

CONTENTS

This Issue in the Journal

- 4 A summary of the original articles featured in this issue

Editorials

- 7 Delay in the treatment of acute coronary syndromes
Robin M Norris
- 10 Proposed changes to the New Zealand Ethics Committees
Frank A Frizelle

Original Articles

- 12 Pre-hospital delay in acute coronary syndromes: PREDICT CVD-18
Daniel Garofalo, Corina Grey, Mildred Lee, Daniel Exeter, Andrew J Kerr
- 23 Uptake of pulmonary rehabilitation in New Zealand by people with chronic obstructive pulmonary disease in 2009
William M M Levack, Mark Weatherall, Julie C Reeve, Christina Mans, Antonia Mauro
- 34 Hazardous patterns of alcohol use are relatively common in smokers: ITC Project (New Zealand)
Nick Wilson, Deepa Weerasekera, Christopher W Kahler, Ron Borland, Richard Edwards
- 42 Diagnosis of disorders of intermediary metabolism in New Zealand before and after expanded newborn screening: 2004–2009
Callum Wilson, Nicola J Kerruish, Bridget Wilcken, Esko Wiltshire, Kathy Bendikson, Dianne Webster
- 51 New Zealanders' knowledge of palliative care and hospice services
Rod D MacLeod, Rachel Thompson, John W Fisher, Kris Mayo, Nathan Newman, Donna M Wilson
- 61 Ocular trauma epidemiology: 10-year retrospective study
Archana Pandita, Michael Merriman
- 70 Aspiration pneumonia and challenges following the Samoa Tsunami in 2009
Tamara Ah Leong-Nowell, Foloto Leavai, Lucilla Ah Ching, Limbo Fiu, Rosemary Wyber, Mitzi Nisbet, David Jones, Tim Blackmore, Tupu Ioane-Cleverley

Viewpoint

- 79 'The way things are around here': organisational culture is a concept missing from New Zealand healthcare policy, development, implementation, and research
Shane L Scahill

Clinical Correspondence

- 90 Are Māori women at increased risk of cardiac complications of Graves disease?
Parul Nigam, Adam Morton
- 93 Nasopharyngeal fibroepithelial polyp in a New Zealand Māori man
Ravi Jain, Subhaschandra Shetty
- 97 Medical image. Quadricuspid aortic valve: a rare cause of aortic regurgitation
Jen-Li Looi, Andrew J Kerr
- 100 Medical image. Spontaneous pneumomediastinum without pneumothorax in idiopathic pulmonary fibrosis
Martyn P T Kennedy, Ahmed Fahim, Graham Smith

Letters

- 103 BPAC recertification plan
Humphrey B Rainey
- 104 Recertification and the Medical Council
Roger M Ridley-Smith
- 106 Auckland District Health Board's new emergency care initiatives
Lance Gravatt
- 108 Integration of emergency department and primary care workload
Ken Greer
- 110 The pharmaceutical industry has a contribution to make to evidence-based healthcare
Kevin Sheehy
- 112 Do New Zealand nurses claim more lumbar spine injuries than the general population? A retrospective study (1995–2009)
Jon Cornwall, Markus Melloh

100 Years Ago in the NZMJ

- 116 Quakery with impunity

Methuselah

- 117 Selected excerpts from Methuselah

Notice

119 Medical Volunteering in Namibia

This Issue in the Journal

Pre-hospital delay in acute coronary syndromes: PREDICT CVD-18

Daniel Garofalo, Corina Grey, Mildred Lee, Daniel Exeter, Andrew J Kerr

In people admitted to Middlemore Hospital with heart attacks and unstable angina we studied the time delay between developing chest pain and coming under potentially life-saving paramedic care. Over a half of people had a delay in the community of over 3 hours. Those from more deprived areas were delayed more than an hour compared with those from less deprived areas. Māori, Pacific, Indian and those from areas of higher deprivation were less likely to travel to hospital by ambulance. Community intervention targeted at more disadvantaged communities and higher risk ethnic groups should be considered as part of an overall strategy to reduce disparity and improve cardiac outcomes.

Uptake of pulmonary rehabilitation in New Zealand by people with chronic obstructive pulmonary disease in 2009

William M M Levack, Mark Weatherall, Julie C Reeve, Christina Mans, Antonia Mauro

Pulmonary rehabilitation is a health intervention for people with respiratory disease that typically involves an 8–16 week programme of group exercise and education. Pulmonary rehabilitation is one of the few health interventions that has been demonstrated to improve physical function and quality of life in people with chronic obstructive pulmonary disease (COPD)—a disorder affecting approximately 120,000 to 320,000 New Zealanders. This survey investigated the total uptake of pulmonary rehabilitation in New Zealand by people with COPD in 2009, and found that currently approximately only 1% of the total population of people who could potentially benefit from pulmonary rehabilitation were participating in it in one year. Given the cost-effectiveness of pulmonary rehabilitation for improving health outcomes for people with COPD, there is a need for far greater investment of healthcare resources in pulmonary rehabilitation and a need for the development of strategies to improve its uptake.

Hazardous patterns of alcohol use are relatively common in smokers: ITC Project (New Zealand)

Nick Wilson, Deepa Weerasekera, Christopher W Kahler, Ron Borland, Richard Edwards

In this study we aimed to describe patterns of alcohol use in a nationally-representative sample of 1376 New Zealand smokers. We found that a third (33.1%) of these smokers had a drinking pattern that was considered hazardous (i.e., AUDIT scores ≥ 8). These figures were much higher than for non-smokers in the NZ Health Survey (at 13.1%). Furthermore, we found that hazardous drinking patterns were

significantly more common among: younger smokers, male smokers, and Māori smokers (and in some analyses for smokers with financial stress and for moderate individual-level deprivation). Given the international evidence that hazardous drinking may impede quitting, policy makers could consider the potential benefits of improved alcohol control as part of the national strategy to curtail the tobacco epidemic and achieve the government's "Smokefree Nation 2025" goal. Such an approach could also reduce this country's high levels of alcohol-related harm and reduce gender and ethnic health inequalities.

Diagnosis of disorders of intermediary metabolism in New Zealand before and after expanded newborn screening: 2004–2009

Callum Wilson, Nicola J Kerruish, Bridget Wilcken, Esko Wiltshire, Kathy Bendikson, Dianne Webster

Inborn errors of metabolism refer to a group of rare genetic chemical disorders. Children with these conditions often present with serious symptoms such as coma. However because these symptoms are usually due to other more common conditions clinicians may not investigate the patient for an underlying metabolic disorder. This is unfortunate as treatment, if commenced very early dramatically improves the outcome. This paper reports the findings of a nationwide 3-year surveillance study that shows that these disorders have been under diagnosed in recent years in New Zealand. A small number of children are likely to have died yearly as a result. The recent introduction of expanded newborn screening, a process whereby key chemicals are measured in the neonatal Guthrie card blood test prior to the child becoming sick, will hopefully improve this situation. The paper further discusses this new form of screening, its advantages and limitations.

New Zealanders' knowledge of palliative care and hospice services

Rod D MacLeod, Rachel Thompson, John W Fisher, Kris Mayo, Nathan Newman, Donna M Wilson

This project surveyed over 1000 New Zealanders' views about palliative care and local hospice services. There was a reasonably good understanding of the concept of palliative care. However, people could not always identify local hospices, with younger people and males more unaware of accessible hospice services. Better education about what hospices do is needed.

Ocular trauma epidemiology: 10-year retrospective study

Archana Pandita, Michael Merriman

This paper gives an insight regarding the eye trauma within New Zealand, illustrates common causes and effects from it. Appropriate safety and precautionary measures can be taken to prevent much eye trauma from happening, reducing visual loss and impact on the community from it.

Aspiration pneumonia and challenges following the Samoa Tsunami in 2009

Tamara Ah Leong-Nowell, Foloto Leavai, Lucilla Ah Ching, Limbo Fiu, Rosemary Wyber, Mitzi Nisbet, David Jones, Tim Blackmore, Tupu Ioane-Cleverley

The medical problems of the tsunami that hit Samoa in 2009 was caused by the pressure of tsunami waves forcing dirty seawater contaminated with all sorts of foreign objects (blunt or sharp), germs, sand and mud to penetrate the skin through to deep tissue and organs such as the lungs causing aspiration pneumonia or “tsunami lung” and can cause blood stream infection, that can kill unless aggressively treated in a timely manner.

This paper reports the challenge of treating tsunami lung victims in a developing country like Samoa, with all the expected problems of limited resources for diagnosis, treatment and monitoring. The challenge was that little is known about how best to treat tsunami lungs, as randomised trials upon which best practice is based are not appropriate or ethical to be carried out in such disaster situations. Despite numerous tsunami disasters, there is no database of tsunami shared-experiences to learn from, particularly for doctors working in undeveloped countries with resources and internet access. Local doctors generally have the best understanding of the local health situation as well as their resource constraints are fundamental for the initial and on-going tsunami response efforts. The management approach taken during the Samoa situation was driven by necessity, locally available resources and the application of basic clinic practice with suggestions from overseas experts.

The outcomes of the Samoa tsunami patients admitted with aspiration pneumonia were very good, only one patient died and all were discharged with no complications on follow-up.

Delay in the treatment of acute coronary syndromes

Robin M Norris

Ventricular fibrillation (VF) is the major mode of death in the acute coronary syndromes (ACS). VF occurs most commonly at or near the onset of an acute ischaemic episode and is most readily treated by defibrillation when it happens within an hour or so of the onset. It follows that the proper management of patients with ACS is to provide them with access to a defibrillator as soon as possible after they call for help. Ambulances carry defibrillators and paramedics are trained to use them.

Delay in coming under care is the most potent avoidable risk factor for patients with developing heart attacks, and the risk from not having immediate access to a defibrillator is greater than the risk of more serious damage to the myocardium from delay in delivery of thrombolysis or primary angioplasty.^{1,2.}

In this issue of the *Journal*, Garofalo and colleagues³ from Middlemore Hospital in Auckland, New Zealand describe the delay from onset of symptoms to defibrillator availability in 805 consecutive patients with ACS admitted over an 18-month period during 2009–2010. Defibrillator availability was defined as the time of arrival at hospital or the time of arrival of an ambulance, whichever was the sooner. Middlemore Hospital was an ideal site for the study since it serves the population of South Auckland with its mix of ethnicity and degree of social deprivation.

Results of the study are important and give cause for concern. The median time from onset to defibrillator availability was nearly 3 hours, the most important determinant of delay being the patient's behaviour in calling for help. For the 43% of patients whose first call was to the ambulance, the median time from onset to defibrillator availability was 1¼ hours, but for the 34% who called their general practitioner the time was 9¼ hours, a full 8 hours longer than if they had called the ambulance.

Māori, Pacific Islanders, and Asian Indians as well as patients from areas of high social deprivation were less likely to call the ambulance. The assumption must be that patients arriving with the longest delays must increasingly be a population of survivors who have had the good fortune to have escaped the complication of VF which happens in perhaps 10–20% of ACS, most commonly over the first few hours. Those who have died before they reach hospital are of course unaccounted for.

Only 20 (2.5%) of the 802 Middlemore patients had a cardiac arrest, and we are not told whether these arrests happened in the ambulance or in hospital or how many survived to be discharged from hospital. Again the assumption must be that an avoidable number of patients died outside hospital because of unnecessary delay.

What needs to be done? First, we need to know to what extent the problems at Middlemore Hospital happen throughout New Zealand. We also need to know the relationship between delay and success of defibrillation both in and out of hospital. This implies collaboration between hospital clinicians and the ambulance service, and a seamless national audit of the treatment of ACS both inside and outside hospital. A

recent publication in this Journal⁴ confirms the vital role of the ambulance service in preventing death from out-of-hospital cardiac arrest.

Second, we need to re evaluate the role of the general practitioner in dealing with patients with putative ACS. Of course the Middlemore data tell us only the bare facts, and there may be legitimate reasons for delay in many cases. Many patients may delay calling the GP until after their symptoms have abated and are probably at low risk of arrest. Many practices may have defibrillators and can safely evaluate patients in their surgeries, ordering an emergency ambulance for those with continuing pain or with ST segment elevation in the electrocardiogram. Nevertheless, the Middlemore study raises important questions.

What happens when a patient with acute chest pain telephones his or her GP? If this happens during working hours, the call will be answered by a receptionist. Is the call referred immediately to the doctor, is the patient given the next available appointment, or are receptionists instructed to advise patients to call 111 for an emergency ambulance? If the call happens outside working hours does the recorded message from the surgery specifically advise patients with emergencies such as acute chest pain, breathlessness or bleeding to call the ambulance directly, or is some less specific advice given?

Nearly 20 years ago, the National Heart Foundation instigated a public educational campaign in New Zealand (*Heart Attack Action!*) which gave the message "Chest pain lasting more than 15 minutes. Call 111 for the ambulance." The aim of the campaign was to raise funds for all ambulances carrying cardiac patients to be equipped with defibrillators so that patients with ACS could be protected during their journey to hospital. But disappointingly only a minority (43%) of the Middlemore Hospital patients called the ambulance, and Māori, Pacific Islanders and socially deprived people were less likely to do so than less deprived European New Zealanders.

A recent publication from the UK Myocardial Ischaemia National Audit Project (MINAP)⁵ on more than 600,000 patients treated between 2003 and 2010 reported that nearly 60% had called the emergency services and the proportion doing so was greater in older than in younger patients.

We need a repeat of *Heart Attack Action!*, but this may not be sufficient because "one-off" campaigns of this type have had limited success.⁶⁻⁸ As with advice against smoking, public health education may take years or decades to be effective, and in other countries (Denmark and Germany in particular⁹) campaigns to reduce patient delay in calling for help are continuing. Here in New Zealand, advice on *Heart Attack Action!* could be usefully combined with public education on optimum dietary and lifestyle factors for primary prevention of coronary heart disease.

Competing interests: None declared.

Author information: Robin M Norris was cardiologist in charge of the Coronary-Care Unit at Green Lane Hospital and Honorary Professor of Cardiovascular Therapeutics at the University of Auckland School of Medicine until 1992. After 1992 he was an honorary consultant cardiologist at the Royal Sussex County Hospital, Brighton, UK from where he directed the UK Heart Attack Study and helped to set up the UK Myocardial Infarction National Audit Project (MINAP). He is now retired.

Correspondence: Dr Robin Norris, 17 Aberdeen Rd, Castor Bay, Auckland, New Zealand. Email: robinnorris@orcon.net.nz

References:

1. The United Kingdom Heart Attack Study Collaborative Group. Effect of time of onset to coming under care on fatality of patients with acute myocardial infarction: effect of resuscitation and thrombolytic treatment. *Heart* 1998;80:114-120.
2. Julian DG, Norris RM. Myocardial infarction: is evidence-based medicine the best? [Viewpoint]. *Lancet* 2002;359:1515-6.
3. Garofalo D, Grey C, Lee M, et al. Pre-hospital delay in acute coronary syndromes: PREDICT CVD-18. *N Z Med J* 2011;125(1348). <http://journal.nzma.org.nz/journal/125-1348/5020>
4. Swain AH, Barry T, Hoyle SR, et al. Outcomes from out-of-hospital cardiac arrest in the Wellington region of New Zealand. Does use of the Fire Service make a difference? *N Z Med J* 2011;12(1344): <http://journal.nzma.org.nz/journal/124-1344/4913/content.pdf>
5. Gale CP, Cattle BA, Woolston A, et al. Resolving inequalities in care? Reduced mortality in the elderly after acute coronary syndromes. The Myocardial Ischaemia National Audit Project 2003-2010. *Eur Heart J*. Advance Access published October 18, 2011.
6. Blohm MB, Hartford M, Karlson BW, et al. An evaluation of the results of media and educational campaigns designed to shorten the time taken by patients with acute myocardial infarction to decide to go to hospital. *Heart* 1996;76:430-4.
7. Gaspoz JM, Unger PF, Urban P, et al. Impact of a public campaign on pre-hospital delay in patients reporting chest pain. *Heart* 1996;76:150-5.
8. Luepker RV, Raczynski JM, Osgwanian S, et al. Effect of a community intervention on patient delay and emergency service use in acute coronary heart disease: the Rapid Early Action for Coronary Treatment (REACT) Trial. *JAMA* 2000;284:60-7.
9. Kristensen SD, Raatikainen MJP, Andersen K, et al. Cardiology practice in Europe in 2011: acute ST elevation myocardial infarction. *Circulation* 2011;124(15):f85-f90.

Proposed changes to the New Zealand Ethics Committees

Frank A Frizelle

The New Zealand House of Representatives' "Inquiry into improving New Zealand's environment to support innovation through clinical trials" was stimulated because of concerns that that New Zealand has lost its advantage as a "good place" to carry out clinical trials.

This Inquiry made 54 recommendations and concluded that "the main elements of the system can be put right at almost no cost, and we believe the returns for New Zealand patients, the health service, and the economy will be significant."

Their main recommendations were:

- Simplify and streamline ethical review processes.
- Promote collaboration between Government departments to coordinate the system.
- Develop a national health research action plan to foster innovation and commercialisation.
- Develop a framework for clinical trial research throughout district health boards (DHBs), to be facilitated by a hub.

The full report is available at:

http://www.parliament.nz/NR/rdonlyres/5AFC2393-05CF-44DB-8BF1-DD501A057408/193163/DBSCH_SCR_5154_InquiryintoimprovingNewZealandsenvi.pdf

The Government's response to this document was mostly supportive and progressed most of the recommendations.

The Government's full response is available at:

http://www.parliament.nz/NR/rdonlyres/5AFA2E8E-F8A3-486F-9F51-1C0FE455D4A8/200866/DBHOH_PAP_21990_GovernmentResponsetoReportoftheHea.pdf

As a result, the Ministry of Health is currently seeking feedback on draft procedural rules for health and disability ethics committees (HDECs), particularly:

- Rules clarifying that HDECs will be expected to check that proposed research has been appropriately peer reviewed, rather than conducting peer review themselves (see section 2 of the draft SOPs).
- Clarifying and reduce the scope of HDEC review (section 3).
- Imposing a 35-day timeline for full review (section 5), and a 15-day timeline for expedited review (section 6).

- Allowing some clinical trials to be reviewed through the expedited review pathway, on the basis of risk (section 6).
- Clarifying that localities (such as DHBs), rather than HDECs, are responsible for ensuring that local research governance issues are addressed (section 10).
- Clarifying when amendments to approved studies themselves require HDEC review (section 11).

The Ministry of Health is currently seeking your feedback on:

- Draft standard operating procedures for HDECs, and
- A draft HDEC application form.

Submissions close at 5pm, Friday 10 February 2012. Full details are available at:

<http://www.ethicscommittees.health.govt.nz/moh.nsf/indexcm/ethics-upcomingchanges>

The Ministry of Health's website also comments that it expects to announce further details of the online HDEC application system in early 2012, and that the number of HDECs will be reduced from 7 to 4, and their size from 12 to 8 members, as of 1 July 2012. The 7 current HDECs will continue to operate normally until this date.

These changes are important for researchers to understand and have input into. Although most researchers will be supportive of these changes, it is important for those involved to spend some time to appreciate what is being suggested.

Competing interests: None declared.

Author information: Frank A Frizelle, Editor, New Zealand Medical Journal.

Correspondence: Professor Frank A Frizelle, Department of Surgery, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand. Fax: +64 (0)3 3640352; email: frank.frizelle@cdhb.govt.nz

Pre-hospital delay in acute coronary syndromes: PREDICT CVD-18

Daniel Garofalo, Corina Grey, Mildred Lee, Daniel Exeter, Andrew J Kerr

Abstract

Aims To study pre-hospital delay, its components and determinants, in patients with acute coronary syndromes (ACS) admitted to Middlemore Hospital Coronary Care Unit.

Methods Consecutive ACS patients admitted between January 2009 and July 2010 were included. Pre-hospital delay was defined as the time from onset of worst symptom(s) to defibrillator availability: either ambulance arrival at the scene or time of hospital arrival (non-ambulance patients).

Results For 805 patients the median delay from symptom onset to defibrillator availability was 174 minutes. Half the cohort had a delay to defibrillator availability of >3 hours. The median delay was an hour longer for patients from areas of greatest deprivation compared with less deprived areas, [208 vs 149 min, respectively ($p=0.015$)], and 7 hours longer for non-ambulance vs ambulance patients, [553 vs 130 min ($p<0.001$)]. Māori, Pacific, Indian and those from areas of higher deprivation were less likely to travel to hospital by ambulance. Of ST-elevation myocardial infarction patients eligible for reperfusion, over two-thirds of the total delay between symptom onset and reperfusion occurred pre-hospital.

Conclusion Community intervention targeted at more disadvantaged communities and higher risk ethnic groups should be considered as part of an overall strategy to reduce disparity and improve cardiac outcomes.

Measurement of performance in the Health Service, with the object of lifting the performance of poorly performing sectors to match the standards of the best, is, as far as cardiac services are concerned, now firmly established in the UK.¹ This has not yet happened in New Zealand, although a start has been made,^{2,3} and a recent Ministry of Health publication,⁴ as an immediate priority, calls for commission of an “audit of access delay for acute coronary syndromes (ACS), stroke and transient ischaemic accidents (patient, health professional and ambulance delays) in several regions.”

Two thirds to three-quarters of deaths from ACS happen outside hospital,^{5,6} and in victims under age 55 this proportion may be more than 90%.⁵ There is also a strong inverse relationship between deaths prevented and delay in coming under care,⁷ the relationship being strongest when “care” is defined as paramedic rather than hospital care. This is because defibrillation administered by ambulance personnel has been shown to be as effective as in hospital, and in that study it was estimated that four times as many deaths had been prevented (up to 30 days after the event) by defibrillation than by thrombolytic treatment.

Two small studies in New Zealand have shown long delays between symptom onset and hospital arrival.^{8,9} We sought to define the components of pre-hospital delay, and its association with factors including ethnicity, socioeconomic status, distance from hospital, which professional help was first sought from and how patients reached hospital. In patients with ST-elevation myocardial infarction (STEMI), in whom considerable attention has been directed to minimise in-hospital delay to treatment, we looked at the relative pre- and in-hospital components of delay.

Methods

Study design—The study was conducted between 1 January 2009 and 31 July 2010. All patients with a final diagnosis of acute coronary syndrome admitted to Middlemore Hospital's Coronary Care Unit (CCU) from the community were included. Only first admissions during the study period were included. Patients referred from other hospitals, from outpatient clinics, or ACS that occurred in other hospital wards were excluded. Middlemore Hospital is the base hospital for 500,000 people living in the Counties-Manukau District Health Board (CMDHB) catchment in Auckland, New Zealand. Although the population is predominantly urban, there are rural communities within CMDHB up to 50 km from Middlemore Hospital.

From 2007 Middlemore CCU has routinely collected demographic, risk factor, diagnostic, investigation and in-hospital outcome data on all ACS patients using Acute Predict, an electronic database.¹⁰ Demographic and laboratory data are auto populated from hospital databases. Demographic data includes ethnicity and the NZ Deprivation Index 2006 (NZDep06). Clinical and angiographic data are entered by medical staff supervised by the research registrar. Data quality is supported by definition fields within the electronic form and 3-monthly scheduled audits of data quality. For the duration of this study, an additional "pre-hospital delay" data set was collected in new fields added within the existing ACS database.

Data were sourced from the ambulance report and the clinical notes. During the duration of the study the medical teams were specifically asked to inquire about time of onset of symptoms and actions in the pre-hospital phase. This information was gathered by a research registrar or a research nurse on day 0 or 1 post-admission to the CCU.

Data and definitions—All eligible patients were asked for the time and date of onset of their most severe symptom(s). Time of onset of symptoms was defined as time of onset of the chest pain or surrogate symptom (breathlessness, syncope, etc). If there was more than one symptom, or more than one episode of symptoms, the most severe symptom or episode would be chosen. If there was more than one episode of symptoms of equivalent severity, the one lasting for longer was recorded as time of onset. In particular cases where the presentation did not fit these definitions, the research registrar would decide whether an accurate estimate of onset time could be made.

For each patient the professional from whom help was first sought was recorded. The "delay to defibrillator availability" was also defined. This was the time elapsed between onset of symptoms and, either ambulance arrival at the scene (for those patients who first sought help from the ambulance service), or arrival at the hospital (for all the other patients). For patients using the ambulance service, times of despatch, arrival at the scene, departure from the scene, and time of arrival at the hospital were taken from the ambulance report. Patients coming to hospital via other means were divided into those who initially sought help from their regular general practitioner (GP), those who attended a primary care emergency centre (A and E), and those who self presented to hospital. In all these non-ambulance cases, time of arrival at the hospital was defined as the time when they were triaged by a nurse in the Emergency Department.

For this study, selected data recorded in the Acute Predict data set was used. This included patient demographics (age, gender and ethnicity) which auto-populated the electronic record from the hospital patient management system. For these analyses ethnicity was categorised in four groups: European, New Zealand Māori, Pacific, Indian, and Other. As a measure of socioeconomic status, the domicile code (where available and valid) for each patient was obtained from the hospital information system. This was linked to the New Zealand Deprivation 2006 index and reported as decile of deprivation from 1 (least deprived) to 10 (most deprived).

The NZDep06 is a small area index of deprivation that provides a score for each meshblock in New Zealand based on nine variables (reflecting eight types of deprivation).¹¹ Type of ACS diagnosis (ST elevation MI [STEMI], Non-ST elevation MI [NSTEMI] and unstable angina [USA]) at discharge was used.

For patients with STEMI who had in-hospital reperfusion, either pharmacological thrombolysis or primary percutaneous coronary intervention (PCI), the in-hospital delay to reperfusion data was obtained (door-to-needle and door-to-balloon time, respectively).

The individual patient meshblock data was used to estimate the distance that an ambulance would need to travel by road from the patient's home to Middlemore Hospital.

Statistical analysis—Statistical analysis was performed using STATA v10.0 software. Numbers and percentages of those in different population and diagnostic groups who had arrived at the hospital via ambulance and self transport were calculated and compared using the chi-squared and analysis of variance statistics. Differences in the time between onset of symptoms and the availability of defibrillation between population and diagnostic groups were examined by calculating medians and interquartile ranges, and these were compared using a non-parametric k-sample test on the equality of medians.

A subanalysis of the onset of symptoms to receipt of reperfusion therapy was performed on those who had experienced a STEMI. These patients were grouped according to reperfusion therapy received (none, thrombolysis and PCI). The median and interquartile range of the times between onset of symptoms and arrival at hospital, and times between arrival at the hospital and reperfusion, were calculated for those who received thrombolysis and PCI.

The study was approved by the Northern X Regional Ethics Committee (NTX/09/04/EXP).

Results

There were 1068 consecutive first admissions with an ACS event. Of these, no data was collected for 176 patients due to the admission occurring predominantly over a weekend or holiday period when research staff were not available.

Of the 892 patients available for the study, 24 were ineligible, and a further 63 patients could not give a precise estimate of time of onset of symptoms. There were therefore 805 people for whom the time from symptom onset to defibrillation availability could be calculated. These people comprise the final cohort used for analysis.

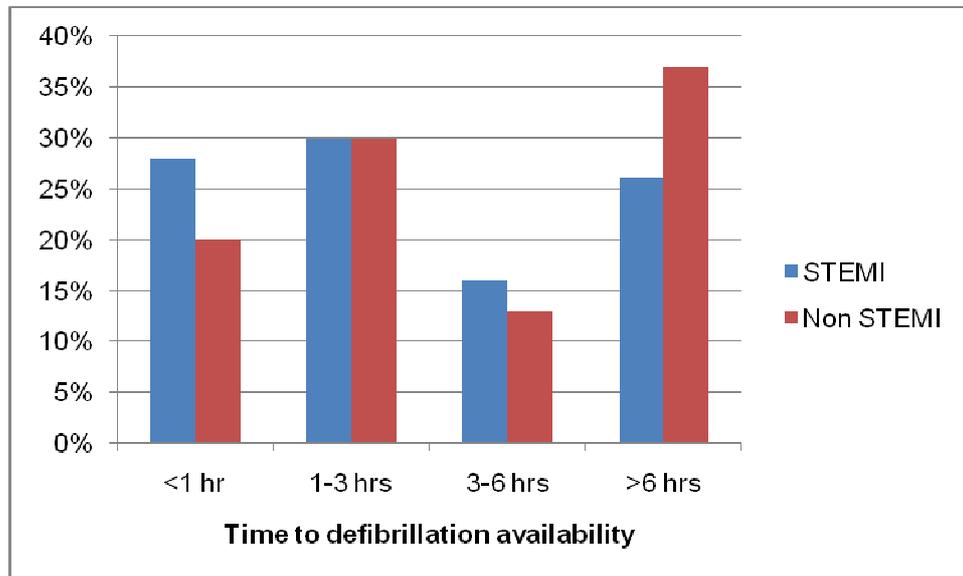
Twenty patients (2%) had a cardiac arrest prior to admission or in hospital, and eight people (1%) died in-hospital.

Overall delay (Figure 1)—Overall median time delay from symptom onset to defibrillator availability was 174 minutes (min), and to hospital arrival was 208 min. Less than a quarter of the cohort had defibrillator availability within an hour of symptom onset, another quarter achieved this under 3 hours, but for nearly a third the delay was greater than 6 hours. Delay for STEMI patients was slightly less than for NSTEMI, but there was substantial overlap.

Ambulance compared with self transport—Table 1 shows demographic and clinical information according to whether patients were transported to hospital by ambulance or self-transport.

Three-quarters of the cohort were men; mean age was 61.3 years (SD 12.4). Half the cohort identified themselves as European, 18% Pacific, 12% Māori and 13% Indian. Almost half of the cohort (48%) lived in areas of high socioeconomic deprivation (NZDep06 9–10).

Figure 1. Time to defibrillation availability in STEMI and NSTEMI patients



Ambulance transport was used by 73% of patients. European patients (82%) were more likely to travel by ambulance than other ethnic groups [Māori (66%), Pacific (63%), and Indian (65%)]. Those from more deprived areas were less likely to come by ambulance. Those coming by ambulance were also slightly older and more likely to have a final diagnosis of STEMI. The time delay from symptom onset to the availability of potentially life-saving defibrillation (ambulance attendance or hospital arrival for those self transported) was approximately four times longer in those not coming by ambulance (130 vs 553 min, respectively, $p < 0.001$). Correspondingly, the time from symptom onset to hospital arrival was markedly longer in those not coming by ambulance.

Delay according to demographics and diagnosis (Table 2 and Figure 1)—For the whole cohort, patients from the most deprived areas (NZDep06 9-10) took an hour longer to come under defibrillator protection than those from less deprived areas (median delay 208 and 149 min, respectively). There were no significant differences by ethnic group, age group or gender. There was a trend towards shorter times in patients with STEMI compared with other ACS, but the median delay in these patients was still just over 2 hours. When this analysis was repeated for delay from onset to hospital arrival, the results were similar (data not shown).

Table 1. Characteristics of pre-hospital delay cohort by method of transport to hospital

Variables	Total cohort (n=805), Col %	Ambulance (n=589), Row %	Self-transport (n=216), Row %	P-value for difference between ambulance & self transport
Gender and age				
Male, n (%)	595 (74%)	433 (73%)	162 (27%)	0.671
Female, n (%)	210 (26%)	156 (26%)	54 (26%)	
Mean age, mean (SD)	61.2 (12.5)	62.2 (12.8)	58.7 (11.1)	0.0004
Ethnicity				
European, n (%)	386 (50%)	318 (82%)	68 (18%)	<0.0001
Māori, n (%)	94 (12%)	62 (66%)	32 (34%)	
Pacific, n (%)	141 (18%)	98 (63%)	52 (37%)	
Indian, n (%)	102 (13%)	66 (65%)	36 (35%)	
Other, n (%)	43 (6%)	30 (70%)	13 (30%)	
Deprivation**				
NZDep06 1-8	396 (52%)	306 (77%)	90 (23%)	0.017
NZDep06 9-10	359 (48%)	250 (70%)	109 (30%)	
Final diagnosis				
USA, n (%)	44 (5%)	31 (70%)	13 (30%)	0.001
Non-STEMI, n (%)	582 (72%)	408 (70%)	174 (30%)	
STEMI, n (%)	179 (22%)	150 (84%)	29 (16%)	
Distance to hospital				
<5 km	159 (20%)	106 (67%)	53 (33%)	0.0003
5–10 km	212 (26%)	143 (67%)	69 (33%)	
10–20 km	225 (28%)	171 (76%)	54 (24%)	
>20 km	209 (26%)	169 (81%)	40 (19%)	
Median time to defibrillation (IQR), min	174 (68, 646)	130 (56, 392)	553 (142, 1700)	<0.0001
Median time to hospital (IQR), min	208 (100, 668)	172 (92, 428)	553 (142, 1700)	<0.0001
Mean time to defibrillation (SD), min	826 (2164)	575 (1508)	1511 (3264)	Mean difference*= 936 (604–1268)
Mean time to hospital (SD), min	852 (2161)	610 (1508)	1511 (3264)	Mean difference*= 901 (569–1232)

*Mean difference (95% confidence interval) between self transport and ambulance groups.

**NZDep06 data missing in 50 people.

Table 2. Median time to defibrillation (interquartile range) for different population/clinical groups

Group	Total cohort (n=805)	P-values	Self transport (n=216)	P-values	Ambulance (n=589)	P-values
Age	174 (68, 646)		553 (142, 1700)		130 (56, 392)	
<50y (n=162)	173 (70, 684)	0.890	558 (172, 1394)	0.255	134 (56, 328)	0.988
50-65y (n=335)	178 (70, 640)		510 (140, 1680)		131 (60, 372)	
>65y (n=308)	165 (61, 641)		665 (148, 2098)		128 (50, 404)	
Gender						
Men (n=595)	164 (64, 646)	0.453	538 (140, 1700)	0.875	120 (48, 408)	0.072
Women (n=210)	184 (84, 676)		630 (148, 1804)		155 (76, 335)	
Ethnicity						
Euro (n=386)	173 (62, 656)	0.627	987 (287, 2091)	0.026	133 (50, 404)	0.901
Māori (n=94)	226 (86, 724)		479 (190, 2454)		146 (56, 404)	
Pacific (n=141)	164 (64, 622)		312 (84, 770)		132 (56, 372)	
Indian (n=102)	163 (76, 774)		591 (161, 1475)		107 (60, 304)	
Other (n=43)	164 (58, 516)		350 (208, 1430)		123 (40, 196)	
Final diagnosis						
USA (n=44)	195 (61, 558)	0.097	480 (138, 2150)	0.01	164 (40, 336)	0.640
NSTEMI (n=582)	189 (76, 760)		615 (220, 1804)		129 (58, 408)	
STEMI (n=179)	128 (50, 378)		120 (64, 536)		130 (44, 360)	
Deprivation						
NZDep06 1-8 (n=396)*	149 (65, 596)	0.015	792 (150, 2084)	0.076	127 (56, 322)	0.150
NZDep06 9-10 (n=359)	208 (76, 732)		510 (140, 1430)		163 (62, 570)	
Distance from hospital						
<5 km (n=159)	162 (72, 584)	0.909	388 (140, 864)	0.883	115 (56, 328)	0.845
5-10 km (n=212)	175 (76, 766)		560 (150, 2084)		134 (58, 436)	
10-20 km (n=225)	164 (58, 576)		557 (120, 1670)		130 (44, 410)	
>20 km (n=209)	186 (68, 640)		653 (222, 2216)		138 (58, 336)	

Delay according to professional first contacted (Table 3)—Patients who called an ambulance directly, 43% of the cohort, had markedly shorter median delays to defibrillation availability than those who presented directly to hospital (76 and 220 min, respectively). The longest delays were in the over 40% who initially presented to their GP or an A and E clinic (556 and 300 min, respectively).

