THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

Audit of acute exacerbations of chronic obstructive pulmonary disease at Waitemata District Health Board, New Zealand
Cheryl Johnson, Martin J Connolly, Shirley Clover, Laura Campbell, Robyn Goonan, Elizabeth Salmon, Michelle Hopley, Martin Phillips, Jaideep Sood

Clinical audit looking at all admissions to North Shore and Waitakere Hospitals with acute exacerbations of chronic obstructive pulmonary disease during May and October 2010. The audit found areas of good adherence to best-practice guidelines particularly in the use of certain investigations and medication prescribing. Areas of poor adherence found included use of non-invasive ventilation (machine to assist breathing), arterial blood gas measurement (indication of blood oxygenation) and referrals to pulmonary rehabilitation. Audit findings are compared to similar local and international audits and recommendations for improvement in care delivery are also made.

Impact of pneumococcal vaccine on hospital admission with lower respiratory infection in children resident in South Auckland, New Zealand
Alison M Vogel, Adrian A Trenholme, Joanna M Stewart, Emma Best, Charissa McBride, Diana R Lennon

Māori and Pacific children living in poor areas of South Auckland are admitted to hospital with pneumonia and other chest infections much more often than other children. A new vaccine (PCV7) against pneumonia was routinely used from 2008. The admissions to hospital with pneumonia were reduced by about one-third during 2009 to 2011. The reduction in pneumonia admissions was much greater for Pacific infants rather than Māori. Despite this reduction the rate of admission to hospital of Māori and Pacific children living in poor areas of South Auckland to hospital with chest infection remains very high.

Severe impact of the 1918–19 pandemic influenza in a national military force
Jennifer A Summers, G Dennis Shanks, Michael G Baker, Nick Wilson

The article documents the impact of the 1918-19 influenza pandemic amongst the New Zealand Expeditionary Forces (NZEF) of World War One (WW1), through the use of modern epidemiological methods. An estimated 930 NZEF pandemic-related deaths were identified, representing 5.1% of all NZEF WW1 deaths. Mortality rates varied significantly by various host and environmental/military factors. These findings suggest that the total number of New Zealanders killed by this pandemic, New Zealand's largest natural disaster ever, needs to be revised upwards.

Awareness and perceived effectiveness of smoking cessation treatments and services among New Zealand parents resident in highly deprived suburbs
Nathan Cowie, Marewa Glover, Robert Scragg, Chris Bullen, Vili Nosa, Judith McCool, Dudley Gentles

Most of the parents in this study were well aware of the Quitline, nicotine gum and patches, but they were less aware of the wider range of effective treatments that are now available, especially treatments like Champix and Zyban which are only available through a doctor. Few smokers thought treatments and services were likely to work—less than half expected the Quitline would work, and expectations were even lower for other treatments and services. Poor awareness and low expectations of quit smoking treatments and services are a barrier to reducing smoking rates in New Zealand. This study reveals the knowledge gaps about quit smoking treatments among Māori and Pacific parents, an important group with high rates of smoking.

Assessing the value for money of pharmaceuticals in New Zealand—PHARMAC's approach to cost-utility analysis ((viewpoint article))
Rachel Grocott, Scott Metcalfe, Paul Alexander, Rachel Werner

Cost-utility analysis (CUA) is a form of economic analysis that provides information on the cost-effectiveness of health technologies. The article describes the process involved in undertaking CUA at PHARMAC, including how PHARMAC reviews the clinical evidence and then uses this information to estimate quality-adjusted life years (QALYs); what costs are included in a CUA; and how this information is combined to obtain an output that can be used to inform decision-making. The article also describes how PHARMAC uses CUA to prioritise pharmaceuticals for funding in New Zealand.

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Reading between the COPD audits: have current GOLD standards lost their lustre?

Catherina L Chang, Robert J Hancox, Lutz Beckert

A decision can only be as good as the information it is based on. In that regard the audit published in this issue of the *NZMJ*[1] helps us to reflect on our care and to improve outcomes for our patients with exacerbations of chronic obstructive pulmonary disease (COPD).

The Waitemata group can be congratulated for their detailed audit on the management of 155 patients admitted to North Shore and Waitakere Hospitals. The authors took a systematic approach using an international audit proforma and validated 18% of their data through blind re-auditing by another investigator. The authors comment that their audit could be compared to the 2006 audit at Waikato Hospital.[2]

So how do their findings compare to Waikato in 2006 and what are the main lessons learned?

- The increasing numbers of women with COPD is highlighted by both cohorts, which include a much higher proportion of women than is traditionally expected.[3] Other patient characteristics and markers of disease severity such as lung function are comparable between the two cohorts, suggesting similar referral and admission patterns. Unsurprisingly, nearly all were current or ex-smokers, but it is reassuring that the proportion of current smokers decreased from 36% in Waikato to 21% in Waitemata, which is the national average smoking rate.[4]

  This is still a high smoking rate and we must not forget that COPD is an essentially preventable disease and each contact with health care professionals is an opportunity to deliver the smoke-free message.

- It is pleasing to note that the mortality rate appears to be lower. The 30-day mortality of the Waikato cohort was 8% and the overall 12-month mortality was 31%; in contrast, the 30- and 90-day mortality of the Waitemata cohort were 4.1% and 6.7% respectively. While this fall in mortality remains unexplained and may be a chance observation, it is in keeping with trends in the UK where the mortality rate has dropped from 15.5% to 13.9%.[5] However, both the mortality rate and the readmission rate of 70% over 2 years remind us that COPD carries significant morbidity and mortality.[6]

- The authors are concerned that despite good evidence of benefit, none of the patients with hypercapnic respiratory failure received non-invasive ventilation (NIV). Several were treated with continuous positive airway pressure (CPAP), but there is little or no evidence for the use of CPAP in this situation and it is unlikely to be as effective as NIV. This is likely to be due to NIV machines being unavailable. NIV is now the standard of care for patients with hypercapnic respiratory failure.[7]

Compared to many health interventions, NIV machines are not expensive and should be readily available. District Health Boards need to commit to providing a small number of machines for emergency use and train staff to use them.

- Despite similar lung function and comorbidity profiles, patients admitted to Waitamata DHB were less likely to have arterial blood gases performed (33% versus 70% for the Waikato audit). The reason for this is difficult to ascertain—it may reflect different hospital assessment protocols, or perhaps the increasing use of venous blood gases in the emergency department. As the authors point out, diagnoses of respiratory failure may have been missed because an arterial blood gas was not done. Based on a British COPD audit, it is likely that about 25% would have had respiratory acidosis[5] and these patients may have benefited from NIV. Unless arterial blood gases are done whenever this is suspected, these patients will not be recognised.
- Waitemata DHB shares, with most hospitals in New Zealand, the problem of poor oxygen prescribing.[8] Oxygen had only been prescribed for 13% of those receiving oxygen. This is an area of major concern, particularly as some patients were receiving high-flow oxygen. Tasmania data clearly demonstrate that high-flow oxygen during an acute exacerbation of COPD increases mortality.[9]

  Oxygen is a potentially dangerous drug with specific indications. Like any drug it should be prescribed and the administration should be accurately documented.[10]
- The final observation we wish to highlight is the length of the course of oral corticosteroids. The Waitemata audit reported a mean duration of 12 days, while the Waikato audit had a mean duration of just four days. A very recent randomised controlled trial has shown that that a 5-day course of prednisone 40 mg is just as effective as a longer course of 14 days.[11] This provides a welcome opportunity to minimise the iatrogenic harm from steroid use.

The data from Waitemata are important. We should be able to improve patients' survival by ensuring the provision of NIV therapy as outlined in international guidelines. We can improve patients' quality of life by referring to pulmonary rehabilitation, supporting smoking cessation, optimising inhaler therapy, and referring to a dietician where indicated. Importantly we can reduce the harm we do to our patients by avoiding uncontrolled oxygen therapy and shortening the course of systemic steroids to 5 days.

After many years of research we still only have three interventions that have been shown to improve survival in COPD:

- Smoking cessation (that could also prevent most COPD altogether),
- Non-invasive ventilation for hypercapnic respiratory failure, and
- Long-term oxygen therapy for patients who are chronically hypoxic.

While it is important to ensure our patients have access to these treatments, the impact of these on the overall survival is likely to be small.

New directions in therapy of COPD are desperately needed. Given that most patients with COPD die of cardiac disease rather than respiratory failure, looking after the cardiovascular system may be of as much benefit to our patients as our current respiratory drugs.[12] In fact, emerging evidence suggest that both beta-agonist and anticholinergic bronchodilators may contribute to cardiac complications in COPD.[13]

Even in the setting of acute exacerbations of COPD, there is emerging evidence that biomarkers of cardiac disease are better predictors of mortality than existing respiratory indicators of severity.[14] Unfortunately it is not known whether the usual cardioprotective medications, such as beta-blockers, will reduce mortality in this population since many of the cardiac studies have excluded patients with COPD.

In conclusion, the data from Waitamata prompt us to reflect on our clinical management of exacerbations of COPD. The findings are broadly similar to the audit from Waikato in 2006 and it is likely they are representative of the care patients receive in other hospitals in New Zealand.

It is pleasing to see that smoking and mortality rates appear to be decreasing, but there are areas for improvement. By now, all DHBs should offer NIV services for patients with acute hypercapnic respiratory failure. We should do less harm by restricting oxygen therapy to those who are hypoxic and titrating the dose to the oxygen saturations. Recent evidence should provide us with confidence to use short, 5-day courses of steroids.

We can improve quality of life by referring patients to pulmonary rehabilitation courses, which should also be available to all patients with COPD. However all these interventions will not hugely improve the prognosis for most patients with COPD.

Furthermore, we need new treatment strategies and perhaps it is time to shift our focus to more achievable goals such as reducing the cardiac complications of COPD. Unfortunately, like most COPD treatments, evidence that this will improve survival is currently lacking.

Competing interests: Nil.

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References:

1. Johnson C, Connolly MJ, Clover S, et al. Audit of acute exacerbations of chronic obstructive pulmonary disease at Waitemata District Health Board, New Zealand. N Z Med J. 2013;126(1378). http://journal.nzma.org.nz/journal/126-1378/5743
2. Chang CL, Sullivan GD, Karalus NC, et al. Audit of acute admissions of chronic obstructive pulmonary disease: inpatient management and outcome. Intern Med J. 2007; 37: 236-41.
3. Roberts CM, Lowe D, Bucknall CE, et al. Clinical audit indicators of outcome following admission to hospital with acute exacerbation of chronic obstructive pulmonary disease. Thorax 2002;57:137–41.

4. Ministry of Health. Tobacco Use in New Zealand: Key findings from the 2009 New Zealand Tobacco Use Survey. Wellington: Ministry of Health; 2010.
5. Royal College of Physicians of London, British Thoracic Society and British Lung Foundation Report of The National Chronic Obstructive Pulmonary Disease Audit 2008: clinical audit of COPD exacerbations admitted to acute NHS units across the UK November 2008.
6. Patil SP, Krishnan JA, Lechtzin N, Diette GB. In-hospital mortality following acute exacerbations of chronic obstructive pulmonary disease. Arch Intern Med 2003; 163: 1180–6.
7. RCP/BTS guideline. The Use of Non-Invasive Ventilation in the management of patients with chronic obstructive pulmonary disease admitted to hospital with acute type II respiratory failure (with particular reference to Bilevel positive pressure ventilation).October 2008 http://www.brit-thoracic.org.uk/Guidelines/NIPPV-NIV-in-Acute-Respiratory-Failure-Guideline.aspx
8. Boyle M, Wong J Prescribing oxygen therapy. An audit of oxygen prescribing practices on medical wards at North Shore Hospital, Auckland, New Zealand N Z Med J 2006;119(1238). http://journal.nzma.org.nz/journal/119-1238/2080/content.pdf
9. Austin MA, Wills KE, Blizzard L, et al. Effect of high flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting: randomised controlled trial BMJ. 2010 Oct 18;341:c5462.
10. O'Driscoll BR, Howard LS, Davison AG; British Thoracic Society. BTS guideline for emergency oxygen use in adult patients. Thorax. 2008 Oct;63 Suppl 6:vi1-68
11. Leuppi JD, Schuetz P, Bingisser R, et al. Short-term vs conventional glucocorticoid therapy in acute exacerbations of chronic obstructive pulmonary disease: the REDUCE randomized clinical trial [published online May 21, 2013]. JAMA. 2013;309(21):2223-31.
12. Mannino DM, Doherty DE, Sonia BA. Global Initiative on Obstructive Lung Disease (GOLD) classification of lung disease and mortality: findings from the Atherosclerosis Risk in Communities (ARIC) study. Respir Med 2006;100:115–22.
13. Gershon A, Croxford R, Calzavara A, et al. cardiovascular safety of inhaled long-acting bronchodilators in individuals with chronic obstructive pulmonary disease. JAMA Intern Med. 2013 May 20:1-9.
14. Chang C, Robinson S, Mills G, et al. Biochemical Markers of Cardiac Dysfunction Predict Mortality in Acute Exacerbations of COPD Thorax. 2011;66(9):764-8.

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Pneumococcal disease in New Zealand and prevailing inequalities, the tip of the lower respiratory infection iceberg

Helen A Petousis-Harris

Streptococcus pneumoniae (*S. pneumoniae*) is a Gram-positive diplococcus with over 90 serotypes identified by the polysaccharide capsule that encloses the cell and contributes to virulence and the cause of pneumococcal disease. The organism is widely carried asymptomatically in the upper respiratory tract and is a common cause of community-acquired pneumonia (CAP), bacterial meningitis, bacteraemia and otitis media (OM).

Development of capsular polysaccharide vaccines against *S. pneumoniae* began early in the 20th Century with the first vaccines marketed in the 1940s. Poor immunogenicity of the polysaccharide vaccine in children under 2 years of age stimulated the development of conjugate vaccines which, unlike polysaccharide vaccines, induce immunological memory, affect nasopharyngeal carriage and are immunogenic in infants and young children.

Since the introduction of routine pneumococcal conjugate vaccination into infant schedules, significant herd immunity has been demonstrated with reductions in invasive pneumococcal disease (IPD) occurring, in both vaccinated persons and their community contacts. New Zealand (NZ) introduced a 7-valent vaccine (PCV7) against pneumococcal disease in 2008 and in 2011 this was replaced with a 10-valent vaccine (PCV10). There is also a 13-valent vaccine (PCV13) available and funded for persons with high-risk conditions or private purchase.

As observed in other countries who have introduced these vaccines, NZ has experienced a dramatic reduction in IPD caused by the serotypes included in the scheduled vaccine, almost to the point of elimination among the vaccine-eligible age groups.[1] Decreases in pneumococcal pneumonia have been observed internationally[2] and are expected in NZ. Early data has reported some reductions in OM in primary care.[3] Conversely there have been small increases in the rates of IPD caused by non-vaccine serotypes in the over 4 year olds.[1]

In 2012 an important paper on the incidence of serious infectious diseases and inequalities in NZ was published.[4] The study showed an increase in acute hospital admissions for infectious diseases in general between 1989 and 2008 and most significantly for LRI. Hospitalisation for pneumonia and influenza almost doubled during this time period and age standardised hospitalisations for Māori and Pacific increased progressively throughout the 1990s. [4]

A decrease in infectious disease hospitalisations for children under five of all ethnicities occurred from the late 1990s to 2008, although the ratio of Māori and Pacific to European increased.[4] This amounts to a modest improvement overall for the youngest members of the population over the most recent years but a substantial widening of inequalities.

To control vaccine preventable infections, vaccine coverage must be high enough and equitable enough socially and geographically so as to prevent transmission of the infection. The immunisation coverage rates for the infant schedule have been improving over the past few years with over 92% of NZ infants fully immunised by their second birthday.

Along with this overall improvement has been the significant reduction in ethnic and deprivation inequities. In 2009, coverage for Māori children was 73%, Pacific 80%, NZ European 82% and Asian 85%: By the end of 2012, the differences were less apparent with Māori 90%, NZ European at 90%, Pacific 93% and Asian 95%.

The socioeconomic differences have also narrowed with just two percentage points between the highest and lowest quintiles. While larger inequities still exist for on-time vaccinations, there has nevertheless been significant progress made. In terms of geographical variation in vaccine coverage for 2 year olds, there is similarity between District Health Boards (DHBs) (10 percentage points between highest to lowest), but this cannot be said for timely administration of the primary series. For example at 6 months of age there is around 25 percentage points difference between highest and lowest performing DHBs.[5]

The impact of the pneumococcal vaccination programme in NZ is reflected in the incidence of IPD. Reductions in IPD caused by vaccine types have been observed in all ethnic groups and all age groups. This is least profound in Pacific children under 2 years of age. The rate per 100,000 in Māori children under 2 years went from 86.6 in 2009 to 45.2 in 2011. In contrast, Pacific children was not so marked, reducing from 64.0 in 2009 to 56.6 in 2011, despite Pacific children having superior immunisation uptake at age 2 years.[1,5]

The study, in this issue of the *NZMJ*,[6] by Alison Vogel and colleagues investigated the impact of pneumococcal vaccination on hospital admissions for lower respiratory infection in Counties Manukau DHB (CMDHB). The ethnic and socioeconomic disparities for hospital admission are consistent with those observed across a range of childhood infectious diseases in NZ.[7] Unlike the pattern observed for IPD since the introduction of the vaccine, there was a significant decline in admissions for pneumonia among Pacific children under 2 years but not Māori children.

The ethnic disparities are troubling. Clearly LRIs have many causes and it is possible that pneumococcal infection is responsible for a lower proportion of these cases in some groups compared with others. This is unlikely due to any variation in vaccine performance between ethnic groups, as IPD caused by PCV7 serotypes in children under 2 years has effectively ceased nationally and it seems reasonable to assume that these serotypes are no longer a cause of LRI.

Despite improvements it is likely factors such as overcrowding, poor housing and access to primary health care still continue to be barriers to achieving more significant reductions in LRI for Māori and Pacific children in CMDHB.[8] There is robust evidence for the effectiveness of pneumococcal vaccination; however there are other important issues at play, including timeliness of vaccination,[9] which is still a challenge particularly for Māori and Pacific infants.

Further research exploring the role of vaccine exposure could help answer some of these questions. Sadly it appears that the burden of bronchiectasis and pneumonia in

CMDHB and presumably nationally may not be particularly amenable to the use of pneumococcal vaccine and require broader strategies,[10] particularly for Māori and Pacific children. However, improving the low timely uptake of vaccine among these children may help.

Competing interests: Nil.

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References:

1. Lim E, Heffernan H. Invasive pneumococcal disease in New Zealand, 2011. Porirua: Institute of Environmental Science and Research Ltd (ESR), 2012.
2. Fitzwater SP, Chandran A, Santosham M, Johnson HL. The worldwide impact of the seven-valent pneumococcal conjugate vaccine. Pediatric Infectious Disease Journal 2012;31(5):501-08.
3. Gribben B, Salkeld L, Hoare S, Jones H. The incidence of acute otitis media in New Zealand children under five years of age in the primary care setting. Journal of Primary Health Care 2012;4(3):205-12.
4. Baker MG, Barnard LT, Kvalsvig A, et al. Increasing incidence of serious infectious diseases and inequalities in New Zealand: a national epidemiological study. The Lancet 379(9821):1112-19.
5. Ministry of Health. National Immunisation Register - Immunisation coverage to year end 2012. Wellington: Ministry of Health, 2013.
6. Vogel AM, Trenholme AA, Stewart JM, et al. Impact of pneumococcal vaccine on hospital admission with lower respiratory infection in children resident in South Auckland, New Zealand. N Z Med J 2013;126(1378). http://journal.nzma.org.nz/journal/126-1378/5743
7. Craig E, Adams J, Oben G, Reddington A, et al. The health status of children and young people in New Zealand. In: New Zealand Child and Youth Epidemiology Service (NZCYES), editor. Dunedin: University of Otago, 2013.
8. Fancourt N, Turner N, Asher MI, Dowell T. Primary health care funding for children under six years of age in New Zealand: Why is this so hard? Journal of Primary Health Care 2010;2(4):338-42.
9. Petousis-Harris H, Grant CC, Goodyear-Smith F, et al. What contributes to delays? The primary care determinants of immunisation timeliness in New Zealand. Journal of Primary Health Care 2012;4(1):12-20.
10. D'Souza AJ, Turner N, Simmers D, Craig E, Dowell T. Every child to thrive, belong and achieve? Time to reflect and act in New Zealand. N Z Med J 2012;125(1352):71-80. http://journal.nzma.org.nz/journal/125-1352/5129/content.pdf

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A disease deadlier than war

Geoffrey W Rice

It is now a truism among historians that before the 20th Century far more people died in wartime from disease than from combat. Down the centuries the so-called 'camp fevers' of typhus and typhoid had been the big killers of all large armies, along with smallpox, while scurvy had decimated the sailors.[1]

During the Crimean War (1854–6) ten times more British soldiers died from dysentery than from enemy fire. The bacteriological revolution pioneered by Pasteur and Koch revolutionised human understanding of major epidemic diseases and enabled effective prevention of most of them by inoculation. Japan was the first country to apply this new knowledge. Routine inoculation against typhoid, smallpox and tetanus, along with delousing to prevent typhus, reduced Japanese deaths from disease in the Russo-Japanese War of 1904–6 to less than a quarter of deaths from enemy action.[2]

The British Army was much slower to respond, as seen during the Boer War in South Africa (1899–1902). Official figures show that five times as many British soldiers died from disease in this conflict as were killed in action or died from wounds. A deadly outbreak of typhoid after the capture of Bloemfontein in March 1900 prompted sweeping reforms in sanitation and hospital organisation.[3] These lessons were applied by the British Army during the First World War with spectacular success. Regular delousing and proper sanitation reduced the rates of sickness to very low levels and kept the old 'camp fevers' at bay. One New Zealand medical officer, when asked what he would remember most about the war, replied that it would be the smell of chlorine-based disinfectants.[4]

Yet in the last year of this healthiest of wars the world was swept by its worst-ever influenza pandemic, which returned in a third wave in the first year of peace. Australia kept out the severe second wave by maritime quarantine, but suffered from the third wave, so this pandemic is known to Australians as the 1919 flu.[5] New Zealand, by contrast, had a shorter but much sharper outbreak, with over 8500 deaths recorded between late October and early December 1918. Māori deaths accounted for almost a quarter of this total, at a death rate seven times that of the Pākehā (New Zealand European) population.[6]

Military personnel were at greater risk of death in this pandemic because they were in the most susceptible age-group, as young adult males, and they were concentrated in training camps and barracks where infection could spread rapidly. Yet civilian deaths from the flu pandemic far outstripped the military deaths of the First World War.

The latest figures from Wikipedia suggest 10 million military deaths, two-thirds of these from combat, whereas the global death toll from influenza across 1918–20 has been estimated at between 50 and 100 million, or 3 to 5% of the world's population at that time.[7]

New Zealand's military personnel suffered serious losses in the 1918 influenza pandemic, and the article by Jennifer Summers, Dennis Shanks, Michael Baker and Nick Wilson, in this issue of the *NZMJ*,[8] adds a significant new chapter to the story of New Zealand's experience of this pandemic.

They have applied the methodology of modern epidemiology to a much more accurate database of victims than was available at the time when I first wrote *Black November*, including the updated NZEF Roll of Honour and the Cenotaph database at the Auckland Museum.

Back then I could only thumb through the bound volumes of New Zealand military deaths held in the office of the Registrar-General in Lower Hutt, trying to count all deaths from influenza, pneumonia and respiratory causes within the pandemic period of late 1918. I was well aware at the time that some of the deaths attributed simply to 'Sickness' might be flu victims, but I had no way of checking them. Nor did I have the means to pursue New Zealand soldiers who had died overseas.

In the course of research for her recently-conferred PhD thesis, Jennifer Summers had access to individual military files and has been able to confirm specific causes of death. She also extended her search to UK sources, finding yet more New Zealand soldiers who died from influenza in British military hospitals. I am pleased but not surprised that she has found another 258 flu-related military deaths to add to my provisional figures in *Black November*. This finding brings the grand total of New Zealanders who died in the 1918 pandemic to 8831.

This is an important new finding, the result of careful and painstaking archival research. Interpretation of the results has benefitted from the methodology developed by Professor Dennis Shanks in his work on influenza deaths in the Australian armed forces.[9] New recruits, men from rural backgrounds, and Māori were most at risk of dying from the flu in the NZEF in 1918.

These new figures establish that pandemic influenza accounted for 5.1% of all NZEF deaths in the First World War. This is remarkably close to the percentage of flu deaths in the Māori population. Since many Māori deaths were never registered in 1918, my own estimate of Māori mortality, augmented by newspaper reports, erred on the side of caution.

It is quite possible that the actual number of Māori deaths (which will never be known, for simple lack of evidence) would push the grand total of New Zealanders who died in the 1918 flu over the 9000 mark. This remains our worst disease-disaster so far. Understanding what happened in 1918, and how the country responded, holds valuable lessons for dealing with a future pandemic threat.

Competing interests: Nil.

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References:

1. Zinsser H. Rats, Lice and History. New York; 1965.

2. McNeill WH. Plagues and Peoples. New York; 1976:261–2.
3. Pakenham T. The Boer War. London: Weidenfeld & Nicolson; 1979:381–2.
4. Rice GW. Black November: The 1918 Influenza Pandemic in New Zealand. Christchurch: Canterbury University Press; 2005:43.
5. Barry JM. The Great Influenza. New York: Viking; 2004:375–6.
6. Rice G. Black November; 2005.
7. Johnson N, Mueller J. Updating the Accounts: Global Mortality of the 1918-1920 'Spanish' Influenza Pandemic. Bull Hist Med. 2002;76:105–15.
8. Summers JA, Shanks GD, Baker MG, Wilson N. Severe impact of the 1918–19 pandemic influenza in a national military force. N Z Med J. 2013;126(1378). http://journal.nzma.org.nz/journal/126-1378/5742
9. Summers JA, Wilson N, Shanks GD. Mortality Risk Differences between Australian and New Zealand Soldiers during the 1918-19 Influenza Pandemic. Poster presentation at 'Influenza 2012' conference, Oxford, UK, Sept 2012. www.otago.ac.nz/wellington/otago039517.pdf

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Audit of acute exacerbations of chronic obstructive pulmonary disease at Waitemata District Health Board, New Zealand

Cheryl Johnson, Martin J Connolly, Shirley Clover, Laura Campbell, Robyn Goonan, Elizabeth Salmon, Michelle Hopley, Martin Phillips, Jaideep Sood

Abstract

Aim To examine management and outcome of patients admitted to Waitemata District Health Board (WDHB) with acute exacerbations of chronic obstructive pulmonary disease (AECOPD) and determine performance according to evidence-based guidelines.

Methods Retrospective chart review of all patients admitted to WDHB hospitals with primary diagnosis of AECOPD during May and October 2010. 195 admissions (156 patients) were audited.

Results Patients comprised 72 females and 84 males; mean age 73.1 years. 96% were ever-smokers. 10% of patients had BMI <18 kg/m^2 and 40% of these received no dietician input. Spirometry was recorded in 72% within the previous 5 years. Chest X-ray was performed in 96% in the first 24 hours and 33% had arterial blood gas (ABG) performed.

Twenty-three patients (29%) had acute respiratory acidosis. Continuous positive airways pressure (CPAP) was used in 11 but none received non-invasive ventilation (NIV). Systemic corticosteroids and antibiotics were prescribed to 87% and 84% respectively. Ten percent of patients were referred for pulmonary rehabilitation (PR).

Overall 90-day mortality was 6.7% with 3.1% inpatient mortality. Mean length of stay was 5 days. 90-day re-admission rate was 44%.

Conclusion Areas of good adherence to best practice guidelines. Room for improvement in use of NIV, ABG and spirometry measurement, and PR referral.

Chronic obstructive pulmonary disease (COPD) is a common condition characterised by progressive decline in lung function and recurrent exacerbations often requiring hospitalisation.

COPD has been estimated to affect 15% of the New Zealand population over 45 years with prevalence rates two-times higher in Māori populations.[1] However, a significant proportion of patients remain undiagnosed. COPD is associated with considerable morbidity and mortality, accounting for over 10,000 admissions annually to New Zealand hospitals.[2] COPD is the fourth leading cause of death nationally, and third in Māori.[3]

Effective management of exacerbations is associated with reduced hospital stay, improved outcomes and better quality of life.[4] Evidence-based guidelines detailing acute and chronic management of COPD have been published.[5] There are few local studies that review whether COPD management conforms to these guidelines.

Waitemata DHB (WDHB) inpatient facilities at North Shore and Waitakere Hospitals receive approximately 950 emergency admissions for acute exacerbations of COPD (AECOPD) annually. The current study examines whether WDHB patients with AECOPD received assessment/treatment according to evidence-based guidelines,[5] with local and international comparison of results.[4,6,7]

Methods

Retrospective chart review of all patients admitted to WDHB with primary diagnosis of AECOPD (International Classification of Diseases {ICD-10} codes; unspecified chronic bronchitis {J42}, emphysema unspecified {J439}, COPD with acute lower respiratory infection {J440}, COPD with acute exacerbation unspecified {441}, other specified COPD {J448} and COPD unspecified {J449}) was performed for May and October 2010 (including patients with repeated admissions and those admitted to hospital or discharged from the Emergency Department [ED]). These months were chosen for comparison with a similar audit at Waikato District Health Board, New Zealand.[6]

Our proforma reflected the 2008 UK National COPD audit[4] modified for updated guidelines.[5] Before the audit, inter-rater reliability was tested on medical records of five AECOPD admissions.

Patients' charts were reviewed by three investigators. Validity was verified by random selection of 35 admissions (18%), blindly re-audited by another investigator.

Mortality data was obtained from WDHB databases and General Practitioners (GPs). Vaccination status was obtained from GPs. Descriptive statistics expressed as percentages of number of patients audited or number of admissions as appropriate.

The regional Ethics Committee confirmed that formal approval was not required.

Results

202 admissions were identified; 7 were excluded with incorrect admission dates, leaving 91 in May and 104 in October, involving 156 patients. None were lost to follow-up.

Patient characteristics—Table 1 details patient characteristics. The majority of patients were living at home and 43% had domiciliary support services. One patient had such services added between May and October. Over 96% were ever-smokers. Two (1.2%) stopped smoking during the audit.

Over 70% of patients had spirometry within the previous 5 years of which 70% had severe or very severe COPD according to the GOLD criteria.[5,8] Comorbidity was estimated using the Charlson Index[9] (composite score of 19 conditions; higher number indicating more comorbidities).

Median Charlson score was 2 (one patient lacked adequate information to calculate score). 115 patients (73.7%) had height/weight data to calculate body mass index (BMI) with mean BMI of 26.0 (SD±8.2) kg/m^2.

Information was collected on pre-exacerbation exercise tolerance using a performance scale taken from the 2008 UK COPD audit[4] (Table 1) and Medical Research Council (MRC) dyspnoea score,[10,11] as estimated from admission documentation.

