

# THE NEW ZEALAND MEDICAL JOURNAL



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# THE NEW ZEALAND MEDICAL JOURNAL



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## EDITORIALS

### Funding health services for older people

**Richard Sainsbury, Professor; Tim J Wilkinson, Associate Professor, Department of Medicine, The Princess Margaret Hospital, Christchurch.**

When Edmund Hillary first saw Mount Everest it is unlikely that he said "What a problem!" He is more likely to have said "What a challenge!" Later, after careful planning and preparation, he was able to observe, "Well, we knocked the bugger off". An ageing population is a predictable phenomenon, not only in New Zealand, but world wide. Politicians, health planners and professionals would do well to adopt the Hillary approach of viewing it as a challenge but sadly the words 'problem' and 'burden' are those that all too frequently dominate discussions on the topic and make more dramatic headlines for the news media.

The English and Scottish Parliaments have responded to the report of The Royal Commission on Long term Care of the Elderly<sup>1</sup> in different ways. The main recommendation (of 24) of the Commission was that the State should fund personal care according to need but living and housing costs should be subject to a co-payment according to means. The English Government rejected the recommendation and has adopted a narrow definition of nursing care, accepting only the cost of care provided by a registered nurse but not the cost of a care worker or health care assistant who carry out the bulk of long term personal care work. Scotland, on the other hand, will fund all long term personal care from within general taxation. A natural experiment has thus been set up and the results will be watched with great interest.

In New Zealand the draft of the 'Health of Older People Strategy' has been released (Ministry of Health draft document of consultation: Health of Older People Strategy, September 2001). This strategy sets out the Government's policy for the future direction of health and support services for older people and provides the framework for service planning, funding and delivery. There are likely to be many submissions but the document is well written and it is to be hoped that the final version achieves its purpose and does not gather dust as some earlier reports about health service planning for older people have done. The draft raises the possibilities of alternatives to hospital care for ill older people. This is a topic that is also being debated in the United Kingdom, the cradle of modern Geriatric Medicine, leading to a whole Supplement in 'Age and Ageing' one of the leading journals in the field.<sup>2</sup> Early Geriatric Medicine was 'practised' in isolated facilities geographically and philosophically separated from the general hospitals.<sup>3</sup> As a result of poor access to investigations and treatments there was much manufactured disability. This was offset, to some extent, by progress in assessment and rehabilitation and a better understanding of the common illnesses of old age. In recent years there has been accumulating evidence of the effectiveness of Geriatric Medicine including a meta-analysis of 28 controlled trials showing unequivocal benefits from geriatric evaluation and management units.<sup>4</sup> Comprehensive

geriatric assessment results in an impressive reduction in functional decline and admissions to long stay nursing care.<sup>5</sup> Our own work also shows that if older people referred for long term care undergo comprehensive assessment, treatment and rehabilitation, many can continue successful community living.<sup>6</sup> When planning new models of care and service innovations for the health care of older people it is vital therefore, that successful models which have been well evaluated, are not discarded.

The pattern of caring for older people with acute illness will inevitably change. A number of alternatives to hospital care for old people have been suggested. These include community hospitals, hospital-at-home schemes, community outreach rehabilitation, rehabilitation in rest homes or continuing care hospitals and the rather nebulous 'intermediate care' - a term coined in the United Kingdom which has a variety of definitions-but which is usually used to describe rehabilitation at venues other than a secondary care hospital. There has been limited evaluation of alternative schemes and further research, particularly in New Zealand, needs to be done. It is both irresponsible and unethical to adopt new styles of care without rigorous evaluation just because they 'sound nice' or might be thought to be cheaper. "A change from the present system must be based on the principle that old people should receive the best possible care".<sup>7</sup> Some new options are potentially positive developments but "some out-of-hospital alternatives could be undesirable with the potential for missed diagnosis, fragmented and substandard care and low expectations"<sup>7</sup> ie, a return to the bad old days. The 'intermediate care' idea requires particularly careful thought. If it is predicated on the ideas that (a) it will be cheaper because of lower overheads in long stay hospitals and (b) that it is easy to separate the 'acute phase' from the 'rehabilitation phase' of an older person's illness, then there will be some unpleasant surprises. Failure to adequately diagnose and treat remediable conditions in older people results in increased disability and a higher cost to the community. Our own work has shown the futility of trying to separate acute care and rehabilitative care in older people.<sup>8</sup> Rehabilitation of ill older people should start on day one. Older people with serious illness can present with altered presentations at the risk of being dubbed 'social admissions' not needing detailed diagnosis and treatment. The message that a call for extra 'social' support can be a marker of hidden 'medical' needs is beginning to sound like a stuck record, but it is a message that may not have been comprehended sufficiently even yet. The counter argument that normal ageing and 'social' needs shouldn't be 'medicalised' is equally relevant. Social and medical needs go hand in hand. This is one of the reasons why comprehensive geriatric assessment has been shown to

work. Teamwork, interdisciplinary communication and a holistic approach are more than catchwords. They are the essence of quality care of older people. Fragmentation of this approach by separating them out in the guise of 'intermediate care' carries real risks.

The need for research on health service innovations for older people is urgent. Such research must be robust and include randomised controlled trials. 'Before and after' studies are inadequate. Assessment of consumer preference and satisfaction, effects on carers and cost effectiveness are also required. Departments of Geriatric Medicine in New Zealand are more than able to conduct this research and venues should include provincial hospitals and rural areas as well as major cities. Investment in research is a priority given the demographic considerations, and it is likely to be well repaid. Effective services for older people benefit all sectors of the health service. Above all, tried and true services should not be abandoned but enhanced. The acid

test of diversionary schemes such as intermediate care is whether they would be acceptable to patients or clients of any age. If a 40 year old would not accept a proposed new 'service' then it is discriminatory to apply them to older people.<sup>3</sup>

**Correspondence.** Professor Richard Sainsbury, Department of Medicine, The Princess Margaret Hospital, Christchurch. Fax: (03) 337 7975; email: richard.sainsbury@chmeds.ac.nz

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## Recovery requires reconciliation

A reliable health service is one factor important to each individual's sense of security. In this week's Journal, Dr Ross Fairgray, concerned about New Zealand's Health Service, calls for recognition of the issues and a start to reconciliation (p143).

Whatever may have been achieved in the last 12 years, the cost has been too great, both financially and in the effects on relationships between staff. It is time for a new concord. The nurses' strike in Christchurch has been a summons to the nation. Morale in the health service is low, and there is difficulty in retention and recruitment of staff. Doctors and nurses do not expect unrealistic salaries, but they should be reasonably in line with other centres in New Zealand and local relativities between individuals should be fair. Although bargaining between hospital boards and staff has concentrated on salaries, of even greater importance to staff are the conditions under which they work.

The pressures to conformity and compliance in the name of economic necessity, and later to market economic theory, have been enormous. Those who resisted the more damaging changes have been excluded from playing a constructive role in the system. In many hospitals health professionals sympathetic to the "reforms" were appointed to represent professional views and this has created divisiveness and lack of trust. Much of the administrative approach has been couched in politically-correct phraseology which bears little resemblance to actual function and relationships. If we are to have an harmonious future we must again have inclusiveness and democratic participation, where all staff can contribute to planning and the development of policy.

The Minister of Health and the government need to make clear their intentions for the public health service. Privatisation of segments of the public health system was one of the aims of the "health reforms", and it is unclear whether

this remains an agenda for our politicians and Treasury. Easton (1999) wrote "...despite a public demand for a reversal of the policy direction of the 1990's... the government... was reluctant to provide adequate funding... it has continued to rely on a paradigm - and those who promoted it - which emphasised the running down of the public provision".

As pointed out by Dr Fairgray, in relation to the health services we have a double problem. The extraordinary power given to the new general management initiated by Labour was compounded by the competitive market promoted by National. Resolution of the resulting difficulties requires an acceptance and admission by both these major parties of the failure of these initiatives, and the need for the re-development of effective, inclusive systems of management and planning. Easton suggests that "failed policies can be changed by changing politicians... (but) there has to be a better way... given it is inevitable some new policies are going to fail, successful politicians are going to have to find ways of apologising and retreating."

At present the government is seeking a strategic plan from each District Health Board. How can these be developed in a realistic way if the parameters are decided in virtual isolation from the workforce? One is likely to end up with another set of tired platitudes that the public and staff have grown weary of over the last 12 years. What we really require is administration and planning of the health service based on effective functional relationships. We think that the Minister of Health and government should consider the views of Dr Fairgray in a constructive spirit.

### The Editors

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## ORIGINAL ARTICLE

**Giardia infection in Auckland and New Zealand: trends and international comparison**

M Ekramul Hoque, *HRC Training Fellow; Virginia T Hope, Senior Lecturer in Environmental Health, New Zealand Environmental and Occupational Health Research Centre (NEOH), Division of Community Health, The University of Auckland and Public Health Physician, Auckland District Health Board; Robert Scragg, Senior Lecturer in Epidemiology, Division of Community Health, The University of Auckland, Auckland.*

**Abstract**

**Aims.** To describe the epidemiological pattern of *Giardia* infection in the Auckland region and compare it with national and international patterns of *Giardia* infection.

**Methods.** Anonymised giardiasis notification data from the Auckland District Health Board for the period July 1996 to June 2000 were analysed by person, place and time. Infection rates and relative risks were calculated and compared with national and international information.

**Results.** Auckland had a significantly higher rate of *Giardia* infection than New Zealand as a whole. Infection rates, which peaked during February-May, were significantly higher in Pakeha/Europeans and Asian/others, compared with Maori/Pacificans. Adjusted notification rates were higher for residents of North Shore and Auckland cities

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than for other areas of Auckland. The crude regional and national notification rates were almost six times the rate of laboratory identification of positive isolates in the UK and four times US reported rates.

**Conclusions.** The higher rates of giardiasis observed in Auckland and New Zealand, in comparison with other developed countries, may be related to environmental or social factors. Missing ethnicity information precludes clear interpretation of variations in notification rate by ethnic group and suggests a need for improvement in data collection. There are opportunities to investigate the influence of risk factors on seasonal changes in notification rates both locally and nationally.

*Giardia* is one of the leading protozoal causes of human gastrointestinal illness. The transmission of *Giardia* is very common in specific groups and settings and varies seasonally.<sup>1-6</sup> Giardiasis is the third most commonly notified communicable disease in New Zealand and the most commonly notified potentially waterborne disease with an incidence rate of 46.6/100 000 population,<sup>7</sup> which is thought to be one of the highest among developed countries.<sup>8</sup> Nearly 2000 *Giardia* positive cases are reported in New Zealand annually, a third of which are from the Auckland region. Despite its high incidence and cost, few reports have evaluated the epidemiological pattern of giardiasis in New Zealand. Most were published before notification was required (1 July 1996) and used sporadic data collected through local health service providers or personal effort.<sup>1,9-11</sup> A review of its occurrence is well overdue.

This paper outlines the results of a review of four years of notifications from the Auckland region aimed at identifying local epidemiological trends in giardiasis notification rates in national and international contexts. The study aims to identify high risk groups and geographic patterns of infection.

**Methods**

**Data sources.** We obtained anonymised raw giardiasis data notified between July 1996 and June 2000 for the Auckland region from the Auckland District Health Board (ADHB). The DHB receives notifications of giardiasis for all three Health Districts, comprising seven local authorities (LAs) in the Auckland region of which four are city councils and three are district councils. The data include general demographic information on notified cases. National information on incident cases for the same period was collected from published reports (NZ Public Health Report). International data on the incidence of giardiasis were downloaded from overseas websites (Centers for Disease Control and Prevention, USA and Public Health Laboratory Service, UK).

**Data analysis.** Data for the Auckland region were analysed by age, gender, area of residence (LA), ethnicity and date of notification. Regional

data were compared with national and international data. Results are expressed in frequencies, rates, relative risks with their confidence intervals and levels of significance. EPI-Info 6.04d & 2000, PEPI 3.01, SAS v8.0 and MS Excel 2000 computer packages were used for statistical analysis and data presentation.

**Results**

Auckland District Health Board received 2510 *Giardia* positive case notifications between July 1996 and June 2000, an average annual infection rate of 58/100 000 population. Gender was not available for eighteen cases (<1%) who were excluded from gender analysis. The mean age ( $\pm$ SEM, years) for all cases was 27.8 $\pm$ 0.40 with females (29.7 $\pm$ 0.57) being slightly older than males (26.0 $\pm$ 0.56). Medians for females and males were similar (31 versus 29) and cases were distributed equally between the genders (50.9% versus 49.1%). A bimodal pattern of infection was observed, peaking in children under five years and in 25-44 year olds (Figure 1), who represented over 20% and 40% of cases, respectively. Children aged under five years had thrice the infection rate of the 25-44 year age group and a twelve fold higher rate than those 10-19 years after adjusting for gender, ethnicity and LA. The gender ratio was reversed among 1-4 and 25-34 year olds. Males had a significantly higher risk of infection in the 1-4 year age group (RR=1.22,95%CI 1.02-1.46;p<0.05) than their female counterparts but a lower risk in the 25-34 year group (RR=0.84,0.71-1.00;p<0.05).

Ethnicity was classified as Pakeha/European, Maori/Pacificans or Asian/others. Ethnicity was not available for a quarter of cases (24.2%). The risk of infection was significantly higher for Pakeha/Europeans (RR=5.91) and Asian/others (RR=3.10) compared to Maori/Pacificans after adjusting for age and gender (Table.1).

The risk of *Giardia* infection for the Auckland region as a whole was significantly higher (RR=1.11,1.06-1.16;p<0.0001) than for New Zealand (Figure 2). Average annual *Giardia*

infection rates (standardised and adjusted) for all LAs within Auckland were higher than the current national incidence (notifications) rate<sup>7</sup> except for Manukau City (Table 2). When biennial average rates were compared, a reduction in rates from July 1996-June 1998 to July 1998-June 2000 was observed for Papakura (-51.6%), Rodney (-27.9%), Franklin (-18.3%) and Waitakere (-4.3%), whereas increased rates were observed for North Shore (+9.6%) and Auckland (+20.7%) when adjusted for age, gender and ethnicity.

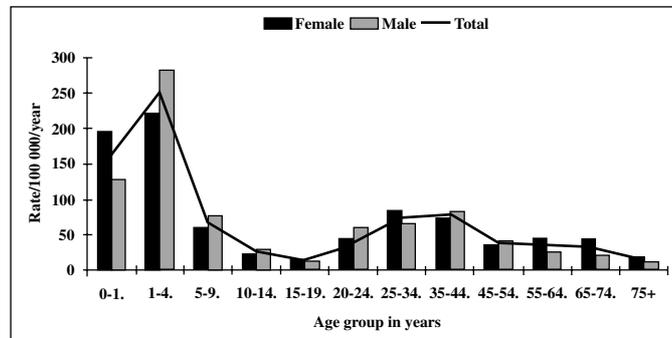


Figure 1. Rates of notified giardiasis in Auckland adjusted for gender, ethnicity and Local Authorities (July 1996-June 2000).