Table 3. Median onset (IQR) of symptoms to defibrillation availability according to professional help first sought

Professional help sought	Number (%)	Median time (IQR), min	P-value for differences between groups	Mean time (SD), min
Ambulance	348 (43%)	76 (34, 195)	<0.0001	269 (769)
Hospital by self referral	109 (14%)	220 (84, 536)		649 (1749)
GP	275 (34%)	556 (162, 1716)		1590 (3201)
Private A and E	65 (8%)	300 (110, 740)		791 (1391)
Other	5 (1%)	2334 (764, 3810)		2241 (1845)
Unavailable/don't know	3 (<1%)			

Components of delay in patients transferred by ambulance (Table 4)—For those patients transferred by ambulance, the major component of delay was decision time, between symptom onset and ambulance despatch. A median of 2 hours elapsed before the decision was made. In contrast, the median time between ambulance despatch and arrival at the scene was just 8 minutes, and the total time from despatch to hospital arrival was a median of 40 minutes. The distance from the hospital contributed on a very small increase in median time from despatch to arrival at the scene, but a greater delay from departure from the scene to hospital arrival.

STEMI delay pre- and in-hospital (Table 5)—Nearly a third of patients with STEMI were not offered potentially lifesaving reperfusion therapy. This was related to an over 6-hour median delay from symptom onset to hospital arrival. Patients who had reperfusion therapy still had a median pre-hospital delay of 2 hours, which is much longer than the in-hospital delays to thrombolysis or primary PCI (median door-to-needle and door-to-balloon times 40 and 84 min, respectively). Over two-thirds of the total delay between symptom onset and reperfusion therapy occurred in the pre-hospital phase.

Table 4. Median times (IQR) in minutes between ambulance departure and arrival at hospital according to distance of home residence from hospital (n=589)

Start to destination	Total	<5km	5-10km	10-20km	>20km	P-value
Symptom onset to ambulance despatch	120 (48, 396)	106 (44, 320)	120 (52, 420)	118 (36, 400)	124 (48, 324)	0.93
Despatch to arrival at scene	8 (4, 12)	6 (4, 8)	8 (6, 12)	8 (4, 12)	8 (4, 18)	<0.0001
Ambulance arrival to departure	14 (8, 20)	12 (8, 16)	14 (8, 18)	14 (10, 20)	16 (8, 24)	0.01
Ambulance departure from scene to hospital	18 (12, 28)	8 (6, 12)	16 (12, 18)	20 (16, 26)	30 (20, 38)	<0.0001
Symptom onset to hospital	172 (92, 428)	135 (86, 344)	164 (90, 470)	176 (88, 448)	188 (108, 402)	0.35

Table 5. Median times (IQR) in the path from onset of symptoms to reperfusion in STEMI patients

Revascularisation	Onset to hospital	Door to reperfusion	Onset to reperfusion
PCI (n=44)	124 min (79, 426)	84 min (66, 116)	272 min (164, 519)
Thrombolysis (n=80)	122 min (65, 225)	40 min (25, 62)	180 min (120, 272)
No reperfusion (n=55)	332 min (128, 944)	–	–

Discussion

Half of all patients with ACS who were admitted to Middlemore CCU spent at least 3 hours of the early and potentially deadly phase of their illness in the community without access to a cardiac defibrillator. This period corresponds to the period when availability of prompt defibrillation is most likely to save lives.⁵ A third of these patients had a greater than 6 hour delay, including a quarter of those with STEMI. When an ambulance was called the ambulance response times were short. Most of the delay occurred prior to calling for an ambulance.

Patients who called an ambulance directly had the shortest delays, and those who initially contacted their GP or an A and E clinic had much longer delays. Although three-quarters of the cohort were transported by ambulance, only 43% of the cohort called an ambulance directly. The others came by ambulance after consulting their GP or an A and E centre. Māori, Pacific and Indian patients, and those from areas of greatest deprivation were less likely to come to hospital by ambulance. Although the mode of transport is likely to be partly determined by symptom severity, it is unlikely that symptom severity varied between ethnic groups. Rather, this lower rate of ambulance contact is likely to be due to a number of factors including cultural, educational and financial reasons. In New Zealand there is a charge for calling an ambulance, which is an obvious disincentive for those from poorer areas.

Apart from the type of transport used, the only other significant predictor of pre-hospital delay was socioeconomic status estimated by the NZDep06 score. Patients from more deprived areas took an hour longer to defibrillator availability. This association between socioeconomic status and longer delays has also been observed in the United States.^{12,13} This delay during a critical phase of the illness may be an important contributor to the known higher cardiovascular mortality rates in poorer New Zealanders.¹⁴

Of the patients presenting with STEMI, a third presented too late to be offered reperfusion therapy, exposing them to both high risk of lethal arrhythmia and poorer late outcomes due to more extensive myocardial infarction. Of those presenting early enough to be offered reperfusion therapy, the ambulance pre-hospital transport times were very good, and in-hospital door-to-reperfusion therapy times were mostly within international targets. Whilst some small improvement in those and the ambulance transport times may be achieved, by far the biggest modifiable delay for all STEMI patients is the delay in seeking professional help.

What are the implications of this delay? Improved prevention and treatment options have led to a marked reduction in age-adjusted cardiovascular mortality rates in Western countries over the last 40 years, but these rates are now levelling off. Whilst some gains will still be made by improving utilisation of evidence-based therapy and timeliness of reperfusion in hospital, the greatest opportunities for further improvement in outcomes are likely to be made in the community, including improved primary and secondary prevention and reducing delays to appropriate management.

Strategies to reduce pre-hospital delays can improve outcomes by reducing both time to defibrillation access in all ACS, and time to reperfusion in the subgroup with STEMI. Because the benefits of early defibrillation availability occur for all ACS and reperfusion gains occur only in the fifth of patients with STEMI, the greatest benefits of getting ACS patients under earlier paramedic care will probably relate to earlier defibrillation availability.

What can be done? Intervention studies to reduce pre-hospital delays have had mixed results. Two European community intervention studies in the 1980's and 90's reduced the median delay to hospitalisation in myocardial infarction from 180 to 138 min, and from 196 to 144 min, respectively.^{15,16} In contrast, a similar study from the United States¹⁷ had shorter delays at baseline and found no significant reduction in delay after community intervention programmes (144 to 138 min).

Another United States study which randomised communities to community intervention versus no intervention also found no effect on delay (140 min in both groups), although they did increase the proportion of patients calling an ambulance.¹⁸ It is interesting that in the Physician's Health Study,¹⁹ involving presumably very health literate individuals, the median delay was 114 min.

On the basis of this and the US studies it has been suggested that it may be difficult to reduce delay to less than 2 hours in view of the varying symptomatic presentations and psychological factors involved in making the decision to call for help.¹⁸ However, in our cohort the median delay to hospital arrival was 208 min, which is at a level seen pre-intervention in the successful European studies.

Furthermore, people from poorer areas have an hour greater delay than those from wealthier areas. These findings suggest that a community intervention programme targeted at more disadvantaged communities and higher risk ethnic groups which encourages earlier call for help directly to the ambulance service may be a useful part of an overall strategy to reduce disparity and improve cardiac outcomes

Study limitations. We have only included patients admitted to CCU. Patients admitted to other wards, usually with multiple co-morbidities or with significant functional or cognitive impairment, were not part of this analysis. We only have information on those who survived to CCU admission. If the median delay time is 3 hours, it is likely that there were patients who delayed calling for help for a similar time who died suddenly. We have no information regarding this number or details of their delay from this study. For those patients who initially contacted their GP or an A and E centre we did not collect the delay between that initial contact and the subsequent time of call for an ambulance or hospital arrival. This would be useful to collect as a sub-study in any future research.

For this study the time of the call for help was estimated from the time of ambulance despatch. We did not have access to the actual call time, but for ACS patients in the Auckland region ambulance despatch typically occurs within a few minutes of receipt of the call (personal communication, Tony Smith, St John's Ambulance Service Clinical Director). The NZDep06, the measure of socioeconomic status used, is an area-based measure and does not take into account all determinants of socioeconomic status at an individual level. As a result it is possible that the effects of socioeconomic status may have been underestimated.²⁰

In this study the distance from hospital was defined as the distance from home estimated using the NZ mesh block data. Some patients will have made the call for help from GP surgeries or from their workplace, which will lead to some underestimation of the effect of this variable. Some A and E centres may have had a defibrillator, which would mean the median delay time to defibrillation availability for patients presenting initially to these centres might be slightly overestimated.

Conclusions

There are significant potentially modifiable pre-hospital delays in patients with ACS. These delays are most marked in those groups known to have worse cardiovascular outcomes, and are likely to be a significant contributor to those poorer outcomes. Consideration should be given to developing a community intervention programme

targeting at-risk communities to encourage earlier call for help directly to the ambulance service, to reduce disparity and improve cardiac outcomes.

Competing interests: None declared.

Author information: Daniel Garofalo, Cardiology Registrar and Research Fellow, Middlemore Hospital, South Auckland; Corina Grey, Public Health Medicine Registrar, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland; Mildred Lee, Data Analyst, Counties Manukau District Health Board, South Auckland; Daniel Exeter, Senior Lecturer, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland; Andrew J Kerr, Cardiologist and Clinical Head of Cardiology, Middlemore Hospital, Counties Manukau District Health Board, South Auckland

Acknowledgements: We acknowledge Dr Robin Norris for his assistance in designing and implementing this study. We also thank Dianne Caveney for her assistance with data collection, Dr Tony Smith from St John's Ambulance service who provided advice, as well as The National Heart Foundation and the Middlemore Cardiology Research Fund who provided financial support for this study.

Correspondence: Andrew Kerr, c/o Dept of Cardiology, Middlemore Hospital, Otahuhu, Auckland 93311, New Zealand. Email: Andrew.Kerr@middlemore.co.nz

References:

1. Birkhead JS, Walker L, Pearson M, et al. Improving care for patients with acute coronary syndromes: initial results from the National Audit of Myocardial Infarction Project (MINAP). *Heart*. 2004;90(9):1004-9.
2. Ellis C, Gamble G, French J, et al., Management of patients admitted with an Acute Coronary Syndrome in New Zealand: results of a comprehensive nationwide audit. *New Zealand Medical Journal*. 2004;117(1197):U953.
3. Ellis C, Gamble G, Hamer A, et al. Patients admitted with an acute coronary syndrome (ACS) in New Zealand in 2007: results of a second comprehensive nationwide audit and a comparison with the first audit from 2002. *New Zealand Medical Journal*. 2010;123(1319):25-43.
4. Ministry of Health, Diabetes and cardiovascular disease: Quality improvement plan. Wellington, NZ; 2008.
5. Norris RM. Fatality outside hospital from acute coronary events in three British health districts, 1994-5. United Kingdom Heart Attack Study Collaborative Group. *BMJ*. 1998;316(7137):1065-70.
6. Chambless L, Keil U, Dobson A, et al. Population versus clinical view of case fatality from acute coronary heart disease: results from the WHO MONICA Project 1985-1990. Multinational MONItoring of Trends and Determinants in CARdiovascular Disease. *Circulation*. 1997;96(11):3849-59.
7. The United Kingdom Heart Attack Study Collaborative Group, Effect of time from onset to coming under care on fatality of patients with acute myocardial infarction: effect of resuscitation and thrombolytic treatment. The United Kingdom Heart Attack Study (UKHAS) Collaborative Group. *Heart*. 1998;80(2):114-20.
8. Tanner H, Larsen P, Lever N, et al., Early recognition and early access for acute coronary syndromes in New Zealand: key links in the chain of survival. *New Zealand Medical Journal*. 2006;119(1232):U1927.
9. Tang EW, Wong C-K, Herbison P. Community hospital versus tertiary hospital comparison in the treatment and outcome of patients with acute coronary syndrome: a New Zealand experience. *New Zealand Medical Journal*. 2006;119(1238):U2078.

10. Kerr AJ, Looi JL, Garofalo D, et al. Acute Predict: a clinician-led cardiovascular disease quality improvement project (Predict-CVD 12). *Heart, Lung & Circulation*. 2010;19(5-6):378-83.
11. White P, Gunston J, Salmond C, et al. Atlas of socioeconomic deprivation in New Zealand, NZDep 2006. Ministry of Health: Wellington; 2008.
12. Schmidt SB, Borsch MA. The prehospital phase of acute myocardial infarction in the era of thrombolysis. *American Journal of Cardiology*. 1990;65(22):1411-5.
13. Sheifer SE, Rathore SS, Gersh BJ, et al. Time to presentation with acute myocardial infarction in the elderly: associations with race, sex, and socioeconomic characteristics. *Circulation*. 2000;102(14):1651-6.
14. Hay D. Cardiovascular disease in New Zealand 2004. A summary of recent statistical information. The National Heart Foundation of New Zealand: Auckland; 2004.
15. Gaspoz JM, Unger PF, Urban P, et al. Impact of a public campaign on pre-hospital delay in patients reporting chest pain. *Heart*. 1996;76(2):150-5.
16. Herlitz J, Blohm M, Hartford M, et al. Follow-up of a 1-year media campaign on delay times and ambulance use in suspected acute myocardial infarction. *European Heart Journal*. 1992;13(2):171-7.
17. Ho MT, Eisenberg MS, Litwin PE, et al. Delay between onset of chest pain and seeking medical care: the effect of public education. *Annals of Emergency Medicine*. 1989;18(7): 727-31.
18. Luepker RV, Raczynski JM, Osganian S, et al., Effect of a community intervention on patient delay and emergency medical service use in acute coronary heart disease: The Rapid Early Action for Coronary Treatment (REACT) Trial. *JAMA*. 2000;284(1):60-7.
19. Ridker PM, Manson JE, Goldhaber SZ, et al. Comparison of delay times to hospital presentation for physicians and nonphysicians with acute myocardial infarction. *American Journal of Cardiology*. 1992;70(1):10-3.
20. Blakely T, Pearce N. Socio-economic position is more than just NZDep. *New Zealand Medical Journal*. 2002;115(1149):109-11.

Uptake of pulmonary rehabilitation in New Zealand by people with chronic obstructive pulmonary disease in 2009

William M M Levack, Mark Weatherall, Julie C Reeve, Christina Mans, Antonia Mauro

Abstract

Aims To estimate of uptake of pulmonary rehabilitation (PR) by people with chronic obstructive pulmonary disease (COPD) in New Zealand in 2009.

Method A postal survey sent to all District Health Boards (DHBs), Primary Health Organisations (PHOs), and other non-government organisations (NGOs) identified as providers of PR. The survey requested information on the characteristics of PR programmes, estimates of the total number of people with COPD who were offered PR, entered PR, and completed PR in 2009.

Results In 2009 PR was provided in 19 of 21 DHB regions by 23 organisations (16 DHBs, five PHOs, one DHB/PHO partnership, and 1 NGO). Twenty-one of these 23 organisations (91%) responded to the survey. In total, 2569 people with COPD were offered PR, 1786 entered PR, and 1378 completed a PR programme in 2009.

Conclusions There is a marked shortfall between the national levels of provision of PR and the prevalence of COPD, with less than 1% of people with COPD participating in PR each year in New Zealand. Incentives, leadership and coordination of services are required at a national level to increase the uptake of PR.

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death and contributes significantly to disability in New Zealand (NZ).¹ The direct health care costs for NZ of COPD in 2003 were estimated to be between \$128 million and \$192 million.² COPD presents a particular burden for Māori, who experience impairments resulting from COPD up to two decades earlier than non-Māori, have a prevalence of COPD twice as high compared to non-Māori, and have higher rates of hospitalisation and death associated with COPD.³

Estimates of the prevalence of COPD in NZ have varied widely. The 2006/2007 NZ Health Survey found that 6.6% (95%CI 5.9–7.3%) of adults over 45 years recalled being told by a doctor that they had chronic bronchitis, emphysema, or COPD.⁴ However, a more recent survey based on clinical assessment of a random sample of residential New Zealanders reported a much higher prevalence.⁵

Using the Global Initiative for Chronic Obstructive Lung Disease (GOLD) definition of COPD, the only New Zealand-based prevalence study to have done so, Shirtcliffe and colleagues reported that 14.2% (95%CI 11.0–17.0%) of adults aged over 40 years had COPD, and further that self-report of having received a doctor's diagnosis of COPD had little relationship with an objective diagnosis.⁵

Based on the figures of Shirtcliffe and colleagues⁵ and NZ population statistics,⁶ it is likely that between 213,400 and 329,800 New Zealanders over 40 years had COPD in

2009, according to the GOLD criteria. Shirtcliffe and colleagues also demonstrated however, that estimates of COPD prevalence differ depending on which clinical criteria are used.

Using a lower limit of normal (LLN) of forced expiratory volume in one second/forced vital capacity as opposed to a fixed ratio of 0.7 to diagnose airflow obstruction (the American Thoracic Society and European Respiratory Society Joint Task Force recommended criteria for diagnosis of COPD), Shirtcliffe and colleagues reported a lower prevalence of COPD: 9.0% (95%CI 6.7–11.3%) of people over 40 years.⁵ This would equate to an estimate of 129,980 to 219,220 New Zealanders over 40 years having COPD in 2009.

Pulmonary rehabilitation (PR)—a multidisciplinary programme usually comprised of 6 to 16 weeks of group-based exercise and education in outpatient or community centres—is one of the few interventions that have been consistently shown to enhance physical function and quality of life (QOL) in people with COPD.

The most recent systematic review of randomised controlled trials indicated that PR results in clinically and statistically significant improvements in quality of life, and statistically significant improvements in exercise capacity.⁷ In people receiving PR after a hospital admission for an exacerbation of COPD, evidence from a systematic review of randomised controlled trials has demonstrated that PR results in a significant reduction in risk of rehospitalisation (pooled odds ratios of 0.13 over 34 weeks) and death (pooled odds ratios of 0.3 over 107 weeks).⁸ PR is thus considered one of the key strategies for management of COPD from a public health perspective.⁹

However, despite the public and personal cost of COPD and the known benefits of PR, little information exists regarding the uptake of PR in NZ. Broad and Jackson suggested that in 2003 it was ‘doubtful if more than 2% of new COPD patients, or 10% of existing patients in New Zealand access such a programme’ (p. 30),² although the origin of these figures is unclear as no methodology was stated.

Overseas reports have indicated a lower uptake, with only 1–2% of all people with COPD having access to PR in Australia,¹⁰ Canada,¹¹ and the UK.¹² From a recent audit of respiratory services in NZ it is known that PR is one of the interventions most frequently highlighted by District Health Boards (DHBs) when discussing their strategies for management of chronic respiratory disorders.¹³

In 2009, 14 of 15 DHBs who completed a survey on management of COPD reported that they ran a PR programme.¹⁴ However, other than these broad indications of service provision, no empirical data exists on the exact number of New Zealanders with COPD who attend PR each year.

The primary aim of this current study was to develop a good estimate of the total uptake of PR by New Zealanders with COPD in 1 year (specifically, 2009), including the number of people who:

- Were offered a place on a PR programme,
- Entered a PR programme, and
- Completed a PR programme.

A secondary aim of this study was to gather further details regarding the characteristics of existing pulmonary rehabilitation services in NZ.

Method

Research design—A postal survey was undertaken involving all providers of pulmonary rehabilitation in NZ. The Multi-regional Ethics Committee confirmed that formal ethical approval was not required for this low risk observational study of health care providers.

Identification of providers—Services were eligible to participate in this study if they provided a PR programme in 2009 consistent with criteria in the latest Cochrane Review on PR⁷: structured exercise for people with chronic respiratory disease over a period of at least 4 weeks, delivered by a health professional in inpatient, outpatient or community settings, with or without additional education sessions or psychosocial support. Programmes that involved patient education only were excluded from the survey.

Prior to undertaking this survey, we created a database of all providers of PR in NZ. In order to identify all possible providers, we approached all DHBs and all Primary Health Organisations (PHOs) to ask about the relevant programmes, if any, they ran. We made contact with the respiratory services in all 21 DHBs (100%) and with 73 of the 81 PHOs (90%) that existed in 2009. Eight PHOs (10%) did not respond to multiple requests by phone, electronic mail or post regarding whether or not they provided PR, and, based on conversations with the DHB providers in their region, were assumed not to have been providers of PR in 2009.

We also contacted all non-government organisations (NGOs) listed by the Asthma Foundation New Zealand as providers of services for people with COPD. We asked about who, if anyone, within these organisations ran, or coordinated, a pulmonary rehabilitation programme, and their contact details. Additionally we made links with potential providers through professional networks among nurses and physiotherapists who specialised in respiratory services. Twenty three organisations were thus identified as providers of PR in 2009 (16 DHBs, five PHOs, one DHB/PHO partnership, and one NGO) and included for data collection.

Data collection—A survey was sent by mail in October 2010 to each of the 23 organisations identified as being a provider of PR in 2009. The survey consisted of a structured questionnaire seeking information about:

- The characteristics of the PR programme(s) offered (e.g. the location, frequency, duration, and design of programmes)
- The types of patient data collected (i.e. whether data was collected on age, ethnicity, lung function physiology) and how this data was collected
- The method used for establishing or confirming a diagnosis of COPD among attendees of PR programmes (if any)
- The type and timing of outcome measures used to evaluate patient progress, and
- The providers' estimates of the number of people with COPD who a) were offered, b) entered, and c) completed a PR programme in 2009.

In order to evaluate the accuracy of the providers' estimates of the provision of PR in 2009 we also sought information on what these estimates were based on (e.g. actual patient records, average PR class sizes, or a 'best guess' of patient numbers based on the respondents' recall). As definitions were likely to differ regarding what might constitute 'completion' of a PR programme, we specified that for the purpose of this survey 'completion' referred to 'the number of people who entered minus the number of people who stopped attending before finishing the PR programme'. (For a full copy of the questionnaire used please contact the corresponding author.)

The survey was piloted by respiratory services in two DHBs, and feedback from these providers was used to revise the structure of the questionnaire before the final version was mailed out. The organisations involved in the pilot also completed the final version of the survey for data collection purposes. In order to enhance the response rate for the survey, we kept the questionnaire short (concentrating on the primary aims of the study), we offered a prize draw (for a contemporary textbook on PR) for services who completed the questionnaire, and we followed up with regular reminders by electronic mail and phone to all potential respondents. Data was analysed using descriptive statistics.

Results

Survey responses—We received data from 21 of the 23 organisations that provided pulmonary rehabilitation in 2009—a 91% response rate. Two PHO providers did not respond.

Regional service delivery—PR was provided in 19 of the 21 DHB regions in 2009. Twelve DHBs (57%) were reported to be the sole providers of PR in their region. Two DHB regions (10%) had a PHO as the sole PR provider. In one DHB region (5%), PR was provided as part of a partnership project between the local DHB and PHO.

In four further DHB regions (19%) the work of providing PR was divided up between the DHB and at least one PHO, with each organisation being responsible for subpopulations within the DHB region (and one PHO providing for a subpopulation of people from two DHBs). One of these regions had three providers of PR—the DHB, a PHO, and a NGO—all providing services to separate subpopulations within the DHB region. In two DHB regions (10%) no PR was provided in 2009, although one of these DHBs had since established a PR programme in 2010.

Characteristics of PR programmes—A summary of the characteristics of the PR programmes provided by the 21 organisations responding to the survey are shown in Table 1. These 21 organisations offered PR programmes in a range of locations including hospital gyms, community gyms, community centres, marae, and individuals' homes. There was a high degree of similarity in the structure of programmes offered, with the majority of organisations (86%; 18/21) offering PR programmes consisting of 6 to 10 weeks of twice weekly exercise, plus education, with a period of assessment before and after the formal period of exercise.

All 21 providers collected data using the 6-Minute Walking Test and 81% (17/21) collected data on QOL using a disease-specific tool—either the Chronic Respiratory Disease Questionnaire or the St George Respiratory Questionnaire. Many providers also collected other forms of standardised outcome data on exercise capacity, severity of breathlessness, depression and anxiety, and body mass or weight. The majority of PR providers (86%; 18/21) collected data on patient ethnicity, and did so largely by asking patients open-ended questions about their ethnicity or by gathering information from existing patient records.

Many organisations also offered variations to the standard programme to better meet the needs of their patient populations. Examples of this included offering programmes consisting of once weekly rather than twice weekly exercise for people who struggled to adhere to more intensive programmes or offering 'rolling' programmes where patients did not need to all start at the same time but could be admitted and discharged from the programme at times that suited them.

One organisation offered an additional 4 weeks of exercise for people identified as likely to particularly benefit from an extended programme. Some incorporated transition to an ongoing community-based exercise group in a public gym or other community facility as part of discharge from the structured PR programme.

Table 1. Characteristics of PR programmes in organisations responding to the survey

Location of PR programmes	Single location only		10/21 (48%)	
	Multiple locations		11/21 (52%)	
	Group programme in a hospital gym		15/21 (71%)	
	Group programme in private or community gyms		6/21 (29%)	
	Group programme in a community centre		6/21 (29%)	
	Group programme in a Marae		2/21 (10%)	
	Individual programmes in hospital gym, home or community		12/21 (57%)	
Method for confirming a diagnosis of COPD	GOLD criteria		11/21 (52%)	
	American Thoracic Society and European Respiratory Society Joint Task Force criteria		1/21 (5%)	
	Lung function tests without specific diagnosis criteria		3/21 (14%)	
	Diagnosis of referring physician		5/21 (24%)	
Patient signs and symptoms without lung function testing		1/21 (5%)		
Structure of PR programmes	Enrolment	'Discrete' PR programme, with set start and end date for whole group	16/21 (66%)	
		'Rolling' PR programmes, with patients joining and leaving at different times	4/21 (19%)	
		Both 'rolling' and 'discrete' programmes	1/21 (5%)	
	Duration	8 weeks	15/21 (71%)	
		6–10 weeks	20/21 (95%)	
		Continuous (patients never formally discharged)	1/21 (5%)	
	Exercise	Twice weekly classes	20/21 (95%)	
		Once weekly classes	1/21 (5%)	
	Education	Run concurrently with exercise classes	21/21 (100%)	
	Outcome evaluation	Pre- and post-PR outcomes collected		18/21 (86%)
		Follow up data (1, 4, 6, or 12 months after completion of PR)		4/21 (19%)
		6-Minute Walk Test		21/21 (100%)
		Incremental Shuttle Walk Test		4/21 (19%)
		Timed Up and Go		1/21 (5%)
		Timed Sit-to-Stand		1/21 (5%)
		Borg Dyspnoea Scale		15/21 (71%)
		Medical Research Council Dyspnoea Score		7/21 (33%)
		Chronic Respiratory Disease Questionnaire		15/21 (71%)
		St. George Respiratory Questionnaire		2/21 (10%)
		Hospital Anxiety and Depression Scale		12/21 (57%)
		Chronic Disease Questionnaire		1/21 (5%)
Body Mass Index		15/21 (71%)		
Weight		2/21 (10%)		
Patient satisfaction with PR programme		18/21 (86%)		
Quality of data collection	Ethnicity	Open-ended question about ethnicity	5/21 (24%)	
		Gathered from existing clinical records	10/21 (48%)	
		Both open-ended questions and clinical records	2/21 (10%)	
		Structure questionnaire	1/21 (5%)	
	Ethnicity data not collected		3/21 (14%)	
	Basis for estimates of programme referrals	Actual patient records		7/21 (33%)
		Informal recall supplemented by some patient records		11/21 (52%)
		Unable to estimate referral numbers		3/21 (14%)
	Basis for estimates of programme enrolments	Actual patient records		13/21 (62%)
		Average PR group sizes and number of PR groups per year		4/21 (19%)
		Informal recall supplemented by some patient records		4/21 (19%)
	Basis for estimates of programme completions	Actual patient records		14/21 (67%)
		Average completion rates per PR group sizes and PR groups per year		3/21 (14%)
		Informal recall supplemented by some patient records		4/21 (19%)

Uptake of PR in 2009—Data on the number of patients entering and completing PR programmes in 2009 was provided by all 21 of the responding organisations. The majority based their figures entirely on actual patient records and approximately a third reported a ‘best estimate’ based either on the average number of enrolments and completions per PR group multiplied by the number of PR groups run in 2009, or based on their and their colleagues’ recall of the total number of participants seen in 2009 (see Table 1 for details).

Three of the 21 organisations (14%) felt unable to estimate the number of people who had been offered a place on a PR programme, with the other 18 organisations (86%) providing an estimate of the number of people offered PR based on actual patient records (33%; 7/21) or a ‘best guess’ based on their recall and any other information they had available (52%; 11/21).

Overall, the 21 organisations provided 105 ‘discrete’ group PR programmes in 2009 (a ‘discrete’ programme being one where all participants begin and end at the same time) with a number of additional patients receiving PR through programmes offering ‘rolling’ admissions or through individualised programmes. The survey responses indicated that in 2009 these 21 organisations offered PR to 2498 people with COPD, enrolled 1736 people with COPD into a PR programme, and assisted 1340 people to complete a PR programme.

In order to accommodate missing data from the two PHOs known to provide PR but who did not respond to the survey, we substituted figures derived from existing data. For these two PHOs we calculated the number of people entering PR based on the mean number of PR enrolments for programmes run by PHOs who did respond to the survey. Furthermore, we estimated the number of people with COPD offered PR and completing a PR programme based on the mean ratio of referrals to enrolments and enrolments to completions in all organisations that did respond to the survey. Likewise, we substituted data on PR referrals for the three respondents to the survey who had felt unable to estimate this data.

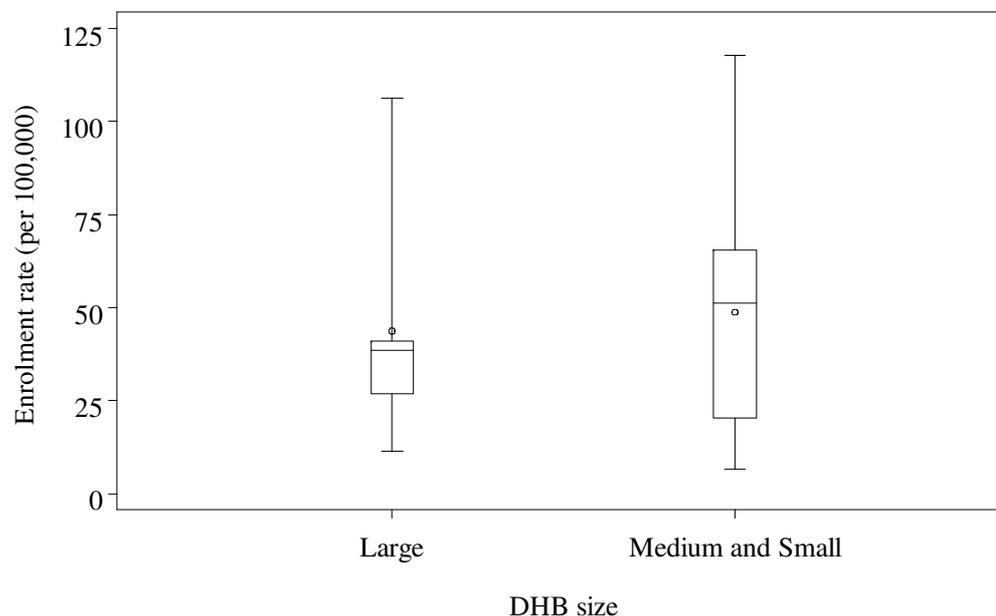
Thus in total we estimated that in 2009, 2569 people with COPD were offered PR, 1786 entered PR, and 1378 completed a PR programme. This means that based on the prevalence figures of Shirtcliffe and colleagues using GOLD criteria for diagnosis of COPD⁵ and NZ population statistics for 2009,⁶ it is likely that only 0.9% of people with COPD were offered PR, 0.7% entered PR, and 0.5% completed PR in NZ in 2009—although these estimated proportions are a little higher if different criteria are used to estimate the prevalence of COPD in NZ (see Table 2).

Sub-analysis on enrolment rates by DHB region (calculated on the basis of number of people with COPD enrolling in PR per 100,000 people in each DHB region)¹⁵ revealed no obvious differences in the number of people with COPD provided PR per capita between small (population <100,000) and medium (population 100,001—150,000) compared to large (population >150,000) DHBs (see Figure 1).

Table 2. Uptake of PR as a percentage of the estimated population of people over 40 with COPD in NZ

Variables	Figures based on data from the 2006/07 National Health Survey using self-reports of having a doctors' diagnosis of COPD ⁴	Figures based on data from Shirtcliffe et al.'s observational study using LLN-defined criteria for diagnosis of COPD ⁵	Figures based on data from Shirtcliffe et al.'s observational study using GOLD standard criteria for diagnosis of COPD ⁵
Percentage of population over 40 estimated to have COPD	6.6% (95% CI 5.9%–7.3%)	9.0% (95% CI 6.7%–11.3%)	14.2% (95% CI 11.0%–17.0%)
Total number of people over 40 estimated to have COPD in 2009 (based on New Zealand population statistics)	128,040 (95% CI 114,460–141,620)	174,600 (95% CI 129,980–219,220)	275,480 (95% CI 213,400–329,800)
Percentage of people over 40 with COPD who were offered PR in 2009	2.0% (1.8%–2.2%)	1.5% (1.2%–2.0%)	0.9% (0.8%–1.2%)
Percentage of people over 40 with COPD who entered a PR programme in 2009	1.4% (1.3%–1.6%)	1.0% (0.8%–1.4%)	0.7% (0.5%–0.8%)
Percentage of people over 40 with COPD who completed a PR in 2009	1.1% (1.1%–1.2%)	0.8% (0.6%–1.1%)	0.5% (0.4%–0.7%)

Figure 1. Box-plot of enrolment rates (number of people entering PR per year) by DHB size



Discussion

To our knowledge this is the first survey that attempts to quantify the numbers of people with COPD being referred to, entering and completing PR in NZ. Overall, this

survey has indicated that there is a marked shortfall between the national levels of provision of PR and the prevalence of COPD, with less than 1% of people who have COPD accessing it each year. This estimate of national provision of PR is lower than has been previously assumed,² despite significant levels of commitment by individual health professionals and pockets of excellence in the provision of PR at a local level around the country. While disappointing, these figures are in fact more in line with findings from similar surveys conducted in Canada¹¹ and the UK.¹²

The current level of provision of PR, in part representing a poor investment of health care resources in these programmes, is unfortunate; particularly given the sound evidence base for PR. High quality randomised controlled trials supporting the effectiveness of PR in improving exercise capacity and QOL have been published since the late 1990s. The first Cochrane review on the subject, supporting the effectiveness of PR, was published in 2003.⁷ Lack of evidence regarding the effectiveness of PR is no longer a credible reason for the low participation rates.

