletcher-Challenge University of Auckland Heart and Health Study); Ricci Harris, Papaarangi Reid, and Philippa Gander (Sleep Survey); and the Ministry of Health (National Nutrition Survey).

The authors also acknowledge and thank the following people who assisted by providing advice or reviewing this paper prior to publication: Robert Scragg, Elizabeth Robinson, Robyn Norton, Margaret Geddes, and Mike MacAvoy.
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References:


Estimated prevalence of cardiovascular disease and distribution of cardiovascular risk in New Zealanders: data for healthcare planners, funders, and providers

Susan Wells, Joanna Broad, Rod Jackson

Abstract

Aims New Zealand cardiovascular risk management guidelines advocate targeted risk assessment based primarily on age, gender, and ethnicity—and recommend drug management for people with a 5-year absolute cardiovascular disease (CVD) risk greater than 15%. To inform service planning and healthcare delivery for district health boards and primary healthcare organisations in New Zealand, we have produced population estimates of CVD prevalence and 5-year absolute CVD risk.

Methods The 1993 Auckland Heart and Health Study provided the data for estimating CVD prevalence and absolute CVD risk distributions using the Framingham CVD risk prediction equation. These estimates were applied to population projections for 2005 based on 2001 New Zealand Census data.

Results Of the projected 2.09 million people aged over 35 years in New Zealand in 2005, approximately 1.5 million (72%) meet national criteria for formal CVD risk assessment. About 151,000 (7%) are estimated to have suffered a non-fatal heart attack or stroke or have angina. A further 262,000 (13%) are estimated to have a 5-year CVD risk greater than 15% based on New Zealand CVD risk charts. This represents around 1 in 5 adults over the age of 35 years in New Zealand for whom pharmacological interventions are recommended according to the New Zealand CVD risk guidelines for the prevention of new or further CVD events.

Conclusions The latest published data available on the burden of CVD risk in New Zealand is now over 10 years old and does not include Maori, Pacific, and other non-European ethnic groups. Current data on the risk profile of adult New Zealanders is required for more accurate service planning. However the information reported here provides a reasonable estimate of the magnitude of the task. Although systematic identification and management of CVD risk in New Zealanders with raised CVD risk will be a major undertaking for healthcare services, it has the potential to produce significant health-gains while reducing health disparities.

Cardiovascular disease (CVD) is the leading cause of death and hospitalisation in New Zealand.¹ There are major disparities in CVD between ethnic groups and significant under-treatment of high-risk patients. Age-specific death rates are two to three times higher for Maori compared with non-Maori in those aged less than 75 years.² CVD prevention and management and the reduction of health inequalities have been targeted as priorities in the New Zealand Health Strategy, He Korowai Oranga (Maori Health Strategy), and Primary Health Care Strategy.

Current New Zealand CVD risk management guidelines recommend targeting CVD risk assessment to all men aged over age 45 years and women aged over 55 years (10
years earlier for people of Maori, Pacific Island, or Indian ethnicity or if they have known CVD risk factors or are at high risk of developing diabetes3 [Appendix 1]).

Treatment recommendations are based primarily on the patient’s estimated absolute risk, with pharmacological treatments recommended for those over 15% 5-year absolute CVD risk.

To inform their health-needs assessments, and to guide healthcare planning and funding decisions, over the previous 2 years we have been asked by district health boards (DHBs) and primary healthcare organisations (PHOs) to provide estimates of the prevalence of CVD and distributions of CVD-risk in the community. As there is limited data on CVD and absolute risk prevalence available in the public domain, we considered that this information would also be useful to other DHBs and PHOs.

Methods

Population estimates were provided by Statistics New Zealand for the projected 2005 adult population aged over 35 years (divided according to 5-year age groups and gender) for the whole of New Zealand, and for each DHB separately. Estimates were based on the 2001 Census usually resident populations, assuming medium fertility, medium mortality, and medium migration term projection methods.

To calculate absolute CVD risk, we used data from the Auckland Heart and Health Study (AHAH)4 to estimate the proportion eligible for risk assessment and to derive estimates of the prevalence of existing cardiovascular disease and cardiovascular risk factors.

The AHAH study was a population-based cross-sectional survey conducted between 1993 and 1994. The study population included 2507 men and women aged 35–84 years and resident within the Auckland region of New Zealand. Age-stratified samples were randomly chosen from central Auckland general electoral rolls with a response rate of 72%. The investigators aimed to include 250 subjects from each 10-year age/sex category. For our purposes, data were reaggregated into 5-year age/sex categories.

Analyses excluded Maori and Pacific Island ethnic groups as the sampling frame did not include the Maori electoral roll and the general electoral rolls significantly under-represented the true proportions of Pacific and Maori peoples within the general population. A detailed account of AHAH study methodology is presented elsewhere.4 The proportion eligible for risk assessment was based as closely as possible to the New Zealand Guideline Criteria (Appendix 1). Data on personal history of gestational diabetes, polycystic ovarian syndrome, known impaired glucose tolerance or impaired fasting glycaemia, and waist circumference were not available. However these omissions are unlikely to add significantly to the number of eligible people.

Previous history of CVD in the AHAH study includes coronary heart disease and previous self-reported stroke—but not transient ischaemic attack, peripheral vascular disease, or previous coronary artery surgery. Coronary heart disease was determined by self-reported myocardial infarction, with hospital admission or angina defined as currently taking nitrate medication. The AHAH study did not collect any data on CVD ‘risk equivalents’ (genetic lipid disorders or diabetes with nephropathy).

The absolute CVD risk over a 5-year period for each individual was estimated using a risk prediction model based on the Framingham Heart Study.5 The model includes gender, age, systolic blood pressure, smoking, total cholesterol:high density lipoprotein (TC:HDL) ratio, diabetes, and interaction terms of age by gender, and diabetes by gender. A cardiovascular event is defined in the risk prediction model as a death related to coronary disease, non-fatal myocardial infarction, new angina, fatal or non-fatal stroke, or transient ischaemic attack—or the development of congestive heart failure or peripheral vascular disease.

Summary measures of risk categories (proportion over 20%, 15–20%, 10–15%, and less than 10% absolute CVD risk) were obtained for each 5-year age-gender group.

For the small number of people (1.5%) missing blood pressure or lipid data we calculated their absolute risk using the age/gender specific median value for that risk factor. Rate smoothing via moving
averages was applied to better reflect the naturally occurring patterns within populations and all estimated counts were rounded.

To estimate the numbers of people meeting risk-assessment criteria (as defined in the guideline), we used:

- Statistics New Zealand 2005 population projections for all men aged over 45 years and women aged over 55 years, plus Maori and Pacific men (aged 35–44 years) and women (aged 45–54 years); and
- An estimate of the proportion of other men aged 35–44 years and women aged 45–54 years with one or more risk factors collected in the AHAH study—these factors included those people with a first-degree family history of premature coronary heart disease or stroke, personal history of smoking, blood pressure of more than 160/95 mmHg, TC:HDL ratio more than 7.0, or obesity (body mass index [BMI] over 30).

Results

Of the projected 2.09 million people aged over 35 years in New Zealand in 2005, approximately 1.5 million (72%) would meet national criteria for formal CVD risk assessment. Table 1 shows the proportion of New Zealanders over 35 years with prior CVD and the distribution of absolute CVD risk in those without prior CVD, by gender and 5-year age group for the estimated 2005 population.

Approximately 151,000 people (7%) have suffered a heart attack or stroke or have angina (or a combination of these). About 272,000 people (13%) are at high (CVD risk 15–20%) or very high (CVD risk over 20%) risk of a new CVD event in the next 5 years. Men are about three times as likely to be at high or very high risk than women (19% vs 7%). A further 10% of the total population over the age of 35 years are at moderate risk (CVD risk 10–15% in 5 years).

For each DHB catchment, aggregated estimates of CVD prevalence and CVD risk distributions are shown (Table 2). The proportion of people aged over 35 years meeting criteria for drug treatment (with prior CVD or an absolute CVD risk over 15% in 5 years) varies from 18.3% to 25.5% according to the demographic structure of the DHB.

Discussion

Current New Zealand guidelines for the management of CVD risk are based on evidence demonstrating that the magnitude of benefit from treatment for an individual patient is directly proportional to their pre-treatment absolute CVD risk. Those who have had a prior CVD event and those at high risk of having a first event have the most to gain from identification and coordinated care. In appropriately targeted patients, the New Zealand guidelines suggest that 55% of future CVD events could be prevented. We estimate that 7 out of 10 New Zealanders over 35 years of age should have a baseline risk assessment; and of those risk-assessed people, 1 out of 5 would meet criteria for drug treatment.

Cardiovascular risk prediction based on the Framingham Heart Study is integral to the New Zealand cardiovascular risk assessment and management guidelines. The Framingham Heart Study was a cohort of mainly white Americans living in Massachusetts, USA in the 1970s and 1980s. The resultant cardiovascular risk prediction equation has been found to accurately predict on a population basis the 5-year risk of hospitalisation or death from a first cardiovascular event in New Zealand men aged 35 to 74 years and women aged 35 to 69 years.
## Table 1. Prevalence and risk of cardiovascular disease in New Zealand population, by age and sex

