THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

CONTENTS

Viewpoint

100 Years Ago in the NZMJ

Medical Images

Methuselah

Letters

Obituaries

Book Reviews

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

This Issue in the Journal

Original articles

The risk of bronchiolitis hospitalisation following administration of a group B meningococcal vaccine in New Zealand

Paul Stehr-Green, Yvonne Galloway, Charlotte Kieft, Anne McNicholas

About 2% of children in their first year of life are admitted to hospital with bronchiolitis, making this disease the most common lower respiratory infection in infants. Most cases are caused by a virus called respiratory syncytial virus (also called RSV), and most cases occur in the winter. Because five infants were hospitalised with bronchiolitis within 2 months of receiving MeNZB™ during the vaccine trials, we looked at the risk of hospitalisation for bronchiolitis in infants after receiving MeNZB. We carried out three separate studies to examine this possible association, and based on the clear results from these studies, we concluded that MeNZB does not result in a higher risk of hospitalisation due to bronchiolitis.

Meningococcal B: tell me everything you know and everything you don't know. New Zealanders' decision-making regarding an immunisation programme

Paul B Watson, Judy Yarwood, Kim Chenery

This paper describes the decision-making process and factors that influenced a group of parent's decisions regarding their children's participation in the MeNZB immunisation programme. Ten parents who consented, 10 parents who did not consent to immunisation and one parent who chose to immunise one of two children were interviewed about their decision-making process. Both groups of parents were influenced by similar factors and followed similar decision-making processes. Parents perception was that their desire for reliable, valid, and balanced information about the MeNZB vaccine was largely unfulfilled.

Evaluation of the Palliative Care Partnership: a New Zealand solution to the provision of integrated palliative care

Eileen McKinlay, Lynn McBain

This paper reports an independent evaluation of a model of palliative care/end-of-life care currently being delivered in primary care settings in the Mid Central District Health Board area. The partnership consists of Arohnanui Hospice, Compass Health, and general practice teams of GPs and practice nurses and is funded by MidCentral District Health Board. The evaluation was conducted by the Department of Primary Health Care and General Practice, University of Otago, Wellington and showed a very effective form of integrated care at modest cost to the funder.

Viewpoint

Did Janet Frame have high-functioning autism?

Sarah Abrahamson

Janet Frame (1924–2004) was one of New Zealand's most well-known authors and unusual personalities. Her formal psychiatric diagnosis has not been clear. Some have suggested that she was simply "different." Dr Sarah Abrahamson (a neurological rehabilitation physician based in Ballarat, Australia) suggests that her difference could be called high-functioning autism. People with this condition have significant difficulties with social interaction with others, have strong interests, and sometimes specific exceptional skills, and often have difficulty with certain aspects of everyday life. These features are clearly described in Janet Frame's autobiographies.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Tobacco control in the Pacific

Don Matheson, Ashley Bloomfield, Debbie Ryan

The article by Rasanathan and Tukuitonga (*Tobacco smoking prevalence in Pacific Island countries and territories: a review*—http://www.nzma.org.nz/journal/120-1263/2742) in this issue of the *Journal* highlights the regional manifestation of global health inequalities with respect to tobacco use.

Progress being made in reducing overall smoking prevalence in Australia[1] and New Zealand[2] is not being matched in Pacific Island countries and territories (hereafter termed 'Pacific states') where rates of tobacco use are higher for males and females. Of particular concern are the high rates of youth smoking that will ensure the continuation of the problem for the next generation in these islands.

Interestingly, smoking rates experienced in Pacific states are not dissimilar to the rates for Pacific and Māori populations in New Zealand.[3] Indeed, 30 years of tobacco control in New Zealand have had little impact on the disparity in smoking prevalence between the general population and Māori, Pacific, and low income communities.

Until recently, smoking prevalence rates in the New Zealand Māori population remained stubbornly around the 50% level, with only the most recent survey showing signs of a reduction 45.2%.[2] The most recent Pacific smoking rate (37.4%) is higher than previous estimates, while the European rate is now 20.6%.[2]

Progress has been more successful, however, in reducing per capita consumption across all ethnic groups. However, even this 'gain' may be partly offset by remaining smokers switching from packaged cigarettes to "roll your owns," which they can roll thinner and extract the nicotine more efficiently through inhaling more frequently and deeply.