The low uptake in NZ is more likely to reflect a lack of direction and financial incentives provided at a public policy level. A recent audit of respiratory services in NZ concluded that there is a 'lack of national leadership and insufficient regional organisation leading to large gaps in service provision of even basic respiratory services' (page 1).¹³ The findings from this survey of PR in NZ are symptomatic of this wider problem.

Some may perhaps consider provision of PR less important than smoking cessation programmes, which have featured highly in both the media and public policy discussions—particularly following recent suggestions to make NZ 'Smokefree' by 2025.¹⁶ While smoking cessation is probably the single most important action a smoker with COPD could take to improve their health and wellbeing¹⁷, it is important to note that even if the ambitious goal of a 'Smokefree' NZ was achieved by 2025, there would still be a further 20–30 years where smoking-related respiratory illnesses continued to be a financial burden for the country because of the insidious nature of these conditions. Furthermore, smoking is not the only (or, some have argued, even the most significant) cause of COPD.¹⁸ Other factors such as childhood respiratory illnesses, socioeconomic status, and air pollution have been also identified as significant contributors to COPD.¹⁸ Smoking cessation, while highly important, will not eradicate the need for PR.

On the positive side, data from this survey have indicated that when PR is provided, it is done so in line with evidence-based guidelines, and as part of publicly funded health services in the large majority of DHB regions. This is a finding worth celebrating.

There are important limitations of this survey. Firstly, just over a third of the respondents were unable to base their reported enrolment and completion rates on actual patient records, relying instead on other sources of information, including informal recall. Secondly, almost half of all respondents did not confirm the diagnosis of people entering their PR programmes using standard diagnostic criteria for COPD and used instead the diagnosis of the referring physician or non-standard diagnostic criteria. This means that we cannot be entirely confident that all people counted in this survey as receiving PR did in fact have COPD.

Thirdly, our survey did not account for people who might have attended more than one PR programme. Fourthly, the survey did not account for people who might have been referred to PR by a general practitioner or hospital physician, but who did not enter a PR programme—potentially resulting in an underestimate of the percentage of people who decline an offer to enrol in a PR programme.

Furthermore, we are aware that there are a handful of community-based exercise groups, attended by people with COPD, which did not meet our definition of a PR programme. Potentially these programmes may also be effective in improving health outcomes for people with COPD, but were not included in our review. Nevertheless, even if our figures on the uptake of PR in NZ in 2009 were doubled the conclusions from this survey would remain the same.

Finally, we did not specifically gather data on the number of participants in each PR programme by ethnicity. Previous research has implied that Māori appear to be under-represented in PR classes nationally, with it being estimated that Māori comprise 11% of PR classes in DHBs¹⁴, despite Māori representing 15% of the NZ population and experience COPD at over twice the rate of non-Māori.³ However, the exact uptake of PR by Māori with COPD still needs to be established empirically.

Some might argue that another limitation of this study was that we did not distinguish between people based on severity of COPD, and that perhaps a restricted resource such as PR could be best utilised if targeted at the group of people most like to benefit. However, given that the current best evidence has indicated that people with moderate, severe and very severe COPD benefit equally from involvement in PR (and insufficient evidence exists to draw firm conclusions about the relative benefit to people with mild COPD)¹⁹ we suggest that interpretation of our survey data on the basis of severity of disease would be misleading.

There is a strong need for continued research into PR in the future. Such research should include investigations of the barriers and facilitators to access and completion of PR programmes. Arguably, given that COPD appears significantly underdiagnosed,⁵ the greatest barrier to improved uptake of PR is lack of identification of people who could benefit from it.

Interventions to increase screening for COPD, such as offering lung function testing in primary care settings as a routine part of the management of smokers and other people at risk of respiratory disease, would be worth investigating. However, data from our survey indicates that even when PR is offered, at least 30% of people with COPD do not subsequently enrol in a PR programme. In order to maximise the known benefits of this intervention, research needs to be conducted to better understand the reasons why people with COPD do not take up an opportunity to participate in PR when it is offered.

Any such future research ought to focus in particular on addressing the needs of Māori, who are inequitably burdened by COPD.³ Finally, future research should also investigate, in a NZ context, the cost-utility of PR in comparison to other medical management strategies in order to further strengthen arguments regarding the importance of this intervention in NZ.

Competing interests: None declared.

Author information: William M M Levack, Senior Lecturer in Rehabilitation, Rehabilitation Teaching and Research Unit, Department of Medicine, University of Otago, Wellington; Mark Weatherall, Professor of Medicine, Department of Medicine, University of Otago, Wellington; Julie C Reeve, Senior Lecturer in Physiotherapy, Faculty of Health and Environmental Science, AUT University, Auckland; Christina Mans, Senior Physiotherapist, Physiotherapy Department, Waikato Hospital, Hamilton; Antonia Mauro, Physiotherapist, Institute of Sport Science and Sport, University of Erlangen-Nuremberg, Nürnberg, Germany

Acknowledgements: We thank Betty Poot and Colleen Stevens from Hutt Valley District Health Board, who piloted our draft survey, as well as all the health professionals who took time to contribute data to this study.

Correspondence: Dr William Levack, Rehabilitation Teaching and Research Unit, Department of Medicine, University of Otago (Wellington), PO Box 7343, Wellington 6242, New Zealand. Email: william.levack@otago.ac.nz

References:

1. Town I, Taylor R, Garrett J, Patterson J, eds. The burden of COPD in New Zealand. Wellington: Asthma and Respiratory Foundation of New Zealand Inc.; 2003.
2. Broad J, Jackson R. Chronic Obstructive Pulmonary Disease and Lung Cancer in New Zealand: Report to The Thoracic Society of Australia and New Zealand; 2003.
3. TMG associates Ltd. Literature Review: Respiratory Health for Maori. Wellington: The Asthma and Respiratory Foundation of New Zealand (Inc.); 2009.
4. Ministry of Health. A Portrait of Health: Key Results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.
5. Shirtcliffe P, Weatherall M, Marsh S, et al. COPD prevalence in a random population survey: a matter of definition. *Eur Respir J*. 2007;30:232-9.
6. Statistics New Zealand. Population Estimates at 30 June 2006–09. 2010. http://www.stats.govt.nz/tools_and_services/tools/TableBuilder/intercensal-population-estimates-tables.aspx
7. Lacasse Y, Goldstein R, Lasserson TJ, Martin S. Pulmonary rehabilitation for chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*. 2006;4:CD003793.
8. Puhan M, Scharplatz M, Troosters T, et al. Pulmonary rehabilitation following exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev*. 2009;1:CD005305.
9. Meyer RJ. Respiratory service in New Zealand: a breath of fresh air is needed. *N Z Med J*. 2009;122:5-9.
10. Australian Lung Foundation. Pulmonary Rehabilitation. Australian Lung Foundation, 2009. <http://www.lungfoundation.com.au/content/view/109/14/>
11. Brooks D, Sottana R, Bell B, et al. Characterization of pulmonary rehabilitation programs in Canada in 2005. *Can Respir J*. 2007;14:87-92.
12. Yohannes A, Connolly M. Pulmonary rehabilitation programmes in the UK: a national representative survey. *Clin Rehabil*. 2004;18:444-9.
13. Garrett J, Chen B, Taylor DR. A survey of respiratory and sleep services in New Zealand undertaken by the Thoracic Society of Australia and New Zealand. *N Z Med J*. 2009;122(1289). <http://www.nzma.org.nz/journal/122-1289/3456>
14. Connolly M, Clinton J, Carswell P, et al. Alleviating the Burden of Chronic Conditions in New Zealand (The ABCC NZ Study) Generic Stocktake Analysis. (Commissioned Report): District Health Board New Zealand; 2009.

15. Ministry of Health. District Health Boards: frequently asked questions. 2010. (Accessed 9 February, 2011, at <http://www.moh.govt.nz/moh.nsf/indexmh/dhb-faq>)
16. Blakely T, Thomson G, Wilson N, et al. The Maori Affairs Select Committee Inquiry and the road to a smokefree Aotearoa. *N Z Med J.* 2010;123:7-17.
17. Godtfredsen NS, Lam TH, Hansel TT, et al. COPD-related morbidity and mortality after smoking cessation: status of the evidence. *Eur Respir J.* 2008;32:844-53.
18. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet.* 2009;374:733-43.
19. Marciniuk DD, Brooks D, Butcher S, et al. Optimizing pulmonary rehabilitation in chronic obstructive pulmonary disease - practical issues: a Canadian Thoracic Society Clinical Practice Guideline. *Can Respir J.* 2010;17:159-68.

Hazardous patterns of alcohol use are relatively common in smokers: ITC Project (New Zealand)

Nick Wilson, Deepa Weerasekera, Christopher W Kahler, Ron Borland, Richard Edwards

Abstract

Aims To describe patterns of alcohol use in a nationally-representative sample of New Zealand smokers.

Methods The New Zealand (NZ) arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) derives its sample from a national survey: the NZ Health Survey (NZHS). From this sample we surveyed adult smokers (n=1376).

Results A third (33.1%) of these smokers had a drinking pattern that was considered hazardous (i.e., AUDIT scores ≥ 8). These figures were much higher than for non-smokers in the NZHS (at 13.1%). In both the univariate and multivariate analyses, hazardous drinking patterns were significantly more common among: younger smokers, male smokers, and Māori smokers (e.g., adjusted odds ratio for the latter: 1.43, 95%CI: 1.05–1.95). The same pattern of more hazardous drinking was also seen (but in the univariate analysis only), for smokers with financial stress and for moderate individual-level deprivation.

Conclusions These findings provide additional evidence that hazardous drinking patterns are elevated in New Zealand smokers overall and particularly in some groups of smokers. Given the international evidence that hazardous drinking may impede quitting, policy makers could consider the potential benefits of improved alcohol control as part of the national strategy to curtail the tobacco epidemic and achieve the government's "Smokefree Nation 2025" goal. Such an approach could also reduce this country's high levels of alcohol-related harm and reduce gender and ethnic health inequalities.

Higher levels of alcohol use are associated with higher levels of smoking¹⁻⁵ and are also associated with lower rates of quitting.⁶⁻¹⁰ For example, one large prospective study, the ITC Four Country Survey, found that individuals who regularly drank heavily had significantly lower rates of quitting smoking than all other participants.¹¹

In New Zealand there is also some literature on the relationship between smoking and alcohol use. One national study reported that smoking was clustered with hazardous alcohol use (high AUDIT scores) at almost twice the expected level.¹² A pooled analysis of five New Zealand surveys found that smoking prevalence was higher with increasing average daily volume of alcohol (and with increasing volume per drinking session) for both Māori and non-Māori, but that the difference in smoking prevalence across levels of alcohol volume was greater among non-Māori.¹³ Also, a study of tertiary students, found that increased AUDIT scores were associated with smoking tobacco.¹⁴

Given this background and ongoing health sector and government interest in addressing the tobacco epidemic in New Zealand,¹⁵ we aimed to further describe alcohol use patterns among smokers in this country.

Methods

Survey—The ITC Project (New Zealand arm) surveyed a nationally representative sample of adult smokers. This study derives its sample from the New Zealand Health Survey (NZHS) which is a national sample with boosted sampling of Māori, Pacific and Asian New Zealanders. Out of the potential respondents in the NZHS, a total of 1376 completed a telephone questionnaire in Wave 1 in 2007/08, giving a response rate of 56.4%. But when considering the NZHS response rate and willingness to further participate, then the overall response rate was reduced further to 32.6%.

Measures—The questions around alcohol use were asked in the NZHS with the first question being if they had had an alcoholic drink in the previous 12 months. Respondents were then asked 10 questions about their alcohol use, covering the volume and frequency of alcohol consumed, alcohol-related problems and abnormal drinking behaviour. These 10 questions were developed by the World Health Organization and comprise the Alcohol Use Disorders Identification Test (AUDIT). As in the published NZHS analyses, we used the international definition of hazardous drinking as an AUDIT score of 8 or more.

For five respondents there were incomplete AUDIT scores (one out of 10 questions incomplete for each). So we imputed the missing values by first examining the responses for those respondents who had given the same answers to the other nine questions (of the AUDIT score) and who belong to the same age, sex and ethnicity groups. Then we randomly selected from those responses of the matched respondents to impute the value for the missing question.

Weighting and statistical analyses—Weighting of the results was desirable given the sampling design (e.g., boosted sampling of three ethnic groups in the NZHS), and non-response for the NZHS and ITC Project Wave 1 survey. Detailed descriptions of the weighting processes are detailed in an online report.¹⁶

Univariate analyses included various socioeconomic status measures covering small area deprivation (NZDep2006), individual deprivation (NZiDep) and financial stress.¹⁷ The logistic regression analysis used the “enter” method for all key variables and fits with a conceptual framework that assumed hierarchical relationships between demographic and sociodemographic factors.¹⁸ Model 1 included demographic variables alone, and Model 2 included additional sociodemographic variables (the two measures of deprivation and a measure of financial stress, all of which have conceptual differences¹⁷).

All analyses were conducted in Stata software (version 10, Stata-Corp, College Station, TX) and all of the presented results were weighted and adjusted for the complex sample design of the NZHS to make the sample representative of all New Zealand smokers. Further details of the methods (including response rates and weighting processes) are available in online *Methods Reports*^{16,17} and related publications.^{19,20}

Results

A third (33.1%) of participants had a drinking pattern that was considered hazardous at the time of the NZHS (i.e., AUDIT scores ≥ 8). Hazardous drinking was more prevalent among younger smokers, with the proportion drinking hazardously declining with increasing age (Table 1).

Male smokers were significantly more likely to have a hazardous drinking pattern, as were Māori and Pacific smokers (compared to European/Others). There was no pattern by small area deprivation but there was a non-linear association with individual deprivation. That is those with moderate individual deprivation (scores of 1 to 4) had a more hazardous pattern than either the least-deprived or most-deprived individuals.

Reporting one of two forms of financial stress was also associated with more hazardous drinking (Table 1).

Table 1. Demographic and sociodemographic characteristics of New Zealand smokers by pattern of hazardous alcohol use (AUDIT score \geq 8, with all the results weighted to adjust for the complex sample design and non-response)

Variables*	Non-hazardous alcohol use (AUDIT score < 8) (row %)	Hazardous alcohol use (AUDIT score \geq 8) (row %)	Crude odds ratios (OR) for hazardous alcohol use (95% CI)
Total (n=1376)	66.9	33.1	–
Age (years)			
18–24 (n=147)	41.0	59.0	8.42 (4.58–15.48)
25–34 (n=339)	64.3	35.7	3.25 (1.94–5.43)
35–44 (n=353)	68.2	31.8	2.73 (1.65–4.52)
45–54 (n=292)	74.3	25.7	2.02 (1.15–3.54)
55+ (n=245)	85.4	14.6	1.00 Referent
Gender			
Men (n=529)	59.3	40.7	2.04 (1.49–2.80)
Women (n=847)	74.8	25.2	1.00 Referent
Ethnicity			
European (includes Other) (n=620)	70.8	29.2	1.00 Referent
Māori (n=607)	57.9	42.1	1.76 (1.29–2.41)
Pacific (n=90)	47.9	52.1	2.63 (1.51–4.58)
Asian (n=59)	86.9	13.1	0.37 (0.14–0.98)
Deprivation level (small area) (quintiles)			
1&2 (least deprived) (n=121)	69.1	30.9	1.00 Referent
3&4 (n=205)	67.0	33.0	1.10 (0.57–2.12)
5&6 (n=238)	69.7	30.3	0.97 (0.50–1.89)
7&8 (n=308)	63.0	37.0	1.32 (0.71–2.44)
9&10 (most deprived) (n=504)	67.5	32.5	1.08 (0.60–1.95)
Individual deprivation NZiDep scores			
0 (least deprived) (n=627)	71.3	28.7	1.00 Referent
1 (n=252)	62.7	37.3	1.48 (0.98–2.24)
2 (n=173)	56.3	43.7	1.93 (1.17–3.19)
3–4 (n=197)	64.7	35.3	1.36 (0.82–2.25)
5–8 (most deprived) (n=126)	69.0	31.0	1.11 (0.59–2.09)
Financial stress			
Unable to pay any important bills on time ... “yes” (n=113), (referent=“no”)	52.8	47.2	1.90 (1.07–3.37)
Not spending on household essentials ... “yes” (n=374), (referent=“no”)	62.3	37.7	1.30 (0.91–1.85)

Note: *See an online Methods Report for more detailed descriptions of all these measures.¹⁷

The associations with high AUDIT scores that remained statistically significant in the logistic regression analysis were the findings for being a younger smoker, being male and being Māori (i.e., Model 2, Table 2). Also Asians had significantly less hazardous drinking compared to European/Others.

Table 2. Logistic regression analysis for hazardous alcohol use (AUDIT \geq 8) among New Zealand smokers

Variables	Adjusted Odds Ratio (aOR) (95% CI)*	
	Model 1 (demographics)	Model 2 (+sociodemographics)
	Hosmer-Lemeshow $\chi^2=7.03$, df=7 (p=0.425)**	Hosmer-Lemeshow $\chi^2=7.04$, df=8 (p=0.533)**
Demographic		
Age (35–49 vs 50+)	1.40 (0.97–2.01)	1.45 (1.01–2.09)
Age (<35 vs 50+)	4.00 (2.66–6.00)	4.31 (2.85–6.51)
Gender (men vs women)	2.21 (1.62–3.02)	2.18 (1.59–3.01)
Māori vs European/Other	1.29 (0.96–1.75)	1.43 (1.05–1.95)
Pacific vs European/Other	1.24 (0.73–2.10)	1.43 (0.83–2.46)
Asian vs European/Other	0.12 (0.05–0.28)	0.12 (0.05–0.29)
Sociodemographic		
Area deprivation deciles (increasing deprivation)	–	0.89 (0.79–1.00)
Individual deprivation using NZiDep ...(any deprivation vs nil)	–	0.82 (0.59–1.13)
Financial stress ...(unable to pay any important bills on time)	–	1.02 (0.60–1.73)

Notes:

* The adjusted odds ratios (aORs) represent the odds for having an AUDIT score of \geq 8 compared to $<$ 8.

** This p-value not adjusted for complex sample design.

Discussion

This study found that a third of smokers had a drinking pattern that was considered hazardous. When compared to the pattern for all non-smoking New Zealand adults in the NZHS, smokers were much more likely to be hazardous drinkers (i.e., 33.1% in smokers in our study versus 13.1% in NZHS non-smokers). Such hazardous drinking is of concern given the evidence suggesting it may impede successful smoking cessation.¹¹ But also alcohol and smoking synergistically increase the risk of some cancers e.g., those of the oral cavity, pharynx, larynx and oesophagus.²¹

The groups with the most hazardous drinking pattern were younger smokers, male smokers and Māori smokers (in both the univariate and multivariate analyses). Therefore such patterns may be contributing to both gender and ethnic health inequalities associated with smoking-related disease and in terms of cancer risk. Indeed, growing ethnic inequalities in cancer mortality (Māori versus non-Māori) have been described in New Zealand.²²

One finding of note was that those with *moderate* individual deprivation (scores of 1 to 4) had a more hazardous pattern than either the least-deprived or most-deprived individuals. The reason for this is unknown in the New Zealand setting, but it could relate to the most-deprived group being less able to afford alcohol or having existing health problems which limit scope for heavy drinking.

A strength of this study was its nationally representative sample. The risk of social desirability bias should have been reduced by the location of the alcohol questions in a large survey and with the use of show-cards for these questions during the face-to-face interview in the NZHS.

A potential weakness is that this study involved a sample which (due to non-participation in the NZHS and then in the ITC Project survey) could have become less representative of the national population of smokers. It is therefore possible that the weighting process (although sophisticated) may not have fully adjusted for non-response bias, potentially affecting the generalisability of the findings to all New Zealand smokers. Therefore such analyses as our one should be repeated in the future with data from the new-version of the NZHS where continuous data are collected (and especially where respondents complete the episodic but more in-depth “tobacco module” questions).

In 2010 the New Zealand Law Commission published a comprehensive review on reducing alcohol-related harm²³ and a Parliamentary Select Committee began examining options for reducing this harm in early 2011. Given the research findings presented here and the international literature, it would seem prudent for New Zealand policy makers to further consider the synergies between alcohol use and smoking. In particular they could:

- Consider the potential benefits for advancing tobacco control via strengthening cost-effective and evidence-based policies that reduce alcohol-related harm.^{24–26}
- Consider higher alcohol taxation levels, given some evidence that tobacco consumption has been found to decline with higher alcohol taxes.²⁷
- Consider raising the legal alcohol purchase age. There is evidence from the United States (US) that this reduces adolescent smoking prevalence.²⁸
- Consider exploring policies to further decouple smoking and drinking by making the outdoor seating areas around cafés and pubs, smokefree. For example, smokefree legislation covering bars and pubs in Scotland was associated with reduced drinking behaviour in these venues among moderate- and heavy-drinking smokers.²⁹

This pattern has also been found in the US.³⁰ Nevertheless, making *outdoor* areas smokefree could be controversial (given only minority smoker support¹⁹)—although it may be generally favoured by non-smoking patrons who often sit outside hospitality venues in New Zealand in summer and are exposed to secondhand smoke.

- Consider additional funding support for health services to allow them to address both heavy drinking and smoking cessation together. There is evidence for both feasibility and effectiveness of this approach.³¹

Nevertheless, as of January 2012 it appeared that the above interventions are not substantively part of the alcohol-reform legislation currently before the New Zealand Parliament. But since this draft legislation will probably be considered further in the 2012 parliamentary term, there is probably still scope for it to be strengthened. Financial pressures on government may also move the case for increased alcohol tax up the political agenda.

Overall, there appears to be a growing divergence between tobacco control and alcohol control in New Zealand with recent years involving a range of new tobacco control initiatives (e.g., multiple tobacco tax rises, a law banning point-of-sale

tobacco displays, extra support for quitting services, smokefree prisons, and government commitment to a “Smokefree Nation 2025” goal).

In summary, it would appear that hazardous drinking patterns are relatively common among New Zealand smokers. Yet if policy makers wish to address such problems then there is scope for using proven interventions relating to reducing heavy alcohol drinking.

Competing interests: Although we do not consider it a competing interest, for the sake of full transparency we note that some of the authors have undertaken work for health sector agencies working in tobacco control.

Author information: Nick Wilson¹; Deepa Weerasekera¹; Christopher W Kahler²; Ron Borland³; Richard Edwards¹

¹ Department of Public Health, University of Otago, Wellington, New Zealand

² Center for Alcohol and Addiction Studies, Brown University, Providence, Rhode Island, USA

³ VicHealth Centre for Tobacco Control, Melbourne, Australia

Acknowledgements: The ITC Project (NZ) team thank: the interviewees who kindly contributed their time; the Health Research Council of New Zealand which has provided the funding (grant 06/453); and our other project partners (see: <http://www.wnmeds.ac.nz/itcproject.html>). We thank Professor Tony Blakely and Dr Fiona Imlach Gunasekara for helpful comments on draft versions of this article.

Correspondence: Dr Nick Wilson, Department of Public Health, University of Otago – Wellington, Box 7343, Wellington South, New Zealand. Fax: +64 (0)4 3895319; email: nick.wilson@otago.ac.nz

References:

1. Chiolero A, Wietlisbach V, Ruffieux C, et al. Clustering of risk behaviors with cigarette consumption: A population-based survey. *Prev Med.* 2006;42:348-53.
2. Dawson DA. Drinking as a risk factor for sustained smoking. *Drug Alcohol Depend.* 2000;59:235-49.
3. Falk DE, Yi HY, Hiller-Sturmhofel S. An epidemiologic analysis of co-occurring alcohol and tobacco use and disorders: findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Res Health.* 2006;29:162-71.
4. Anthony JC, Echeagaray-Wagner F. Epidemiologic analysis of alcohol and tobacco use. *Alcohol Res Health.* 2000;24:201-8.
5. Kahler CW, Strong DR, Papandonatos GD, et al. Cigarette smoking and the lifetime alcohol involvement continuum. *Drug Alcohol Depend.* 2008;93:111-120.
6. Augustson EM, Wanke KL, Rogers S, et al. Predictors of sustained smoking cessation: A prospective analysis of chronic smokers from the Alpha-Tocopherol Beta-Carotene Cancer Prevention Study. *Am J Public Health.* 2008;98:549-55.
7. Hymowitz N, Cummings KM, Hyland A, et al. Predictors of smoking cessation in a cohort of adult smokers followed for five years. *Tob Control.* 1997;6:S57-62.
8. Osler M, Prescott E, Godtfredsen N, et al. Gender and determinants of smoking cessation: A longitudinal study. *Prev Med.* 1999;29:57-62.
9. Sorlie PD, Kannel WB. A description of cigarette smoking cessation and resumption in the Framingham Study. *Prev Med.* 1990;19:335-45.

10. Dollar KM, Homish GG, Kozlowski LT, et al. Spousal and alcohol-related predictors of smoking cessation: a longitudinal study in a community sample of married couples. *Am J Public Health*. 2009;99:231-3.
11. Kahler CW, Borland R, Hyland A, et al. Alcohol consumption and quitting smoking in the International Tobacco Control (ITC) Four Country Survey. *Drug Alcohol Depend*. 2009;100:214-20.
12. Tobias M, Jackson G, Yeh LC, et al. Do healthy and unhealthy behaviours cluster in New Zealand? *Aust N Z J Public Health*. 2007;31:155-63.
13. Bramley D, Broad J, Jackson R, et al. Cardiovascular risk factors and their associations with alcohol consumption: are there differences between Maori and non-Maori in Aotearoa (New Zealand)? *N Z Med J*. 2006;119:U1929.
14. Kypri K, Langley JD, McGee R, et al. High prevalence, persistent hazardous drinking among New Zealand tertiary students. *Alcohol and Alcoholism*. 2002;37:457-64.
15. Blakely T, Thomson G, Wilson N, et al. The Māori Affairs Select Committee Inquiry and the road to a smokefree Aotearoa. *N Z Med J*. 2010;123(1326):7-18.
16. Clark R. Summary of method for calculating estimation weights for Wave 1 of the 2007 International Tobacco Control Policy Evaluation Project (ITC) – New Zealand Arm Wollongong: University of Wollongong Centre for Statistical and Survey Methodology, 2008. <http://www.wnmeds.ac.nz/academic/dph/research/HIRP/Tobacco/itcproject.html>
17. Wilson N. Methods report for the New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) (Updated 2009). Wellington: University of Otago, Wellington, 2009. <http://www.wnmeds.ac.nz/itcproject.html>
18. Victora CG, Huttly SR, Fuchs SC, et al. The role of conceptual frameworks in epidemiological analysis: a hierarchical approach. *Int J Epidemiol*. 1997;26:224-7.
19. Wilson N, Blakely T, Edwards R, et al. Support by New Zealand smokers for new types of smokefree areas: national survey data. *N Z Med J*. 2009;122(1303):80-9.
20. Wilson N, Weerasekera D, Edwards R, et al. Characteristics of smoker support for increasing a dedicated tobacco tax: National survey data from New Zealand. *Nicotine Tob Res*. 2010;12:168-73.
21. IARC. Tobacco smoke and involuntary smoking (volume 83). Lyon, France: International Agency for Research on Cancer (IARC), 2004. <http://monographs.iarc.fr/ENG/Monographs/vol83/index.php>
22. Blakely T, Tobias M, Atkinson J, et al. Tracking Disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health, 2007.
23. New Zealand Law Commission. Alcohol In Our Lives: Curbing the Harm (NZLC R114). Wellington New Zealand Law Commission, 2010. <http://www.lawcom.govt.nz/ProjectReport.aspx?ProjectID=154>
24. Cobiac L, Vos T, Doran C, et al. Cost-effectiveness of interventions to prevent alcohol-related disease and injury in Australia. *Addiction*. 2009;104:1646-1655.
25. Chisholm D, Rehm J, Van Ommeren M, et al. Reducing the global burden of hazardous alcohol use: a comparative cost-effectiveness analysis. *J Stud Alcohol*. 2004;65:782-93.
26. WHO Regional Office for Europe. Evidence for the effectiveness and cost-effectiveness of interventions to reduce alcohol-related harm. Copenhagen: World Health Organization Regional Office for Europe, 2009. <http://www.euro.who.int/document/E92823.pdf>
27. Jimenez S, Labeaga JM. Is it possible to reduce tobacco consumption via alcohol taxation? *Health Econ*. 1994;3:231-41.
28. Dee TS. The complementarity of teen smoking and drinking. *J Health Econ*. 1999;18:769-93.
29. McKee SA, Higbee C, O'Malley S, et al. Longitudinal evaluation of smoke-free Scotland on pub and home drinking behavior: findings from the International Tobacco Control Policy Evaluation Project. *Nicotine Tob Res*. 2009;11:619-26.
30. Picone GA, Sloan F, Trogdon JG. The effect of the tobacco settlement and smoking bans on alcohol consumption. *Health Econ*. 2004;13:1063-80.

31. Kahler CW, Metrik J, LaChance HR, et al. Addressing heavy drinking in smoking cessation treatment: a randomized clinical trial. *J Consult Clin Psychol.* 2008;76:852-62.

Diagnosis of disorders of intermediary metabolism in New Zealand before and after expanded newborn screening: 2004–2009

Callum Wilson, Nicola J Kerruish, Bridget Wilcken, Esko Wiltshire, Kathy Bendikson, Dianne Webster

Abstract

Aim The purpose of this study was to compare the rate of diagnosis of inborn errors of intermediary metabolism (IEMs) in New Zealand in the 3 years before and after the commencement of expanded newborn screening (ENBS) in December 2006

Method The cases diagnosed during the period January 2004 to December 2006 were compared to a subsequent cohort, December 2006–December 2009, when ENBS was available in NZ

Results The total number of patients diagnosed in the 3 years prior to the introduction of EBNS was 15. In the following 3 years 42 cases were diagnosed. Thirty cases were diagnosed by ENBS. Two were diagnosed after investigation of older siblings in the families of the EBNS cases. Seven cases presented clinically with IEMs either because they had conditions that are not detectable with EBNS or they presented as older children born prior to December 2006. Three cases of carnitine-acylcarnitine translocase deficiency (CACT) presented on day 1 with symptoms and were diagnosed prior to the day 2 sample for EBNS being obtained.

Conclusion ENBS has resulted in an increase in the number of patients diagnosed with IEMs in New Zealand

Inborn errors of intermediary metabolism (IEMs) are genetic defects resulting in enzyme deficiencies of biochemical pathways and in particular those of amino acid, organic acid and fatty acid metabolism. The corresponding medical conditions are known as the aminoacidopathies, organic acidemias and the fatty acid oxidation disorders (FAODs) respectively. The clinical features of these diseases include symptoms such as encephalopathy that results from the accumulation of a toxic substrate, such as leucine in the aminoacidopathy maple syrup urine disease, or from the deficiency of energy providing products such as ketones in the FAODs.

The IEMs are individually rare, clinically heterogeneous, conditions that primarily affect young children. Without treatment the outcome is often poor. However with early diagnosis and treatment the prognosis for most conditions is favourable. A previous report documented that these IEMs were under-diagnosed in New Zealand and concluded that this was almost certainly due to the absence of newborn screening for these conditions.¹

With the advent of expanded newborn screening (ENBS), a procedure by which amino acids and acylcarnitines are quantified in the newborn Guthrie card blood spot using tandem mass spectrometry, it was hoped that the under-diagnosis of these

conditions would be rectified as over 20 different metabolic diseases can be identified with this technique (Table 1).

Table 1. Inborn errors of metabolism that can be diagnosed by expanded newborn screening

<p>Fatty acid oxidation disorders</p> <ul style="list-style-type: none">• Carnitine uptake defect• Carnitine palmitoyltransferase 1 deficiency (CPT1)• Carnitine palmitoyltransferase 2 deficiency (CPT2)• Carnitine-acylcarnitine translocase deficiency (CACT)• Medium chain acyl-CoA dehydrogenase deficiency (MCAD)• Long-chain L-3-OH acyl-CoA dehydrogenase deficiency (LCHAD)• Very long-chain acyl-CoA dehydrogenase deficiency (VLCAD)• Trifunctional protein deficiency• Multiple acyl-CoA dehydrogenase deficiency (GA II) <p>Aminoacidopathies</p> <ul style="list-style-type: none">• Phenylketonuria (PKU)• Homocystinuria (Hcy)• Maple syrup urine disease (MSUD)• Argininase deficiency• Argininosuccinic acidemia• Citrullinemia type 1 (CIT 1)• Citrullinemia type 2 (CIT II)• Tyrosinemia type II (TYR) <p>Organic acidopathies</p> <ul style="list-style-type: none">• Glutaric acidemia type I (GA1)• Beta ketothiolase deficiency• Isovaleric acidemia• Methylmalonic acidemia (Cobalamin disorders–CblC)• Methylmalonic acidemia (mutase deficiency) (MMA)• Holocarboxylase synthetase deficiency (HCS)• Propionic acidemia• HMG-CoA lyase deficiency• 2 Methyl 3 hydroxybutyric acidemia• 3 Methyl glutaconic acidemia• 3 Methylcrotonyl carboxylase deficiency (3-MCC) <p>Other</p> <ul style="list-style-type: none">• Vitamin B12 deficiency
--

The purpose of this study was to compare the numbers of patients with disorders of intermediary metabolism diagnosed in New Zealand in the 3 years before the commencement of ENBS in December 2006, with the numbers diagnosed in the first 3 years of ENBS.

Method

From January 2004 to December 2009 cases diagnosed by the Newborn Metabolic Screening Unit at LabPlus in combination with the Starship Children's Hospital clinical metabolic team in Auckland were notified to the New Zealand Paediatric Surveillance Unit (NZPSU). In addition, paediatricians in New Zealand were sent monthly questionnaires, via email or regular post, from the NZPSU, asking

whether they had diagnosed an inborn error of metabolism over the previous month. If they had they were sent a further questionnaire regarding the exact diagnosis along with aspects of the clinical presentation and immediate outcome. The Auckland, Wellington, and Christchurch laboratories that either perform the relevant metabolic investigations or facilitate samples being sent to the appropriate tertiary laboratories in Australia were also contacted and asked to report cases.

Cases were identified to the authors by initials, diagnosis and date of birth and notifications were screened to remove multiple notifications of single cases.