<table>
<thead>
<tr>
<th>Projected population</th>
<th>N</th>
<th>( \text{Prior CVD} )</th>
<th>CVD risk &gt;20%</th>
<th>CVD risk 15-20%</th>
<th>CVD risk 10-15%</th>
<th>CVD risk &lt;10%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35-39</td>
<td>145,600</td>
<td>160</td>
<td>0.1</td>
<td>320</td>
<td>0.2</td>
<td>650</td>
</tr>
<tr>
<td>40-44</td>
<td>154,600</td>
<td>1,550</td>
<td>1.0</td>
<td>1,210</td>
<td>0.8</td>
<td>1,480</td>
</tr>
<tr>
<td>45-49</td>
<td>143,900</td>
<td>3,380</td>
<td>2.3</td>
<td>1,810</td>
<td>1.3</td>
<td>3,130</td>
</tr>
<tr>
<td>50-54</td>
<td>126,700</td>
<td>6,900</td>
<td>4.8</td>
<td>3,240</td>
<td>2.6</td>
<td>6,410</td>
</tr>
<tr>
<td>55-59</td>
<td>116,300</td>
<td>8,250</td>
<td>7.1</td>
<td>9,730</td>
<td>8.4</td>
<td>12,350</td>
</tr>
<tr>
<td>60-64</td>
<td>89,600</td>
<td>10,240</td>
<td>11.4</td>
<td>14,540</td>
<td>16.2</td>
<td>15,280</td>
</tr>
<tr>
<td>65-69</td>
<td>70,300</td>
<td>10,640</td>
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<td>20,920</td>
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<td>14,730</td>
</tr>
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<td>70-74</td>
<td>57,600</td>
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<td>21,130</td>
<td>36.7</td>
<td>10,900</td>
</tr>
<tr>
<td>75-79</td>
<td>46,200</td>
<td>13,480</td>
<td>29.2</td>
<td>21,090</td>
<td>45.7</td>
<td>6,500</td>
</tr>
<tr>
<td>80-84</td>
<td>29,000</td>
<td>9,990</td>
<td>34.5</td>
<td>13,950</td>
<td>48.1</td>
<td>2,920</td>
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<tr>
<td>85+</td>
<td>17,500</td>
<td>6,100</td>
<td>35.0</td>
<td>8,580</td>
<td>49.2</td>
<td>1,700</td>
</tr>
<tr>
<td><strong>All men</strong></td>
<td>997,300</td>
<td>83,370</td>
<td>8.4</td>
<td>116,530</td>
<td>11.7</td>
<td>76,040</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35-39</td>
<td>157,500</td>
<td>150</td>
<td>0.1</td>
<td>310</td>
<td>0.2</td>
<td>620</td>
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<tr>
<td>40-44</td>
<td>164,800</td>
<td>350</td>
<td>0.2</td>
<td>590</td>
<td>0.4</td>
<td>880</td>
</tr>
<tr>
<td>45-49</td>
<td>149,700</td>
<td>510</td>
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<td>0.5</td>
<td>1,320</td>
</tr>
<tr>
<td>50-54</td>
<td>129,400</td>
<td>2,480</td>
<td>1.9</td>
<td>1,430</td>
<td>1.1</td>
<td>2,180</td>
</tr>
<tr>
<td>55-59</td>
<td>118,200</td>
<td>3,900</td>
<td>3.3</td>
<td>1,980</td>
<td>1.7</td>
<td>4,300</td>
</tr>
<tr>
<td>60-64</td>
<td>91,700</td>
<td>5,800</td>
<td>6.3</td>
<td>2,090</td>
<td>2.3</td>
<td>4,830</td>
</tr>
<tr>
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<td>75,000</td>
<td>8,480</td>
<td>11.3</td>
<td>3,240</td>
<td>4.3</td>
<td>5,790</td>
</tr>
<tr>
<td>70-74</td>
<td>63,100</td>
<td>10,870</td>
<td>17.2</td>
<td>5,230</td>
<td>8.3</td>
<td>6,430</td>
</tr>
<tr>
<td>75-79</td>
<td>55,900</td>
<td>13,110</td>
<td>23.5</td>
<td>5,900</td>
<td>10.6</td>
<td>8,070</td>
</tr>
<tr>
<td>80-84</td>
<td>44,300</td>
<td>11,620</td>
<td>26.2</td>
<td>4,870</td>
<td>11.0</td>
<td>7,650</td>
</tr>
<tr>
<td>85+</td>
<td>40,300</td>
<td>10,780</td>
<td>26.7</td>
<td>4,280</td>
<td>10.6</td>
<td>7,120</td>
</tr>
<tr>
<td><strong>All women</strong></td>
<td>1,089,900</td>
<td>68,050</td>
<td>6.2</td>
<td>30,700</td>
<td>2.8</td>
<td>49,180</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>2,087,200</td>
<td>151,420</td>
<td>7.3</td>
<td>147,230</td>
<td>7.1</td>
<td>125,220</td>
</tr>
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</table>

Note:
1. Usually resident population aged over 35 years projected to 2005 from 2001 census counts, sourced from Statistics NZ, Feb 2005.
2. Prior CVD estimates based on smoothed rates from Auckland Heart & Health Survey (1992-3 data for non-Maori, non-Pacific people), for age & sex groups, and includes self-reported heart attack (with hospital admission) or stroke, angina (on nitrates), but not PVD, PTCA, CABG or genetic lipid disorder.
3. CVD Absolute risk estimates calculated using by Framingham 5-year CVD risk equation applied to Auckland Heart and Health Survey data.
Table 2. Prevalence and risk of cardiovascular disease (CVD) in New Zealand population, by District Health Board

<table>
<thead>
<tr>
<th>District Health Board</th>
<th>Projected population</th>
<th>Prior CVD</th>
<th>CVD risk &gt;20%</th>
<th>CVD risk 15-20%</th>
<th>CVD risk 10-15%</th>
<th>CVD risk &lt;10%</th>
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</thead>
<tbody>
<tr>
<td>Northland</td>
<td>81,000</td>
<td>6,420</td>
<td>7.9</td>
<td>6,850</td>
<td>8.5</td>
<td>5,220</td>
</tr>
<tr>
<td>Waitemata</td>
<td>248,800</td>
<td>17,180</td>
<td>6.9</td>
<td>18,500</td>
<td>7.4</td>
<td>13,920</td>
</tr>
<tr>
<td>Auckland</td>
<td>202,100</td>
<td>13,280</td>
<td>6.6</td>
<td>14,330</td>
<td>7.1</td>
<td>10,550</td>
</tr>
<tr>
<td>Counties Manukau</td>
<td>199,500</td>
<td>12,830</td>
<td>6.4</td>
<td>13,740</td>
<td>6.9</td>
<td>10,810</td>
</tr>
<tr>
<td>Waikato</td>
<td>169,900</td>
<td>12,950</td>
<td>7.6</td>
<td>13,890</td>
<td>8.2</td>
<td>10,330</td>
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<tr>
<td>Lakes</td>
<td>51,600</td>
<td>3,770</td>
<td>7.3</td>
<td>4,030</td>
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<td>3,060</td>
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<tr>
<td>Bay of Plenty</td>
<td>106,000</td>
<td>9,020</td>
<td>8.5</td>
<td>9,730</td>
<td>9.2</td>
<td>7,030</td>
</tr>
<tr>
<td>Tairawhiti</td>
<td>21,900</td>
<td>1,640</td>
<td>7.5</td>
<td>1,770</td>
<td>8.1</td>
<td>1,310</td>
</tr>
<tr>
<td>Taranaki</td>
<td>56,800</td>
<td>4,750</td>
<td>8.4</td>
<td>5,150</td>
<td>9.1</td>
<td>3,650</td>
</tr>
<tr>
<td>Hawkes Bay</td>
<td>79,600</td>
<td>6,410</td>
<td>8.1</td>
<td>6,950</td>
<td>8.7</td>
<td>5,030</td>
</tr>
<tr>
<td>MidCentral</td>
<td>83,100</td>
<td>6,820</td>
<td>8.2</td>
<td>7,390</td>
<td>8.9</td>
<td>5,310</td>
</tr>
<tr>
<td>Whanganui</td>
<td>33,800</td>
<td>2,870</td>
<td>8.5</td>
<td>3,150</td>
<td>9.3</td>
<td>2,210</td>
</tr>
<tr>
<td>Capital and Coast</td>
<td>133,600</td>
<td>9,060</td>
<td>6.8</td>
<td>9,760</td>
<td>7.3</td>
<td>7,330</td>
</tr>
<tr>
<td>Hutt</td>
<td>70,000</td>
<td>4,910</td>
<td>7.0</td>
<td>5,320</td>
<td>7.6</td>
<td>3,950</td>
</tr>
<tr>
<td>Wairarapa</td>
<td>22,500</td>
<td>1,950</td>
<td>8.7</td>
<td>2,110</td>
<td>9.4</td>
<td>1,510</td>
</tr>
<tr>
<td>Nelson Marlborough</td>
<td>75,400</td>
<td>6,060</td>
<td>8.0</td>
<td>6,490</td>
<td>8.6</td>
<td>4,730</td>
</tr>
<tr>
<td>West Coast</td>
<td>17,500</td>
<td>1,330</td>
<td>7.6</td>
<td>1,400</td>
<td>8.0</td>
<td>1,070</td>
</tr>
<tr>
<td>Canterbury</td>
<td>248,300</td>
<td>19,530</td>
<td>7.8</td>
<td>21,260</td>
<td>8.6</td>
<td>15,130</td>
</tr>
<tr>
<td>South Canterbury</td>
<td>31,700</td>
<td>2,870</td>
<td>9.1</td>
<td>3,140</td>
<td>9.9</td>
<td>2,180</td>
</tr>
<tr>
<td>Otago</td>
<td>96,200</td>
<td>8,070</td>
<td>8.4</td>
<td>8,780</td>
<td>9.1</td>
<td>6,190</td>
</tr>
<tr>
<td>Southland</td>
<td>57,700</td>
<td>4,370</td>
<td>7.6</td>
<td>4,710</td>
<td>8.2</td>
<td>3,440</td>
</tr>
</tbody>
</table>

Note:
1 Usually resident population aged over 35 years projected to 2005 from 2001 census counts, sourced from Statistics NZ, Feb 2005.
2 Prior CVD estimates based on smoothed rates from Auckland Heart & Health Survey (1992-3 data for non-Maori, non-Pacific people), for age & sex groups, and includes self-reported heart attack (with hospital admission) or stroke, angina (on nitrates), but not PVD, PTCA, CABG or genetic lipid disorder.
3 CVD Absolute risk estimates calculated using the Framingham 5-year CVD risk equation applied to Auckland Heart and Health Survey data.
Extrapolating data collected over 10 years ago from 2507 Aucklanders to the New Zealand population in 2005 requires caution. However they are the only currently available data and are given as ‘ball park’ estimates only. Our findings may underestimate the true prevalence of CVD in New Zealand as they are based on a study that did not include Maori and Pacific people who have higher risk of CVD. Furthermore, these estimates do not include those who have had a sole diagnosis of other cardiovascular disease including transient ischaemic attack, acute coronary syndrome, percutaneous coronary intervention (PCI), or peripheral vascular disease. However, many people with these diagnoses will have other manifestations of atherosclerotic disease. For example, studies of patients presenting with intermittent claudication indicate that around 50% have evidence of coronary disease on clinical history and ECG,\(^8,9\) (and 90% have coronary disease angiographically\(^10\)).

Nevertheless, the magnitude of the reported CVD risk burden is likely to be reasonable because the effect of excluding Maori and Pacific people, and some CVD diagnoses will be offset by declining secular trends in CVD morbidity and mortality over the previous 10 years.\(^2\)

Despite the limitations of the data, this aggregate information is much needed by PHOs and DHBs to guide service planning and health care delivery particularly with new funding streams (for example the CarePlus programme) and requirements to fulfil key quality indicators for their population’s health. The major challenge for primary care is to systematically identify those people most at risk and to ensure they are appropriately managed. If this occurs, the potential to reduce this leading cause of mortality and morbidity in New Zealand, while also reducing disparities between ethnic and socioeconomic groups, will be substantial.