The currently unmet challenge both within countries and between countries is to address inequalities in tobacco use and not inadvertently worsen them. In approaching tobacco-related inequalities in the Pacific, we need to learn from our experience in tackling inequalities in New Zealand, take advantage of the global tools that are now available to all countries through the WHO Framework Convention on Tobacco Control (FCTC), and support the innovative solutions that arise from the Pacific states themselves.

Recent New Zealand experience has shown the comprehensive legislation, such as the *Smokefree Environments Amendment Act 2003* that eliminated tobacco smoke exposure in indoor workplaces and public places, is successful at reducing inequalities between groups.

Once the legislation came into force, the exposure of Māori workers to secondhand smoke in indoor workplaces dropped from 27% (2003) to 9% (2006), and for non-Māori from 19% to 8% over the same period.[4] At the same time, the cumulative effects of an increasingly smokefree environment is having a positive impact on smoking initiation, with a marked and ongoing reduction in smoking initiation by young teenagers in all ethnic and socioeconomic groups.[5]

While there was no significant decline in the proportion of young people whose parents smoked between 2001 and 2006, the proportion of Pacific 14 to 15 year olds exposed to smoking in their homes declined significantly from 34.6% in 2001 to 27.4% in 2006.[5] This demonstrates the power of broad environmental approaches to making progress in non-communicable disease control. In addition, widespread environmental 'prompts' to smokers to quit and increased availability of smoking cessation services will both increase the number of quit attempts and the likelihood that they will be successful.

New Zealand played a major role in the development of the FCTC (a world first as a global convention to curb a non-communicable disease), and continues to take a leading role in its implementation.[6] Interestingly, the convention has been extremely popular, with 149 ratifying countries at last count, making it one of the most widely affirmed Treaties in the UN system. New Zealand is already compliant with most of the mandatory provisions of the FCTC, but there are large gaps in the tobacco control policies and programmes of most Pacific states.

The FCTC will greatly assist tobacco control efforts in Pacific states. The Treaty effectively creates a globally agreed blueprint for tobacco control available to all countries, enabling them to leapfrog several developmental steps in their control programmes and move quickly to what is internationally accepted as best practice. The existence of a global consensus provides firm backing for states that will inevitably be challenged by tobacco industry interests.

The value of the FCTC in this respect can be seen in the experience of Canada where lengthy legal challenges by the tobacco industry to national tobacco control legislation were recently rejected, partly because of the mandate now conferred by the FCTC on ratifying parties.[7]

NZAID, the overseas development arm of the New Zealand Government, is funding a programme to develop capacity in the Pacific to develop tobacco control programmes and implement the FCTC.[8] The overall goal for the Tobacco Control in the Pacific (TCIP) programme is to support the efforts of Pacific states in countering the adverse health, social and economic impacts of tobacco use.

This initiative has involved two stages: Stage 1 took place from 2003-2004 in Tonga and the Cook Islands, with Stage 2 being implemented over 2005-2007 in Samoa, Solomon Islands, Vanuatu, and Tuvalu.

An additional strength for Pacific states is their history of developing uniquely Pacific approaches specific to their size and cultural context. Examples of this include the "Healthy Islands" Policy,[9] and the developing response to the obesity epidemic; for example, both Fiji and Samoa have acted at a national level to restrict the availability of fatty foods.[10,11] Approaches to tobacco will benefit from similarly inspired local solutions that small states can apply.

The stakes for effective non-communicable disease (NCD) control in the Pacific are high, as the disease burden associated with it will impact and overwhelm local health responses, as well as potentially strain those of neighbouring countries such as New Zealand and Australia. Altruism and self interest combine to make this a priority. Our ability to share experience and directly assist approaches that work will be important for tobacco control, but even more so for other drivers of NCDs such as obesity and alcohol related harm where the evidence for effective interventions has not been as well researched and evaluated.