The cases diagnosed during the period December 2006–December 2009 when expanded newborn screening was available in NZ were compared to those diagnosed in the previous 3 years from January 2004 to December 2006. During this time ENBS was not available and thus these patients were diagnosed clinically or following a previous sibling being diagnosed.

Patients with phenylketonuria were not included in this study as this relatively common condition has been screened for in New Zealand for a number of decades and while the screening methodology has changed recently to mass spectrometry it is most unlikely that this has led to any change in the incidence.

This study was approved by the Lower South Regional Ethics Committee.

Results

The number of patients with IEMs diagnosed in New Zealand during the study period 2004–2009 can be seen in Tables 2 and 3.

Table 2. Disorders of intermediary metabolism diagnosed in New Zealand: 2004–2006

Disease	Method of initial diagnosis	Number	Outcome
MCAD	Clinical	2	Good
MSUD	NBS	1	Good
GAI	Clinical	1	Poor
Ketothiolase	Clinical	1	Good
HCS	Clinical	1	Died
MADD	Clinical	1	Poor
VLCAD	Clinical	1	Good
NKH	Clinical	4	Died
OTC	Clinical	3	Good

See Table 1 for full names of detected conditions.

From Jan 2004–Dec 2006 inclusive there were approximately 175,000 births in New Zealand.² During this period the majority of patients were diagnosed clinically apart from the one maple syrup urine disease patient who was diagnosed by a specific newborn screening test in use at the time, identifying elevated leucines by enzymatic assay. The total number of patients diagnosed was 15, of whom four had non-ketotic hyperglycaemia (NKH) while three had ornithine transcarbamylase (OTC) deficiency. Only small numbers of each other individual metabolic condition were diagnosed with the overall incidence being 1 in 11,666 (Table 2).

Table 3. Disorders of intermediary metabolism diagnosed in New Zealand: December 2006–2009

Disease	Method of initial diagnosis	Number	Outcome (Oct 2010)
MCAD	13 ENBS, 1 sibling	14	Good
CACT	Clinical-pre ENBS	3	2 Good, 1 died
Citrullinemia	ENBS	5	Good-benign
IVA	ENBS	3	Good-benign
VLCAD	ENBS	1	Good
MADD	ENBS	2	1 Good, 1 died
Carnitine uptake	NBS-maternal case	1	Maternal-good
3-MC	ENBS	3	Good-Benign
GA-1	2 ENBS, 1 sibling	3	Good
Homocystinuria	Clinical-born prior to 2006	2	Good
NKH	Clinical	2	Died
L-2(OH)glutaric	Clinical-born prior to 2006	1	Good
OTC	Clinical	2	Died

See Table 1 for full names of detected conditions.

From Dec 2006–Dec 2009 inclusive there were approximately 185 000 births in New Zealand.² During this time 30 cases of IEMs were diagnosed via EBNS. Two additional cases were diagnosed after investigation of older siblings in the families of these EBNS cases. Seven cases presented clinically with IEMs either because they had conditions that are not detectable with EBNS (NKH and OTC) or they presented as older children born prior to December 2006 (homocystinuria and L-2(OH) glutaric aciduria). Three cases of carnitine-acylcarnitine translocase deficiency (CACT) presented on day 1 with symptoms and were diagnosed prior to the day 2 sample for EBNS being obtained (Table 3). Medium chain acyl-CoA dehydrogenase deficiency (MCAD), with 14 cases, was by far the biggest contributor to the overall incidence of 1 in 4302 during this period

Excluding NKH and OTC, disorders not able to be detected by current ENBS, some 22 extra cases of IEM were diagnosed by ENBS during the period.

Discussion

There has been a dramatic increase in the number of cases of IEMs that have been diagnosed in New Zealand since the advent of ENBS. The overall rate of diagnosis has risen from around 1 in 12000 to either 1 in 4400 if all conditions are included or 1 in 6000 if those conditions that are general thought to be benign are excluded (3-MCC and benign forms of citrullinaemia and IVA). This increase has been mainly due to the ability to screen for MCAD, a disease that is generally considered to fulfil most screening criteria.^{3–5} MCAD is a disorder resulting in a relative inability to convert medium chain fat into energy. This process is needed during times of catabolic stress.

The condition is especially important in young children as they have reduced glycogen stores and are more prone to significant intercurrent illness. It is during these times that the children become catabolic and as they cannot produce ketones adequately they become hypoglycaemic and encephalopathic. They often die if left untreated for a few hours in this state. This disease is easily and successfully treated with patient/parent education stressing the need to feed the child a high calorie diet

during times of unwellness and if the child is not taking this feed or there are any other concerns then to have a low threshold for admission to hospital for intravenous feeding until they are well enough to feed normally.

Without screening roughly a third of MCAD children present clinically with life-threatening hypoglycaemia and encephalopathy and are eventually diagnosed while a third either never get diagnosed correctly despite presenting with classical clinical features or die from their disease prior to a diagnosis. The remaining third never have symptoms and thus don't get diagnosed.⁶⁻⁹ It is thus clear that lives will have been saved in the 3 years since screening for MCAD, as part of ENBS, commenced.

Three cases of glutaric academia type I (GA-1), all with as yet good outcome, were diagnosed in the later cohort compared with one case, with a poor outcome, in the early cohort. GA 1 is another disease that also often presents secondary to an intercurrent illness. Unlike MCAD which tends to result in either death or a relatively normal outcome GA-1 often results in severe neurodisability.^{10,11} While not all patients with GA 1 suffer disease, most do, and it seems likely that at least two of the children diagnosed through screening would have had very significant if not fatal disease without ENBS.^{11,12}

No cases of clinically significant metabolic diseases from the other organic acidemias (for instance methylmalonic and propionic acidemia) were diagnosed by screening during the 2006–2009 period. This is likely to reflect chance as we have no evidence that such a diagnosis was missed.

The optimal time, regarding sensitivity and specificity, for EBNS is at 48 hours of age with a subsequent small delay of a few days to allow for transport and laboratory processing of the Guthrie card. Thus patients can become unwell prior to the results being available. During the study period three neonates presented with profound hypoglycaemia and cardiac dysfunction on day 1; symptoms classical for the long chain fatty acid oxidation disorders. All three, despite being unrelated and from different ethnic groups were later shown to have the extremely rare condition CACT, a disorder in the transport of fat into the mitochondria.

While it was extremely useful to be able to establish the diagnosis rapidly with the local availability of tandem mass spectrometry in two of the cases the diagnosis was already strongly suspected clinically and successful treatment commenced while in the third clinical management was, for a variety of reasons, less than optimal and the child died. However, it is likely that the availability of expanded newborn screening increases the awareness of the possibility of an IEM, and improves the likelihood of a final diagnosis being reached in such cases.

One of the MCAD cases was also significantly symptomatic with hypoglycaemia and liver disease secondary to metabolic decompensation prior to the results of EBNS becoming available. This illustrates the importance of clinicians who care for neonates to continue to request urgent metabolic investigations if they have clinical suspicion of an IEM rather than waiting for the results of screening.

The very long-chain acyl-CoA dehydrogenase deficiency (VLCAD) cases represent an area of difficulty with ENBS. The phenotype is dependent on both the genotype and the environmental or more correctly the physiological state. VLCAD is another disorder resulting in a relative inability to convert fat into energy. Patients with severe

VLCAD defects can present, like CACT, prior to a screening result becoming available, during the normal early neonatal catabolic phase. However the majority of patients diagnosed with VLCAD, especially during the era of ENBS, have a mild form of the disease whereby they become symptomatic not with childhood illnesses and moderate periods of starvation like MCAD patients but with prolonged exercise, during which time, if not accompanied by a good oral intake of calories, they can experience rhabdomyolysis and cardiac dysfunction.¹³ Thus some patients diagnosed with a disease in the first week of life via ENBS may not be at any risk of symptoms until much later in life, if at all.

Our two cases, one from each cohort, illustrate this. The patient from 2004–2006 was diagnosed as an adult after recurrent episodes of rhabdomyolysis during periods of moderately severe exercise (2 hours plus mountain biking) accompanied by relatively poor caloric intake while the case from 2006–2009 has never been symptomatic despite a few typical childhood illnesses, albeit with the precaution of the parents knowing to maintain a good oral intake during these times, after being diagnosed by ENBS.

Because of this problem of potentially diagnosing patients who will only become symptomatic if exposed to quite significant physiological stress it has become important to clarify where the screening laboratory/metabolic service ‘draws the line in the sand’ as to what one ‘calls’ a disease and thus notifies the family about. As yet, there is no clear international agreement on whether biochemical, enzymological or molecular findings for VLCAD are the best discriminators for defining likely future disease.^{14,15} A close working relationship between the screening laboratory and the clinical metabolic team is thus essential with the latter having enough resources to provide a rapid and thorough service to the whole of the country.

There are a number of IEMs that are probably not clinically significant and yet are diagnosable by ENBS. There remains considerable debate about some of these conditions. Centres in Australasia, for example, consider short chain acyl Co-A dehydrogenase deficiency (SCAD) to be a benign condition and thus should not be screened for whereas many centres in the United States and continental Europe feel that this is indeed a condition that warrants screening.^{16–18}

There are some reports of the negative consequences of informing a family of a ‘disease’ that is not really a disease at all.^{19,20} In New Zealand we have a particularly high incidence of a benign form of citrullinaemia due to a high rate of this in the Niuean population and a secondary molecular test has been developed to identify these patients rapidly and the decision what to inform the family is based on this.²¹

Likewise there are diseases such as multiple acyl CoA dehydrogenase deficiency (MADD) which in some instances are not always responsive to optimal treatment and if one considers the ability to successfully treat to be one of the main tenets of screening then the merits of EBNS for this disorder could be debated. However many of these conditions are themselves clinically heterogeneous and there is little doubt that some forms are very responsive to treatment. In addition, the ability to successfully treat a condition should not be seen as the sole reason for screening as there are other advantages of early diagnosis such as the avoidance of a ‘diagnostic odyssey’ and potential for future pregnancy risk to be discussed through genetic counselling.

The case of carnitine uptake disorder illustrates the interesting phenomenon whereby biochemical abnormalities in the screened child may reflect primary disease in the mother. With this case it was the mother who had a defect in the transporter for carnitine, a substance required for the metabolism of fatty acids and whose deficiency can lead to hypoglycaemia and encephalopathy. Her subsequent low carnitine levels resulted in the fetus also having very low levels and thus being at risk of disease. A simple short course of carnitine supplements cured the baby while the mother requires life-long carnitine supplements. Vitamin B12 deficiency in women, usually due to dietary reasons, can also be diagnosed in a similar manner based on the elevated levels of Vitamin B12 dependent substrates in the blood of the screened newborn.

During the short study period we had no evidence that conditions that are diagnosable by ENBS were missed with screening and presented clinically at a later date

Non ketotic hyperglycaemia (NKH) and ornithine transcarbamylase deficiency (OTC) are two relatively common disorders of intermediary metabolism that are not easily detected by ENBS due to a lack of specificity of key diagnostic metabolites. They are diagnosed on an episodic clinical basis and there has been understandably no change in the incidence between the two periods.

Although they have arguably characteristic phenotypes of severe neonatal seizures and unexplained encephalopathy respectively it is likely that they remain under-diagnosed, based on the dramatic increase in prevalence in the equally clinically characteristic ENBS condition of MCAD with the commencement of screening. Additionally, 'mild' cases of these disorders, who would benefit greatly from appropriate management, certainly cannot be detected at present. This is especially relevant in New Zealand as both NKH and OTC have a high incidence in the Māori and Pacific peoples respectively.²²

There are many other metabolic diseases that are not currently screened for currently by ENBS, including the glycogen storage diseases, mitochondrial disorders, the peroxisomal diseases, and the various disorders of purines, pyrimidines, lipids, metals, protein glycosylation, creatine, cholesterol, and neurotransmitters. These conditions are also likely to be under-diagnosed.

In summary this study has shown the ENBS has resulted in an increase in the number of patients diagnosed with IEMs. While it is too early to be definitive regarding how beneficial this has been it is likely that this has resulted in a number of lives per year being saved. This study supports the findings from a number of other centres that ENBS is an important recent addition to newborn screening and to the diagnosis of metabolic diseases.

Competing interests: None declared.

Author information: Callum Wilson, Metabolic Paediatrician, Newborn Metabolic Screening Unit, LabPlus, Auckland City Hospital, Auckland; Nikki Kerruish, Paediatric Research Fellow, Department of Paediatrics and Child Health, Dunedin School of Medicine, University of Otago, Dunedin; Bridget Wilcken, Metabolic Physician, Director, NSW Biochemical Genetics and Newborn Screening Services, The Children's Hospital at Westmead, Sydney, Australia; Esko Wiltshire, Senior Lecturer, Department of Paediatrics and Child Health, University of Otago Wellington; Kathy Bendikson, Newborn Metabolic Screening Programme, Ministry

of Health, Penrose, Auckland; Dianne Webster, Director, Newborn Metabolic Screening Programme, LabPlus, Auckland City Hospital, Auckland

Correspondence: Dr Callum Wilson, Metabolic Paediatrician, Newborn Metabolic Screening Unit, PO Box 872, Auckland, New Zealand. Fax: +64 (0)9 3074978; email callumw@adhb.govt.nz

References:

1. Wilson C, Kerruish NJ, Wilcken B, et al. The failure to diagnose inborn errors of metabolism in New Zealand: the case for expanded newborn screening. *N Z Med J.* 2007;120(1262). <http://journal.nzma.org.nz/journal/120-1262/2727/content.pdf>
2. Births, Deaths and Marriages - Whanautanga, Matenga, Marenatanga. http://www.dia.govt.nz/diawebsite.NSF/wpg_URL/Services-Births-Deaths-and-Marriages-Index?OpenDocument
3. Schulze A, Lindner M, Kohlmuller D, et al. Expanded newborn screening for inborn errors of metabolism by electrospray ionization-tandem mass spectrometry: results, outcome, and implications. *Pediatrics.* 2003;111:1399-406.
4. Wilcken B, Wiley V, Hammond J, Carpenter K. Screening newborns for inborn errors of metabolism by tandem mass spectrometry. *N Engl J Med.* 2003;348:2304-12
5. Wilcken B, Haas M, Joy P, et al. Outcome of neonatal screening for medium-chain acyl-CoA dehydrogenase deficiency in Australia: a cohort study. *Lancet.* 2007;369:37-42.
6. Iafolla AK, Thompson RJ Jr, Roe CR. Medium-chain acyl-coenzyme A dehydrogenase deficiency: clinical course in 120 affected children. *J Pediatr.* 1994;124:409-15.
7. Wilson CJ, Champion MP, Collins JE, et al. Outcome of medium chain acyl-CoA dehydrogenase deficiency after diagnosis. *Arch Dis Child.* 1999;80:459-62
8. Derks TG, Reijngoud DJ, Waterham HR, et al. The natural history of medium-chain acyl CoA dehydrogenase deficiency in the Netherlands: clinical presentation and outcome. *J Pediatr.* 2006;148:665-670.
9. Bjugstad KB, Goodman SI, Freed CR. Age at symptom onset predicts severity of motor impairment and clinical outcome of glutaric acidemia type 1. *Pediatrics.* 2000 Nov;137(5):681-6.
10. Wilcken B, Haas M, Joy P, Wiley V, et al. Expanded newborn screening: outcome in screened and unscreened patients at age 6 years. *Pediatrics.* 2009;124:241-8.
11. Heringer J, Boy SPN, Ensenauer R, et al. Use of guidelines improves the neurological outcome in glutaric aciduria type I. *Ann Neurol.* 2010;68:748-52
12. Bijarnia S, Wiley V, Carpenter K, et al. Glutaric aciduria type I: outcome following detection by newborn screening. *J Inherit Metab Dis.* 2008;31:503-7
13. Spiekerkoetter U Mitochondrial fatty acid oxidation disorders: clinical presentation of long-chain fatty acid oxidation defects before and after newborn screening. *J Inherit Metab Dis.* 2010;33:527-32
14. Spiekerkoetter U, Haussmann U, Mueller M, et al. Tandem mass spectrometry screening for very long-chain acyl-CoA dehydrogenase deficiency: the value of second-tier enzyme testing. *J Pediatr.* 2010;157:668-73
15. Olsen RK, Dobrowolski SF, Kjeldsen M et al. High-resolution melting analysis, a simple and effective method for reliable mutation scanning and frequency studies in the ACADVL gene. *J Inherit Metab Dis.* 2010;33:247-60.
16. Lindner M, Hoffmann GF, Matern D. Newborn screening for disorders of fatty-acid oxidation: experience and recommendations from an expert meeting. *J Inherit Metab Dis.* 2010;33:521-6
17. Arnold GL, Koeberl DD, Matern D, et al. Delphi-based consensus clinical practice protocol for the diagnosis and management of 3-methylcrotonyl CoA carboxylase deficiency. *Mol Genet Metab.* 2008;93:363-70.

18. Wilcken B The consequences of extended newborn screening programmes: Do we know who needs treatment? *J Inherit Metab Dis.* 2008;33:501-6
19. Gurian EA, Kinnamon DD, Henry JJ, Waisbren SE. Expanded newborn screening for biochemical disorders: the effect of a false-positive result. *Pediatrics.* 2006;117:1915-21.
20. Hewlett J, Waisbren SE. A review of the psychosocial effects of false-positive results on parents and current communication practices in newborn screening. *J Inherit Metab Dis.* 2006;29:677-82
21. Marquis-Nicholson R, Glamuzina E, et al. Citrullinemia type I: molecular screening of the ASS1 gene by exonic sequencing and targeted mutation analysis. *Genet Mol Res.* 2010;9:1483-9.
22. Wilson C, Unpublished data, 2011.

New Zealanders' knowledge of palliative care and hospice services

Rod D MacLeod, Rachel Thompson, John W Fisher, Kris Mayo, Nathan Newman, Donna M Wilson

Abstract

Aim This project investigated New Zealanders' views about palliative care and local hospice services.

Method A representative population-based sample of 1011 New Zealanders completed an online survey.

Results The age, gender, and geographic region of the 1011 participants were broadly representative of the New Zealand population. Varying awareness of hospice services and palliative care were displayed among respondents, with age and gender influencing awareness.

Conclusions There was a reasonably good understanding of the concept of palliative care. However, participants could not always identify local hospices, with younger people and males more unaware of accessible hospice services. Low levels of understanding point to the need for continued public education so that the holistic nature of palliative care is understood and accessible hospice services are sought when required.

There were 28,964 deaths registered in New Zealand in 2009, with 14,480 male and 14,484 female deaths according to Statistics New Zealand.¹

The number of deaths will rise rapidly in the years ahead with population aging and population growth, so it can be anticipated that many more people will personally need to use hospice/palliative care services to assist them when dying and many more people will have family or friends needing hospice/palliative care services.

Hospice New Zealand is a national organisation that exists to support member hospices in their work of caring for people who are dying and their families. Their primary goal is to give voice to the interests, views and concerns of member hospices. Their work is in keeping with the 2001 New Zealand Palliative Care Strategy.² The aim of this Strategy was to further a systematic and informed approach to the provision and funding of palliative care services through the implementation of the following vision:

All people who are dying and their family/whānau who could benefit from palliative care have timely access to quality palliative care services that are culturally appropriate and are provided in a co-ordinated way.

Strategy 6 of that document relates to informing the public about palliative care services. In particular, it states “Public information specific to each District Health Board area is necessary to:

- Outline the public’s rights/entitlement to palliative care services.
- Describe the services offered by palliative care providers.
- Provide information on what the public should expect from a palliative care service.”

Nearly one decade later, it is of concern that New Zealanders’ knowledge of palliative care and hospice services has not yet been researched. In New Zealand, hospices are independent charitable organisations providing care and support free of charge for all who are dying. This care extends beyond the physical – as it includes social, emotional and spiritual aspects of each unique person’s life. For the purposes of this survey the terms hospice and palliative care have been used interchangeably.

The New Zealand definition of palliative care identifies a similar aim and includes (as hospice does) the support of the individual’s family, whānau and other caregivers into bereavement.³ Awareness of hospice services and the nature of palliative care may still be low, even among health professions.

For many, hospice is still perceived as a ‘place to die’ rather than as a philosophy of care and so people may not seek support from hospices until near the very end of their life. We also do not know much about public attitudes to death and dying, and the anxiety associated with them.

Tomas-Sabado and Limonero claim that ‘our attitudes to death and dying are shaped by many things such as cultural perspectives of illness and the religious and spiritual beliefs of our family and community.’⁴ We do know, from the recent Australian National Community Education Initiative, that ‘talking about dying and death is not something that comes naturally to Australians.’⁵

Palliative Care Australia, the national peak body for palliative care has, over the last 13 years, held a National Palliative Care Awareness Week in an attempt to address a perceived lack of understanding and preparedness for death in Australia. In the UK, the Dying Matters coalition (www.dyingmatters.org) encourages people to talk death in general about their wishes towards the end of life in particular. No similar initiatives are yet available in New Zealand.

Method

Under the auspices of Hospice New Zealand, the researchers approached The Nielsen Company, an international market research company, to conduct a survey. The questions asked were identical to those asked in similar studies conducted in a number of other countries (with minor modification for our on-line methodology), as there is a plan to compare research findings. Ethics approval was obtained from the Northern X Regional Ethics Committee of the New Zealand Health and Disabilities Ethics Committee (MEC/10/27/EXP).

The survey components reported here comprised:

- A. 17 items on the purpose and practice of palliative care and hospice services. Response to these questions were sought using a 5-point Likert scale from *strongly disagree* to *strongly agree*.
- B. 16 items on the participants, 12 on socio-demographics (e.g. gender, age, marital status, education, state of health, strength of connection with family, importance of religion and

spirituality to them, and personal experience with palliative care and death) C. 15 items on attitudes on dying and death, again on a 5-point Likert scale.

C. 1 open-ended question to gauge awareness of their nearest hospice.

Data were collected via an online survey using the Nielsen *Your Voice* panel, an online community designed to provide members with a forum to voice their opinions on a number of matters (www.yourvoice.net.nz).

The survey commenced on 27 July 2010 and continued until 2 August 2010 when the online tool was taken down off the Internet. The survey was expected to take under 11 minutes to complete, and a representative population-based sample of 1011 was anticipated and obtained. Invitations to panellists (18 or over) were sent via e-mail which contained a link to the survey. A sampling matrix was used to ensure that the number of surveys sent to different age groups, regions, and gender was in line with the makeup of the New Zealand (18+) population.

The results were also weighted by age, gender and region to account for any minor imbalances the sampling matrix was unable to account for.

Results

Respondents were distributed throughout the country; 48% were male and 52% female. The age and gender of participants broadly represent New Zealand's population (Table 1) with an over-representation of NZ European responders.

Table 1. Population sample characteristics

Age (years)	Survey %	Ethnicity	Survey%	New Zealand population %*
18–20	4	NZ European	85	77
20–29	18	Māori	6	15
30–39	19	Pacific	–	7
40–49	20	Chinese	3	10
50–59	17	Indian	2	
60–69	15	All Other	9	
70+	7	Total	105 †	†

* www.socialreport.msd.govt.nz; † Some identified 2 nationalities.

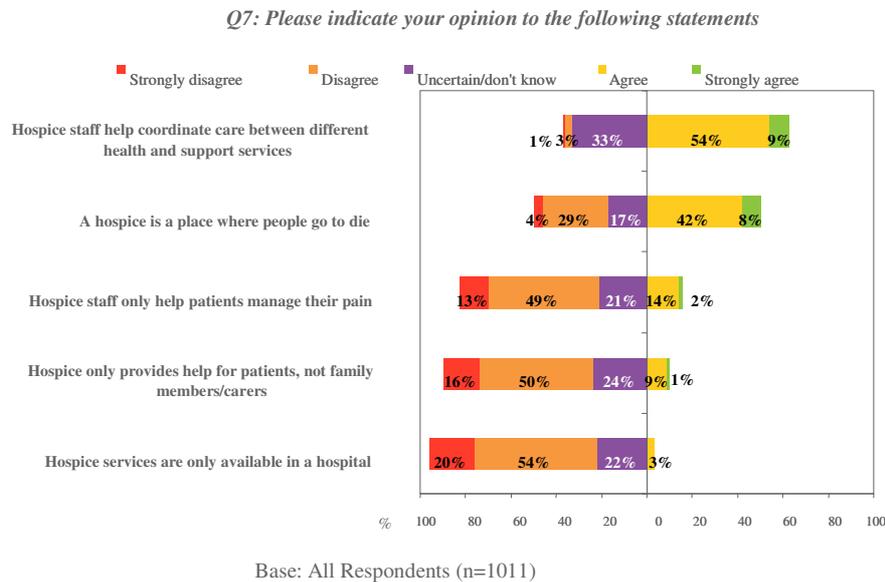
Awareness of hospice activities

Where differences between subgroups and the total sample are identified here these are all statistically different at 95% level.

Figure 1 demonstrates the New Zealand public's awareness of hospice and hospice activities. Only half of the respondents agreed that a hospice is a place where people go to die, while two-thirds agreed or strongly agreed that hospice staff help coordinate care across health and support services.

Just over a third of respondents (35%) correctly named their local hospice. Many respondents simply assumed that their local hospital was where their local hospice was located, but others thought it was in retirement villages or even at the Ronald McDonald House.

Figure 1. Differences by age



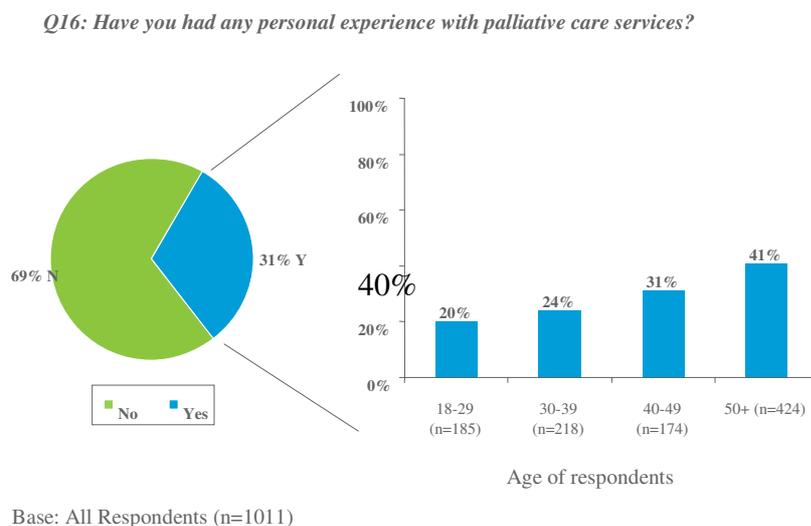
People aged 50 and over had greater confidence that hospice staff coordinate care between a number of different health and support services (71% agreeing cf. 63% average for all respondents). Respondents aged 50 and older also illustrated more accurate perceptions of hospices, as they were more likely to *strongly disagree* with the following statements:

- Staff only help patients manage their pain (19% cf. 13% average for all respondents).
- Hospices only provide help for patients, not family members/carers (20% cf. 16% average for all respondents).
- Hospice services are only available in hospitals (27% cf. 20% average for all respondents).

In comparison, people under the age of 30 were the least likely to be accurate in their responses to the above three statements.

Experience of palliative care—Figure 2 illustrates that around three in ten respondents indicated they had had personal experience with palliative care services. Experience increased with age, with people aged 50 or older twice as likely as those under the age of 30 to have had experience.

Figure 2. Personal experience of palliative care services



Of the two main types of community palliative or hospice care services available, inpatient care was the most likely service that New Zealanders reported experience with. However, 1 in 10 respondents did not know what type of palliative care service they had been exposed to, and this gap was more likely to be the case for those under the age of 30 (24% cf. 9% average for all respondents).

Understanding of palliative care

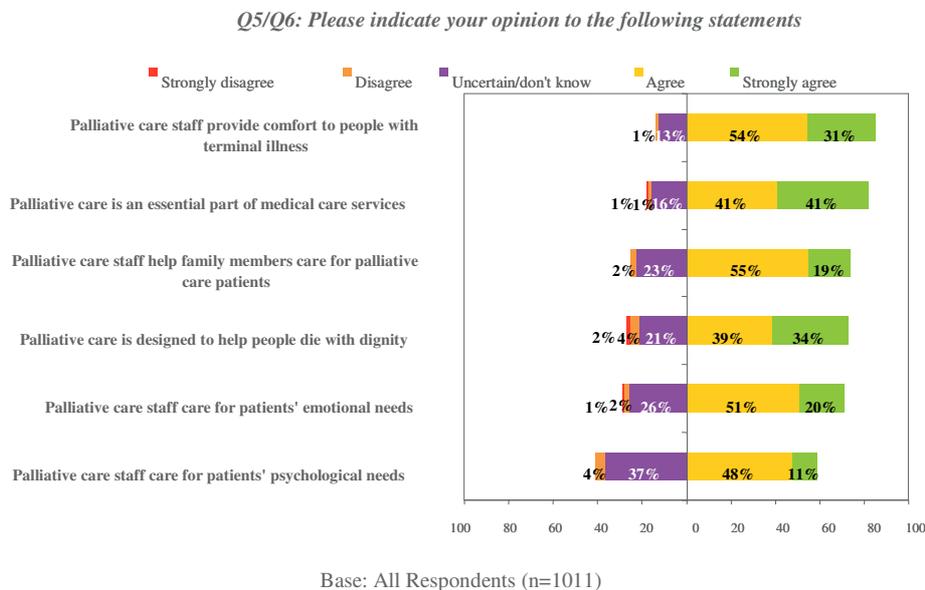
Figure 3 reveals that the respondents as a whole were most in agreement with the statement “palliative care staff provide comfort to people with terminal illness,” followed closely by “palliative care is an essential part of medical care services” and “palliative care staff help family members care for palliative care patients.”

Differences by age—Age was again found to be a factor in public support for palliative care, as people aged 50 or over placed more value on palliative care, with half (51%) strongly agreeing that it is an essential part of medical care services as compared to 41% for all respondents. This older age group also had more accurate perceptions of palliative care as evidenced by higher *strongly agree* scores for the following statements:

- Palliative care staff provide comfort to people with terminal illness (43% cf. 31% average for all respondents)
- Palliative care is designed to help people die with dignity (50% cf. 34% average for all respondents)
- Palliative care staff care for patients’ emotional needs (27% cf. 20% average for all respondents)

- Palliative care staff care for patients' psychological needs (17% cf. 11% average for all respondents).

Figure 3. Opinions about palliative care statements (part 1)



Opinions about palliative care

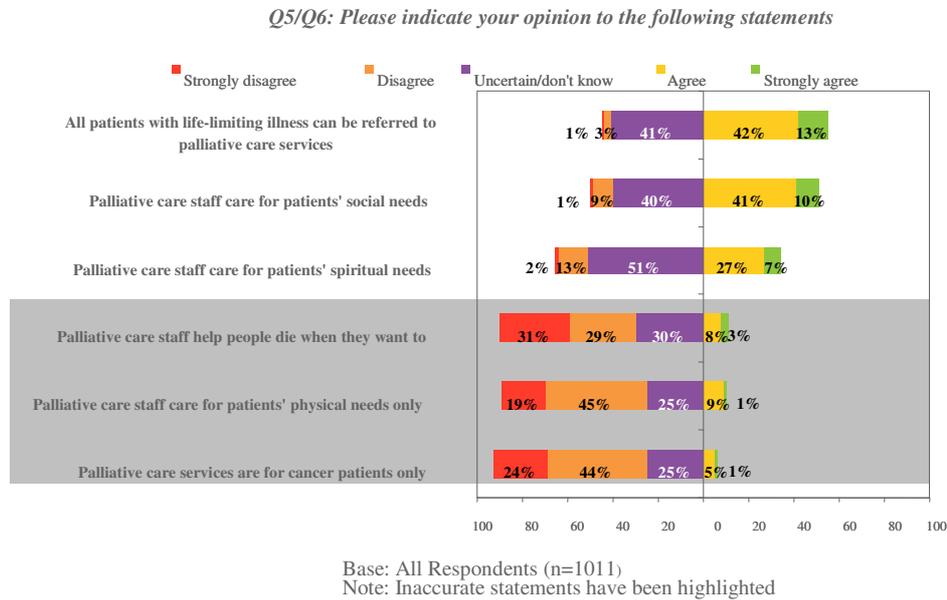
The majority of respondents recognised accurate inaccurate statements about palliative care (see Figure 4), but more than half were unsure about spiritual care services being provided in hospices.

Differences by age—Age was a factor in knowledge of palliative care services, as people aged 50 or over were more likely to know that palliative care staff care for patients' social needs (15% cf. 10% average for all respondents). In addition, people aged 50 or over were more likely to disagree or strongly disagree with three inaccurate statements:

- Palliative care staff care for patients' physical needs only (74% cf. 64% average for all respondents).
- Palliative care staff help people die when they want to (e.g. physician assisted suicide – 66% cf. 59% average for all respondents).
- Palliative care services are for cancer patients only (76% cf. 68% average for all respondents).

Those under the age of 30 were again more likely to be uncertain or not have an opinion about any of the statements about palliative care.

Figure 4. Opinions about palliative care statements (part 2)



Differences by gender—Females were revealed as more informed about hospice, as shown by three key findings:

- Females were more likely to strongly disagree that hospices only provide help for patients, not family members/carers and that hospice services are only available in hospitals (21% and 23% respectively cf. 10% and 17% for males).
- Females were also more likely to strongly agree that hospice staff help coordinate care between different health and support services (12% cf. 6% for males).

There was also a clear gender difference in perceptions of palliative care with females more likely than males to agree or disagree with all statements depending on their accuracy.

Personal experience with death of a family member or close friend

To provide context for this section we evaluated respondents' personal experience with death, their current state of health, importance of religion and religious group they identify with, importance of spirituality, and level of connection with family.

Around 8 in every 10 respondents have personally experienced the death of a close friend or family member (we take this to mean have been bereaved rather than a more literal experience of being at the bedside). Irrespective of age, personal experience of death is very high, with seven in every ten respondents under the age of 30 also having personally experienced the death of a close friend or relative.