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**Acknowledgements:** The Auckland Heart and Health Study was funded by the Health Research Council and the National Heart Foundation while Dr Wells is the recipient of a National Heart Foundation Research Fellowship. We also thank Elizabeth Robinson and Patricia Metcalf for their helpful comments and feedback.

**Correspondence:** Dr Susan Wells, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Private Bag 92019, Auckland 1. Fax: (09) 373 7494; email: s.wells@auckland.ac.nz

**References:**


### Appendix 1. Target population criteria for CVD risk assessment

<table>
<thead>
<tr>
<th>RECOMMENDATIONS: WHO SHOULD BE ASSESSED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular risk assessments are recommended:</td>
</tr>
<tr>
<td>• from the age of 45 years for asymptomatic men without other known risk factors</td>
</tr>
<tr>
<td>• from the age of 55 years for asymptomatic women without other known risk factors.</td>
</tr>
<tr>
<td>Cardiovascular risk assessments are recommended 10 years earlier for Māori (from the age of 35 years for men and 45 years for women).</td>
</tr>
<tr>
<td>Cardiovascular risk assessments are recommended 10 years earlier for Pacific peoples and people from the Indian subcontinent (from the age of 35 years for men and 45 years for women).</td>
</tr>
<tr>
<td>Cardiovascular risk assessments are recommended annually from the time of diagnosis for people with diabetes.</td>
</tr>
<tr>
<td>Cardiovascular risk assessments are recommended:</td>
</tr>
<tr>
<td>• from the age of 35 years for men with other known cardiovascular risk factors or at high risk of developing diabetes</td>
</tr>
<tr>
<td>• from the age of 45 years for women with other known cardiovascular risk factors or at high risk of developing diabetes.</td>
</tr>
<tr>
<td>These people will have one or more of the following risk factors:</td>
</tr>
<tr>
<td>• family history of premature cardiovascular disease in a first-degree male relative (parent or sibling) under 55 years or female relative under 65 years</td>
</tr>
<tr>
<td>• family history of diabetes in a first-degree relative (parent or sibling)</td>
</tr>
<tr>
<td>• personal history of gestational diabetes</td>
</tr>
<tr>
<td>• personal history of polycystic ovary syndrome</td>
</tr>
<tr>
<td>• personal history of current or recent smoking</td>
</tr>
<tr>
<td>• prior blood pressure of more than 160/95 mm Hg*</td>
</tr>
<tr>
<td>• prior 1:CHDL ratio of more than 7*</td>
</tr>
<tr>
<td>• known IGT or IFG (see Table 22)</td>
</tr>
<tr>
<td>• obesity (BMI ≥30*) or truncal obesity (waist circumference ≥100 cm* in men or ≥90 cm* in women).</td>
</tr>
</tbody>
</table>

**Source:** New Zealand Guidelines Group. The assessment and management of cardiovascular risk; 2003 (reference #3).
Oral angioedema secondary to ACE inhibitors, a frequently overlooked association: case report and review

Miriam Hurst, Marianne Empson

Angioedema is a common problem; in clinical practice it is usually either idiopathic or secondary to allergens or medications. Angiotensin converting enzyme (ACE) inhibitors and nonsteroidal anti-inflammatory medications (NSAIDs) are the most common medications responsible for angioedema. ACE inhibitor-induced angioedema can occur at any stage of treatment and is potentially life-threatening. However, this condition is under-recognised, and many patients who present to immunology outpatients clinics with typical histories of ACE inhibitor-induced angioedema are still receiving these medications. We present an illustrative case of ACE inhibitor-induced angioedema from Auckland, New Zealand, followed by a discussion aimed at helping doctors recognise and manage this association.

Case report

A 71-year-old European woman was referred to Auckland City Hospital’s Immunology Clinic for investigation of angioedema. Some months earlier, she woke with tongue swelling that impaired her speech and made breathing difficult. An ambulance was called and she received three doses of intramuscular adrenaline but with no improvement.

At the emergency department she was given nebulised adrenaline, steroids, and antihistamines, with gradual resolution of her symptoms, and she was observed in intensive care overnight. She was discharged with a prescription for an EpiPen® (a self-injecting adrenaline device, DEY L.P., Napa, California); no other changes were made to her medication.

She also reported three milder episodes of angioedema over the preceding 2 years, one episode affecting the tongue and the other two her cheeks. No other triggers had been identified. She had been diagnosed with hypertension 3 years previously and started on cilazapril 5 mg once daily and pindolol 15 mg once daily. Other medical problems included a history of osteoarthritis, osteoporosis, eczema, and hay fever. She had no history of food or drug allergies.

ACE inhibitor-induced angioedema was thought to be the most likely diagnosis for all her episodes of angioedema. Her beta-blocker therapy could have contributed to the severity of her reaction and lack of response to adrenaline. This diagnosis was discussed with her GP and arrangements were made for her to alter her current antihypertensive medications under his guidance.

The patient was informed of her diagnosis and its implications, and instructed in the use of her EpiPen.
Causes of angioedema

Angioedema is a nonpitting oedema involving deeper layers of the cutaneous and mucosal tissues caused by allergic or nonallergic reactions.\textsuperscript{1,2} Although any part of the body may be affected, the commonest sites for skin involvement are the perioral and periorbital tissues.\textsuperscript{2} Angioedema involving the tongue and throat can be fatal due to asphyxia.\textsuperscript{1} Gastrointestinal angioedema may present with abdominal pain, nausea, and diarrhoea; other organ systems are rarely affected.\textsuperscript{1}

A good clinical history is essential in establishing the diagnosis and determining possible causes. Factors to consider include timing and duration of the episode(s), particularly in relation to any possible triggers such as foods or environmental stresses (e.g. heat, cold, exercise). Drug history, especially new medications or over the counter agents such as NSAIDs, is also important, as are any associated symptoms (such as urticaria), other diseases (autoimmune or atopic), and family history.

Angioedema can be part of an IgE-mediated allergic reaction (most commonly to foods such as peanuts, tree nuts, or shellfish; or medications such as penicillin) or non-allergic as with ACE inhibitors, most NSAID reactions, and chronic idiopathic urticaria/angioedema.\textsuperscript{3} Rare causes include congenital or acquired deficiencies in C1 esterase inhibitor (C1-INH).\textsuperscript{2} Most cases of angioedema in adult clinical practice are not IgE-mediated—a significant proportion of cases are due to ACE inhibitors.

ACE inhibitors in clinical practice

ACE inhibitors are used widely in New Zealand. According to New Zealand’s drug-buying agency, PHARMAC, in the year ending June 2004, just over 1 million prescriptions were filled for ACE inhibitors (value estimated from graph).\textsuperscript{4} The main clinical indication for ACE inhibitors is hypertension; however, ACE inhibitors are also used to treat congestive heart failure and diabetic nephropathy, and to significantly decrease morbidity and mortality after myocardial infarction.\textsuperscript{5}

Adverse effects

ACE inhibitors are generally well tolerated. Significant adverse effects include hypotension, renal impairment, and cough. Cough is a side-effect specific to ACE inhibitors, reported in 5 to 20% of patients; it is thought to result from bradykinin and/or prostaglandin accumulation.\textsuperscript{6}

Angioedema is a less common reaction that has been associated with all ACE inhibitors. Most ACE inhibitor-induced angioedema involves the tongue, pharynx, or perioral tissues. No diagnostic test exists at present; the diagnosis is made on the classical history and ingestion of the ACE inhibitor. Visceral angioedema with ACE inhibitors is rare, but has been reported, and needs to be considered in the context of recurrent abdominal pain where no other cause has been identified.\textsuperscript{7}

ACE inhibitors and angioedema

Incidence—Initial estimates of the incidence of angioedema during ACE inhibitor treatment were around 0.1%,\textsuperscript{6} although rates of up to 2% have subsequently been observed.\textsuperscript{8} Observational studies suggest a rate of 0.1% per year of treatment, resulting in an overall risk of up to 1% after 10 years of treatment.\textsuperscript{1} The majority of
reactions are in the first week of treatment but they can occur at any time; in addition, episodes of angioedema have been reported after stopping ACE inhibitor treatment.\(^1\) Although these episodes are predominantly in the first month after treatment cessation, we have observed episodes up to 3 months afterwards.

People with a history of idiopathic angioedema are at an increased risk of ACE inhibitor-induced angioedema.\(^8\) For unknown reasons, studies indicate a higher incidence in African-Americans (4.5-fold more than in white Americans).\(^9\) There are no Maori or Pacific Island data. Episodes of angioedema may also be triggered by trauma, such as intubation or endoscopy.\(^10\) Patients experiencing ACE inhibitor-associated cough are not significantly more likely to develop ACE inhibitor-associated angioedema.

One case series indicated that ACE inhibitor-associated angioedema may account for up to 40% of all angioedema presentations to the emergency department.\(^11\) Despite this, the role of ACE inhibitors may be overlooked. In one study, patients presenting with recurrent angioedema while taking ACE inhibitors had their ACE inhibitors stopped on only 3 of 14 occasions;\(^12\) in another series of 6 patients with ACE inhibitor-associated angioedema who presented on 9 occasions, ACE inhibitors were identified as a possible cause only once.\(^13\) Our clinical experience also suggests that a significant number of patients referred to us for investigation of angioedema are still on ACE inhibitors at the time of referral.

**Severity**—Studies suggest hospitalisation for ACE inhibitor-induced angioedema patients was necessary in roughly half of all cases.\(^12,14\) Intubation or tracheostomy was required in 5–16% of hospitalised patients\(^12,14,15\) and 4 deaths were reported in one early study, although 2 were thought to be unrelated to ACE inhibitor usage.\(^14\) In a United States study, a coroner’s review of 2000 autopsies from 1998 to 2000 found 7 deaths from ACE inhibitor-induced angioedema of the tongue, all in African Americans aged 51 to 65 years.\(^16\)

Failure to identify ACE inhibitors as a cause and cease treatment may be associated with increasing severity of the reaction. In one study of 82 patients with ACE inhibitor-induced angioedema, 45% required hospitalisation for their initial presentation with angioedema; however, 64% required hospitalisation for recurrent reactions, and rates of intubation also increased from 5% to 18%.\(^12\)

**Proposed mechanisms**—The exact mechanism of ACE inhibitor-associated angioedema is not yet certain. Immunological and complement-based mechanisms are thought to be unlikely, as there is no increase in IgE levels and a lack of other allergic phenomena. Significant complement or C1-INH deficiencies have not been demonstrated. Current theories focus on the effects of the ACE inhibitor on the renin-angiotensin-aldosterone system.