Competing interests: None.

Disclaimer: Don Matheson, Ashley Bloomfield, and Debbie Ryan are employees of the Ministry of Health and the views expressed in this paper do not necessarily reflect the views of the Ministry of Health.

Author information: Don Matheson, Director International Relations; Ashley Bloomfield, Chief Advisor Public Health and Acting Manager, Tobacco Policy and Implementation; Debbie Ryan, Chief Advisor Pacific Health; Ministry of Health, Wellington

Correspondence: Don Matheson, Director International Relations, Ministry of Health, PO Box 5013, Wellington, New Zealand. Fax: +64 (0)4 816 2340; email: don_matheson@moh.govt.nz

References:

1. Australian Institute of Health and Welfare. Statistics on drug use in Australia 2006. Drug Statistics Series No. 18. Cat. no. PHE 80. Canberra: AIHW; 2007. http://www.aihw.gov.au/publications/phe/soduia06/soduia06-c00.pdf
2. Ministry of Health. Tobacco Trends 2006: monitoring tobacco use in New Zealand. Wellington: Ministry of Health; 2006. http://www.moh.govt.nz/moh.nsf/pagesmh/5658?Open
3. Ministry of Health. New Zealand Tobacco Use Survey 2006. Wellington: Ministry of Health; 2007. http://www.moh.govt.nz/moh.nsf/indexmh/nz-tobacco-use-survey-2006?Open
4. Edwards R, Bullen C, O'Dea D, et al. After the Smoke has Cleared: Evaluation of the Impact of a New Smokefree Law. Wellington: Ministry of Health; 2006. http://www.moh.govt.nz/moh.nsf/by+unid/A9D3734516F6757ECC25723D00752D50?Open
5. Scragg R. Report of 1999-2006 Year 10 Snapshot Smoking Surveys: Prepared for ASH and Health Sponsorship Council. Auckland: University of Auckland; 2007. http://www.ash.org.nz/pdf/Reportof2006Year10Survey_FINAL.pdf
6. Hon Damien O'Connor, Associate Minister of Health. Global Award Recognises New Zealand as a Leader in Tobacco Control. Media Release 14 September 2007. http://www.beehive.govt.nz/ViewDocument.aspx?DocumentID=30631
7. Canada (Attorney General) v. JTI-Macdonald Corp., 2007 Supreme Court of Canada 30. June 28 2007. Paragraph 10. http://scc.lexum.umontreal.ca/en/2007/2007scc30/2007scc30.html
8. Emma Dunlop-Bennett (Development Programme Manager). Personal Communication. Pacific Regional Health and Education NZAID; September 2007.
9. World Health Organization. Regional Office for the Western Pacific. Madang commitment towards healthy islands, 14-15 March 2001. Meeting of the Ministers of Health for the Pacific Island Countries (4th: 2001: Madang, Papua New Guinea). Manila: WHO Regional Office for the Western Pacific; 2001. http://whqlibdoc.who.int/wpro/2001/WPR_ECP_DPM_2001.pdf
10. Evans M, Sinclair R, Fusimalohi C, Liav'a V. Globalization, Diet, and Health: An Example from Tonga. Bulletin of the World Health Organization. 2001:79;856–62. http://www.who.int/bulletin/archives/79(9)856.pdf
11. Radio New Zealand International. Samoa government bans import of US turkey tail meat; 25 April 2007. http://www.rnzi.com/pages/news.php?op=read&id=31750

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Economics can be good for health, but it needn't be so dismal

Eric Crampton

One can easily see why some come to call economics the "dismal science."[1] The letter by Wilson, Edwards, and Mansoor in this issue of the *NZMJ* entitled *Economics can be good for health: need for rational policy without the influence of vested interests* (http://www.nzma.org.nz/journal/120-1263/2757) suggests to me that anything fun should be taxed until we're left with a rather dreary existence devoid of risk, excitement, or sensation. However, for a more positive assessment, one ought look perhaps to a real economist rather than those invoking the discipline's name in pursuit of their personal agendas.