Seasonal patterns of giardiasis in the Auckland region and in New Zealand were almost identical, although the Auckland region had a slightly higher rate throughout (Figure 2). A similar seasonal pattern was observed in the UK. Infection/notification rates peaked in late summer and early autumn and dropped in winter. This seasonal variation was significant (Edward's test:  $\chi^2=15.0, df=2; p<0.001$ ) in the region. A comparison of two year's average annual rates of giardiasis notifications in New Zealand (July 1996-June 1997), positive laboratory tests for giardiasis in the UK (Jan 1996-Dec 1997), and reported giardiasis cases from 41 states in the US (Jan 1996-Dec 1997) showed that the New Zealand rate (55.68/100 000) was much higher than the UK (10.22/100 000) and the US (15.39/100 000) rates. The Auckland rate (57.59/100 000) was significantly higher (RR=2.07, 1.94-2.21;  $p<0.0001$ ) than that of New York (27.80/100 000) which was double that for the US as a whole.

## Discussion

Communicable disease surveillance is an essential prerequisite for health service delivery in the community. An organised surveillance system based on disease reporting by general practitioners (GPs) is in place in many countries.<sup>7,12</sup> Regular interpretation and appropriate dissemination of surveillance data and evaluation of surveillance performance are important in disease containment and prevention.<sup>13</sup> The underreporting of disease notifications has been highlighted previously<sup>14,15</sup> and this is often influenced by social and regional factors.<sup>12</sup>

This study found that more than one-third of giardiasis cases in New Zealand were reported from the Auckland region. A bimodal infection pattern peaking in the 1-4 year

and 25-44 year age groups has been documented elsewhere.<sup>4,6,10,11,16</sup> Higher rates in 1-4 year old males and in 25-44 year old females requires further investigation. These findings support a major role for person-to-person transmission of the disease and indicate a possible route of transmission between children and their parents and/or caregivers which may be facilitated by environmental exposures eg, childhood centre attendance, household contact,<sup>10,17,18</sup> or nappy handling.<sup>19</sup> The low incidence rates among teenagers are not unexpected.<sup>15</sup> The relatively low incidence in infants and a declining rate after 45 years are consistent with overseas observations.<sup>20,21</sup> The limited activities of older people and infants may reduce transmission of the disease.<sup>22</sup>

The deprivation profiles (NZDep96) for Maori and non-Maori in New Zealand suggest Maori are disadvantaged.<sup>23</sup> In this study, the group without ethnicity was evenly distributed across the LAs. However, the absence of ethnicity in 25% of cases suggests that the ethnic data should be treated with caution. In comparison with ethnic representation in the Auckland population and excluding those of unknown ethnicity, Pakeha/European were over-represented by 20%, and Maori/Pacificans and Asian/others were under-represented by 19% and 3% respectively. This is consistent with other New Zealand studies<sup>1,24</sup> but is inconsistent with the reported inverse relationship of the disease with socio-economic status,<sup>25</sup> suggesting under-diagnosis in some ethnic groups. This is supported by a substantial difference between crude (5.60) and age adjusted (12.60) relative risks for Pakeha/Europeans analysed separately. The under-representation of Maori/Pacificans and possibly of Asian/others appears to exaggerate the risk in Pakeha/Europeans.

The sustained increase in infection rates for Auckland and North Shore could be real or artifactual.<sup>9</sup> Auckland City has a high number of visitors, possibly contributing to incidence rates. The lowest infection rate, reported in Manukau City, again suggests under-reporting or effect modifications influenced by ethnicity and/or socio-economic factors. Lower infection rates in Waitakere and in Franklin are possibly due to under-diagnosis or the absence of environmental risk factors. A dramatic decrease in giardiasis in Papakura District coincides with a number of changes made by the local water supplier in the last 2-3 years which include expansion and improvement in reticulated supply (personal communication: Michael Morgan, United Water International Pty Ltd, Papakura, Auckland). Each Council in Auckland, which is primarily urban, either operates or sub-contracts its own local water network operation after it receives bulk water from Watercare Services Ltd. In contrast, Districts, which are mostly rural, are more likely to have a smaller proportion of their population on a reticulated water supply. Reticulated supplies have previously been associated with reduced risk.<sup>26</sup> However, the main factors that contributed to these changes in infection rates among LAs are yet to be determined.

The seasonal pattern of *Giardia* infection in Auckland and New Zealand is similar to that in England and Wales with a

Table 1. Relative risk of *Giardia* infection by ethnic group in the Auckland region.

Ethnicity	Female n(%)	Male n(%)	Total n(%)	Adjusted for Age & gender RR (95% CI)
Maori/Pacificans*	62 (4.9)	57 (4.7)	121 (4.8)	1.0
Pakeha/European	855 (67.4)	776 (63.4)	1637 (65.2)	5.91 (4.93-7.09) <sup>†</sup>
Asian/Others	59 (4.7)	85 (6.9)	144 (5.8)	3.10 (2.44-3.94) <sup>†</sup>
Unknown	292 (23.0)	306 (25.0)	608 (24.2)	-
All	1268 (100.0)	1224 (100.0)	2510 (100.0)	5.07 (4.24-6.07) <sup>†</sup>

\*Reference group: †include cases gender not specified; † $p<0.0001$ .

late summer and early autumn peak. This is consistent with other reports of seasonality,<sup>1,2,4-6</sup> although, giardiasis is more prevalent in the cooler months in tropical and subtropical countries.<sup>27-29</sup> A peak incidence in Auckland during February and March, continuing throughout April and May, suggests outdoor activity in these months may occur at temperatures similar to those in cooler months elsewhere.

**Table 2. Rate of giardiasis/100 000 population in Auckland region by Local Authorities (LAs)**

LAs	Maori*	European*	Asian & others*	†All ethnicity†
Manukau City	10.52	51.27	31.86	45.05
Waitakere City	7.54	47.55	45.52	48.69
Franklin District	7.72	49.20	29.12	56.52
North Shore City	11.08	59.75	30.40	60.99
Auckland City	14.89	70.62	36.63	65.08
Papakura District	20.60	78.75	20.73	75.73
Rodney District	27.18	71.22	53.06	87.64
Auckland Region†	11.77	63.73	34.12	57.59

\*Adjusted for age and gender; †adjusted for age, gender and LAs; ‡adjusted for age, gender and ethnicity; §include cases ethnicity not specified; currently giardiasis incidence rate in New Zealand is 46.6/100 000.

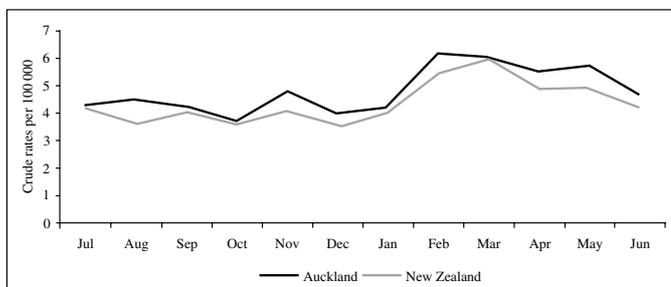


Figure 2. Average monthly giardiasis notifications in Auckland and New Zealand (July 1996-June 2000).

A four fold increased rate in Auckland, and in New Zealand, compared to the UK is surprising. If under-reporting is assumed to be at a similar level to that previously found in New Zealand, (37-50%, Hill P et al, ADHB and Hoque ME et al, NEOH – unpublished data), this difference would be only slightly reduced. A similar rate difference was also observed in comparison with the US notification records; although rates were not consistent between the states and notification is voluntary in the US where substantial under-reporting is suspected.<sup>12</sup>

This study highlights a need for improvement in reporting and in data completeness. Factors which possibly influence the higher incidence in New Zealand could be behavioural, climatic, environmental or related to the virulence or genotype of the parasites. Further study of the seasonality and situational characteristics of the infection could aid understanding of giardiasis. Analysis of national surveillance

data will provide further insight into *Giardia* epidemiology in New Zealand which may contribute to enteric infectious disease policy at a national level.<sup>30</sup>

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# Infective endocarditis – a twelve year surgical outcome series

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## Abstract

**Aim.** To review the clinical course and outcome of patients with infective endocarditis proceeding to surgical treatment in the South Island of New Zealand.

**Methods.** A retrospective review of all cases of infective endocarditis requiring cardiac surgery, excepting homograft replacement between 1989 and June 2001 was performed. All patients treated at both cardiothoracic units over this time frame in the South Island of New Zealand were included.

**Results.** A total of 29 patients, ten females and nineteen males, age range 31-79 years (mean 55) underwent surgery. 27 patients had native valve endocarditis, two infection of prosthetic valves. A variety of causative micro-organisms were isolated, and all patients received aggressive intravenous antibiotic therapy. Heart failure was the

predominant indication for surgical intervention. Fifteen patients underwent aortic, nine mitral, three combined and two replacement of infected prosthetic valves. There were five peri-operative deaths (17% mortality) and significant morbidity in a further eleven patients (38%). Of the 23 survivors available to follow-up none have recurrent endocarditis, with an average disease free survival of 35 months.

**Conclusions.** Patients who require valve surgery for endocarditis have significant peri-operative morbidity and mortality. Long-term outcome in survivors, however, is extremely good with a prognosis similar to those undergoing elective valve replacement surgery. Mycotic cerebral aneurysms are an emerging important cause of early deaths.

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Infective endocarditis remains a serious and difficult clinical management problem. Infections of prosthetic cardiac valves or infections with atypical organisms are especially difficult to cure medically and often require valve surgery. Additionally, native valve destruction by infection often necessitates emergency valve replacement. The aim of this review was to assess the clinical course of all patients recently undergoing valve surgery for infective endocarditis in the South Island of New Zealand. Any patient requiring an aortic root replacement because of extensive disease or infectious destruction was referred to Green Lane Hospital, Auckland for homograft replacement. Green Lane is the only New Zealand centre with a tissue bank performing homograft replacements. This involved less than ten patients over the twelve year period, and they are not included in this series. An assessment of other series outcomes and guidelines for valve replacement in endocarditis was also performed.

## Methods

A retrospective review of all patients with infective endocarditis undergoing cardiac surgery (excepting homografts) between January 1989 and June 2001 was conducted. Patients were identified by means of a computerised surgical audit system established at both cardio-thoracic centres in Dunedin and Christchurch Hospitals. All patients successfully treated receive six monthly or annual cardiology assessment so follow-up was readily sourced. Demographic, infectious agent, antibiotic therapy, surgical procedure and short plus long term outcomes were recorded for all cases.

## Results

29 patients underwent valve surgery for infective endocarditis. There were ten female and nineteen male patients with an age range of 31-79 years (mean 55). The majority were European with two New Zealand Maori patients. One of the European and one of the Maori patients had pre-existing rheumatic heart valvular disease. No other Polynesian patients were treated surgically, reflecting both the low population density of Polynesians in the South Island, and the low incidence of rheumatic heart disease in the south.

Seven patients were previously completely well with no comorbid conditions. In terms of valvular disease, nine had aortic stenosis (seven on the basis of a congenital bicuspid valve), and one aortic incompetence due to previous rheumatic heart disease. Five patients had abnormal mitral valves, three due to prolapse, one with moderate mitral regurgitation from childhood rheumatic fever. Two patients had significant renal impairment (baseline serum creatinine greater than 0.15 mmol/L) and another patient had dialysis dependent renal failure secondary to analgesic nephropathy. Four patients had significant pre-existent ischaemic heart disease, one had systemic lupus erythematosus (also the patient with renal failure), sarcoidosis occurred in another patient, three were hypertensive, three had mild to moderate thrombocytopenia, and one was a non-insulin dependent diabetic.

In terms of pre-existing cardiac disease, 22 patients were in New York Heart Association classification 1 or 2 and seven were in class 3. The causative micro-organisms isolated are shown in Table 1. No patient was known to have undergone dental or other bacteremia-producing procedure preceding the development of endocarditis.

Table 1. Microorganisms causing endocarditis.

Causative Micro-Organism	No.
<i>Staphylococcus aureus</i>	8
<i>Staphylococcus epidermidis</i>	2
<i>Streptococcus viridans</i>	12
Other*	6
No Organism Isolated	1

\* One case each of *Staphylococcus lugdunensis*, *Streptococcus pneumoniae* & *bovis*, *Escherichia coli*, *Corynebacterium* and *Candida albicans*.

The patient's own native valve was affected in 27 of the 29 cases and in two there was infection of a previously implanted prosthetic valve. All patients received aggressive medical therapy with intravenous antibiotics. Initial empirical therapy was based predominantly on a benzyl penicillin plus gentamycin regimen. Intravenous flucloxacillin was added if

*Staphylococcus aureus* was suspected. Empirical therapy was altered once culture and sensitivity results were to hand. Common specific antibiotics used included vancomycin, rifampicin and various gram positive spectrum first or second generation cephalosporins. The only patient with gram negative *E. coli* endocarditis was treated with ceftriaxone and ciprofloxacin with gentamycin being avoided due to moderate renal impairment. *Candida albicans* was managed medically with amphotericin B and fluconazole. This patient had undergone a previous whipples pancreaticoduodenectomy for cholangiocarcinoma of the distal common bile duct, but was not immunosuppressed. All patients received aggressive antibiotic therapy for at least six weeks, the longest course being for three months.

Indications for surgical intervention are show in Table 2. The average time interval between diagnosis, commencement of medical therapy and valve surgery was 41 days (range of 1-210 days). Six cases were surgical emergencies, 20 urgent and three elective. Fifteen patients underwent aortic, nine mitral, three combined mitral and aortic and two prosthetic valve surgery. No right sided valve surgery was performed reflecting not only its lower incidence but also the very low level of intravenous drug abuse in the South Island of New Zealand.

**Table 2. Indications for Surgery.**

Indication for Surgery	No.
Left Ventricular Failure*	25
Septic embolisation†	7
Myocardial Abscess	2
Fungal Endocarditis	1

The majority had surgery for worsening heart failure due to severe valve damage.

\*Two patients rapidly developed florid cardiogenic shock due to valve destruction.

† Two patients had associated severe *Staphylococcal* septicaemia.

Two patients underwent mitral valve repair alone, one in association with replacement of an infected aortic valve, and the remaining patients had valve replacement. 22 patients had a St Jude mechanical valve placed after native valve excision, four Medtronic valves, one a Carbomedics valve, and in one, a Bjork-Shirley valve was used. The average cross-clamp time was 75 minutes (range 36-158 minutes), and the average bypass time was 106 minutes (range 52-183 minutes). A total of four patients required pericardial patching of associated intra-cardiac abscess cavities, three needed coronary artery bypass grafting, one a pre-operative intra-aortic balloon pump placement for severe cardiogenic shock, and two pacemaker placement before surgery for complete heart block (one permanent, one temporary).