Normally, ACE converts angiotensin I to angiotensin II (ATII); see Figure 1. It also catalyses the breakdown of bradykinin and other vasoactive substances such as substance P.\(^1\) Blocking ACE with an ACE inhibitor decreases levels of ATII and increases levels of bradykinin, a vasoactive peptide which acts on a constitutively-expressed B2 receptor (found on smooth muscle and vascular endothelium) to cause vasodilation and increased vascular permeability. The increase in bradykinin is thought to be responsible for the angioedema seen in ACE inhibitor-induced angioedema.
Some studies have shown increased levels of bradykinin in patients with ACE-induced angioedema versus controls; however, patients with ACE dysfunction do not have increased rates of angioedema. Acute external trauma can increase concentrations of vasoactive substances and may be responsible for the connection between intubation and angioedema.

In addition to being metabolised by ACE, bradykinin is also metabolised by the specific peptidases aminopeptidase P, carboxypeptidase N, and dipeptidyl peptidase IV (DPPIV). Studies in small numbers of patients have shown lower levels of carboxypeptidase N, aminopeptidase P, or DPPIV in patients experiencing ACE inhibitor-induced angioedema, compared with controls; however, results have not been consistent across all studies.

Management of ACE inhibitor-induced angioedema

Acute attack—Angioedema sparing the airway can usually be managed conservatively with antihistamines with or without steroids and discontinuation of the causative medication. However, when the tongue and upper airway are involved, intramuscular adrenaline should be used (although its efficacy is uncertain as there are
no controlled trials) and some patients may even require an artificial airway (e.g. intubation or cricothyroidectomy).

Fresh frozen plasma has been successfully used in treating patients with severe ACE inhibitor-induced angioedema that has not responded to other treatments.\textsuperscript{21,22} C1 esterase inhibitor concentrate has also been used successfully in one case,\textsuperscript{23} although the reason for this is uncertain, as patients with ACE inhibitor-induced angioedema have normal C1 esterase inhibitor levels.

**Long term**—ACE inhibitor-induced angioedema is a class effect, so patients experiencing angioedema with a particular ACE inhibitor should not be switched to another ACE inhibitor.\textsuperscript{1,6} Calcium channel blockers and/or thiazides are appropriate as alternative antihypertensives. Beta blockers are contraindicated in the initial setting because of the risk of recurrent episodes and their antagonistic effects on adrenaline; however, they could be used once it has been established that the angioedema has not recurred after stopping the ACE inhibitor. Generally an interval of at least 6 months would be recommended to exclude the possibility of idiopathic or non-ACE inhibitor-related causes of angioedema.

Patients with other causes of angioedema may have their episodes exacerbated by concurrent ACE inhibitor usage so these should still be avoided in these patients.

Patients with a history of laryngeal oedema or life-threatening reactions should be given an EpiPen in case of recurrence and a MedicAlert® bracelet should be considered.

**Alternative medications: are AT II receptor blockers safe?**—ATII receptor blockers have been used as alternatives to ACE inhibitors in patients unable to tolerate these drugs because of cough.\textsuperscript{8} However, literature reviews indicate several cases of angioedema associated with ATII receptor blockers,\textsuperscript{8,24,25} with at least one case requiring emergency tracheotomy,\textsuperscript{25} although the overall incidence appears lower than that with ACE inhibitors. There is a predominance of cases associated with losartan, although this may represent prescribing bias.

In one paper, 6 of 19 patients with angioedema associated with ATII blockers had previous histories of ACE inhibitor-associated angioedema,\textsuperscript{24} hence suggesting that ATII blockers may not be an acceptable alternative to ACE inhibitors in patients with ACE inhibitor-induced angioedema.

A recent retrospective review of 64 consecutive patients with ACE inhibitor-related angioedema found that 2 of the 26 patients commenced on an ATII blocker had further angioedema secondary to the ATII blocker.\textsuperscript{26} The potential benefits of the ATII blocker need to be considered against the risks of further angioedema when deciding whether a trial of these medications is warranted.

**The future**

Usage of ACE inhibitors is likely to increase and consequently the number of patients experiencing ACE inhibitor-induced angioedema will also increase. Observational studies indicate that ACE inhibitors are currently significantly under-prescribed in certain patient populations, such as those who have experienced acute myocardial infarctions.\textsuperscript{5} Combination therapy with both ACE inhibitors and ATII blockers has been suggested in patients with diabetic nephropathy as a way to limit proteinuria and
delay (or prevent) disease progression;\textsuperscript{27} it is not clear how common angioedema will be with this combination.

Newer agents currently in development for treatment of hypertension and/or heart failure include omapatrilat, which inhibits both ACE and neutral peptidases; rates of angioedema with this medication are currently unknown.\textsuperscript{28} However, if the pathogenesis of ACE inhibitor-induced angioedema can be identified, it may be possible to screen patients before treatment and identify those at higher risk of this significant complication.\textsuperscript{29}

New agents such as icatibant\textsuperscript{30} (a selective bradykinin receptor B2 antagonist) and DX-88\textsuperscript{31} (a kallikrein inhibitor) are currently in clinical trials for the treatment of acute attacks of hereditary angioedema, and may in the future have a role to play in the treatment of ACE inhibitor-induced angioedema.

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**References:**


Foville’s syndrome masquerading as Wernicke’s encephalopathy

Nizam Ahmed, Asifa Riaz, Ashfaq Shuaib, Zaeem Siddiqi

Wernicke’s encephalopathy is often associated with alcohol abuse, and characterised by gait ataxia, mental confusion, and ophthalmoplegia. Circumstantial evidence in the absence of a thorough neurological evaluation can lead to misdiagnosis and potential mismanagement.

We present a case evaluated in the emergency department and suspected of Wernicke’s encephalopathy based on high blood-alcohol level and weakness of extraocular muscles. Later neurological evaluation lead to a diagnosis of pontomedullary infarct.

This case illustrates the importance of a thorough neurological examination, and the complementary role of the history and examination in establishing the diagnosis.

Case report

A 49-year-old Caucasian man with a history of chronic alcohol abuse consumed 26 fluid ounces (780 ml) of vodka the night before presentation. At midnight, he went to bed well. His wife awoke at 0230 am and noted the patient making gagging sounds and not moving his right side. He was taken to the Emergency Department at a local hospital where the casualty officer witnessed a seizure. He was subsequently transferred to the University of Alberta Hospital. The patient also had paralysis of extraocular movements and a blood alcohol level of 18 mmol/L.

Wernicke’s encephalopathy was suspected and patient was treated with diazepam and thiamine. Neurology Service was then consulted for further evaluation.

On physical examination, the patient’s vital signs were normal. He was awake and oriented, but somewhat drowsy. Left pupil was 2.5 mm and right 3.5 mm; both reactive to light. Pupillary asymmetry was more pronounced in the dark. He had a very mild ptosis on the left eyelid with accompanying anhidrosis of the left face.

He complained of double vision on left lateral gaze, and examination of extraocular muscles revealed left gaze palsy and failure of left adducting saccades. Facial sensations were normal. He had left facial weakness of lower motor neuron type. Gag reflex was absent.

Two days later, right palatal elevation was better than left. Speech was mildly dysarthric. Rest of the cranial nerve functions were preserved. There was no pronator drift. Strength was normal in both upper and lower extremities. Pinprick was diminished on the right. Vibration sense was intact. Reflexes were 3+ in both the right upper and lower extremities and 2+ in the left upper and lower extremities. Plantar reflexes were equivocal. His gait was unsteady and ataxic.

The patient subsequently underwent diffusion and perfusion-weighted magnetic resonance imaging (MRI) scan, which demonstrated an acute infarct in left posterior
mesial pons and upper medulla, left inferior cerebellar hemisphere, and a small lesion in the right inferior cerebellar hemisphere. A conventional cerebral angiogram was remarkable for complete occlusion of the left vertebral artery as well as complete filling defect of the left posterior inferior cerebellar artery.

Based on an irregular appearance of the vertebral thrombosis, vertebrobasilar dissection was considered in the differential diagnosis. Some supply was noted in the distribution of the left anterior inferior cerebellar artery. Additional abnormalities were noted in the basilar artery (“tongue of thrombus”) and left posterior cerebral artery (occluded).

We suspect that the clinical signs noted in our patient were related mainly to ischaemia of left lower median pons and lateral medulla (Figure 1).

**Figure 1. Diffusion weighted axial MRI images demonstrating (a) left posterior mesial pontine infarct, (b) left posterior lateral medullary infarct, and (c) left cerebellar infarct**

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**Discussion**

The history and physical examination were suggestive of an acute pontomedullary infarct. The high blood alcohol level in this case was a “red herring” and confounding factor. The seizure was most likely related to alcohol consumption. Part of his deficits were consistent with Foville’s syndrome;\(^2\) paralysis of conjugate gaze to the left (with paralysis of abduction on the left and adduction on right), lower motor neuron type left seventh nerve palsy, and weakness of contralateral arm and leg. However, the additional symptoms (dysarthria, dysphagia, and decreased pinprick) lead to suspicion of medullary involvement.

Figure 2 and Table 1 present an approximate neuroanatomic localisation of the structures involved in our patients.
Figure 2A. Cross section of the pons demonstrating the infarct in the left posterior ventral tegmentum (red) and area of transient ischemia (green).

Figure 2B. Cross section of the medulla demonstrating the approximate location of the infarct (red) in the left lateral medulla along with the location of the cranial nerve nuclei involved (?).
Table 1. Clinical signs with neuroanatomical localisation in our patient

<table>
<thead>
<tr>
<th>Number</th>
<th>Sign</th>
<th>Localisation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Paralysis of conjugate gaze to the left</td>
<td>Left abducens nucleus or parapontine reticular formation</td>
</tr>
<tr>
<td>2</td>
<td>Adductor palsy on the left</td>
<td>Left medial longitudinal fasciculus</td>
</tr>
<tr>
<td>3</td>
<td>Lower motor neuron type left facial weakness</td>
<td>Left seventh nerve nucleus or fascicle</td>
</tr>
<tr>
<td>4.</td>
<td>Transient right hemiparesis</td>
<td>Transient ischaemia of the uncrossed corticospinal tracts on the left</td>
</tr>
<tr>
<td>5.</td>
<td>Dysarthria and dysphagia</td>
<td>Nucleus ambiguous on the left</td>
</tr>
<tr>
<td>6.</td>
<td>Right hemisensory loss to pinprick</td>
<td>Lateral spinothalamic tract on the left</td>
</tr>
<tr>
<td>7.</td>
<td>Miosis on the left</td>
<td>Sympathetic fibres on the left</td>
</tr>
</tbody>
</table>

Crossed paralysis (ipsilateral cranial nerve palsies with contralateral motor deficits) is a useful neurological sign localising to the brainstem. Acute presentation is most typical of acute ischaemia (stroke).