Scores could be determined for performance status and MRC score in 53% and 50% of patients respectively. Of these, 53% had a performance status of limited activity or worse and 61% had MRC ≥4 indicating maximum exercise tolerance 100 metres or few minutes on flat ground. Over 78% (123/156) had influenza vaccination during 2010 and 21% (32/156) had pneumococcal vaccination in the previous 5 years.

Table 1. Baseline patient characteristics

Characteristic	Number (%) / (n=156)
Sex	
Males	84 (53.8)
Females	72 (46.2)
Age (SD)	73.1 years (±11.8)
Males	73.5 years (±12.3)
Females	72.5 years (±11.4)
Ethnicity	
New Zealand European	98 (62.8)
Māori	19 (12.3)
Other European	28 (17.9)
Pacific Island	7 (4.5)
Other Ethnicity	3 (1.9)
Not documented	1 (0.6)
Living circumstance	
Private residence - with others	98 (62.8)
Private residence - alone	32 (20.5)
Independent Unit (†)	11 (7.1)
Rest home/Private hospital	8 (5.1)
Not documented	7 (4.5)
Domiciliary support	
With support – alone	28 (19.0)
With support – with others	35 (23.6)
No support – alone	12 (8.1)
No support – with others	65 (43.9)
Not documented	8 (5.4)
Smoking status	
Ex-smoker (*)	118 (75.7)
Current smoker	32 (20.5)
Life-long non-smoker	6 (3.8)
Number of pack-years smoked (SD)	40.4 (±23.8)
Not documented	25.6%
Medical Research Council (MRC) dyspnoea score	
Grade 1	13 (8.3)
Grade 2	6 (3.8)
Grade 3	15 (9.6)
Grade 4	35 (22.4)
Grade 5	19 (12.3)
Not documented	68 (43.6)
Performance scale	
Normal activity	12 (7.7)
Strenuous activity limited	32 (20.5)
Limited activity but self-care	28 (17.9)
Limited self-care	21 (13.5)
Bed/chair bound	0 (0)
Not documented	63 (40.4)
Body mass index	
Underweight < 18	15 (9.6)
Normal 18–25	41 (26.3)
Overweight 25–30	28 (17.9)
Obese >30	31 (19.9)
Unable to calculate	41 (26.3)
Spirometry within 5 years	112 (71.8)
Median post-bronchodilator FEV_1 (range)	0.97 (0.23–3.52)
Mild – $FEV_1 \geq 80\%$ predicted (%)	5 (4.5)
Moderate – FEV_1 50–79% predicted (%)	29 (25.8)
Severe – FEV_1 30–49% predicted (%)	47 (42.0)
Very severe – $FEV_1 < 30\%$ predicted (%)	31 (27.7)

(†) Independent units refer to residences often attached to a residential care facility in which residents maintain a high degree of independence; (*) Patients stopping smoking before admission were considered ex-smokers.

Assessment and management—Of those admitted, 95% (166/175) were admitted under the care of general medical teams run by general physicians with one admitted to the Intensive Care Unit (ICU). Eight patients (5%) were admitted under the care of general medical teams run by respiratory physicians.

Table 2 details inpatient investigations. A chest radiograph (CXR) was obtained from 96% of patients (188/195) within 24 hours of admission. All were reported by radiologists either during the admission or after discharge. It was difficult to ascertain whether the report was acted on by the treating clinician. Arterial blood gas (ABG) was obtained from 33% within 24 hours of admission and from 41% during hospitalisation.

Of those undergoing ABG, 23/79 patients (29.1%) had acute or acute-on-chronic respiratory acidosis with median pH of 7.25 (range 7.06–7.34). Of these, 82% had repeat ABG within 4 hours of the index measurement. Oxygen usage was documented in 12 patients with respiratory acidosis with 50% on >5L/min.

Table 2. Investigations

Investigation	Number (%)
Chest X-ray	188 (96.4)
COPD-related changes	52 (27.7)
Normal	43 (22.8)
Infective/inflammatory changes	29 (15.4)
Congestive heart failure	15 (8.0)
Other (cancer, scarring, atelectasis, plaques)	49 (26.1)
Admission arterial blood gas (range)	65 (33.3)
Median pH	7.38 (7.10-7.48)
Median pCO_2 (kPa)	6.9 (3.9-14.5)
Documented oxygen usage	29 (45%)

Spirometry was performed in 11% of patients (21/195) prior to discharge. There was no reason for the omission in 168 (86%) patients (excluding those that died). Of those, 87/156 (55.8%) had spirometry within the previous 5 years.

Nineteen patients (10%) were reviewed by respiratory physicians and 45 (23%) by Assessment Intervention Respiratory Service (AIRS). Physiotherapist or dietician review was recorded in 88 (45%) and 32 (16%) patients respectively. Nine of the 15 patients with BMI <18 and 3 of 31 with BMI >30 were reviewed by dieticians.

Continuous positive airways pressure (CPAP) therapy was given to 14 patients (7.2%). One patient had CPAP pre-admission for obstructive sleep apnoea. Of 13 receiving CPAP acutely, eight (62%) received this within 3 hours of admission and six (46%) had treatment plans.

Median pH of those receiving CPAP was 7.25 (range 7.06-7.42); 11 had respiratory acidosis (compensated in three) and two had metabolic acidosis. No patients received non-invasive ventilation (NIV). Mortality in those with acute or acute-on-chronic respiratory acidosis receiving CPAP vs. not was 1 patient vs. 3 respectively (p=NS).

Over 80% of patients received systemic corticosteroids and antibiotics. Twenty-six patients (13.3%) did not receive systemic corticosteroids. Median duration of corticosteroid prescription was 12 days with 31% of patients receiving 7–14 day course.[5] Nine were receiving long-term corticosteroids.

Based on sputum characteristics or infective changes on CXR, 103 patients (53%) had clear indication(s) for antibiotics and 70 (36%) did not. There was insufficient information to determine antibiotic appropriateness in 22 patients (11%). Of those without indication(s), 46 (66%) received antibiotics. Four patients with clear indication(s) did not receive antibiotics within 24 hours of admission.

Nicotine replacement therapy (NRT) was prescribed to 19/32 (59.4%) current smokers and 10 recent ex-smokers. Three current smokers declined NRT. Of the 26 patients (13%) for whom oxygen was prescribed within the first 24 hours, 19 (73%) had ABG taken within the same time period. Thirteen (68%) were hypoxic with median pO_2 of 7.2kPa. Eleven (42%) had a repeat ABG.

Of 156 patients, 141 (90%) were on inhaled therapy before admission. Of the 195 admissions, 10 had no inhaled therapy charted (nine discharged from ED). Inhaler technique was reviewed in 41 patients (21%).

Outcomes—Table 3 summarises outcomes. There were six inpatient deaths (3%) – five in May, one in October. Two further deaths occurred within 30 days of the index admission. Overall 90-day mortality rate was 6.7% (data unavailable for one patient).

Cause of death was COPD in seven, unrecorded in three, and other causes in three (lung cancer, hyperkalaemic renal failure and congestive cardiac failure). Of the inpatient deaths, four had acute respiratory acidosis and three did not receive CPAP or NIV. Reviewing COPD mortality predictors,[12–15] three had inadequate data to calculate BMI; five had BMI <18; three had BMI >30.

Mean age of those deceased was 75.9 (SD±7.9) years. Mean number of admissions in the preceding 24 months was 3.2 (SD±3.6). Spirometry was recorded in 10 with median FEV_1 0.89L. One was receiving home oxygen. Median Charlson index was 3. Sample size was inadequate to do further statistical analysis.

Table 3. Outcomes

Outcome	Number (%)
Mortality	
Inpatient	6 (3.1)
30-day	8 (4.1)
90-day	13 (6.7)
Readmissions	
Within 90 days	86 (44.1)
Within 24 months	138 (70.8)
Mean length of stay (range)	5.2 days (1 hour – 38 days)

Mean length of stay (LOS) was 5.2 (median 4.0) days. Thirty-one patients (16%) were discharged with oxygen, including 12 (6%) new prescriptions. Of the 156 patients, 18 (11.5%) had home oxygen before admission with three new prescriptions between

May and October. No patients were accepted onto an early discharge scheme (EDS). Of those discharged, 175 patients (90%) were not referred to pulmonary rehabilitation (PR). Four had recently completed PR and two declined.

Of the 195 admissions, 86 were re-admitted with AECOPD within 90 days. Mean interval between admissions was 32 (SD±22.3) days. Of the 156 patients, 103 had been admitted with AECOPD within the *prior* 24 months with mean interval between admissions of 151 (range 1–713) days. Mean number of admissions per patient within the *prior* 24 months was 2.8 and 3.4 for May and October cohorts respectively.

Of the 35 charts re-audited, 203 inter-observer errors were found (mean 5.8 per patient). Of these, 148 were major errors e.g. number transposition or omission, and 55 minor errors. Overall error rate was 6.9%.

Discussion

This was a comprehensive retrospective audit of AECOPD admissions. There were four main concerns revealed; poor utilisation of non-invasive ventilation (NIV), limited use of ABG and spirometry, and referral to PR.

NICE guidelines recommend use of NIV in patients with persistent hypercapnic ventilatory failure.[5] In our audit, no patients with acute respiratory acidosis received NIV. In the UK audit, 12% received NIV and over half had documented plans. However, even in the UK audit following a regular audit series with the British Thoracic Society, 50% of those qualifying for NIV did not receive it.[4]

Further, 15% of those receiving CPAP did not have acute respiratory acidosis. A trend to inappropriate use of NIV was highlighted in the UK audit where 11% had metabolic acidosis. In that analysis, this subgroup had higher mortality suggesting both inappropriate use in metabolic acidosis and delayed use as a 'treatment-of-last-resort'.[16] In Waikato, 13% of patients received ventilatory support, and nearly 90% had hypercapnic respiratory failure.[6]

At WDHB during the audit, access to NIV was limited to high dependency unit or ICU and CPAP was available in ED only. However, since this audit a ward-based NIV service has been established and NIV is available on a dedicated medical ward under the supervision of respiratory physicians and led by a NIV clinical nurse specialist. ED also plans to replace the use of CPAP with appropriate use of NIV in patients admitted with hypercapnic respiratory failure.

Only 41% of patients had ABG taken during admission (only one third in the first 24 hours). These figures are significantly lower than comparable (UK and Waikato), audits where 70–90% of patients had ABGs.[4,6,7] ABG is recommended in all AECOPD admissions[5] as identification of hypercapnic respiratory failure is imperative.

Of those having ABG, 30% had respiratory acidosis. There was poor documentation of oxygen usage during ABG measurement, making interpretation of results difficult. Only 13% had oxygen *prescribed* within 24 hours of admission and this was monitored by repeated ABG in under 50%. This figure is similar to the 2008 UK audit (16%)[4], implying an unknown number of patients received oxygen potentially outside safe parameters with risk of respiratory depression.

Of those with respiratory acidosis, 50% received high flow oxygen (>5L/min). Whilst high oxygen flow rates may have been appropriate in some of these patients, oxygen prescription should always be titrated to a target oxygen saturation.[17]

Spirometry had been performed in 72% of patients within the previous 5 years. This was higher than UK figures.[4] However, NICE guidelines recommend spirometry at diagnosis and before discharge.[5] It was disappointing that nearly 45% patients without discharge spirometry had not had this investigation performed previously as this may have been their first COPD presentation and hence a "missed opportunity". Reasons may include patient's respiratory status or lack of knowledge of availability.

Only 7% of our patients had PR referral at discharge. NICE guidelines recommend PR be available to all appropriate patients including those recently hospitalised.[5] PR reduces likelihood of further admissions and provides education about warning signs of AECOPD.[18]

Whilst the current audit was focused on AECOPD, referral for PR should be considered integral to discharge planning. Potential reasons for low referral rates may include lack of knowledge of availability or focus being on acute issues. However, WDHB has a multidisciplinary AIRS team (nurse specialists/physiotherapist) providing post-discharge support and PR in patients' homes. During the audit period, the AIRS team saw 85 audit patients as outpatients.

Almost half of all patients were re-admitted within 3 months with mean interval between admissions being only 1 month. The UK audits had lower 90-day re-admission rates (31% and 33%).[4,7] Our higher rate may reflect premature discharge, lack of social support, lack of free primary care (vs. UK), or the fact that most patients had severe or very severe airways obstruction.

Our 90-day and inpatient mortality rates were low at 6.7% and 3.1% respectively. This is encouragingly lower than other reported figures.[4,6,7] A recent study has shown improvement in long-term survival following AECOPD hospitalisation.[19]

Potentially, our mortality may be artificially low due to incomplete data. However, mortality rates were determined through GPs (most likely to complete death certification in the community) and WDHB databases (accounting for inpatient deaths post index admission). Mean LOS was longer than that documented of other medical patients (3.8 days).[20] LOS in AECOPD in other studies has been reported at 3–10 days.[4,6,7,19]

It was disappointing that no patients were accepted into an EDS. At WDHB, an EDS was previously available through AIRS but was discontinued during the audit due to lack of patients meeting standard clinical inclusion criteria.[5] In the UK audit, 18% were accepted onto such schemes.[4]

Such differences are not easily explicable by variations in severity or social connectedness of the groups studied; median FEV_1 and mean age were identical, our patients had worse ABG measurement on admission, but the UK group contained more patients living alone and considerably more with MRC dyspnoea score ≥4. This highlights the difficulties in duplicating care models between different healthcare systems.

Ours was an ageing population, though 90% lived independently and under 50% received support services. However, 31% of patients had a performance status of limited activity or worse, i.e. difficulty leaving the house. There was also a large proportion without performance status documentation. This is concerning as knowledge of performance status is integral to clinical decision-making. Further, we discovered low levels of support for patients with very limited exercise capacity. This has been previously documented and likely relates to poor detection of functional impairment.[21]

There were several areas of patient care strengths. NICE guidelines recommend CXRs in all patients presenting to hospital with AECOPD.[5] In this audit, 96% of patients had CXR within 24 hours of admission. This is very similar to the Waikato audit.[6] Only 23% of CXRs were normal, highlighting their importance in patient care.

Systemic corticosteroids were prescribed to 87% of patients. NICE guidelines recommend that corticosteroids be used in *all* AECOPD patients presenting to hospital. A dose of 30mg of prednisone/prednisolone daily for 7-14 days is recommended.[5] In the current audit, median duration was 12 days, i.e. significant minority received >14 days treatment. Indeed, nine were receiving long-term corticosteroids (for which there are no indications in COPD).[5] In Waikato's audit 83% received corticosteroids, but median duration was 4 days.[6]

Antibiotics were prescribed to most patients. However, NICE guidelines recommend that antibiotics are only given to those with increasing sputum purulence or clinical/radiographical changes suggesting pneumonia.[5] Only 53% of our patients had such indication(s). In four patients antibiotics were indicated but not prescribed within 24 hours. Mean LOS in these patients was under 3 days with no inpatient or 90-day deaths. Thus omission of antibiotics early in admission did not seem to negatively impact outcome (albeit a very small sub-sample).

BMI could be calculated in 74% of patients, which is pleasing given its prognostic significance.[13,14] Less than 10% of patients had a BMI <18 (an independent predictor of mortality in COPD[13,14]). However it is concerning that 40% of these did not receive dietician review, as weight gain may improve mortality.[13] Ideally all WDHB patients should have Malnutrition Universal Screen Tool (MUST) scores[22,23] documented, to determine need for dietician referral.

Reasons for the lack of referral may include reduced recognition of need or focus on acute aspects of care. Dietician review can be considered an outpatient intervention; however, it is imperative that appropriate patients are identified during admission and follow-up arrangements made. In Waikato's audit, only 25% patients saw dieticians.[6]

Influenza vaccination had been given to 78% of patients and pneumococcal vaccination to 21%. In the 2008 UK audit, 85% had had influenza vaccination within the previous year.[24] These figures are suboptimal as NICE guidelines recommend all should be offered annual influenza vaccinations and a pneumococcal vaccination.[5] New Zealand guidelines recommend pneumococcal vaccination in this population (especially those >65 years)[25] however its cost ($60–70 to patients[26]), may explain the lower uptake.

NICE guidelines recommend NRT prescription in all appropriate patients.[5] There were 13 smokers who did not have NRT prescribed despite the Ministry of Health's

current Health Target for 95% of hospitalised smokers to be provided help to quit smoking.[27]

Hospital admission provides opportunity to review patients' inhaler technique which occurred in only 21% of patients. Guidelines recommend regular review of technique by competent healthcare professionals.[5]

Our audit has limitations. There is potential for error from incorrect coding. Coding is dependent on discharge diagnosis which may be inaccurate. This was a retrospective audit, however no patients were excluded or 'lost-to-follow-up'. The selected months may be unrepresentative of the entire year.

Limited data was collected for some areas of management including sputum cultures, antibiotic prescription according to sputum results and whether radiographic changes were acted on by clinicians. Descriptive statistics were used, due to the low numbers in comparator groups making statistical analysis difficult. Auditing of charts by three different investigators introduces error due to interpretation. However we estimated our error rate at 6.9%.

A number of recommendations can be made—improvements in admission/ assessment procedures in particular ABG measurement, use of spirometry and NIV, PR referral, systemic corticosteroid/NRT prescription, judicious antibiotic use, and documentation of performance status. Educational sessions have been held for medical staff to raise awareness of the findings.

Current WDHB guidelines for the management of AECOPD are being updated incorporating information on NIV and appropriate pharmacological management. The introduction of an oxygen and NIV service will streamline access to home oxygen and provide appropriate use of NIV on a dedicated ward with aim for development of a dedicated respiratory ward.

Policies regarding the use of NIV at WDHB have been created which include recommendations for ABG monitoring and oxygen prescription. We intend to repeat the audit process in 2012/2013 and we anticipate improvements in the use of NIV, home oxygen and overall management of AECOPD.

Competing interests: Nil.

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References:

1. Broad J, Jackson R. 2003. Chronic obstructive pulmonary disease and lung cancer in New Zealand [published November 2003; cited December 2012]. Available from: http://www.asthmafoundation.org.nz/research/burden-of-respiratory-disease-2/
2. Ministry of Health. 2009. Publicly funded hospital casemix events 1 July 2006 to 30 June 2007 [published March 2009; cited October 2011]. Available from: http://www.moh.govt.nz/moh.nsf/indexmh
3. Ministry of Health. 2011. Mortality and demographic data 2008 [published August 2011; cited October 2011]. Available from: http://www.moh.govt.nz/moh.nsf/indexmh
4. National COPD Audit 2008 Steering Group. 2008. Report of the National Chronic Obstructive Pulmonary Disease Audit 2008: clinical audit of COPD exacerbations admitted to acute NHS units across the UK [published November 2008; cited October 2011]. Available from: http://www.brit-thoracic.org.uk/delivery-of-respiratory-care/national-copd-resources-and-outcomes-project.aspx
5. NICE clinical guideline. 2010. CG101 - Chronic obstructive pulmonary disease [published June 2010; cited October 2011]. Available from: http://www.nice.org.uk/guidance/CG101
6. Chang CL, Sullivan GD, Karalus NC et al. Audit of acute admissions of chronic obstructive pulmonary disease: inpatient management and outcome. Intern Med J. 2007;37:236-41.
7. Price LC, Lowe D, Hosker HSR et al. UK National COPD audit 2003: impact of hospital resources and organisation of care on patient outcome following admission for acute COPD exacerbation. Thorax. 2006;61:837-42.
8. Global Initiative for Chronic Obstructive Lung Disease. 2011. Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease. [published December 2011; cited December 2012]. Available at: http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html
9. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis. 1987;40:373-83.
10. Fletcher CM, Elmes PC, Fairbairn AS, Wood CH. Significance of respiratory symptoms and the diagnosis of chronic bronchitis in a working population. BMJ. 1959;2:257-66.
11. Stenton C. The MRC breathlessness scale. Occup Med. 2008;58:226-27.
12. Martinez FJ, Foster G, Curtis JL et al. Predictors of mortality in patients with emphysema and severe airflow obstruction. Am J Respir Crit Care Med. 2006;173:1326-34.
13. Schols AM, Slangen J, Volovics L, Wouters EF. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1998;157:1791-97.
14. Gray-Donald K, Gibbons L, Shapiro SH, et al. Nutritional status and mortality in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 1996;153:961-66.
15. Almagro P, Calbo E, Ochoa de Echaguen A, et al. Mortality after hospitalisation for COPD. Chest. 2002;121:1441-8.
16. Roberts CM, Stone RA, Buckingham RJ, et al. Acidosis, non-invasive ventilation and mortality in hospitalised COPD exacerbations. Thorax. 2011;66:43-8.
17. O'Driscoll BR, Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. Thorax. 2008;63Suppl 6:vi1-68.
18. Puhan M, Scharplatz M, Troosters T et al. Pulmonary rehabilitation following exacerbations of chronic obstructive pulmonary disease. Cochrane Database Syst Rev. 2009:CD005305.
19. Almagro P, Salvado M, Garcia-Vidal C, et al. Recent improvement in long-term survival after a COPD hospitalisation. Thorax. 2010;65:298-302.
20. Ministry of Health. 2010. Annual Report for the year ended 30 June 2010 [published October 2010; cited October 2011]. Available from: http://www.health.govt.nz/publications/annual-reports/annual-report-year-ended-30-june-2010

21. Yohannes AM, Roomi J, Connolly MJ. Elderly people at home disabled by chronic obstructive pulmonary disease. Age Ageing. 1998;27:523-5.
22. Stratton RJ, Hackston A, Longmore D et al. Malnutrition in hospital outpatients and inpatients: prevalence, concurrent validity and ease of use of the 'malnutrition universal screening tool' (MUST) for adults. Br J Nutr. 2004;92:799-808.
23. Harris DG, Davies C, Ward H, Haboubi NY. An observational study of screening for malnutrition in elderly people living in sheltered accommodation. J Hum Nutr Diet. 2008;21:3-9.
24. National COPD Audit 2008 Steering Group. 2008. Report 5 of the National Chronic Obstructive Pulmonary Disease Audit 2008: survey of COPD care within UK general practices [published December 2008]. Available from: http://www.brit-thoracic.org.uk/delivery-of-respiratory-care/national-copd-resources-and-outcomes-project.aspx
25. Ministry of Health. 2011. Immunisation Handbook 2011 [published May 2011; cited October 2011]. Available from: http://www.moh.govt.nz/moh.nsf/indexmh/immunisation-handbook-2011
26. MIMS Online. 2011. [cited October 2011]. http://www.mimsonline.co.nz/AbbreviatedInfo.aspx?pcode=5797#CustomInfo
27. Ministry of Health. 2011. Health Targets 2011/12 [published August 2011; cited October 2011]. Available from: http://www.moh.govt.nz/moh.nsf/indexmh/healthtargets-targets

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Impact of pneumococcal vaccine on hospital admission with lower respiratory infection in children resident in South Auckland, New Zealand

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Abstract

Aim To assess the change in admission rates for all Lower Respiratory Infection (LRI) including pneumonia for children resident in Counties Manukau District Health Board (CMDHB) with the introduction of the Pneumococcal Conjugate Vaccine 7 valent (PCV7) in June 2008.

Method National Minimum dataset ICD10 coded LRI admissions to any NZ hospital August 2001–July 2011 for children <2 year resident in CMDHB were analysed using Poisson regression, omitting 1 August 2008 to 31 July 2009, the first-year post vaccine introduction.

Results Pneumonia but not bronchiolitis admissions have been declining since 2001. Pneumonia admissions decreased significantly after PCV7 introduction (incidence risk ratio (IRR) (95% CI) 1.51 (1.08–1.77), additional to the gradual decline since 2001. There was significant decline for Pacific children post PCV7 introduction IRR 1.70(1.39, 2.07) but not for Māori children, IRR 1.05 (0.78–1.40). Māori and Pacific children are at increased risk of admission with LRI compared to European children (relative risk (RR) (95%CI) 4.6 (4.3–5.0) and 5.0(3.7–5.3) respectively) as are those living in Decile 9, 10 compared with those from other deciles, RR 1.43 (1.36–1.50).

Conclusion The introduction of PCV7 is associated with reduced admissions for pneumonia in young children yet there has been less impact for Māori in CMDHB.

New Zealand (NZ) children have very high rates of admission with lower respiratory tract infection (LRI) when compared with other developed countries.[1–7] Lower respiratory illness is the most common cause of admission in children 0–14 years in Counties Manukau District Health Board (CMDHB) after the neonatal period.[6]

CMDHB has a significantly higher burden of respiratory disease than the rest of New Zealand with rates of admission for bronchiolitis almost double those of other regions.[6] Pacific, Māori and disadvantaged children bear a disproportionate burden.[6] Furthermore, Grant et al have also shown that Pacific children admitted to hospital with pneumonia have a greater disease severity.[9]

Admission to hospital with pneumonia under 2 years of age is linked to the later development of chronic lung disease including bronchiectasis in indigenous populations in Australia, Alaska and New Zealand.[10–12]

The population of CMDHB, particularly Māori and Pacific children and young people, have very high rates of bronchiectasis.[6] Any reduction of pneumonia

admissions to hospital is likely to reduce later complications of chronic lung disease for our population.

Pneumococcal disease is a common cause of pneumonia in childhood but is also a leading cause of invasive disease, particularly in infancy. Studies using different techniques and child populations have demonstrated that bacterial infection with *Streptococcus pneumoniae* (*S. pneumoniae*), viral/bacterial co-infection and viral infection are common in children admitted to hospital with community-acquired pneumonia (CAP).[13–15]

Pneumococcal conjugate vaccines including 7 (PCV7), 9 (PCV9), 10 (PCV10) and 13 (PCV13) serotypes have been developed and subjected to randomised double-blind controlled trials.[16–19] Studies of the impact of PCV7/9 vaccines on CXR diagnosed pneumonia in young children shows an efficacy varying between 20 and 65% (per protocol analysis).[16–20]

A time series analysis based on the discharge diagnosis from the US Nationwide Inpatient sample compared admission rates before and after routine PCV7 immunisation in the US and reported a 39% [22–52] reduction in all cause admission rates for pneumonia in children under 2 years of age.[20]

A comprehensive summary of the impact of PCV7 worldwide has recently been published.[20] PCV7 was introduced as part of the routine immunisation schedule in NZ in June 2008, backdated to infants born on or after 1 January 2008.

This analysis was undertaken to provide background for a study of the viral aetiology of admissions to KidzFirst Hospital (CMDHB) in children under 2 years, in the 1 year prior to and the 2 years following PCV7 introduction.

The population in Counties Manukau is estimated to be 500,600 in 2011 (454,700 in 2006). In the birth cohort of 2010 29% of births were Māori, 32% were Pacific, 20% were European, and 17% Asian/Indian. It is estimated that in 2012 60% of children aged 0–3 years in CMDHB live in Decile 9 and 10 regions.[6]

The goal of this analysis was to assess the change in numbers of admissions of children under the age of 2 years hospitalised with pneumonia and other LRI over a 10-year period encompassing the introduction of PCV7 to the national immunisation schedule in June 2008.

Methods

A list of admissions to hospital for children younger than 2 years of age resident in CMDHB with an ICD-10 principal diagnosis code for acute bronchiolitis (J210, J218, J219), pneumonia (J100, 120-2, 129, 13-14, 150, 152, 154, 155, 159, 160, 180, 181, 189, 204, 209, 851, 852, 869), unspecified lower respiratory tract infection (J101,22), whooping cough (A370, 379) and bronchiectasis (J47) listed as the principal diagnosis on the discharge record was obtained from the National Minimum Data Set (NMDS) for the years 1 August 2001–31 July 2011.

The ICD10 coded diagnosis is made by trained medical information staff based on the discharge summary and clinical notes. NMDS includes all admissions of three hours and over. We analysed admissions with length of stay ≥1 day, i.e. excluding those who were admitted and discharged on the same day, but including readmissions and short stay patients if they stayed ≥1 day.

Admissions of CMDHB children to any hospital in NZ were included. A transfer between hospitals was counted as the same admission. Ethnic-specific hospitalisation rates were calculated using the Ministry of Health's level 1 prioritisation algorithm[21] and categorised into three groups: Māori, Pacific and Other.

Socioeconomic deprivation index for each child was estimated using the NZDep2006 index for their residential address at diagnosis.[22] The NZDep2006 combines 9 variables from the 2006 NZ Census. Individual area scores are then ranked and placed on an ordinal scale from 1 to 10, with Decile 10 representing the most deprived 10% of small areas.

In order to look at changes in admissions for LRI after the introduction of the pneumococcal vaccine, hospital admissions during the year immediately following the vaccine introduction (1 August 2008 to 31 July 2009) were omitted.

A Poisson regression was run with age (<1 year and 1–<2 years), ethnicity (Māori, Pacific, other), gender, decile [1–10], year, pre or post vaccine introduction, and diagnosis (pneumonia, bronchiolitis, other LRI) as explanatory variables and the number of admissions in the year as the outcome.

The log of number of births in CMDHB in the appropriate year, ethnicity, decile, and gender category was included as an offset with the births in the year the period began used for children under 1 year of age, and the previous year for the 1 year olds (e.g. for the year from 1 August 2001 to 31 July 2002 the 2001 births were used for under ones and 2000 births for 1 year olds).

Initially the 3 way interaction of diagnosis, year and pre/post was examined to see if there was a difference in the change of slope from pre to post vaccine introduction in the different diagnoses. When this difference could not be demonstrated the two-way interactions were examined.

Data from the National Immunisation Register (NIR) for the periods 1 July 2009 to 30 June 2010 and 1 July 2010 to 30 June 2011 for the CMDHB were accessed for those reaching 6 months of age and those reaching 12 months of age within the time period with analysis by total group, by ethnicity and by deprivation.[23]

Results

Ethnic and socioeconomic disparity—Over the period 1 Aug 2001–31 July 2011 Māori and Pacific children aged <2 years resident in CMDHB, were at increased risk compared with European children of admission with LRI to any NZ hospital, relative risk (RR) (95%CI) 4.6 (4.3–5.0) and 5.0(3.7,5.3) respectively. Also children living in Decile 9 or 10 regions were at increased risk of admission compared to other deciles, RR 1.43 (1.36–1.50).

Time trends—Figures 1 and 2 illustrate admission rates for pneumonia and bronchiolitis of children who are resident in CMDHB, and admitted to any NZ hospital, over the period 1 Aug 2001–31 July 2011.