Average intensive care stay post-operatively was four (range 1-21) days. Average total hospital stay from diagnosis to discharge was 30 (range 6-79 days). There were five early (<30days) post-operative but no late post-operative deaths. Three patients died of intra-cerebral haemorrhages (two underwent emergency craniotomy). Two patients succumbed to early endocarditis recurrence with florid heart failure and overwhelming sepsis. Overall mortality was 5/29 patients (17%).

Significant morbidity (cardiac tamponade requiring re-exploration, renal failure, subarachnoid haemorrhage) occurred in 11/29 patients (38%). Minor morbidity (temporary renal impairment, pneumonia, pleural effusion, atrial fibrillation, mild left ventricular failure, pseudomembranous colitis) affected a further 7/29 (24%) patients.

One patient was an American tourist who developed *Staphylococcus aureus* aortic endocarditis whilst on holiday in New Zealand and was lost to long term follow-up.

Of the 23 surviving patients available during a mean follow-up of 38 months, none had late recurrence of endocarditis. One underwent repeat aortic valve replacement for prosthetic failure, although at the time of re-operation there was no evidence of infection. All blood and valve tissue cultures were negative. One patient who underwent mitral valve repair has moderate mitral valve regurgitation managed medically. One has moderately severe left ventricular failure due to ventricular hypokinesia and ischaemic heart disease. Four patients have subsequently died of other causes (ischaemic small bowel, colorectal cancer, ischaemic heart disease). The average overall disease free survival is 35 months, with the majority of patients being managed in good health on warfarin and no, or low-dose anti-failure medication.

## Discussion

The epidemiology of infective endocarditis has changed.<sup>1</sup> Degenerative valvular disease and mitral valve prolapse have replaced rheumatic heart disease as the most common predisposing conditions. The average age of patients has also increased. In this series three patients had pre-existing mitral prolapse, nine aortic stenosis (seven on the basis of a bicuspid valve), and two rheumatic fever leading to valvular incompetence. The average age was 55 years, the oldest being 79 years.

This series additionally demonstrates that patients who fail medical management of endocarditis and require surgical intervention have significant morbidity and mortality (in the order of 38 and 17% respectively). Clearly, these are extremely sick patients with a life threatening infection. The mortality rate is in line with other reported series.<sup>2-4</sup> Moon et al<sup>5</sup> reported an operative mortality of 18 ± 2% for 306 patients undergoing valve replacement for left sided endocarditis between 1964 and 1995. Karps' series of 135 patients with native valve endocarditis reported a 14% overall mortality, 6% in those with mild heart failure and 33% in those with severe failure.<sup>6</sup>

Neurological complications are associated with high mortality rates in infective endocarditis. Of the five deaths in our series, three were due to intracranial haemorrhage possibly the result of mycotic aneurysms. Intracranial mycotic aneurysm as a complication of infectious endocarditis occurs in about 2-5% of cases.<sup>7</sup> Although the risk of rupture is small, it is usually a catastrophic event (fatality over 80%).<sup>8</sup>

Fever, headache or focal neurological symptoms should raise clinical concern and all endocarditis patients should have a low index of suspicion for CT scan and cerebral angiography. Cerebral aneurysms remain, however, a formidable diagnostic challenge, and have an unpredictable clinical course. Most patients, as in this series, are completely asymptomatic, the first presentation often being a catastrophic intracranial haemorrhage. Two of the three cases in this series underwent emergency craniotomy, one being deemed inoperable. All three patients died.

Indications for neurosurgical intervention must be evaluated on an individual basis.<sup>9</sup> Follow-up MRI or angiography is recommended for all patients with mycotic aneurysms to assess the response to antibiotic therapy, to detect new aneurysms and to detect those aneurysms with no response or enlargement.<sup>10</sup> It remains unclear if early valve surgery in cases of large or mobile vegetations, or when systemic embolisation has occurred, will decrease the risk of mycotic cerebral aneurysms.

Valvular surgery may be indicated for infection control and/or deteriorating haemodynamics. Goals of surgery are to remove, as far as possible, all infected para-valvular tissue,

debride necrotic tissue, drain abscesses and improve mechanical function by replacement or repair of infected valves. Reparative surgery may be performed for lesions producing minor valve incompetence or for closure of small perforations using autologous pericardial tissue. Presence of all but small vegetations or major incompetence dictates the need for valve replacement.

Mechanical valves, as extensively used in this series, provide the longest term freedom from re-operation with its associated risks, although this is offset by the risks associated with lifelong anticoagulation. Fatal anticoagulant-associated bleeding has been estimated at 0.1 to 0.2 events per 100 patient years.<sup>11</sup> The incidence of non-fatal events is difficult to ascertain.

Tissue valves are acceptable for older patients with endocarditis. There is no firm information on whether tissue valves have higher resistance to subsequent infection than mechanical valves. None of the patients who had mechanical valves in this series developed reinfection. Some authors choose homografts for prosthetic valve endocarditis due to their apparent resistance to infection.<sup>12</sup> Homografts provide the additional advantage of allowing aortic root replacement where there is extensive root destruction. Green Lane Hospital is the only New Zealand centre utilising homografts in endocarditis. Patients referred to Auckland for such replacement were not included in this series.

The effectiveness of surgical treatment depends on several major parameters. Important variables include haemodynamic status, the infecting organism, the presence of myocardial abscesses (annular abscesses are more frequent with prosthetic endocarditis), and the presence of pre-operative complications such as emboli, renal failure and whether the endocarditis involves a native or prosthetic valve.<sup>13</sup>

The long term survival of patients treated surgically in this series with mechanical valve replacement was very good. Of the 23 patients available for follow-up only one had undergone further valve surgery and none had documented endocarditis recurrence. Of the 23 original patients, nineteen (83%) remain alive and well, and four have died of other causes. Surgery for life threatening endocarditis is associated with initially high

peri-operative morbidity and mortality, but long-term outcome in survivors is extremely good. Such patients probably have a lower incidence of endocarditis recurrence and better functional status if treated surgically rather than medically.<sup>14</sup> Others<sup>6</sup> have certainly documented this although the present series cannot draw strong conclusions in this regard. Surgical treatment is required in up to 30-40% of patients in whom valve function deteriorates during medical treatment.<sup>4,15,16</sup>

The absolute indications for surgery in infective endocarditis remain moderate or severe heart failure, unstable prosthesis or major systemic emboli, uncontrolled sepsis and fungal endocarditis.<sup>14</sup> Heart failure is the most common cause of death in patients with infective endocarditis and is the prime indication for valve replacement. Initial morbidity and mortality is high, but survivors have a similar prognosis to those undergoing elective valve replacement surgery.

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## The physical examination – an anachronistic practice?

Where would medicine be without the physical examination? Along with taking the medical history, examining a patient is at the very core of the practice of medicine. The techniques used link us to our past giants: Auenbrugger, who introduced the art of percussing the chest; Laennec, who pioneered auscultation with his rudimentary stethoscope; and Osler, whose acclaimed teaching of the art of observation in medicine reinforced the central role of the physical examination.

But has the ascendancy of techno-medicine rendered the physical examination anachronistic? As William J Mayo noted in the last century: “Sometimes I wonder whether today we take sufficient care to make a thorough physical examination before our patient starts off on the round of the laboratories, which have become so necessary that oftentimes we do not fully appreciate the value of our five senses in estimating the condition of the patient.”

In a recent essay, *Must doctors still examine patients?*, the US physician Colin Phoon writes: “If physical examination indeed serves primarily as a diagnostic tool, then it is theoretically possible to replace the examination with technology ...” But he also remarks that there is a growing disenchantment by patients with technomedicine that reveals the innermost secrets of their bodies, and argues that “... most patients expect a ‘laying on of the hands’ by a physician ... and it is likely that most physicians are appreciated for spending the time to perform a careful and thorough physical examination of their patients.”

Machines distance people, whereas patients instinctively value a sense of connectedness. If doctors wish to be more than robotic gatekeepers to machines, they will need to preserve the humanity in their practice and still examine their patients.

Martin B Van Der Weyden. *Med J Aust* 2001; 175: 449.

# Social class mortality differences in Maori and non-Maori men aged 15-64 during the last two decades

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## Abstract

**Aims.** This investigation uses data from 1996-97 to update previous studies of social class mortality differences in Maori and non-Maori New Zealand men aged 15-64 years.

**Methods.** Numerator data were obtained from the national death registrations and denominator data were from the 1976, 1986 and 1996 censi. For each social class, age standardised death rates in Maori and non-Maori men were calculated for amenable, non-amenable and all causes of mortality.

**Results.** Maori male mortality was significantly higher than non-Maori mortality in each social class and for the total population for amenable (overall RR = 5.3(CI = 4.0-6.9)), non-amenable (overall RR = 2.4(2.2-2.6)) and all causes of

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mortality (overall RR = 2.4(2.3-2.6)). The social class mortality differences within Maori (relative index of inequality was 3.3) were markedly greater than non-Maori class differences (RII = 1.5).

**Conclusions.** The persistently high Maori mortality rates, when controlled for social class, indicate that the poor state of Maori health cannot be explained solely by relative socioeconomic disadvantage. The high Maori rate of potentially preventable deaths indicates that the health sector is still not meeting the serious health needs of many Maori. The social class mortality gradient within Maori underlines the need to address disparities within Maori.

The disadvantaged health profile of Maori in comparison with non-Maori has been well established across a number of health indicators.<sup>1,3</sup> Previous studies have investigated the social class mortality differences in Maori and non-Maori males aged 15-64 years for the time periods 1975-1977<sup>4,5</sup> and 1985-1987.<sup>6</sup> These studies reported a trend for decreasing mortality for both Maori and non-Maori, but with continued strong social class mortality differences within each ethnic group.

New Zealand has undergone significant change since the period covered by our previous analyses.<sup>6</sup> The combination of major macroeconomic reform, health sector restructuring, Maori health and social service development as well as an increased recognition of the need of mainstream health providers to be responsive to Maori health needs may have had an impact upon Maori health status. The means of classifying both ethnicity and occupations have also changed over this period.

This paper updates these previous studies for the period 1996 - 97 by applying the revised methods of ethnic and occupational classification to investigate social class differences in Maori and non-Maori male mortality.

## Methods

The methods used in this study were similar to those used in the previous studies of male mortality for the time periods 1975 to 1977<sup>4,5</sup> and 1985 to 1987.<sup>6</sup> The current study involved an analysis of all deaths in New Zealand men aged 15-64 years during 1996 to 1997, as well as a reanalysis of male mortality data for 1975-1977 and 1985-1987. The focus on this age group allows the use of occupationally based social class codings. It also means that the studies are examining premature death, as life expectancy for both Maori and non-Maori has been above 64 years for almost all of this time period.<sup>7</sup> The methodology for analyses of the data from the earlier two time periods has been described previously.<sup>4,6</sup> Ethnic-specific analysis of the most recent data was limited to a two-year time period as the ethnicity definitions changed in September 1995,<sup>8</sup> making ethnic specific mortality data for that year unusable.<sup>9</sup> Numerator data (on deaths of men aged 15-64 years during 1996 and 1997) were obtained from the New Zealand Health Information Service (NZHIS). The denominator data for all men aged 15-64 years were obtained from Statistics New Zealand using data from the 1996 Census. As in the previous studies,<sup>4,6</sup> women had to be excluded as the information on occupation contained on the women's death certificates was inadequate for social class categorisation (strictly speaking, these should be referred to as occupational classes rather than

social classes, but we have retained the latter term for consistency with previous publications).

For this study we categorised deaths as either Maori or non-Maori. For the period 1996-97 the standard 'Maori ethnic group' definition was applied<sup>10</sup> - namely a deceased was identified as Maori if Maori was listed as one of their (or their only) ethnic groups. Similarly, denominator figures were based upon Census 1996 data for the Maori ethnic group. For 1985-87 the numerator data (ie. for those deceased) were for those with 50% or more Maori ancestry, with the denominator data being sourced from the Sole Maori population from the 1986 Census. For 1975-77 Maori ethnicity was defined in both the numerator and denominator figures as being those individuals with 50% or more Maori ancestry. This change in definitions means that the Maori mortality rates cannot be directly compared over time.

The previous analyses<sup>4,6</sup> were based on the Elley-Irving socioeconomic scale with individuals allocated to a social class on the basis of the 1968 New Zealand Standard Classification of Occupations (NZSCO).<sup>11</sup> It was not possible to analyse the 1996-1997 mortality rates using the Elley-Irving classification as both the 1996 census data and the 1996-1997 mortality data use the 1990 NZSCO system of occupational coding,<sup>12</sup> and there is no straightforward mapping between the coding systems.<sup>13</sup> Therefore the 1996-97 data were analysed using the recently developed New Zealand Socioeconomic Index (NZSEI)<sup>14</sup> which utilises the NZSCO-90 codes. Thus, both the denominator (1996 census) and numerator (deaths during 1996-1997) data were grouped into the standard six NZSEI categories using the NZSCO codes. Previous analysis<sup>14</sup> has shown that the NZSCO-90 codes contain insufficient information for valid social class classification of farmers and agricultural workers, so they were excluded from the analysis. For this study classes 1 and 2 were combined due to the low numbers of Maori men in class one. The data on the occupationally unclassifiable category (which includes students, retired persons, invalids, and the unemployed) were also excluded from the class-specific analysis as the different approaches of the census (which records current occupation) and the death certificate (which records the current or most recent occupation) make interpreting the results problematic.

SAS and Excel were used to calculate directly age-standardised mortality rates per 100 000 person-years for Maori and non-Maori for all deaths, each social class grouping and all classes combined. All data were classified into five-year age groups and Segi's world population<sup>15</sup> was used as the standard population. Maori/non-Maori relative risks were calculated for all deaths, each social class grouping and all classes combined.

The social class mortality trends were assessed using a weighted linear regression of the age-standardised (or age-specific) class-specific mortality rates in order to estimate the slope of the overall age-standardized social class mortality trends.<sup>16,17</sup> These mortality rates were regressed against the social class scores (in percentiles) for the midpoint of each social class

category, with the regression weighted by the inverse variance of the rate for each class. The resulting regression coefficient and intercept were then used to estimate the Relative Index of Inequality<sup>16</sup> (RII) which is the predicted relative risk for the 100th percentile (intercept + 100\*slope) against the 0th percentile (intercept only).

The rates in non-Maori were also standardized for social class (using the social class distribution of Maori in that time period as the standard) in order to estimate the proportion of the Maori excess mortality that was attributable to differences in social class distribution between Maori and non-Maori. These analyses were also conducted separately for 'amenable' and 'non-amenable' causes of mortality. The concept of amenable mortality involves 'sentinel health events' which are judged to be largely avoidable given appropriate medical intervention. The list of diseases used to define amenable mortality in the current study was the same as used in previous analyses<sup>4,6</sup> and comprises: hypertensive heart disease; tuberculosis; asthma; chronic rheumatic heart disease; appendicitis; acute respiratory disease; bacterial infections; Hodgkin's disease; abdominal hernias; acute and chronic cholecystitis; deficiency anaemias; pneumonia and bronchitis.