Wilson et al suggest that rational economic analysis calls for large increases in tobacco and alcohol taxes to reduce consumption—taxing obesogenic and subsidising "healthier" food options follow as the next "rational" suggestions. Given New Zealand's rather high rates of STDs, I await their calls for large taxes on unprotected sex and subsidisation of condoms or, in the alternative, strict regulation of sex outside of a monogamous partnership.

Just as differential hazards call for differential taxes on beer and wine, so too might they recommend higher taxes on particularly risky sexual activity. It seems that, for them, reducing health (and, possibly, environmental) risks is all that we really should include in a social welfare function.

Of course, most economists would disagree vehemently. Raising taxes does tend to reduce consumption and, where consumption generates large negative externalities (costs borne by uninvolved parties) can even be efficient: Pigovean taxes (taxes proportionate to those external costs) can push us closer to socially-optimal outcomes. But, there is no inefficiency caused by people choosing to live lifestyles they view as preferable despite the health costs.

If I decide to enjoy a shorter life rather than eek out a miserable existence without wonderfully-marbled steaks, a beer or several, or even the occasional cigar, zero inefficiency is induced thereby.[2]

Some try to build a case for market failure based on "fiscal externalities": with a publicly-funded health system, I may offload the costs of my choices onto the public purse and take too many risks. But, economic theory suggests that we really oughtn't worry too much.[3] Many economists view these not to be inefficiency-inducing externalities at all.

To the extent that fiscal externalities are a problem, the only two solutions that push us towards efficiency are:

- Massively comprehensive sets of Pigovean taxes and subsidies on every choice that may be affected (again, see my caution about taxes on sex, above), or, more feasibly
- The elimination of public health care.[3]

I tend to think that the losses of either approach are much larger than the losses incurred simply by leaving things alone. It is more socially costly to try and correct all of the distortions caused by people consuming too much risk because of the public health system than it is to simply bear the cost of the distortions.

Moreover, what evidence there is suggests that to the extent smoking induces a "fiscal externality," the sign of the effect is wrong: smokers pay more in cigarette taxes than they ever cost the public purse. They die earlier of cheaper diseases and collect less in superannuation than do non-smokers.[4] And, as a 10% increase in cigarette taxes correlates with a 2% increase in obesity,[5] one wonders whether increased cigarette taxes consequently require further increases in taxes on fatty foods.

I suppose that the key difference between economists and public health researchers is that economists are typically happier to let individuals be the judges of their own wellbeing. If people make choices that we wouldn't make for ourselves, we're happy for them to do so as long as they're not hurting anybody else in the process.

"De gustibus non est disputandum":[6] we should not criticise individuals' preferences. Too often, researchers couch paternalistic arguments in allegations of market failure to give the cloak of scientific efficiency to their prescriptions. Doing so is just bad economics. A more honest approach would first specify that the authors want to tell everyone how to live their lives, then present the set of Pigovean instruments as an efficient way of inducing the consumption choices they view as better than that which people would otherwise choose for themselves.

Be not ashamed of your paternalism: embrace it! But, if others disagree, don't blame shadowy special interests for the failure of your policy prescriptions; rather, concede that most people really don't like it when others try to tell them how to live, even if following the advice would lead to slightly longer (but less interesting) lives.

Wilson et al specifically criticise the government's planned policies on climate change. I confess to puzzlement as to the relevance of this issue for a medical journal. Given the goal of reducing emissions, most economists prefer carbon taxes[7] though many prefer trading as second-best where Pigovean taxes are not politically feasible.

Economists also are divided as to whether the entire enterprise is worth the effort: reducing carbon emissions sufficiently to have any effect on warming may well prove far more costly than simply bearing the costs of the warming and reallocating the saved monies to environmental areas where the "bang for the buck" is much higher.[8,9]

But few economists would follow Wilson et al in arguing that a carbon trading system that allocates initial carbon emission rights to current emitters is irrational. Once the system is set up, most economists would argue it's entirely irrelevant (on efficiency grounds) to whom initial credits are allocated: people will trade the emission rights to achieve the efficient allocation of the rights.[10]

Moreover, allocating rights initially to polluters makes the whole thing politically feasible. Such allocations might not be "fair" under some ethical frameworks, but others may well deem it unjust to ask the agricultural sector to bear the lion's share of the costs of appeasing Green Party voters' sensibilities. However, such allocations certainly do not violate efficiency or conditions for "rational policy." Couching value statements as matters of efficiency, again, is bad economics.