Foville’s syndrome, first described by French neurologist Achille Foville in 1858, is a rare brainstem syndrome with ipsilateral lower motor neuron type facial paralysis, conjugate gaze palsy to the side of the lesion and contralateral hemiparesis. The syndrome characterises a specific neuroanatomic localisation rather than aetiology. It has been reported in association with tuberculomas, vascular occlusion from tuberculous meningitis, and posterior circulation ischaemia.

The crossed neurological signs in our patient were very characteristic of the localisation. First described in 1881, Wernicke’s encephalopathy has several clinical features that simulate brainstem ischaemia. Pathology may involve periventricular areas of the brainstem thus affecting the sixth and third nerve nuclei. Lateral gaze palsies and conjugate gaze palsies are common.

Our patient suffered a pontomedullary cerebrovascular accident (CVA), in close association with heavy alcohol consumption. Although the gaze palsy, nystagmus, dysartrhia, and ataxia in the presence of a high alcohol level and seizure could easily masquerade as Wernicke’s encephalopathy; lower motor neuron type facial palsy was not a typical feature. Furthermore, lack of response to thiamine and acute onset were more in keeping with a vascular event (Table 2).

**Conclusion**

Despite significant advances in neuroimaging, neurology continues to be a discipline where history and physical examination can provide essential clues to the localisation and possible aetiology. In the presence of confounding factors, differentiating between the presenting symptoms and coexisting conditions can help with the diagnosis.

Our case elaborates some common overlapping clinical features between Wernicke’s encephalopathy and brainstem ischaemia. It further emphasises that circumstantial evidence, although useful, should be considered in context of a broad differential diagnosis.
Table 2. Comparison of Foville’s syndrome and Wernicke’s encephalopathy against our patient

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Foville’s syndrome (from ischaemia)</th>
<th>Wernicke’s encephalopathy</th>
<th>Our patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood alcohol level</td>
<td>Normal</td>
<td>Can be elevated</td>
<td>Elevated</td>
</tr>
<tr>
<td>Onset</td>
<td>Acute</td>
<td>More gradual</td>
<td>Acute</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Can be present</td>
<td>Usually present</td>
<td>Present</td>
</tr>
<tr>
<td>Horizontal gaze palsy</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Facial paralysis</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>Absent</td>
<td>Can be present</td>
<td>Present secondary to medullary ischaemia (nucleus ambiguous)</td>
</tr>
<tr>
<td>Disturbance of consciousness</td>
<td>Absent</td>
<td>Present</td>
<td>Patient was alert and oriented despite a high blood alcohol level</td>
</tr>
<tr>
<td>Response to thiamine</td>
<td>None</td>
<td>Improvement in ocular deficits</td>
<td>None</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Structural pathology</td>
<td>Lower medial pons</td>
<td>Paraventricular regions of the thalamus and hypothalamus, mammillary bodies, periaqueductal region of the midbrain, and floor of the fourth ventricle. Superior cerebellar vermis is often involved.</td>
<td>Dorsal medial area of the left lower pons involving the sixth nerve nucleus and fascicle of the seventh nerve, lateral medulla, and left cerebellar hemisphere.</td>
</tr>
</tbody>
</table>

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References:

Interferon beta, PHARMAC, and political directives: in the best interests of people with multiple sclerosis?

Harry McNaughton, Nicola Kayes, Kathryn McPherson

Abstract
Interferon beta is prescribed for people with multiple sclerosis in an attempt to reduce the number of relapses occurring and to slow progression of disability. The current cost of the subsidy provided by PHARMAC for this drug is over NZ$5 million annually and is likely to rise. The history of funding decisions for interferon beta, the evidence for cost-effectiveness of the drug, and other possible ways of improving outcomes for people with multiple sclerosis other than this particular pharmaceutical subsidy are considered in this article. The authors conclude that the evidence for cost-effectiveness of interferon beta is not compelling, and other options need to be considered in an integrated package of health services for all people with MS in New Zealand.

Drugs
Interferon beta-1-alpha (Avonex)
Interferon beta-1-beta (Betaferon)

Indication
Relapsing remitting multiple sclerosis meeting criteria set by MSTAC (Multiple Sclerosis Treatment Advisory Committee)

Table 1. Numbers of patients and cost (NZ$) of interferon beta in 2005 (personal communication, PHARMAC, March 2006)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Patients on drug in 2005</th>
<th>Average cost per patient per year</th>
<th>Total cost in 2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avonex</td>
<td>120</td>
<td>$13,823</td>
<td>$1,658,751</td>
</tr>
<tr>
<td>Betaferon</td>
<td>244</td>
<td>$14,960</td>
<td>$3,650,261</td>
</tr>
<tr>
<td>Totals</td>
<td>364</td>
<td>$14,585</td>
<td>$5,309,012</td>
</tr>
</tbody>
</table>

Evidence for effectiveness
This remains controversial. A systematic review for the Cochrane Collaboration concluded that there was evidence for modest benefit in reducing the number of relapses (but not hospital admissions) and possibly delaying the accumulation of disability. For occurrence of exacerbations the relative risk was 0.80 (95% CI 0.73–0.88) and for progression of disease relative risk was 0.69 (95% CI 0.55–0.87) 2 years after randomisation.

The authors of that review point out that if the drop-outs from the treatment (interferon beta) arm are assumed to have progressed, which other evidence suggests
is a reasonable assumption, then any positive effect of interferon beta on progression is lost. Various authors have pointed out problems with these trials which include possible inadequate randomisation, documented loss of blinding, short follow-up periods, loss to follow-up, difficulty with interpretation of 'progression', publication bias and issues around funding of the trials by pharmaceutical companies. For example, regarding unblinding of participants, in one study, 80% of subjects in a higher dose interferon arm correctly guessed their treatment allocation.

Most neurologists in New Zealand nevertheless would be comfortable with the published evidence for effectiveness and accept that the benefits are modest on prevention of relapses and would at least hope that there was a positive effect on delaying progression of disability.

**Evidence for cost-effectiveness**

This is even more controversial. Estimates of cost-effectiveness have ranged between about £27,000 and £800,000 per QALY saved with beta-interferon. Probably the most authoritative of these was published in 2003. Chilcott et al's most optimistic figure of £42,000 per QALY saved depends on the horizon used (i.e. how long treatment with interferon beta continues) and they make a series of unsupported assumptions which tend towards favouring use of the drug.

Following an exhaustive consultative process (including two appeals from UK neurologists, the UK MS Society, and pharmaceutical manufacturers), in 2001 the National Institute for Health and Clinical Excellence (NICE) in the UK concluded that: "on the balance of their clinical and cost effectiveness neither beta interferon nor glatiramer acetate is recommended for the treatment of multiple sclerosis (MS) in the NHS in England and Wales." NICE instead proposed a system of 'risk-sharing' with pharmaceutical companies. Some authors have suggested that the evidence for effectiveness of azathioprine is as good as that for beta-interferon and at a price which is substantially lower.

**Side effects and long-term risk**

As many as 50% of patients receiving interferon beta report a constellation of 'flu-like symptoms'. More fatigue was also reported in patients receiving interferon beta than controls (17% vs 12% respectively). A range of haematological and liver enzyme abnormalities were reported statistically more frequently in interferon-treated compared to placebo-treated patients. Very long-term risk, especially related to cancer risk, is unknown.

**The New Zealand situation**

Before 1999, New Zealand patients could receive beta interferon if they were prepared to pay for it themselves. In 1999, after extensive lobbying from neurologists and the MS Society, Annette King (the then Minister of Health) announced that she would direct the Board of PHARMAC to make beta interferon available to a limited number of people with MS, with those patients being screened by a panel of neurologists. The funding cap was set at 180 people with that cap being removed in 2002.
It seems that the decision to fund interferon beta was made at least partly for political rather than clinical reasons. On the one hand, PHARMAC’s general manager, Wayne McNee was reported in the New Zealand Herald as having ‘twice turned down funding for beta-interferon because the benefit for most patients was low, relative to the cost. He said …while beta-interferon is beneficial for some patients, it is difficult to find out exactly who is going to benefit before they try it. At $20,000 per patient per year, this drug is simply too expensive to subsidise for everyone with MS, when we know the majority will not benefit significantly.’

On the other hand, the then Minister of Health, Annette King, said in her press release…we made this pledge during the election campaign, and it was a decision supported by the health select committee which heard compelling evidence that the previous government took little notice of. Multiple sclerosis is a disease with little or no treatment possible, and the Government wants to do what it can to help sufferers who can benefit from this drug treatment. Until this decision, New Zealand was one of only two countries in the world mean enough not to fund these drugs.

**Alternative approaches for patients with multiple sclerosis**

One of the criteria that PHARMAC cite for making funding decisions is: the cost-effectiveness of meeting health needs by funding pharmaceuticals, rather than by using other publicly funded health and disability support services.

Are there any other treatments available that are similarly effective, or more effective, than beta interferon and/or carry less risk and/or are likely to be more cost-effective? One candidate is activity-based exercise programmes, for which there is a systematic review including nine high-methodological-quality RCTs with 260 participants. That review demonstrated strong evidence in favour of exercise therapy compared to no exercise therapy in terms of muscle power function, exercise tolerance functions, and mobility-related activities, although not on health-related quality of life.

Moderate evidence was found for improving mood. These trials suffer from some of the same flaws as the interferon beta trials, particularly loss of blinding, small numbers, and limited follow-up. Nevertheless, with the information on activity-based interventions and MS available, one might imagine that there would be investment in physiotherapy and other rehabilitation services in the community for people with MS?

On the contrary, the Health Funding Authority Disability Support Services division drastically reduced funding for these services in the late 1990s by summarily redefining rehabilitation as not including ongoing therapy services for people with chronic and/or deteriorating conditions (which became ‘maintenance’) and withdrawing funding for these services. Community rehabilitation services run by the then Crown Health Enterprises which were responsible for managing large numbers of patients with MS were scaled back and have not recovered. This would appear to conflict with Objective 7 of the New Zealand Disability Strategy which is to ‘create long-term support systems centred on the individual.’

Whilst interferon beta may be exciting insofar as it is directed at the underlying pathology of MS, it is not a cure and many people with MS will inevitably accumulate disability, with or without this drug. An important component in the treatment of MS.
is managing the array of symptoms people with MS experience and the issues they face as a consequence of their disability.

A multidisciplinary approach has been recommended for the management of symptoms and disability, including a combination of exercise, education, professional support (e.g. occupational therapy, neuropsychology), psychosocial support, and pharmacologic intervention. Few people with MS in New Zealand currently have access to a full range of such services.