Figure 1. Pneumonia admissions for CMDHB resident children aged <2 years with stay ≥1 day to any NZ hospital facility

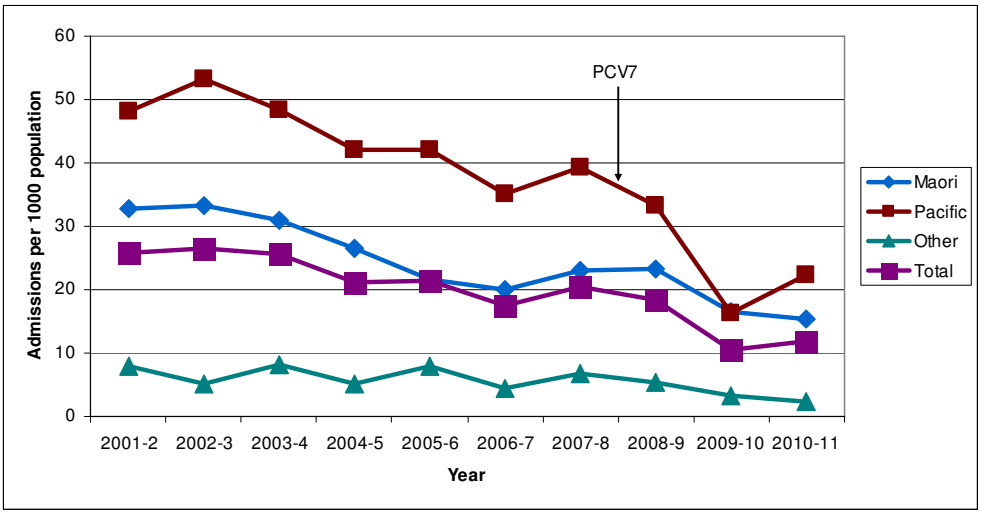
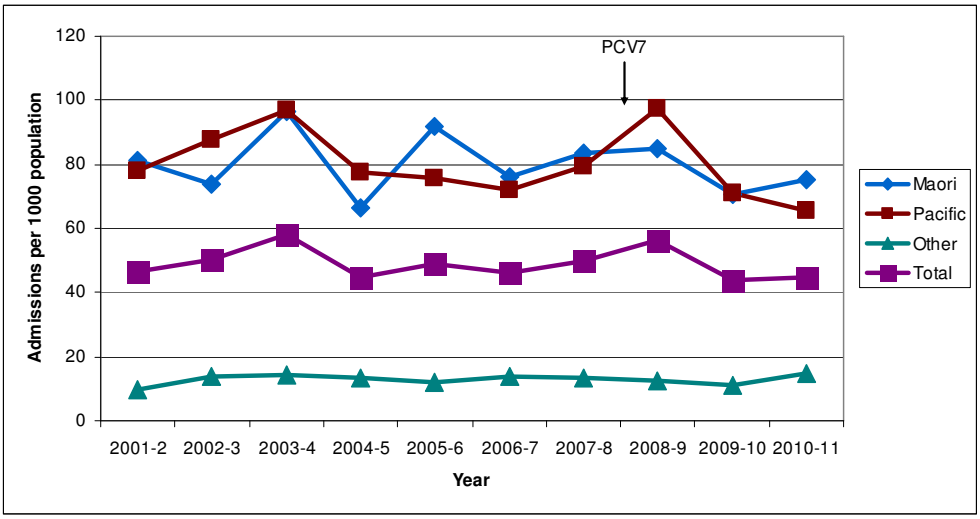


Figure 2. Bronchiolitis admissions for CMDHB resident children aged <2 years with stay ≥1 day to any NZ hospital facility



Poisson regression analysis—Initially the rate of change over time in admissions was modelled to be able to differ pre and post the vaccine introduction and for different diagnoses. However differences in these rates of change over time could not be shown so a constant rate of change pre and post vaccine introduction was assumed within diagnosis category.

In contrast there was strong evidence that the change in rate in admissions over time differed by diagnosis for the three diagnoses ($p<0.0001$). The size of their step in rates after vaccine introduction was also significantly different ($p=0.003$). Therefore the three diagnoses (pneumonia, bronchiolitis and other LRI) were examined separately.

There was strong evidence of a general decrease over time in rates of pneumonia admissions (incidence risk ratio (IRR) (95% CI) for a change of 1 year 0.96 (0.94–0.98), $p<0.0001$) and of an additional step down in rates of pneumonia admissions after vaccine introduction (IRR pre to post 1.51(1.08–1.77) $p<0.0001$) (Figure 1, Table 1).

There was no evidence of the step in pneumonia admissions after vaccine introduction differing by age ($p=0.34$), or decile ($p=0.53$). However there was some evidence of a difference according to ethnicity ($p=0.05$). For Māori, although there was strong evidence of a general decline over the 10-year period in admissions for pneumonia ($p<0.0001$), no step from pre to post vaccination could be demonstrated ($p=0.76$). However a drop from pre to post vaccination could be demonstrated for Pacific ($p<0.0001$) and other ethnicities ($p=0.003$) (see Table 1).

We also investigated whether there was a difference in the change in pneumonia rates from pre to post vaccine introduction in different age groups with age in four 6-month categories, in case the effect was different in those <6 months. However an age difference in the effect could still not be shown, ($p=0.11$).

There was still a significant reduction from pre to post vaccine introduction even in the youngest group, with an effect size very similar to the other age groups (IRR 1.48, 1.53, 1.49 and 1.64, for 0–6 months, 6 months–1 year, 1 year–18 months, 18 to 24 months respectively).

Table 1. Comparison of LRI admissions for CMDHB resident children aged <2 years with stay ≥ 1 day Pre vs Post PCV7 vaccine introduction

Variables	IRR*	95%CI	P
Pneumonia	1.51	1.08–1.77	<0.0001
Pacific	1.70	1.39–2.07	<0.0001
Māori	1.05	0.78–1.40	0.76
Other	1.93	1.26–2.98	0.003
Bronchiolitis	1.07	0.98–1.17	0.15
Other LRI	1.28	0.97–1.77	0.08

A general change over time could not be demonstrated for bronchiolitis (p=0.12, IRR for a change of 1 year 0.99) nor could a change from pre to post vaccine introduction (p=0.15 (Figure 2, Table 1).

For other LRI there was also no evidence of a general decrease over time (p=0.72, IRR 0.99) and only possible weak evidence of a change from pre to post vaccine introduction (p=0.08).

The National Immunisation Register (NIR) has data on immunisation coverage at 6 months and 12 months of age by DHB which is relevant to the age group in this study (see Table 2). By age 6 or 12 months, 3 doses of PCV7 would have been received if all doses given on time. At least 2 doses are needed for effectiveness.[24]

Māori rates are the lowest of all ethnic groups, being about 20% less than those of other ethnicities at 6 months and still at least 10% less at 12 months.

Table 2. Immunisation coverage (%) by age for CMDHB resident children

Variables	July 2009–June 2010		July 2010–June 2011	
Age group	6 months	12 months	6 months	12 months
Total	67	86	66	88
NZ European	75	91	73	90
Māori	53	77	51	79
Pacific	66	89	66	90
Asian	86	94	88	96
Other	75	86	73	89

Discussion

The analysis of hospital admissions for the 10-year period 2001–2011 for CMDHB-resident children has clearly demonstrated a gradual reduction in pneumonia admissions from 2001–2011 with no significant change in admissions for

bronchiolitis. A further step-down in admissions for pneumonia occurred in the 2 years after the introduction of PCV7 to the immunisation schedule in addition to the gradual reduction over the whole decade. This step-down was seen for children of Pacific and other ethnicities, but has not been seen for Māori children. There was no significant step-down effect for bronchiolitis admissions. The magnitude of this reduction in pneumonia is very comparable to international studies.[20]

The gradual reduction in pneumonia admissions in CMDHB, one of the most disadvantaged urban areas in New Zealand with very high Māori and Pacific numbers of children, contrasts with the rise in admissions to hospital for infectious diseases overall as described by Baker up to 2008.[25] However it is consistent with their observed decline in admission rate for the under 5-year group.

Their data for the periods 1999–2003 and 2004–2008 demonstrates a reduction in rate of hospitalisation for infectious diseases (ID) under 5-years of age for European (4608/100,000 to 3856/100,000) and Pacific (8857/100,000 to 8147/100,000). Māori children did not show a change in the rate of ID admission for the same time period and age group, (7971/100,000 to 7918/100,000).

In addition a social gradient was reported in hospitalisations for children age 0–14 years from 2000-2011, including admissions for bronchiolitis and pneumonia, by the Child and Youth Epidemiology service of the Paediatric Society of New Zealand, in the New Zealand Children's Social Health monitor.[26] There was an overall reduction in these admissions for CMDHB from a peak in 2001 contrasting with no change in these admission rates for the rest of New Zealand. Rates for CMDHB Māori and CMDHB Pacific were higher than the non CMDHB Māori and Pacific populations.

A number of factors may have influenced the findings in CMDHB. The Healthy Housing programme is a combined housing improvement, health and social intervention which has been implemented in nearly 6500 social housing (Housing New Zealand) homes in CMDHB between 2000 and 2011. Jackson et al[27] have reported on the outcomes for 3410 of these Healthy Housing interventions with a significant reduction in preventable childhood respiratory hospital admissions using a counterfactual design.

The majority of families served by Healthy Housing were living in areas of high deprivation and were of Pacific ethnicity (personal communication Jude Woolston, manager). Health care may also have improved with a number of DHB primary care strategies[28] aimed at reducing barriers to Primary Care access for children. Immunisation rates have improved and are now at 88% completion at 12 months of age.[23]

Since 1997 there has been an active programme of post discharge follow-up in the community using paediatric home care nurses and this has not changed over this time period.

Immunisation reports demonstrate that Māori have 10% lower completion rates than Pacific at 6 and 12 months.[23] We are unaware of reports specifically comparing barriers to primary care access for Māori compared with Pacific.[29] Delayed or incomplete immunisation would reduce the effectiveness of PCV7 because pneumonia presents from a young age.[6]

Pacific children live in more overcrowded circumstances and more live in Decile 10 areas than Māori[30] both of which increase the likelihood of nasopharyngeal carriage of *S.pneumoniae*[31] and also increase the likelihood of a high nasopharyngeal load which may then link to rates of pneumonia.[32] These hypotheses could also be a link to the increased vaccine effectiveness for Pacific infants.

Our observed reduction in pneumonia admissions for Pacific children brings their rates now to equivalent with Māori children. However both remain much higher than those for European and other ethnicities in New Zealand and populations overseas, although not as high as in aboriginal children.[1–5,9,33–35]

There are some limitations to this study. Hospital discharge data has to be interpreted with caution. Diagnostic shift from pneumonia to another diagnosis such as bronchiolitis or asthma could cause a gradual reduction as seen above however admissions for these entities over the same period have remained the same or reduced. Coding practice has been unchanged in this time period.

The admissions analysed were those with a recorded length of stay ≥1 day which is defined as greater than or equal to 3 hours and present at midnight. This will include all children admitted to the inpatient unit except rare cases admitted after midnight and then discharged before the end of the day. Patients who presented to ED just prior to midnight with a stay >3 hours but not admitted to a ward will have been included.

We have analysed admissions rather than patients as this represents workload for the health services and overall burden for the population. A number of children had multiple admissions. Our 'years' ran from 1 August through to 31 July of the following year. This does mean each year's worth of data includes two different winter seasons with different viral patterns in each winter.

We have used the birth cohort born to mothers resident in CMDHB from NMDS as the denominator population as it reflects the fast growing birth rate and changing demography of CMDHB over this period. Problems have been identified with the allocation of ethnicity in hospital data demonstrating that it under represents Māori ethnicity.[36,37]

The ethnicity data collection protocol was introduced in 2004 and there is evidence that Māori ethnicity was under reported in relation to other ethnicities in the late 90's and early 2000's. There is no assessment of the accuracy of the allocation of ethnicity at birth since the protocol was introduced in 2004. Ethnicity is self identified in the same way as for other admissions.

There was a significant decline in pneumonia hospitalisation in infants <24 months following the introduction of the Pneumoccocal Conjugate Vaccine 7 valent (PCV7) in CMDHB in addition to the gradual decline since 2001. This reduction post vaccination introduction appeared to be less in Māori children.

Ongoing effort to maximise on time vaccination for infants of all ethnicities is essential. Further study to understand the factors underlying both the decline in admission rates over the decade and the difference in the change in rates in Māori and Pacific since the introduction of PCV7 is important.

Despite the improvements since the introduction of PCV7 Māori and Pacific continue to bear an unequal burden of LRI including pneumonia. This requires urgent solutions particularly as pneumonia can lead to long term morbidity and early mortality.[38]

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References:

1. Wickman M, Farahmand BY, Persson PG, Pershagen G. Hospitalization for lower respiratory disease during 20 yrs among under 5 yr old children in Stockholm County: a population based survey. Eur Respir J. 1998;11:366-70. Epub 1998/04/29.
2. Peck AJ, Holman RC, Curns AT, et al. Lower respiratory tract infections among american Indian and Alaska Native children and the general population of U.S. Children. Pediatr Infect Dis J. 2005;24:342-51. Epub 2005/04/09.
3. Lee GE, Lorch SA, Sheffler-Collins S, et al. National hospitalization trends for pediatric pneumonia and associated complications. Pediatrics. 2010;126:204-13. Epub 2010/07/21.
4. MacIntyre CR, McIntyre PB, Cagney M. Community-based estimates of incidence and risk factors for childhood pneumonia in Western Sydney. Epidemiol Infect. 2003;131:1091-6. Epub 2004/02/13.
5. Yorita KL, Holman RC, Steiner CA et al. Severe bronchiolitis and respiratory syncytial virus among young children in Hawaii. Pediatr Infect Dis J. 2007;26:1081-8. Epub 2007/11/29.
6. Craig E, Adams J, Oben G et al on behalf of the NZ Child and Youth Epidemiology Service. The Health Status of Children and Young People in the Northern District Health Boards. NZ Child and Youth Epidemiology Service November 2011. Available from http://dnmeds.otago.ac.nz/departments/womens/paediatrics/research/nzcyes/pdf/Rpt20
7. Grijalva CG, Nuorti JP, Arbogast PG, et al. Decline in pneumonia admissions after routine childhood immunisation with pneumococcal conjugate vaccine in the USA: a time-series analysis. Lancet. 2007;369:1179-86. Epub 2007/04/10.
8. Craig E AP, Jackson C. The Health Status of Children and Young People in Counties Manukau 2008 [cited 2012 April 20]. Available from: http://www.cmdhb.org.nz/About_CMDHB/Planning/Health-Status/Child-Youth/HealthStatus-of-Children-YoungPeople.pdf
9. Grant CC, Pati A, Tan D, et al. Ethnic comparisons of disease severity in children hospitalized with pneumonia in New Zealand. J Paediatr Child Health. 2001;37:32-7. Epub 2001/02/13.
10. Valery PC, Torzillo PJ, Mulholland K,et al. Hospital-based case-control study of bronchiectasis in indigenous children in Central Australia. Pediatr Infect Dis J. 2004;23:902-908.

11. Singleton RJ, Redding GJ, Lewis TC, et al. Sequelae of severe respiratory syncytial virus infection in infancy and early childhood among Alaska Native children. Pediatrics. 2003;112:285-90.
12. Edwards EA, Metcalfe R, Milne DG, Thompson J, Byrnes CA. Retrospective review of children presenting with non cystic fibrosis bronchiectasis: HRCT features and clinical relationships. Pediatr Pulmonol. 2003;36(2):87-93. Epub 2003/07/02.
13. Cevey-Macherel M, Galetto-Lacour A, et al. Etiology of community-acquired pneumonia in hospitalized children based on WHO clinical guidelines. Eur J Pediatr. 2009;168:1429-36. Epub 2009/02/25.
14. Juven T, Mertsola J, Waris M, et al. Etiology of community-acquired pneumonia in 254 hospitalized children. Pediatr Infect Dis J. 2000;19:293-8. Epub 2000/04/27.
15. Michelow IC, Olsen K, Lozano J, et al. Epidemiology and clinical characteristics of community-acquired pneumonia in hospitalized children. Pediatrics. 2004;113:701-7. Epub 2004/04/03.
16. Black S, Shinefield H, Fireman B, et al. Efficacy, safety and immunogenicity of heptavalent pneumococcal conjugate vaccine in children. Northern California Kaiser Permanente Vaccine Study Center Group. Pediatr Infect Dis J. 2000;19:187-95. Epub 2000/04/05.
17. Black SB, Shinefield HR, Ling S, et al. Effectiveness of heptavalent pneumococcal conjugate vaccine in children younger than five years of age for prevention of pneumonia. Pediatr Infect Dis J. 2002;21:810-5. Epub 2002/09/28.
18. Klugman KP, Madhi SA, Huebner RE, et al. A trial of a 9-valent pneumococcal conjugate vaccine in children with and those without HIV infection. N Engl J Med. 2003;349:1341-8. Epub 2003/10/03.
19. Cutts FT, Enwere G, Jaffar S, et al. Efficacy of nine-valent pneumococcal conjugate vaccine against pneumonia and invasive pneumococcal disease in The Gambia: randomised, double-blind, placebo-controlled trial. Lancet. 2005;365:1139-46.
20. Fitzwater SP, Chandran A, Santosham M, Johnson HL. The Worldwide Impact of the Seven-valent Pneumococcal Conjugate Vaccine. Pediatr Infect Dis J. 2012;31:501-8. Epub 2012/02/14.
21. Ministry of Health. 2004. Ethnicity Data protocols for the Health and Disability Sector. Wellington: Ministry of Health.
22. Salmond C, Crampton P, Atkinson J. NZDep2006 Index of Deprivation. Wellington: Department of Public Health, University of Otago, Wellington; 2007:61.
23. MoH. National and DHB immunisation data. Wellington: Ministry of Health; [updated 2011 August 1; cited 2012 April 6]; Available from: http://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/immunisation-coverage/national-and-dhb-immunisation-data
24. Whitney CG, Pilishvili T, Farley MM, Schaffner W, et al. Effectiveness of seven-valent pneumococcal conjugate vaccine against invasive pneumococcal disease: a matched case-control study. Lancet. 2006;368:1495-502.
25. Baker MG, Barnard LT, Kvalsvig A, et al. Increasing incidence of serious infectious diseases and inequalities in New Zealand: a national epidemiological study. Lancet. 2012; 379:1112-9. Epub 2012/02/23.
26. New Zealand Child and Youth Epidemiology Service. The Children's Social Health Monitor New Zealand - Health and Wellbeing Indicators 2009 [cited 2012 March 15]. Available from: http://www.nzchildren.co.nz/hospital_admissions.php
27. Jackson G, Thornley S, Woolston J,et al. Reduced acute hospitalisation with the healthy housing programme. Journal of epidemiology and community health. 2011;65:588-93. Epub 2011/02/02.
28. CMDHB. Primary Health Care Plan 2007-2010 2007 [cited 2012 March 15]. Available from: http://www.cmdhb.org.nz/About_CMDHB/Planning/Primary-Care-Plan/CMDHB-PHCplan-2007-2010.pdf

29. New Zealand Heart Foundation. New Zealand Guidelines for Rheumatic fever. 3. Proposed Rheumatic fever primary prevention programme. May 2009. Accessed at http://www.heartfoundation.org.nz/uploads/Rheumatic%20Fever%20Guideline%203(4).pdf
30. Schluter P, Carter S, Kokaua J. Indices and perception of crowding in Pacific households domicile within Auckland, New Zealand: findings from the Pacific Islands Families Study. N Z Med J. 2007;120:U2393. Epub 2007/02/06.
31. Jacoby P, Carville KS, Hall G, et al. Crowding and other strong predictors of upper respiratory tract carriage of otitis media-related bacteria in Australian Aboriginal and non-Aboriginal children. Pediatr Infect Dis J. 2011;30:480-5.
32. Vu HT, Yoshida LM, Suzuki M, et al. Association between nasopharyngeal load of Streptococcus pneumoniae, viral coinfection, and radiologically confirmed pneumonia in Vietnamese children. Pediatr Infect Dis J. 2011;30:11-8.
33. Grant CC, Scragg R, Tan D, et al. Hospitalization for pneumonia in children in Auckland, New Zealand. J Paediatr Child Health. 1998;34:355-9. Epub 1998/09/04.
34. Moore H, Burgner D, Carville K, et al. Diverging trends for lower respiratory infections in non-Aboriginal and Aboriginal children. J Paediatr Child Health 2007;43:451-7.
35. O'Grady KA, Torzillo PJ, Chang AB. Hospitalisation of Indigenous children in the Northern Territory for lower respiratory illness in the first year of life. Med J Aust. 2010;192:586-90. Epub 2010/05/19.
36. Rumball-Smith J, Sarfati D. Improvement in the accuracy of hospital ethnicity data. N Z Med J. 2011;124:96-7. Epub 2011/09/29.
37. Cormack D, McLeod M. Improving and maintaining quality in ethnicity data collections in the health and disability sector. Te Rōpū Rangahau Hauora A Eru Pōmare: Wellington 2010.
38. Chang AB, Bell SC, Byrnes CA, et al. Chronic suppurative lung disease and bronchiectasis in Australia and New Zealand. Med J Aust. 2010; 193:356-65.

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Severe impact of the 1918–19 pandemic influenza in a national military force

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Abstract

The impact of pandemic influenza on the New Zealand Expeditionary Force (NZEF) in 1918–19 has never been studied using modern epidemiological methods. Therefore we analysed mortality and descriptive data from various sources for these military personnel. An estimated 930 NZEF personnel deaths from pandemic influenza occurred in 1918–19, making it the main cause of disease deaths, and representing 5.1% of all NZEF deaths from World War One (WW1). The epidemic curve was much more drawn out in the Northern Hemisphere compared with the Southern Hemisphere.

Mortality rates varied markedly by setting (e.g. in military camps, by country and by hemisphere). Significantly higher mortality rates were found amongst NZEF personnel: aged 30–34 years, those of Māori ethnicity, those with a rural background, and those who left New Zealand for Europe in 1918.

In conclusion, this work documents the heavy mortality burden from pandemic influenza amongst this national military force and highlights the large variations in mortality rates through host and environmental factors.

The 1918–19 influenza pandemic occurred during the final stages of World War One (WW1). It occurred in a series of waves (of varying severity) and claimed the lives of an estimated 50+ million worldwide.[1] The earliest outbreaks thought to be due to pandemic influenza occurred in March 1918 at two military training camps in the United States.[2, 3] However, reports of the influenza pandemic and its high mortality did not reach New Zealand (NZ) officials until September 1918,[4] by which time the first deaths of New Zealanders from this pandemic had already occurred.

Some of these first deaths occurred amongst the NZ Expeditionary Force (NZEF) onboard the troopship the His Majesty's New Zealand Troopship (HMNZT) *Tahiti* in September 1918.[5] The impact of this pandemic has been described in other military groups around the world,[2,3,6–9] however the impact on NZEF personnel (Figure 1) has only be incompletely described to date.[10-15]

Figure 1. Daily Mail (London): ANZACS in France. Off to the trenches (circa 1916) Alexander Turnbull Library. Official war pictures, no. 153. [Postcard. ca 1916]. Reference Number: Eph-POSTCARD-WWI-01. 2012 [cited January 2013]; Available from: http://timeframes.natlib.govt.nz/

Military camps in NZ were hit hard by influenza in late 1918. Narrow Neck Camp in Auckland was the one of the first populations in NZ to experience an influenza outbreak; an October wave affected 30–40% of the camp (with no deaths), and the November wave affected ~50%.[10,12,15]

In the Featherston Camp (Wairarapa), over 3,220 troops required hospitalisation during the second wave period (November 1918), from a camp comprised of around 8000 men (40.3%).[13] The camp's hospital was quickly overwhelmed with the influenza outbreak.

Awapuni and Trentham Camps both experienced the pandemic's November wave and like other institutions with confined populations in NZ, such as mental hospitals and prisons, reported high morbidity and mortality rates.[10]

Around 600 NZEF personnel located overseas and in NZ were reported as dying from pandemic influenza in 1918.[10,12] However, given the limited detail on the epidemiology of the pandemic we thought this figure was likely to be an underestimate.

Consequently, this study aimed to provide a more detailed description of the impact (at least in terms of mortality) of the pandemic amongst the NZEF personnel.

Methods

Historical context—Historical information was obtained from a variety of documentary sources, describing particular aspects of the influenza pandemic amongst the NZEF personnel in various locations and times.[5,11,16] One of the authors (JS) also conducted interviews with various military historians.

Archival records (known as the Chronicles[17]) containing hospital pandemic morbidity information on NZEF personnel in the United Kingdom was accessed (as part of a PhD thesis[18]). However the lack of clearly defined admission totals meant that it was not possible to extrapolate case fatality estimates without consulting the personnel file for each hospitalised individual, which was beyond the scope of this study.

Presently, there are no other known archival records from this period that contain individualised morbidity information for this military population. Therefore, this current study focuses on mortality from the pandemic.

Denominator data—Individualised data (Cenotaph dataset) on the vast majority of NZEF personnel were obtained from the Auckland War Memorial Museum. These data were used as the main source for variable denominator populations after adjustment for pre-pandemic NZEF personnel deaths.

Additional denominator information was obtained from various sources: New Zealand military camp size numbers;[10, 13] Monthly totals for the NZEF personnel numbers during WW1;[16] and estimates of the numbers of NZEF personnel stationed overseas during various time periods.[11,19]

Mortality data—An electronic dataset (Roll of Honour [RoH]) covering all deaths amongst NZ military personnel during WW1 was obtained courtesy of the compiler, Professor Peter Dennis (University of New South Wales at the Australian Defence Force Academy). The total number of NZEF personnel deaths during WW1 is estimated to be 18,000+.[19]

Individual archival records were then used to confirm an individual as a pandemic influenza case. Initial investigations identified 1113 potential influenza cases, based on NZEF personnel dying from disease or unclear causes in 1918 and 1919. For the purposes of this analysis, pre-pandemic or 'herald' waves (prior to 1918) were not explored further.

The individual military files for each of these NZEF personnel were accessed to confirm the specific cause of death. The military files were from the following sources: digitised PDF personnel files held at Trentham Military Camp NZ (permission obtained in January 2011 from the NZ Defence force), online PDF military files freely available online through the Archives New Zealand website (www.archway.archives.govt.nz), NZ Death Registers held at the NZ Department of Internal Affairs: Births, Deaths and Marriages (permission obtained in December 2011), online death notices freely available online through the NZ National Library Papers Past website (www.paperspast.natlib.govt.nz/cgi-bin/paperspast), and the publicly available Casualty Rolls held in Archives New Zealand.

Cases of pandemic influenza were defined as NZEF personnel whose specified cause of death was one of the following: influenza, pneumonia and/or bronchitis in the time considered to be the pandemic period as described in results.

Deaths occurring at sea (aside from those identified onboard the HMNZT *Tahiti*) were excluded as the numbers were negligible and were not part of any documented influenza outbreak.

Demographic and military data—The variables of place of death, first deployment year, and military rank were obtained from the RoH and Cenotaph datasets. Not all records held information on the variables of interest, and it was assumed for this analysis that there was an equal distribution of missing data amongst the individual NZEF records.

Military rank was divided into four categories based on a key military text.[20] These categories consist of officers, non-commissioned officers, health-care workers and others (mainly privates).

Few records within the Cenotaph dataset (n=1,186) contained age information. Therefore, a 1% simple random sample (adjusted for those still alive during the pandemic) was used as a basis for estimating the age at enlistment for the entire NZEF.

Given the difficulty in obtaining date-of-birth (DoB) information, a larger number of randomly selected records (n=1521) were researched using the three above methods, to obtain a sample size of 1000.

Age at time of enlistment was derived from DoB records. These records were obtained from a number of sources: the Cenotaph dataset, Casualty Rolls, and an online searchable dataset which was used in a previous study[5] for births, deaths and marriages in NZ.[21]

All NZEF personnel identified as pandemic deaths were assigned to one of the following three ethnic groups: European/Other, Māori or Pacific peoples.

The classification of Māori is described in a previous study.[14] For Pacific peoples this classification was defined as having come from a Pacific Island country and having: (i) a Pacific name; or (ii) having a parent with a Pacific name; or (iii) coming from a village (because some Europeans were living in the major towns in some Pacific Islands at this time). All other personnel were categorised as 'European/Other'.

As the Cenotaph dataset does not identify ethnicity for each individual, a 1% sample of the Cenotaph dataset was used to estimate the ethnic distribution of NZEF personnel as a whole. The sample was selected using a simple random sampling method, after restriction to NZEF personnel who were still alive at the start of the pandemic period.

All listed pre-enlistment occupations were classified and given a measure of occupational class (based on occupations in the 1919 year)[22] and a rurality index was used (based on pre-enlistment address and pre-enlistment occupation), as described in previous studies.[5,23]

As only a small number of women were in the NZEF (n=536, mainly nurses, and with n=3 deaths from pandemic influenza) we did not conduct further analyses by gender.

Analysis—All statistical work was performed using MS Excel 2007, EpiInfo, and SAS version 9.1 software.

Results

Time and place of outbreaks—From 27 August 1918 to March 1919, a distinct increase in the number of non-combat deaths was observed amongst NZEF personnel in the Northern Hemisphere.

These deaths can be described as consisting of two waves; a late 1918 wave (27 August 1918 to 31 December 1918) and an early 1919 wave (1 January 1919 to 31 March 1919). Outside of this period there is little evidence for an influenza outbreak causing any mortality. This fits with historical records,[3] which reported little mortality from a first pandemic wave amongst the NZEF in France. From April 1919 onwards, the total number of personnel recorded as dying from non-combat causes returned to pre-pandemic levels.

NZEF personnel in the Southern Hemisphere experienced a distinct increase in the number of non-combat deaths occurring between 1 November to the end of December 1918. The timing of these deaths and the available specific disease records strongly support the evidence for a November pandemic outbreak amongst the NZEF in the Southern Hemisphere, mainly occurring in NZ.

From January 1919 onwards, the numbers of non-combat deaths returns to pre-pandemic levels, providing little evidence of a subsequent pandemic wave amongst this population.

Estimated total of cases—The final estimated pandemic influenza mortality burden amongst the NZEF was 930 deaths. Using the two estimated denominator values of the total number of NZEF personnel, adjusted to those alive during the pandemic gives a mortality rate of 8.8 per 1000 (Table 1).