## Results

Table 1 shows the social class distribution based upon the 1976, 1986 and 1996 censuses. The percentage unclassifiable by social class increased during this period especially in Maori. The percentage of deaths during 1996-97 that were unclassifiable for social class was 33.1% for Maori and 19.3% for non-Maori males.

**Table 1. Social class distribution of Maori and non-Maori New Zealand males aged 15-64 years in 1976, 1986 and 1996.**

Social Class	1976 Census		1986 Census		1996 Census	
	Elley-Irving (E-I)		Elley-Irving (E-I)		NZSEI	
	Maori	Non-Maori	Maori	Non-Maori	Maori	Non-Maori
1	0.8	5.2	0.8	5.6	1.4	3.3
2	2.0	8.9	2.1	10.6	5.6	12.1
3	7.1	21.5	8.4	19.9	7.3	14.1
4	16.7	25.2	16.8	23.2	13.0	16.0
5	36.0	18.7	29.8	15.8	17.7	12.7
6	17.3	7.3	14.9	6.8	8.2	5.6
Unclassifiable	20.2	13.2	27.3	18.1	41.4	28.5
Farmer	-	-	-	-	5.4	7.7
Total	100%	100%	100%	100%	100%	100%

As shown in Table 2, in 1996-97 the Maori mortality rate for all men aged 15-64 years was 721 per 100 000 person years compared with 297 for non-Maori, giving a relative risk for Maori of 2.4 (CI = 2.3-2.6). The Maori relative risk was significantly greater than one for all males, all individual class groupings and for all classes combined in 1996-97. Table 2 also indicates that in 1996-97 the social class mortality differences were greater for Maori than non-Maori in both absolute and relative terms. For Maori the RII was 3.3 while that for non-Maori was 1.5.

Mortality rates standardised for social class (Table 2) indicate that in 1996-97 only 4% (435-409) of the excess Maori mortality (1119-409 = 710) was attributable to social class differences between Maori and non-Maori. Also shown on Table 2 are the corresponding findings for the time periods 1975-77 and 1985-87, with the social class groupings based upon the Elley-Irving scale.

The findings for amenable mortality are shown in Table 3. For 1996-97, the Maori relative risk for mortality from amenable causes is 5.3 (CI = 4.0-6.9)—with adjustment for social class only reducing this excess to 4.8 (CI = 3.4-5.8). However, although the absolute rate of mortality from amenable causes is much greater in Maori than in non-Maori, the relative social class differences were similar within each ethnic group with a RII of 2.5 for Maori and 3.6 for non-Maori.

As shown on Table 4, the findings for non-amenable mortality in 1996-97 indicate a Maori relative risk of 2.4 with adjustment for social class altering this to 2.6 (CI = 2.2-2.6).

There is a stronger class gradient in Maori non-amenable mortality (RII = 3.3) than non-Maori (RII = 1.4).

Figure 1 depicts the Maori and non-Maori male mortality rates for each social class grouping for the 1996-97 period. Figure 2 illustrates the Maori and non-Maori male mortality rates from amenable causes for the same time period.

## Discussion

There are methodological problems in this type of study that have been outlined in previous papers<sup>4,6,13</sup> and in the wider literature.<sup>18</sup> One issue is the potential for misreporting of occupation on the death certificate and in the Census, especially as the death certificate requires the current or most recent occupation while the Census requires the current occupation. This numerator/denominator bias may impact upon the mortality rates for specific social classes, especially class 1 (A Blakely, personal communication), but its overall impact on the social class gradient appears to be negligible.<sup>13</sup> Its impact in this study is further minimised by the aggregation of data for classes 1 and 2.

A further issue with this type of study is the misclassification of ethnicity in both the Census and in the mortality data. The 1995 Birth Deaths and Marriages Act has resulted in consistency in ethnicity questions between the death certificate and the Census as well as a 97% completion of the ethnicity field on the death certificates.<sup>9</sup> However, the death certificate ethnicity question may be being interpreted as referring to sole Maori ethnicity rather than Maori ethnic group.<sup>19</sup> If true, this would mean that Maori deaths remain under-reported and that the Maori mortality rates reported here are still underestimates.

A third issue is that the use of different social classification systems in 1985-87 and 1996-97 has made the results for the last two periods not directly comparable. This is not crucial in itself, as the RII takes into account the relative proportions in the different social class categories and this can be used to compare findings using different scales provided that both scales are monotonic. However, the combination of the different ethnic and social classifications, as well as the increase in unclassifiable data means the populations under study in the most recent time period are different to those of the earlier time periods. Therefore it is not possible to say if mortality rates or differentials have changed, but just whether they persist.

The reduction of detail in the more recent occupational codes is likely to increase the misclassification of occupation and has resulted in the exclusion of farmers from the analysis. As the misclassification would affect non-differentially both the numerator and denominator data for 1996-97, the effect of the occupational misclassification would be to reduce the class mortality gradient.

A further issue with this study is the high and increasing proportion of occupationally unclassifiable data. The increase is presumably due in part to the striking increase in unemployment and joblessness over this period (that had a disproportionate impact upon Maori)<sup>20,21</sup> as well as increasing participation in post-compulsory education. The difference between the classifiability of the denominator data and the numerator data results from different methods of asking about occupation in the Census and death certificates. This difference in questions also precludes any analysis of information about unemployed workers.

As the highest rates of unemployment amongst Maori and non-Maori are in the low skilled occupations,<sup>22</sup> the high rate of unclassifiability would be likely to reduce the mortality rates for the less advantaged social classes and hence the overall class mortality gradient. As the Maori workforce has proportionately more low skilled workers than the non-

**Table 2. Age-standardized mortality per 100 000 person-years and age-standardized rate ratios during 1975-1977, 1985-1987 and 1996-1997 in New Zealand males aged 15-64 years, by Elley Irving (E-I) or NZSEI socioeconomic category.**

Category	1975-1977 (E-I)			1985-1987 (E-I)			1996-1997 (NZSEI)			95% CI
	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	
Pooled Rate (all men aged 15-64)	864	485	1.8	622	418	1.5	721	297	2.4	2.3-2.6
Class 1-2	399	455	0.9	488	371	1.3	559	339	1.6	1.3-2.1
Class 3	859	490	1.8	600	380	1.6	841	410	2.1	1.7-2.5
Class 4	709	531	1.3	649	501	1.3	1012	457	2.2	1.9-2.6
Class 5	880	633	1.4	608	627	1.0	1455	466	3.1	2.8-3.5
Class 6	1399	785	1.8	1061	571	1.9	1512	440	3.4	2.9-4.1
Pooled rate (classes 1-6)	953	548	1.7	689	470	1.5	1119	409	2.7	2.6-2.9
Ratio class 6 to class 1-2	3.5	1.7		2.2	1.5		2.7	1.3		
Intercept	516	408		487	312		536	337		
Slope	8.09	2.84		3.95	3.22		12.24	1.59		
Relative index of inequality	2.6	1.7		1.8	2.0		3.3	1.5		
Pooled rate classes 1-6 standardised for social class	953	626	1.5	689	548	1.3	1119	435	2.6	2.4-2.7
Proportion of Maori excess due to social class	19%			36%			4%			

**Table 3. Age-standardized mortality per 100 000 person-years and age-standardized rate ratios during 1975-1977, 1985-1987 and 1996-1997 in New Zealand males aged 15-64 years, by Elley Irving (E-I) or NZSEI socioeconomic category for amenable causes of mortality.**

Category	1975-1977 (E-I)			1985-1987 (E-I)			1996-1997 (NZSEI)			95% CI
	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	
Pooled Rate (all men aged 15-64)	100	21	4.7	46	16	2.8	38	7	5.3	4.0-6.9
Class 1-2	12	17	0.7	43	9	4.6	22	6	3.9	1.5-9.9
Class 3	44	16	2.7	46	13	3.6	58	5	11.8	4.8-28.2
Class 4	47	21	2.2	33	17	1.9	42	11	3.8	1.6-9.2
Class 5	90	28	3.3	41	29	1.4	46	11	4.3	2.2-8.4
Class 6	219	46	4.8	102	23	4.3	72	14	5.2	2.2-12.5
Pooled rate (classes 1-6)	102	22	4.6	51	17	3.1	45	9	5.3	3.7-7.6
Ratio class 6 to class 1-2	18.3	2.7		2.4	2.6		3.2	2.4		
Intercept	8.8	11.7		24.2	6.5		25.8	3.7		
Slope	1.76	0.21		0.46	0.21		0.39	0.09		
Relative index of inequality	21.0	2.8		2.9	4.2		2.5	3.6		
Pooled rate classes 1-6 standardised for social class distribution of Maori	102	29	3.5	51	22	2.3	45	10	4.5	3.4-5.8

Maori workforce<sup>22</sup> and proportionally more unemployment<sup>23</sup> the Maori class gradient would be reduced to a greater extent than the non-Maori gradient.

Further issues are associated with the use of class-based concepts in the analysis of ethnic specific statistics. First, ethnicity and social class are not completely independent variables. The NZSEI is based on an algorithm relating age, education and income for each occupational code,<sup>14</sup> and the Maori population is disproportionately represented in the groups with lower education,<sup>23</sup> income and occupation (by NZSEI)<sup>14</sup> outcomes. Secondly, if social class is intended as an indicator of 'social status', then the concept of occupational

derived class is less applicable in Maori society, where social status is more a result of social roles and whakapapa than occupation. Furthermore an individualised measure of social status can overlook the more collective nature of Maori society<sup>24</sup> and the possibility that larger families and broader social obligations may result in individual income being a poor proxy for actual disposable income.<sup>25</sup> The NZSEI is derived from Census based information upon age, education and income levels in various occupations. While these issues mentioned above may limit the utility of social class measures in understanding variations in Maori health and social outcomes, the use of an occupationally-based index provides

**Table 4. Age-standardized mortality per 100 000 person-years and age-standardized rate ratios during 1975-1977, 1985-1987 and 1996-1997 in New Zealand males aged 15-64 years, by Elley Irving (E-I) or NZSEI socioeconomic category for non-amenable causes of mortality.**

Category	1975-1977 (E-I)			1985-1987 (E-I)			1996-1997 (NZSEI)			95% CI
	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	Maori	Non-Maori	Ratio	
Pooled Rate (all men aged 15-64)	764	464	1.7	575	402	1.4	683	289	2.4	2.2-2.6
Class 1-2	387	438	0.9	445	362	1.2	537	334	1.6	1.3-2.1
Class 3	815	474	1.7	554	367	1.5	783	405	2.1	1.6-2.6
Class 4	662	510	1.3	616	484	1.3	969	446	2.2	1.8-2.6
Class 5	790	605	1.3	567	598	1.0	1408	456	3.1	2.7-3.5
Class 6	1180	739	1.6	959	548	1.8	1440	426	3.4	2.8-4.1
Pooled rate (classes 1-6)	851	526	1.6	638	532	1.2	1074	401	2.7	2.5-2.9
Ratio class 6 to class 1-2	3.0	1.7		2.2	1.5		2.7	1.3		
Intercept	514	397		461	306		507	334		
Slope	6.17	2.62		3.49	3.01		11.8	1.49		
Relative index of inequality	2.2	1.7		1.8	2.0		3.3	1.4		
Pooled rate classes 1-6 standardised for social class distribution of Maori	851	597	1.4	638	535	1.2	1074	426	2.6	2.3-2.7

a useful proxy to compare the combined effect on different populations of these key socio-economic factors operating at an individual level.

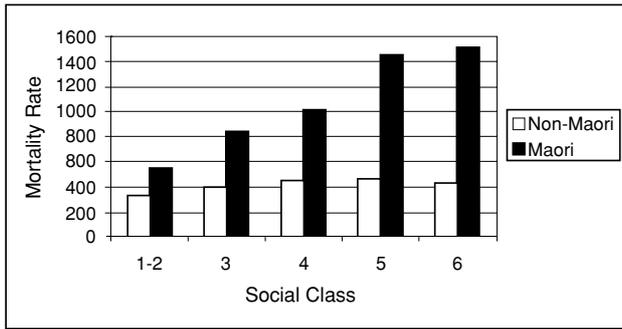


Figure 1. New Zealand male age-standardised mortality rates (per 100 000 person years) by NZSEI occupationally defined social class and ethnicity 1996-1997.

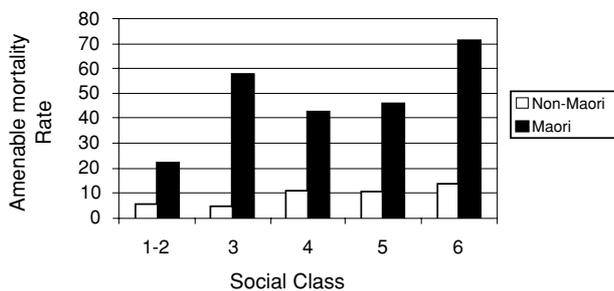


Figure 2. New Zealand male age-standardised amenable mortality rates (per 100 000 person years) by NZSEI occupationally defined social class and ethnicity 1996-1997.

While there are a range of variables impacting on the validity of the data and methods used in this study, there are several interesting aspects to the findings. Firstly, Maori males are more than twice as likely to die prematurely than non-Maori, despite Ministry of Health publications highlighting Maori health disparities since 1960.<sup>26</sup>

Secondly, Maori have significantly higher mortality rates than non-Maori for each class grouping as well as for all men combined. The mortality rates for Maori in the highest occupational grouping were higher than those of non-Maori in the lowest occupational grouping for amenable, non-amenable and all causes of mortality. Maori mortality remains higher than that of non-Maori even when the mortality rates are adjusted for social class. This indicates that the higher Maori mortality is more than just a result of Maori being over-represented in the lower social classes as suggested by a recent critic of Maori focussed social policies.<sup>27</sup> (Chapple S. Maori Socio-economic disparities: unpublished observations). Some of this residual difference may be due to the impact of non-individualised components of socio-economic position (such as housing, number of dependents and total household disposable income) or to socio-economic factors that are not measured adequately by occupation-based class scales.<sup>28</sup> The excess mortality may also reflect a range of socio-economic factors including the impact of downward social mobility (in addition to actual position) on health outcomes, especially for specific age groups.<sup>29</sup>

Thirdly, the social class mortality differences were greater within Maori than within non-Maori. This indicates that high Maori mortality is not inevitable and the diversity of health outcomes within Maori. The social class mortality differences within Maori could be due in part to the negative effects of the economic restructuring of the last decade that

have fallen disproportionately on certain groups within the Maori population.<sup>3,21</sup>

Most importantly, much of the higher Maori mortality is preventable despite the relatively restricted definition of amenable used in this study.<sup>3,7</sup> Thus, the relative risk for mortality in Maori was higher for amenable (RR=5.3) than for non-amenable (RR=2.4) mortality. On the other hand, the social class differences within Maori were about equal for amenable (RII = 2.5) and non-amenable (RII = 3.3) mortality, indicating that reducing mortality rates in Maori requires interventions with regards to both amenable and non-amenable causes of death.