The difference in the number of patients considered eligible for interferon beta between the lifting of the funding cap in 2002 (230:180 receiving the drug plus 50 on the waiting list) and the latest PHARMAC figures for 2005 (364 patients—see Table 1) represents an increase of around 25–30 additional patients receiving interferon beta per year.

At an average cost per patient per year of $14,585 this means new funding of $365,000–$437,000 per year that PHARMAC needs to find every year. By 2010, this will be around $2 million per year more than 2005. The reason for the increase in patients receiving interferon beta is uncertain, making an assumption that there has been no increase in the incidence of MS in the last 4 years. Either the rate of people meeting the starting criteria is higher than the rate of people on the drug meeting the stopping criteria, or there has been some relaxation of entry criteria since the funding cap was lifted, or a combination of the two.

**Conclusion**

Multiple sclerosis is a relatively common neurological condition with often severe consequences for people affected by it. There is no known cure. Concerns about the quality of the research underpinning claims of effectiveness of interferon beta on both reducing relapses and slowing progression are appropriate. Nevertheless, in a situation where there is nothing definitely better to offer, it is not unreasonable for neurologists to want to provide this treatment at a reasonable cost (preferably free) to patients.

The decision to initially fund the drug in New Zealand appears to have had a political element and was not simply made on effectiveness or cost-effectiveness grounds. Little if any consideration appears to have been given to other means of improving outcomes for people with MS in New Zealand, despite this being part of PHARMAC's decision-making criteria.

The question that needs to be asked is whether the current $5 million per year spend on interferon beta for MS is a good idea or not. If there was an independent body—with representatives from the MS Society, neurologists, rehabilitation providers, and PHARMAC; and with a mandate to recommend how this money should be spent to ensure better outcomes for all people in New Zealand with MS (including the 90% or so who don't receive interferon beta)—would they recommend spending all of it on this drug? We think not.

The change to DHB structures was supposed to deliver coordinated care for people suffering disease and disability in New Zealand. The story of interferon beta reminds us that the old 'silos' approach to health funding is still alive and well.

If the projected increase in PHARMAC spending on interferon beta holds true, and even if it doesn't, PHARMAC will have some difficult decisions to make and may yet
have to follow the lead of NICE in the UK, that the funding of interferon beta does not represent value for money, whatever the political consequences of that decision.

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**References:**

Puerperal abscess of uterus—drainage—recovery

This case report was written by William Young, M.D., Wellington and published in the New Zealand Medical Journal 1906, Volume 5 (19), p38

Mrs. B., aged 23, was admitted into Hospital on the 20th September, 1905. She complained of severe abdominal pain, and stated that three days ago, whilst riding in an electric car, she was severely jolted and immediately felt great pain in the hypogastric region, the pain later extending to the navel and loins. She further stated that she had menstruated regularly.

On admission her temperature was 102° Fahr., her pulse 120, and respiration 30. The bowels had not moved for four days. A feeling of nausea was complained of, but there was no vomiting. There was some distension of the lower part of the abdomen, with pain and tenderness. A blood-stained vaginal discharge, somewhat offensive, was noticed.

Suspecting a ruptured extra-uterine pregnancy, possibly with suppuration, after consultation with several colleagues I decided to open the abdomen. However, an evacuant enema produced a copious motion, so we decided to wait till the following morning.

At night the woman, who had been living apart from her husband, admitted that she had had a miscarriage a few days previously, and that her previous story was false.

On the following morning, assisted by Drs. Ewart and Robertson, I curetted the uterus, removing portions of placenta. We found a distinct rounded swelling on the right side of the uterus, which we explored by an abdominal incision. This swelling proved to be a subperitoneal abscess of the uterine wall, near the fundus on the right side. With the usual precautions we evacuated the abscess and then stitched the uterus, close to the site of the abscess, to the edges of the lower part of the abdominal wound. Leaving in a drainage-tube we closed the rest of the abdominal wound.

The abscess cavity discharged freely, and the patient left hospital well with the wound quite healed on the 3rd November.

Remarks.—This case illustrates the unreliability of a patient’s testimony, however circumstantial it may be.

The method we adopted of draining the uterine abscess by stitching the uterus to the edges of the abdominal wound was very effective, and we think shortened convalescence.
Differentiate hepatic abscess from simple cyst

Shih-Hung Tsai, Wei-Chou Chang, Shi-Jye Chu, Chin-Pyng Wu, Ning-Chi Wang

A 47-year-old previous healthy man presented to the emergency department due to a 2-day course of fever and rigor. He denied having any systemic illness and habitual drinking or illicit drug abuse. Physical examinations were unremarkable. Laboratory data showed white blood cell count of 18,500 µL, C-reactive protein of 18.6 mg/dL, alanine aminotransferase of 76 U/L, and bilirubin of 1.6 mg/dL. The urine analysis and chest radiography were normal.

Contrast-enhanced computed tomography (CT) of the abdomen showed two cystic lesions over segment 8 and 2 respectively (Figure 1A and 1B, white arrows). To confirm the nature of these cystic lesions, magnetic resonance imaging (MRI) with gadolinium enhancement was performed.

Figure 1

Questions:

Which one is the culprit lesion and what is the diagnosis?
Diagnosis?—Hepatic abscess over segment 8

MR imaging with gadolinium enhancement in the identical levels revealed that the lesion over segment 8 was of inhomogenous hypointensity (black arrow) with rim enhancement and wedge-shaped hyperaemic change (asterisk) in fat-saturated T1 weighted image (Figure 2A and B)—thus indicating a hepatic abscess. Ultrasound-guided percutaneous aspiration obtained 4 ml of pus-like material. His blood and pus cultures subsequently yielded *Klebsiella pneumoniae*. The patient had a full recovery after being treated with a 2-week course of intravenous ceftriaxone.

Figure 2

Hyperintense mural enhancement on early gadolinium-enhanced images and the presence of peri-abscess hyperintensity (either circumferential or wedge-shaped) are characteristic findings of a pyogenic abscess in MRI. Modalities such as a diffusion-weighted image and apparent diffusion coefficient maps are able to differentiate hepatic abscesses from cystic or necrotic liver tumours.

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References:


Cannabis lung—maybe

We all know that cigarette smoking harms the lung, but what about cannabis? A recent report features a 39 year old man with heavy marijuana use and cigarette smoking, and 3 weeks of weight loss, fevers, dry cough, and right-sided pleuritic chest pain. Radiology of his lungs demonstrated “a pattern of large peripheral paraseptal bullae, which is compatible with marijuana smoking and differs to the uniformly distributed centrilobular pattern seen with standard cigarette smoking in a man of similar age and similar cigarette-smoking history.” The authors speculated on the causation of the atypical emphysema pattern. And, finally they asserted that it has been estimated that four marijuana cigarettes causes symptoms of cough and sputum and pulmonary histological changes equivalent to 20 tobacco cigarettes.

Int Med J 2006;36:270–1

Cannabis lung—maybe not

By chance, the next paper your scribe read was on the same topic. The opening gambit—“What is the evidence that cannabis smoking causes bullous lung disease and what is its association, directly or indirectly, with pneumothorax?” In their systematic search they found 10 cases in four reports where the authors have described bullous lung disease in association with cannabis smoking. Their ages were 24 to 46, nine were male and all were also tobacco smokers. They discovered one large prospective histopathological study in 241 known cannabis smokers which did not mention bullae. And a much smaller pathological report from Australia of ‘bong lung’ in 10 patients who underwent resection of bullous lung disease. And their conclusion? Cannabis smoking and emphysema are common, so the two will occur together by coincidence. Pay your money and take your choice.


Case reports of suspected adverse drug reactions—the case for and against

Some, including myself, believe they are useful as an early warning system. There is support for this view. One review in the BMJ in 2004 stated that more than half of suspected adverse drug reactions were confirmed by subsequent, more detailed research. The opposite conclusion is reached in a recent paper. The authors studied case reports of suspected adverse drug reactions published in 1997 and established whether each case report had been followed by more definitive studies. Their review included 63 suspected adverse reactions and they found that most (52/63, 83%) had not yet been subjected to further detailed evaluation. Perhaps the not yet is significant as their follow-up period was only 5 years.

BMJ 2006;332:335–8
Drug companies and the profession

“All gifts (zero dollar limit), free meals, payment for time for travel to or time at meetings, and payment for participation in online CME from drug and medical device companies to physicians should be prohibited.” A bold statement from leading United States members of our profession. In support of this view they point out, for example, that the rate of drug prescriptions by physicians increases substantially after they see sales representatives, attend company-supported symposia, or accept samples. They are also sceptical about the concept of disclosure of conflicts of interest which adorn articles in our journals. Why? Because they are seldom verified and are really only the opinion of the author—it is easier to disclose it and then proceed as though it did not exist. An ethical dilemma.

Osteoarthritis, chondroitin sulfate, and glucosamine

The dietary supplements glucosamine and chondroitin sulfate have been advocated (especially by those who retail them) as safe and effective options for the management of symptoms of osteoarthritis. Results from a multi-center, double-blind, placebo-controlled study throw some doubts on this subject. 1583 patients were randomised to receive glucosamine or chondroitin, or both, or placebo. And the results? Glucosamine and chondroitin sulfate alone or in combination did not reduce pain effectively in the overall group of patients with osteoarthritis of the knee. Safe—yes. Effective—no.
Outcome measures in the management of chlamydial genital infection in New Zealand

Available surveillance data suggests *Chlamydia trachomatis* is endemic amongst New Zealand youth. The National Screening Unit is reviewing whether a screening programme should be developed in New Zealand. Ideally, the effectiveness of any such intervention should be reflected in a reduced prevalence of pelvic inflammatory disease and associated sequelae as well as reduced further transmission of infection. However, in the short-term, more pragmatic outcome measures need to be considered.

National clinical guidelines and standards for the management of genital chlamydial infection have been developed for the UK; suggested primary outcome measures include the proportion of the positive diagnoses treated within 4 weeks and partner notification success rates. Treatment of uncomplicated infection is straightforward with the availability of single-dose azithromycin. Yet, if diagnosis does not result in immediate treatment or there are poor rates of partner notification then an increased likelihood of further transmission results, with a reduction in the impact of testing on disease incidence, and an increased risk of complications.

During March–August 2005, 303 cases (292 patients) of confirmed uncomplicated chlamydial infection were diagnosed at the Hamilton Sexual Health Clinic. Of the 292 patients, 56% were female with a median age of 21 years 5 months (15 yrs 1 mth–44 years 8 mths). Self-reported ethnicity was NZ European 45%, Maori 49%, Pacific 2%, other/not given 5%, compared to the self-reported ethnicity for all clinic visits during the same period of NZ European 60%, Maori 29%, Pacific 1%, other/not given 10%.