Figure 5. Personal experiences of death (part 1)

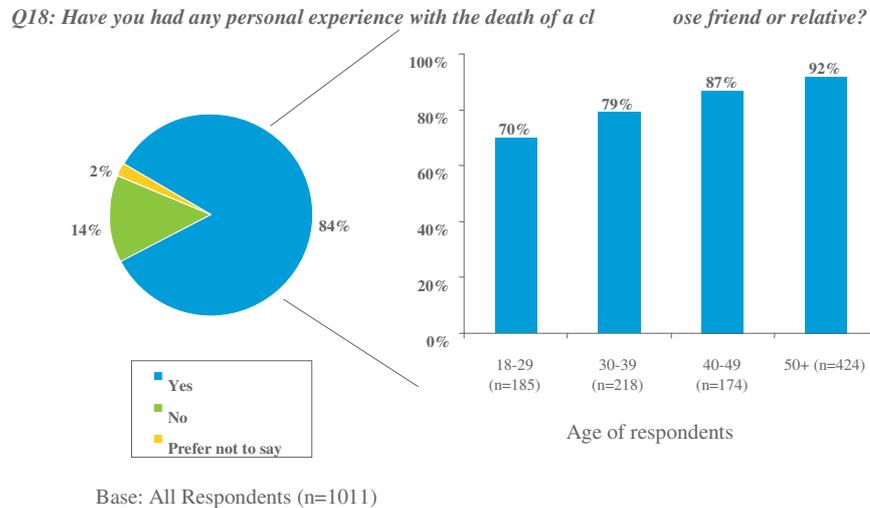
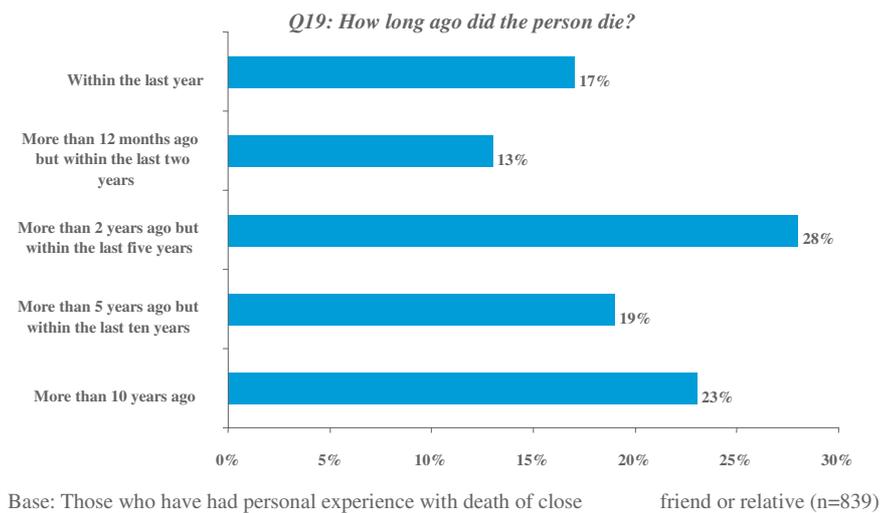


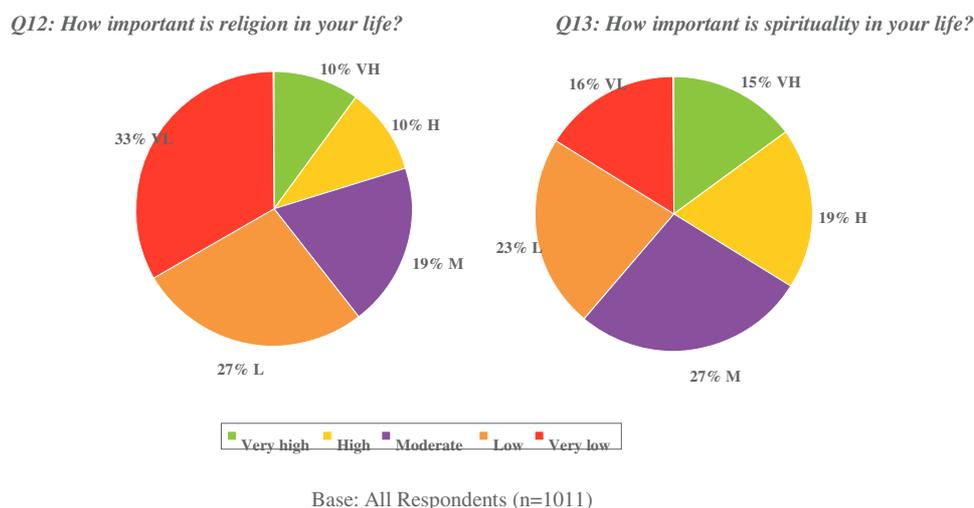
Figure 6 illustrates that for the majority of respondents the death of a close friend or relative occurred more than 2 years ago.

Figure 6. Personal experiences of death (part 2)



Religion was considered important (both high and very high importance) by only two in every 10 respondents (Figure 7). In comparison, one third of respondents (34%) considered spirituality to be of high or very high importance in their life.

Figure 7. Importance of religion and spirituality



Differences by age—Although people aged in their thirties were more likely to say religion was of *very low* importance (41% cf. 33%), the importance of spirituality was consistent across age groups.

Differences by gender—The only difference in importance of spirituality was the greater likelihood for females to say it was of high or very high importance in their life (40% cf. 28% for males). In terms of strength of connection with family, just over three quarters believe they have a strong or very strong connection with their own family.

Discussion

This research revealed that although New Zealanders generally understand the concept of palliative care, 65% of respondents were unable to name their local hospice. Despite high levels of awareness that hospice support is designed for both patients and families, there were lower levels of understanding of the psychosocial and spiritual services available in hospices. Not surprisingly, people under the age of 30 were commonly the most unaware of hospice services. However, 82% of all respondents believe palliative care is an essential health service.

These findings reflect the experiences and expectations of a society where palliative care has been a part of some districts' health services for over 30 years. It is possible that some respondents had experience of hospice that did not include all the services

that could be available, so skewing their responses. For example, not all hospices have care coordination or spiritual care available specifically.

One of the other findings that needs further attention is that a significant number (34%) of New Zealanders consider spirituality to be of high or very high importance in their life. Given that the survey also identifies that a majority of people do not think, or were uncertain, that palliative care staff address spiritual needs, this is something that providers of care may need to attend to.

It is important for the achievement of Strategic goal 6 of the NZ Palliative Care Strategy that education of the public continues so that the true nature of hospice and palliative care services is understood and sought when required.²

Competing interests: None declared.

Author information: Rod MacLeod, Honorary Clinical Professor, Department of General Practice and Primary Health Care, School of Population Health, University of Auckland, Auckland; Rachel Thompson, Funding & Communications Advisor, Hospice New Zealand, Wellington; John W Fisher, Honorary Senior Fellow, School of Rural Health, Faculty of Medicine, University of Melbourne, Melbourne, Australia; Kristine Mayo, Associate Director, Client Service, The Nielsen Company, Wellington; Nathan Newman, Senior Client Service Executive, The Nielsen Company, Wellington; Donna M Wilson, Professor – Faculty of Nursing, University of Alberta, Edmonton, Canada

Acknowledgements: The authors gratefully acknowledge the support of Hospice New Zealand in facilitating this project as well as Mundipharma NZ Ltd for funding the research.

Correspondence: Rod MacLeod, Department of General Practice and Primary Health Care, School of Population Health, University of Auckland, Auckland, New Zealand. Fax: +64 (0)9 3737624; email: rd.macleod@auckland.ac.nz

References:

1. http://www.stats.govt.nz/browse_for_stats/population/estimates_and_projections/demographic-trends-2010/chapter4.aspx
2. King A. The New Zealand Palliative Care Strategy, Wellington, Ministry of Health; 2001.
3. Palliative care in New Zealand. Ministry of Health. 2010. <http://www.moh.govt/palliativecare>
4. Tomas-Sabado J, Limonero JT. Religiousness and death anxiety. In: Ambrose SD (ed), Religion & psychology: New research. USA: Nova Publishers; 2006:107-122.
5. Palliative Care Australia. National Community Education Initiative. 2009. <http://www.palliativecare.org.au/Default.aspx?tabid=1949>

Ocular trauma epidemiology: 10-year retrospective study

Archana Pandita, Michael Merriman

Abstract

Aims This study is performed to determine the epidemiology and incidence of ocular trauma in Waikato, New Zealand.

Methods The data was analysed on age, sex, ethnicity and type of trauma. Visual acuity (VA) at presentation and discharge was recorded. Details of slit lamp examination were noted.

Results There were a total of 821 injuries. Men had higher rate of ocular trauma than women (74% vs 26%, $p < 0.001$). Mean age was 31 years for males and 37 years for females respectively. Highest number of ocular trauma was seen in age group 15–20 (11.5%). There were 253 open globe injuries (OGI) and 568 closed globe injuries (CGI) ($p < 0.001$). The most frequent causes of eye injuries in men were related to outdoor activities (25.9%) and work (20.7%). In women, outdoor activity was also the highest cause (10%).

The annual rate of ocular trauma was 20.5 per 100,000 populations. Final VA of $\geq 6/12$ was found in 590 eyes, $6/12$ – $6/60$ in 143 eyes and $\leq 6/60$ in 88 eyes. Primary or secondary vitreoretinal procedures were performed in 54 eyes. There were three cases (0.3%) of endophthalmitis (inflammation of the internal coats of the eye).

Conclusion This study provides insight into epidemiology of ocular trauma in Waikato, New Zealand.

Ocular trauma is a major public health issue. Ocular trauma mostly commonly occurs at work, at home, during sport activities, motor vehicle crashes or interpersonal trauma. Reported risk factors are male gender, workplace, road accidents, alcoholism and lower socioeconomic class.^{1–3} It is a significant but preventable cause of blindness worldwide.

Ocular trauma has an impact on the healthcare system and also the wider economy due to time off work. Negrel and Thylefors reported that worldwide 1.6 million people are blind secondary to ocular injuries, 2.3 million with low visual acuity bilaterally and 19 million with unilateral blindness or low vision.⁴

Around 29,000 eye injuries occur in Australia annually.¹ Hospital admissions are not a complete record of prevalence and risk factors for ocular trauma because mild to moderate injuries such as corneal foreign bodies and corneal abrasions are not included as they are treated as an out patient. However nearly all severe and blinding injuries can be captured by using hospital admission data. Not only is the trauma often preventable but appropriate management of injuries can have a significant reduction on the burden of the visual impairment.

Waikato is the fourth largest region in New Zealand and has a population of just over 400,000. It includes Hamilton, a large urban centre (41% of population), but mainly

comprises rural areas and small towns (59% of population). The purpose of this retrospective study is to determine the incidence and risk factors associated with ocular trauma in Waikato.

Waikato Hospital provides secondary and tertiary care to the region and serves as a major referral centre for a geographical area of 25,000 km². The department of Ophthalmology at Waikato Hospital offers both emergency and specialised care for ocular diseases and conditions for patients of all age groups.

A better understanding of the risk factors associated with it can help to design targeted campaigns to reduce the incidence of ocular trauma in community and develop effective plans for disseminating eye injury prevention material to the public.

Methods

For this study case notes were reviewed with ocular trauma who presented to Waikato Hospital from January 1999 to December 2008. Patient records were identified by computer search using Waikato Hospital's RUS codes (Resource Utilization System, equivalent to ICD codes) for all patients admitted.

Ocular injury was defined as any injury affecting eye or adnexa identified as principal discharge diagnosis. RUS codes for ocular trauma included contusion, penetrating wounds of orbit with and without foreign body, perforation, rupture, ocular laceration with and without prolapse or loss of intraocular tissue, injury of conjunctiva and corneal abrasion.

Patient data extracted included age, sex, ethnicity, type of trauma (sharp/blunt), location and nature of trauma, chemical injuries and burns. Visual acuity at the time of presentation and discharge was recorded using the Snellen's visual acuity chart. Details of slit lamp examination including fundus examination were noted.

Any computed tomography and ultrasound findings were recorded. Primary and secondary repair including adjuvant treatment was noted. Medical and surgical history was recorded. Any alcohol consumption at the time of trauma was noted. Injuries were classified by the standardized international classification of ocular trauma, Birmingham Eye Trauma Terminology system (BETTS).⁵

Statistical analysis was performed in conjunction with a professional biomedical statistician. Frequency distributions were created for injury type and cause. The eye injury rate was calculated using denominator obtained from NZ institute of statistics. Statistical analysis of quantitative data was performed for all variables. Frequency analysis was performed by the Chi-squared test. One way analysis of variance (ANOVA) was used to evaluate difference in parametric variables. Chi-squared test or Fischer exact test was used as appropriate. All p values were two tailed and p-value less than 0.05 was considered statistically significant.

Results

There were total of 821 patients who were admitted to Waikato Hospital with ocular injuries between January 1999 and December 2008. Among these 52 % were New Zealand European, 35% were Maori and 13% were from other ethnicity (mainly Asian, Pacific, or Middle East). NZ Europeans are 80% of total Waikato population and Maori are the 15% of population.

Adjusting for age and gender, men had higher rate of ocular trauma than women (74% vs 26%, $p < 0.001$, Pearson's Chi-squared test). The mean age for all patients in this study was 35.5 years (range 0–98 years). Mean age for males was 31.0 (± 20.8) and for females was 37.0 (± 24.7 ; $p = 0.005$ ANOVA test). Median age was 42 years. No significant difference in frequency of right vs left eye injuries was noted ($p = 0.522$).

Figure 1. Eye trauma incidence in various age groups over 10 years period

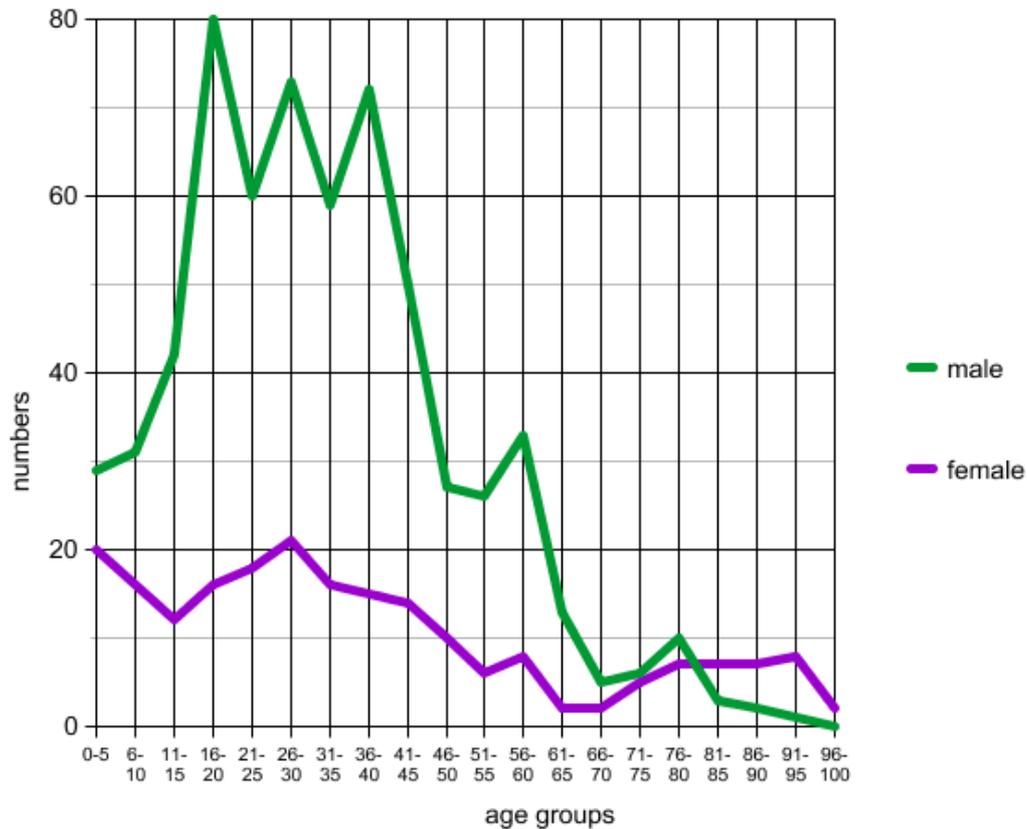


Table 1. Activity when eye injury occurred

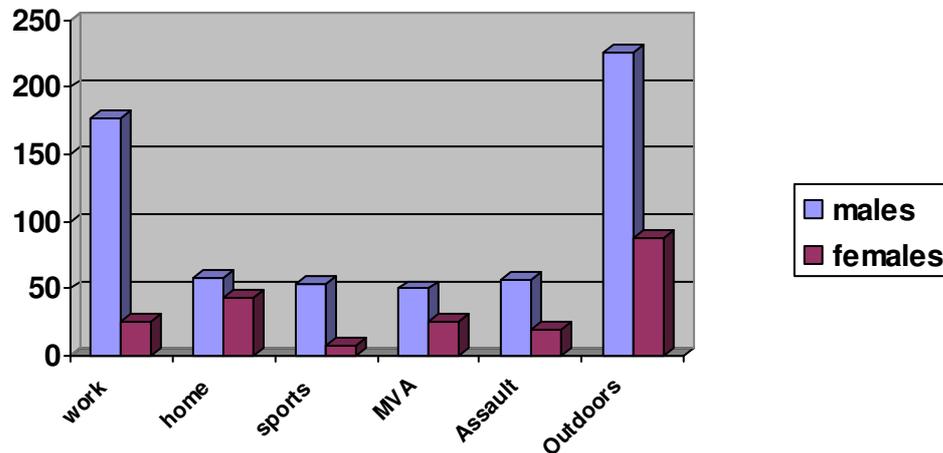
Activity	No. of eyes	Percentage	SE Asia ²⁰	Australia ¹
Assault	81	9.8	8.8	3.0
Motor Vehicle Accident	65	7.9	13.1	1.9
Sports	62	7.6	3.1	3.3
Falls at home	31	3.7		
Work (professional & DIY)	203	24.7	48.1	60
Hammering/chiselling/chainsawing	71			
Fencing/farm related	89			
Others	44			
Chemical	61	7.5	4.7	
Fireworks	5	0.7	1.8	
Others	313	38.1	20	

DIY=do it yourself work.

The incidence of eye injury was seen to be decreasing with increasing age. The maximum number of injuries was seen in the age group 16-20 years and 26-30 years (11.5% and 11.3% respectively, Fig 1). 21.5% of the New Zealand population is less than 15 years and 12.3% is above 65 years age. The younger age group had assault and metal work as a main cause of ocular injuries.

Elderly people had ocular injuries mainly from falls. Using the Birmingham Eye Trauma Terminology System (BETTS), contusion affecting the anterior segment occurred most frequently among different forms of ocular trauma (Table 2).

Figure 2. Frequency of injury by gender and activity



There was a correlation between activity when injured and gender ($p < 0.001$; Pearson Chi-squared test) (Figure 2). Outdoor activity related injuries accounted for 34.8% of injuries in men followed by work related injuries (29%). The most frequent cause of ocular trauma in women was outdoor activity related (43%) followed by home related work (20%). Assaults accounted for 9.2% of all injuries and among these alcohol use was documented in 73.6%.

Table 2. Type of eye injury

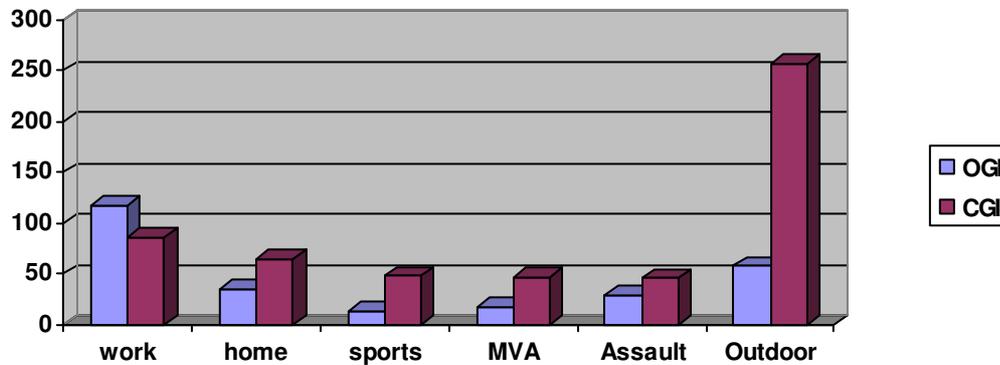
BETTS	No. of eyes	Percentage
Contusion	461	56.1
Lamellar laceration	107 (corneal 102, conjunctival 5)	13.0
Penetrating injury	170	20.7
Perforating injury	5	0.6
Intraocular foreign body	19	2.3
Rupture	59	7.2

BETTS=Birmingham Eye Trauma Terminology System.

There were 253 open globe injuries and 568 closed globe injuries ($p < 0.001$; Pearson's Chi-squared test). There was a significant difference in the frequency of open and closed globe injuries in work related injuries (58% open vs 42% closed, $p = 0.044$; Fisher's exact test), sports-related injuries (23% open vs 77% closed, $p < 0.005$; Fisher's exact test), outdoor related activities (17% open vs 83% closed, $p < 0.005$; Fisher's

exact test) and in MVA-related injuries (28% open vs 72% closed, $p=0.001$; Fisher's exact test) respectively (Figure 3).

Figure 3. Frequency of injury by open globe/closed globe



A vitreoretinal procedure was performed in 54 eyes and three among these had repeat vitrectomy to improve vision. Nineteen eyes had vitrectomy for intraocular foreign bodies, 15 metal and 4 glass. Seventeen eyes had vitreoretinal procedures for retinal detachment repair. Another 17 eyes had reasons such as retinal haemorrhages, lens dislocation, decreased visual acuity after primary repair. One had vitrectomy for dislocated intraocular lens after blunt trauma.

There were 82 ocular injuries presenting to Waikato Hospital on average per year with an incidence rate of 20.5 (CI 19.3–21.5) per 100000 population (Fig 4). Incidence rate for CGI was 14.2/100,000 and for OGI was 6.3/100,000 population. Overall 590 eyes had BCVA (Best corrected visual activity) $\geq 6/12$ (71.8%), 143 had VA between 6/12 (17.4%) and 6/60 and 88 eyes had VA $\leq 6/60$ (10.7%) at final follow-up visits.

Generally it was seen that eyes injured with blunt objects had final BCVA of 6/12 or better compared to those injured by sharp objects. The majority of chemical injuries (82%) happened at work with sodium hydroxide and sodium hypochlorite being the dominant agents. Chemical injuries occurring at home (18%) were usually due to cleaning agents. All the chemical and firework injuries had full recovery of VA except one firework injury in a corneal graft patient whose VA was reduced from 6/9 to hand movements.

Figure 4. Incidence of ocular injuries per year

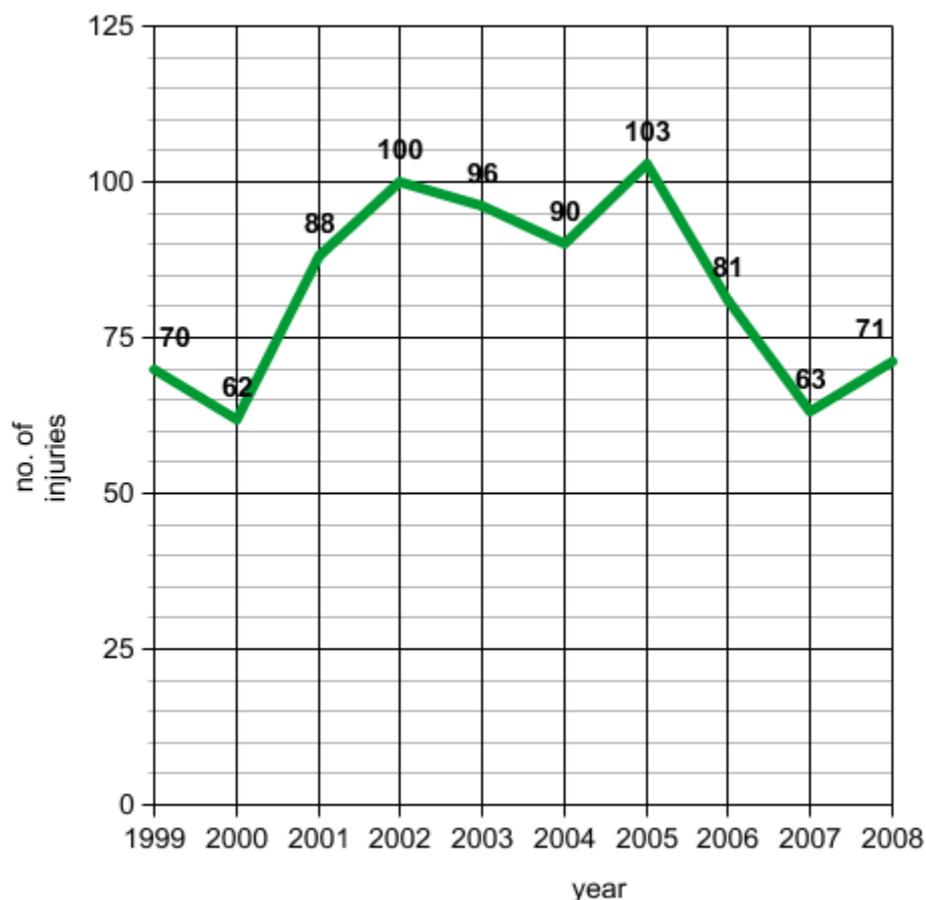


Table 3. Source of trauma for Visual acuity less than or equal to 6/60

Causes	No. of eyes (88)	Percentage
Assault (9 enucleations, 3 pthisis)	24	27.3
Farm related/fencing/wiring (5 enucleations, 2 evisceration)	18	20.5
Falls (1 enucleation, 2 evisceration)	9	10.2
MVA (6 enucleations, 1 evisceration, 1 pthisis)	8	9.1
Hammering/chiselling/chainsawing (2 enucleations, 1 evisceration)	8	9.1
Sports (3 enucleations)	7	7.9
Gunshots (1 enucleation)	2	2.3
Fireworks	1	1.1
Others	11	12.5

The most common primary surgery was reconstitution of the globe integrity with repositioning or excision of extruded ocular contents and suturing of the wound. There were a total of 27 (3.2%) enucleations performed during this period that included 12(44.4%) primary and 15(55.6%) secondary enucleations.

Six (0.7%) eviscerations were performed, three for penetrating eye injuries, one for endophthalmitis resulting from IOFB (intraocular foreign body) and one each for complications from motor vehicle accident and scleral rupture.

Of the 19 cases of IOFB three developed endophthalmitis (two metallic and one organic). Two had bacillus species identified, the other had negative cultures. Two of these were treated with intravitreal antibiotics and vitreoretinal procedures and third one underwent evisceration.

Four eyes (0.5%) developed phthisis. No case of sympathetic ophthalmia (inflammation to both eyes following trauma to one eye) was seen in this study. Angle recession was noted in 1.5% (n=13) eyes.

Discussion

Ocular trauma is an important cause of visual loss and is frequently preventable. This study documents the nature of the ocular trauma over a 10-year period. We must acknowledge that only inpatients are included. However most patients with sight-threatening injuries are admitted.

Some closed globe injuries are treated as outpatients and will not be included in this study (e.g. Commotio retinae). Data was collected based on discharge coding, so some injuries would have been left out for example multisystem trauma with relatively mild eye injuries. As a retrospective study there will be recording bias. The vision at presentation and final follow-up was not recorded for every injury. Not all patients had complete treatment at Waikato Hospital so their final outcome may not be known.

Estimation of ocular trauma rates depends on the data source. Hospital data does not represent the total number of patients with eye trauma as not all the less severe injuries will present to hospital, but it will provide useful information regarding sight-threatening injuries which are of greatest concern.

This study provides insight into epidemiology of ocular trauma in New Zealand. It also supports the previous reports that ocular trauma may represent a significant cause of visual loss in the population.

The rates of eye injuries requiring hospital admission range from 8-57/100,000 population.^{1,6-10} This study showed an incidence rate of 20.5/100,000 which is more than other reports from United States but comparable to Australia (21/100,000).¹ This could be related to factors such as increased outdoor activities in this region and comparatively easier access to public health system.

As a large geographic area, Waikato has an extensive road network that could contribute to a greater number of road traffic accidents compared to other regions. Some of our patients identified and included were from outside our region but treated at Waikato Hospital due to their location at time of injury e.g. MVA, or transferred to our centre for specialised treatment (e.g. vitreoretinal surgery).

Higher frequency of ocular injury in men was seen in our study population and occurred in all age groups. A higher male preponderance may be related to occupational exposure, participation in dangerous sports and hobbies, alcohol use and risk taking behaviour.¹¹ Other studies also reported higher rate in men compared to women.^{1,3,15}

This study showed that sports and motor vehicle accidents contribute to about 15% of ocular injuries. Rugby was found to be the commonest cause of sports injury followed by tennis and squash. Other less common sports eye injuries were from fishing, soccer, boating, golf, shooting and paint ball.

Work-related equipment was found to be the other major contributory risk factor for ocular injuries. Among these, lawn mowing caused highest eye injuries and others included fencing, hammering, tree pruning and grinding.

Excess alcohol consumption was identified as a contributing factor in approx 13.8% of injuries in our cohort, predominantly in assault and MVA activities. Of the 88 eyes with severe visual loss, assault and MVA caused 36%. Three quarters of eye injuries due to assault involved alcohol consumption.

Closed globe injuries have better prognosis compared to open globe injuries.¹² Pieramici et al described that good presenting visual acuity of 6/60 or better is associated with less incidence of enucleation.¹³ This is consistent with other studies and is an important prognostic factor when counselling these patients. However vitreoretinal procedures in eyes with vision of hand movements resulted in measurable vision improvement in subsequent follow-up in some of our patients.

This study detected three cases of post traumatic endophthalmitis. This was 1.1% of the open globe injuries. No post operative endophthalmitis was seen. Post-traumatic endophthalmitis is reported in 2–12% of eyes with open globe injuries in other studies.¹⁴⁻¹⁹

Risk factors include delayed presentation, delayed commencement of antibiotics and presence of intraocular foreign body. This study has a comparatively low rate of endophthalmitis which is probably a consequence of prompt presentation, early antibiotic treatment and surgery.

There was a single gunshot perforating wound in the ten years of our study. This eye had pars plana vitrectomy, lensectomy and laser vision improved to 6/5 BCVA.

Ocular trauma is a significant cause of visual disability. Typically it impacts younger people and may dramatically affect their future, independence and work. Health education and appropriate preventive measures should therefore be directed at these high risks.

Our study shows that educating workplaces and people doing high risk tasks such as drilling, grinding, chain sawing and farm fencing would be a good target to reduce ocular injuries. Adequate eye protection is often a simple step. Education of the community regarding the high association of alcohol and eye injuries leading to blindness from MVA and assaults could be a useful in changing society's attitude to alcohol use.

Competing interests: None declared.

Author information: Archana Pandita, Registrar; Michael Merriman, Consultant; Department of Ophthalmology, Waikato Hospital, Hamilton

Correspondence: Archana Pandita, 26 Kentwood Drive, Wellington 6037, New Zealand. Email: panditaarchana@yahoo.com

References:

1. McCarty CA, Fu CL, et al. Epidemiology of Ocular Trauma in Australia. *Ophthalmology* 1999; 106:1847-52.
2. Thylefors B. Epidemiological patterns of ocular trauma. *Aust NZ J Ophthalmol* 1992; 20:95-8.
3. Cillino S, Casuccio A, et al. A five-year retrospective study of the epidemiological characteristics and visual outcomes of patients hospitalized for ocular trauma in Mediterranean area. *BMC Ophthalmol* 2008, 8: 6.
4. Negral AD, Thylefors B. The global impact of eye injuries. *Ophthalmic Epidemiology* 1998;5:43-69.
5. Kuhn F, Morris F, et al. The Birmingham Eye Trauma Terminology System (BETTS). *J Fr Ophthalmol* 2004; 27:206-10.
6. McGwin G, Xie A, Owsley C. The rate of eye injuries in the United States. *Arch Ophthalmol* 2005; 123:970-76.
7. Desai P, MacEwen CJ, et al. Incidence of cases of ocular trauma admitted to hospital and incidence of blinding outcome. *Br J Ophthalmol* 1996;80:592-96.
8. Wong TY, Tielsch JM. A population based study on the incidence of severe ocular trauma in Singapore. *Am J Ophthalmol* 1999;128:345-51.
9. Wong TY, Klein BE, et al. The prevalence and 5-year incidence of Ocular trauma-The Beaver Dam Eye Study. *Ophthalmology* 2000;107:2196-202.
10. Loon SC, Tay WT, et al. Prevalence and risk factors of ocular trauma in an urban south-east Asian population: the Singapore Malay Eye Study. *Clin and Exp Ophthalmology* 2009;37:362-67.
11. Koo L, Kapadia MK, et al. Gender differences in etiology and outcome of open globe injuries. *J Trauma* 2005; 59:175-78.
12. Knyazer B, Levy J, et al. Prognostic factors in posterior open globe injuries. *Clin and Exp Ophthalmology* 2008; 36:836-41.
13. Pieramici DJ, MacCumber MW, et al. Open globe injuries. Update on types of injuries and visual results. *Ophthalmology* 1996;103:1798-803.
14. Smith ARE, O'Hagan SB, Gole GA. Epidemiology of open and closed globe trauma presenting to Cairns Base Hospital, Queensland. *Clin Experiment Ophthalmol* 2006;34:252-9.
15. Soliman MM, Macky TA. Pattern of ocular trauma in Egypt. *Graef's Arch Clin Exp Ophthalmol* 2008;246:205-12.
16. Peyman GA, Carroll CP, Raichand M. Prevention and management of traumatic endophthalmitis. *Ophthalmology* 1980;87:320-4.
17. Fan JC, Neiderer RL, et al. Infectious endophthalmitis : clinical features, management and visual outcomes. *Clin and Exp Ophthalmol* 2008;36:631-36.
18. Zhang Y, Zhang MN et al, Jiang CH, et al. Endophthalmitis following open globe injury. *Br J Ophthalmol* 2010; 94:111-14.
19. Essex RW, Yi Q, Charles PG, et al. Post traumatic endophthalmitis. *Ophthalmology* 2004;111:2015-22.
20. Chang CH, Chen CL, et al. Hospitalized eye injury in a large industrial city of South-Eastern Asia. *Graef's Arch Clin Exp Ophthalmol* 2008; 246:223-228.

Aspiration pneumonia and challenges following the Samoa Tsunami in 2009

Tamara Ah Leong-Nowell, Foloto Leavai, Lucilla Ah Ching, Limbo Fiu, Rosemary Wyber, Mitzi Nisbet, David Jones, Tim Blackmore, Tupu Ioane-Cleverley

Abstract

On 29 September 2009, a large tsunami struck the Samoan Islands in the South Pacific Ocean, causing 142 deaths and large numbers of casualties. 199 patients presented to the emergency department within the first 72 hours. Twenty-nine patients were admitted with respiratory symptoms and histories of aspirating contaminated seawater and were diagnosed with tsunami-associated aspiration pneumonia. These patients were initially treated with empiric antibiotics based on drug availability and published experience after the Asian Boxing Day Tsunami of 2006. Antibiotic treatment was subsequently modified with sputum culture information. The good outcomes of the Samoa Tsunami patients may be attributed to early initiation of appropriate antibiotics and timely coordinated management.

Samoa is a small independent developing island nation in South Pacific Ocean with a 2009 population of 183,203 people.¹ On 29 September 2009, a large tsunami struck the south east coast of the main island of Upolu following an offshore undersea earthquake of 8.1 magnitude.² The worst affected areas were low-lying villages about 2 hours drive across the island from the capital, Apia. The Tupua Tamasese Meaole (TTM) National Hospital in Apia, was the major treatment centre for casualties from the tsunami.