The findings of this study portray a disturbing picture of persistently high levels of inequality in premature death between Maori and non-Maori as well as high levels of inequality within Maori. The high levels of Maori preventable death emphasise the need to make health policy and services more responsive to Maori health needs. Even if disparities between Maori and non-Maori are now decreasing, as has been claimed,<sup>27</sup> the continued existence of large disparities in such basic public health indicators as mortality, particularly mortality from amenable causes, indicates how far we have to go.

The persistence of inequity also reflects the fact that socio-economic differences in premature mortality are the consequences of a lifetime of exposure to the socio-economic determinants of health.<sup>30</sup> Improving the access to and suitability of health services to Maori health needs could reduce the high rates of preventable mortality amongst Maori. It will, however, do little to address the socio-economic disparities that underlie these differences and that will perpetuate health inequalities as the current population experiencing them ages.<sup>30</sup>

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## Changing patterns of hospital admissions for patients with rheumatic diseases

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### Abstract

**Aims.** To audit trends in hospital admission of patients with rheumatic diseases to a rheumatology unit.

**Methods.** Numbers of admissions for 1990-1999 were reviewed. Notes of patients admitted in 1993, 5, 7 and 9 were reviewed for principal diagnosis and other data.

**Results.** There was a 75% decline in admissions during the study period. There was a significant fall in admissions for control of active rheumatoid arthritis which declined from 53% to 21% of total admissions ( $p=0.004$ ).

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**Conclusions.** In the last decade in Otago there has been a substantial decrease in patient admissions for treatment. This was mainly due to decreased admissions for treatment of flares of rheumatoid arthritis and its complications. Determining if this was due merely to more rigorous admission criteria or improved effectiveness of treatment of rheumatoid arthritis will be important for guiding decisions on healthcare services for patients with rheumatic diseases.

In recent years we have become aware of a substantial decline in hospital admissions in the Rheumatology Service at Dunedin Hospital. This has been, for example, noticeable with respect to student teaching, with increased teaching in the outpatient setting. It has been our impression also that there have been fewer admissions for treatment of acute, uncontrolled rheumatoid arthritis. To determine if these subjective impressions were correct we decided to audit hospital admissions to our service over the last decade to document the extent of any reduction, and to identify any particular diagnostic categories that may have been especially affected.

### Methods

We reviewed the number, diagnostic category, and day stay of rheumatology inpatients in Dunedin Public Hospital over the last decade. Inpatient and day stay numbers for the decade 1990-1999 were obtained. Each admission was counted as a single event, although each event did not always represent a different person.

Inpatient and day stay patients from 1993, 1995, 1997 and 1999 were identified by NPI number and admission data, and their notes were retrospectively reviewed. Information collected included age, principal diagnosis, duration of admission, and category of admission ie, inpatient or day stay. Categories of inpatients were as described in a European audit.<sup>1</sup> Long stay patients (>28 days) were reviewed according to diagnostic categories.

Detailed data for inpatients in 1991 were not available for review regarding diagnosis although total number of admissions was present. Full admission figures were not available for 1992 and therefore could not be included.

### Results

Over the ten year period a 75% reduction in admissions was demonstrated (Figure 1). A progressive decline in inpatient numbers occurred from the early 1990's. This was continuous rather than stepwise and may be starting to plateau at the end of the 1990's.

The mean day stay ( $\pm$ SD) remained relatively stable changing from 9.72 (9.16) in 1993 to 8.34 (6.76) days in 1999 (NS, Student's t test). Prolonged day stay (>21 days) was not restricted to vascular causes or sepsis but included rheumatoid arthritis (n=8), spondyloarthritis and seronegative arthritis

(n=3), connective tissue disease (n=2), lumbar spinal stenosis (n=2) and one each of infection and vasculitis. The long stay patients' conditions showed a slow shift over the decade with eventual loss of non-inflammatory diagnoses altogether. The prolonged stay was generally a reflection of disease severity and poor admission functional status rather than actual diagnosis.

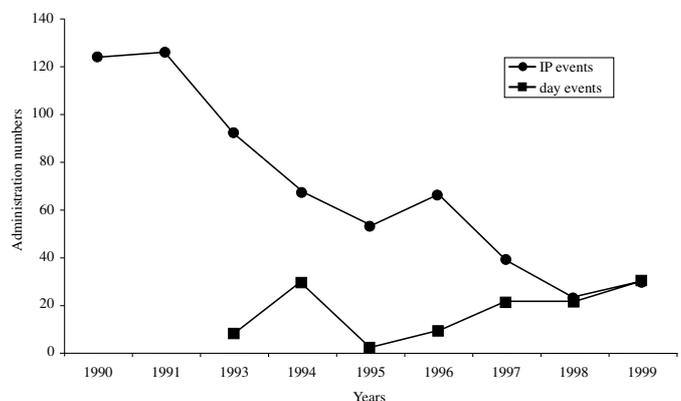


Figure 1. Change in the number of inpatients, and day-stay events 1990-1999. IP = inpatient.

As well as a decline in total admissions there was a decline in the proportion due to rheumatoid arthritis flares or complications. These were more precipitous in their decline than other diagnoses and now represent a smaller proportion of all inpatients. The reduction from 53% to 21% of total patients (Chi-squared 8.2) is highly significant ( $p=0.004$ ). The different categories of diagnosis are seen in Table 1. Some conditions such as osteoarthritis, have disappeared from the inpatients subtypes altogether.

### Discussion

A general decline in rheumatology admissions has been noted world wide and a variety of reasons have been

proposed including political health reform, changes in treatment practice and patient driven factors.<sup>2-5</sup> In New Zealand, major health 'reforms' have continued since the mid 1980's. Of interest, the introduction of the Regional Health Authority system coincided with a dip in inpatient numbers in 1993. A further reduction in 1997 may be attributable to change in the Rheumatology Unit location from General Medicine to the Musculoskeletal service, a Clinical Practice Group comprising orthopaedics and rheumatology. Under this system admissions have been to an orthopaedic ward potentially causing concern for medical patients with severe systemic illness. However, such a change would not, in itself, lead to a further reduction in admissions for control of active rheumatoid arthritis.

**Table 1. Number of inpatients and diagnostic category for the years 1993-1999. Rheumatoid arthritis shown as a percentage of the total number of admissions.**

Admission type	1993	1995	1997	1999
RA flare	29	15	7	3
RA complication	20	10	10	3
Seronegative arthritis	3	8		3
CTD	14	5	9	3
Juvenile arthritis	2	1		
OA	7			
PMR/GCA	4	5	3	3
Other	11	10	9	11
Vasculitis	2	0	1	3
RA as % of IP	53%	47%	43%	21%

RA = rheumatoid arthritis. CTD = connective tissue disease. OA = osteoarthritis. PMR = polymyalgia rheumatica. GCA = giant cell arteritis.

Reduction in admissions for control of severe active rheumatoid arthritis or its complications was the main contributor to the overall reduction in inpatient events. Our data do not allow us to determine the cause for this. There may be a simple explanation such as tightened admission policy due to changes in health administration and facilities as outlined. However, it must also be recognised that during this decade the main change in rheumatology practice has been a substantial change in the management of rheumatoid arthritis. This includes early introduction of disease

modifying agents.<sup>6-11</sup> Such treatment has demonstrable ability to reduce disability<sup>12</sup> and mortality<sup>13</sup> due to rheumatoid arthritis. The changes continue and it will be interesting to see if the recent introduction of newer agents such as leflunomide and 'biologicals' such as tumour necrosis factor blocking agents<sup>14</sup> have a further impact on admissions for patients with rheumatoid arthritis, should such agents eventually become available (with subsidies) to New Zealand patients.

In conclusion, we have demonstrated a sharp reduction in admissions to hospital in Otago in the last decade for treatment of rheumatic diseases. The main contributor was a significant fall in admissions for management of rheumatoid arthritis flares and complications. Determining if this fall has occurred in other areas, and if it is due to improved treatment of rheumatoid arthritis, is relevant to the economics of health care in New Zealand and provision of services for patients with rheumatic diseases.

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## Perfect teeth without the trauma of frequent trips to the dentist

Toothache and fillings could soon be a thing of the past. On both sides of the Atlantic, scientists are developing vaccines aimed at eliminating dental caries, one of the most common infectious diseases.

The vaccines target the bacterium *Streptococcus mutans*, which damages teeth by secreting large amounts of lactic acid that erodes tooth enamel. The bug also makes a sticky substance that helps it cling to tooth surfaces, forming a furry layer called plaque.

Martin Taubman, Daniel Smith and their team at the Forsyth Institute in Boston are developing a vaccine against *S. mutans* that they hope can be given to children aged 18 months to 3 years. "We found that's the best time to immunise," says Taubman. If toddlers fight off the bacteria before they have fully colonised the mouth, he says, the vaccine could give lifelong protection.

Rather than attack *S. mutans* directly, the vaccine targets the enzyme that makes the goo it uses to stick to teeth. Deprived of this anchor, the bugs should easily be swept away when people brush their teeth.

When adults were given an oral version of the vaccine, they produced antibodies to the bacteria, Taubman found. And rats given the vaccine as a nasal spray secreted bacteria-fighting antibodies in their saliva, where it is needed to eradicate the bugs.

New Scientist 2001, 15th Sept.

## Education to address medical error – a role for high fidelity patient simulation

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### Abstract

**Aims.** To describe and evaluate a simulation based course that emphasizes the role of teamwork in the management of both crises and errors.

**Methods.** The course allowed participants to experience and manage simulated crises. Emphasis was placed on important error management strategies such as communication, leadership and delegation of workload. A computerized mannequin that is physiologically and pharmacologically responsive was used to run life-like crisis scenarios. The scenarios were videotaped and reviewed during a debriefing discussion after each crisis. Scenarios were alternated with tutorials that addressed error management, communication and medico-legal issues. Participants evaluated the courses using 5-point Likert scales and free comments.

NZ Med J 2002; 115: 133-4

**Results.** In 1999 and 2000, 172 participants (34% of New Zealand anaesthetists) attended one of these courses. Evaluation forms were received from 151 participants (88%). The global evaluations had median scores of 4 or 5 and all respondents would recommend the course to others. The responses from 50 participants indicated that the course should be repeated at least every two years.

**Conclusion.** New Zealand anaesthetists found this an acceptable and useful form of training. Teamwork is an effective strategy in crisis management and error reduction and is worthy of consideration within the broader context of medical education.

Human error in healthcare is now recognized as a major problem<sup>1</sup> and it may result in adverse events in up to 7.7% of New Zealand hospital patients.<sup>2</sup> Principles of error management clearly need to become part of mainstream medical education and there is currently a paucity of educational interventions addressing this specific problem.

In this paper we describe our initial experience with a workshop-based course that has been developed to provide education in crisis management and in the recognition and management of error. Particular emphasis was placed on leadership, communication and the appropriate delegation of workload. These are the principles that are seen as important in error management training in other high-risk occupations such as aviation.<sup>3</sup> The course has been called Effective Management of Anaesthesia Crises (EMAC) and it makes extensive use of a METT™ Human Patient Simulator that has been installed in a high-fidelity mock patient-care environment at Wellington Hospital. The simulator breathes, consumes oxygen, exhales carbon dioxide, has heart sounds, palpable pulses and is interfaced with sophisticated physiological and pharmacological models. Very realistic scenarios are thus possible and this represents a major advance over the simple mannequins that are used to teach cardiopulmonary resuscitation. Although the specific context of this course has been the management of crises in anaesthesia, the general concepts have widespread implications.

In this paper we describe the EMAC course and the participant evaluations from the first two complete years of operation.

### Methods

Between January 1999 and December 2000, EMAC courses were run as either half-day or full-day workshops, with 4-6 participants and 2-3 instructors at each workshop. Participants were a mixture of anaesthesia registrars and specialists; and the instructors were mostly specialist anaesthetists from Wellington Hospital.

Each workshop began with an introduction to the simulator. This was followed by a team-building exercise that helped to develop cohesion amongst the participants by using an analogy to introduce crisis management concepts. In this 'ice-breaking' exercise, participants formed a circle, and then a tennis-ball was thrown to one of the participants. This person caught the ball, called-out a word written on the ball ('ECG', 'oxygen', 'pulse-

oximeter', 'drugs', 'BP', 'fluids', 'CPR' or 'charting') and then threw the ball to another participant, who repeated the task. The number of balls was then rapidly increased until the group was unable to successfully manage the task. During the discussion that followed this 'crisis', participants generally identified all the important principles needed for successful crises management, but from this non-threatening context.

The remainder of the workshop consisted of simulated crises, alternating with debriefing sessions and tutorials. The scenarios involved situations that anaesthetists might expect to meet in the operating theatre or the emergency department and called for a range of procedural and organizational skills. The participants rotated through a range of roles including the primary doctor (the 'hot-seat'), the colleague who comes to provide help ('first responder') or as a member of the 'crash-team'. Each scenario had the personnel required to imitate a typical clinical team and so realistic interpersonal interactions took place. The scenarios were videotaped and each crisis was followed by a debriefing discussion during which the videotapes were reviewed. This took place in a non-judgmental and collegial environment that was intended to encourage reflective practice.

The tutorials addressed both the clinical problems from the scenarios and the psychological underpinnings of crisis management, error management and the medico-legal consequences of error. In the half-day course there were two scenarios with one tutorial, whereas the full-day course typically had six scenarios and three or four tutorials.

Participants were asked to complete a 2-3-page anonymous evaluation form before leaving the centre. There was global evaluation of the course as a whole and then evaluation of the individual parts. The evaluations used five-point Likert scales and unstructured written comments. We also asked a question about the need to maintain secrecy of the scenario content. This was done because we had followed the international tradition of ensuring that participants were not forewarned of the content of scenarios. During the last half of 2000 we added a question to determine how frequently participants thought that they should undertake training of this nature.

Data from the Likert scales are summarised in Table 1. The free comments were mostly positive and are not included in this report.

### Results

During 1999 and 2000, 172 anaesthetists attended EMAC courses and 151 (88%) completed the course evaluation forms. Of the 151 evaluations, 58 were from half-day, and 93 from full-day courses. The 172 participants represent 34% of the 512 anaesthetists documented in the New Zealand Medical Council Workforce Survey.<sup>4</sup>

The global evaluations indicated that participants found the course useful. There was no real variation between the global evaluation scores, and more than 75% of respondents scored

components of the course as 4 or 5 (Table 1). All 151 respondents would recommend the course to others. 84 participants responded to the question asking if the scenarios should be kept secret and 78 (93%) wanted the content to remain a secret. There were 50 responses to the question asking how often anaesthetists should undertake this form of training and the median was twelve months (interquartile range 12-24 months).