Table 1. Sociodemographic and military characteristics of New Zealand troops dying of pandemic influenza (1918–1919)

Variable (denominator populations)	Pandemic-related deaths (N)*	Mortality rate (per 1000 population)	Crude mortality rate ratios (95% CI)
Setting and time			
Second Wave, Northern Hemisphere (n=58,399)	255	4.4	1.00 Reference
Third Wave, Northern Hemisphere (n=48,954)	89	1.8	0.4 (0.3–0.5)
Southern Hemisphere/Transit (n=51,844)	519	10.0	2.3 (2.0–2.7)
At sea	67	60	–
Total for all NZEF personnel using total estimated NZEF personnel alive during pandemic period (n=105,520)	930	8.8	–
Age group (years)			
Under 20 (n=2321)	5	2.2	1.0 Reference
20–24 (n=36,193)	258	7.1	1.4 (1.4–8.0)
25–29 (n=30,706)	288	9.4	4.4 (1.8–10.5)
30–34 (n=17,094)	195	11.4	5.3 (2.2–12.9)
35–39 (n=10,130)	112	11.1	5.1 (2.1–12.6)
40–44 (n=5909)	48	8.1	3.8 (1.5–9.5)
45+ (n=2958)	21	7.1	3.1 (1.2–8.2)
Ethnicity			
European/Other (n=103,199)	887	8.6	1.0 Reference
Māori (n=1,583)	32	20.2	2.4 (1.7–3.3)
Pacific (n=739)	11	14.9	1.7 (1.0–3.1)*
Occupational class (pre-enlistment)			
1–3 (highest most privileged) (n=4757)	38	8.0	1.0 Reference
4–6 (n=38,028)	289	7.6	1.0 (0.7–1.3)
7–9 (n=62,735)	439	7.0	0.9 (0.6–1.2)
Rurality index (base on location and occupation)			
0 (highly urban) (n=46,218)	228	4.9	1.0 Reference
1–2 (n=28,174)	210	7.5	1.5 (1.3–1.8)
3–4 (n=15,723)	137	8.7	1.8 (1.4–2.2)
5–6 (n=9,391)	90	9.6	1.9 (1.5–2.5)
7–8 (highly rural) (n=6015)	64	10.6	2.2 (1.6–2.8) †
Military rank			
Privates / Other (n=83,995)	728	8.7	1.0 Reference
Non-commissioned officers (NCOs) (n=16,881)	143	8.5	1.0 (0.8–1.2)
Officers (n=4049)	37	9.1	1.1 (0.8–1.5)
Health care workers (n=595)	3	5.0	0.6 (0.2–1.8)
Deployment year in the NZEF			
1914/1915 (n=32,036)	150	4.7	1.0 Reference
1916 (n=32,094)	181	5.6	1.2 (1.0–1.5) ‡
1917 (n=28,717)	123	4.3	0.9 (0.7–1.2)
1918 (n=12,673)	192	15.2	3.2 (2.6–4.0) §

*p-value = 0.044 (1-tailed test)

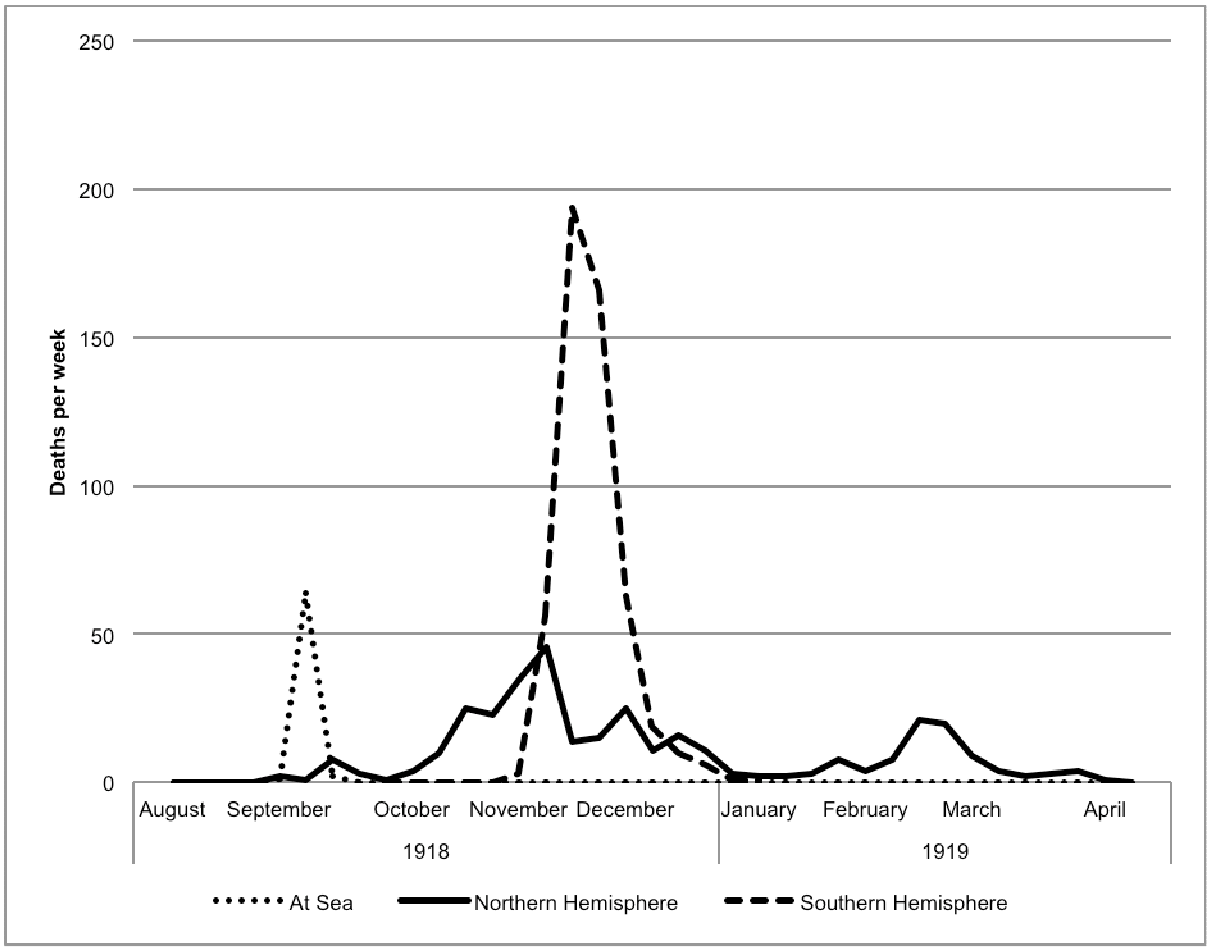
†Chi Square for Linear Trend = 53.31 (p-value <0.0000001)

‡p-value = 0.046 (1-tailed test)

§Chi Square for Linear Trend = 75.57 (p-value <0.00001)

The pandemic wave passing through the full NZEF is estimated to have occurred over a period of 33 weeks. However, the Southern Hemisphere experienced a shorter pandemic period compared to the Northern Hemisphere (Figure 2), consistent with other research.[19]

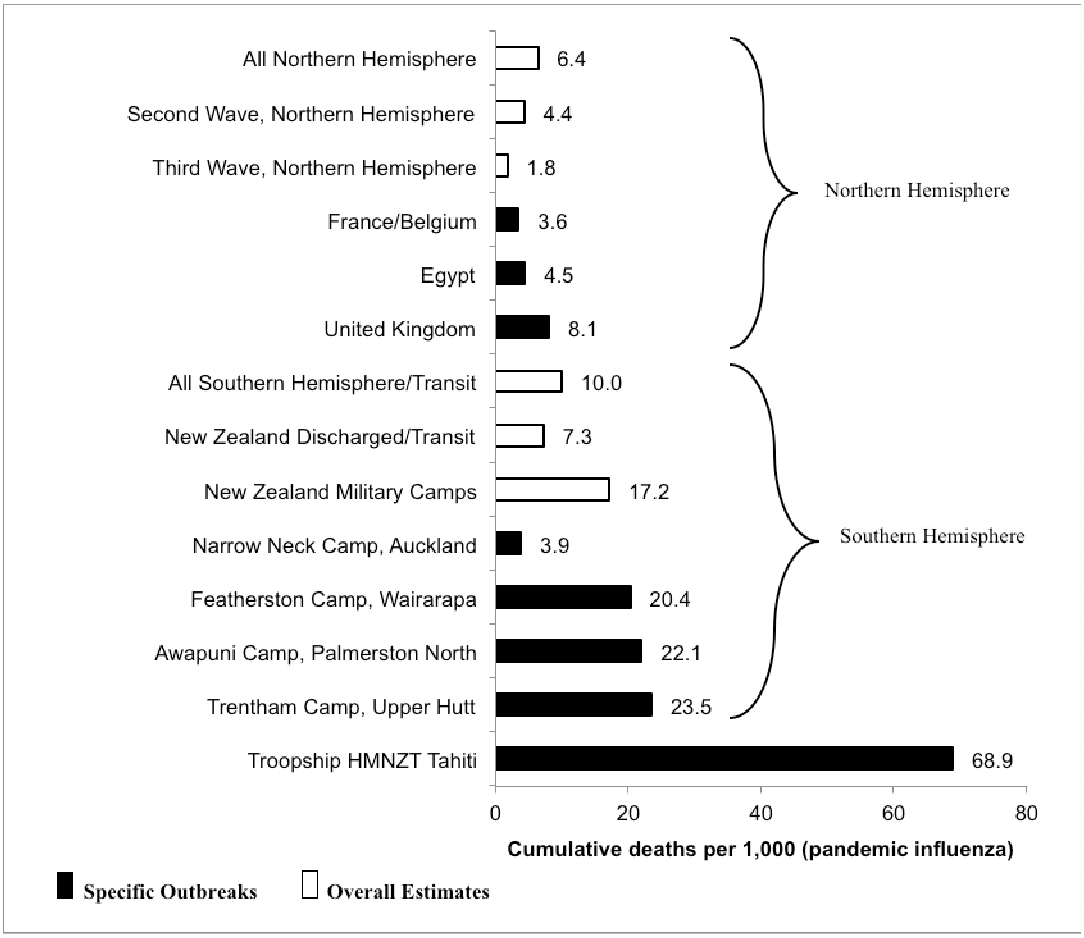
Figure 2. Pandemic Influenza deaths amongst NZEF personnel (1918–1919)



Specific outbreaks: Figure 3 shows estimated mortality rates for specific outbreaks and regional outbreaks of pandemic influenza amongst the NZEF, with the denominator values obtained from various sources.[5,10,13,16,19]

A reliable denominator value could not be determined for NZEF personnel located in the Southern Hemisphere (outside military camps), therefore the total includes NZEF personnel who were in transit during the pandemic.

Figure 3. Pandemic influenza mortality rates amongst NZEF personnel at different locations



Ethnicity—Both Māori and Pacific NZEF personnel experienced significantly higher pandemic mortality rates (20.2 per 1000 and 14.9 per 1000) when compared to European/Other NZEF personnel (9.2 per 1000) (Table 1). The majority of European/Other and Māori pandemic deaths occurred in the Southern Hemisphere, whilst deaths of Pacific personnel were equally distributed in both hemispheres.

Deployment year—NZEF personnel for whom 1918 was the first year of recruitment experienced the highest pandemic mortality rate (15.2 per 1000), which was statistically significant when compared to those whose first embarkments occurred in 1914–15 (with a mortality rate of 4.7 per 1000) (Table 1).

Age distribution—The average age for NZEF personnel dying from the pandemic was 29.0 years, slightly higher than the estimated average age for all NZEF personnel during 1918 of 28.0 years of age. The largest proportion of deaths occurred amongst the 25–29 year olds, consisting of 31.1% of pandemic cases (Table 1). However, the highest mortality rate was experienced by 30–34 year olds (11.4 per 1000), which was statistically significant when compared to under 20 year olds.

Occupation and rurality—NZEF personnel classified in the higher (most privileged) occupational classes of 1 to 3, experienced a higher mortality rate compared to the other occupational classes, but these differences were not statistically significant (Table 1).

NZEF personnel classified as highly rural in the rurality index had statistically significant higher mortality rates when compared to those from urban areas (Table 1). The Chi-squared value for linear trend for mortality rates with increasing rurality index was statistically significant (p-value <0.001).

Mortality by military rank—Similar mortality rates were experienced by Privates/Others, Non-Commissioned Officers and Officers (8.7, 8.5 and 9.1 per 1000 respectively). Health care workers experienced the lowest mortality rate amongst all military ranks (5.0 per 1000). However, there were no significant statistical differences between any military rank.

Discussion

The influenza pandemic of 1918-19 was a substantial cause of death amongst the NZEF personnel during and after WW1. The estimate of 930 pandemic deaths identified in this study indicates that previous assessments,[10,12] of the mortality burden amongst the NZEF were underestimates. This finding suggests that the total number of New Zealanders killed by the pandemic, NZ's largest natural disaster ever, needs to be revised upwards.[10] For example, this current study adds 258 more military specific deaths from the pandemic to the total found by Rice (n=603), resulting in a new grand national New Zealand total of 8831.

Overall, the mortality from this pandemic accounted for 28.3% of all estimated deaths from disease in the military in WW1, and an estimated 5.1% of all deaths amongst the NZEF personnel of WW1.

This research provides evidence that NZEF personnel located in the Southern Hemisphere military camps during the pandemic suffered disproportionately severely, in terms of mortality, compared to the Northern Hemisphere based NZEF and discharged/transiting NZEF personnel.

The Northern Hemisphere NZEF personnel experienced a more temporally dispersed outbreak, with a second pandemic wave in early 1919. There was no evidence in this mortality-orientated study of a subsequent pandemic wave occurring in 1919 amongst Southern Hemisphere/transiting NZEF personnel, which is consistent with the general NZ population with no increased mortality rates occurring post 1918.[10]

The majority of NZEF personnel who died from influenza in the Northern Hemisphere during 1919 were troops awaiting transport back to NZ after discharge.

The high pandemic mortality experienced by NZEF personnel who left for Europe in 1918 is consistent with other research in other military populations (Australia, USA and Britain) which suggests that 'fresh' recruits suffered disproportionately during the pandemic compared with 'seasoned' troops.[8,9,24,25]

Some commentators have suggested that newly recruited soldiers lacked immunity due to lack of exposure to influenza infections earlier in 1918. This mechanism may also partially explain the low mortality rate observed in Narrow Neck Camp

(previously exposed to the reportedly mild wave in October 1918[12, 26]) relative to the other three main military camps.

The lower overall mortality rate for personnel in the Northern Hemisphere and amongst health care workers (although not significant, is consistent with a previous study[25]) suggests that some level of acquired immunity may have existed in these sub-sections of the NZEF. This has been hypothesised in previous research regarding a range of WW1 military populations in Europe.[7]

Of the three regions in the Northern Hemisphere in which mortality rates were able to be calculated, it is notable that NZEF personnel located in France/Belgium experienced the lowest mortality rate. This was potentially partially due to the veteran status of the fighting soldiers, with few recent recruits. It is also perhaps another example of the potential protective role of immunity from a greater exposure to other infectious pathogens (e.g. to the bacteria that caused the secondary pneumonia in many influenza-related deaths in 1918).

The pattern of mortality rate by age, with a peak experienced by 30 to 34 year olds in this study is consistent with findings amongst the general NZ population[10,27] and in other populations.[28–30] There is still no established explanation for this unique and distinctive pattern of mortality by age during the influenza pandemic of 1918–19.

Māori and Pacific NZEF personnel experienced a much higher pandemic mortality rate when compared to the European/Other ethnic grouping. The findings for Māori personnel are consistent with other research which found a marked ethnic gradient in mortality in the overall NZ population in this and two subsequent influenza pandemics.[10,14]

Previous research investigating indigenous populations differential mortality outcomes during the 1918–19 pandemic has suggested that this may be the result of having lower socio-economic status (relative to the European/Other ethnic group), higher levels of rurality (potentially less acquired immunity through past respiratory pathogen exposure), poorer access to health care, and possibly higher rates of comorbidities.[14,31,32]

All these factors may have been relevant in the military setting—as there is no evidence for differential access to health care or to other basic provisions such as food and accommodation amongst the NZEF personnel in WW1.

A history of rural location was a risk factor in this study and not a protective factor as suggested by previous studies amongst the NZ civilian population,[23] and civilian populations in Australia and the USA.[24] However, the latter may have been due to social-distancing during the pandemic—which would not be relevant in crowded military populations during WW1.

In this military population, a history of rurality may instead have been a proxy indicator of lower cumulative exposure to respiratory pathogens and therefore possibly lower immunological protection when exposed to the influenza pandemic strain and associated bacterial pathogens.

It is plausible that other strains of influenza and pneumonia were circulating both during and pre/post pandemic periods. There are no known serological samples that exist from this NZEF population; but given that the NZEF personnel mortality burden

between 1915–1917 averaged a total of around ~330 non-combat deaths per year, it is plausible to assume that of the 1143 non-combat deaths which occurred amongst NZEF personnel during the pandemic period alone, the 930 pandemic deaths identified in this study are close to the accurate total.

A further limitation of this study is that all the mortality rate ratios are not age adjusted. However, this was not felt to be important for this study given that most of the NZEF personnel were in a fairly narrow age-band. Nevertheless, multivariate analyses has been undertaken as part of an unpublished PhD thesis as part of a case-control study.[18] When published, this will provide information on risk factors with age adjusted results (e.g. enlistment year and military rank as risk factors).

Examining the course of the influenza pandemic amongst the NZEF personnel is of strong historical and epidemiological importance. In terms of epidemiology, the study of past pandemics has the potential to provide knowledge beneficial to the planning and management of future pandemics. For example, the current study has demonstrated evidence that personnel of different ages but in similar regions experienced strikingly different mortality rates, a strong indication of the impact that host factors had during this pandemic.

Whilst it is plausible that pre-existing immunity (to virological and/or bacterial pathogens) provided protection amongst certain groups of the NZEF, it is not possible to be certain due to lack of surviving pathological specimens. The 1918-19 influenza pandemic represents one of the worst pandemics, of any kind, experienced by humans in recorded history. Therefore understanding the great lethality of this pandemic is relevant to preparations for future pandemics.

As more historical records become available and are analysed, the events that occurred around 1918 can potentially provide today's health sector planners a better basis for justifying allocation of scarce health care resources to pandemic preparations and to particular populations when another influenza pandemic arises.

Competing interests: Nil.

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References:

1. Murray CJ, Lopez AD, Chin B, et al. Estimation of potential global pandemic influenza mortality on the basis of vital registry data from the 1918-20 Pandemic: a quantitative analysis. Lancet. 2006;368:2211-8.
2. Byerly C. Fever of War New York: New York University Press; 2005.
3. Crosby AW. America's Forgotten Pandemic: The Influenza of 1918 New York: Cambridge University Press; 2003.
4. O'Neill CEJ. Court of Inquiry regarding H.M.N.Z.T "Tahiti". London: Archives New Zealand; 1918.
5. Summers JA, Wilson N, Baker MG, Shanks GD. Mortality Risk Factors for Pandemic Influenza on New Zealand Troop Ship, 1918. Emerg Infect Dis. 2010;16:1931-7.
6. Soper MGA. The Influenza Pneumonia Pandemic in the American Army Camps during September and October, 1918. Science. 1918;48:451-76.
7. Oxford J, Lambkin R, Sefton A, et al. A hypothesis: the conjunction of soldiers, gas, pigs, ducks, geese and horses in Northern France during the Great War provided the conditions for the emergence of the "Spanish" Influenza Pandemic of 1918-1919. Vaccine. 2005;23:940-5.
8. Brundage J, Shanks D. Deaths from bacterial pneumonia during the 1918-19 Influenza Pandemic. Emerg Infect Dis. 2008;14:1193-9.
9. Barry J, Viboud C, Simonsen L. Cross-protection between successive waves of the 1918-1919 Influenza Pandemic: epidemiological evidence from US army camps and from Britain. J Infect Dis. 2008;198:1427-34.
10. Rice G. Black November: The 1918 Influenza Pandemic in New Zealand. 2nd ed. Christchurch: Canterbury University Press; 2005.
11. Erye J, Lowe E. Report upon the Autumn Influenza Epidemic (1918) as it affected the N.Z.E.F. in the United Kingdom. Lancet. 1919;(April 5, 1919):553-60.
12. Carbery A. The New Zealand Medical Service in the Great War, 1914-1918. Auckland: Whitcombe and Tombs Limited; 1924.
13. Sertsou G, Wilson N, Baker M, et al. Key transmission parameters of an institutional outbreak during the 1918 influenza pandemic estimated by mathematical modelling. Theor Biol Med Model. 2006;3(38).
14. Wilson N, Barnard Telfar L, Summers J, et al. Differential Mortality Rates by Ethnicity in 3 Influenza Pandemics Over a Century, New Zealand. Emerg Infect Dis. 2011;18:71-7.
15. Maclean F. Challenge for health: a History of Public Health in New Zealand. Wellington: RE Owen, Government Printer; 1964.
16. N.Z.E.F Base Records Office. Graph showing the N.Z.E.F Overseas by Monthly Totals – including Embarkations, Disembarkations, and Final Disposition – from Mobilization to Demobilization. In: Pugsley C, editor. On the fringe of hell: New Zealanders and military discipline in the First World War. Auckland: Hodder & Stoughton Ltd; 1991.
17. Anne Bromell Collection. Chronicles of the N.Z.E.F., 1916-1919. [cited January 2012]; Available from: http://www.ancestry.com.au
18. Summers JA. (2013). The Burden and Risk Factors for Death from the 1918-19 Influenza Pandemic amongst the New Zealand Military Forces of World War One (Thesis, Doctor of Philosophy). University of Otago. Abstract available from: http://hdl.handle.net/10523/3766
19. NZ History. 2012 [cited January 2012]; Available from: http://www.nzhistory.net.nz
20. Pugsley C. Te Hokowhitu a Tu. The Maori Pioneer Battalion in the First World War (2nd Ed). Auckland: Raupo Publishing (NZ) Ltd; 1995.

21. Births Deaths and Marriages New Zealand. Births Deaths and Marriages Historical Records. 2012 [cited January 2009 to January 2012]; Available from: http://bdmhistoricalrecords.identityservices.govt.nz/Home/
22. Olssen E, Hickey M. Class and occupation: the New Zealand reality. Dunedin: Otago University Press; 2005.
23. McSweeny K, Coleman A, Fancourt N, et al. Was rurality protective in the 1918 Influenza Pandemic in New Zealand?". N Z Med J. 2007;120:U2579.
24. Paynter S, Shanks G. Host and environmental factors reducing mortality during the 1918-19 Influenza Pandemic. Epidmiol Infect. 2011;139:1425-30.
25. Shanks G, MacKenzie A, Waller M, Brundage J. Low but highly variable mortality among nurses and physicians during the Influenza Pandemic of 1918-19. Influenza Other Respi Viruses. 2011;5:213-9.
26. Cooper Cole C. Influenza epidemic at Bramshott in September-October, 1918. Br Med J. 1918;2(23 November):566-8.
27. Bryder L. The 1918 Influenza Epidemic in Auckland [Unpublished MA Thesis, University of Auckland]. University of Auckland; 1980.
28. Mamelund S. A socially neutral disease? Individual social class, household wealth and mortality from Spanish Influenza in two socially contrasting parishes in Kristiania 1918-19. Soc Sci Med. 2006;62:923-40.
29. Andreasen V, Viboud C, Simonsen L. Epidemiological Characterization of the 1918 Influenza Pandemic Summer Wave in Copenhagen: Implications for Pandemic Control Strategies. J Infect Dis. 2008;197:270-8.
30. Chowell G, Viboud C, Simonsen L, et al. Mortality Patterns associated with the 1918 Influenza Pandemic in Mexico: evidence for a spring herald wave and lack of preexisting immunity in older populations. J Infect Dis. 2010;202:567-75.
31. Mamelund S. Spanish Influenza mortality of ethnic minorities in Norway 1918-1919. Eur J Popul. 2003;19:83-102.
32. Mamelund S. Geography may explain adult mortality from the 1918-20 Influenza Pandemic. Epidemics. 2011;3:46-60.

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Awareness and perceived effectiveness of smoking cessation treatments and services among New Zealand parents resident in highly deprived suburbs

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Abstract

Aim To describe the awareness and perceived effectiveness of smoking cessation treatments and services among a population of mainly Māori and Pacific parents in South Auckland, New Zealand.

Method Parents of pre-adolescent children from 4 schools were surveyed from 2007–2009 using a self-complete questionnaire. Awareness and perceived effectiveness of cessation treatments and services were analysed by smoking status, ethnicity, gender and age. Relative risks were calculated using log-binomial regression to establish differences between smokers and non-smokers.

Results Awareness of Quitline, nicotine gum, and nicotine patch was higher among smokers (94%, 91%, 90%) than non-smokers (87%, 73%, 64%). Low percentages of smokers reported cessation interventions as effective (only 41% for Quitline—the intervention perceived effective by most). Awareness of varenicline, bupropion and nortriptyline was the lowest among both smokers and non-smokers (<31%).

Conclusion Poor awareness and low perceived efficacy of smoking cessation treatments and services among priority groups are barriers to accelerating the reduction of smoking prevalence in New Zealand.

Tobacco smoking is a major cause of mortality and morbidity globally.[1] In New Zealand (NZ), smoking contributes to an estimated 5000 deaths annually, around 17% of total mortality.[2,3]

Despite a comprehensive tobacco control programme that includes regular price increases, graphic health warnings on packets, restricted tobacco advertising and marketing, smokefree workplaces, social marketing campaigns and government funded cessation support and subsidised treatments,[4,5] smoking prevalence is still around 20% overall and disproportionately higher among Māori (the indigenous people of NZ), Pacific Island communities, and in areas of high socioeconomic deprivation.[6]

The NZ toll-free Quitline and the ready availability of subsidised nicotine replacement therapy (NRT) are central components of the NZ tobacco control programme. They represent a significant investment by Government in providing cessation support to smokers.

Nicotine patches, gum and lozenges are available through the Quitcard (exchange voucher) programme at only $NZ3 (increasing to $NZ5 from 1 January 2013) for an 8-week supply.[7] Quitcards are issued by the Quitline service (phone, SMS or online

support), primary care physicians, and available through trained Quitcard providers, largely healthcare workers based in community or hospital settings. NRT can also be purchased at full price over the counter in supermarkets and community pharmacies.

A number of treatments purporting to aid cessation—hypnosis, acupuncture, electronic cigarettes and Nicobrevin—are promoted as ways to stop smoking but are not recommended by the New Zealand Smoking Cessation Guidelines due to a lack of evidence of effectiveness.[8]

Interest among smokers in a number of settings in using alternative treatments appears to be high,[9–14] but reported use of alternative cessation treatments is low.[12,15–18] Nevertheless, 23% to 39% of smokers and recent quitters surveyed in the USA considered acupuncture and hypnosis to be effective treatments.[19,20]

Disappointingly, effective cessation treatments are underused.[15] In 2009, only 2.1% of an estimated 70,000 smokers in a large multi-ethnic suburban population in Auckland, New Zealand's largest city (population 1.4 million), reported having used subsidised NRT in a quit attempt,[21] although older smokers and females were more likely to have used NRT than younger smokers and males respectively.[21]

A 2008 analysis of national Quitline data found that only 8.2% of the smoking population from this same area had ever called for quitting support.[22] Analysis of Quitcard redemptions over six months in 2007 showed that only two thirds of callers from this region who had been provided with a Quitcard had exchanged it for NRT.[23]

Attempting to quit smoking without the assistance of behavioural support and or cessation treatments is common in New Zealand (just 34% use cessation products/advice).[15] It is also common around the world, with two-thirds to three-quarters of quit attempts proceeding without assistance.[16, 24]

Internationally, there appears to be a commonly-held belief among smokers that cessation treatments are unnecessary and that strong self-control and a desire to quit are sufficient.[9,10,25] The use of medications may be perceived as a sign of weakness, reflecting an inability to deal with one's problems and to be in control of one's own willpower and motivation,[10,26] and using medications or treatments may be seen as not really quitting.[27]

Studies in a range of countries have found that smokers commonly believe cessation treatments are ineffective at helping them quit.[9–12,28–31] In some of these surveys, cessation treatments were viewed with caution, as smokers perceived that they could be addictive, and that cessation treatments would not address the reasons people smoke.[10,27–29]

Further barriers to the use of cessation medication include concerns about the safety and side effects of treatments, particularly the risks of overdosing on nicotine, or that they may be more dangerous than cigarettes. Studies in New Zealand and the USA have found that many smokers mistakenly perceive that it is the nicotine in cigarettes that causes tobacco-related cancers.[32,33]

In order to identify strategies for increasing the use of evidence-based treatments among population groups with the highest smoking prevalence (Māori, Pacific Island and people of lower socioeconomic position), we set out to examine their awareness

of cessation medications and quit support services, and perceptions of their effectiveness.

Methods

We analysed data from a large quasi-experimental smoking prevention trial, *Keeping Kids Smokefree*,[34] conducted from 2007–2010 in South Auckland, an urban area of high ethnic diversity and socioeconomic deprivation.

The study aimed to test interventions to reduce uptake of smoking among pre-adolescents.[34] An allied objective of the study was to increase smoking cessation among parents. A detailed account of the method of data collection and recruitment is described elsewhere.[34] In brief, baseline and follow up surveys of a singular parent/caregiver of participating children were conducted between 2007 and 2009.

Data for the current study were drawn primarily from follow-up surveys. Demographic information on ethnicity, gender and smoking status was drawn from the 2007 baseline survey. Students delivered the questionnaires to parents and returned them once completed. Data were entered into Epi Info,[35] checked and validated against the original questionnaire by a data entry supervisor at the study centre.

Participants were asked to indicate their awareness and or perceived effectiveness of various smoking cessation treatments and services. The treatments and services included in the questionnaire represented the majority of the smoking cessation treatment options available in NZ at the time (nicotine patch, gum, inhaler, Microtab and lozenge), three pharmacotherapies available on prescription (bupropion, nortriptyline, and from 2009, varenicline), and three alternative treatments (hypnosis, acupuncture and Nicobrevin).

The services included were Quitline and the Aukati Kai Paipa programme (a face-to-face smoking cessation service aimed at Māori, and delivered by local Māori community health providers around NZ).

For the purpose of analysis, four response options (*I have never heard of this treatment/service* (not aware)*; I have heard of it but I don't know if it works* (aware, not perceived effective)*; I don't think this treatment/service works* (aware, not perceived effective)*; I think this treatment/service works* (aware, perceived effective) were recoded into two binary variables (Aware/not aware; perceived effective/not perceived effective). The variable 'not perceived effective' is taken to denote not only perceived ineffectiveness, but also encompasses uncertainty over the effectiveness of the treatments/services.

Participants specified whether they had ever smoked, and if so, how much tobacco they currently smoked. Participants who had never or no longer smoked were coded as non-smokers, while participants reporting smoking at least one cigarette per day were coded as current smokers. The questionnaire did not distinguish between daily and non-daily smoking or account for people smoking less than one cigarette per day. Participants identified their ethnicity by selecting from *Māori*, *Pākehā/European*, *Pacific Island*, or *Other*.

Participants selecting *Pacific Island* or *Other* were asked to specify their ethnicity in a free text field. Multiple responses were prioritised to a single category in the order Māori, Pacific Island, Asian and New Zealand European/Pākehā using Statistics New Zealand's prioritisation standard.[36]

Age was grouped into three age ranges *16-24*, *25-44*, and *45 and over.* Gender was inferred from the participant's relationship to the school student, with gender roles such as *mother*, *aunty*, *other female caregiver* being coded as *female* and gender roles such as *father*, *uncle*, and *other male caregiver* being coded as *male*.

Frequencies and cross tabulations were performed to describe awareness and perceived effectiveness of each treatment by age, gender, ethnicity and smoking status. Differences between age, gender and ethnic groups were assessed using Pearson's Chi-squared tests. In cases where one or more cells had a value of less than 5, Fisher's exact test was used. A p-value < 0.05 was considered statistically significant. Data was analysed using SAS v 9.2 (SAS Institute., Cary, NC, US).

Relative risks (RR) were calculated using log-binomial regression to establish differences between smokers and non-smokers while adjusting for ethnicity. Differences in awareness or perceived effectiveness between smokers and non-smokers were considered to be statistically significant if the 95% confidence intervals (CI) did not include the value one.

As the survey instrument was a self-complete questionnaire, it was not uncommon for some questions to be left unanswered or for responses to be invalid. The denominator reported is that of valid responses excluding missing data.

Ethics approval for the study was obtained from the University of Auckland Human Participants Ethics Committee (Ref. 2006/416). The study was registered with the Australian and New Zealand Clinical Trials Registry (ACTRN12611000591954).