142 people died following the disaster, representing 0.08% of the total population.³ 199 patients presented to the emergency department within the first 72 hours. This paper describes the cohort of patients who were assessed by the medical team and diagnosed with tsunami-associated aspiration pneumonia.

The National Hospital's immediate response during the acute phase was coordinated and performed by the Samoa Disaster Medical Team. The Samoan team was supported by the Australian Quick Response Team (who arrived the day after the tsunami) and subsequently by the New Zealand Disaster and Emergency Response Team. Samoan volunteer doctors and nurses from New Zealand, Australia, United States, Canada and other parts of the world arrived in the days following the tsunami and were incorporated into the team.

This violent disaster resulted in much orthopaedic and surgical trauma, however the majority of victims had medical rather than surgical injuries. Tsunami-associated aspiration pneumonia is a unique condition which occurs when patients are submerged by tsunami waves resulting in the inhalation of saltwater, sand, foreign bodies and waste matter, usually under pressure.

Care for these patients was provided by the local staff in the TTM Medical Unit, which at the time of disaster consisted of one house surgeon, four registrars and a

general medical consultant who also assumed the role of disaster administrator. Following the tsunami, an improvised tsunami ward was immediately opened and largely run by junior medical staff. Any patient that was under the surgical team who was identified by either the anaesthetist or surgical team as having aspirated were also referred to the medical team for further assessment.

This retrospective descriptive case series describes the cohort of patients who had tsunami-associated aspiration pneumonia including the clinical characteristics, treatment and outcome. It provides an opportunity to reflect on the lessons learnt and to consider recommendations for the future.

Methods

The 29 patients admitted with aspiration pneumonia to TTM Hospital within 72 hours of the tsunami impact were included in this retrospective descriptive case series.

The diagnosis of tsunami-associated aspiration pneumonia was made in patients who were submerged, had respiratory symptoms with supporting clinical and radiological findings. All patients with aspiration pneumonia had bilateral infiltrates on chest X-ray imaging. Acute respiratory distress syndrome (ARDS) was defined as the development of bilateral infiltrates associated with poor oxygenation despite high flow oxygen and no clinical evidence of left ventricular failure. Some of these 29 patients also had tsunami related barotrauma, psychological problems, soft tissue injuries and concurrent pulmonary tuberculosis.

Patients who were admitted to the surgical unit were excluded from this review, unless they were specifically referred to the medical unit for management and had a history consistent with tsunami-associated aspiration pneumonia.

A chart review was completed to obtain information on clinical characteristics, antibiotic treatment, radiological findings and outcome. Culture information is reported on sputum samples that were collected between 2 to 5 days following the tsunami. Follow-up data is also provided from clinics that occurred at two, six and 7 months following the tsunami.

Results

Of the 29 aspiration pneumonia patients identified, seven (24%) were male and the median age was 41 years (range 14–95). Five (17%) patients had oxygen saturations of less than 70% on room air at presentation. Most patients with aspiration pneumonia had multiple complications as outlined in Table 1.

Table 1. Medical diagnoses in aspiration pneumonia patients

Aspiration pneumonia
ARDS
Lobar collapse
Rib fractures
Pneumothorax
Pulmonary tuberculosis
Soft tissue infections and fasciitis
Vertigo
Psychological

The radiological findings in the patients with tsunami-associated aspiration pneumonia included bilateral pulmonary infiltrates in all those admitted, rib fractures in two patients, a pneumothorax in two patients and a left lower lobe collapse in one

patient. The lobar collapse was presumably due to the aspiration of a foreign body and resolved with physiotherapy.

Five patients (17%) out of the 29 included in the cohort had presumed acute respiratory distress syndrome (ARDS) and all of these patients had oxygen saturations of less than 70% at presentation.

A series of cases and medical issues that were observed following the tsunami are described below:

- A 21-year-old male with severe respiratory distress initially improved clinically and radiologically on broad spectrum antibiotics and high flow oxygen, but then acutely deteriorated 6 days later. A repeat chest X-ray showed interval changes of widespread reticulonodular infiltrates. He had a brief period of non-invasive ventilation in the form of CPAP provided by a borrowed machine from outside the hospital. This was poorly tolerated due to poor fitting of the only available nasopharyngeal interface and was discontinued a short period later.

Seventy-two hours after discontinuing CPAP he had a further acute deterioration due to a large pneumothorax that required an intercostal drain. Throughout his hospital admission he expectorated black sand mixed with purulent sputum. His sputum cultured *Citrobacter* spp. and *Pseudomonas aeruginosus*. On day 12 intravenous meropenem 1g 8 hourly was commenced with clinical improvement. Arterial blood gases were not able to be performed.

- A 95-year-old presented more than 36 hours after the tsunami died after she declined ongoing active medical intervention. The remaining 28 patients all survived to discharge from hospital.
- One patient was observed to have bilateral upper lobe infiltrates at presentation and an additional diagnosis of smear positive tuberculosis was also made.
- Skin and soft tissue infections were common; one patient was initially admitted to the high dependency unit (HDU) with aspiration pneumonia and on day 3 was also found to have thigh fasciitis and was transferred to New Zealand on the next available flight. The other one was diagnosed with perineal infection and required a prolonged admission
- Vertigo was one of the most common complaints after the third day following the tsunami and was attributed to eardrum trauma from the tsunami waves.
- There were a number of tsunami medical patients who had an acute stress reaction and for these patients psychological concerns were an immediate and ongoing priority.

Microbiology—Sputum was cultured from 15 of the initial patients who presented with aspiration pneumonia (Table 2). An organism was subsequently identified from 12 of these cultures. Five patients had polymicrobial cultures. *Streptococcus* spp. was identified in seven patients and *Pseudomonas aeruginosa* in six cultures. Three

patients grew *Citrobacter* spp., two cultured *Proteus* spp., and there was one culture each of *Klebsiella* spp., *Pantoea* spp., and *Enterobacter* spp.

Table 2. Isolates cultured from sputum from tsunami aspiration pneumonia patients

Species	N
<i>Streptococci</i> spp.	7
<i>Pseudomonas aeruginosa</i>	6
<i>Citrobacter</i> spp.	3
<i>Proteus</i> spp.	2
<i>Klebsiella</i> spp.	1
<i>Pantoea</i> spp.	1
<i>Enterobacter</i> spp.	1

Antibiotic treatment—Several antibiotics were used as initial empiric treatment however this was rationalised by day 2 to a standard four antibiotic regimen (IV gentamicin, IV cefuroxime 750mg 8 hourly, oral metronidazole 400mg 6 hourly and oral cotrimoxazole 960mg 12 hourly) for all patients that required hospital admission due to aspiration pneumonia.

All patients with respiratory distress at initial presentation had received dexamethasone and frusemide however this was discontinued after 24 hours. Patients were discharged on either oral amoxicillin-clavulanate 625mg three times daily or oral cotrimoxazole 960mg twice daily unless cultures suggested that other treatment was required such as ciprofloxacin 500mg twice daily to treat *Pseudomonas aeruginosus*.

Expectant treatment was recommended for vertigo. Intervention for psychological sequelae was provided for patients with persistent concerns as required.

Follow-up—Three follow-up clinics were conducted for aspiration pneumonia patients at 2, 6 and 7 months following the tsunami. Chest X-rays were repeated in all patients at 2 months, and in any patient who had ongoing symptoms at 6 and 7 months following the tsunami.

Only one of the 29 patients required a further admission shortly after their initial discharge and was diagnosed with both a psoas abscess and septic arthritis of the ankle. This patient had severe respiratory compromise at the time of the initial admission and these subsequent infections were either due to secondary seeding or suppression of these infections due to antibiotic treatment for pneumonia so that they were not identified until after all antibiotic treatment was stopped. He had normal saturations (100% on room air) at follow-up but had persisting extensive small nodules on chest X-ray

Seventeen (59%) patients attended the 2-month follow-up clinic. Eleven (38%) patients reported ongoing respiratory symptoms predominantly with cough and sputum production. A further three (10%) patients reported weight loss following the tsunami. Most patients had resolution of chest X-ray abnormalities but a number had persisting nodular infiltrates or peri-bronchial inflammation.

Thirteen (47%) patients returned for a follow-up assessment at 6 months. Only those with continuing cough and or dyspnoea were asked to be followed up by the Respiratory specialist the following month. All those seen had recovered both clinically and radiologically including the patient with pulmonary tuberculosis. To date there have been no identified long-term pulmonary complications.

Discussion

The immediate use of antibiotic polytherapy was based on the pathogens that had been identified as causing aspiration pneumonia and skin infections in the 2004 Boxing Day Asian Tsunami. Tsunami-associated aspiration pneumonia in Asia was complicated by infection with multi-resistant organisms and *Burkholderia pseudomallei*, the latter of which has not previously been reported in Samoa.

Given limited local pathogen data, the medical team asked tsunami patients where they were found; many recalled being rescued on land used for pigsties, refuse tips, septic tanks, cemeteries, or the road. Empirically, the initial four antibiotics used provided cover for water borne pathogens such as *Vibrio* and *Aeromonas*, plus other potential pathogens including *Pseudomonas* and *Nocardia*.

Co-trimoxazole was used to cover for *Nocardia* which is commonly found in soil. Unlike the Boxing Day Asian Tsunami, carbapenem antibiotics are not routinely available in Samoa so were not used except for in one case of the young man with complicated pulmonary infection who failed to settle on available treatment.

The sputum culture information provided by the Samoan microbiology laboratory was invaluable for targeting ongoing antibiotic treatment. The laboratory was not able to test for *Nocardia* but did identify a number of patients who had pneumonia due to Enterobacteriaceae including a number of organisms with potential for inducible beta-lactamase activity (sometimes referred to as “ESCAPP” organisms).

Streptococcal species were also identified from sputum in a number of patients. These organisms were not speciated and may have represented normal oral flora rather than a being a pathogenic organism.

In the course of writing this article a retrospective literature review was performed to compare the Samoa experience with international experience post tsunami and confirmed that the Boxing Day Tsunami literature was the only information available to assist our empirical antibiotic treatment.

It was difficult to know how much Indian Ocean microbiology data could be extrapolated to the Samoa situation but it was assumed that *Vibrio* might be a contributing pathogen. It was not possible to locate any aspiration pneumonia microbiology relating to the Papua New Guinea tsunamis of 1998 and 2000 that would have provided relevant Pacific experience.

Of particular concern was *Burkholderia pseudomallei* which had caused a number of severe cases of melioidosis in the Indian Ocean tsunami setting.^{4,9-11} This Gram-negative bacterium is endemic in soil and surface water of South East Asia and North Australia and cases have been reported nearby Papua New Guinea and New Caledonia.^{10,12} Diabetes is an important risk factor for severe necrotising pneumonias and Samoa had 23.1% of the adult population in a 2002 survey.^{13,14}

The majority of the Samoa Tsunami patients who were admitted to hospital for surgical injuries had been submerged and had also inhaled contaminated seawater. Nevertheless, their trauma injuries took precedence over their respiratory problems and it is possible that the number of patients with aspiration pneumonia may have been greater than were clinically diagnosed. These patients received similar empirical antibiotic treatment as those with aspiration pneumonia that would have been sufficient to treat pulmonary infection.

A number of key challenges were identified as listed below:

- The use of dexamethasone in aspiration pneumonia is not usually recommended, however it was administered to this cohort for the initial 24 hours and the outcomes were generally good.
- High flow oxygen was routinely used however there was no established facility to use invasive or non-invasive ventilation. An attempt was made to establish one severely hypoxic patient who was not maintaining adequate oxygen saturations despite high-flow oxygen on non-invasive ventilation by using a donated CPAP machine. There may be benefits to having non-invasive ventilation available in future emergencies.
- Treatment was largely empiric for most patients but it was presumed that there may have been contributing Gram negative and waterborne organisms. The large influx of acutely unwell patients meant that all clinical services including the microbiology laboratory were stretched beyond capacity. Antibiotic treatment was rationalised as culture information became available. Laboratory equipment failure added to the challenges with renal function and arterial blood gas tests not being available for the initial 10-day period. Gentamicin levels cannot be performed in Samoa and so gentamicin was discontinued as soon as clinical response was observed but was continued in patients with severe sepsis.
- The Radiology Department resources were adequate, but many patients had portable bedside chest X-rays performed with anterior posterior views limiting the clarity of images especially in obese patients.
- Codeine phosphate and paracetamol were the only readily available analgesics for severe chest-wall pain. Poor analgesic control limited physiotherapy and potentially delayed recovery in some patients.
- Regular and effective chest physiotherapy was essential for the initial management of aspiration pneumonia but was limited as there was only one physiotherapist for the entire hospital.

Lessons from the Samoan Tsunami—Most tsunami victims were submerged in contaminated seawater. Respiratory problems with lung injuries and ARDS predominated and early onset of sepsis and necrotizing fasciitis complications can occur. A number of infections involved more than one organism. Infections due to atypical organisms and fungi were not reported from the Samoa Tsunami and although some of these would require a specialised laboratory to identify it was reassuring that all but one patient (who had extensive pneumonia) responded to empiric therapy that did not include antifungal treatment.

Treatment of surgical injuries tended to be undertaken with urgency, and it is important not to overlook accompanying immersion lung injury. Other cohorts have reported that patients with delayed diagnosis of tsunami aspiration pneumonia generally have worse clinical outcomes.¹⁵

The internet literature search of previous tsunami experiences was very helpful given most medical workers have no prior experience of tsunami disasters. However there were difficulties in accessing many of the desired articles due to subscription only databases. Local staff recommend the establishment of a single international database for the management of medical problems for tsunami victims based on previous tsunami evidence data that is readily available and easily accessible at a time of need.¹⁵

Prompt discussions with infectious disease (ID) physicians, locally or internationally, at an early stage are crucial for local practitioners. In country projects to identify common local pathogens and to determine antibiotic sensitivities would be invaluable, particularly in resource limited countries where there maybe a scarcity of antibiotic choices. Knowledge of local antibiograms would also assist in improving daily clinical practices.

Clear lines of communication between clinicians, Ministry of Health and disaster organisations, both locally and internationally, is important to help facilitate access to required resources during the initial acute period. Our team presented a list of required medical needs to the disaster team coordinators on day 3.

Local doctors generally have the best understanding of the local health situation as well as resource constraints. The role of international health professionals should be to enhance local capacity and to provide expert knowledge to assist the local medical personnel.¹⁵

Few health professional came with prior tsunami experience and knowledge, therefore briefing at the earliest opportunity is essential. The medical team was able to inform newly arrived colleagues on the nature of tsunami injuries together with our treatment approach to help guide management of these patients.³

Generally, when international disaster health teams depart, the devastated country is left with the enormous task of dealing with continuing and additional problems in the medium and long term. Hence links with local Primary Health Care Providers were enhanced by quickly producing a “Post-Tsunami Medical Information Booklet” and antibiotic guidelines to raise awareness and guide management of tsunami related medical problems.³

Recommendations—The ideal medical team should consist of physicians (General, Infectious Disease, Respiratory), microbiologists and physiotherapists. Support should be provided by a laboratory with regard to the identification of atypical and seawater borne pathogens. Antibiotics such as a carbapenem, clindamycin and co-amoxicillin-clavulanate should be rapidly available. Equipment such as pulse oximeters, non-invasive ventilation, chest drains and possibly a bronchoscope need to be at hand.

Managed coordination and streamlined procurement and allocation of appropriate resources are essential. The local Internal Medicine Unit, in conjunction with the

overseas physicians, played an important early strategic role in the management of patients with tsunami related respiratory and medical problems in a disaster situation where resources are limited.

Conclusions

Tsunami patients admitted with aspiration pneumonia in the Samoa disaster had very good outcomes for these patients, which can be explained by the early initiation of appropriate treatment and antibiotics provided by a well coordinated local team. This management approach was driven by necessity, locally available resources and the application of basic clinical practice guided by overseas expert knowledge. Literature reviews support the interventions taken.

This paper illustrates the importance of sharing tsunami experiences both locally and internationally to improve disaster management response and practice. Acquired knowledge, expertise, resources and lessons learnt should be integrated into disaster response policies in tsunami prone regions. Local medical staff is fundamental to the success of initial and ongoing responses to any future disasters.

Competing interests: None declared.

Author information: Tamara Ah Leong-Nowell, Senior Medical Registrar, Internal Medicine, Tupua Tamasese Meaole (TTM) National Hospital, Apia, Samoa; Foloto Leavai, Junior Medical Registrar, Internal Medicine, TTM Hospital, Apia, Samoa; Lucilla Ah Ching, Junior Medical Registrar, Internal Medicine, TTM Hospital, Apia, Samoa; Limbo Fiu, Head of Unit, Clinical Services ACEO, Internal Medicine, TTM Hospital, Apia, Samoa; Rosemary Wyber, Junior Registrar, Internal Medicine, TTM Hospital, Apia, Samoa; Mitzi Nisbet, Infectious Disease Specialist, Infectious Disease Department, Auckland Hospital, Auckland; David Jones, Respiratory Physician, Wellington Hospital, Wellington; Tim Blackmore, Infectious Diseases Physician and Microbiologist, Capital & Coast District Health Board, Wellington; Tupu Ioane-Cleverley, Medical Doctor, Wellington Hospital, Wellington

Acknowledgements: The authors thank Glenn Fatupaito, Agnes Iosefa and Claire Matheson for their assistance with this study.

Correspondence: Tamara Ah Leong-Nowell, email: tcnowell@gmail.com or Tupu Ioane-Cleverley, email: tupu@pti.co.nz

References:

1. Samoa Bureau of Statistics (SBS) <http://www.sbs.gov.ws/tabid/3277/language/en-US/Home.aspx>
2. United States Geological Survey (USGS). <http://earthquake.usgs.gov/earthquakes/recenteqsww/Quakes/us2009mdbi.php>
3. Samoa Medical Unit report: Ioane-Cleverley T. "Medical Team at Tupua Tamasese Meaole (TTM) National Hospital Report of Response to the Samoa Tsunami, 29 September 2009." Samoa Tsunami report sent to TTM Hospital Samoa and to the New Zealand Ministry of Health, October. 2009.
4. Allworth A. Tsunami lung: a necrotising pneumonia in survivors of the Asian tsunami. *MJA* 2005;182(7):364.
5. Potera C. Infectious disease: In disaster's wake: tsunami lung. *November 2005*;113(11): A734.

6. Peter JV, John P, et al. Corticosteroids in the prevention and treatment of acute respiratory distress syndrome (ARDS) in adults: meta-analysis. *BMJ* 2008;336:1000-1009.
7. Garzoni C, Emonet S, Legout L, Benedict R. Atypical infections in tsunami survivors. *Emerging infectious diseases* 11. 10. 2005 1591-2.
8. Kongsangdao S, Bunnag S, Siriwiwattanakul N. Treatment of survivors after the tsunami. *N Eng J Med* 352;25 2654-5
9. Athan E, Allworth A, Engler C, et al. Melioidosis in tsunami survivors *Emerging Infectious Diseases* 2005;11(10):1637-8.
10. Le Hello S, Currie B, Godoy D, et al. Melioidosis in New Caledonia. *emerging infectious diseases*. 2005;11(10):1607-9.
11. Chierakul W, Winothai W, Wattanawaitunechai C, et al. Melioidosis in 6 tsunami survivors in southern Thailand. *Clin Infect Dis* 2005 Oct 1;41(7):982-90.
12. Currie B, Fisher D, Howard D, et al. The epidemiology of melioidosis in Australia and Papua New Guinea. *ActaTropica* 2000;74(2-3):131-127.
13. Suputtamongkol Y, Chaowagul W, Chetchotisakd P, et al. Risk factors for melioidosis and bacteremicmelioidosis. *Clinical Infect Dis* 1999;29(2):408-13.
14. Viali S, Pasa E, Quested C, et al. Samoa STEPS survey. Preliminary Report, Ministry of Health, Samoa, 2004.
15. Ioane-Cleverley T, Nowell T, Ah Ching L, et al. "Physicians In The First Wave To The Samoa Tsunami Rescue, 29 Sept 09", WCIM Oral Presentation, Melbourne, Australia, March 2010.

‘The way things are around here’: organisational culture is a concept missing from New Zealand healthcare policy, development, implementation, and research

Shane L Scahill

Abstract

Internationally, healthcare sectors are coming under increasing pressure to perform and to be accountable for the use of public funds. In order to deliver on stakeholder expectation, transformation will need to occur across all levels of the health system. Outside of health care it has been recognised for some time that organisational culture (OC) can have a significant influence on performance and that it is a mediator for change. The health sector has been slow to adopt organisational theory and specifically the benefits of understanding OC and impacts on performance. During a visit to health research units in the United Kingdom (UK) I realised the stark differences in the practice of health reform and its evaluation.

OC is a firmly established concept within policy development, implementation and research in the UK. Unfortunately, the same cannot be said for New Zealand. There has been unrelenting reform and structural redesign, particularly of the primary healthcare sector under multiple governments over the past 20 to 30 years. However, there has been an underwhelming focus on the human aspects of organisational change. This seems set to continue and the aim of this viewpoint is to introduce the concept of OC and outline why New Zealand policy reformists and health services researchers should be thinking explicitly about OC. Culture is not solely the domain of the organisational scientist and current understandings of the influence of OC on performance are outlined in this commentary. Potential benefits of thinking about culture are argued and a proposed research agenda is presented.

During a visit to prominent health service research units in the United Kingdom (UK) I was struck by the stark differences in the development, implementation and research activity associated with health policy reform.

Discussions with academic leaders and their staff highlighted an understanding and focus toward *Organisational Culture* (OC) as an important concept within health reform. These individuals have considerable political clout, being advisors to the National Health Service (NHS). To a great academic leaders of this type have ensured that the concept of OC is not a forgotten ingredient of organisational change. They have not allowed the term OC to become a buzzword; confined to the hallways of health service research units or policymaking departments.

These leaders have actively promoted an understanding of OC through their research which influences policymakers and those working at the coal face. The agenda is for OC to assist efforts to improve health systems through a focus on both structural and social change.¹

A definition of organisational culture

OC has been described in lay terms as ‘the way things are around here’²; the commonly held beliefs and values about the ‘way(s) we think and act’³; within organisations.³⁻⁵ The most commonly cited definition of OC in the healthcare literature is one by Edgar Schein:

“...the pattern of shared basic assumptions – invented, discovered or developed by a given group as it learns to cope with its problems of external adaption and internal integration – that has worked well enough to be considered valid and, therefore to be taught to new members as the correct way to perceive, think, and feel in relation to those problems”²

OC has been borrowed from anthropology, denoting the collective thinking that drives normal behaviour within a group with common goals.⁴⁻⁸ Organisations are human systems that engage in activities that create and distribute value for key stakeholders in order to ensure organisational longevity.⁹⁻¹¹

Both structural and human components need to be considered when understanding organisational change. Understanding the way(s) we think and act collectively at the organisational level is important.

Why bother studying the culture—effectiveness link?

When attempting to develop and implement health policy, OC is an important concept for change at multiple levels within the health system.^{1,12} Outside of the healthcare sector, OC is recognised as an important factor in influencing organisational effectiveness and success.^{8,13-19}

Supported by popular literature^{2,19-21} OC has largely been conceptualised as one of many organisational factors that influence success. OC is a variable, easily manipulated by leadership to improve productivity, competitiveness and financial sustainability.^{16,18} The health sector has been slower than other sectors to adopt organisational development strategies.

Slowly, OC has been embraced as an important concept in the USA²²⁻²⁴ and the UK.^{1,25-29} Interest was based on the realisation that to deliver effective health care within a defined fiscal cap, human change is required in addition to structural considerations.

The same cannot be said for the historical development of OC in New Zealand. Here OC is relatively unknown, unspoken, unwritten and under-researched phenomenon in health circles. In this viewpoint I outline the international drive for healthcare transformation; aimed at improving performance.

I argue the importance of OC and present the learning from research predominantly from the UK. The potential impact on New Zealand policy and practice through ignoring the concept of OC is outlined and a research agenda is presented.

Organisational culture as a mediator for change

In recent years the health sector has come under increasing pressure to perform and to be made accountable for use of public funds.^{30,31} Despite subtle differences in developed healthcare systems, one thing is commonplace; the unrelenting thrust by central policymakers for accountability and performance. All the while, disease states

are becoming increasingly complex, with more therapeutic options and limited resource. This is an international phenomenon and New Zealand has not bucked the trend.

Increased complexity alongside disasters such as the Shipman and Bristol Heart scenarios led to realisation by UK policymakers that safe service provision and greater productivity cannot be squeezed out of the system through structural change alone. OC has become a significant concept for understanding aspects of organisational transformation at all levels.

The definition of culture and the manifestations outlined can be thought about and applied to the context of organisations within the following three levels of the healthcare sector:

- The macro level—policy and policy setting organisations such as the Ministry of Health and the Pharmaceutical Management Agency (PHARMAC)
- The meso level—the health funders and planners including District Health Boards (DHBs) and PHOs
- The micro level—the healthcare providers including organisations such as general practice and community pharmacy

Over two decades policymakers in the UK have made explicit the need for OC transformation in order to reconfigure health care and demonstrate performance gains. The rationale was two fold. First, with regard to implementation, there has been a focus on aspects such as clinical governance which provides a framework for both structural and OC transformation at the ‘meso’ and ‘micro’ levels.

Second, significant research streams have been developed and continue to be well supported by the UK Government. This is through realisation that policy development and implementation will be better informed and more successful through an understanding of OC as a facilitator of change. The focus of these research streams is on the influence of OC on organisational performance, particularly within the secondary care sector.

New Zealand health reform and the lack of focus on OC

A system wide shift from a focus on the individual to population health outcomes has not been without its challenges in New Zealand. Improved service delivery and resultant health gains were expected to occur through *structural* redesign of the health system. This structural change is evidenced by the incremental morph of primary care support organisations, over the past two decades, under Labour and National Governments.

In addition to structural change, the focus has been on applying financial models in an attempt to make the system more efficient. This argument is supported by the introduction in the late 1980s of Independent Practitioner Associations (IPA) and subsequently Primary Health Organisations (PHOs). These structural changes cemented ‘middle’ level structures within the health sector.

Significant resources were allocated for the development of IPAs. Later through PHOs, capitation based funding was being applied in order to shift focus from patient

to population and to increase utilisation of the multi-disciplinary primary care team. Neither of these objectives has been achieved in full; possibly due to divergent professional subcultures³² found within and across primary and secondary health care. A divide between clinicians and management and the 'rise of managerialism' is likely to have contributed to this.³³

The National Government under the guidance of Health Minister, Rt Hon Tony Ryall calls for *Better, Sooner, More Convenient Primary Health Care*.³⁴ The main driver for delivery of care is infrastructure rather than OC change. With the development of large Integrated Family Healthcare Centres (IFHC) there is the expectation that general practices will amalgamate and health professionals will 'just get on with it'.

The expected gain in sector-wide efficiency is based on the notion that larger, co-located service providers will result in improved integration across all levels of the healthcare sector and higher performance. There is no consideration of history or sub-cultures that have manifest over long periods of time. Improved multidisciplinary teamwork, patient experience and health outcomes are expected through structural change, which masquerades as innovation by service co-location.

There is little evidence cited in government policy to support the notion that structural change and co-location will have the desired effect of improved healthcare performance. The IFHC strategy may prove to be less successful than expected, unless there is greater thought about the human aspects of change. There is an agenda for devolution of services from secondary to primary care but no money set aside for the development of IFHC.

Apart from physical collocation and service design, little thought has been given to the interaction (or lack of) between primary and secondary care; based on fundamental differences in the values, beliefs and behaviour. There is a strong focus on clinical leadership under the National Government and the tension between this and non-clinician management does not seem to have been considered.

There is a literature that advocates for simultaneous change across all levels of the health system, through establishing crises.¹² It is unfortunate that this is represented in New Zealand by 'rattling the health sector cage' from above.

There is a completely naive expectation that secondary care will happily devolve half of its activity to PHOs without a struggle and PHOs will easily amalgamate, whilst General Practices with vastly different cultures will also calmly band together in certain locations whilst funding themselves into IFHCs. The notion that DHBs will happily transfer their role to the private sector through this development of a two tier American style healthcare system will be interesting to observe.

The most common reasons for failure of organisational change activities are lack of development of a guiding coalition, not making explicit the short and long term gains and not embedding this into the culture.³⁵ 'Because a representative of the government has said so' or 'the government wants it done that way' has been common dialogue bouncing around the corridors of the health sector over the past two years.

Our politicians and their advisors need to rethink the human aspects of their policies; particularly the 'rattling cage' approach and the likelihood of healthcare organisations

co-operating because they are all put in the same building. It certainly doesn't work with politicians so why would it work within the much more complex arena of health care, where there is considerably more at stake.

Within the primary care sector there is no clear plan of how organisations or professional groups with divergent and disparate cultures might work together under one roof. This expectation is not likely to achieve anything more than discontent, a loss of productivity and job satisfaction and the continued mass exodus of the New Zealand health workforce that Professor Gorman the Health Workforce New Zealand Executive Director has talked about.^{36,37}

The tenets of New Zealand primary healthcare policy are not dissimilar to the UK. There has been the call for improvement in access to services alongside the provision of quality health care. This is expected through technological integration and multidisciplinary teamwork.^{38,39} The difference between the UK and NZ is the realisation in the UK that to effect change one has to understand the 'way things are around here' to begin with and how this might change in the future. The concept of OC is understood and deemed to be important.

Organisational culture has been made explicit in UK health policy development, implementation and research for more than a decade. Within the discipline of health services research much financial resource, time and energy and academic brainpower has been devoted to studying the influence of OC on performance. The aim of this work has been to better understand mechanisms of change within the complex environment of healthcare provision at multiple levels. The other main agenda has been to expose the healthcare sector to the realisation that physical system reconfiguration is not the sole lever for organisational change.

UK health policy is currently directing the dissolution of Primary Care Trusts which has a flow-on effect at the 'middle' level of planning, as well as for general practices at the coal face. General practitioner (GP) commissioning is expected to be the mechanism for change at these levels. Moving to this approach is likely to require structure and process change in order to achieve the health outcomes expected by policymakers.

For some time, UK healthcare policy has been explicit in its call for *cultural* as well as *structural* change in order to achieve delivery of accessible, equitable and high quality health care.¹ There will be a residual understanding of OC as the UK transitions into this next wave of reform; the GP commissioning of services. This is arguably the greatest policy shift which directly affects practitioners at the coal face and the idea that OC change may be required as part of the process will not be a new one, at least not at the policymaking level!

What New Zealand has missed out on

A lack of discourse and associated research activity on OC in New Zealand could have significant implications for stakeholders; particularly those who have an interest in healthcare performance. In the UK, healthcare reform has developed with a focus on OC as a significant facilitator of change.^{1,40-43}

This has followed several strands which we could learn from in New Zealand:

Realisation of the importance of OC by policymakers—Major disasters relating to patient safety in the UK (Bristol Heart and Harold Shipman) resulted in a re-focus on the place of human activity in health service delivery. The development of clinical governance by policymakers was expected to curb flaws in health service delivery through a focus on culture as a lever for organisational change.^{1,41,43}

I am unaware of formal evaluation in New Zealand however, it appears that clinical governance activity has been limited to the mandatory requirement of the District Health Board New Zealand PHO Performance Framework (PPF). In 2005 and 2006 Dunedin based Best Practice Advocacy Centre (BPAC) undertook education sessions about clinical governance as part of the PPF program. This hardly constitutes the embedding of clinical governance as part of OC.

Recognition of the importance of OC by health service researchers—It has been increasingly recognised that OC has an influence on the performance of healthcare systems. There has been a flurry of interest in applying organisational theory to the healthcare sector, particularly in the UK and USA. Instruments to measure OC have been assessed^{44,45} and the conceptual methodological challenges of studying in this area have been identified.^{25,28,29} If culture is conceptualised as *the way(s) we think and act* within organisations³ then OC must have an influence on performance. It then becomes a matter of determining *in what way(s)* so that levers of change can be identified.

Apart from research within the community pharmacy sector, health service researchers in New Zealand have been relatively naïve to the benefits of pursuing an OC research agenda.^{3,46} The energy put into studying OC in the UK has not been replicated in New Zealand. As a result, we have a poorer understanding of approaches to organisational change, how OC manifests, and the ways in which dimensions of OC may mediate performance in both positive and negative ways.

Recognition by policymakers of the need to understand and evaluate OC change—The recognition by health policymakers, funders and planners of the need to understand and evaluate OC in health care, and the keen interest by health service researchers to do so, has resulted in a flourishing research agenda in the UK.

Studies suggest that performance in health care is contingent upon particular manifestations of OC. High and low performing hospitals in the UK demonstrate divergent patterns of OC.^{25,47} It is also possible that high performance may influence OC in recursive ways. That is, different OC may develop from high performing healthcare organisations over time, although more work is required in this area.⁴⁷

In the UK, Mannion and colleagues have noted a shift in the OC of healthcare organisations as a result of an increasingly market driven sector.⁴⁸ Understanding this sought of change is best undertaken through a cultural lens using robust and systematic research techniques. Such an approach has been followed in the UK.

There is a high awareness amongst clinical governance managers in the UK of the need for cultural renewal.⁴⁹ One third of managers surveyed were using OC assessment tools.⁵⁰ I am unaware of similar data available for New Zealand although I

suspect that the uptake of the notion of OC and the assessment tools will be at a considerably lower level.

The gaps: A research agenda for New Zealand

Deciphering the influence of culture on performance is a challenging area to study. Both are difficult to conceptualise, operationalise and separate as distinct concepts.^{25,28,29} Novel methodological approaches need to be thought about to achieve this. The majority of studies have been undertaken in the context of the secondary care sector and more OC oriented research is required in primary care.

Studies have involved OC associated with leadership²⁷ with little focus on the collective whole, who carry the culture within an organisation.^{7,17} Some work has been undertaken in the area of professional subcultures³³ but a lot more needs to occur in primary care. There is an increasing realisation that stakeholders contribute significantly to service co-production and are likely to be involved in co-production of service delivery²⁶ and so OC research that involves patients and communities will be important.

There is a need to consider the place of OC in influencing system-wide change. This is particularly the case under the devolution of services from secondary to primary care and the development of IFHC. I have previously discussed OC as a mediator for change and this section outlines this with respect to unanswered questions in the New Zealand context.

The micro level – the practice level—Exploring barriers and facilitators to multi-disciplinary teamwork between general practitioners, primary care nurses, community pharmacists and practice managers will be required under the IFHC model. The potential influence of professional subcultures on performance should be high on the research agenda. The PFP describes a list of performance indicators which reflect population health outcomes that are important to all New Zealanders.⁵¹

Experience as a clinical governance group member and Chair across a number of PHOs suggests there is marked variation in general practice performance. It is likely that some of the variance in performance is due to the different OCs that manifest as part of these practices. These differences need to be identified and understood. Adopting a cultural lens to achieve this is appropriate.