Wilson et al conclude by calling for independent agencies with "real power to introduce and implement effective policy decisions in the interests of health, the environment, and social justice." If there were real social consensus as to the goals to

be achieved, and their relative weightings as compared to everything else in life, perhaps they would be correct. Of course, we ought to insulate an independent "Tobacco Control Authority" not only from tobacco lobbyists but also from the puritans[11] who view any tobacco use as sin to be extinguished.

There seems to be a great deal of money these days (mostly coming from the public purse) funding moral crusades against the demon weed; I, for one, wonder about the tenure or continued funding prospects of the health researcher finding risks of secondhand smoke to be overblown.[12]

More importantly, we do not have widespread social agreement regarding the goals which Wilson et al wish to achieve by employing economic incentives. People reasonably disagree as to what constitutes social justice, the appropriate tradeoffs between health and happiness, and the weighting of environmental amenities as compared to wealth and economic growth. Such is the hurly-burly of normal politics, and such oughtn't be shunted away to arms-length agencies charged with forcing us to lead lives devoid of flavour.

I'll take their recommendations, as I do my dinner, with more than a pinch of salt.

Competing interests: The author has received funding neither from evil tobacco lobbyists nor from anti-tobacco zealots. He doesn't smoke, but won't think less of you if you do; he'll just ask that you refrain during dinner or in his home.

Author information: Eric Crampton, Senior Lecturer, Department of Economics, University of Canterbury, Christchurch

Correspondence: Eric Crampton, Department of Economics, University of Canterbury, Private Bag 4800, Christchurch. Email: eric.crampton@canterbury.ac.nz

References:

1. Levy D. How the Dismal Science Got its Name: Classical Economics and the Ur-Text of Racial Politics. University of Michigan Press; 2002.
2. Buchanan J, Stubblebine WC. Externality. Economica. 1962;29:116:371–84.
3. Browning E. The myth of fiscal externalities. Public Finance Review. 1999;27:1:3–18.
4. McLeod R, Patterson D, Jones S, et al. Tax review 2001 final report. Treasury paper. Wellington: New Zealand Government; 2001;v. http://www.treasury.govt.nz/taxreview2001/finalreport/index.html
5. Rashad I, Grossman M. The economics of obesity. The Public Interest. 2004;156:104–12.
6. Stigler G., Becker G. De gustibus non est disputandum. American Economic Review. 1977;67:2:76–90.
7. Mankiw NG. Raise the gas tax. The Wall Street Journal, 20 October 2006.
8. Lomborg B. Global crises, global solutions. Cambridge University Press; 2004.
9. Weitzman M. A Review of The Stern Review on the Economics of Climate Change. Journal of Economic Literature. 2007;XLV:703–24.
10. Coase R. The problem of social cost. Journal of Law & Economics. 1960;III:October:1–44.
11. Action on Smoking and Health. ASH position on snus. Smokefree Times. 2006;82:4. http://www.ash.org.nz/system/_doc.php?id=871
12. Libin K. Whither the campus radical: Academic freedom. The National Post (Canada). 2007;28 September.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

The risk of bronchiolitis hospitalisation following administration of a group B meningococcal vaccine in New Zealand

Paul Stehr-Green, Yvonne Galloway, Charlotte Kieft, Anne McNicholas

Abstract

Aim During the Phase II clinical trials for a new group B meningococcal vaccine in New Zealand, six study participants (including five children who had been vaccinated with this vaccine) were hospitalised due to acute bronchiolitis. We examined more closely the potential association between bronchiolitis hospitalisation and this vaccine.