**Table 1. Evaluation of the course.**

Global Evaluations (Anchors to Likert scale)*	n	Median (interquartile range)
Overall content? (very dissatisfied - very satisfied)	150	5 (4-5)
Useful to professional practice (no value - very useful)	151	5 (4-5)
Would you recommend it to others? (never - whole heartedly)	151	5 (5-5)
Realism? (very unrealistic - very realistic)	150	4 (4-4)

\*Likert scale scores range 1-5.

The evaluations of individual components were frequently incomplete and so no useful comparison could be made between the different sections of the course.

## Discussion

We have described our initial experience with a workshop-based course, using tutorials and high fidelity patient simulation, in anaesthesia education. The aims were centred on the need to improve teamwork in crisis management and in the minimization of error. Great emphasis was placed upon leadership, communication and the delegation of responsibility. Very little emphasis was placed upon the technical aspects of crisis management. The emphasis upon teamwork and behaviour sets this course apart from other forms of medical education and this approach warrants consideration in both undergraduate medical education and advanced training. A large proportion of New Zealand anaesthetists (34%) have attended these workshops and their evaluations show that the courses were valued, would be recommended to others and should be repeated at least every two years. The enthusiastic feedback from participants was consistent with the international experience<sup>5-8</sup> and has been remarkable, particularly because this sort of exercise is usually viewed as intimidating<sup>7</sup> and is expensive to undertake (course fee over \$500 per day).

Effective Management of Anaesthetic Crises (EMAC) courses were developed as a local adaptation of the Anaesthesia Crisis Resource Management (ACRM) Courses<sup>5</sup> that are offered at many centres around the world. Aviation was the first industry to develop Crew Resource Management (CRM) training, and this was because some form of human error precedes at least 70% of 'accidents'.<sup>3</sup> This high degree of association between error and accidents is similar in most high-risk vocations and it is believed that effective teamwork can reduce the risk of accidents. This type of training is mandatory in many commercial airlines.

Crises are characterized by an increased workload in terms of the volume, complexity or pace. A mismatch arises when task demands become excessive for one person and an inevitable consequence is failure to complete all required tasks. In addition, crises are associated with deterioration in performance and an increased risk of error.<sup>9</sup> The increased risk of error during a crisis makes this type of course particularly useful when developing specific education to address medical error. Simulation provides the opportunity to reliably create problems

that tend to trigger errors and which can only be solved with effective teamwork, and does so in an environment where there is no clinical risk. Effective teamwork is crucial because it allows a greater output of work and a reduced error rate.

In aviation, crew resource management training results in improved crew performance and reduced error rates.<sup>9,10</sup> This improvement has been shown by examining attitudes towards team-behaviour, with cockpit observations and from anecdotal reports of actual crises. In anaesthesia there are many anecdotal reports of improved crisis management after high fidelity simulation training, in addition to two publications.<sup>6,11</sup> One study reported self-evaluation of "real" clinical crises<sup>6</sup> and the other was a qualitative evaluation of crisis management in a simulator.<sup>11</sup> If these improvements can be carried into the clinical arena then patient safety should improve.

Healthcare professionals need to find ways to reduce error and the need for team training has been stressed by the most recent report from the Institute of Medicine.<sup>12</sup> Courses such as ours are one way of addressing this educational need. Recent data from aviation have emphasized the role of professional, organizational and national culture upon the effectiveness of Crew Resource Management training; different approaches are needed for different cultures.<sup>13</sup> The EMAC course is noteworthy because it has rapidly become widely accepted by New Zealand anaesthetists.

In conclusion, we have described a form of crisis management training that has been successfully implemented in New Zealand. This is a novel form of medical education by virtue of the emphasis placed upon team behaviour and error reduction, in contrast to technical and scientific content. Although much of the course content is highly specific to the domain of anaesthesia, the general concepts related to teamwork and error reduction are worthy of consideration in the broader context of medical education.

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## Evolution of the degree Doctor of Medicine at the University of Otago

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For the last 119 years, the University of Otago has offered the degree Doctor of Medicine (MD) for medical graduates who complete a course of advanced study. Over this period the requirements for the degree have undergone a gradual evolution from a qualification suitable for a medical specialist to an unsupervised higher research degree.

### Historical overview<sup>1,2</sup>

The earliest statutes for the degree MD at Otago were published in the University of New Zealand Calendar of 1883. The requirements for admission were that the candidate should be not less than 24 years of age, have graduated Bachelor of Medicine at least two years prior to enrolling and during this time have engaged in full-time medical practice or study. The candidate was required to present a thesis and pass written and oral examinations in one of the following groups of subjects; anatomy and physiology, surgery and anatomy, medicine and pathology, or public health and jurisprudence.

The first MD graduate was William Ledingham Christie who had the degree conferred in 1890. Prior to this several MD degrees were conferred *ad eundem gradum* on MD graduates from Universities in the United Kingdom and Europe.

In 1887, the requirement of a degree in arts or science was added to the statutes, although this was deleted in 1902 when the statutes were extensively revised. In this revision candidates were not admitted to the MD course for at least three years after graduating Bachelor of Medicine, although this was reduced to two years for graduates who had been employed for one year in a large hospital (of at least 100 beds) or in a European hospital. In order to qualify for the MD degree a candidate was required to present a thesis introducing observations on some branch of Medicine; and pass examinations in general medicine and one of the following; diseases of the chest, disease of the digestive and urinary systems, nervous diseases, diseases of children, midwifery or mental diseases.

In 1921, one year's service as a house surgeon, where duties included both medical and surgical work, also provided one year's exemption from the three year post graduate rule. In this revised regulation, it was stipulated that a published paper could be substituted for a thesis containing original observations and the examination requirements were amended. Candidates were now required to pass a written paper in general medicine and therapeutics, and a clinical examination with report and commentaries on two medical cases. In addition, practical knowledge of clinical laboratory methods and the application to medical diagnosis and treatment of electrical and mechanical appliances, was also examined. For those candidates engaged in teaching or research for two years, the clinical examination was replaced by a slanted examination directed towards the candidates' area of special study.

Service in a recognised hospital or medical school rather than in a European hospital was substituted as a condition of exemption from one year's post-graduate employment in 1926.

In 1928, the option of presenting a published paper in lieu of a thesis was removed, while in 1934 a literature review relating to the subject of the thesis was added as a requirement. The 1934 statutes also added the rider that if the thesis was of sufficient merit, the candidate could be exempted the examination components of the degree. In such instances the degree would be awarded *with distinction in thesis*. The importance of the clinical component of the examination was further eroded in 1952 by the option of substituting research related papers for both parts of the clinical examination. In 1958, a pass in the examination for Membership of the Royal Australasian College of Physicians (MRACP) qualified the candidate for exemption from all of the clinical examination for the MD degree. In 1963 this provision was extended to include a pass in other examinations of a standard similar to the MRACP.

The regulations for the degree were extensively revised in 1965 and the examination requirement was removed. At this time the minimal qualifying post-graduate period was extended from three to five years.

Further changes in the regulations were made in 1973 and the candidate was required to declare which proportion of the work was done in collaboration with others, and if the work was not independent, the candidate was required to identify the supervisor or collaborator. In 1994 this regulation was altered to remove any reference to supervision, with the candidate being required to identify their own work in the situation where co-authored work was submitted.

The MD regulations were again refined in 1999 following a recommendation from the Board of Studies for Higher Degrees, that the standard of the degree should satisfy its higher doctorate status. The degree is now awarded upon the submission of a collection of *published contributions of special excellence* or a thesis reporting an *original investigation of special excellence*.

### The doctoral hierarchy

Traditionally the doctoral degree stands at the head of the academic pantheon, being awarded for original research of a high standard. In recent years there has been some deviation from this standard practice and in some disciplines a doctoral degree is now awarded upon satisfactory completion of prescribed course work in addition to a corpus of original research. In 1998, the Committee on Academic Programmes of the New Zealand Vice-Chancellors' Committee (CUAP) presented a series of guidelines for doctoral qualifications in New Zealand Universities.<sup>3</sup> In these guidelines three categories of doctoral degree were defined upon the basis of the requirements leading to the award of the degree.<sup>1</sup>

1. *The Supervised Doctorate*, which includes the Doctorate of Philosophy (PhD), requires the presentation of supervised original research in the form of a thesis and is designed to prepare the candidate for an independent research career.<sup>2</sup>

2. *The Named Doctorate* is usually discipline-specific and includes a significant component of coursework in addition to the presentation of a thesis reporting the results of

supervised research. The requirements for the award of a Named Doctorate varies between institutions and in Australian Universities the coursework component varies from 17% to 67% of the total proportion of work required.<sup>4</sup> In New Zealand, both CUAP and the New Zealand Qualifications Authority require that the research component must contribute no less than two thirds of the overall credit for the degree and this may include thesis work, a collection of research elements or works of artistic or creative merit.<sup>5</sup>

3. The *Higher Doctorate*, which is awarded for independent work of special excellence. Candidates are required to submit a substantial and coherent collection of published works which make a significant contribution to the advancement of knowledge.

In their earlier form, the requirements for the MD satisfied the definitions of a Named Doctorate with an emphasis on clinical training rather than original research. Prior to the advent of the specialist colleges the MD degree served as a specialist qualification, however this role has been supplanted by the College Fellowship. When the Register of Specialists was established in New Zealand in 1971, the MD degree from Otago was listed as a recognised specialist qualification for the speciality of pathology only, with the proviso that the qualification must have been obtained under the University regulations that required both examination and the presentation of a thesis.<sup>6</sup> On 31 March 1980 the Medical Practitioners (Registration of Specialists) Regulations were amended and all reference to the MD was removed from the list of approved qualifications for specialist registration.<sup>7</sup>

With the removal of the requirement for a clinical component, the MD became neither a Supervised nor Higher Doctorate, as the degree was awarded for unsupervised work reported in thesis form rather than as a collection of published works. Historically, the MD was often taken by medical graduates who were in the early years of their career and as such fulfilled the role of a junior doctorate. Despite this, and the limited research component of the pre-1965 MD, the degree has always been considered by the University of Otago to be a Higher Doctorate, being listed as such in University statistics and having academic regalia similar to other Higher Doctorates. The most recent changes in the MD regulations have, to a large extent, addressed this ambiguity with the degree now being awarded for unsupervised work of special excellence. At present the regulations are in evolution and it is anticipated that in the future the degree will only be awarded following the submission of a substantial corpus of published work of high academic

merit. The University of Otago considers the PhD to be the appropriate qualification for the junior post-graduate researcher, as this provides for formal supervision and training in research methods and is available to undergraduate medical students through the intercalated MB, ChB, PhD programme. The development of advanced information technology linkages now allows for PhD supervision off-campus. This negates the argument that the MD was the appropriate research degree for candidates who were involved in clinical work and did not have direct access to University facilities.

At Australian Universities the academic standard of the MD is somewhat variable, ranging from a PhD equivalent to that of a higher doctorate of equal standing with the Doctor of Science degree.<sup>8</sup> In recent years some Australian Universities have raised the status of their MD<sup>9</sup> and at the newly established Medical School at James Cook University it is proposed that the MD will require a substantial collection of publications amassed over a period of at least ten years. It has been argued that the purpose of a supervised doctorate is to provide advanced learning in research techniques and that, because of an absence of formal supervision, the MD lacks credibility as to its scientific rigour.<sup>10</sup> The recent changes to the MD, both in Australia and at the University of Otago, have addressed this problem and the degree is now more suited to the established academic, who may already hold a supervised postgraduate research degree and who has amassed a significant research portfolio.

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## NHS breast screening report contradicts Cochrane findings

The NHS Cancer Screening Programmes insisted this week that breast screening saved lives despite recent claims the contrary by the Cochrane centre. Julietta Patnick, national coordinator of the NHS Cancer Screening Programmes, said that breast cancer screening "reduces the risk of a woman dying from the disease." This statement contradicts the findings of a Cochrane review published last month. The cochrane review concluded that "currently available reliable evidence does not show a survival benefit of mass screening for breast cancer (and the evidence is inconclusive for breast cancer mortality)." The abstract of the Cochrane review can be accessed at [www.cochrane.org/cochrane/revabstr/ab001877.htm](http://www.cochrane.org/cochrane/revabstr/ab001877.htm)

The 2001 breast screening programme's annual review, published this week, said that "more women are being screened and more cancers are being detected than ever before" and that this "should help to improve women's chances of survival."

After the launch of the review, Mrs Patnick said that research published in the *BMJ* last year suggested that breast screening was effective, contrary to the claims of the Cochrane Review. This research showed that between 1990 and 1998 breast cancer mortality fell by 21.3% in women aged 55-69. Of this fall, 6.4% was attributed to screening (2000; 321: 665-9).

BMJ 2001; 323: 1088.

## How the pharmacogenetics of cytochrome P450 enzymes may affect prescribing

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Understanding variation in drug metabolism is essential for drug prescribing so that efficacy is assured and adverse effects are minimised. Drug therapy during much of the 20th century has been dominated by 'one dose fits all'. Advances in clinical pharmacology have led to the replacement of this approach by one of dose-individualisation to take into account variability between patients. A substantial portion of variability in drug response is genetically determined, with other factors such as age, diet, other diseases and concurrent therapy playing important contributory roles.<sup>1</sup> A major cause of the genetic variation is polymorphisms in the cytochrome P450 enzymes that mediate metabolism of many drugs.

### The cytochrome P450 enzymes

The cytochrome P450 (CYP) enzymes are a group of haem-proteins found largely within the endoplasmic reticulum of hepatocytes. The enzymes are also found in high concentrations in the small intestine, and at lower concentrations in the kidneys, lungs and brain.<sup>2</sup> It is thought that the multiplicity of CYP enzymes arose during co-evolution of plants and animals, because plants depend on animals for propagation or reproduction but they must also develop defense mechanisms such as toxins to protect against destruction.<sup>3</sup>

Many CYP gene families are found in humans. CYP1, CYP2 and CYP3 are the major families responsible for the oxidative metabolism of a wide range of drugs, environmental chemicals and pollutants (or "xenobiotics").<sup>4</sup> Metabolism of such compounds can be considered in two phases. In phase I, the substance is modified by hydroxylation and other reactions catalysed by cytochrome P450 enzymes. In phase II, specific enzymes convert these activated compounds into more polar metabolites that are water soluble and readily excreted from the body. These enzyme systems protect the body against xenobiotics, and in addition they metabolise a diversity of endogenous substances including steroids, bile acids, fatty acids, prostaglandins, biogenic amines, and lipid hydroperoxides.