The meso level—At the funding, planning and implementation level clinical governance activities would be one focus of OC shift. Clinical governance groups have been a pre-requisite for PHOs entering the PPF however there is little knowledge of how this is operating in primary care.

Despite top-down health policy, the devolution of services from secondary to primary care organisations may also be influenced by OC; the values and beliefs which underlie each of these organisations which are significantly different. No better is this demonstrated than the recent ‘cage rattling’ at a national level and subsequent restructuring of DHB teams and PHOs across the greater Auckland area.

The macro level—Within the macro level—national policymakers; New Zealand healthcare policymaking and evaluation needs to align with other developed countries and be more explicit about the importance of OC as a mediator for change within

complex healthcare environments. Lack of consideration of humanistic aspects of organisational change provides the thrust of this paper.

In other developed countries such as the UK, there has been a systematic approach to the development of quality service provision and the identification of patterns of OC in both high and low performing organisations. UK policymakers have incrementally developed a way forward with respect to healthcare reform.

In New Zealand we have not been so broad or systematic. It appears the recent emphasis on change is at the implementation level where priorities stem from doing things 'because representatives of government said so' and 'government wants it done this way, before the election'. This is a reflection of the 'cage-rattling' approach alluded to previously.

Another macro-level issue is the fact there are a large number of PHOs serving disparate communities yet there is little understanding of the OC of these Boards. It would be useful to consider the increased policy focus on ethnic and socioeconomic disparities ('reducing inequalities') from the previous Labour Government and to explore whether this policy focus has changed organisational values, behaviours and thinking. It would also be interesting to know whether this has had an impact on organisational performance and if so, in what ways?

Amalgamation of PHOs is occurring, some joining through their own processes of rationalisation, others through government or local DHB coercion. Either way, there is little understanding of or consideration for the differing OCs and how this will affect top-down merger processes, and the implementation of programmes.

Over the years I have seen IPA and PHO amalgamations that seem rational on paper however, they have fractured due to incompatible OCs. The same could occur with IFHC development at the practice level. IFHC are more likely to be a merge of existing general practices, with their own cultures than as new practices outright.

The development of IFHC will be gradual and action research will be required to ensure that new IFHC learn from established centres. More important is the macro-level question of whether IFHC centres will demonstrate improved health gains over the system unchanged.

Conclusion

Internationally, healthcare sectors are under pressure to be accountable for the use of public monies and performance. In order to deliver on stakeholder expectation transformation across all levels of the healthcare system may be required. In the UK health sector there has been a focus on structural and systems change and human or social change.

Cultural change was introduced into policy development and implementation from the outset of reform. The UK Government has supported research which helps to understand structural and human change processes that influence health service delivery. In this paper I challenge current health policymakers, funders and planners, primary care support organisations and health research units to embrace the notion that organisational culture is too important to ignore.

Organisational culture does influence organisational effectiveness and determining the ways in which this occurs will impact on the success of all levels of health care in New Zealand.

Competing interests: None declared. The views expressed are those of the author and cannot be attributed to the University of Auckland, or any other third party in any manner.

Author information: Shane L Scahill, Senior Health Research Scholar, School of Pharmacy, University of Auckland

Acknowledgements: I thank the University of Auckland for awarding the Senior Health Research Scholarship and for funding the field visit; academic leaders at two Health Service Research Units in the UK for their time and guidance; and Associate Professor Amanda Wheeler for the time and energy she put into reviewing this manuscript.

Correspondence: Shane L Scahill, Senior Health Research Scholar, School of Pharmacy, University of Auckland, New Zealand. Fax: +64 (0)9 3677192; email: s.scahill@auckland.ac.nz

References:

1. Davies HTO, Nutley SM, Mannion R. Organisational culture and quality of health care. *Qual Health Care*. 2000;9:111-119.
2. Schein EH. *Organisational Culture and Leadership*. 1st ed. San Francisco, CA: Jossey-Bass; 1985.
3. Scahill SL, Harrison J, Carswell P, Babar ZUD. Organisational culture: an important concept for pharmacy practice research. *Pharm World Sci*. 2009;31(5):517-521.
4. Pettigrew AM. On studying organizational cultures. *Admin Sci Quart*. 1979;24:570-581.
5. Smircich L. Concepts of culture and organizational analysis. *Admin Sci Quart*. 1983;28:339-358.
6. Allaire Y, Firsirotu ME. Theories of Organisational Culture. *Organ Stud*. 1984;5:193-226.
7. Alvesson M. *Understanding organizational culture*. London: Sage; 2002.
8. Schein EH. *Organisational Culture and Leadership*. 3rd ed. San Francisco: Jossey-Bass; 2004.
9. Sanchez R., Heene A. *The New Strategic Management: Organisation, Competition and Competence*. New York: John Wiley & Sons, Inc; 2004.
10. Krell TC. Organizational longevity and technological change. *Journal of Organizational Change Management*. 2000;13(1):8.
11. Montuori LA. Organizational longevity: Integrating systems thinking, learning and conceptual complexity. *J Org Change Manag*. 2000;13(1):61-73.
12. Ferlie EB, Shortell SM. Improving the Quality of Health Care in the United Kingdom and the United States: A framework for change. *Milbank Q*. 2001;79(2):281-315.
13. Bate P. *Strategies for Cultural Change*. Oxford, UK: Butterworth-Heinemann Ltd; 1994.
14. Cameron K, Quinn RE. *Diagnosing and Changing Organisational Culture: Based on the Competing Values Framework*. 1st ed. USA: Addison Wesley; 1999.
15. Denison DR, Mishra AK. Toward a Theory of Organisational Culture and Effectiveness. *Organ Sci*. 1995;6(2, March-April):204-223.
16. Kotter J, Heskett J. *Corporate culture and performance*. New York: Free Press; 1992.
17. Martin J. *Organisational Culture: Mapping the Terrain*. 1st ed. Thousand Oaks: Sage; 2002.
18. Ouchi WG. *Theory Z*. Reading, MA: Addison-Wesley; 1981.
19. Peters TJ, Waterman RH. *In search of excellence*. New York: HarperCollins; 1982.
20. Peters TJ. *Thriving on Chaos*. New York: Knopf; 1987.
21. Schein EH. "Organisational Culture". *Am Psychol*. 1990;45(2):109-119.

22. Gerowitz MB. Do TQM Interventions Change Management Culture? Findings and Implications. *Qual Manage Health Care* 1998;6(3):1-11.
23. Gerowitz MB., Lemieux-Charles L., Heginbothan C. Top management culture and performance in Canadian, UK and US hospitals. *Health Serv Manage Res.* 1996;9:69-78.
24. Shortell SM., Jones RH., Rademaker AW., et al. Assessing the Impact of Total Quality Management and Organizational Culture on Multiple Outcomes of Care for Coronary Artery Bypass Graft Surgery Patients. *Med Care.* 2000;38(2):207-217.
25. Davies HTO, Mannion R, Jacobs R, Powell AE, Marshall MN. Exploring the Relationship between Senior Management Team Culture and Hospital Performance. *Med Care Res Rev.* 2007;64(1):46-65.
26. Hyde P, Davies HTO. Service design, culture and performance: Collusion and co-production in health care. *Hum Relat.* 2004;57(11):1407-1426.
27. Mannion R., Davies HTO., Marshall MN. Cultural characteristics of "high" and "low" performing hospitals. *J Health Organ Manage.* 2005;19(6):431-439.
28. Scott T, Mannion R, Davies H, Marshall M. *Healthcare Performance and Organisational Culture.* 1 ed. Oxford: Radcliffe Medical Press; 2003a.
29. Scott T, Mannion R, Marshall MN, Davies HTO. Does organisational culture influence health care performance ? A review of the evidence. *J Health Serv Res Pol.* 2003;8(2):105-117.
30. Gibberd RW. Performance measurement; is it now more scientific? *Intl J Qual Health Care.* 2005;17(3):185-186.
31. Saltmarshe D, Ireland M, McGregor JA. The performance framework: A systems approach to understanding performance management. *Pub Admin Develop.* Dec 2003;23(5):445-456.
32. Martin J. *Cultures in Organisations: Three Perspectives.* 1st ed. New York: Oxford University Press; 1992.
33. Degeling P, Maxwell S, Kennedy J, Coyle B. Medicine, management, and modernisation: a danse macabre? *BMJ.* March 22, 2003 2003;326(7390):649-652.
34. Ministry of Health. *Better, Sooner, More Convenient Primary Health Care: Ministry of Health;* 2007.
35. Kotter J. Leading change: Why transformation efforts fail. *HBR.* 1995;73(2):59-67.
36. Woodfield C. Doctor loyalty without extra dosh. <http://www.nbr.co.nz/article/doctor-loyalty-without-extra-dosh-cw-86583>; 22 February 2011
37. Wynyard R. Workforce talk knocks socks off. *NZ Doctor.* 2005;June 29:10.
38. Ministry of Health. *New Zealand Primary Health Care Strategy: New Zealand Ministry of Health;* 2001.
39. Ministry of Health. *Primary Health Care Strategy: Implementation Work Programme 2005-2010: New Zealand Ministry of Health;* 2006.
40. Davies HTO. Understanding organizational culture in reforming the National Health Service. *JR Soc Med.* 2002;95(3):140-142.
41. Freeman T, Peck E. Culture made flesh: Discourse, performativity and materiality In: Braithwaite J, Hyde P, Pope C, eds. *Culture and climate in health care organizations.* London: Palgrave MacMillan; 2010.
42. Mannion R, Davies HTO. *Cultures for performance in healthcare: Evidence on the relationship between organisational culture and organisational performance in the NHS.* York: Centre for Health Economics, University of York; 2003.
43. Scally G, Donaldson LJ. Clinical governance and the drive for quality improvement in the new NHS in England. *BMJ.* 1998;317(7150):61-65.
44. Jung T, Scott T, Davies HTO, et al. Instruments for exploring organizational culture: A review of the literature. *Pub Admin Rev.* 2009;69(6):1087-1096.
45. Scott T, Mannion R, Davies HTO, Marshall MN. The quantitative measurement of organizational culture: A review of available instruments. *Health Serv Res.* 2003;38(3):923-945.

46. Scahill SL. Improving community pharmacy practice by studying organizational theory. *Southern Med Rev.* 2008;1(1):18-20.
47. Mannion R, Davies HTO, Marshall MN. Cultural characteristics of "high" and "low" performing hospitals. *J Health Organ Manage.* 2005;19(6):431-439.
48. Mannion R, Davies HTO, Harrison S, Konteh FH, Jacobs R, Walshe K. Changing management cultures in the English National Health Service. In: Braithwaite J, Hyde P, Pope C, eds. *Culture and climate in health care organizations.* London: Palgrave MacMillan; 2010.
49. Konteh FH, Mannion R, Davies HTO. Clinical governance views on culture and quality improvement. *Clin Govern Int J.* 2008;13(3):200-207.
50. Mannion R, Konteh FH, Davies HTO. Assessing organisational culture for quality and safety improvement: A national survey of tools and tool use. *Qual Safe Health Care.* 2009;18(2):153-156.
51. District Health Boards New Zealand. PHO Performance Management Programme. DHBNZ; 2005.

Are Māori women at increased risk of cardiac complications of Graves disease?

Parul Nigam, Adam Morton

Abstract

We present a case series of three women of New Zealand Māori ethnicity, who presented to the emergency department of a Brisbane hospital (Brisbane, Australia) with symptomatic cardiac complications of Graves disease requiring hospital admission.

We raise the question as to whether individuals of Māori ethnicity are genetically susceptible to cardiac complications of thyrotoxicosis.

Case 1—A 35-year-old woman of New Zealand Māori ethnicity presented to the Emergency Department with palpitations, dyspnoea and muscle weakness. Graves disease had been diagnosed 4 years earlier but she had ceased carbimazole therapy 4 months prior to presentation. The only other significant history was of mild episodic asthma. Her cardiovascular risk factors included a 10-pack year smoking history but no other history of hypertension, dyslipidaemia or any significant family history of cardiac disease.

On clinical examination she was in atrial fibrillation (AF) with a ventricular rate of 120 beats per minute (bpm), blood pressure (BP) was 120/80 mmHg, jugular venous pressure (JVP) was elevated 5 cm, heart sounds were dual with a clear chest. Thyroid was diffusely enlarged with a bruit on auscultation, and there was ophthalmopathy with tethering of the right medial rectus muscle. Free thyroxine (FT4) was more than 77 pmol/L (normal 10–20 pmol/L), free triiodothyronine (FT3) was greater than 46 pmol/L and thyroid stimulating hormone (TSH) was suppressed.

Echocardiography revealed normal left ventricular size with global moderate systolic dysfunction (left ventricular ejection fraction 45%), moderately dilated right ventricular size with moderate dysfunction, moderate tricuspid regurgitation and moderate pulmonary hypertension with estimated right ventricular systolic pressure (RVSP) 60 mmHg. The woman was treated with prednisone 50 mg mane, carbimazole 60 mg per day, verapamil, digoxin and anticoagulation.

One month later her FT4 was 17.1 pmol/L and FT3 was 7.6 pmol/L. Repeat echocardiography 6 weeks after initial presentation showed normal left ventricular function (ejection fraction 55–60%), normal right ventricular function and improvement in pulmonary hypertension with RVSP 38 mmHg. She reverted to sinus rhythm and proceeded to thyroidectomy.

Case 2—A 75-year-old Māori woman presented to the Emergency Department because of palpitations, fatigue and heat intolerance. Her cardiac risk factors included a history of hypertension for the past 3 years and dyslipidaemia requiring medication. She was an ex-smoker.

On examination she was in AF with a ventricular rate of approximately 120 bpm, BP was 120/80 mmHg, JVP was elevated 4 cm with V waves, heart sounds were dual and chest was clear. There was a nodular goitre approximately 60 g in volume with no bruit and no ophthalmopathy. FT4 was 68.2 pmol/L, free T3 was 23.6 pmol/L and TSH was suppressed. Echocardiography revealed normal left ventricular size with globally impaired function with an estimated ejection fraction of 37%. There was mild right ventricular impairment, moderate to severe tricuspid regurgitation, and estimated RVSP was elevated at 45 mmHg. TSH receptor antibodies were elevated at 25 consistent with Graves disease.

She was treated with carbimazole 40 mg/day, prednisone 25 mg per day, propranolol 80 mg per day and was also anticoagulated. Two weeks later her FT4 was 27 pmol/L and FT3 was 7.2 mol/L and she remained in atrial fibrillation. Repeat echocardiography 6 weeks after her initial presentation revealed improvement in left ventricular function (estimated LVEF 45–50%) and pulmonary hypertension (RVSP 33 mmHg).

Two months after her initial presentation it was noted she had reverted to sinus rhythm, and repeat echocardiography one year after presentation was completely normal. Her thyrotoxicosis remains in remission 12 months after cessation of drug therapy.

Case 3—A 41-year-old woman of Māori ethnicity was brought to the Emergency Department having been noted to have tachycardia after a minor motor vehicle accident. Thyrotoxicosis due to Graves disease had been diagnosed 9 months earlier; however she had ceased thionamide medication 3 months prior to presentation. She had a 10-pack-year smoking history but no other significant cardiac risk factors.

On examination her heart rhythm varied between atrial fibrillation with ventricular rate of approximately 120 bpm, and atrial flutter with a 2:1 block. Blood pressure was 110/70 mmHg, JVP was elevated 4 cm with V waves, and clinically the pulmonary component of her second heart sound was loud. She had a diffuse goitre approximately 120 g with no bruit. FT4 was 57 pmol/L and FT3 more than 46 pmol/L. She reverted to sinus rhythm following a single dose of intravenous metoprolol, and was commenced on carbimazole 40 mg mane, prednisone 50 mg mane and atenolol.

Echocardiography showed normal left ventricular size and function (EF 60–65%), moderately dilated right ventricular size with normal function, severe mitral and tricuspid regurgitation with normal valve structure, and moderate pulmonary hypertension with RVSP 57 mmHg. Next echo done 4 weeks later revealed preserved left ventricular systolic function of 65%, right ventricular systolic pressure (RVSP) improved to 48 mmHg with moderate tricuspid and mitral regurgitation. She was clinically and biochemically euthyroid.

Asymptomatic pulmonary hypertension on echocardiography is common in individuals with hyperthyroidism. Siu et al found 47% of individuals with hyperthyroidism had pulmonary arterial systolic pressures greater than 35 mmHg, and Suk et al reported a prevalence of pulmonary hypertension of 44% in untreated Graves disease patients.^{1,2} The pulmonary hypertension resolved upon achieving an euthyroid state.

Symptomatic thyrocardiac involvement requiring hospital admission however is uncommon, and these three cases of women of Māori ethnicity who have presented in the last 6 years represent at least one-third of patients who have required hospital admission for management of symptomatic thyrocardiac disease at our hospital in the last decade.

The prevalence of overt hyperthyroidism in 2 general practices in Hamilton, New Zealand was found to be 0.2%, with no statistical difference between the Māori and New Zealand European population after adjustment for age and gender. According to 2006 Census data, individuals identifying as being of Māori ethnicity represent only 0.007% of the population in Brisbane.

Our experience raises the question as to whether symptomatic cardiac involvement is more common in women of Māori ethnicity with Graves disease. This would seem more likely to be due to a genetic susceptibility, rather than the severity of thyrotoxicosis due to medication non-adherence.

Our cases did have some family history of thyroid disorders, but no significant family history of thyrocardiac disease, that we could identify. Interestingly, we have also cared for a gentleman of Māori ethnicity who developed thyrotoxic periodic paralysis.

A previous study found a 37-fold over-representation for Polynesians (Māori and Pacific Islander) compared with New Zealand Europeans for thyrotoxic periodic paralysis.⁴ This raises the possibility that individuals of Polynesian ethnicity are at increased risk for both cardiac and skeletal muscle complications of thyrotoxicosis.

It is possible our experience is due to chance. We feel, however, the possible association between Māori ethnicity and cardiac involvement with thyrotoxicosis warrants further examination, particularly given the implications for Māori women of childbearing age, and the adverse outcomes that may be associated with pulmonary hypertension and cardiac disease in pregnancy.

Author information, Parul Nigam, Advanced Trainee Registrar; Adam Morton, Staff Specialist, Department of Endocrinology, Queensland Diabetes Centre, Mater Adult Hospital, South Brisbane, Queensland, Australia

Correspondence: Adam Morton, Department of Endocrinology, Queensland Diabetes Centre, Mater Adult Hospital, Raymond Terrace, South Brisbane, Queensland 4101, Australia. Email: Adam.Morton@mater.org.au

References:

1. Siu CW, Zhang XH, Yung C, et al. Hemodynamic changes in hyperthyroidism-related pulmonary hypertension: a prospective echocardiographic study. *J Clin Endocrinol Metab.* 2007;92: 1736-42.
2. Suk JH, Cho KI, Kim M, et al Prevalence of echocardiographic criteria for the diagnosis of pulmonary hypertension in patients with Graves' disease: before and after antithyroid treatment. *J Endocrinol Invest.* 2011 Mar 7. [Epub ahead of print]
3. Gibbons V, Conaglen JV, Lillis S, et al. Epidemiology of thyroid disease in Hamilton (New Zealand) general practice. *Aust N Z J Public Health.* 2008 ;32:421-3.
4. Elston MS, Orr-Walker BJ, Dissanayake AM, Conaglen JV. Thyrotoxic, hypokalaemic periodic paralysis: Polynesians, an ethnic group at risk. *Intern Med J.* 2007;37:303-7.

Nasopharyngeal fibroepithelial polyp in a New Zealand Māori man

Ravi Jain, Subhaschandra Shetty

Abstract

Adult nasopharyngeal polyps have not previously been described in the literature. We present the case of a 42-year-old New Zealand Māori man who presented with a large, 11cm mobile mass in his nasopharynx. We discuss his history and management, emphasising the need for early assessment and intervention.

Case report

A 42-year-old Māori man presented to a rural New Zealand emergency department after coughing up a globular, mobile, sausage-shaped tissue mass that appeared to be “stuck” at the back of his throat. He held it forward out of his mouth to prevent it slipping backwards in to the oropharynx, which caused anxiety and respiratory distress (Figure 1). He gave a one-day history of recurrent, intermittent choking and gagging lasting for a few seconds at a time with the sensation of a pharyngeal foreign body that he was unable to cough up. A similar episode had occurred one year previously. Since then he had mild pharyngeal irritation and discomfort without dysphagia. He was a heavy cigarette smoker.

Fiberoptic nasopharyngoscopy demonstrated that the lesion was attached to the right nasopharyngeal wall anterior to the opening of the eustachian tube (Figure 2). No other obvious abnormalities were noted and the decision to remove this mass urgently was made. After securely clamping the mass under general anaesthesia, the soft palate was retracted, the lesion was cauterised at its base and completely excised. A formal rigid pharyngoesophagoscopy revealed no further abnormality. The postoperative recovery was uneventful.

Macroscopically the lesion was a sausage-shaped mass appearing to be covered with congested pharyngeal mucosa and measuring 11×3cm. Microscopically it was consistent with a fibroepithelial polyp covered by respiratory epithelium. The stroma comprised elasto-fibrous tissue and inflammatory cells. There was no thickening of the basement membrane or evidence of malignancy (Figure 3).

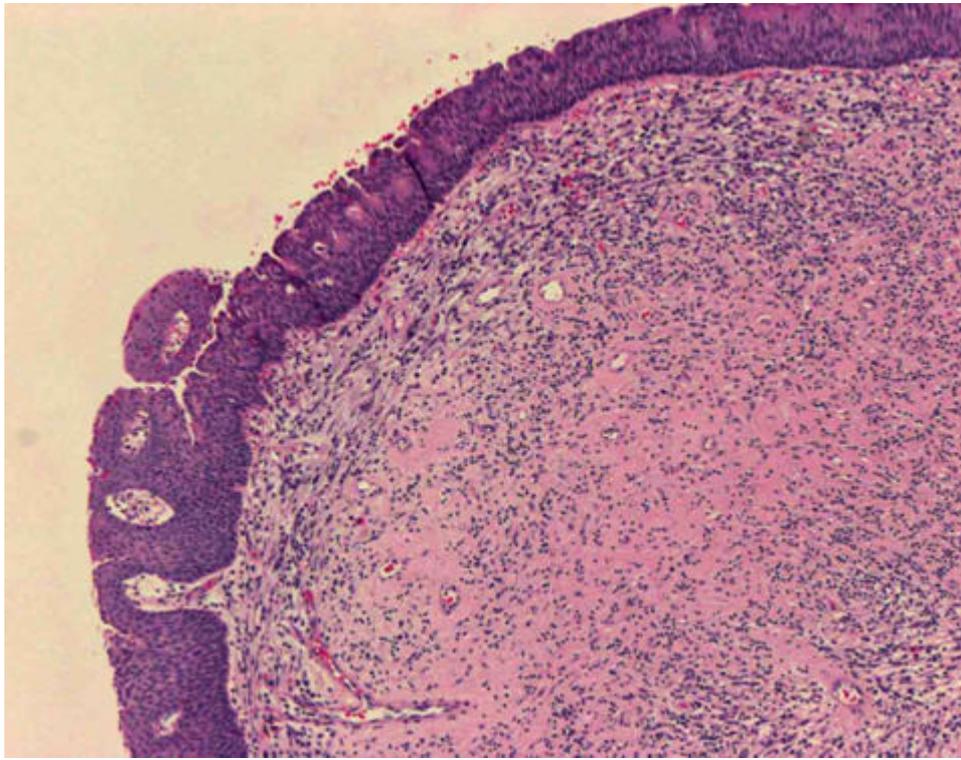
Figure 1. Pre-op securing of polyp with ribbon gauze



Figure 2. Intraoperative, macroscopic appearances of nasopharyngeal polyp



Figure 3. Microscopic appearances consistent with fibroepithelial polyp covered with respiratory epithelium



Discussion

Polyps of the upper digestive tract are classified according to their predominant histological component and include fibroma, fibromyxoma, fibrolipoma, angioliopoma or fibroepithelial polyps. Fibroepithelial polyps of the pharynx and upper airways are rare in medical literature and, to our knowledge they have not previously been described in the adult nasopharynx.

Fibroepithelial polyps are benign polypoid lesions originating from mesodermal tissue and are comprised of varying amounts of loose fibrovascular connective tissue interspersed with fat cells, covered by a squamous epithelium.^{1,2} They are most commonly found in the skin, gastrointestinal, lower respiratory and genitourinary systems and are predominantly seen in males aged 40 to 70, although cases involving women and children have also been described.¹⁻³ Malignant transformation has been reported but is extremely rare.²

The exact aetiology of fibroepithelial polyps is unknown although a few theories have been proposed. One such theory relates to development of polyps following focal loss of elastic tissue.⁴ Another theory is that the polyps are a mixture of various tissue elements that could represent a slowly enlarging haemartoma of the lamina propria.³ In the upper airway, documented lesions have been mostly been reported in the literature as arising from the hypopharynx at Killan's dehiscence between the superior

and inferior cricopharyngeal muscles or at Laimer's triangle between the cricopharyngeus muscle and the proximal end of the oesophagus.^{2,5}

Polyps of the hypopharynx in children and neonates are often discovered as asymptomatic masses by routine examinations. Extremely rare cases exist with polyps arising from the tonsillar region of the oropharynx and nasal turbinates.^{1,5,6} The differential diagnosis for polyps in the nasopharynx should include hairy polyps, which are also rare and usually seen in infants, although cases in adults have been described.⁷

Our case highlights several key points. Despite the high risk of asphyxiation, this patient remained relatively asymptomatic for several years. Oesophageal extension of the polyp was causing some mild intermittent dysphagia. The location of the polyp in the nasopharynx enabled flexible nasopharyngoscopy to visualise the attachment of the polyp, however a barium swallow or CT scan would have delineated between the more common hypopharyngeal polyps.

In this case we felt early management was paramount due to the threat of impending airway compromise. Radiographic studies were not waited for. Multiple cases have been described in recent literature of laryngeal polyps becoming impacted in the airway with subsequent asphyxiation, cerebral anoxia and death.^{2,8} We therefore emphasise the need of early recognition and resection of these polyps for future cases.

Author information: Ravi Jain, Department of Otolaryngology – Head and Neck Surgery, North Shore Hospital, Auckland; Subhaschandra Shetty, Department of Otolaryngology, Head and Neck Surgery, Northland DHB, Whangarei

Correspondence: Dr Subhaschandra Shetty, Department of Otolaryngology, Head and Neck Surgery, Northland DHB, Private Bag 9742, Whangarei, New Zealand.
Email: subhash@nhl.co.nz

References:

1. Peric A, Matkovic-Jozin S, Vukomanovic-Durdevic B. Fibroepithelial polyp arising from the inferior nasal turbinate. *J Postgrad Med.* 2009;55(4):288.
2. Drenth J, Wobbes T, Bonenkamp JJ, Nagengast FM. Recurrent esophageal fibrovascular polyps: case history and review of the literature. *Dig Dis Sci.* 2002 Nov;47(11):2598–2604.
3. Pham AM, Rees CJ, Belafsky PC. Endoscopic removal of a giant fibrovascular polyp of the esophagus. *Ann. Otol. Rhinol. Laryngol.* 2008 Aug.;117(8):587–590.
4. Mangar W, Jiang D, Lloyd RV. Acute presentation of a fibroepithelial pharyngeal polyp. *J Laryngol Otol.* 2004 Sep.;118(9):727–729.
5. Borges A, Bikhazi H, Wensel JP. Giant fibrovascular polyp of the oropharynx. *AJNR Am J Neuroradiol.* 1999 Oct.;20(10):1979–1982.
6. Farboud A, Trinitade A, Harris M, Pflleiderer A. Fibroepithelial polyp of the tonsil: case report of a rare, benign tonsillar lesion. *J. Laryngol Otol.* 2010 Jan.;124(1):111–112.
7. Jarvis SJ, Bull PD. Hairy polyps of the nasopharynx. *J Laryngol Otol.* 2002 Jun.;116(6):467–469.
8. Carrick C, Collins KA, Lee CJ, et al. Sudden death due to asphyxia by esophageal polyp: two case reports and review of asphyxial deaths. *The American Journal of Forensic Medicine and Pathology.* 2005 Sep.;26(3):275–281.

Quadricuspid aortic valve: a rare cause of aortic regurgitation

Jen-Li Looi, Andrew J Kerr

Quadricuspid aortic valve (QAV), a rare congenital anomaly, is an uncommon cause of aortic regurgitation. Echocardiography is the first-line imaging modality in valvular heart disease but cardiovascular magnetic resonance (CMR) is becoming an important tool. We present two cases of aortic regurgitation in patients with quadricuspid aortic valve seen on CMR.

Hurwitz and Roberts¹ described seven morphological types of quadricuspid valve based on the anatomic appearance of the valve.² The first case demonstrates a type A QAV where there are four aortic sinuses and four equally sized cusps. There is incomplete leaflet closure in diastole resulting in AR (Figure 1A).

The leaflets fail to close down normally into the aortic annular plane resulting in a central regurgitant orifice arising above the annular plane at the mid-sinus level. In systole the slightly undersized leaflets fail to fold back normally into each sinus resulting in a “square” systolic orifice (Figure 1B).

The second case illustrates an even more unusual variant of QAV. In this case there are four sinuses and four leaflets but each pair of leaflets is fused to produce a functionally bicuspid valve with the typical “fish-mouth” appearance of a BAV (Figures 2A & 2B).

Figure 1A. Short axis view of QAV in diastole, with a small central coaptation defect (arrow)

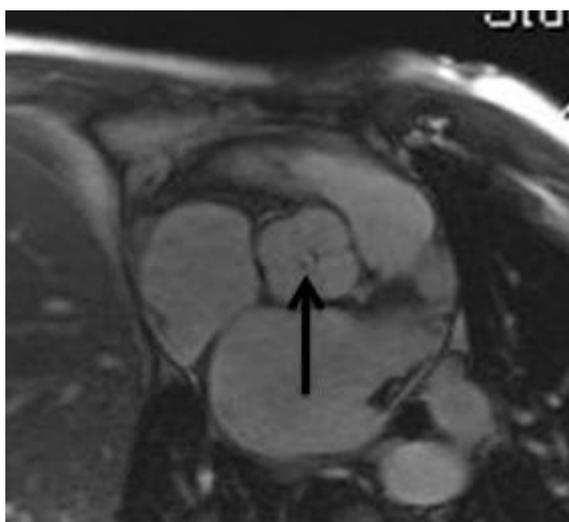


Figure 1B. Short axis view of QAV in systole demonstrating a square systolic orifice (arrow)

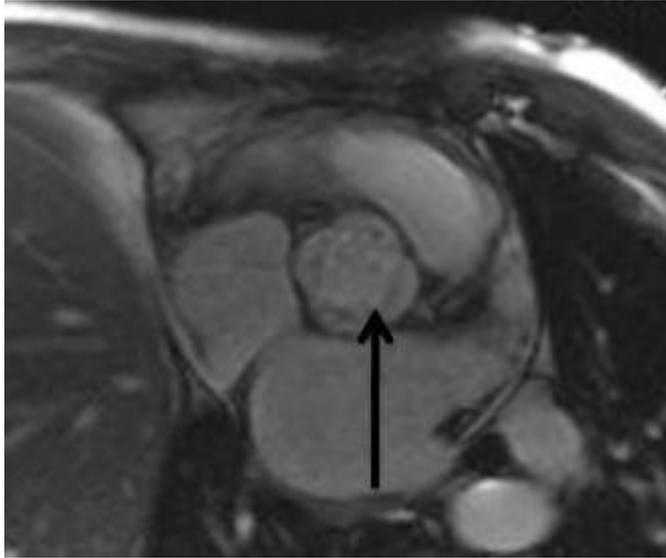


Figure 2A. Short axis view of QAV in diastole, with a small central coaptation defect (arrow)

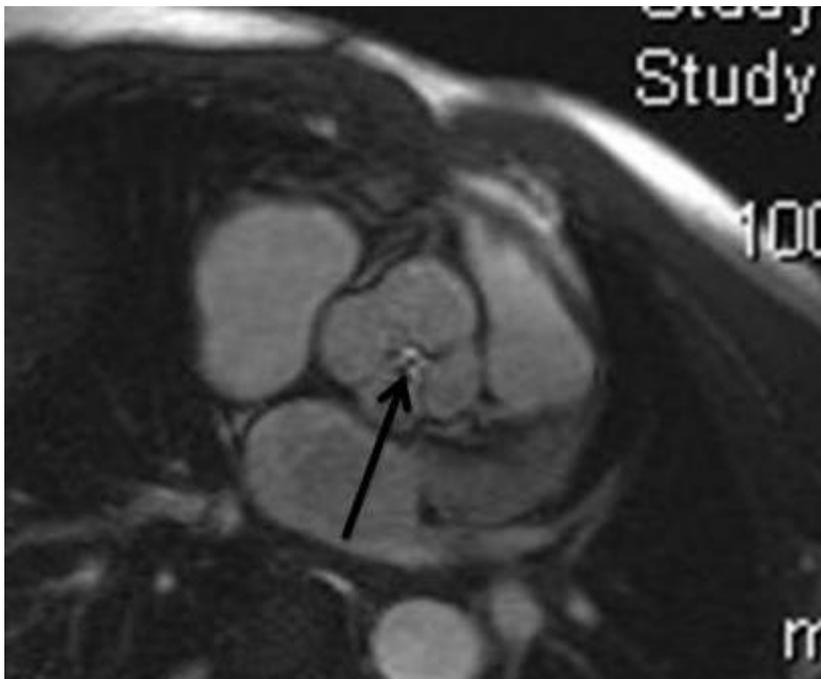
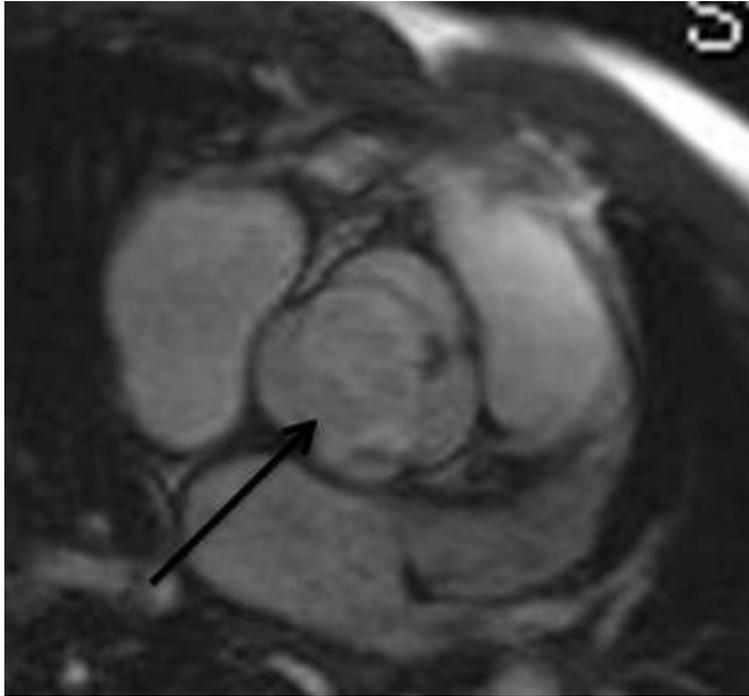


Figure 2B. Short axis view of QAV during systole demonstrating “fish-mouth” appearance (arrow)



Author information: Jen-Li Looi, Registrar in Cardiology; Andrew J Kerr, Cardiologist and Clinical Head of Cardiology; Department of Cardiology, Middlemore Hospital, Otahuhu, Auckland

Correspondence: Dr Jen-Li Looi, Department of Cardiology, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland, New Zealand. Fax: +64 (0)9 2709746; email: ljl517@yahoo.co.nz

References:

1. Hurwitz LE, Roberts WC. Quadricuspid semilunar valve. *Am J Cardiol.* 1973 May;31(5):623-6.
2. Feldman BJ, Khandheria BK, Warnes CA, et al. Incidence, description and functional assessment of isolated quadricuspid aortic valves. *Am J Cardiol.* 1990 April 1;65(13):937-8.