Of the 303 cases, 298 received appropriate treatment; 5 patients did not respond to recall efforts. Immediate treatment was given to 46%, thought likely to have chlamydial infection, either because of symptoms or as known sexual contacts. The median time to treatment for those not receiving same-day-treatment was 7 days (2–92), with 76% of all cases treated within 1 week and 96% within 4 weeks.

‘Successful’ partner notification proved difficult to quantify; patient-reported successful notification and treatment of current partners was often recorded but could not be verified if partners attended elsewhere for treatment. Reported notification and treatment of current partners was more likely than notification of casual contacts or of previous partners.

Our review affirmed appropriate management of chlamydial infection, including prompt availability of test results and an effective procedure for recalling untreated patients, but we feel time-lines could be improved. Over the last 6 months, 83% of newly-registered clinic attendees provided a mobile phone number in their ‘preferred contact number(s)’ and 53% provided a landline phone number. To improve timeliness of communicating positive test results, we have begun trialing the use of text-messaging software to supplement use of phone calls and letters.
Of note, 109 (37%) patients reported previous chlamydial infection, and 11 (4%) were diagnosed with a further episode of chlamydial infection during the 6-month review. In all 11 cases, it was more than 2 months since their previous infection, and all reported a change of partner. This highlights issues of patient education about safer sex following treatment for an STI and about the risks of subsequent infection. It also suggests that New Zealanders should be offered re-screening 3 months after treatment for chlamydia—as recommended overseas but not yet common practice in New Zealand.

There are many issues to be addressed to improve the sexual health of New Zealanders; here is yet another—ensuring timely, appropriate treatment of those with chlamydial infection as well as of their partners.

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References:

Accessed April 2006.

2. British Association for Sexual Health and HIV Clinical Effectiveness Group. Clinical effectiveness guideline for the management of Chlamydia trachomatis genital tract infection; September 2001. Available online. URL:  
Accessed April 2006.

http://www.sign.ac.uk/guidelines/fulltext/42/section7.html  
Accessed April 2006.

http://www.cdc.gov/STD/treatment/4-2002TG.htm#Chlamydia  
Accessed April 2006.
Screening for pharyngeal *Chlamydia trachomatis* in asymptomatic men who have sex with men

There has been some debate on the value of screening for chlamydia from the pharynx. Testing recommendations endorsed by the Australasian College of Sexual Health Physicians suggest all men who have had sex with another man in the previous year should be offered pharyngeal culture for gonorrhoea (as well as other testing) but do not advise pharyngeal chlamydial testing.\(^1\)

The 2002 Centers for Disease Control recommendations do include pharyngeal testing “if the patient concerned regarding exposure during fellatio or cunnilingus”.\(^2\) They recommend culture or direct fluorescent tests but do not recommend nucleic acid amplification tests (NAATs) because of limited published evaluation. In a study of 264 genitourinary clinic attenders by Winter et al the prevalence of pharyngeal chlamydia was 1.5% in 194 women and zero in men although they do not report the proportion of men who have sex with men (MSM).\(^3\)

An early study\(^4\) not using NAATs reported zero prevalence in a group of 160 men who have sex with men (MSM) and another study found 4.3% prevalence in a group of 51 MSM. Lister et al\(^5\) investigated the Roche COBAS Amplicor assay for testing of extragenital chlamydia and concluded that the use of this NAAT produced valid results.

We audited samples collected for chlamydia testing from the oropharynx from 1 February 2002 to 1 December 2003 at Christchurch Sexual Health. The NAAT used was BD Probetec, a strand displacement amplification test which has an inhibition control. These were collected from 297 clinic attenders and also included combinations of urine, urethral, rectal, and cervical samples as well. There were 18 females in this group who were negative at all sites tested. The remaining 279 males were mostly MSM.

All oropharyngeal samples were negative and no inhibition was detected. Two samples were positive from the rectal site only, 3 from urine samples only, and 1 positive from rectal and urine samples. Over this time period the total number of attenders tested for chlamydia from any sample was 4918 and, of these, 7.1% were positive.

In line with the Australian guidelines and based on this audit of a local population, we do not routinely test for oropharyngeal chlamydia in MSM.

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References:


Response to the article by Lynn et al: Older patients in the nephrology clinic—should they be referred?

Lynn, Sainsbury, and Searle—in their NZMJ article Older patients in the nephrology clinic—should they be referred? (http://www.nzma.org.nz/journal/118-1225/1728)—retrospectively reviewed 61 patients over the aged of 65 years who were referred to a nephrology clinic, usually with an elevated creatinine or reduced glomerular filtration rate (GFR). Only 50% required a change in management and only six were recommended to undergo renal biopsy. In the majority, a clinical diagnosis was possible.

The authors concluded that (as most were asymptomatic and did not warrant intensive investigation) elderly patients probably did not need referral until GFR was <30 ml/min. They raised the concern that nephrology services would be overwhelmed by patients aged over 65 if either a predicted 31,300 patients in Canterbury with a GFR <60 ml/min, or even the smaller number with a GFR <30 ml/min (930), were referred.

Since the majority of published studies showing slowing of progressive renal disease after intervention have been conducted in diabetic nephropathy—which is less prevalent in this elderly population (than amongst younger newly diagnosed end-stage chronic kidney disease [CKD])—disease prevention/amelioration is less likely to be useful in this group.

These conclusions are predictable but not appropriate. Most renal failure is asymptomatic in the absence of a systemic cause, until GFR is well below 30 ml/min. This is the reason for screening. The mean GFR in the cohort was 32 ml/min, thus suggesting appropriate or even somewhat delayed referral practice.

The implied message to general practitioners—that they should not ask nephrologists for specialist advice (with the suggestion of more guidelines to aid “appropriate referral”) even where practitioners have only limited experience of patients with renal disease—may help nephrology services, but is unhelpful to patients and the medical community. Indeed, it is out of step with New Zealand guidelines in the treatment of diabetic renal disease, and with recommendations of nephrology societies in most developed countries for renal disease in general.

Late referral is associated with higher mortality on dialysis as noted. More importantly, early referral to a nephrologist results in better preservation of renal function. Early referral also allows triaging into those requiring further investigation, and also defines those where further investigation is not appropriate. Here, identification of patients with renal failure who may benefit from erythropoietin therapy remains helpful, as the second most common cause of anaemia over age 50 is reduced GFR. Similarly, there is good evidence that both rate of progression and risk of end-stage renal failure are reduced by ACE inhibitors in patients with proteinuric non-diabetic renal disease.

The authors confuse consultative nephrology services that provide expert advice to patients and referring practitioners with provision of significant diagnostic or
therapeutic interventional services, such as renal biopsy and dialysis. As with other specialties both types of consultative service are required. If nephrology services are “overwhelmed” by the epidemic of diabetes for example, they may require expansion, rather than seeking ways to hand over consultative services to another subspecialty group or simply forcing patients to remain with less than adequately supported primary care providers.

This confusion is not uncommon and highlights how nephrology services in Australia and New Zealand have often been viewed only from the perspective of a need for intervention with renal replacement therapy. Unfortunately this approach has not only limited availability of nephrologists for consultative services, it has produced significant local de-skilling (cf. the US for example) in relation to consultative nephrology practice in a range of diseases directly related to kidney function including fluid and electrolyte disorders, hypertension, and management of acute renal failure in intensive care units. This approach now threatens the provision of good preventative medical practice in the approach to CKD.

Detailed guidelines for management of CKD stage 3 (GFR 30–60 ml/min) patients will allow primary care givers to be more effective in management. However, reduced GFR <60 ml/min remains an appropriate indication for nephrology clinic referral for diagnosis and assessment of prognosis, “even in the elderly”.

Patients with stable CKD and no requirement for intervention should be discharged or managed in partnership with the primary care giver. Late referral is simply locking the door after the horse has bolted.

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References:


Memories of Dr Charles Sorrell


Charlie was a friend and partner to members of the Herne Bay Medical Centre from the 1960s to the 80s when it strove to give the best medical service to the community.

Charlie was a brave man. Doctors of our generation lucky enough to miss the battles listened to soldiers returning from the Second World War. We knew that Charlie had been one of those courageous men who at night crawled under the wire digging out land mines before an advance. Being such a big man must have been a problem. He faced the loss of a child and wife, and his final illness, with the same patience and courage.

Charlie was a great GP, a "doctor’s doctor”. He kept up-to-date. An attentive and warm listener, he was loved by his patients who were attracted also by his honest straightforward manner.

Charlie was the best of partners. He worked hard and cheerfully, never complaining, always doing more than his share in busy times. He willingly did his nights and weekends on duty as well as extra time to help a partner or the patients of neighbouring doctors.

At practice meetings he was a rock of good sense and quickly defused tensions with his "wicked" humour. I personally felt confident that with Charlie as a partner our practice’s future was assured.

Charlie did not seek wealth or personal advancement but he was a fortunate man. His family and vocation filled his life. A skilled "do-it-yourself man", he rebuilt his first house. Though he had an eye for good cars, he did not indulge it.

There are many GPs and Specialists today who were inspired by GPs like Charlie in their student years, and his work lives on through them.

Simon Cotton
Retired GP
Grey Lynn, Auckland
Sir Brian Gerald Barratt-Boyes

Sir Brian Barratt-Boyes was born in Wellington on 13 January 1924. He was an outstanding person in so many ways; highly talented with flawless dexterity and skill as a surgeon, innovative and insightful as a scientist, a brilliant teacher, and a wonderful man. He was held in the highest regard by colleagues and patients.

His death, following a fourth cardiac bypass operation, has left colleagues with an overwhelming sense of sadness and of loss, but also wonderful memories, of times of immense joy, of immense satisfaction, of being extremely challenged, and memories of a man with enormous determination, with attention to the finest detail, of a man who had achieved so much, and of a man living life to the fullest.

Sir Brian’s life has affected the life of many others in innumerable different ways and will continue to do so in the future.

Sir Brian’s academic journey began in Wellington at Johnsonville Primary and Wellington College and followed on to Otago University where he graduated MB, ChB in 1946. He was an outstanding pianist and at one stage considered a concert career. He became a fellow of the Royal Australasian College of Surgeons in 1952 being the first to not have undergone any training outside of New Zealand and was awarded a Master of Surgery in 1962. He undertook a Fellowship at the Mayo Clinic in Cardiovascular Surgery from 1952–1955. During this time he worked in the Cardiac Catheterization Laboratory and did seminal work on measurements of normal heart pressures and cardiac outputs. It was here that he met his friend and colleague, John Kirklin.