### Factors affecting CYP450 expression and activity

Variability in drug response is at least partly attributable to individual differences in the expression or function of CYP450 enzymes. This variability may be due to age, enzyme induction or inhibition, disease, or polymorphisms within genes encoding CYP450 enzymes.

*Enzyme inhibition and induction.* A range of factors can influence the expression of CYP450 enzymes, such as dietary substances, hormonal changes, environmental chemicals, pollutants and drugs. These may lead to a reduction (inhibition) or an increase (induction) in the activity or level of CYP enzymes, with important clinical implications. Enzyme inhibition may lead to adverse reactions if the drug is administered in a biologically active form, or reduced clinical efficacy if the drug is a pro-drug requiring metabolic activation.<sup>5</sup> In contrast, the recommended dosage of a drug

may be ineffective if it is co-administered with an inducer.

*CYP450 gene polymorphisms.* A genetic polymorphism is defined as a 'Mendelian or monogenic trait that exists in the population in at least two phenotypes (and presumably at least two genotypes), neither of which occurs at a frequency of less than 1-2%'. If the frequency of a genetic variant is lower than this, it is termed a rare trait or mutation.<sup>6</sup>

Variation in the DNA sequence of genes encoding drug metabolising CYP enzymes can abolish, reduce or increase the expression and activity of the enzyme, giving rise to distinct subgroups in a population that differ in their ability to metabolise certain drugs. Individuals with the normal genotype ('wild type') are termed extensive metabolisers (EM) while those with defective copies of a CYP gene are defined as poor metabolisers (PM). People with duplication or increased activity of their CYP genes may be ultrarapid metabolisers (UM). In cases where alleles lead to reduced (but not abolished) function of CYP genes, individuals may display an intermediate metaboliser (IM) phenotype.

If the variant enzyme is quantitatively significant in determining the fate of a particular drug within the body then there may be important clinical consequences of the genetic status. A PM may accumulate a drug excessively.<sup>7</sup> Conversely, UMs may require significantly higher concentrations of a drug than the 'recommended' dose to reach therapeutic concentrations.<sup>8</sup>

Of the CYP450 enzymes known to mediate drug metabolism, pharmacologically significant polymorphisms have been reported in several, as summarised below.

### CYP2D6

CYP2D6 is involved in the metabolism of around 20% of all currently prescribed and 'over the counter' drugs, including many antidepressants, antipsychotics, beta-blockers, antiarrhythmics, antihypertensives and opioids<sup>9</sup> (Table 1). There are clear examples of the clinical relevance of CYP2D6 polymorphisms.

*Codeine.* Codeine (methylnorphine) analgesia depends upon hepatic biotransformation to morphine catalysed by CYP2D6.<sup>7</sup> In experimental pain models a lack of analgesic effect of codeine in CYP2D6 PMs has been attributed to lack of formation of morphine.<sup>7</sup> The implication of this polymorphism is that the 5-10% of Caucasians that are PM may not experience adequate codeine analgesia.

*Perhexiline.* This effective anti-anginal agent was introduced into New Zealand in 1974. It lost favour because of an unacceptable incidence of peripheral neuropathy and hepatitis.<sup>10</sup> At the time, the importance of CYP variability was not appreciated. Perhexiline is metabolised by CYP2D6 and PM individuals are more likely to accumulate drug and suffer toxicity.<sup>11</sup> Therapeutic drug monitoring assists in the appropriate dosing of perhexiline, but a knowledge of the genotype might be even more helpful.

*Phenformin.* This biguanide antidiabetic agent preceded metformin. Phenformin was withdrawn from practice

**Table 1. Characteristics of polymorphic CYP450 enzymes involved in drug metabolism.**

Approximate proportion of drugs metabolised by this CYP	CYP2A6 1%	CYP2C9 5-10%	CYP2C19 2-3%	CYP2D6 25%
Caucasian PM	1-3%	6-13%	2-5%	5-10%
Asian PM	15%	2-3%	18-23%	1-3%
African PM	nd	nd	3-5%	1-3%
Polynesian PM	nd	nd	14%	0%
Maori PM	nd	nd	7%	5%
Major substrates*	Nicotine Methoxyflurane Halothane	NSAIDs <b>S-warfarin</b> <b>Phenytoin</b> Tolbutamide Losartan	Tricyclic Antidepressants <b>Omeprazole</b> <b>Pantoprazole</b> Barbiturates R-warfarin Phenytoin Moclobemide Proguanil	Lipophilic $\beta$ -blockers Tricyclic Antidepressants <b>SSRIs</b> <b>Tramadol</b> <b>Codeine</b> Neuroleptics <b>Perhexiline</b> <b>Propafenone</b> Flecainide Mexiletine

\*Bold indicates clinically proven effect of poor metaboliser phenotype. nd=no data.

around 20 years ago because of an unacceptable incidence of lactic acidosis, now thought to be due to increased phenformin concentrations as a result of CYP2D6 PM status.<sup>12</sup> Metformin, by contrast, is cleared by renal elimination and avoids the problems of CYP2D6 genetic polymorphism.

*Timolol eye drops.* This preparation has been associated with untoward and unacceptable systemic adverse effects. When first observed this seemed almost inconceivable. However it became apparent that timolol was a CYP2D6 substrate and that it was the PMs that were effectively amplifying the response to the small amount of systemic absorption that occurs.<sup>13</sup> Other  $\beta$ -blockers such as propranolol, metoprolol and labetalol are also metabolised by CYP2D6.

*Flecainide.* It is possible, though unproven, that the problems of flecainide illustrated by the Cardiac Arrhythmia Suppression Trial-1 (CAST-1) study may have been related to CYP2D6 PM status.<sup>14</sup> This antiarrhythmic agent and some others such as encainide and propafenone are metabolised by CYP2D6.

### CYP2C9

CYP2C9 contributes in whole or in part to the metabolism of many clinically important drugs such as fluoxetine, phenytoin, tolbutamide, S-warfarin and numerous non-steroidal anti-inflammatory drugs.<sup>15</sup> Clinical significance of polymorphisms in CYP2C9 has been established, at least for warfarin.<sup>16</sup>

*Warfarin.* The use of warfarin is complicated by large interindividual variation in response for a given dose. As a consequence, it is difficult to predict the correct maintenance dose of warfarin for an individual patient and daily doses range from 0.5mg to 60mg. The low therapeutic index of warfarin, and particularly haemorrhagic complications, makes dose-individualisation very important.

Warfarin is a mix of two stereoisomers, R-warfarin and S-warfarin, which are metabolised by different CYP450 enzymes. The anti-coagulant activity of S-warfarin is three times greater than that of R-warfarin, and this form is metabolised to an inactive metabolite by CYP2C9 and excreted in the bile.<sup>17</sup>

Two variant CYP2C9 alleles have been identified that result in 5% and 12% of wild type enzyme activity. A strong association has been found between patients that carry one or more CYP2C9 variant alleles and a requirement for lower doses of warfarin.<sup>16</sup> Carriers of variant CYP2C9 alleles also appear to be at greater risk of developing bleeding complications on warfarin medication than wildtype patients. Therefore, genotyping patients for CYP2C9 variants prior to administering warfarin may increase the safety and efficacy of this widely used drug (Figure 1).

### CYP2C19

CYP2C19 catalyses the metabolism of many important drugs including omeprazole and pantoprazole, certain barbiturates, some antidepressants, R-warfarin and the anti-malarial agent proguanil. Seven CYP2C19 PM alleles have been described, and individuals who are homozygous for these alleles may have an altered response to a CYP2C19 substrate.<sup>18</sup> Patients with PM alleles have much reduced metabolism of a given dose of omeprazole or pantoprazole compared with those of EM status.<sup>19</sup> A study examining the efficacy of omeprazole and amoxicillin treatment for peptic ulcers associated with *Helicobacter pylori* found that individuals with a CYP2C19 PM genotype had significantly higher cure rates than EMs on this dual therapy,<sup>20</sup> suggesting a concentration-response effect.

### CYP3A4 & CYP3A5

The CYP3A subfamily are of pharmacological significance because they collectively form the largest portion of liver CYP protein and metabolise between 45% and 60% of all currently used drugs, including calcium channel blockers, statins, protease inhibitors, steroids, macrolides andazole antifungal agents. A number of pesticides, non-ionic detergents and carcinogens are also substrates of CYP3A.

Considerable inter-individual variation in CYP3A4- and CYP3A5-mediated metabolism has been observed. Part of this variability may be attributed to drug interactions. CYP3A gene expression can be induced by barbiturates, glucocorticoids and rifampicin. Conversely, CYP3A catalytic activity can be strongly inhibited by antifungal agents (eg, ketoconazole), macrolide antibiotics (eg, erythromycin) or by

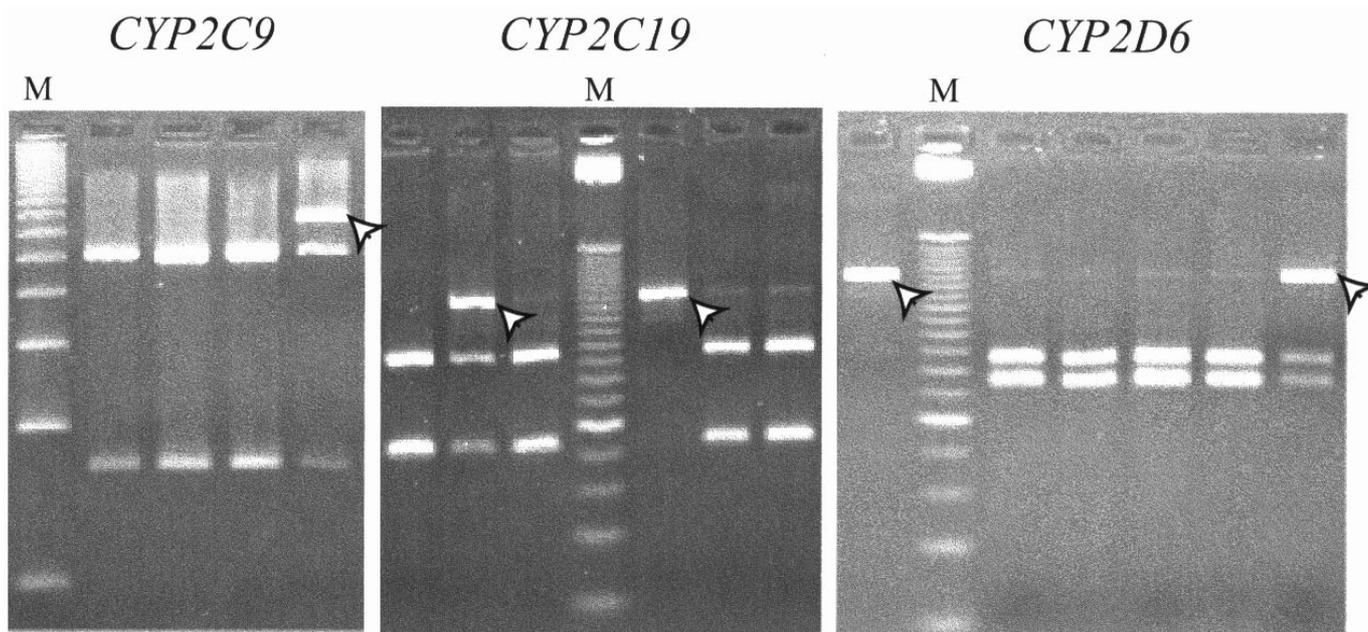


Figure 1. Examples of simple DNA assays for the detection of CYP2C9, CYP2C19 and CYP2D6 PM variants. DNA from each patient is subjected to polymerase chain reaction (PCR) which amplifies the region of interest. Treatment of the resulting DNA fragment with a restriction enzyme that is sensitive to differences in the sequence of bases comprising the fragment is diagnostic for variants of each gene. Each lane on the agarose gels represents the genotype of a single patient, and PM variants of each gene are indicated by an arrow. Bands in each patient sample are sized using a DNA size marker (M).

foodstuffs such as grapefruit juice.<sup>21</sup> Recently a number of polymorphisms have been identified within the CYP3A4 and CYP3A5 genes and it is possible though unproven, that these allelic variants may contribute to variability in CYP3A metabolism.

### Interracial differences in allele frequencies

The study of CYP genes and CYP enzymes in different populations has revealed substantial interracial differences in the frequency of particular phenotypes. In many cases this variability can be explained by the differential occurrence of specific variant alleles (Table 1). For example, 5-10% of Caucasians are CYP2D6 PMs<sup>22</sup> and 6-13% are CYP2C9 PMs,<sup>23</sup> whereas amongst Asians and Africans this percentage appears much lower for both enzymes.

To our knowledge there are only two studies on the frequency of CYP450 phenotypes in New Zealand Maori. Wanwimolruk et al (1995)<sup>24</sup> phenotyped 101 Maori subjects for CYP2D6 and CYP2C19 activities, and found 5% of this sample population were CYP2D6 PMs, and 7% were CYP2C19 PMs. The incidence of CYP2D6 PM phenotypes in Maori appears to be similar to frequencies found in New Zealand Caucasians. In contrast, the frequency of CYP2C19 PM phenotype is higher in Maori than Caucasians but lower than the usual ranges (18-23%) reported in Asians. Furthermore, no research has been published on the allele frequencies of pharmacologically significant CYP450 genes in this New Zealand population. Considering the substantial differences in allele frequencies found across other races and the impact this may have on prescribing, it would seem a matter of some urgency that allele frequencies for CYP450 and other pharmacologically significant genes be established for Maori, Pacific Islanders and other major ethnic groups represented in New Zealand.

### Conclusions

A greater awareness of pharmacologically significant CYP450 polymorphisms is needed to minimise adverse

drug reactions and therapeutic failures. Knowledge of pharmacogenetics is increasing rapidly, particularly since the completion of the Human Genome Project.<sup>25</sup> Methods for detecting polymorphisms are becoming increasingly accessible (as illustrated in Figure 1), and simple DNA tests can detect most known variants.<sup>26</sup> However, understanding the impact of these genetic variants on drug responses lags behind our ability to test for variant genes, and further research is required to determine the potential clinical utility of these tests. In future, when a patient presents with an unusual reaction to a medication it may be worth investigating whether the cause of the reaction has a pharmacogenetic basis. As genotyping costs come down, it may become feasible to routinely screen patients for a wide range of important polymorphisms in advance of drug treatment. Finally, it would be of value to establish the frequencies of CYP450 genetic variants in New Zealand Maori and Pacific Island populations, particularly in situations where such groups show elevated risk of adverse drug reactions relative to Caucasians.

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## Monsanto accused of cover-up over Alabama pollution

Monsanto, the drugs company that has invoked the ire of environmentalists because of its involvement with genetically modified foods, is about to face a far older controversy.