Results

The response rate was 67% (3722/5557) over the three years of parent follow-up surveys. Participants surveyed were mainly female (74.5%), aged 25–44 years old (71.0%). Approximately 43% were Pacific Island, 25% were Māori, 23% Asian, and almost 10% were NZ European.

Smoking prevalence was 27.6% but there were significant differences in smoking prevalence by ethnicity and age group with Māori (51.7%) and 25–44 year olds (29.4%) having higher smoking prevalence (Table 1).

Table 1. Participant characteristics and smoking status

Variables	All participants	Smoking status known		P-value*
		Non-smoker (row %)	Smoker (row %)	
Gender	3095			
Female	2306	1483 (72.7)	558 (27.3)	0.73
Male	789	519 (72.0)	202 (28.0)	
Ethnicity	3151			
Māori	784	329 (48.3)	352 (51.7)	<0.0001
Pacific	1349	924 (74.7)	296 (24.3)	
Asian	731	612 (91.8)	55 (8.2)	
NZ European	287	162 (68.6)	74 (31.0)	
Age group	3456			
16-24	115	72 (80.0)	18 (20.0)	0.004
25-44	2453	1407 (70.6)	587 (29.4)	
45+	888	530 (76.1)	166 (23.9)	
Smoking prevalence	2835	2053 (72.4)	782 (27.6)	<0.0001

*Calculated from Pearson's Chi-square

Awareness of cessation treatments and services—Participants were most aware of the Quitline, nicotine gum, and nicotine patch, followed by hypnosis, acupuncture and Nicobrevin. Participants were less aware of newer forms of NRT (inhaler, lozenge, Microtab), prescription only treatments (varenicline, bupropion, nortriptyline), and the Aukati Kai Paipa service, with between half to a quarter of participants being aware of these treatments. Smokers were significantly more likely to be aware of the nicotine patch, nicotine gum, hypnosis, Nicobrevin and the Quitline service, than non-smokers (Table 2).

Table 2. Awareness of treatments and services by smoking status

Treatment (n)	Awareness of treatment			RR** (95%CI)
	All participants* N (%)	Smokers N (%)	Non-smokers N (%)	
Quitline (2568)	2291 (89.2)	672 (94.2)	1619 (87.3)	1.04 (1.01–1.07)
NRT Gum (2442)	1894 (77.6)	616 (90.7)	1278 (72.5)	1.08 (1.04–1.13)
NRT Patch (2493)	1875 (75.2)	628 (90.1)	1247 (69.4)	1.11 (1.06–1.16)
Hypnosis (2346)	1396 (59.5)	473 (73.1)	923 (54.3)	1.06 (1.001–1.133)
Acupuncture (2331)	1373 (58.9)	432 (67.5)	941 (55.6)	1.00 (0.94–1.07)
Nicobrevin (2339)	1215 (51.9)	420 (65.4)	795 (46.8)	1.10 (1.02–1.19)
NRT Inhaler (2340)	1080 (46.2)	296 (46.5)	784 (46.0)	0.95 (0.86–1.06)
NRT Lozenge (2335)	949 (40.6)	280 (43.7)	669 (39.5)	1.08 (0.96–1.21)
Aukati Kai Paipa (2127)	754 (35.4)	233 (39.8)	521 (33.8)	0.91 (0.80–1.03)
NRT Microtab (2335)	839 (35.9)	229 (35.8)	610 (36.0)	0.95 (0.84–1.09)
Varenicline/Champix (1644)	463 (28.2)	131 (30.2)	332 (27.4)	1.03 (0.86–1.23)
Bupropion/Zyban (2314)	654 (28.3)	190 (29.7)	464 (27.7)	0.99 (0.85–1.15)
Nortriptyline/Norpress (2320)	613 (26.4)	167 (26.3)	446 (26.5)	0.96 (0.81–1.13)

*Numerator is sum of non-smokers and smokers.

**Relative Risk with 95% Confidence Intervals adjusted for ethnicity. Non-smoker was the reference group.

Female smokers had significantly ($p<0.05$) higher awareness of the Quitline (96.3% vs 88.2%), nicotine gum (93.7% vs 82.9%), nicotine patch (92.8% vs 83.1%), hypnosis (76.0% vs 65.8%), acupuncture (72.2% vs 65.8%), and Nicobrevin (70.4% vs 50.0%) than male smokers.

There were no significant differences in awareness of newer NRT (inhaler, lozenge, Microtab), prescription only treatments (varenicline, bupropion, nortriptyline), or the Aukati Kai Paipa service by gender among smokers (data not shown).

There were significant differences in awareness of the Quitline, nicotine gum and patch, hypnosis, acupuncture, Nicobrevin and the Aukati Kai Paipa service by ethnicity amongst smokers (Table 3). Māori, Pacific, and NZ European smokers were more aware of the Quitline than Asian smokers.

Māori and NZ European smokers were more aware of nicotine gum and patch, hypnosis, acupuncture, and Nicobrevin than Pacific and Asian smokers. Māori smokers were more aware of the Aukati Kai Paipa service than smokers of other ethnic groups.

Smokers aged 16–24 years old were significantly ($p<0.05$) more aware of the nicotine Microtab (than smokers aged 25–44, and 45 years or older) (66.7% vs 33.9% and 38.9%), and bupropion (60.0% vs 28.5% and 31.0%). There were no significant differences in awareness of other treatments or services by age group (data not shown).

Table 3. Ethnic differences in awareness of treatments among smokers

Treatment (n)	Awareness of Treatment (smokers)					P-value
	All* N (%)	Māori N (%)	Pacific N (%)	Asian N (%)	NZ European N (%)	
Quitline (709)	609 (94.4)	309 (95.4)	256 (95.5)	40 (81.6)	64 (94.1)	0.007†
NRT gum (676)	613 (90.7)	294 (95.5)	221 (86.7)	38 (79.2)	60 (92.3)	<0.0001†
NRT patch (693)	625 (90.2)	299 (94.3)	229 (87.1)	35 (72.9)	62 (95.4)	<0.0001†
Hypnosis (644)	471 (73.1)	241 (82.0)	157 (64.6)	23 (51.1)	50 (80.6)	<0.0001
Acupuncture (637)	430 (67.5)	219 (75.5)	148 (60.9)	20 (45.5)	43 (71.7)	<0.0001
Nicobrevin (639)	418 (65.4)	216 (74.0)	143 (59.3)	17 (38.6)	42 (67.7)	<0.0001
NRT inhaler (634)	294 (46.4)	137 (47.9)	103 (42.6)	21 (47.7)	33 (53.2)	0.410
NRT lozenge (638)	280 (43.9)	123 (42.9)	104 (42.1)	20 (42.5)	33 (55.0)	0.321
Aukati Kai Paipa (584)	232 (39.7)	134 (48.4)	72 (34.6)	8 (20.5)	18 (30.0)	<0.0001
NRT Microtab (637)	228 (36.8)	104 (36.0)	78 (32.2)	20 (46.5)	26 (41.3)	0.230
Varenicline/Champix (433)	131 (30.3)	58 (31.0)	46 (27.2)	11 (31.4)	16 (38.1)	0.565
Bupropion/Zyban (637)	190 (29.8)	90 (30.8)	69 (28.9)	15 (34.1)	16 (25.8)	0.776
Nortriptyline/Norpress (632)	168 (26.6)	75 (25.5)	64 (27.1)	16 (36.4)	13 (21.0)	0.338

*Numerator is sum of each ethnicity †Fisher's exact test

Perceived effectiveness of smoking cessation treatments and services

Smokers were most likely to perceive the Quitline as effective, followed by nicotine patch, the Aukati Kai Paipa service, and nicotine gum (Table 4). However, smokers were significantly less likely to perceive the Quitline, nicotine gum or patch, Nicobrevin, or the Aukati Kai Paipa service as effective, compared with non-smokers.

Table 4. Perceived effectiveness of treatments by smoking status

Treatment (n)	Perceived effectiveness of Treatment			RR** (95% CI)
	All participants** N (%)	Smokers N (%)	Non-Smokers N (%)	
Quitline (2291)	1180 (51.5)	278 (41.4)	902 (55.7)	0.77 (0.69–0.85)
NRT gum (1894)	495 (26.1)	125 (20.3)	370 (29.0)	0.73 (0.61–0.88)
NRT patch (1875)	601 (32.1)	179 (28.5)	422 (33.8)	0.83 (0.71–0.97)
Hypnosis (1396)	258 (18.5)	88 (18.6)	170 (18.4)	0.97 (0.76–1.23)
Acupuncture (1373)	201 (14.6)	59 (13.7)	142 (15.1)	0.91 (0.68–1.23)
Nicobrevin (1215)	255 (21.0)	72 (17.1)	183 (23.0)	0.74 (0.57–0.96)
NRT inhaler (1080)	177 (16.4)	32 (10.8)	145 (18.5)	0.70 (0.48–1.02)
NRT lozenge (949)	166 (17.5)	36 (12.9)	130 (19.4)	0.79 (0.55–1.13)
Aukati Kai Paipa (754)	259 (34.6)	60 (25.8)	199 (38.2)	0.63 (0.49–0.81)
NRT Microtab (839)	131 (15.6)	25 (10.9)	106 (17.4)	0.72 (0.47–1.11))
Varenicline/Champix (463)	86 (18.6)	24 (18.3)	62 (18.7)	0.99 (0.63–1.55)
Bupropion/Zyban (654)	113 (17.3)	30 (15.8)	83 (17.9)	0.94 (0.63–1.41)
Nortriptyline/Norpress (613)	93 (15.2)	21 (12.6)	72 (16.1)	0.83 (0.52–1.33)

*Numerator is sum of non-smokers and smokers.

**Relative Risk with 95% Confidence Intervals adjusted for ethnicity. Non-smoker was the reference group.

Male smokers were significantly more likely than female smokers to perceive nicotine gum (26.1% vs 17.7%, p<0.05) and nicotine lozenge (19.7% vs 10.3%, p<0.05) to be

effective. There were no significant differences in perceived effectiveness by gender for any other treatments (data not shown).

NZ European smokers (36%) were more likely to perceive hypnosis as an effective treatment than Māori (16.6%), Pacific (16.6%), or Asian smokers (17.4%). Asian smokers were more likely to perceive the nicotine inhaler as an effective treatment (28.6%) than Māori (8.0%), Pacific (13.6%), or NZ European smokers (3.0%). There were no other significant differences in perceived effectiveness by ethnicity among smokers (data not shown).

Smokers aged 45 years and over (35.2%) were more likely to perceive nicotine patch as an effective treatment than smokers aged under 25 (6.7%), and aged 25–44 (27.4%). Smokers aged over 45 years were also more likely to perceive acupuncture (age 45 plus, 23.6%, vs age 16-24, 9.1%, and age 25-44, 11.3%) and Nicobrevin (age 45 plus 25.3% vs age 16-24, 0.0%, and age 24–44, 15.6%) as effective treatments than younger smokers. There were no other significant differences in perceived effectiveness by age group among smokers (data not shown).

Discussion

In this study conducted mainly with Māori and Pacific parents of pre-adolescent children, resident in a socio-economically deprived area of Auckland, awareness of Quitline (89%), gum (78%) and patch (75%) was high, and Quitline (52%), Aukati Kai Paipa (35%), and patch (32%) were the services and treatment considered most likely to be effective.

At the time of this study the Quitline had been in place for seven years and patch and gum were first subsidised in 2000. Therefore, the higher awareness of these could be in part attributable to their longer period of availability, the resulting higher profile via advertising of Quitline and earned media coverage of subsidised NRT. Quitline also supplies Quitcards, giving access to the subsidised patch and gum (and lozenge), so these would be expected to be highly correlated.

Our findings that smokers were more aware of smoking cessation treatments and services than non-smokers, and yet were generally less likely to perceive any particular cessation treatment as effective are consistent with the literature.[9–11,29–31,37,38]

It is concerning that awareness of hypnosis, acupuncture and Nicobrevin persists at a higher level (52-60%) than the wider range of recommended cessation treatments (26-46%).

We had expected awareness of the nicotine lozenge to be higher, since it was first subsidised in 2008. Nortriptyline and bupropion were subsidised from 2003 and 2009 respectively and varenicline partially so from 2010, but these prescription medicines are recommended as second line therapies after NRT in the New Zealand Smoking Cessation Guidelines. Contraindications also limit the number of smokers able to use these treatments.

Slightly lower awareness of patch and gum among Pacific and Asian people and lower awareness of Quitline among Asian people is unsurprising as campaigns promoting cessation have predominantly targeted NZ Europeans and Māori, who in

2008/2009 made up 70% and 21% of Quitline registrations respectively,[39] while Pacific and Asian clients accounted for just 6% and 3% of registrations.

Knowledge of cessation treatments often comes from smokers' social networks, particularly other smokers[14,28] rather than health professionals. While knowing someone who had successfully quit by using an evidence-based cessation treatment could be a powerful influence on decision making processes,[14,30] knowing someone who used an evidence-based treatment without success could influence decision making away from using such treatments.

Before smoking cessation treatments such as NRT or prescription medications were widely available, smokers who quit smoking mostly did so without professional support.[40] Qualitative research conducted in New Zealand 2009/2010 found Māori and Pacific Island smokers perceived willpower as sufficient for quitting smoking.[41]

Furthermore, there was a notion amongst some that smoking addiction was a myth, or just a habit, and that if smoking is not really addictive, then an external intervention such as a medication or a stop smoking service could not be of any assistance.[41]

Dessaix et al[42] argued that smokers place high value on use of willpower in a quit attempt, but that when they used treatments such as NRT, there was an expectation that the treatment should act like a magic bullet, that the treatment would do the quitting on their behalf, and when the quit attempt is not successful, blame is placed upon the treatment for not working.

Strengths and limitations—The study had a large sample size of 3722. It is a strength that all parents of the four participating schools were approached, meaning there was near full enumeration of the sample rather than using only a random subset.

Three-quarters of participants were mothers of child-bearing age of mainly Māori and Pacific ethnic groups. This could be seen as a strength as it provides hitherto unavailable information on a priority population group for tobacco control efforts: Māori women of child-bearing age have the highest prevalence of smoking (61% of Māori women aged 20–24 and 55% of Māori women aged 25–44 smoke tobacco) in New Zealand,[43] followed by Pacific women (29% aged 15-64).[2]

Māori women have a high prevalence of smoking while pregnant (45% at registration with a maternity carer, and 29% smoking following delivery).[44]

This study was cross-sectional in nature so no conclusions can be made about temporal associations. The modest response rate of 67% and the differential rate of invalid responses between explanatory variables such as age, gender, ethnicity and smoking status, and the treatment items in the questionnaire weakens any conclusions drawn.

Participants were prompted with the names of treatments and services, which may have inflated awareness of some treatments—unprompted responses may provide a truer representation of awareness of treatments, consequently a self-complete questionnaire may not be the appropriate survey instrument.

Conclusions and recommendations—If NZ is to attain its smokefree 2025 goal of less than 5% smoking prevalence, the rate of decline must accelerate—at the current rate of declining prevalence (23.7% in 1996, 20.7% in 2006) it will take generations

to arrive at a smokefree society.[45] One way to accelerate this is to provide smokers with better and more frequent access to effective support so that they can make 'more supported quit attempts more often'.[46]

While other aspects of a tobacco control program (taxes, smokefree environments, media campaigns) generate motivational tension, and make triggers to quit more salient,[47] treatments need to be readily available and smokers need to be attracted to using them. Lack of awareness of treatments and services, and perceived lack of efficacy are two barriers that should be addressed.

Recommendations for triggering quit attempts and wider use of effective cessation treatments and services among Māori, Pacific peoples and other priority populations have included culturally salient group quit smoking contests aimed at triggering clusters of smokers to quit, with greater access to treatments and behavioural support; offering smoking cessation more directly to smokers in the community; and greater use of communications media such as phone, email and social networking platforms.[48]

Campaigns and events to trigger mass quitting, such as World Smokefree Day, No Smoking Day,[49] and Stoptober[50] could be synergistically deployed to raise awareness of effective and available treatments thereby triggering more supported quitting. More could be done to raise awareness of some of the newer treatments available, particularly the prescription only treatments, and the NRT inhaler.

More effort needs to be placed on emphasising the safety and effectiveness of NRT and other cessation treatments. This could be done using the warning labels on tobacco packaging to communicate directly to smokers.[32]

Surveys of NZ doctors, midwives, and nurses in 2006/7 found that knowledge of evidence-based smoking cessation treatments was poor,[51, 52] though knowledge should have improved with the release of the NZ Smoking Cessation Guidelines and the introduction of the government health target *Better help for smokers to quit* and associated ABC programme.[46,53]

Further research is needed to assess if health professionals have greater awareness and perceptions of treatments and services than previously found. Deficiencies found should be addressed with training and education at undergraduate and postgraduate levels.

Competing interests: Nil.

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References:

1. Eriksen M, Mackay J, Ross H. The Tobacco Atlas. 4th ed. Atlanta, GA: American Cancer Society; 2012.
2. Ministry of Health. Tobacco Trends 2008:A brief update on tobacco use in New Zealand. Wellington: Ministry of Health; 2009.
3. Ministry of Health. Mortality and Demographic Data 2008. Wellington: Ministry of Health; 2011.
4. Laugesen M, Swinburn B. New Zealand's tobacco control programme 1985-1998. Tobacco Control. 2000;9:155-62.
5. Wilson N, Thomson G, Edwards R. Use of four major tobacco control interventions in New Zealand: A review. New Zealand Medical Journal. 2008;121:71-86.
6. Ministry of Health. Tobacco Use in New Zealand: Key Findings from the 2009 New Zealand Tobacco Use Survey. Wellington: Ministry of Health; 2010.
7. NRT and Quitcards. The Quit Group, 2012. URL: http://www.quit.org.nz/69/helping-others-quit/research/nrt-and-quitcards
8. Ministry of Health. New Zealand Smoking Cessation Guidelines. Wellington: Ministry of Health; 2007.
9. Hammond D, McDonald PW, Fong GT, Borland R. Do smokers know how to quit? Knowledge and perceived effectiveness of cessation assistance as predictors of cessation behaviour. Addiction. 2004;99:1042-8.
10. Vogt F, Hall S, Marteau TM. Understanding why smokers do not want to use nicotine dependence medications to stop smoking: Qualitative and quantitative studies. Nicotine & Tobacco Research. 2008;10:1405-13.
11. Roddy E, Antoniak M, Britton J, et al. Barriers and motivators to gaining access to smoking cessation services amongst deprived smokers – a qualitative study. BMC Health Services Research. 2006;6:147.
12. Sood A, Ebbert JO, Sood R, Stevens SR. Complementary treatments for tobacco cessation: A survey. Nicotine & Tobacco Research. 2006;8:767-71.
13. Jackson N, Prebble A, Rose CS. Perceptions of smoking cessation products and services among low income smokers. London: Health Development Agency; 2002. https://nice.org.uk/nicemedia/documents/perceptions_smoking_cessation.pdf
14. Burgess D, Fu SS, Joseph AM, et al. Beliefs and experiences regarding smoking cessation among American Indians. Nicotine & Tobacco Research. 2007;9:19-28.
15. Ministry of Health. New Zealand Tobacco Use Survey 2008: Quitting results. Wellington: Ministry of Health; 2009.

16. Shiffman S, Brockwell S, Pillitteri J, Gitchell J. Individual differences in adoption of treatment for smoking cessation: Demographic and smoking history characteristics. Drug and Alcohol Dependence. 2008;93:121-31.
17. Lillard DR, Plassmann V, Kenkel D, Mathios A. Who kicks the habit and how they do it: Socioeconomic differences across methods of quitting smoking in the USA. Social Science & Medicine. 2007;64:2504-19.
18. West R, Zhou X. Is nicotine replacement therapy for smoking cessation effective in the "real world"? Findings from a prospective multinational cohort study. Thorax. 2007;62:998-1002.
19. McMenamin SB, Halpin HA, Bellows NM. Knowledge of Medicaid Coverage and Effectiveness of Smoking Treatments. American Journal of Preventive Medicine. 2006;31:369-74.
20. Public Perceptions about the Effectiveness of Tobacco Cessation Products and Services. RWJF Research Highlight Number 20. 2007. URL: http://www.rwjf.org/files/publications/other/Research%20Highlight%2020%5B3%5D.pdf
21. Thornley S, Jackson G, McRobbie H, et al. Few smokers in South Auckland access subsidised nicotine replacement therapy. New Zealand Medical Journal. 2010;123:16-27.
22. The Quit Group. The Quit Group: Quitline Client Analysis Report – July 2007-June 2008. Wellington: The Quit Group; 2008. http://www.quit.org.nz/file/research/Yearly%20Callers%20Report%20200807-200906.pdf
23. Li J. Redemption of Quit Cards distributed through Quit Services, July – December 2007. Wellington: The Quit Group; 2009. http://www.quit.org.nz/file/research/NRT%20Redemption%20Report%20Jul-Dec%202007%20FINAL%2020090420.pdf
24. Chapman S, MacKenzie R. The Global Research Neglect of Unassisted Smoking Cessation: Causes and Consequences. PLoS Med. 2010;7:e1000216.
25. Bansal MA, Cummings KM, Hyland A, Giovino GA. Stop-smoking medications: Who uses them, who misuses them, and who is misinformed about them? Nicotine & Tobacco Research. 2004;6:303-10.
26. Balmford J, Borland R. What does it mean to want to quit? Drug and Alcohol Review. 2008;27:21-7.
27. Kishchuk N, Tremblay M, Lapierre J, et al. Qualitative investigation of young smokers' and ex-smokers' views on smoking cessation methods. Nicotine Tob Res. 2004;6:491-500.
28. Fu SS, Burgess D, van Ryn M, et al. Views on smoking cessation methods in ethnic minority communities: A qualitative investigation. Preventive Medicine. 2007;44:235-40.
29. White M, Bush J, Kai J, et al. Quitting smoking and experience of smoking cessation interventions among UK Bangladeshi and Pakistani adults: the views of community members and health professionals. J Epidemiol Community Health. 2006;60:405-11.
30. Wiltshire S, Bancroft A, Parry O, Amos A. 'I came back here and started smoking again': perceptions and experiences of quitting among disadvantaged smokers. Health Education Research. 2003;18:292-303.
31. Leatherdale S, Shields M. Smoking cessation: intentions, attempts and techniques. Health Reports. 2009;20:31-9.
32. Wilson N, Peace J, Edwards R, Weerasekera D. Smokers commonly misperceive that nicotine is a major carcinogen: National survey data. Thorax. 2011;66:353-4.
33. Mooney ME, Leventhal AM, Hatsukami DK. Attitudes and knowledge about nicotine and nicotine replacement therapy. Nicotine & Tobacco Research. 2006;8:435-46.
34. Glover M, Scragg R, Nosa V, et al. Keeping Kids Smokefree: rationale, design, and implementation of a community, school, and family-based intervention to modify behaviors related to smoking among Maori and Pacific Island children in New Zealand. International Quarterly of Community Health Education. 2009;30:205-22.
35. Centres for Disease Control and Prevention. Epi Info. 3.5.1 ed. Atlanta: Centres for Disease Control and Prevention; 2008.

36. Statistical Standard for Ethnicity 2005. Statistics New Zealand, 2005. URL: http://www.stats.govt.nz/reports/analytical-reports/review-measurement-of-ethnicity.aspx
37. De Zwart KM, Sellman JD. Public knowledge and attitudes regarding smoking and smoking cessation treatments. New Zealand Medical Journal. 2002;115:219-22.
38. Etter J-F, Perneger TV. Attitudes toward nicotine replacement therapy in smokers and ex-smokers in the general public. Clinical Pharmacology & Therapeutics. 2001;69:175-83.
39. The Quit Group. The Quit Group: Quit Service Client Analysis Report – July 2008 – June 2009. Wellington: The Quit Group; 2009.
40. Public Health Commission. Tobacco Products: The Public Health Commission's Advice to the Minister of Health. Wellington: Public Health Commission; 1994.
41. Glover M, Nosa V, Watson D, Paynter J. WhyKwit: A Qualitative Study of What Motivates Maori, Pacific Island and Low Socio-Economic Peoples in Aotearoa/New Zealand to Stop Smoking. Auckland: Centre for Tobacco Control Research; 2010. http://www.fmhs.auckland.ac.nz/soph/depts/sch/atc/_docs/WhyKwit%20Report.pdf
42. Dessaix A, Murphy M, Perez D, et al. Quitting is easy, not smoking is hard: research with smokers of low socio-economic status. Incite: Journal of the Cancer Institute NSW. 2009.
43. The Quit Group, Ministry of Health. Maori Smoking and Tobacco Use 2009. Wellington: Ministry of Health; 2009.
44. Dixon L, Aimer P, Fletcher L, et al. Smokefree Outcomes with Midwife Lead Maternity Carers: An analysis of smoking during pregnancy from the New Zealand College of Midwives midwifery database information 2004-2007. New Zealand College of Midwives. 2009;40.
45. Laugesen M. Snuffing out cigarette sales and the smoking deaths epidemic. N Z Med J. 2007;120:U2587. Review
46. Ministry of Health. Implementing the ABC approach for Smoking Cessation: Framework and work programme. Wellington: Ministry of Health; 2009.
47. West R, Sohal T. "Catastrophic" pathways to smoking cessation: findings from national survey. BMJ. 2006;332:458-60.
48. Glover M, Cowie N. Increasing delivery of smoking cessation treatments to Maori and Pacific smokers. New Zealand Medical Journal. 2010;123:125.
49. Kotz D, Stapleton JA, Owen L, West R. How cost-effective is 'No Smoking Day'? Tobacco Control. 2011;20:302-4.
50. Mercer D. 268,000 pledge to quit smoking for 'Stoptober' The Independent 2012. http://www.independent.co.uk/life-style/health-and-families/health-news/268000-pledge-to-quit-smoking-for-stoptober-8229016.html
51. Glover M, Paynter J, Bullen C, Kristensen K. Supporting pregnant women to quit smoking: postal survey of New Zealand general practioners and midwives' smoking cessation knowledge and practices. New Zealand Medical Journal. 2008;121:53-64.
52. Wong G, Fishman Z, McRobbie H, et al. Smoking and Nurses in New Zealand. ASH-KAN Aotearoa: Assessment of smoking history, knowledge and attitudes of nurses in New Zealand. Auckland: ASH, New Zealand; 2007. http://www.ash.org.nz/site_resources/library/Research_commisoned_by_ASH/ASH_KAN_Aotearoa_Smoking_and_nurses_in_New_Zealand.pdf
53. Ministry of Health. Health Targets 2009/10. Wellington: Ministry of Health; 2009.

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Assessing the value for money of pharmaceuticals in New Zealand—PHARMAC's approach to cost-utility analysis

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Abstract

Cost-utility analysis (CUA) is a form of economic analysis that has been used by PHARMAC for nearly 20 years. It is also used by many health funding and assessment agencies internationally. So what is CUA and why is it so important?

This article describes the process involved in undertaking CUA, including critical appraisal of clinical evidence; transforming the evidence to estimate quality-adjusted life years (QALYs); estimating costs; and how this information is combined to obtain an output that can be used to inform decision-making. The article also describes how PHARMAC uses CUA to prioritise pharmaceuticals for funding in New Zealand.

PHARMAC, the Pharmaceutical Management Agency, is a Government agency that decides which pharmaceuticals will be subsidised in New Zealand. These decisions are made using nine decision criteria;[1] one of these criteria is cost-effectiveness.

The relative cost-effectiveness of a pharmaceutical is assessed using cost-utility analysis (CUA). PHARMAC has been undertaking CUA to inform decision-making for nearly 20 years.[2–9] CUA is also used by many health funding and assessment agencies internationally.[10–12]

Relevance of cost-utility analysis to clinical practice

The basic principle of economic analysis, including cost-utility analysis, is that choices need to be made between alternative uses of resources, as resources (e.g. money, personnel, time, etc.) will always be insufficient to support all possible activities. Therefore, by choosing to use resources one way, we forgo other opportunities to use the same resources. Such decisions are made by consumers and health professionals daily, be it as simple as deciding how much time to allocate to each patient, or whether to go out for dinner versus cooking at home. All options are associated with various costs (financial and non-financial) and benefits that need to be considered.

With pharmaceuticals, demand will always exceed our ability to pay, therefore choices are inevitable. CUA provides decision makers with information on the health gains and costs associated with various funding options, so informed decisions can be made. These analyses are based on technical clinical and economic information (as detailed in this article), and PHARMAC staff often seek further advice from clinicians when undertaking economic analysis. This includes advice on the relevance of empirical evidence to the New Zealand population; expected long-term outcomes (in cases where trials end too early); and quality of life impacts. The understanding of the components of CUA helps clinicians provide the specialist advice that PHARMAC

requires when modelling the benefits and costs of a pharmaceutical. Further, through the understanding of the inputs of a CUA, clinicians can better understand how the results of a CUA are generated, and why certain treatments may be considered less cost-effective than others.

So what exactly is CUA and why is it so important?

Cost-utility analysis

CUA is a form of economic analysis that quantitatively assesses the health outcomes and costs of a proposed treatment compared with an alternative treatment (often current clinical practice).[13,14]

Another form of economic analysis that is well-known is cost-effectiveness analysis. CUA differs from cost-effectiveness analysis in the way that health outcomes are measured. With cost-effectiveness analysis, outcomes are measured in common units, such as life years saved or myocardial infarctions prevented. However, as the outcome measures are diverse, it is difficult to compare the cost-effectiveness of treatments for different health conditions.

With CUA, health outcomes are measured using a common currency, usually quality-adjusted life years (QALYs). QALYs take into account changes in patients' health-related quality of life as well as duration of survival. This enables comparison between the cost-effectiveness of interventions that treat different conditions.

This article describes how CUA is undertaken at PHARMAC (as outlined in the recently updated Prescription for Pharmacoeconomic Analysis[8,9]). Note that different approaches to CUA may be used by other organisations. PHARMAC has undertaken extensive consultation on the CUA methodology used (Endnote 1), and will continue to review these methods.

Researching and critiquing clinical evidence

Before a CUA is undertaken, there needs to be evidence of net clinical benefit (i.e. that benefits exceed harms), and evidence of relative clinical benefit (i.e. that the treatment is more effective compared with current clinical practice).

Funding applications to PHARMAC generally include some evidence of clinical effectiveness. PHARMAC staff review the applications and ensure that all key evidence is included. In most cases this information is provided to the Pharmacology and Therapeutics Advisory Committee (PTAC) for advice on the pharmaceuticals and their benefits (Endnote 2).

When reviewing the clinical evidence, PHARMAC recommends that well-conducted randomised controlled trials (RCTs) and meta-analyses are the preferred data sources. In the absence of valid RCTs, it is recommended that evidence from the highest available level of study design should be considered with reference to the limitations of the study design.

Evidence is critically appraised, using frameworks such as the Graphic Appraisal Tool for Epidemiology (GATE),[15,16] and assessment undertaken on the applicability of the trial to the New Zealand health sector. The following table (Table 1) outlines key factors to consider when critically appraising a clinical trial.