Spontaneous pneumomediastinum without pneumothorax in idiopathic pulmonary fibrosis

Martyn P T Kennedy, Ahmed Fahim, Graham Smith

A 58-year-old man with a background of idiopathic pulmonary fibrosis (IPF) presented with a sudden onset of breathlessness following repeated coughing. He was a life-long non-smoker. On examination, there was evidence of extensive subcutaneous emphysema involving his neck and chest. He was haemodynamically stable with oxygen saturations of 95% breathing room air.

A chest radiograph, obtained at presentation, showed evidence of pneumomediastinum (Figure 1) as well as presence of subcutaneous air in a diffuse pattern.

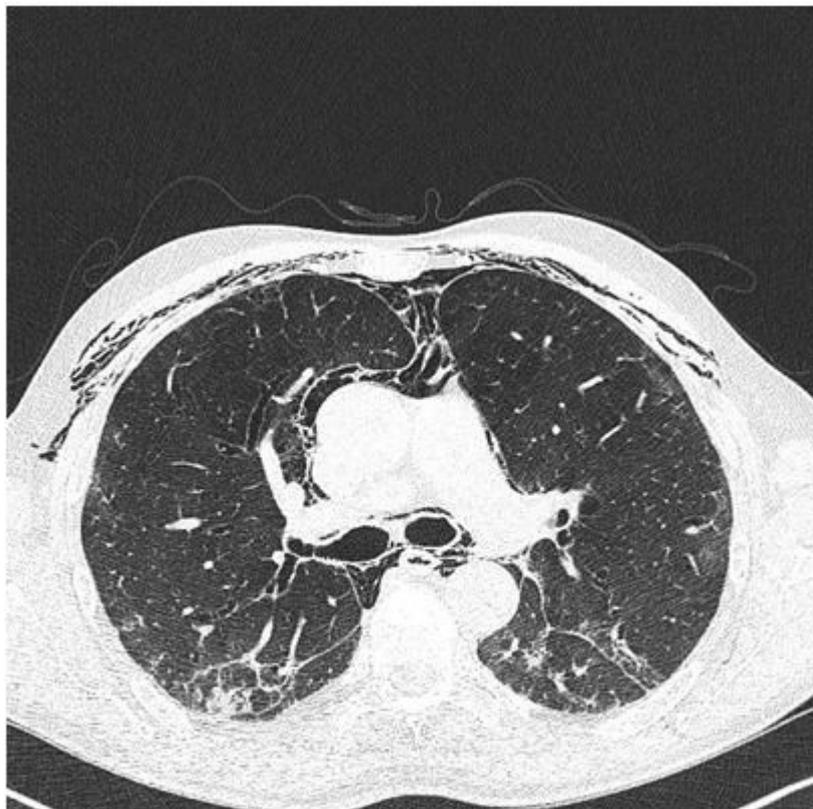
Figure 1. Chest radiograph showing evidence of subcutaneous emphysema and pneumomediastinum



A thoracic computed tomography (CT) scan (Figure 2) was obtained to further characterise the extent of pneumomediastinum and evaluate for co-existing

pneumothorax. The CT scan confirmed the radiographic findings of mediastinal air collection with no evidence of either a pneumothorax or oesophageal rupture.

Figure 2. Images from a CT scan showing subcutaneous emphysema and pneumomediastinum without any co-existing pneumothorax



The patient was treated conservatively with spontaneous improvement in symptoms and significant resolution of pneumomediastinum on follow-up chest radiographs.

This case highlights the importance of considering spontaneous pneumomediastinum (SPM) following repeated attacks of cough in a patient with IPF. Although pneumothorax is commonly associated with SPM, this case suggests that this diagnosis should be considered even in the absence of a pneumothorax. The likely mechanism of the development of SPM is alveolar rupture secondary to increased intrathoracic pressure.^{1,2}

In this particular case, the increased intrathoracic pressure is believed to be the result of extensive cough. CT scan is the diagnostic modality of choice in suspected SPM as it provides more precise information regarding the presence, extent of extra-alveolar air and co-existing pneumothorax.³

Author information: Martyn PT Kennedy, SHO; Ahmed Fahim, SpR; Graham Smith, Consultant; Respiratory Medicine, Pinderfields Hospital, Mid Yorkshire NHS Trust, Wakefield, England

Correspondence: Dr Martyn Kennedy, c/o Dr G Smith, Pinderfields Hospital, Aberford Road, Wakefield WF1 4DG, UK. Email: martynkennedy@doctors.org.uk

References:

1. Macklin CC. Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. *Arch Intern Med.* 1939;64:913-926.
2. Abolnik I, Lossos IS, Breuer R. Spontaneous pneumomediastinum. A report of 25 cases. *Chest.* 1991;100:93-95.
3. Franquet T, Giménez A, Torrubia S, et al. Spontaneous pneumothorax and pneumomediastinum in IPF. *Eur Radiol.* 2000;10:108-13.

BPAC recertification plan

The Medical Council has now raised the educational hurdle for those practising “in a general scope of practice in a collegial relationship”. These doctors must accept the proposed programme at their next annual re-registration and pay \$1200 for the privilege.

The Council has some explaining to do. BPAC is an unknown quantity to many of us. We should have been told, firstly, why the change is necessary—“extensive consultation during 2009 and 2010” may indeed have taken place, but what was the consulting about, and what were the conclusions? What are the qualifications of BPAC in the field of setting educational programmes and who precisely were the perpetrators of this plan? Was any other group asked for an opinion? We perhaps know that BPAC publish “Best Practice”, that they are independent and have five shareholders, but I get the sense there is an ivory tower element about the whole process.

I have a very clear sense of *déjà vu*, going back to the 1980s, when Peter Anyon battled the Council, among other matters, on its role in supervising education. He pointed out that the Council had failed in its duty to keep the profession informed of what they were on about, and I suggest that this new plan is just another example. They would be unwise to ignore the reaction of the profession at that time.

We are generally intelligent people, and given enough information, can make reasoned conclusions. We can't work in a vacuum. Cynically one could suggest that the arrival of their letter was timed to reach us just before the holidays, so that, with luck, the recipients would lose any enthusiasm to contest the proposal.

I would hope not.

Humphrey B Rainey
Upper Hutt

Recertification and the Medical Council

The Medical Council must be stopped before it does any further harm. For a while there have been ominous rumblings, but it was just before Christmas that I got the 3 pages of bumf that spelled out its laborious and expensive intentions. The news for doctors registered in “a general scope of practice” is grim. There will be a Recertification Programme that will cost them, each year, in money, the sum of \$1200.00, and, in time, 50 hours spent away from their families and the places where they carry out their professional work. This programme has been agreed with BPAC, an entity naively described as an independent not-for-profit organisation. Instinct tells me that this cannot be good news.

I had begun to wonder if the GPs understood what was going on, or whether they were, as usual, too weak and disorganised and inattentive (as they were during the theft of the Maternity Services) to do anything about it, when help arrived from a totally unexpected quarter. Naturally, the resistance to the Medical Council does not come from the general practitioners, nor was it culled from the pages of either *Medspeak* or *Vital Signs*.

The *Dominion Post* ran a brief story quoting the reactions of the New Zealand Resident Doctors’ Association to what the Medical Council was thrusting upon us, and the whole of their News Release is on the Association’s website.

Their objections have been issued over the name of Dr Curtis Walker, and in my view that young man is going places. For one thing, he exhibits a concern for the welfare of the taxpayers that I had not supposed his group to be even remotely capable of.

The news release, dated 16th December 2011, can be found at www.nzrda.org.nz. It begins;

“300% hike in house officer fees destined for bureaucratic coffers. More than \$1.2 million is to be taken away from patient care.”

It is all good stuff. I agree with it. The new recertification proposal, it is claimed, is totally unnecessary for doctors working in hospitals under consultant supervision. We can add that it will do nothing for general practice, except drive up the costs.

What, may we ask, is meant by an Essentials Test, described by Dr John Adams, the Chairperson of the Council, as “an interactive online test focusing on both clinical knowledge and also *knowledge of Council’s statements*, [my italics] cultural competence and professionalism (on entry to the profession, and then every three years).”?

This looks to me like Big Brother at work, and encountering no opposition at all from the private practitioners. At the present time, the GPs have no effective voice, and the New Zealand Resident Doctors’ Association can, by contrast, count themselves lucky.

I spoke with a Wellington GP who has the “general” ticket, and who is very distressed by the proposals outlined above. He says the “generalists” are the minority, but I do not suppose that the “vocationally registered” will fare any better. The distinction

between the two groups is meaningless. The Royal New Zealand College of General Practitioners, determined to dominate general practice, occupies a large amount of prime office space in Central Wellington, and the doctors should demand an examination of the financial situation of both that College and the Medical Council. These bodies are in the comfortable position of being able to generate as much rubbish as they wish, and to ask for whatever they want in order to do it.

Roger M Ridley-Smith
Retired GP
Wellington

Auckland District Health Board's new emergency care initiatives

We are pleased to read of Auckland District Health Board's (ADHB's) initiatives to remove the cost barriers to out-of-hours care, the diversion of ambulances to accident and medical (A&M) clinics and funding schemes that pay for some treatments and tests in the community such as ultrasound for deep vein thrombosis (DVT) and intravenous antibiotics for various infections.¹

Coroner Shortland has recently made the following recommendation, which would appear to be supportive of the ADHB's initiatives above.²

Protocol for pre-hospital parenteral antibiotics

- That the Royal New Zealand College of General Practitioners initiate a national working group to develop a protocol for the administration of pre-hospital parenteral antibiotics.
- That the protocol includes the signs and symptoms of suspected bacterial sepsis and indicators for the taking of blood culture samples, in patients without haemorrhagic rash.
- That the Royal Australasian College of Physicians and the national clinical working group for the New Zealand ambulance sector be included amongst those invited to participate in this working group.

The recent article by Morris and Brandaranayake³ entitled "Pre-hospital antibiotics for meningococcal disease remains low" highlights part of the need for such a national integrated approach as recommended by Coroner Shortland. The authors' study notes that while the early use of antibiotics remains a goal of care in the seriously ill patient, and despite the considerable publicity regarding meningococcal deaths, only 23% of patients with signs of meningococcal septicaemia, 26% of patients with signs of meningitis and 37% of patients with signs of both meningococcal septicaemia and meningitis received pre-hospital antibiotics. The authors conclude that "*More focus on primary care attention to early administration of antibiotics on suspicion of meningococcal disease remains a worthwhile recommendation with such a potentially life-threatening illness.*"

Coroner Shortland made further recommendations relevant to Dr Parke's article, including:

Early warning scoring system for assessing physiological instability

- The Royal New Zealand College of General Practitioners develop and propagate an objective for assessing physiological instability, which integrates multiple physiological markers.
- A national clinical working group for the New Zealand ambulance sector develop and promulgate an objective for assessing physiological instability, which integrates multiple physiological markers.

We commend these national integrated Coronial recommendations to the relevant parties as a means of not only potentially improving clinical outcomes for seriously ill patients but also of better coordinating care between the emergency department (ED) and primary care in New Zealand.

Lance Gravatt
Auckland
gravatt@ihug.co.nz

References:

1. Parke T. Diversion of emergency acute workload to primary care: an attractive private sector alternative to public hospital emergency departments? N Z Med J. 16 December 2011;124(1347):6-9. <http://journal.nzma.org.nz/journal/124-1347/5002/content.pdf>
2. Coroner Shortland. Inquest into the death of ZACHARY GRAVATT. 1 November 2011; CSU-2009-AUK-000932.
3. Morris B, Bandaranayake D. Pre-hospital antibiotics for meningococcal disease remains low. N Z Med J. 23 September 2011;124(1343):86. <http://journal.nzma.org.nz/journal/124-1343/4895/content.pdf>

Integration of emergency department and primary care workload

Tim Parke's editorial in the 16 December 2011 issue of the *NZMJ* is a timely one as it articulates the resistance of the Clinical Director of Auckland Hospital Adult Emergency Department (ED) to efforts that seek to provide more appropriate care for primary care type patients who currently visit their service. That this is the view also held by the clinical directors of the EDs of all our major hospital EDs is a matter of concern that needs to be addressed.

With sensible moves to integrate the care of patients, primary and secondary, overseas and in New Zealand the interface between EDs and primary care was always going to receive attention.

The document "Guidance for New Zealand EDs regarding the interface with primary health care" published in June 2011 by the Ministry of Health is a useful summary of a considered way forward.

Tim has argued several points of view as to why it is appropriate for primary care type patients to be seen when they self refer to ED. While I agree that these patients usually are not the cause of delays in ED and in most cases can be seen quickly and easily there, I do not agree that it is clinically appropriate or financially sustainable for this practice to continue unchallenged. The persuasive logic of the patient care integration movement necessitates a close look at how EDs operate and are funded in this country.

I suspect many readers will be surprised to hear that New Zealand EDs are funded \$326 currently for each patient that they see and discharge within 3 hours of first clinician contact. This funding from the Ministry of Health is officially labelled as ED06001 or Emergency Dept Level 6 funding. Within this group are the majority of patients who might also have been seen and treated in Primary Care at a cost of less than one third of this figure.

In Wellington Hospital's ED last year there were 19,531 patients funded under this category. It is useful that Tim, in his editorial, points to the potential loss of funding to EDs if this group of patients are seen in primary care. This is the stumbling block that exists if we are to expect EDs to cooperate with the integration of primary and secondary care at their interface with patients. It is the elephant in the room that has been left unaddressed for too long with the recent growth and expansion of ED services in our hospitals.

By funding EDs \$326 for each primary care appropriate patient seen, we operate a disincentive for sensible moves to integrate health provision. Instead of putting forward other arguments for primary care appropriate patients being seen in their departments, it would be more helpful if ED clinicians advocated widely for change in how the Ministry of Health allocates their funding. They should be appropriately funded for patients who require the secondary level of acute emergency care they are

expert at providing not tied to seeing primary care patients and then having to mount a clinical defence for this way of operating.

We now have much improved hospital front door emergency care for critically ill patients in New Zealand and we should only applaud and support this. The funding for this should be transparent and not involve a perverse incentive for primary care appropriate patients to continue to be seen in our EDs.

Ken Greer
Northland Village Surgery
Wellington

The pharmaceutical industry has a contribution to make to evidence-based healthcare

A letter published in the 14 October 2011 issue of the *NZMJ* entitled *A policy of no pharmaceutical industry sponsorship: a case for health equity* states the opinion “we believe that there are no workable models to balance ethical, education and commercial demands”. This extreme view is unlikely to allow for the best use of the resources and capabilities that each group involved in delivering healthcare has to offer.

We believe that avoiding interaction with the industry that develops some of the health professions most effective (and cost-effective) tools is not tenable. The pharmaceutical industry strives to develop an exceptionally high standard of evidence for the medicines on which doctors rely to achieve health benefits for their patients. There is no other industry that spends as much resource and time on ensuring a product is capable of achieving the claims made about it, with products taking up to 15 years in development to meet international regulatory standards.

Regulators (e.g. Medsafe) and funders (PHARMAC) recognise that the industry is the primary holder of the information essential to the evidence-based use of their medicines. These agencies expect a very high standard of evidence to be developed and presented to them in applications for registration and funding; and rely on the industry to do this.

The industry, in discussion with regulators and other stakeholders has generated substantial self regulatory processes that provide checks and balances to ensure the integrity of evidence development and marketing based on this evidence. Yes, there have been examples of people and companies involved in unacceptable practices, as with any human endeavour, but the broader industry works hard to ensure its’ practices are highly ethical and constantly improved.

The industry strives to provide balanced information to clinicians because the financial sustainability of the industry depends on being recognised as a credible source of prescribing information. There are also built-in incentives for industry self regulation to work, and competitors actively monitor and respond to any marketing that is deemed inappropriate. The Medicines New Zealand Code of Practice (Edition 15 available on our website: <http://www.medicinesnz.co.nz>) has recently been updated to incorporate international moves towards increasingly robust self regulation.

New Zealand Medical Association’s *Consensus Statement on the Role of the Doctor in New Zealand* describes a need for doctors to “advocate for the patient and advise about all treatment options”.² We believe that it is not possible for this to be achieved without being fully informed about the treatment options available. It is also not adequate to rely on PHARMAC to provide information or advice about all treatment options, clinicians must have access to the information that companies are best placed to provide.

We strongly believe that the pharmaceutical industry is a legitimate partner to clinicians and other stakeholders in delivering the optimal healthcare to New Zealand patients.

Disclosure: I am employed by Medicines New Zealand, a membership organisation for the research-based pharmaceutical companies providing medicines to New Zealand.

Kevin Sheehy
General Manager
Medicines New Zealand
Wellington

References:

1. Pinhao Chao P. A policy of no pharmaceutical industry sponsorship: a case for health equity. N Z Med J. 2011 Oct 14;124(1344):115-6. <http://journal.nzma.org.nz/journal/124-1344/4916/content.pdf>
2. New Zealand Medical Association. Consensus statement on the role of the doctor in New Zealand. N Z Med J. 2011 Nov 4;124(1345):117-20. <http://journal.nzma.org.nz/journal/124-1345/4947/content.pdf>

Do New Zealand nurses claim more lumbar spine injuries than the general population? A retrospective study (1995–2009)

In New Zealand, low back pain (LBP) has the highest incidence of all work-related diseases,¹ with the Accident Compensation Corporation (ACC) spending more than \$350 million annually on claims for lumbar spine injuries.²

Determining lumbar injury frequency by occupation is important as it allows for targeted risk analysis to determine causes and develop strategies to prevent injury. It also highlights the injury risks associated with individual vocations. Nursing is recognised as an occupation associated with high lumbar spine injury risk with annual and lifetime injury prevalence reported at 40-50% and 35-80% respectively.³ Such a high annual prevalence for work-related LBP in New Zealand nurses is comparable to nursing populations in other industrialised countries.^{4,5}

A recent survey demonstrated that the annual prevalence of LBP did not differ for occupations of nursing, postal and office workers, and was as high as 57%.⁶ However it remains unclear whether nurses in New Zealand have a higher risk of lumbar injury than other occupations,³ and whether the costs for such injuries differ to other lumbar injury groups. Therefore, we explored the relationship between the number and cost of ACC-registered lumbar spine injury claims in nurses compared to the general population for work-related and non-work-related injuries in New Zealand between 1995 and 2009.

All data covered the period 1995–2009. Injury statistics were retrieved from ACC for all registered lumbar spine injury claims for individuals aged 20 to 69 years.⁷ Population and employment statistics were accessed from Statistics New Zealand.⁸ Data on registered nurses were acquired via the New Zealand Nursing Council.⁹

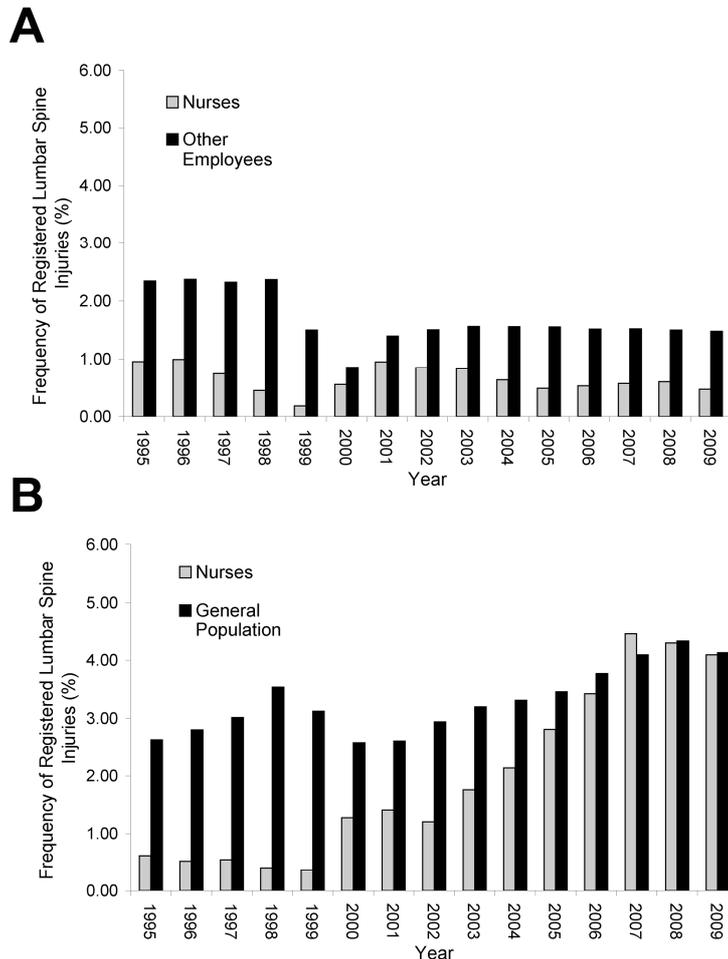
Lumbar injury claim data was standardised for age and population and analysed using STATA 11.0 (StataCorp, Texas) and SPSS 17 (IBM, California) statistical software. Frequency of lumbar spine injury claims was analysed using a Poisson regression analysis, with total costs of lumbar injury claims and annual average cost per registered lumbar injuries analysed using a linear regression model. Statistical significance was set at $p < 0.05$.

Between 1995 and 2009 nurses claimed significantly fewer work-related ACC-registered lumbar injuries (0.7% vs 1.7%) and non-work related (2.0% vs 3.3%) lumbar injuries than the general population (Figure 1). Lumbar injury claims were more likely for the general population (Odds Ratio = 45.06, 95% CI 44.97 to 45.11, $p < 0.0001$) with nurses registering 43% fewer claims than the general population.

Claims for lumbar spine injury for all groups increased by 1.7% annually (significant). For both nurses and the general population, lumbar injuries not related to work were more likely to be registered (Odds Ratio = 1.439, 95% CI 1.437 to 1.446, $p < 0.0001$).

Figure 1. Frequency of ACC-registered lumbar spine injuries in New Zealand nurses and general population aged 20 to 69 from 1995 to 2009

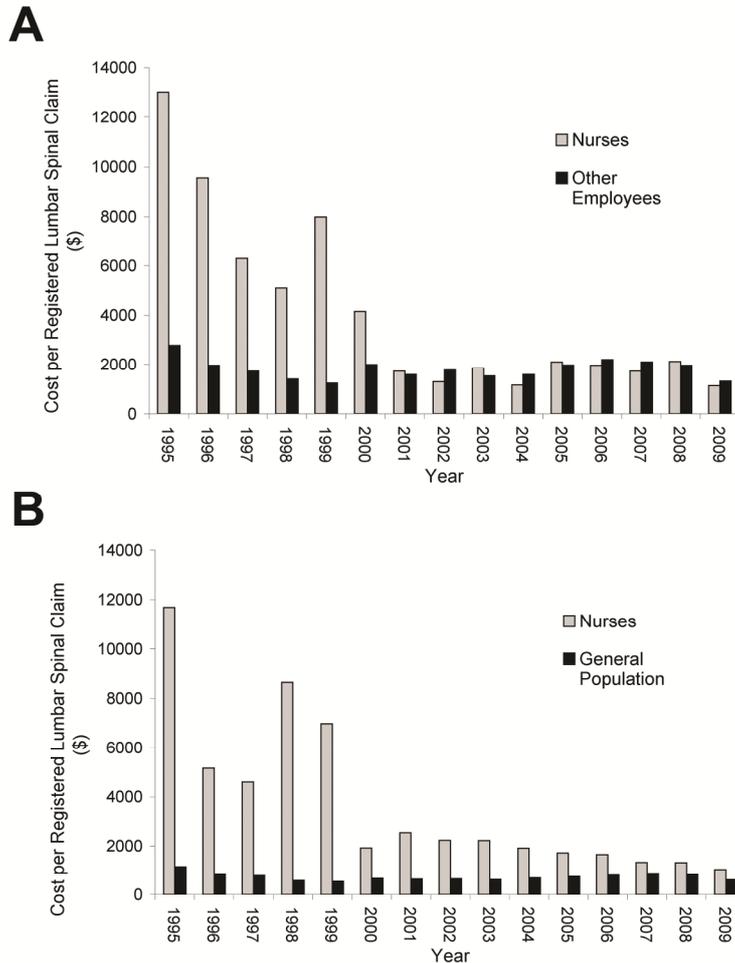
(A: Work-related injuries. B: Non-work-related injuries)



The total cost of ACC-registered lumbar spine injuries was \$2.1 billion, with no significant change in total cost with year. Nurse lumbar injuries represented 2.4% of the total cost (\$49.7 million total, \$3.5 million per annum) and work-related nurse injuries 1% (\$20 million, \$1.4 million per annum).

Costs per nurse's claim were on average three times more expensive than those of the general population ($p < 0.0001$) (Figure 2), with costs per claim decreasing with time ($p < 0.0001$). No significant difference was found for cost per claim in work and non-work related injuries (nurses and non-nurses).

Figure 2. Total costs per ACC-registered lumbar spine injury claim in New Zealand nurses and general population aged 20 to 69 from 1995 to 2009 (A: Work-related injuries. B: Non-work related injuries)



Between 1995 and 2009 nurses registered significantly fewer claims for both work and non-work related lumbar injuries compared to other groups. Interestingly, the frequency of nurse's non-work related injury has increased to closely match non-nurses while the cost of each injury still remains higher. The corresponding work data for the end of this period shows less frequent nurse injury and similar costs to the general working population. Therefore, on average ACC-registered lumbar injuries in nurses were of greater severity and required more costly interventions than those of the general population though the trend shows costs are decreasing for both groups and converging.

New Zealand nurses did not suffer from either work or non-work lumbar injury as frequently as the general population over the study period, indicating that as an occupation nursing has different lumbar injury claim patterns to other groups. However, results should be interpreted with caution. Policy alterations following

changes in government are potentially responsible for data fluctuations, with the influence of lumbar injury claims to private workplace insurance agencies an unknown factor. Nevertheless, the dynamic nature of some data suggests continued surveillance of the figures is warranted to determine the extent of this relationship over time. This would allow the facilitation of adequate prevention strategies and appropriate vocational education for those suffering lumbar spine injuries.¹⁰

Jon Cornwall

Postdoctoral Fellow, Department of Anatomy
University of Otago, Dunedin
jon.cornwall@anatomy.otago.ac.nz

Markus Melloh

Centre for Musculoskeletal Outcomes Research (CMOR)
Dunedin School of Medicine
University of Otago, Dunedin

References:

1. Pearce N, Dryson E, Feyer A, et al. The burden of occupational disease and injury in New Zealand: Report to the associate minister for labour. In: Committee NOHaSA, editor. Wellington: NOHSAC; 2004.
2. ACC. The 'back problem' in New Zealand. In: ACC, editor. Wellington; 2006.
3. Hignett S. Work-related back pain in nurses. *J Adv Nursing*. 1996;23:1238-46.
4. Coggan C, Norton R, Roberts I, Hope V. Prevalence of back pain among nurses. *NZ Med J*. 1994;107:306-8.
5. Leighton D, Reilly T. Epidemiological aspects of back pain: the incidence and prevalence of back pain in nurses compared to the general population. *Occup Med (London)*. 1995;45(5):263-7.
6. Harcombe H, McBride D, Derrett S, Gray A. Prevalence and impact of musculoskeletal disorders in New Zealand nurses, postal workers, and office workers. *Aust NZ J Pub Health*. 2009;33:437-41.
7. The Accident Compensation Corporation of New Zealand. ACC Business Service Centre, PO Box 795, Wellington, New Zealand.
8. Statistics New Zealand. www.stats.co.nz Data retrieved 12 July 2011.
9. The New Zealand Nursing Council. PO Box 9644, Wellington 6141, New Zealand.
10. Pheasant S, Stubbs D. Back pain in nurses: epidemiology and risk assessment. *Appl Ergonomics*. 1992;23(4):226-32.

Quackery with impunity

(But is this law? Ay, marry, is't.—*Hamlet*.)

Published in NZMJ 1911 May;10(38):1.

A lady in the prime of life consulted a Wellington doctor about two years ago and was advised to undergo an operation for the removal of a fibroid tumour about the size of a hen's egg. Neglecting this advice, she took a long course of treatment from a company that agreed to remove the tumour by "rational means without operation." At great cost, she bought their medicaments, and the chief part of the treatment was the use of so-called pastilles, which caused a daily discharge of blood, clot and eschar.

With gladness the poor creature preserved in glass jars these materials, and was repeatedly assured by the company that what she had collected was the tumour coming away. She became ghastly anaemic and faint, and finally could not leave her bed, and then she sent for the doctor who had seen her previously. He found the tumour was the size of a football! The patient died of anaemia and heart failure, or more plainly, her life was vilely cast away through the improper treatment of quacks, who batten on helpless suffering women.

Each of this tribe of infamous charlatans deserves the curse that fell on Shylock—"O be thou damn'd, inexorable dog...for thy desires are wolfish, bloody, starv'd and ravenous."

The Law cannot raise the dead, but can it not protect the living? Oh, that those who have authority would hoist Lord Nelson's favorite battle-signal—"Engage the enemy more closely!" They are but half-hearted in the struggle.

Efficacy of a herpes simplex vaccine?

Both herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2) can cause primary infection of the genital tract, and HSV-1 infection has become an increasingly frequent cause of genital disease. Recurrence of genital disease in people with HSV-2 antibodies happens in some 10–25% and is distressing. Even more distressing is transmission of HSV from infected women to neonates as this may lead to severe neurologic disease or death in the newborn. Current strategies to prevent such include condom usage and antiviral drugs. However, a prophylactic vaccine would be better.

In this trial in 8323 women, a candidate HSV vaccine containing glycoprotein D was found to be ineffective in preventing HSV-2 infection. However it was effective in preventing HSV-1 genital disease. Good but not good enough.

N Engl J Med 2012;366:34-43

Systematic screening for occult cancer in elderly patients with venous thromboembolism

A controversial topic explored in a prospective study from France. Fifty consecutive elderly patients (median age 80 years) with proven deep venous thrombosis and/or pulmonary embolism were screened for occult cancer by clinical, laboratory (including tumour markers) and radiological investigations (abdominal ultrasound, chest X-ray and a thoraco-abdominopelvic computed tomography scan).

The screening strategy did not detect several cancers that became overt clinically over the next 12 months. They conclude that a full history, clinical examination and routine laboratory investigations might be the optimal first-line strategy to detect cancer after the diagnosis of venous thromboembolism in elderly patients, but regular clinical examinations during follow-up are warranted. They do not favour blood tumour markers with the exception of PSA in elderly men.

Int Med J 2011;41:769-75.

Blood pressure targets recommended by guidelines for high-risk patients: <140/90 or <130/80?

Hypertension treatment guidelines recommend that blood pressure (BP) be lowered to <140/90 mmHg, but that a reduction to <130/80 mmHg be adopted in patients at high cardiovascular (CV) risk because of diabetes mellitus, renal disease or prior cardiovascular event. This study involved 12554 such hypertensive patients with a high CV risk. Patients were divided into four groups according to the proportion of in-treatment visits before the occurrence of an event (<25% – >75%) in which BP was reduced to <140/90 or <130/80 mmHg). And their conclusions were that the more frequent achievement of the BP targets recommended by guidelines led to cerebrovascular and renal protection, but did not increase cardiac protection.

Overall, CV protection was favourably affected by the less tight but not by the tighter BP target. In an editorial it is noted that at the present time only a minority of patients with hypertension meet the current recommended targets in either the general hypertensive population or the high risk group. So he recommends that the current targets should not be revised until further evidence is presented.

Circulation 2011;124:1727-36 & 1700-02.

2-cm versus 4-cm surgical excision margins for primary cutaneous melanoma thicker than 2 mm

Apparently the optimal excision margins for such melanomas are controversial. This randomised study involved 936 patients, half of whom had a 2-cm excision margin and the other half a 4-cm margin. After a median follow-up of 6.7 years the authors report no significant difference in overall survival or recurrence-free survival. There was also no difference in the death rate from melanoma in the two treatment groups. The authors also note that those having the wider excision may have worse cosmetic results and may need skin grafts which may lead to complications. Consequently they conclude that their findings suggest that a 2-cm resection margin is sufficient and safe for patients with cutaneous melanoma thicker than 2 mm.

Editorial commentators noted that the trial findings are welcome. However, as the trial was planned as an equivalency trial with 2000 patients but only recruited 936 patients they believe the study should be classed as an unplanned non-inferiority trial, which showed that a 2-cm margin was not inferior to a 4-cm margin.

Lancet 2011;378:1635-42 & 1608-9.

Treatment of recurrent pericarditis with colchicine

Recurrence of pericarditis is common—up to 30% and up to 50% after a first recurrence. Colchicine has been reported to be useful and this randomised placebo controlled trial sets out to elucidate. 120 patients with a first recurrence of pericarditis were randomly assigned to receive either placebo or colchicine, 1.0 to 2.0 mg on the first day followed by a maintenance dose of 0.5 to 1.0 mg/d for 6 months. At 18 months the recurrence rate was 24% in the colchicine group and 55% in the placebo group. These results represent an absolute risk reduction of 0.31 and a relative risk reduction of 0.56 (number needed to treat, 3). In addition colchicine reduced the persistence of symptoms at 72 hours and the mean number of recurrences. Hence, such treatment is recommended. Presumably colchicine treatment effects in pericarditis are analagous to the benefit seen in treating the synovitis of gout. The authors caution that pericarditis of proven bacterial or neoplastic causes were excluded from their trial.

Ann Intern Med 2011;155:409-14.

Medical Volunteering in Namibia

This notice can be viewed at <http://journal.nzma.org.nz/journal/125-1348/5042/content.pdf>

((Libraries, print out the PDF at the link above then replace this page))