A case brought by 3500 residents of a small town in Alabama in which Monsanto is accused of covering up the contamination of the local rivers and land over a 50-year period was due to reach court this week. Monsanto has already paid \$80m on legal settlements related to the case.

The case relates to a factory in Anniston, Alabama, that produced the now banned industrial coolants, PCBs. It is alleged in documents reported by the Washington Post last week that fish placed in a local creek in tests during the 1960s floated to the surface within 10 seconds, spurting blood and with skin flaking off.

The internal memos, which will be presented as evidence, claim that Monsanto was aware more than 30 years ago that its chemicals were contaminating fish in local rivers.

Monsanto has since spent \$40m cleaning up the creek and landfills that were filled with toxic waste. The clean-up operation has included the demolition of about 100 homes and businesses near the factory.

PCBs were used in a wide range of products, from paint to newsprint and bread packaging, before being banned in 1979.

Monsanto has conceded that it would take a tougher line on toxic waste under today's more stringent environmental controls but argues that in the context of the 1960s and 1970s regulations and levels of knowledge the company has done nothing wrong.

The problem gained national attention when a local fisherman caught deformed bass in a local creek in the mid-1990s. Alabama governors then issued their first warning against eating fish from local creeks.

David Teather. *Guardian Weekly* 2002, 10-16th Jan, p10.

## New Zealand showed the risks of policies that foster inequality

New Zealand was the perfect field trial for structural reform. It was small, geographically separate, and has a single-house parliament dominated by the executive. Moreover it was infected with all the toxins that the reformers said were poisoning capitalism: import controls, capital controls, strong trade unions, a redistributive welfare state, a large state sector. New Zealand was hooked on all the bad drugs.

In 1984 the country's Labour government said that this all had to change. It started by deregulating interest rates, removing international capital restrictions, floating the currency and removing agricultural subsidies. Then, having got the taste for change, it scrapped regulations on business, abolished import quotas, enshrined price stability in law as the sole object of monetary policy, forced workers into individual contracts, announced that budget deficits would eventually be banned, cut income taxes and slashed welfare benefits. This was not a detox regime: it was cold turkey.

As a consequence of these reforms, inequality in New Zealand grew more rapidly than in any other country. The government created an underclass where none had existed before. But purged of its addiction, it was hailed as the country that the rest of the West should emulate, the role model that had dared to do what even Margaret Thatcher would not do in Britain, and was all the better for it.

In a brutal sense the New Zealand experiment was worthwhile. It highlighted the ineffectiveness and risks of policies that deliberately foster inequality. New Zealand has shown the world how not to do it.

Larry Elliott. *Guardian Weekly* 24-30/1/02. p14.

## Come back Doctor Finlay!

NZ Med J; 2002; 115: 141

“W;t” (sic), a recent local theatre production, has medical interest. Vivian Bearing is a professor of English literature specialising in John Donne, the metaphysical Elizabethan poet.<sup>1</sup> Donne, obsessed by death, wrote the “Holy Sonnets” in which he engages in a witty dialogue with God and death eventually conquering death by dying himself – “And death shall be no more; death, thou shalt die.”<sup>2</sup> Bearing is successful and in control of her work, her life and John Donne’s poetry until she is found to have ovarian cancer. She then starts a dialogue about her own death and the parallel with Donne in the “Holy Sonnets” is full of exciting possibility – Donne challenges death and God whilst Bearing challenges death and the medical profession.

Bearing opens by explaining that her illness will progress to death as the play unfolds. Excitement soon gave way to dismay, as the play quickly became a stinging attack on the medical profession. Oncologist behind desk tells Bearing in surgical cap and gown that she has a fatal illness with a cheery good morning, how are you? you have cancer, sit down! Audience laughed nervously. Junior doctor has no interest in clinical medicine or patients but is impassioned by the power of molecular biology. Only female nurse shows any human decency. There are scenes of medical humiliation. Teaching ward round focuses on her ovaries but fails to engage Bearing’s ascerbic wit. Pelvic examination has tense, theatrical build up as Bearing mounts obstetric stirrups, decently facing away from audience. Junior doctor starts examination, remembers testily that hospital protocol requires chaperone, then wanders off to look for nurse leaving Bearing with legs akimbo. Audience laughed nervously again but with disapproval. The resuscitation scene is the worst indignity. Junior doctor starts CPR against Bearing’s instruction to achieve the best possible survival statistics for the current drug trial. Eventually, caring nurse wrestles doctor off dying patient. Audience gasped.

Why did playwright Margaret Edson write such a vitriolic attack on the profession and is there any truth in the medical monsters that she created? The programme note provided a clue. Before writing “W;t” at the age of 30 in 1991, Edson worked as a physical therapy aide and unit clerk in a cancer and AIDS unit of a large American teaching and research hospital during the early trials of AZT. It would be easy to dismiss “W;t” as American, out of date, or a feminist diatribe except that it is a major success. “W;t” received a local opening night standing ovation and a string of international awards including the 1999

Pulitzer Prize. So why does “W;t” capture the imagination of the public but touch a raw nerve of the profession? The programme note again provided the answer in describing the medical establishment as “supposedly” on Bearing’s side, but clearly, the doctors are not on Bearing’s side. They are adversaries.

In the last few years, the mutual trust between patient and doctor has been eroded for one reason or another as more patients complain and more doctors practice defensive medicine. There is also a presumption nowadays that the doctor is not necessarily the natural patient advocate, and that doctors and patients are adversaries often enough to justify the range of patient advocacy services that we have today. Not so long ago, the presumption was different. After working as a general practitioner in Glasgow in the 1930s, AJ Cronin wrote “The Citadel”, a story of the idealistic struggle of a young doctor to maintain his integrity against the attractions of worldly success.<sup>3</sup> The long running television series “Doctor Finlay’s Casebook” was based on Cronin’s characters and was recently revived.<sup>4</sup> Dr Finlay is a rather too earnest family practitioner, and Dr Cameron, his senior partner, is an unreformed paternalist, but there is never any doubt that they both stand by their patients with dedication, compassion and trust. If his medical fiction is based on his experience as a doctor, Cronin must have trusted his patients and his patients must have cared for him. This paradox is at the heart of the relationship as much today as it was in Cronin’s day. Unless they trust their patients, doctors will become more and more defensive, and unless they care for their doctors, patients will become more and more like clients.

From time to time, when yet another media story makes the profession seem more like “W;t” than “Dr Finlay’s Casebook”, a keynote address of a world conference comes to mind and lifts the spirit. The invited lecturer, after 50 years of world class contribution to medicine, said that one of the most effective weapons that the doctor has in the struggle against tuberculosis is kindness.<sup>5</sup>

### Scrutator

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## Attributing medical errors to 'the system': the new Accident Compensation legislation

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NZ Med J; 2002; 115: 142

On 1 April 2002, the new accident compensation legislation comes into force. The Injury Prevention, Rehabilitation, and Compensation Act 2001 includes a change to the definition and scope of 'medical error' under the medical misadventure provisions which is likely to have an impact on practitioners and on the organisations within which they work. This article considers this change.

The core part of the definition of 'medical error' remains unchanged. 'Medical error' continues to mean the failure of a registered health professional to observe a standard of care and skill reasonably to be expected in the circumstances (ie, negligence). If a person suffers a personal injury that is caused by a medical error, then in most situations the person will be covered by the Act.

However, there is a change in the range of entities who can be found to have committed 'medical error'. Previously, 'medical error' could only be found where the error could be attributed directly to a particular registered health professional. If the error could not be attributed to an individual's sub-standard conduct, there could be no 'medical error' and therefore no cover. Systemic errors or errors made by the organisation in general were not included. The new legislation changes this. After 1 April, if the treatment is provided at the direction or under the management of an organisation, and an error occurs which cannot readily be attributed to a particular registered health professional, the failure of the organisation to meet a reasonable standard of care will amount to a 'medical error'.<sup>1</sup> It will no longer be necessary to be able to pin the error onto an individual health professional in order to be covered. If the error cannot be attributed to an individual, it will be enough to attribute the sub-standard care to the health provider (eg, district health board, private hospital).

This represents a significant amendment which at first glance appears to be consistent with the widespread call to move away from blaming individuals for mistakes and concentrating more on the role played by the wider system. However, whilst the change is a welcome one in that it will improve the fairness of the process so that a patient who suffers as a result of a medical error is not declined cover simply because the error cannot be attributed to an individual, the change is unlikely to assist in reducing the blame mentality that many feel prevails in the health sector. The amendment is not a movement from attributing responsibility to individuals towards attributing responsibility to the system. In assessing whether a 'medical error' has occurred, the Act requires that there is first an

attempt to attribute the error to a registered health professional. It is only where the error cannot 'readily be attributed' to an individual that consideration can be given to attributing the error to the organisation.

It is likely that this amendment will result in an increase in the legalisation of the ACC process. Lawyers have already become increasingly involved in making representations as to whether an incident should amount to 'medical error', largely because of the increase in the seriousness of the consequences to a doctor when there is a finding of 'medical error'. These consequences include the referral of the matter to the Medical Council. The widening of the definition of 'medical error' opens the way for significant debate as to whether the error should be attributed to an individual practitioner or whether it is in fact a systemic error. There is likely to be debate as to what amounts to a systems error. Practitioners, understandably, are likely to want to raise the wider causative factors leading to the error in an attempt to ensure the responsibility for the error is attributed to the organisation as a whole rather than to themselves. A consequence of this may well be that there is an increase in the debate as to who is responsible for a particular error. This is likely to legalise the process, and will do nothing to assist in creating a harmonious, blame-free environment. Legalisation of the process, and the inevitable accompanying delays, are also likely to be to the detriment of the claimant.

One of the purposes of the ACC legislation is to ensure fair compensation for loss from injury. To this extent the amendment is entirely reasonable. However, the express "overriding goals" of the new Act are the minimisation of "both the overall incidence of injury in the community, and the impact of injury on the community".<sup>2</sup> Many experts believe that one way of reducing the incidence of injury from medical error is by eliminating the 'blame' culture from the hospital environment. If this view is correct, then the amendment may prove to be a step in the wrong direction in that it may actually accentuate the blame culture. This issue perhaps highlights the difficulties involved in implementing a process which is both fair to all and which has a long-term utilitarian focus of improving the system for the benefit of everyone.

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1. Injury Prevention, Rehabilitation, and Compensation Act of 2001, Section 33(2).  
2. Injury Prevention, Rehabilitation, and Compensation Act of 2001, Section 3.

## Reform - Regression - Renaissance

Ross A Fairgray, *Previously Medical Superintendent-in-Chief, Canterbury Area Health Board, Christchurch.*

NZ Med J 2002; 115: 143

The recent industrial action that threatened patient care services in Christchurch gave me genuine concern. My justification for comment arises from twenty years of medical administration in Christchurch. When I retired, voluntarily, in 1989, I was responsible for all aspects of patient care services and for all associated staff. Guiding principles for me included the belief that medical administration is important; goodwill and trust between Board, Managers, Nurses and Doctors is absolutely essential; that those with authority must always accept responsibility; that whilst authority can be delegated, responsibility cannot, and that those who plan must also manage. Whilst some staff disagreed with aspects of this policy virtually all agreed that patient care was paramount. By and large this situation did prevail as indicated by the fact that in 1989, even in so-called shortage specialities like anaesthetics and radiation oncology, there were no vacant positions.

Industrial action has recently involved diverse groups throughout New Zealand including geriatric staff, nurses and radiation therapists, suggesting wide spread "dis-ease". This has confirmed earlier predictions by health professionals about staff shortages and declining standards. These predictions, often denounced as political grandstanding were of course practical realistic assessments. Prolonged disruptive industrial action was extremely unlikely a generation ago because health staff accepted that they were "their brothers' keepers". The health reforms with their emphasis on market forces, efficiency, cost effectiveness and competition do not appear to have improved overall standards of care but have certainly altered goodwill and related intangibles. Few could dispute that in recent years health services have been plagued with discord, strife, loss of trust, apparent excessive administrative costs and ongoing staff shortages, without the desired gain in quality or quantity of care.

Health workers, like all involved in social services share the ethos that absolute priority must be given to providing competent, compassionate care. This philosophy makes withdrawal of labour untenable. As a result health workers are always vulnerable when negotiating conditions of employment. Employers therefore are required to act reasonably and fairly when negotiating. Until the late 1980's this was usually the case. National salary scales with increments based on seniority or responsibility limited disparities between various parts of New Zealand and there was minimal industrial action.

The radical health reforms initiated under Labour in the late 1980's and continued by National, upset the established ethos and balance. Managers frequently devoid of clinical training and experience were given positions of virtually total executive authority. The belief "that health services should be managed like any other industry" led to a significant loss of goodwill between clinical and management staff. Instead of allies the two groups became opponents. The loss of goodwill became greater when executive staff received salaries out of proportion to previous relativities, and bonuses. Moreover the enthusiasm of some managers to accept authority and an

international level of salary without accepting ultimate responsibility irked many clinical staff. Staff who spoke against the reforms were further incensed when they were labelled as defenders of a failing traditional system or were said to be frightened conservatives who feared new technology or change. The preferment of clinicians who supported the new financial focus in health increased mistrust and dissension among staff.

Successive governments have failed to appreciate the negative effects of declining goodwill. It has taken ten years for it to become obvious that there is a recruitment and retention problem that has led to a crisis situation in New Zealand health services. Not surprisingly, clinical staff in all disciplines have opted to work overseas. The present unhappy state of affairs is bad for everyone - patients, families, staff, managers and governments.

How to effect a renaissance and revive what was a successful national health service is crucial for every New Zealander. Such a task is difficult but far from impossible because the majority of health workers genuinely wish to provide competent care devoid of industrial disharmony.

Reconciliation is urgently needed. Political parties, especially National and Labour, must publicly acknowledge that the market force health reforms have not been successful despite genuine efforts by some individuals. Until this is acknowledged health will continue to be an emotive vote-catching political football. Reconciliation is also needed between clinical and administrative staff. Appointees with experience in clinical care and responsibility must again be given senior management positions with real executive authority and not merely advisory roles. To ensure the proper exercise of clinical judgement, policy-making committees in District Health Boards must contain health workers who have been elected by their peers. This will improve work satisfaction and reduce industrial unrest.

Salary scales at local and national level must be addressed so that executive salaries are no longer completely out of step with clinical salaries, and relativities between clinicians are fair. Readjustment may take time and may involve a growth differential in which clinical staff receive increments greater than those for executive staff. This may cause the resignation of some managers but without clinical staff, services will cease!

Since a health service cannot have an unlimited budget clinical staff must be involved in determining priorities and negotiating fair practice, which may involve rationing.

If changes in the philosophy and practice of health administration are not made, and market forces continue to dominate, problems will become worse. Goodwill between staff and the morale of health professionals must be restored. The system which leads managers and clinicians to become adversaries must be changed so that they can cooperate in the development of competent and compassionate care.

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