Table 1. Key factors to consider in critical appraisal of clinical trials

Internal validity—How reliable are the trial results?	
Availability of data	Were all available trial data used? Were there quality controls (e.g. was the trial published in a peer-reviewed journal)?
Number of patients	Was the sample size large enough to rule out effects due to chance (i.e. false negatives and false positives)? Was the effect large enough to be statistically significant even in a small sample size?
Method of randomisation, including adequate concealment	Was there likely to be any selection bias or confounding? Was there adequate reporting of appropriate randomisation and how this was kept concealed? Were patients, clinicians and assessors blinded?
Length and completeness of follow-up	Were patients followed for an adequate time period? How often were patients assessed? Was analysis by intention-to-treat (including drop-outs and deaths)?
Selection of endpoints	Were the endpoint/outcome measures relevant?
External validity—How relevant are the trial results?	
Patient population	Was the patient population in the trial similar to those considered for funding?
Comparator	Was the comparator consistent with current clinical practice in New Zealand?
Dose, formulation and administration regimen	Were these consistent with recommended treatment regimes in New Zealand?

Throughout the article a theoretical worked example is provided to illustrate how a CUA can be constructed and how the results are generated. Each section of this article that details how to undertake CUA will refer to this worked example, building on information in the previous section, with the results of the CUA generated towards the end of the article.

Worked example: A new pharmaceutical is available for advanced bowel cancer (Treatment A). There is already a treatment funded and used widely for advanced bowel cancer (Treatment B). Patients require treatment for 6 months.

On reviewing the clinical evidence, it is established that there has been one randomised controlled trial that has assessed the effectiveness of Treatment A compared with Treatment B for treating advanced bowel cancer. Critical appraisal of the trial indicates that it is of high quality.

Transforming the clinical evidence to estimate QALYs

Economic analysis has two distinct phases:

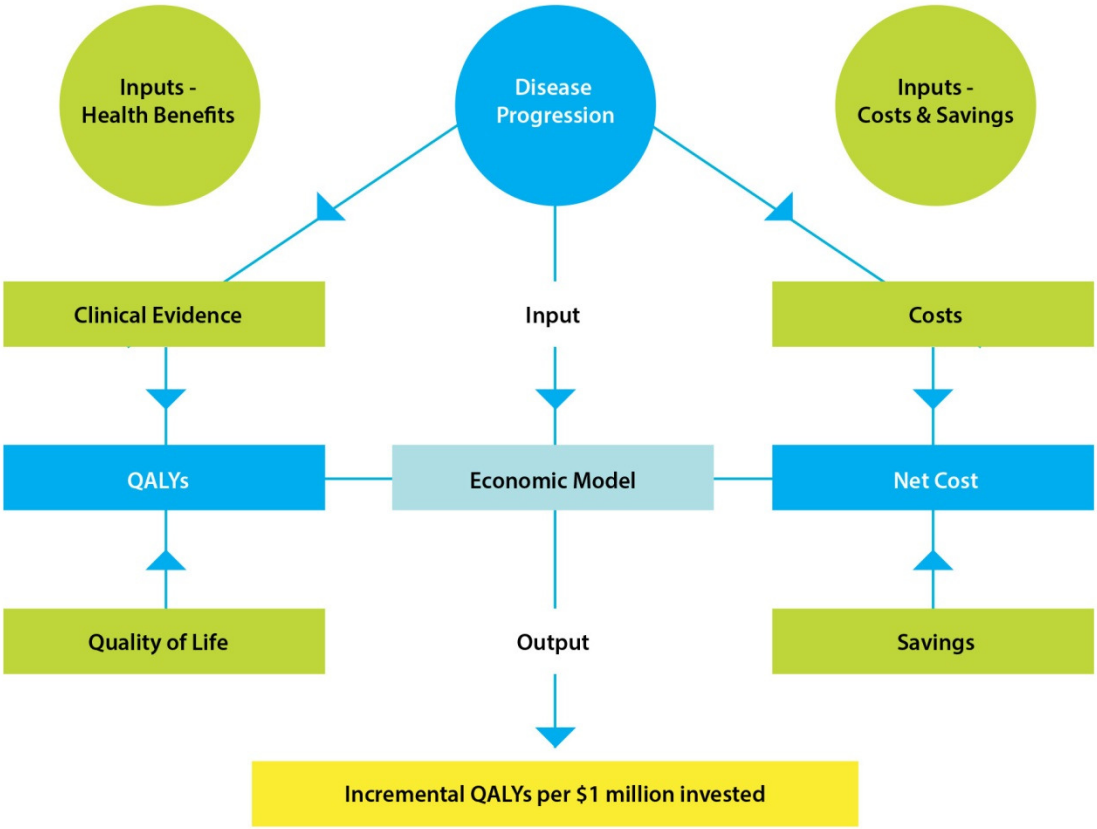
- Gathering evidence; and
- Processing evidence to estimate the effectiveness and relative cost-effectiveness of the pharmaceutical for the proposed indication(s) in the New Zealand clinical setting.

The second phase involves developing economic models that combine information on:

- Natural disease progression;
- Clinical effectiveness of the pharmaceuticals (usually obtained from clinical trial data);
- Health-related quality of life; and
- Health sector costs and savings (including those incurred in hospitals).

This is illustrated in the diagram below (Figure 1).

Figure 1. Economic model inputs and output

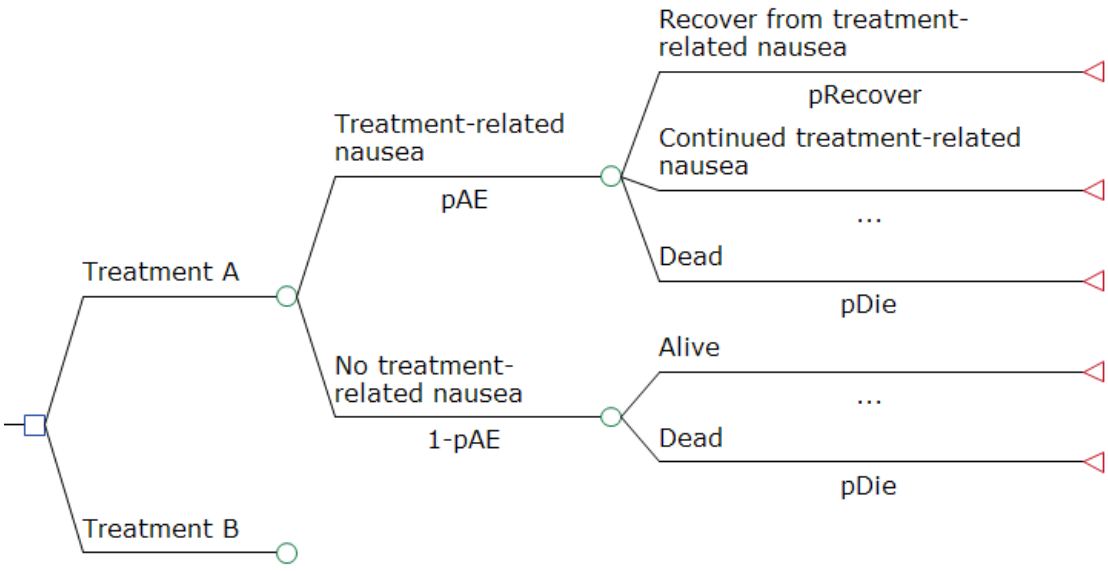


Economic models usually consist of a series of branches, representing the expected health outcomes of treatments. When constructing a model the first task is to define the disease in terms of different states that represent clinically important events in the disease process. Transition probabilities are then assigned for movement between these states.[17,18] These probabilities are often based on data from clinical trials, for example the probability of response to treatment, probability of an adverse event, etc.

PHARMAC models often take a lifetime horizon (based on the age and gender-specific life-expectancy of the target population) in order to capture the longer term effects of the disease and proposed treatment. However, in some cases the duration of a trial may be too short to show the full impact of treatment. It may therefore be necessary to extrapolate the trial data, based on assumptions regarding the expected long-term outcomes.[19–21] For example, many of the trials for smoking cessation treatments evaluated quit rates at 1 year, even though longer-term quit rate data is more relevant. Assumptions therefore need to be made regarding the proportion of patients who would relapse after 1 year.

Assumptions are unavoidable in CUA, however these are made as robust as possible. PHARMAC staff always test the analysis to determine how sensitive the results are to key inputs.

A model is constructed, as illustrated below:



Worked example: The clinical trial assessing the benefit of the proposed treatment for advanced bowel cancer (Treatment A), compared with current treatment (Treatment B), reports the following:

- Treatment A extends survival by approximately 2 months (average survival of 12 months) compared with Treatment B (average survival of 10 months);
- Patients administered Treatment A had lower rates of treatment-related nausea and vomiting compared with patients administered Treatment B.

Estimating benefits—quality-adjusted life years

Health outcomes in CUA are measured in a common unit—quality-adjusted life years (QALYs). QALYs measure the effect of changes in life expectancy and health-related quality of life that result from treatment. QALYs are the most widely used measure for integrating the effect of treatment on survival and health-related quality of life; however they have been subject to some debate (Endnote 3).[22–34]

Health-related quality of life—The health-related quality of life component of the QALY takes into account a range of factors that affect people's quality of life, such as ability to undertake and enjoy leisure activities, freedom from pain, and ability to work and be independent. Health-related quality of life is included in a CUA in the form of a utility value. A utility represents the strength of preference or desirability for a specific level of health status or outcome.

Substantial empirical data are available on the preferences people place on various combinations of factors affecting quality of life. These data have been obtained through various methods, including large surveys that ask participants to rate their health according to the dimensions of the instrument used. Various instruments are available.[35]

In the New Zealand setting, PHARMAC estimates the health-related quality of life (or utility) associated with a health state using the EuroQol 5D (EQ-5D) instrument. This particular tool consists of five dimensions (mobility, self-care, usual activities, pain/discomfort, and anxiety/depression) and three levels (no problems, some problems and extreme problems). In order to derive utility values that represent New Zealand population preferences, a survey was undertaken of 3000 randomly selected New Zealanders, and was completed by 1360 people.[36]

The authors of the survey undertook regression analysis, and produced 245 unique utility values, each representing the net aggregate impact of physical, emotional, and social functioning on quality of life. These utility values range from 1 (perfect health) to 0 (death), and also include negative values (states considered to be worse than death). Such values can be applied to a range of diseases and conditions.

Using the EQ-5D, PHARMAC obtains utility values through a process of mapping descriptions of the symptoms patients experience in the health state to the relevant generic health states in the EQ-5D. The utility value generated is then validated through published literature and/or expert clinical advice.

Quality-adjusted life years—Once information is obtained on the impact of the disease on health-related quality of life, and this information is used to obtain utility values, the QALYs can then be estimated. QALYs are calculated by multiplying the duration of time spent in a health state by the health-related quality of life weight (i.e. utility score) associated with that health state. Under the QALY framework, one QALY is equivalent to living one year in perfect health, or two years at half of perfect health, and so on.

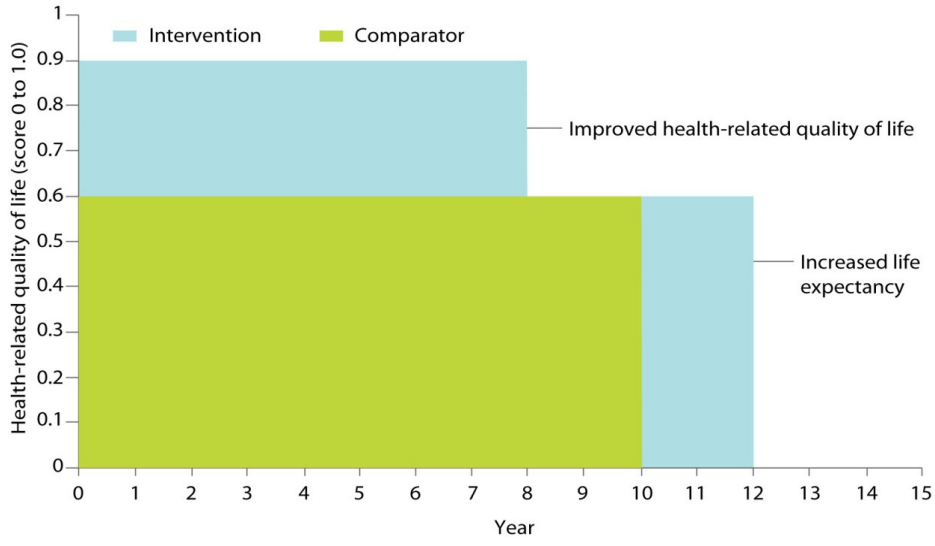
In the example below (Figure 2), life expectancy (the number of years left before death) is 6.00. Quality-adjusted life expectancy (the number of QALYs left before death) is 4.75. This is calculated by multiplying each life year by the average quality

of life experienced in that year [(4×1)+(1×0.5)+(1×0.25)]. This is equivalent to the area under the curve.

Figure 2. Schematic of quality-adjusted life expectancy

The diagram below (Figure 3) illustrates how a theoretical intervention may gain QALYs through both improving patient quality of life and life extension. Without treatment the QALYs are estimated to be 6.00 (life expectancy of 10 years with a quality of life of 0.6). However with the intervention life-expectancy is extended by 2 years, and quality of life improved to 0.9 for 8 years, and then reduces to 0.6 for remaining 4 years. The estimated QALYs are 9.60 [(8×0.9)+(4×0.6)]. Therefore the intervention results in a gain of 3.60 QALYs (9.60−6.00).

Figure 3. Schematic of QALY gains



Worked example: Clinical evidence indicates that patients administered Treatment B (current treatment) have higher rates of treatment-related nausea and vomiting, impacting their quality of life. Using the EQ-5D, it is assumed that patients administered Treatment B have a health-related quality of life of 0.4, and patients administered Treatment A (proposed treatment) have a health-related quality of life of 0.6 (on a scale of 0–1).

As outlined previously, patients administered Treatment A have an average survival of 12 months, and patients administered Treatment B have an average survival of 10 months. The QALY for patients administered Treatment A is therefore estimated to be 0.60 (1×0.6), compared with 0.33 (10/12×0.4) for patients administered Treatment B.

The additional QALY gain of Treatment A compared with Treatment B is therefore estimated to be 0.27 (0.60–0.33).

Estimating costs

PHARMAC CUAs are undertaken from the perspective of the funder (with regards to PHARMAC's decision criteria[1]). PHARMAC CUAs therefore aim to include all relevant costs (and savings) to the heath sector resulting from funding a treatment.[5] This includes:

- Pharmaceutical costs;
- Hospital costs;
- Outpatient costs (for example, laboratory tests, specialist visits, etc.); and
- Direct patient healthcare costs that Government partially subsidises (for example, General Practitioner visits and residential care). The cost included in the CUA is the cost to the Government plus the additional cost to the patient.

CUAs undertaken by PHARMAC therefore include more than just pharmaceutical costs, and take into account any financial impact a treatment may have on the healthcare sector.[5] For example, a CUA on a new oral treatment to replace an infusion will take into account all potential cost-offsets to the health sector associated with patients no longer requiring an infusion.

PHARMAC does not however include the cost-offsets from inability to work, as this is taken into account when estimating QALYs (from reduced health-related quality of life). In addition, valuing lost income would likely bias against treatment that benefit those who do not earn an income (for example, the elderly and children).

Worked example: The total cost per patient of 6 months' treatment with Treatment A is $5500, compared with a cost of $900 per patient for Treatment B. In addition, Treatment B is an infusion administered at a hospital outpatient unit, at a cost of approximately $1500 per patient. Treatment A is an oral treatment.

The evidence indicates that 10% of patients in the clinical trial needed to be hospitalised due to severe nausea and vomiting with Treatment B, compared with none (0%) of the patients taking Treatment A. The cost per hospitalisation is estimated to be $2500. The total cost of Treatment A is therefore $5500, compared with a total cost of Treatment B of $2650 [$900+$1500+$250 ($2500×0.1)].

The additional cost of Treatment A compared with Treatment B is therefore estimated to be $2850 ($5500–$2650).

Putting it all together—the output of CUA

At PHARMAC the results of CUA are expressed as the difference in QALYs gained divided by the difference in costs between treatments. This provides us with information on the amount of additional health benefit that would be gained as a result of the additional expenditure, and is expressed as the QALYs gained per dollar spent.[9] This is shown in the following formula:

$$\text{QALYs gained per \$ spent} = \frac{\text{(QALYs of intervention) – (QALYs of alternative), discounted by year}}{\text{(costs of intervention) – (costs of alternative), discounted by year}}$$

This result can then be multiplied by $1 million to provide information on QALYs gained per million dollars spent, which is a more useful way of expressing the results due to the scale of the investments and values involved.

Another way of expressing the CUA result is the more commonly used 'cost per QALY' (i.e. the additional cost of treatment per QALY gained). For example, a treatment may have a cost per QALY of $10,000, indicating that the treatment costs an additional $10,000 to gain 1 QALY (which is the same as 100 QALYs gained per $1 million).

Note that cost per QALY and QALYs gained per $1 million are the same information, but presented in a different way. PHARMAC uses the measure QALYs gained per $1 million to emphasise the additional health gain for the investment.[37–41]

All future costs and benefits are discounted to the present value[42] using the current annual discount rate of 3.5% (Endnote 4).[43]

The inputs and assumptions in CUAs are always tested to determine how sensitive the results are to various assumptions, such as any uncertainty in long-term benefits of treatment (beyond the period of the trials).

Worked example: As outlined in the QALY section, the additional QALY gain of Treatment A compared with Treatment B is 0.27. The additional cost of funding Treatment A is $2850 (refer to the cost section).

The QALYs gained per $1 million are therefore the additional QALYs divided by the additional cost, multiplied by 1 million (0.27/$2850×$1M). This gives a result of about 95 QALYs gained per $1 million spent.

Therefore, for every million dollars of the total health budget invested in the new medicine (Treatment A), an additional 95 units of benefit (QALYs) would be gained. This result can also be presented as a 'cost per QALY' (additional net cost divided by QALY gain), giving a result of approximately $10,600 (i.e. costing the overall health sector $10,600 for each QALY gained).

Use of CUA in decision-making

PHARMAC uses CUA to compare the cost-effectiveness of a pharmaceutical with other pharmaceuticals that could be funded instead. As a proposal to invest in a pharmaceutical can only be considered 'cost-effective' in comparison with another proposal, PHARMAC does not have a cost-effectiveness threshold (or pre-determined

QALYs per million invested amount) that indicates whether or not a pharmaceutical is 'cost-effective'. Also, cost-effectiveness is only one of nine decision criteria used by PHARMAC.[5]

A proposal may be more cost-effective than another but rate poorly on other decision criteria and, therefore, may not be funded. In addition, what is considered to be cost-effective varies with the amount of funding available. This is not just in terms of the total budget each year, but also the available budget anticipated in the future.[37,41]

Once sufficient information on a proposal is available (such as PTAC priority and cost-effectiveness), this information is compiled and considered by PHARMAC according to its nine decision criteria[1] (outlined in Table 2 below).

Table 2. PHARMAC's decision criteria

PHARMAC's decision criteria are:

- The health needs of all eligible people within New Zealand.
- The particular needs of Māori and Pacific peoples.
- The availability and suitability of existing medicines, therapeutic medical devices and related products and related things.
- The clinical benefits and risks of pharmaceuticals.
- The cost-effectiveness of meeting health needs by funding pharmaceuticals, rather than by using other publicly-funded health and disability support services.
- The budgetary impact (in terms of the pharmaceutical budget and the Government's overall health budget) of any changes to the Pharmaceutical Schedule.
- The direct cost to health service users.
- The Government's priorities for health funding, as set out in any objectives notified by the Crown to PHARMAC, in PHARMAC's Funding Agreement, or elsewhere.
- Any other criteria that PHARMAC thinks are relevant. PHARMAC will carry out the necessary consultation whenever we intend to take any 'other criteria' into account.

Pharmaceuticals awaiting funding consideration are prioritised against other expenditure options (either listing of new pharmaceuticals or expanding access to existing pharmaceuticals), with the overall aim of identifying those proposals that would provide the best health outcomes if funded. PHARMAC conducts regular prioritisation reviews of all active proposals.

In summary, cost-utility analysis provides valuable information on the amount of additional QALYs (or health benefits) that can be gained from the budget available. This information can be used to inform prioritisation decisions. It is a particularly useful tool for organisations working within a constrained budget who are seeking to identify proposals that offer the most health gains relative to their cost.

Competing interests: The authors are PHARMAC staff. PHARMAC is currently reviewing its Operating Policies and Procedures, including its decision criteria. More information about the review can be found at http://www.pharmac.health.nz/about/operating-policies-and-procedures/decision-criteria-consultation

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Endnotes:

1. For further information on PHARMAC's consideration of consultation responses to the PFPA version 2, please refer to the following webpages: http://www.pharmac.health.nz/ckeditor_assets/attachments/12/consultation-responses.pdf
2. http://www.pharmac.health.nz/ckeditor_assets/attachments/14/details-key-amendments.pdf
3. PTAC is PHARMAC's primary clinical advisory committee, and there are also a number of PTAC subcommittees, made up of experts in specialist clinical fields such as cardiology and oncology. PTAC makes recommendations to PHARMAC for the assignment of high, medium, or low priorities for proposals; or that a proposal be declined or referred to a subcommittee. PTAC uses the same decision criteria as PHARMAC[1] when evaluating pharmaceuticals.
4. Despite the advantages of using a single indicator to measure effectiveness, QALYs have been debated on ethical and operational grounds.
5. A key criticism is that QALYs assume uniform preferences (i.e. each QALY has equal value regardless to whom it accrues). This criticism is based on the results of CUAs often being applied within a utilitarian framework. However, CUA is capable of being applied to achieve the desired distribution of QALYs through attaching weights to the estimated QALY gains.
6. Some argue that QALY calculations bias against elderly due to their shortened life expectancy resulting in fewer QALY gains. However empirical evidence concerning the public's view on this issue indicates that most would support the favouring of younger people, entailed by the QALY approach. Also note that it is the marginal differences in QALYs which count as benefits, not the average length of survival.[22-24]
7. Discounting is used to compare treatments that have costs and benefits that occur at different times. The extent to which future benefits and costs are discounted in comparison with the present is reflected in the discount rate. As the discount rate increases, future benefits and costs become less important when compared with benefits and costs occurring in the present.[42] PHARMAC's current discount rate of 3.5% is based on the five-year average real risk-free long-term government bond rate for New Zealand.

References:

1. PHARMAC. Operating policies and procedures of the Pharmaceutical Management Agency ("PHARMAC"), Third Edition, January 2006. http://www.pharmac.govt.nz/2005/12/22/231205.pdf
2. Bennett W, McNee W, Metcalfe S, Wright JM. Use of statins In New Zealand, subsidy of statins is limited to particular groups of patients. BMJ 1997;315:161. http://www.bmj.com/content/315/7122/1615
3. Braae R, McNee W, Moore D. Managing pharmaceutical expenditure while increasing access. The pharmaceutical management agency (PHARMAC) experience. Pharmacoeconomics. 1999 Dec;16(6):649-60.
4. Moodie P, Metcalfe S, McNee W. Response from PHARMAC: difficult choices. N Z Med J. 2003;116:U361. http://journal.nzma.org.nz/journal/116-1170/361/

5. Metcalfe S, Dougherty S, Brougham M, Moodie P. PHARMAC measures savings elsewhere to the health sector. N Z Med J. 2003;116:U362. http://journal.nzma.org.nz/journal/116-1170/362/
6. A prescription for Pharmacoeconomic Analysis: methods for cost-utility analysis, Version 1.1 PHARMAC: Wellington, New Zealand, 2004. http://www.pharmac.health.nz/ckeditor_assets/attachments/6/pfpa-v-1-1.pdf
7. Prescription for Pharmacoeconomic Analysis: methods for cost-utility analysis, Version 2 PHARMAC: Wellington, New Zealand, 2007. http://www.pharmac.health.nz/ckeditor_assets/attachments/7/pfpa-2_0.pdf
8. Prescription for Pharmacoeconomic Analysis: methods for cost-utility analysis, Version 2.1 PHARMAC: Wellington, New Zealand, 2012. http://www.pharmac.govt.nz/2012/06/26/PFPAFinal.pdf
9. Grocott R, Metcalfe S. PHARMAC's updated guidelines for cost-utility analyses, with new QALYs per $1M metric. N Z Med J. 2012;125:U5274. http://journal.nzma.org.nz/journal/125-1358/5274/
10. National Institute of Health and Clinical Excellence. Guide to the Methods of Technology Appraisal. London: NICE, 2008. http://www.nice.org.uk/media/B52/A7/TAMethodsGuideUpdatedJune2008.pdf
11. Canadian Agency for Drugs and Technologies in Health. Guidelines for the Economic Evaluation of Health Technologies. 3rd edition. Ottawa: CADTH, 2006. http://www.cadth.ca/media/pdf/186_EconomicGuidelines_e.pdf
12. Pharmaceutical Benefits Advisory Committee. Guidelines for Preparing Submissions to the Pharmaceutical Benefits Advisory Committee, version 4.3. Canberra: PBAC, 2008. http://www.health.gov.au/internet/main/publishing.nsf/Content/pbacguidelines-index
13. Drummond MF, Sculpher MJ, Torrance GW, O'Brien B, Stoddart GL. Methods for the Economic Evaluation of Health Care Programmes. 3rd ed. Oxford: Oxford University Press, 2005.
14. Siegel JE, Torrance GW, Russell LB, Luce BR, Weinstein MC, Gold MR. Guidelines for pharmacoeconomic studies. Recommendations from the panel on cost effectiveness in health and medicine. Panel on Cost Effectiveness in Health and Medicine. Pharmacoeconomics. 1997;11:159-68.
15. Jackson R, Ameratunga S, Broad J, Connor J, Lethaby A, et al. The GATE frame: critical appraisal with pictures. Evid Based Med. 2006;11:35-8.
16. Evidence-Based Practice and Critical Appraisal (updated 4 February 2011). Effective Practice, Informatics & Quality Improvement (EPIQ), at Epidemiology and Biostatistics, School of Population Health, Faculty of Medical and Health Sciences, University of Auckland. http://www.fmhs.auckland.ac.nz/soph/depts/epi/epiq/ebp.aspx
17. Sonnenberg FA, Beck JR. Markov models in medical decision making: a practical guide. Med Decis Making. 1993 Oct-Dec;13(4):322-38.
18. Briggs A, Sculpher M. An introduction to Markov modelling for economic evaluation. Pharmacoeconomics. 1998 Apr;13(4):397-409.
19. Brennan A, Akehurst R. Modelling in health economic evaluation. What is its place? What is its value? Pharmacoeconomics. 2000 May;17(5):445-59.
20. Weinstein MC. Recent developments in decision-analytic modelling for economic evaluation. Pharmacoeconomics. 2006;24(11):1043-53.
21. Soto J. Health economic evaluations using decision analytic modeling. Principles and practices--utilization of a checklist to their development and appraisal. Int J Technol Assess Health Care. 2002 Winter;18(1):94-111.
22. Kawachi I, Bethwaite P, Bethwaite J. The use of quality-adjusted life years (QALYs) in the economic appraisal of health care. N Z Med J. 1990 Feb 14;103(883):46-8.
23. Schwartz S, Richardson J, Glasziou PP. Quality-adjusted life years: origins, measurements, applications, objections. Aust J Public Health. 1993 Sep;17(3):272-8.

24. Prieto L, Sacristán JA. Problems and solutions in calculating quality-adjusted life years (QALYs). Health Qual Life Outcomes. 2003 Dec 19;1:80.
25. Schwappach DL. Resource allocation, social values and the QALY: a review of the debate and empirical evidence. Health Expect 2002;5:210-22.
26. Wagstaff A. QALYs and the equity-efficiency trade-off. J Health Econ 1991;10(1):21-41. Erratum in: J Health Econ 1993 Jul;12(2):237.
27. Dolan P, Shaw R, Tsuchiya A, Williams A. QALY maximisation and people's preferences: a methodological review of the literature. Health Econ. 2005 Feb;14(2):197-208. http://onlinelibrary.wiley.com/doi/10.1002/hec.924/pdf
28. Singer P, McKie J, Kuhse H, Richardson J. Double jeopardy and the use of QALYs in health care allocation. J Med Ethics. 1995;21:144-50. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1376689/pdf/jmedeth00296-0016.pdf
29. Harris J. Double jeopardy and the veil of ignorance--a reply. J Med Ethics. 1995;21:151-7. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1376690/pdf/jmedeth00296-0023.pdf
30. McKie J, Kuhse H, Richardson J, Singer P. Double jeopardy, the equal value of lives and the veil of ignorance: a rejoinder to Harris. J Med Ethics. 1996 Aug;22(4):204-8. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1376998/pdf/jmedeth00303-0014.pdf
31. Smith MD, Drummond M, Brixner D. Moving the QALY forward: rationale for change. Value Health 2009;12 Suppl 1:S1-4. http://www.ispor.org/meetings/invitational/QALY/Paper1.pdf
32. Weinstein MC, Torrance G, McGuire A. QALYs: the basics. Value Health 2009;12 Suppl 1:S5-9. Erratum in: Value Health. 2010 Dec;13(8):1065. http://www.ispor.org/meetings/invitational/QALY/Paper2revised.PDF
33. Nord E, Daniels N, Kamlet M. QALYs: some challenges. Value Health 2009;12 Suppl 1:S10-5. http://www.ispor.org/meetings/invitational/QALY/Paper3.pdf
34. Lipscomb J, Drummond M, Fryback D, Gold M, Revicki D. Retaining, and enhancing, the QALY. Value Health 2009;12 Suppl 1:S18-26.
35. Nord E. A review of synthetic health indicators. Background paper prepared for the OECD Directorate for Education, Employment, Labour, and Social Affairs, June 1997.
36. Devlin NJ, Hansen P, Kind P, Williams AH. Logical inconsistencies in survey respondents' health state valuations – a methodological challenge for estimating social tariffs. Health Economics. 2003; 12:529-44.
37. Metcalfe S, Rodgers A, Werner R, Schousboe C. PHARMAC has no cost-effectiveness threshold. N Z Med J. 2012;125:99-101. http://journal.nzma.org.nz/journal/125-1350/5083/
38. Craig BA, Black MA. Incremental cost-effectiveness ratio and incremental net-health benefit: two sides of the same coin. Expert Rev Pharmacoecon Outcomes Res. 2001;1:37-46. http://www.expert-reviews.com/doi/pdf/10.1586/14737167.1.1.37
39. Zethraeus N, Johannesson M, Jönsson B, Löthgren M, Tambour M. Advantages of using the net-benefit approach for analysing uncertainty in economic evaluation studies. Pharmacoeconomics. 2003;21:39-48. http://adisonline.com/pharmacoeconomics/Abstract/2003/21010/Advantages_of_Using_the_Net_Benefit_Approach_for.3.aspx
40. Metcalfe S, Grocott R. Comments on "Simoens, S. Health economic assessment: a methodological primer. Int. J. Environ. Res. Public Health 2009, 6,2950-2966"—New Zealand in fact has no cost-effectiveness threshold. Int J Environ Res Public Health. 2010;7:1831-4. http://www.mdpi.com/1660-4601/7/4/1831/
41. Grocott R. Applying Programme Budgeting Marginal Analysis in the health sector: 12 years of experience. Exp Rev Pharmacoecon Outcomes Res 2009;9:181-7 http://www.expert-reviews.com/doi/abs/10.1586/erp.09.2
42. West RR, McNabb R, Thompson AG, Sheldon TA, Grimley Evans J. Estimating implied rates of discount in healthcare decision-making. Health Technol Assess. 2003;7(38):1-60.