In 1956 he held a Nuffield Travelling Scholarship at Bristol University. In 1957 he returned to New Zealand as a senior thoracic surgeon at Green Lane Hospital. In 1965 he became the surgeon in charge.

He became a Fellow of the Royal Society in 1970 and was awarded an Honorary Professor of Surgery at the University of Auckland in 1971. He was awarded an Honorary Fellowship of the American College of Surgeons in 1977, the Royal College of Surgeons in 1985, and the American College of Cardiology in 1989. He was awarded an Honorary DSc by Colorado University in 1985. He was President of the Cardiac Society of Australia and New Zealand from 1986–1987.

He received numerous Honours and awards from almost every country in the world, including from his Holiness the Pope. Last year he was awarded the Distinguished Alumni Award from the Mayo Clinic for accomplishments in medical practice, education, and research.
In 1966 he was awarded a CBE, and in 1971 he was knighted at the age of 47 for his pioneering work on replacing the aortic valve of the heart with a homograft valve. His original idea was to invert the valve so that the cusps were not in the way when it was inserted, allowing visibility for the valve ring to be sown in. His second major achievement was his perfection of the technique of hypothermia and circulatory arrest. The idea originated from a Japanese registrar who came to Green Lane Hospital for training.

Sir Brian tested the technique first in the laboratory and then in babies, allowing surgeons to operate on a still, bloodless heart. This technique changed the practice of paediatric heart surgery and opened up the treatment of many congenital heart defects that previously were untreatable. Again the results of cardiac surgery at Green Lane Hospital were among the best in the world and the Green Lane Hospital surgical results became the standard by which other cardiac centres compared their own results.

His third monumental achievement was his book coauthored with the American surgeon John Kirklin which was simply called Cardiac Surgery. This book recorded many of the results of surgery at Green Lane Hospital. The American Journal of Cardiology called it “absolutely magnificent, it is one of the very best medical books ever published, it is a classic from the beginning”. The International Journal of Cardiac Surgery said “This work is phenomenal and almost defies adequate description”. It is considered the bible for cardiac surgeons worldwide and is now in its third edition.

Many of Sir Brian’s achievements are not listed on his curriculum vitae such as training many cardiac surgeons who are now scattered worldwide saving the lives of 1000s of patients each year; critiquing and challenging other surgeons’ work; developing new cardiac units throughout Asia; and teaching junior doctors and nurses.

Sir Brian was extremely hard working, he demanded the highest standards of medical care without compromise, and he imbued a group of wonderful people with the same high standards; the surgeons, cardiologists, anaesthetists, perfusionists, pathologists, and nurses all had enormous respect for each other and there was an intense focus on teamwork and improving patient care. Huge strides were made in understanding the anatomical detail of congenital heart defects and developing new surgical techniques.

His influence worldwide was enormous. His reputation being so high that doctors at Harvard Medical School, arguably one of the most prestigious in the US where patients with complex heart disease are referred for treatment, referred a gravely ill famous patient to have surgery in New Zealand by Sir Brian, knowing that the patient would receive the very best treatment in the world.

That Sir Brian chose to live and work in New Zealand in the face of numerous offers to work overseas deserves the utmost admiration and has benefited this nation enormously. Sir Brian was indeed one of New Zealand’s favourite sons.

In 1989 at an American College of Cardiology Convocation, at which Sir Brian was awarded a very prestigious Honorary Fellowship, this citation was made using the term physician, and calling him the greatest physician of the century. The term physician was used not because they had forgotten that he was a surgeon, but because
they wanted to acknowledge his enormous contributions to medicine in general as well as his humanistic and caring attributes.

Sir Brian lived the history of cardiology and cardiac surgery, to which he had contributed so much, with himself undergoing four cardiac operations and left main coronary artery stenting. He pioneered the first use of cardiopulmonary bypass in New Zealand in 1958. The first coronary artery bypass at Green Lane Hospital was performed in 1969 and in 1974 he underwent the same operation.

In the early 1980s internal mammary artery grafting was introduced. In 1983 he underwent repeat bypass grafting using a left internal mammary graft. In the 1990s complete arterial grafting was developed and in 1997 he underwent his third bypass operation using a free right internal mammary artery graft. Recently left main artery stenting has been introduced, and in 2004 his left main coronary artery was stented. Two weeks before his death he underwent aortic and mitral valve replacement.

Sir Brian was revered by his patients and, up until his death, Sir Brian was still receiving letters from grateful former patients and he always wrote back thanking them for their kind words and wishing them all the best in the future.

Sir Brian retired from surgical practice in 1989 to his beloved farm “Green Hills” near Waiwera. He grew grapes, played tennis, worked on his book, and spent time with his family.

Sir Brian continued his research on the long-term results of valve replacement right up until he went to Cleveland for his fourth heart operation. This was not a retirement project but important, original work that would influence the care of future patients. He also continued to lecture throughout the world and to give advice about the design of new heart valves.

Sir Brian wanted to be remembered as a surgeon who helped both the young and the old. There is no doubt that he will be remembered for that, as well as for the wonderful legacy of surgeons that he has trained throughout the world; but he will also be remembered as a lovely man who had a wonderful journey and who enriched many lives. Sir Brian’s impact on young surgeons and their careers will be an ongoing legacy.

Sir Brian’s life demands respect and admiration, but it was his gentleness, his love of life, and his compassion that made colleagues and patients love him.

Sir Brian is survived by five sons (David, Mark, John, Stephen, and Simon) and his wife Sara whom he married in 1986.

Rārangi maunga, tū tonu, tū tonu
Rārangi tangata, ngaro noa, ngaro noa

You have gone,
but your mountain is everlasting

Professor Harvey White (Director of the Coronary Care and Green Lane Cardiovascular Research Unit, Green Lane Cardiovascular Service, Auckland City Hospital, Auckland) wrote this obituary.

The photograph is courtesy of Remembering and appears at their interactive memorial site entitled Always in our hearts: [http://www.remembering.co.nz/tribute2.asp?REMID=102](http://www.remembering.co.nz/tribute2.asp?REMID=102)
GRANTS AWARDED MARCH 2006

At the March meeting of the Scientific Advisory Group of the National Heart Foundation, a total of 15 limited budget grants were awarded. The awards included 3 Small Project Grants and 12 Travel Grants.

SMALL PROJECT GRANTS

Dr Naylin Bissessor
Cardiovascular Research Unit, Auckland City Hospital
Natriuretic peptides and exercise capacity in mixed valvular heart disease
$14,782 for a period of 1 year.

Dr Patrick Gladding
Cardiovascular Research Unit, Auckland City Hospital
Plavix response in coronary intervention (PRINC)
$15,000 for a period of 1 year.

Dr Hayden McRobbie
Clinical Trials Research Unit, Faculty of Medical & Health Sciences, University of Auckland
Nicotine oral pouch acceptability and efficacy trial
$14,900 for a period of 6 months.
TRAVEL GRANTS

Ms Denise Barlow
Cardiac Care Section, National Heart Foundation of NZ
13th World Conference on Tobacco OR Health, Washington DC, USA

Ms Maea Hohepa
Centre for Physical Activity & Nutrition Research, Auckland University of Technology
American College of Sports Medicine Conference, Denver, Colorado, USA

Ms Vivienne Homer
Department of Molecular Pathology, Canterbury Health Laboratories
XIV International Symposium on Atherosclerosis, Rome, Italy

Dr Rod Lea
Department of Population Environmental Health, ESR
11th International Congress of Human Genetics, Brisbane, Australia

Dr Hayden McRobbie
Clinical Trials Research Unit, Faculty of Medical & Health Sciences, University of Auckland
13th World Conference on Tobacco OR Health, Washington DC, USA

Ms Sarah Molyneux
Biochemistry Unit, Canterbury Health Laboratories
XIV International Symposium on Atherosclerosis, Rome, Italy

Dr Barry Palmer
Department of Medicine, Christchurch School of Medicine & Health Sciences, University of Otago
World Congress of Cardiology 2006, Barcelona, Spain

Dr Melinda Parnell
Department of Surgery & Anaesthesia, Wellington School of Medicine & Health Sciences, University of Otago
European Resuscitation Council Conference, Stavanger, Norway

Ms Tania Slatter
Department of Biochemistry, University of Otago
The Scandinavian Atherosclerosis Conference, Humlebaek, Denmark

Dr Martin Stiles
NHF Research Fellow, Department of Cardiology, Royal Adelaide Hospital, Australia
Heart Rhythm 2006, Boston, Massachusetts, USA
Dr Mark Wallace-Bell
National Addiction Centre, Department of Psychological Medicine, Christchurch School of Medicine & Health Sciences, University of Otago
13th World Conference on Tobacco OR Health, Washington DC, USA

Dr Robyn Whittaker
Clinical Trials Research Unit, Faculty of Medical & Health Sciences, University of Auckland
Randomised Controlled Trials Course, Oriel College, Oxford, UK
National Heart Foundation: 2006 Grant Applications


((Libraries please print out PDF above and replace this page))
Malaria control in complex emergencies: an inter-agency field handbook


Also available for free as a pdf at:
http://www.who.int/malaria/docs/ce_interagencyfhbook.pdf

Malaria remains an enormous problem causing over 1 million deaths per year globally. There is even some (but not conclusive) evidence that climate change may be exacerbating this problem in parts of the world (e.g. Lancet 2006;367:859–69).

The malaria problem also substantially overlaps geographically with the large number of complex emergencies around the world. These are situations where civilian populations are affected by war or civil strife, food shortages, and population displacement (with an estimated 135 million people affected from such emergencies in 2000).

Malaria is often an important additional cause of morbidity and mortality in these emergencies.

This recently published handbook focuses on achieving malaria control in complex emergencies—particularly during the acute phase of the emergency. The handbook aims to provide planners, policymakers, field programme managers, and medical coordinators with practical guidance on addressing the malaria control needs of local populations and displaced people. The content is appropriately detailed, comprehensive, and the whole handbook is well structured. There is very good use made of mini-case studies and diagrams.

Given that New Zealand volunteers, health workers, and military personnel regularly contribute to assisting with emergencies in developing countries, this handbook will be of potential value to such people. This is particularly so given the presence of malaria in South East Asia and in parts of the Pacific (e.g. Papua New Guinea, Solomon Islands, and Vanuatu). Indeed, given all the useful information about malaria prevention and treatment in this handbook, it may also be useful for those working in malaria-risk areas that are not involved in complex emergencies.

The attractive layout may make the purchased copy (e.g. from the WHO bookstore: http://www.who.int/bookorders/index.htm) a better choice than downloading the freely available pdf.

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