Methods We used descriptive comparisons, a cohort analysis, and a matched case-control study to examine the potential association of bronchiolitis hospitalisation with the vaccine using New Zealand Health Information Service hospital discharge data and vaccination data from the National Immunisation Register.

Results The distribution of hospitalised bronchiolitis cases throughout New Zealand immediately following the introduction of the vaccine was consistent with historical (pre-vaccine) patterns. Similarly, all point estimates for relative risk (cohort analysis) and odds ratio (case-control study) for assessing the potential association between bronchiolitis hospitalisation and the vaccine were less than 1.00.

Conclusions We concluded that this vaccine is not associated with an increased risk of hospitalisation for bronchiolitis.

Acute bronchiolitis is the most common lower respiratory tract infection in children aged under 1 year; approximately 2% of all children are hospitalised with bronchiolitis in their first year of life.[1] The majority of cases are caused by respiratory syncytial virus, with peak disease incidence in the winter season.[2,3]

During the Phase II clinical trials for New Zealand's group B meningococcal vaccine (MeNZB™), six study participants aged from 3 to 9 months were hospitalised for bronchiolitis (Personal Communication: Oster P, Novartis Vaccines and Diagnostics S.r.1, September 2005). All of these cases occurred during the winter/spring months, with one case in a child who had not received the group B meningococcal vaccine and five cases who had onset of symptoms between 19 and 53 days after vaccination with the group B meningococcal vaccine.

After the conclusion of the formal post-marketing safety monitoring programme,[4] we examined the potential association between vaccination and the risk of hospitalisation for bronchiolitis in infants (i.e. aged 6 weeks through 11 months).

Methods

Descriptive comparison—We compared the seasonal distribution of bronchiolitis hospitalisations during 1996–2000 (i.e. prior to the introduction of the vaccine) with those during 2004–2005 (i.e. immediately prior to and following the introduction of the vaccine) in New Zealand.

We identified cases among infants using data in the New Zealand Health Information Service hospital discharge dataset, defined as follows:

- For the time period January 1996–December 2000, we used International Classification of Diseases [ICD]-9-CM code 466.1 (Acute bronchiolitis); and
- For the time period January 2004–December 2005, we used the ICD-10-AM code J21 (Acute bronchiolitis).

To avoid double-counting readmissions for the same episode of bronchiolitis, we excluded admissions that occurred within 14 days after a prior admission for bronchiolitis in the same patient.

Cohort analysis—We assessed the increased risk of hospitalisation for bronchiolitis in infants throughout New Zealand during the 30 days following vaccination using a cohort analysis.[5] This 30-day latency period approximates the mean/median of the latencies in the five cases who received the group B meningococcal vaccine in the Phase II clinical trial.

We also examined the possibility of an increased risk of bronchiolitis hospitalisation within 60 days after vaccination as part of a sensitivity analysis. Using hospital discharge data from 2005, we identified all cases among infants where the ICD-10-AM code J21 was recorded in any of the diagnosis fields.

We included all cases that occurred in infants who were eligible to receive the vaccine in their respective District Health Board (DHB) [numerators]; as in the descriptive comparisons, we excluded readmissions that occurred within 14 days after a prior admission for bronchiolitis in the same infant.

We defined cases that occurred within the specified 'risk interval' following vaccination (i.e. within 30 [or 60] days after vaccination) as 'vaccinated' cases. For the purpose of these analyses, we defined all bronchiolitis cases that occurred outside the risk interval as 'non-vaccinated' cases.

We estimated person-time-at-risk [denominators] using Statistics New Zealand population estimates for 2005 and vaccination details from the National Immunisation Register. We calculated overall person-time-at-risk (in days) by multiplying the 2005 estimated population aged 6 weeks through 11 months (proportionalised from the under 1 year estimate) in each DHB by the number of days in the observation period that infants were eligible to receive the vaccine in their DHB.

We estimated person-days for the vaccinated group in the specified risk interval by multiplying the number of doses received by 30 (or 60) days. Similarly, we estimated person-days for the non-vaccinated group by subtracting the number of person-days for the vaccinated group from the overall number of person-days.