43. Grocott R, Metcalfe S. PHARMAC's updated guidelines for cost-effectiveness analyses, with new discount rate. N Z Med J. 2007;120:U2641. http://journal.nzma.org.nz/journal/120-1258/2641/

THE NEW ZEALAND MEDICAL JOURNAL
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Negative pressure dressing around the airway

James J Johnston, Felix Mariano, David Vokes

Abstract

Background Negative pressure wound therapy (NPWT) is an effective modality in most areas of the body and is associated with more rapid healing. However the use of negative pressure remains a challenge in managing complex wounds of the head and neck region.

Methods and Results We present the case of a patient with a laryngectomy stoma and an adjacent defect. This was successfully closed in ten days using a novel dressing system comprising Aekin cohesive circular dressings, a Shiley cuffed non-fenestrated size 6.0 tracheostomy tube, and a NPWT device.

Conclusions Until now NPWT dressings in the head and neck region have been limited by wounds that develop around a laryngectomy stoma or tracheostomy site. We have described the successful use of a negative pressure dressing around the airway owing to the combined use of a tracheosotomy tube and the appropriate dressing.

Negative pressure wound therapy (NPWT) is effective in most areas of the body and is associated with more rapid healing.[1]

Wound care as a medical concept has a long history dating back as far as 2100 BC, and various methods have been described: from the use of wine and water to cleanse wounds, all the way through to surgical debridement or amputation of the affected area.[2] Many advances have been made in the field of wound care in recent times, probably none more significant than the introduction of asepsis.

Modern wound dressings are designed to optimise wound healing through reducing bacterial contamination, absorbing wound discharge, and preventing transfer of infectious substance into the wound.[3] With the resultant decreased incidence of wound site infection, wound care innovation has skyrocketed.

Negative pressure as a means of assisting wound healing was first documented in 1993 by Fleischman et al.[4] There is much evidence to support the use of NPWT dressings in most areas of the body, however there are very few reports describing its use in the head and neck region.[2]

NPWT as a system is controlled by a computer that is able to generate and maintain negative pressure. It does so via a tube attached to a foam and drape dressing. Foam is placed into the wound and an adhesive drape placed over the top to create an air tight seal. Continuous negative pressure is generated at the wound site and the wound is reviewed every three days.[5]

This is the first report of the use of NPWT dressing over and around the airway in a patient with a laryngectomy stoma and adjacent pharyngocutaneous fistula.

Case report

The subject of this report is an 80-year-old male who underwent total laryngectomy, total thyroidectomy and bilateral selective neck dissections for a locally advanced squamous cell carcinoma of the left glottis (Stage T4aN0M0). He developed an early pharyngeal leak, that was initially treated with a pectoralis major muscle flap and a salivary bypass tube. Despite this saliva continued to leak from the pharynx and a pharyngocutaneous fistula developed.

A second pectoralis major flap successfully closed the fistula. However after this surgery the superior skin flap pulled away from the stoma, leaving moderate sized cavity adjacent to the stoma. See Figure 1a and Figure 1b.

Closure of this cavity was initially attempted using NPWT by inserting a size 8 endotracheal tube to provide an airway, and then placing the NPWT dressing over the tube. See Figure 2. However his configuration leaked air from under the NPWT dressing film and was not tolerated by the patient secondary to discomfort. This was removed and an Aekin cohesive circular dressing was placed over the stoma site, and then a Shiley cuffed non-fenestrated size 6.0 tracheostomy tube was inserted into the trachea to provide an airway.

A second Aekin cohesive circular dressing was placed over the tracheostomy tube with the NPWT sponge in the defect cavity and NPWT dressing film overlying. See Figure 3. NPWT machine was set on continuous negative pressure at 125mmHg medium intensity. No air leak was noted and this dressing was tolerated well by the patient.

Figure 1a. Laryngectomy stoma with adjacent defect

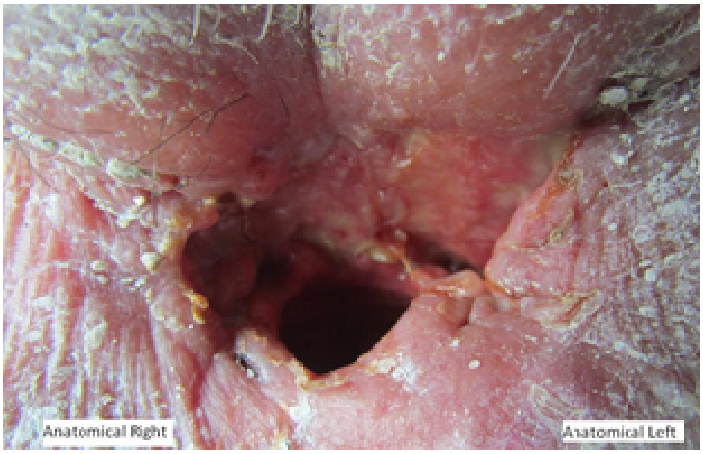


Figure 1b. Laryngectomy stoma with adjacent defect

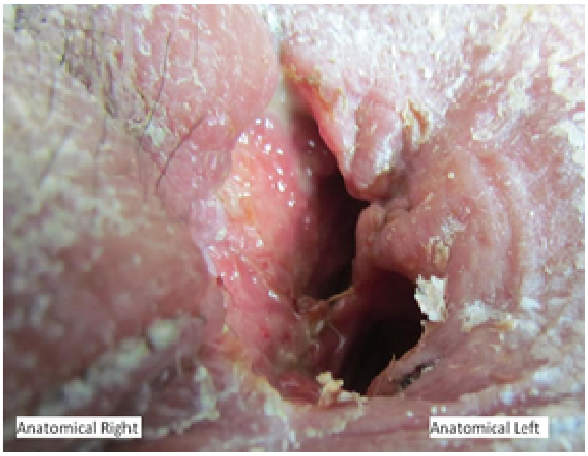
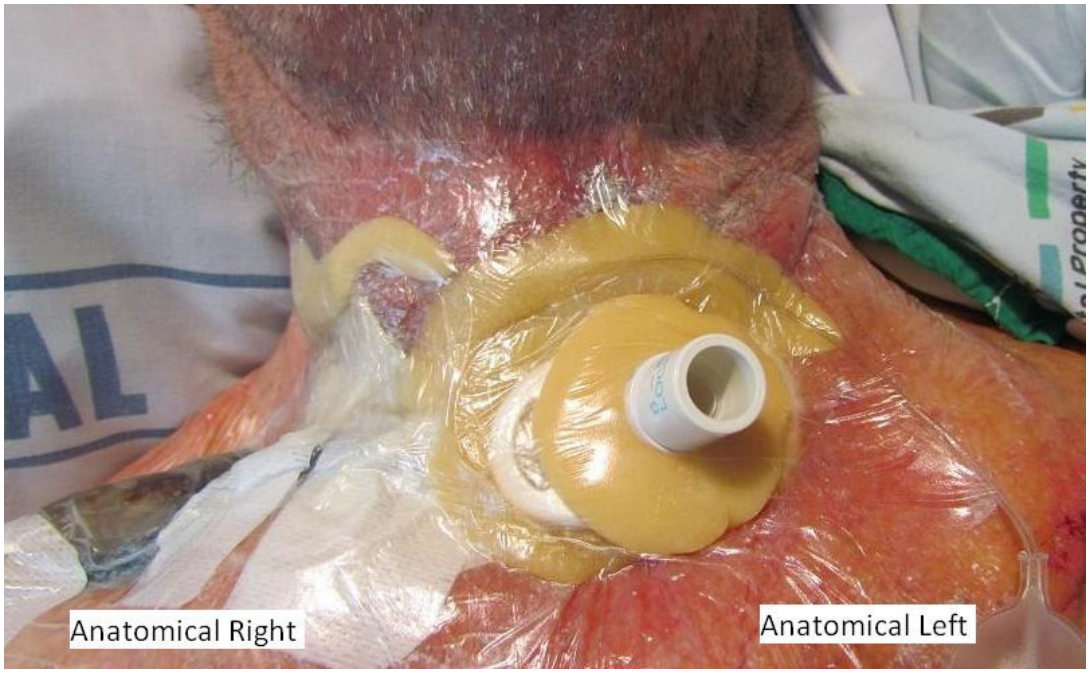


Figure 2. First attempt at cavity closure using NPWT by inserting a size 8 endotracheal tube to provide an airway, and then placing the NPWT dressing over the tube

Figure 3. Second attempt at cavity closure using Aekin cohesive circular dressing placed over the stoma site, then a Shiley cuffed non-fenestrated size 6.0 tracheostomy tube inserted into the trachea to provide an airway. A second Aekin cohesive circular dressing placed over the tracheostomy tube with the NPWT sponge in the defect cavity and NPWT dressing film overlying.



After day 3 the dressing was removed and granulation tissue was noted. See Figure 4. A NPWT dressing was reapplied and removed after day 6 where further granulation tissue was noted and only a small defect was seen with neck extension. See Figure 5.

Figure 4. Day 3 post NPWT application—the dressing was removed and granulation tissue was noted

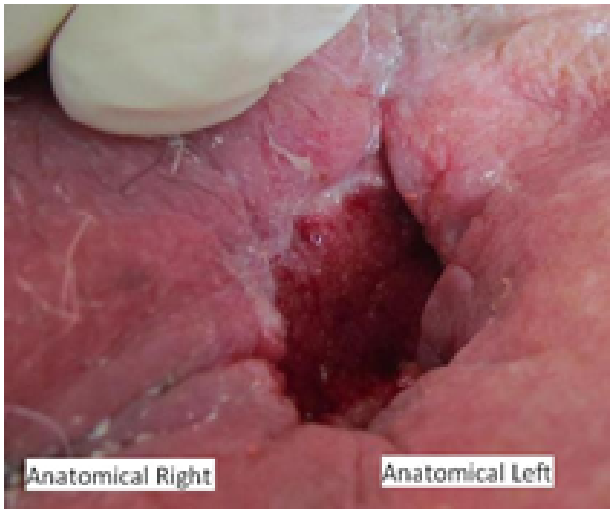
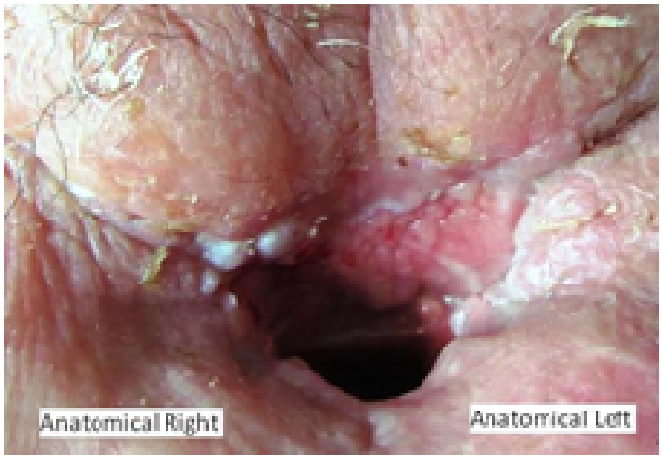
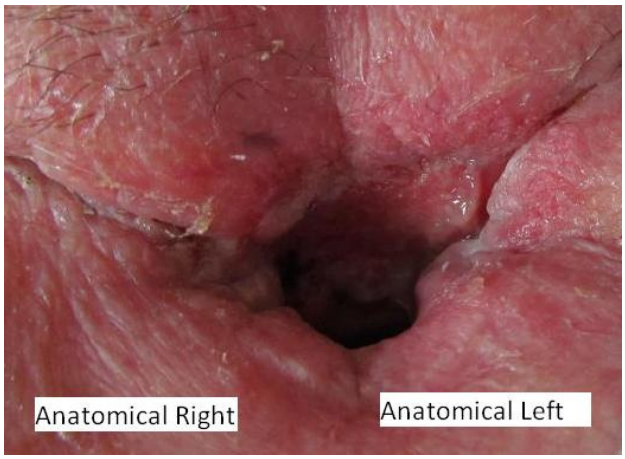


Figure 5. Day 6 post NPWT application—further granulation tissue was noted and only a small defect was seen with neck extension



At this point two vertical mattress sutures were placed to close the residual defect. A NPWT dressing was once again reapplied and removed on day ten where the cavity was noted to be completely healed. See Figure 6.

Figure 6. Day 10 post NPWT application—the cavity was noted to be completely healed



Prior to NPWT dressing closure of the defect patient had a gastrograffin swallow that demonstrated a small leak from the pharynx. Following closure of the defect the gastrograffin swallow was repeated and appearances were improved. After the second barium swallow the patient was commenced on an oral diet successfully.

Discussion

Negative pressure as means to assist open wound closure is a method first described in 1993 by Fleischman et al.[4] This concept was later expanded by Argenta et al[6] who developed the NPWT device in 1997. This device uses continuous or intermittent negative pressure through a foam layer that is applied directly to the wound surface.

It is postulated that negative pressure dressings have four main effects on the wound surface that may assist with wound healing. These are as follows: the prevention of infection, mechanical stress placed on cells at the wound surface, an increase in local blood flow, and the removal of factors that inhibit wound healing.[2]

As outlined by Dhir et al[2], there are advantages to the NPWT dressing system. These include the need to change the dressing only once every three days, thus decreasing the need for multiple daily dressing changes. Healing occurs more rapidly and the enhanced epithelialisation decreases the wound size and often allows the defect to be closed by primary measures.[2]

Conclusions

Until now NPWT dressings in the head and neck region have been limited by wounds that develop around a laryngectomy stoma or tracheostomy site. This is secondary to the inability of achieving an adequate air tight seal over and around the airway. As a result air leaks occur and the patient may experience discomfort. We have described the successful use of a negative pressure dressing around the airway owing to the combined use of a tracheosotomy tube and the appropriate dressing.

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References:

1. Chiummariello S, Guarro G, Pica A, Alfano C. Evaluation of negative pressure vacuum-assisted system in acute and chronic wounds closure: our experience. G Chir. 2012 Oct;33(10):358-62.
2. Dhir K, Reino AJ, Lipana J. Vacuum-assisted closure therapy in the management of head and neck wounds. Laryngoscope. 2009;119:54-61.
3. Triller C, Huljev D, Smrke DM. Application of modern wound dressings in the treatment of chronic wounds. Acta Med Croatica. 2012 Oct;66 Suppl 1:65-70.
4. Fleischmann W, Strecker W, Bombelli M, et al. Vacuum sealing as a treatment of soft tissue damage in open fractures. Unfallchirurg. 1993;96:488-92.
5. Benech A, Arcuri F, Poglio G, et al. Vacuum-assisted closure therapy in reconstructive surgery. Acta Otorhinolaryngol Ital. 2012 June;32(3):192–7.
6. Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. Ann Plast Surg 1997;38:563-77.

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An unusual cause of pleural effusion

Shwan Karim, Ya-Shu Chang

Systemic amyloidosis is uncommon and can affect different organs in different patients. Pleural effusion and pericardial effusion due to systemic amyloidosis are rare. To date, only a few reports have been reported in the literature.

We report an interesting case of systemic amyloidosis causing heavy proteinuria, pleural effusion and pericardial effusion.

Case report

A previously well 72-year-old lady presented with a 10-day history of worsening dyspnoea, chest tightness and palpitation.

She was afebrile on presentation. Heart rate was 130 beats per minute and the ECG showed atrial fibrillation. Her respiratory rate was 30 breaths per minute. Heart sounds were dual with no added sounds.

Chest examination revealed dull percussion notes up to mid zones with absent breath sounds consistent with bilateral pleural effusions. She was clinically in right heart failure with raised jugular venous pressure and significant peripheral oedema.

A chest X-ray showed newly developed large bilateral pleural effusions and significantly increased cardiothoracic ratio comparing to her previous chest X-rays. Serum albumin was low at 21 g/L.

Echocardiogram showed large pericardial effusion, maximum dimension 3.6 cm anterolateral to left ventricle. There was evidence of diastolic compromise of right ventricle. Her heart rate and symptom of dyspnoea improved following pericardiocentesis.

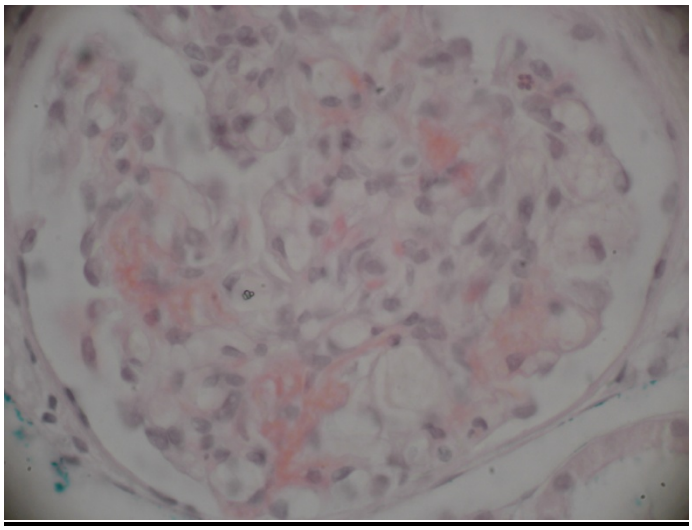
The pleural fluid was drained but quickly re-accumulated. The fluid was transudate according to Light's criteria: Pleural LDH 126 U/L (serum LDH 306 U/L), protein 24 g/L (serum protein 63 g/L). Urine sample showed heavy proteinuria (1.5 g/L [0–0.15 g/L]).

Renal biopsy showed deposition of fibrillary material measuring 9 to 13 nanometers in thickness that were eosinophilic in nature and stained positive with Congo red consistent with amyloidosis (Figure 1).

Supplementary serology showed IgG lambda band (5.6 g/L) and high lambda light chains levels. The diagnosis was AL amyloidosis and she was promptly started on chemotherapy (bortezomib [Valcade], cyclophosphamide and dexamethasone).

She responded well to the treatment with no further hospital admission. Her serum protein electrophoresis normalised. No recurrence of pericardial effusion since commencement of chemotherapy. She had had no recurrence of pleural effusion in the first 8 months following chemotherapy. She developed small pleural effusion subsequently but responded to oral frusemide.

Figure 1. Renal biopsy showing positive Congo red staining



Discussion

Amyloidosis is a systemic illness with extracellular deposition of protein fibrils of low molecular weight, insoluble and β pleated. Two commonest forms are AL (light chain) and AA (reactive) or secondary amyloidosis.

AL amyloidosis is the most common and severe form. It is a plasma cell dyscrasia with a detectable monoclonal immunoglobulin in the serum or monoclonal light chains in the urine. This could be due to any haematological disorder including monoclonal gammopathy of unknown significance, Waldenstrom's macroglobulinemia and multiple myeloma.

The most commonly affected organs are kidneys, lungs and heart. It commonly presents with heavy proteinuria, nephrotic syndrome, heart failure, pleura effusions, oedema and organomegaly.

Hypoalbuminaemia and nephrotic range proteinuria are traditionally believed to be contributing to formation of pleural effusion. A retrospective study[2] of 636 AL patients, published by Berk's Editorial in 2003, found nephrotic range proteinuria and hypoalbuminaemia in combination with restrictive AL cardiomyopathy does not induce pleural effusions.

It is believed that pleural amyloid infiltration plays a central role in persistent pleural effusions. The amyloid impairs pleural lymphatic drainage system and probably promotes pleural fluid secretion.[2]

Biopsy is paramount for diagnosis and can be obtained from affected organs, such as kidney and commonly abdominal fat pad aspirate for the ease of access and the high sensitivity and specificity.[4]

Electron microscopy and post-staining with Congo red show green birefringence under polarised light. Further characterisation of biopsies is vital to distinguish between AL and AA amyloidosis using immunohistochemical staining (looking for Kappa or lambda suggesting AL variant).

The management of amyloidosis is mainly divided into haematopoietic cell transplantation or chemotherapy. A chemotherapy approach for a patient unfit for transplant, using mephalan and dexamethasone, is generally recommended although in recent years changes towards other regimens have been implemented including the use of bortezomib, cyclophosphamide, lenalidomide and thalidomide.[5,6]

Cyclophosphamide-bortezomib-dexamethasone (CyBorD) produces a rapid and complete haematological response in most AL amyloidosis patients but the use of this regimen warrants further trials.[7]

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References:

1. Rafii R, Leslie K, Heo J, Chan A. A 71 year old woman with an unusual cause for pleural effusions. Chest. 2011;139(5):1237-41.
2. Berk JL, Keane J, Seldin DC, et al. Persisten pleural effusions in primary systemic amyloidosis: etiology and prognosis. Chest. 2003 Sep;124(3):969-77.
3. Toyama K, Oka H, Obata K, Ogawa H. Primary systemic amyloidosis with bloody pericardial effusion. Internal Medicine. 2009;48:821-6.
4. Gameren II V, Hazenberg BP, Bijzet J, Van Rijswijk MH. Diagnostic accuracy of subcutaneous abdominal fat tissue aspiration for detecting systemic amyloidosis and its utility in clinical practice. Arthritis Rheum. 2006 Jun;54 (6):2015-21.
5. Desport E, Bridoux F, Sirac C, et al. AL amyloidosis. Orphanet Journal of Rare Diseases. 2012 Aug 21;7(1):54.
6. Qiu ZX, Wang MJ, Wang LH, et al. Clinical investigation of primary amyloidosis with autologus hematopoietic stem cell transplantation. Zhonghua Xue Ye Xue Za Zhi. 2012 Mar;33(3):187-90.
7. Mikhael JR, Schuster SR, Jimenez-Zepeda VH, et al. Cyclophosphamide-bortezomib-dexamethasone (CyBorD) produces rapid and complete response in patients with AL amyloidosis. Blood 2012 May 10;119(19):4391-4.

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A puzzling lady with persistent wheeze and pulmonary nodules

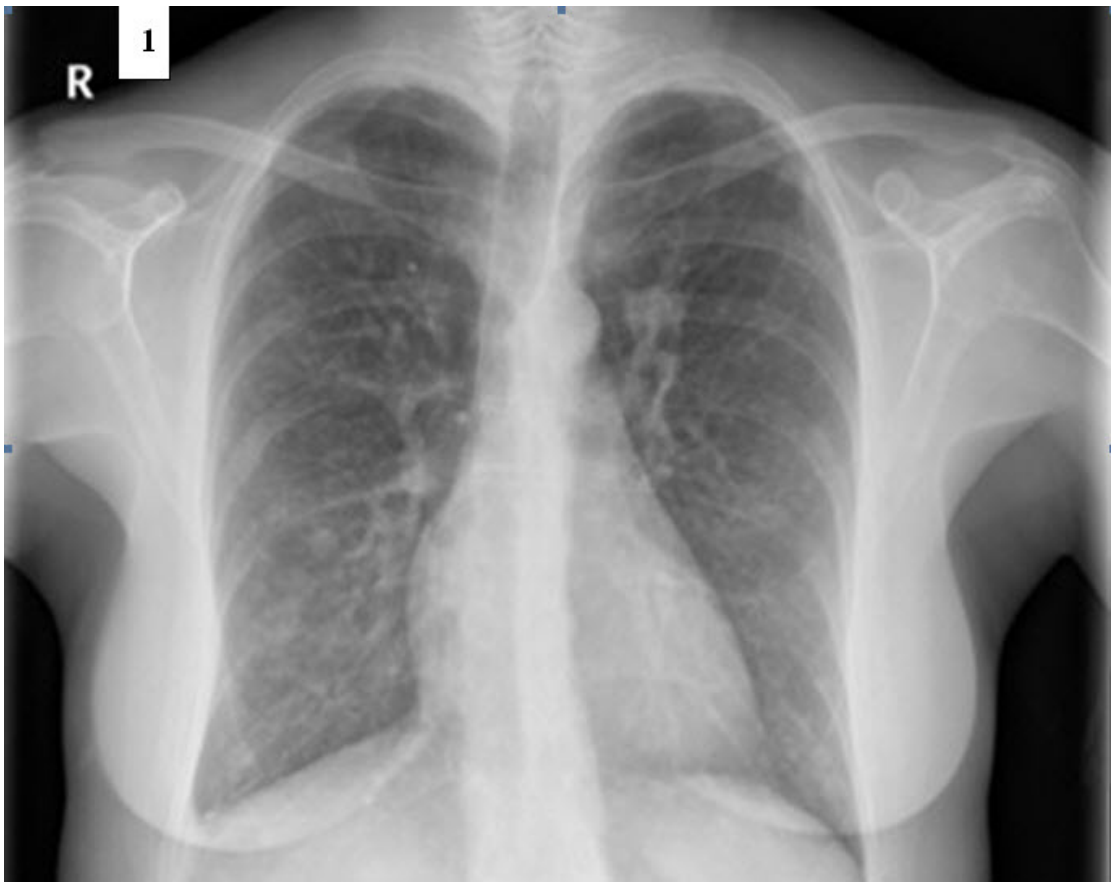
Akshay Dwarakanath, Arunesh Kumar

Clinical—A 35-year-old lady with a background history of cough variant asthma, presented with a history of increasing breathlessness, cough and persistent wheeze.

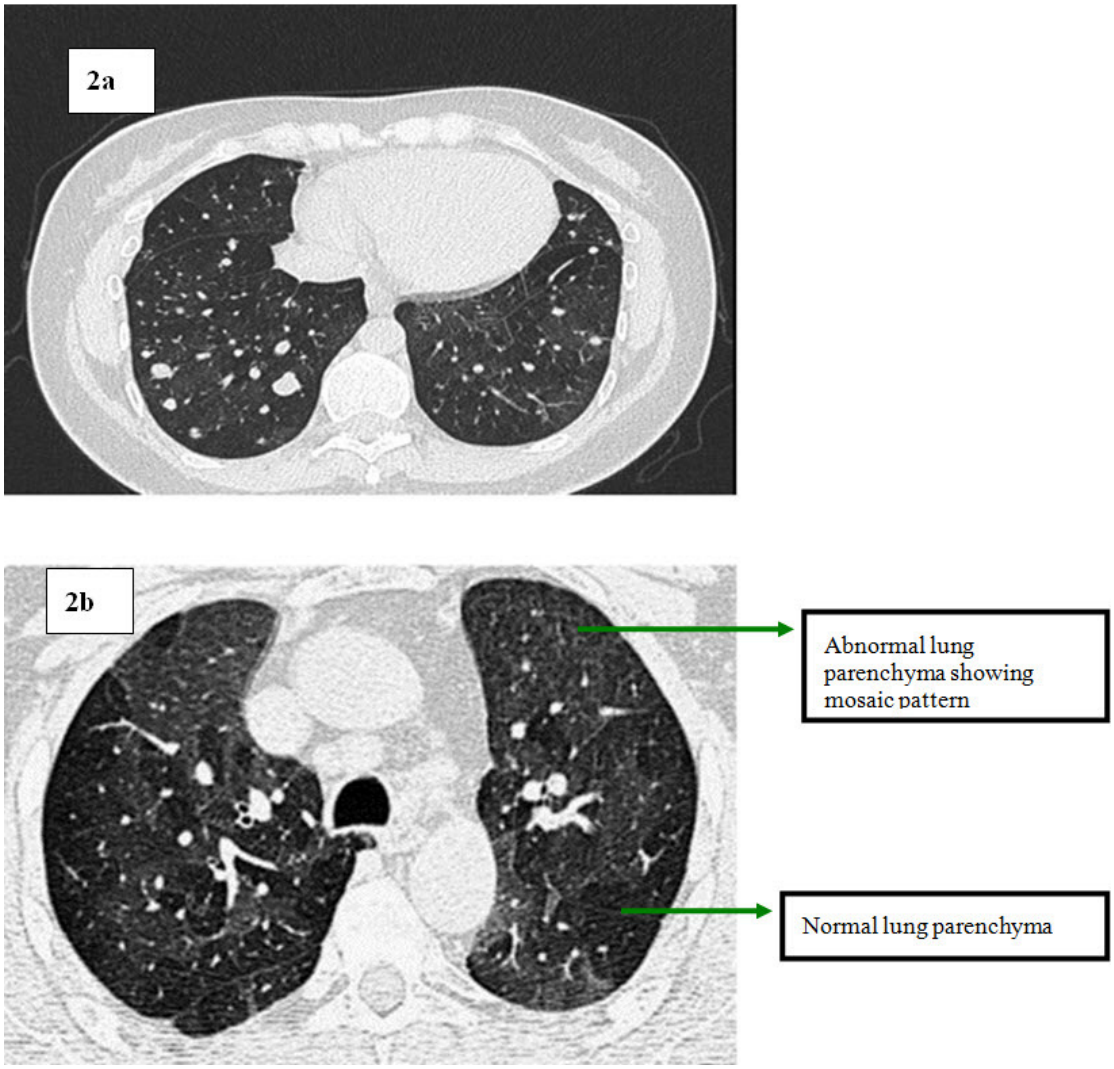
Examination revealed bilateral polyphonic wheeze. Pulmonary function tests showed evidence of predominant obstructive defect with no significant reversibility on bronchodilator testing.

Her chest X-ray (Figure 1) and high resolution computed tomography (HRCT) are shown below (Figures 2a & 2b).

Figure 1. Chest X-ray showing hyperinflation and multiple nodules throughout both lungs. No areas of confluent consolidation, pleural effusion or collapse. Normal mediastinal and cardiac contours

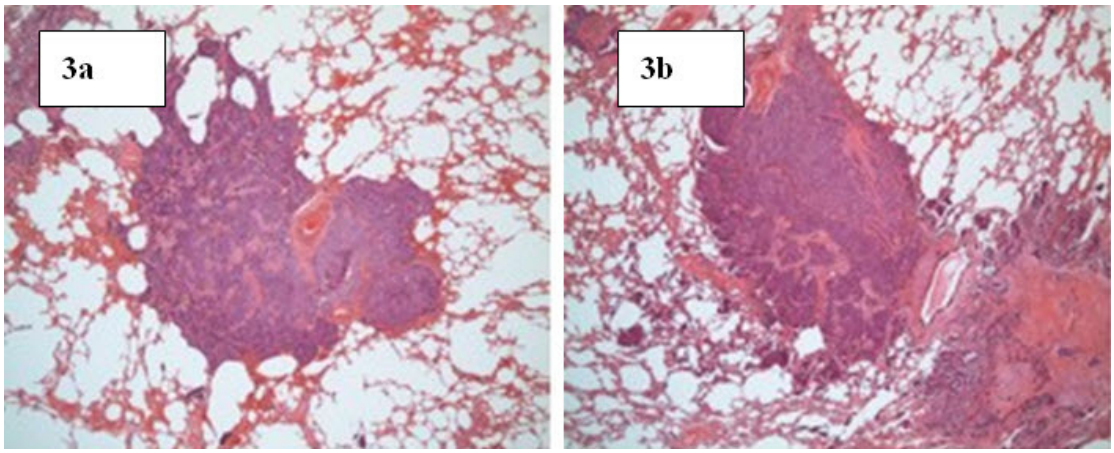


Figures 2a & 2b. A high-resolution computed tomography scan showing multiple bilateral subcentrimetric pulmonary nodules and mosaic pattern of different attenuation



A VATS (video assisted thoracoscopic surgery) lung biopsy (Figures 3a & 3b) showed evidence of multiple tumourlets with neuro-endocrine cell hyperplasia associated with early peribronchial fibrosis that was highly suggestive of diffuse idiopathic pulmonary neuro-endocrine cell hyperplasia (DIPNECH) lung.

Figures 3a & 3b. VATS lung biopsy showing neuro-endocrine cells. These are dark blue nodules, which have a uniform appearance. They have a granular chromatin with small nucleoli and small amounts of cytoplasm, the largest measuring up to 4mm maximally. They are forming small rosettes and are present *in situ*, growing along the bronchioles and forming small nests within a fibrous stroma



Discussion—Diffuse idiopathic pulmonary neuro-endocrine cell hyperplasia (DIPNECH) is a rare disorder first described in the early 1950s,[1] but was not fully recognised and named until 1992.[2] It is considered as a preneoplastic lesion of carcinoid.[3]

Two different modes of clinical presentation are reported. The first is with symptomatic disease, typically non-productive cough and dyspnoea, which are not necessarily progressive, and an obstructive lung function profile.

A misdiagnosis of asthma or COPD is common with no response seen to inhaled corticosteroids or bronchodilators. The second mode of presentation is through surgical referral, typically for resection of a pulmonary nodule or nodules that have been found incidentally on imaging performed for reasons unrelated to DIPNECH. The airflow obstruction has an indolent course, but very rarely progress with severe obliterative bronchiolitis.[4]

Chest X-ray images may be normal or show solitary or multiple pulmonary nodules. Nodules are usually seen on chest CT. Associated features include airway dilatation, bronchial wall thickening, air trapping, mosaicism and atelectasis.[4] Bronchoalveloar lavage will show evidence of lymphocytosis in symptomatic patients.[4] Tissue biopsy is required to establish the diagnosis.

The treatment options are limited, surgical excision of lung carcinoids and Lung transplantation can be considered in severe disease.[5]

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Acknowledgement: We thank our patient for giving the consent.

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References:

1. Felton WL, Liebow AA, Lindskog GE. Peripheral and multiple bronchial adenomas. Cancer 1953;6:555–66.
2. Aguayo SM, Miller YE, Waldron JA Jr, et al. Idiopathic diffuse hyperplasia of pulmonary neuroendocrine cells and airways disease. N Engl J Med 1992;327:1285–8.
3. Chong S, Lee KS, Chung MJ, et al. Neuro-endocrine tumors of the lung: clinical, pathologic, and imaging findings. Radiographics 2006;26:41-57.
4. Davies SJ, Gosney JR, Hansell DM, et al. Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia: an under-recognised spectrum of disease. Thorax 2007;62:248–52.
5. Sheerin N, Harrison NK, Sheppard MN, et al. Obliterative bronchiolitis caused by multiple tumourlets and microcarcinoids successfully treated by single lung transplantation. Thorax. 1995;50:207-209.

THE NEW ZEALAND MEDICAL JOURNAL
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An unexpected finding in a patient with cough

Victoria Mayoral Campos, Claudia Bonnet Carrón, Beatriz Carro Alonso, Cristina Puebla Macarrón, José Luis Benito Arévalo

Clinical presentation—A 28-year-old male with a 5-month history of productive cough and weight lost. He was smoker of 10–15 cigarettes a day. There was no fever or haemoptysis. On examination, the patient had a normal respiratory rate, normal oxygen saturation, and temperature and auscultation of heart, lungs, and abdomen was normal. Morphological, biochemical and hormonal blood parameters did not show any significant abnormality.

A chest radiograph was obtained at presentation (Figure 1). A thoracic computed-tomography (CT) scan (Figure 2) was also obtained. *What is the diagnosis?*

Figure 1. Chest radiograph

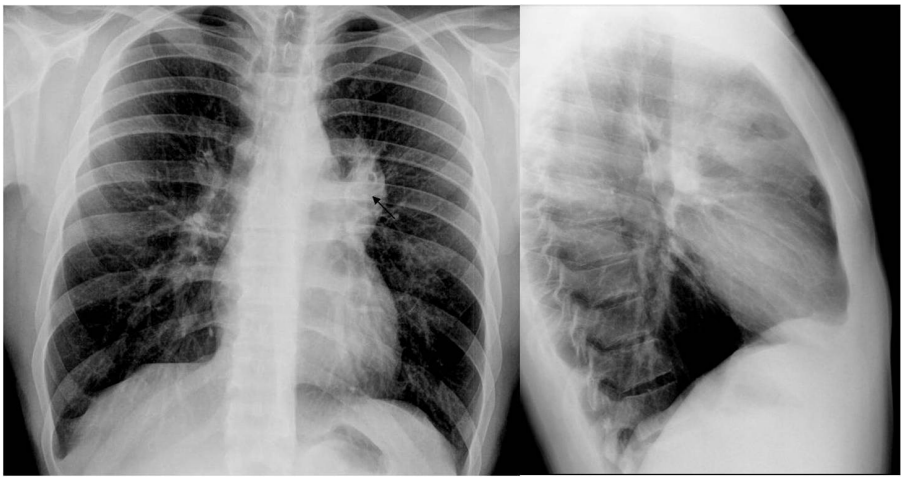
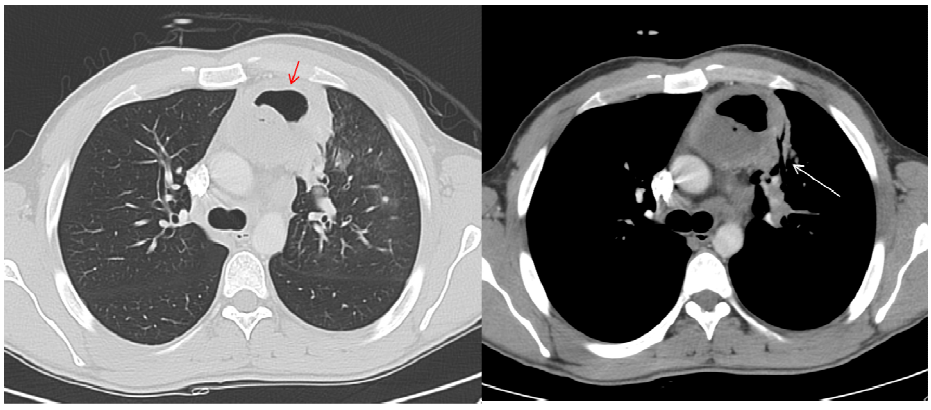


Figure 2. Images from a CT scan



Answer—The Chest radiograph demonstrates a well circumscribed mass, with smooth contour and with central cavitation, which has the radiological characteristics of an *anterior mediastinal mass*.

The CT scan confirmed the radiographic findings of anterior mediastinal mass with air collection inside (short arrow), adenopathies and the clinical suspicion of mediastinal lymphoma. The mass extends to the adjacent lung parenchyma and produce the broncogram sign (long arrow).

Discussion—Anterior mediastinal masses can be identified when the hilum overlay sign is present and the posterior mediastinal lines are preserved.[1] The hilum overlay sign is present when the normal hilar structures project through a mass, such that the mass can be understood as being either anterior or posterior to the hilum (arrow Figure 1).

The four principal anterior mediastinal masses are thymoma, lymphoma, germ cell tumours or retrosternal goitre. The thyroid gland is intimately related to the trachea so enlargement may disrupt the middle and posterior mediastinal lines.

Primary mediastinal lymphoma is a rare entity comprising only 10% of lymphomas in the mediastinum.[2] It usually has a bimodal distribution of incidence, peaking in young adulthood and again after age 50 years. It can have the same appearance as any other type of anterior mediastinal mass on plain films.[3] It can have central cavitation but this finding is extremely rare. The mass can extends to the adjacent lung parenchyma.

The mediastinoscopy with biopsy confirmed the diagnosis of Hodgkin's lymphoma, mixed cellularity type, stage IVB, because there was lung extension.

The patient was treated with six cycles of ABVD, and posterior radiotherapy. There was improvement in symptoms and significant resolution of the mediastinal mass on follow-up CT scan.

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References:

1. Whitten CR, Khan S, Munneke GJ, Grubnic S. A diagnostic approach to mediastinal abnormalities. Radiographics. 2007;27:657-71.
2. Duwe BV, Sterman DH, Musani AI. Tumors of the mediastinum. Chest. 2005;128:2893-909.
3. Brown LR, Aughenbaugh GL. Masses of the anterior mediastinum: CT and MR imaging. AJR Am J Roentgenol. 1991;157:1171-80.

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Debate over tobacco and film ratings should be evidence-based

Paynter and Chapman's letter to the editor[1] criticising Maubach et al's[2] editorial proposing that films with smoking be given an adult content rating because of the strong scientific consensus that smoking in movies causes youth to smoke, misquotes our paper[3] on the amount of smoking in youth-rated films in the United States (US).

In 2010, among all top-grossing films youth-rated in the US (G, PG, PG-13), 69.3% were tobacco-free, not 88.2%, as Paynter and Chapman state. They also ignore the fact that in New Zealand (and Australia), the level of exposure to smoking onscreen in youth-rated films is much higher than in the US. They also ignore the fact that since 2010 there has been a substantial rebound in the amount of smoking in youth-rated films in the US.

Readers considering Paynter and Chapman's arguments should consider the care with which the quote the relevant scientific evidence.

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References:

1. Payner J, Chapman S. Is censorship of films a useful solution to the problem of covert tobacco advertising? New Zealand Medical Journal. 2013;126(1376). http://journal.nzma.org.nz/journal/126-1376/5692/
2. Maubach N, Hoek J, Edwards R, et al. Smoking in children's films—covert tobacco advertising causing smoking uptake or much ado about nothing? New Zealand Medical Journal 2013;126(1375). http://journal.nzma.org.nz/journal/126-1375/5666
3. Glantz SA, Mitchell S, Titus K, Polansky JR, Kaufmann RB, Bauer UE. Smoking in Top-Grossing Movies – United States. MMWR 2010;60(27);909-913
4. Polansky J, Titus K, Lanning N, Glantz S. Smoking in top-grossing US movies, 2012. UCSF Center for Tobacco Control Research and Education http://escholarship.org/uc/item/3j69r912

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The 'moral flabbiness' of compulsory apologies

In a recently published Health and Disability Commissioner (HDC) investigation (Case 11HDC00957), a rather unusual recommendation was made.

The case examined the standard of care provided by a midwife who failed to discuss with the parents about Vitamin K administration during the antenatal period, and also failed to perform a PKU test within an appropriate period after birth. The baby was admitted to hospital with neonatal jaundice and later required an urgent craniotomy and evacuation of a subdural haematoma.

The HDC found the midwife in breach of the Code of Rights. The Commissioner required that the midwife "provide a written apology to Mr and Mrs B and a separate apology to Baby B, suitable for her to read when she is sufficiently mature to do so, apologising for Ms A's breaches of the Code. The apology is to be provided to HDC for forwarding by 24 June 2013."[1]

The requirement to provide an apology to a baby "for her to read when she is sufficiently mature to do so" is rather unusual. The midwife had already faced a competence review, HDC investigation, and has now been referred by the Commissioner for potential disciplinary proceedings.

She described being "profoundly" affected by the case and has given up independent practice. What words should the midwife find to say sorry in a way that a previously harmed (but now well recovered) child can read at some future date? On top of a recommended apology to the parents, it seems excessive and hollow to recommend that an errant health professional undergo yet another ritual of subjugation.

However, it provides a good opportunity to reflect on the appropriateness of HDC recommending (but in effect requiring, given HDC's policy of publicly naming providers who fail to comply with its recommendations) that a health professional apologise to *any* consumer.

Alfie Kohn, in his book *Unconditional Parenting*, writes

> "A more specific example of everyday behaviourism: Perhaps you've met parents who force their children to apologize after doing something hurtful or mean. ("Can you say you're sorry?") Now, what's going on here? Do the parents assume that making children speak this sentence will magically produce in them the feeling of being sorry, despite all evidence to the contrary? Or, worse, do they not even care whether the child really is sorry, because sincerity is irrelevant and all that matters is the act of uttering the appropriate words? Compulsory apologises mostly train children to say things they don't mean—that is, to lie."[2]

The requirement to provide an apology to harmed or otherwise aggrieved consumers is a common recommendation in HDC investigations. However, it would seem that HDC is acting like the behaviourist parent Kohn describes.

As Lee Taft has passionately argued for over a decade, apologies need to be authentic. Unfortunately, Taft fears we have slipped into 'moral flabbiness', readily dishing out and accepting lame apologies: "Apologies are being conflated. We don't know the distinction between an apology that seeks to repair and an apology that is just a social grace or damage control."[3]

All too often, the apologies that are provided to consumers, to fulfil HDC's requirement, ring hollow and the moral dimension of apology is totally subverted. If a health professional needs to be told to apologise, then there is little point. As Taft argues, "Authentic apology is reserved for the morally courageous who seek for themselves and their patients the deep healing authentic apology inspires."[4]

The HDC should reconsider its current practice of compulsory apologies.

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References:

1. Health and Disability Commissioner. Decision 11HDC00957. http://www.hdc.org.nz/decisions--case-notes/commissioner%27s-decisions/2013/11hdc00957 [accessed 27 June 2013]
2. Kohn A. Unconditional Parenting: Moving from Rewards and Punishments to Love and Reason. New York: Atria Books, 2005.
3. Hall C. Ethics consultant is a master of apologies. Dallas News, 12 June 2010: http://www.dallasnews.com/business/columnists/cheryl-hall/20100612-Ethics-consultant-is-a-master-of-4304.ece [accessed 27 June 2013]
4. Taft L. Apology and Medical Mistake: Opportunity or Foil? Annals Health Law 2005;14:55-94.

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Treatment of the Insane (part 1)

Excerpt of an Editorial published in NZMJ 1913 Dec;12(48):653–6.

It is much to the credit of the legislators and people of this could try that a great deal has been done to succour the weak and the distressed, and, in this respect, we are said to lead the world. This is no doubt very flattering to our self-complacency, although it be granted that in a new country it is comparatively easy to avoid many of the evil and unfair conditions that prevail in more populous old-world communities. New brooms sweep clean, and chimneys collect soot in proportion to the time they have been in use.

In New Zealand, the State is very generous in providing a superabundance of good general hospitals, maternity homes for the working classes, nurses for the country districts, free advice for feeding anybody's babies, medical inspection of school children, pensions for the aged, and a multiplicity of holidays for the tired. Even felons in the gaols are not cut off from the milk of human kindness, and are provided with the luxury of false teeth, if required, for the mastication of substantial fare.

It is indeed a very beautiful and touching sight to see our candidates for parliamentary honours and honorariums, before a political election, almost on the verge of tears in their protestations of earnest desire to help the sick, the unfortunate, and the distressed. We hope it is no very cynical asperity on our part if we point out what is, perhaps, only a coincidence, and reveal the fact that the recipients of all this beneficence are either voters or prospective voters, and are capable of rewarding their benefactors. But there is a pathetic class of people whose woes are fit to wring the heart, and whose very helplessness should be their strength. They cannot tell their wants, for they have been bereft of power to know them, but the State has taken charge of them, and bears a terrible responsibility.

Successive Governments have been warned of the deplorable state of the insane in this country, formerly by Dr. Macgregor, latterly by Dr. Hay, and all that has been done is to temporise. Changing the name of a lunatic asylum to that of mental hospital does not convert a madhouse twenty-five years behind the times into a modern institution for the humane treatment of the mentally afflicted. Blundering and dallying for years with the building of a new asylum most urgently required is not the way to overcome the hideous overcrowding and herding together of the insane.

The poor creatures are like him "Who cometh in with vanity, and departeth in darkness, and his name shall be covered with darkness." It is well-known to many medical men, and if not also to politicians it is inconceivable, that in one at least of the large asylums in New Zealand a new patient is thrust first of all into the refractory ward. This is certain to aggravate his madness. At various times in New Zealand hundreds of the insane have had to sleep lying in corridors and on the floors. Far too many noisy patients are crowded into the one ward and out-Bedlam Bedlam, because the parsimony of the Government will not permit of proper classification. All this goes on, and yet money is voted for objects of no particular urgency or necessity.

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Medical emergencies on commercial airline flights

In this paper from Pittsburgh, USA, the researchers have reviewed records of all calls to a medical communications centre from five domestic and international airlines that represented approximately 10% of the global passenger flight volume from 1 January 2008 through 31 October 2010.

There were 11,920 in-flight medical emergencies resulting in calls to the centre (1 medical emergency per 604 flights). Syncope and presyncope were responsible for just over one-third of the calls. Respiratory symptoms (12.1%) and nausea or vomiting (9.5%) were the next most common. In nearly half (48.1%) of the emergencies, in-flight medical assistance was provided by physician passengers. Follow-up data revealed that about a quarter of the subjects were transported to hospital and 8.6% required admission. Possible stroke, cardiac or respiratory symptoms were the commonest causes for hospital admission.

N Engl J Med 2013;368:2075–83.

Cardiovascular events after clarithromycin use in lower respiratory tract infections

Is the use of clarithromycin in the setting of acute exacerbations of chronic obstructive pulmonary disease (COPD) or community acquired pneumonia associated with excess cardiovascular events?

This question is addressed in this paper which is a report of two prospective cohort studies carried out in the UK. The COPD cohort included 1343 patients and there were 1631 patients in the pneumonia cohort. Comparisons of subsequent cardiovascular events were noted between those patients who were treated with one or more courses of clarithromycin and those who did not have such treatment. The researchers report that at one year follow-up there were more hospital admissions for cardiovascular events in those treated with clarithromycin. The hazard ratio in the COPD cohort was 1.50 and it was 1.68 in the pneumonia cohort.

BMJ 2013;356:f1235.

Computed tomographic colonography versus colonoscopy

Colonoscopy is the gold-standard test for investigation of symptoms suggestive of colorectal cancer but computed tomographic colonoscopy (CTC) is an alternative, less invasive test.

A problem arises with CTC in as much that a suspicious lesion will require subsequent colonoscopy. This paper reports on a randomised study to compare the rates of additional colonic investigations after CTC or colonoscopy. Apparently 30% of the patients in the CTC cohort required additional studies compared with 8.2% in the colonoscopy group.

The detection rates for colorectal cancer large polys were 11% in both groups. CTC missed 1 of 29 colorectal cancers and colonoscopy missed none (of 55). The researchers conclude that “guidelines are needed to reduce the referral rate after CTC. For most patients, however, CTC provides a similarly sensitive, less invasive alternative to colonoscopy.”

Lancet 2013;381:1194–202.

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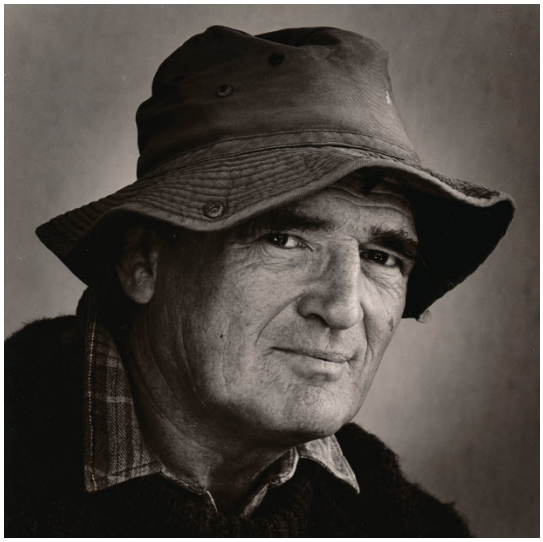
Hugh Cameron Burry

Born Christchurch, 29 October 1930; died Hanmer Springs, 18 June 2013.

Hugh Burry had the rare rugby distinction of beating the 1956 Springboks twice, the 1957 All Blacks, the 1957 Fijians and the 1959 Lions. Yet the Canterbury loose forward, renowned for his "reading" of a game, did not play for his country until 1960, as medical studies and practice got in the way.



One victory over South Africa came with the NZ Universities side. The others came with Canterbury, including a win against the All Blacks who had just returned from Australia.

Burry toured South Africa with the All Blacks in 1960. A groin injury restricted him to 11 games but he still scored eight tries.

He played 41 times for Canterbury, from 1955 to 1962. With John Graham and Kel Tremain, he formed the best back row in provincial rugby at the time. But there was more to Burry than rugby.

His death recently, at 82, followed a distinguished medical career. Among many achievements were his pioneering role in sports medicine, convincing international rugby authorities to make law changes that would help prevent head and neck injuries at the scrum, and attaining a professorship at the University of Melbourne.

Ever modest, he then retired to the farming life he had always hankered for, at Hanmer Springs. Wife Barbara says: “He was a man of wit, wisdom and modesty. He was an absolute out-and-out, one-eyed Cantabrian.” In Melbourne, he supported Essendon in Australian Rules because their colours were red and black.

Burry grew up in Christchurch, attending Fendalton Primary School and winning a scholarship to Christ’s College. He was an outstanding sportsman who, it was said, would have to choose between rugby and cricket, as he could represent New Zealand in either.

While studying medicine at Otago University, he played for the university club. While a house surgeon at Christchurch Hospital, he played for Canterbury University and won selection for NZ Universities.

He worked in general practice at New Brighton from 1957 to 1965 and switched to playing for New Brighton. He supported the club for the rest of his life, latterly as patron. Barbara says it was nothing for Burry in his retirement to drive from Hanmer Springs to New Brighton to watch a game – a three-hour return trip.

He became a medical registrar at Christchurch Hospital in 1965. His interest in rheumatology then took him to Guy's Hospital, London, as a registrar. He became director of rheumatology, rehabilitation and physiotherapy at Guy's, and then a lecturer at London University. Burry sat on committees, boards and panels at Guy's. He became consultant physician to the Sports Council of Great Britain.

He played social rugby, coached the Guy's Hospital team, was on the coaching advisory committee to the English rugby union, and then took up refereeing in London.

With this background in playing, coaching and refereeing, and his experience with rugby injuries as a doctor, the sports authorities might have been expected to take notice of his views on the dangers of modern scrummaging. But it was a battle for Burry to gain recognition of the problem, let alone get something done about it.

He was a lone figure warning of potentially life-threatening injuries from poor scrum techniques. Faced with International Rugby Board (IRB) obduracy, he had his ideas and evidence published in the *British Medical Journal*. The concerns he raised led the authorities eventually to make changes to the scrum laws.

Burry returned to New Zealand in 1976. He worked for the Wellington Hospital Board before becoming Associate Professor of Rheumatology at Wellington Clinical School. He took roles in organisations concerned with arthritis, rheumatism, sports injuries, accident compensation, disabilities and rehabilitation.

Married previously to Pamela Blackie, with whom he had three sons, he met and married Australian-born pharmacist Barbara Allen in 1984.

He became chairman of the New Zealand Rugby Union's medical advisory committee and a member of the IRB's medical advisory committee. He oversaw medical services for the first Rugby World Cup in 1987.

He moved to Australia in 1988 as Professor of Rehabilitation Medicine at Melbourne University. This included roles as Director of Rehabilitation Medicine at Royal Melbourne Hospital and Essendon District Memorial Hospital.

Burry retired in 1991. Settling at Hanmer Springs, he worked part-time as clinical director at Burwood Hospital's physical disabilities unit and conducted research for ACC. He was a self-employed consultant in rheumatology and rehabilitation medicine until 2000.

He and Barbara took up farming and animal husbandry at Hanmer Springs, setting up and running a perendale sheep stud. Burry enjoyed reading, bridge, fishing, tramping, music and gardening.

Survived by wife Barbara, sons Mark, Andrew and Michael and 10 grandchildren.

Mike Crean of *The Press* wrote this obituary, which originally appeared in their 29 June 2013 edition under the headline *All Black played valuable role in sports medicine.* The photograph of Hugh was taken by Barbara in 1997.

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Barrie David Evans

10 February 1948 – 16 December 2012; BDS University of Wales (1971); MBBS University of London (1977); MD University of London (1985); MRCP UK (1980); FRACP 1989; FRCP Edinburgh (1998).

Barrie David Evans was born in Cardiff, Wales, the son of Trevor Evans, a coalminer, and his wife Gladys (nee Hussey).

Barrie was the younger of their two children. He and his brother Ryvan grew up in Cardiff and attended The Cathedral School, Llandaff and Kings College, Taunton, Somerset.

Barrie qualified as a dental surgeon, with distinction, in 1971. He subsequently completed a medical degree at St Georges Hospital in London, qualifying in 1977.

He gained his Physicians specialist qualification in 1980 and trained in medical oncology at the Royal Marsden Hospital in London.

He worked there as Senior House Physician, Research Registrar, and finally Senior Medical Registrar.

His research included the use of high-dose cyclophosphamide in the laboratory and this formed the basis of his thesis for his Doctor of Medicine degree from the University of London in 1985. This research subsequently led him to be involved in a clinical trial using this approach in patients with small cell lung cancer.

Barrie also had a special interest in platinum analogues and was involved in Phase-II and Phase-III clinical trials using cisplatin and carboplatin in ovarian and other tumours. While at the Royal Marsden Barrie worked closely with and forged lifelong friendships with a number of renown oncologists including the late Professor Tim McElwain, the late Dr Eve Wilshire, and Professors Ian Smith and Martin Gore.

In 1981, Barrie married Justine Anderson, a New Zealand nurse whom he had met at the Royal Marsden Hospital. They had two children: Dr (Tom) Thomas Evans (Medical House Officer Auckland District Health Board) and Miss Charlotte Evans (Law and Commerce Student University of Auckland).

When Barrie had completed his specialist training there were no consultant positions available in the United Kingdom. Professor Tim McElwain, himself a New Zealander, suggested he should look at New Zealand and consider returning to the UK when a suitable job became available or if he did not settle in New Zealand.

Thus Barrie moved from the Royal Marsden Hospital to work as the sole Specialist Medical Oncologist at Palmerston North Hospital, New Zealand in 1985. The Oncology "Team" there consisted of only Barrie and a Charge Nurse, who had also trained at the Royal Marsden. It was a challenging job with wide-ranging clinical responsibilities, very long hours and a lot of travel.

Despite the difficulties he successfully developed the medical oncology service for the Central North Island that involved the introduction of standardised protocols for use in the network of regional centres.

In 1988, shortly after a second medical oncologist was appointed in Palmerston North, he moved to the Oncology Unit at Auckland Hospital, continuing his work there as a Specialist Medical Oncologist until he retired in the later part of 2012 due to ill health.

In addition to his work in the public-hospital system, Barrie also consulted in private practice in Auckland for 20 years. He, together with his wife Justine, provided a very personalized and dedicated oncology service for their patients.

Throughout his career, Barrie was involved in many aspects of cancer research, publishing over 70 papers in peer-reviewed journals on various aspects of cancer medicine and research. He presented at many international and national meetings.

Barrie had great experience in most areas of medical oncology with a special interest and expertise in gynaecological oncology, particularly ovarian cancer and gestational trophoblastic disease. He was Chairman of the New Zealand Gynaecology Oncology Group for 12 years and was on the Executive Committee and Deputy Chairman of the Australasian Gynaecology Oncology Group. He was Secretary of the Organising Committee for the First New Zealand International Symposium of Gynaecological Cancer and was instrumental in bringing it to Auckland in 1995. He was also appointed to the Medical Practitioners Disciplinary Tribunal (1997–2003) and to the Editorial Board of Cancer Treatment Reviews (1993–2004).

Barrie loved his medical career and was an outstanding oncologist. He was a superb and compassionate clinician and he was utterly dedicated to giving each and every one of his patients the best possible care. Barrie always gave a lot of time and thought to his patient's problems.

In difficult situations, he would go that extra mile, including consulting internationally when appropriate. Barrie was very supportive of his colleagues in medical oncology but also his colleagues in radiation oncology, surgery, pathology and radiology. He loved the multidisciplinary approach and was greatly respected by his colleagues.

In 1996, Barrie was appointed Honorary Associate Clinical Professor in the School of Medicine, University of Auckland in recognition of his research work and teaching skills. In Auckland, he continued his involvement in a variety of international, national and local clinical trials.

He was involved in the studies of locally developed new cancer drugs, including as the Principle Investigator for the Phase-I trial of "DACA". He had a profound influence on countless registrars during their training. They learnt at first hand from Barrie not only about research but also the importance of dedication to patients, being meticulous in your work and how to be a true professional.

Barrie also greatly enjoyed his life outside of work. He was a great sportsman with a love of all sports, but especially cricket and rugby. He had excelled at cricket and rugby at school, going on to play representative rugby for Cardiff Seconds where he was halfback and number two to that great international halfback Gareth Edwards.

He also played for the Glamorgan Seconds cricket team. In Auckland, in his spare time, Barrie became a high-level cricket coach. He spent many hours supporting both Tom and Charlotte in their sports, including coaching and umpiring.

Fishing was a particular love of Barrie's. This started with salmon fishing in Scotland. In New Zealand most of his trout fishing was around Turangi but in December most years Barrie and Tom, along with several other friends, would fly into the back country to fish in the Rangitikei River. Barrie and Tom also spent many hours together fishing for snapper and kingfish in the Hauraki Gulf.

Barrie had a great love of his family and was justifiably extremely proud of Justine, Tom and Charlotte. Barrie recognised the amazing support Justine provided – at home, at work in his private practice and with Tom and Charlotte.

Barrie was very generous and always a gentleman. He was a very wise man who always gave well considered and sound advice. He understood what was important to people and he did all that he could to help them achieve it. Barrie had a wonderful sense of humour and he was a great raconteur with a large stock of amusing stories and jokes.

Barrie was a wonderful, caring and talented man, with a great sense of fun and is greatly missed by his family, friends, and colleagues.

John H L Matthews (Oncologist, Auckland), a friend and colleague of Barrie, wrote this obituary.

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Erratum

Kirsten J Coppell, Jim I Mann, Sheila M Williams, Emmanuel Jo, Paul L Drury, Jody C Miller, Winsome R Parnell.

Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey.

N Z Med J. 1 Mar 2013;126(1370):23–42.

http://journal.nzma.org.nz/journal/126-1370/5555 and
http://journal.nzma.org.nz/journal/126-1370/5555/content.pdf

Further examination of the 2008/09 Adult Nutrition Survey data revealed a substantial number of people around the lower HbA1c cut-off for pre-diabetes, as defined by the American Diabetes Association (5.7%–6.4%). Following identification of a data storage problem, the data were re-examined, and it was noticed that some participants had been misclassified as having a 'normal' HbA1c level.

The revised pre-diabetes prevalence results are presented in the tables. The prevalence of pre-diabetes is higher than previously published, with the overall prevalence being 25.5%. The prevalence of diabetes was not affected.

Please go to full text and PDF links above for the corrected copy.