We estimated the relative risk (with test-based 95% confidence intervals) of hospitalisation for bronchiolitis within 30 (and 60) days after any dose of the vaccine, and then separately for each individual dose for which we identified more than five vaccinated cases.

Case-control study—We conducted a matched case-control study of infants identified through hospital discharge data. We defined the observation period for the case-control study as 3 February 2005 (i.e. the date on which the vaccination of infants aged under 6 months commenced in New Zealand) through 31 December 2005, to coincide with the time period during which the vaccine was available for all infants eligible for this study.

We identified cases using the following selection criteria:

- Aged 6 weeks through 11 months at the time of hospital admission;
- Hospital admission during 3 February 2005 through 31 December 2005;
- ICD-10-AM code J21 was recorded in any of the diagnosis fields; and
- First hospitalisation for bronchiolitis (this criterion differed, albeit slightly, from that used in the descriptive comparisons and cohort analysis.)

We limited eligible controls to those infants who had equivalent opportunities to be vaccinated and to be hospitalised for bronchiolitis as did the cases using the following criteria:

- Aged 6 weeks through 11 months at the time of hospital admission;
- Hospital admission during 3 February 2005 through 31 December 2005;
- No hospital admissions for bronchiolitis during the study period;
- ICD-10-AM-coded conditions not related to any infectious process, for which there was no extant evidence of an association with any vaccine, and which would not affect the child's likelihood of being vaccinated (e.g. injury); and
- First hospitalisation for this type of event.

We attempted to match up to four controls per case by gender, DHB of residence, month of admission, year of admission, month of birth, and year of birth.

As in the cohort analysis, we focused our primary analysis on all hospitalised cases of bronchiolitis that had occurred within 30 days after receipt of any dose of the vaccine, but we also examined the possibility of an increased likelihood of having been vaccinated with any dose within 7 or 14 days prior to hospital admission as part of a sensitivity analysis. In calculating odds ratios and the exact 95% confidence intervals, we used an extension of the McNemar test that accounted for the variable number of matched controls.[6]

Results

Descriptive comparison—Over 80% of all hospitalised bronchiolitis cases that occurred in New Zealand during 1996 through 2000 were among infants, for whom there were 15,753 cases (annual mean: 3151), which corresponded to an average annual rate of 6263.8 admissions per 100,000 infants. Among these cases, the median age at onset of symptoms was 5.2 months, with boys comprising a majority (9325 cases, or 61.1%).

By comparison, in 2004 through 2005, there were 7150 hospitalised bronchiolitis cases among infants (annual mean: 3573 cases and median age: 5.7 months), which corresponded to a mean annual rate of 7038.7 admissions per 100,000 infants. As in 1996 through 2000, boys comprised a majority (4402 cases, or 61.6%).

Although rates of hospitalisation for bronchiolitis appeared to be increasing throughout the overall time period covered by our study, there was a similar seasonal pattern of bronchiolitis hospitalisations in the two time periods we examined, with a marked winter peak occurring from July to September (Figure 1).

Of particular relevance to our study, we noted that the seasonal increase in the winter of 2005 occurred as expected, coincidentally at the same time that the group B meningococcal vaccine vaccination rates began to increase (Figure 1).

Cohort analysis—In 2005, a total of 165,126 vaccine doses were administered to infants throughout New Zealand. We identified a total of 3215 hospitalised bronchiolitis cases in 2005 that met the case-definition, of which 913 (28.4%) occurred within 30 days and 1479 (46.0%) occurred within 60 days after vaccination with the group B meningococcal vaccine.

The point estimates for every relative risk for all doses combined and separately for each of the first three doses using either a 30 or 60 day risk interval were less than 1.00 (Table 1), indicating no evidence for a statistically significant increased risk of hospitalisation for bronchiolitis either in 30 or 60 days following receipt of the vaccine.

Figure 1. Average rate of bronchiolitis admissions, by month (1996–2000) and rate of bronchiolitis admissions and number of MeNZB™ doses administered, by month and year (2004–2005), infants aged 6 weeks through 11 months, New Zealand

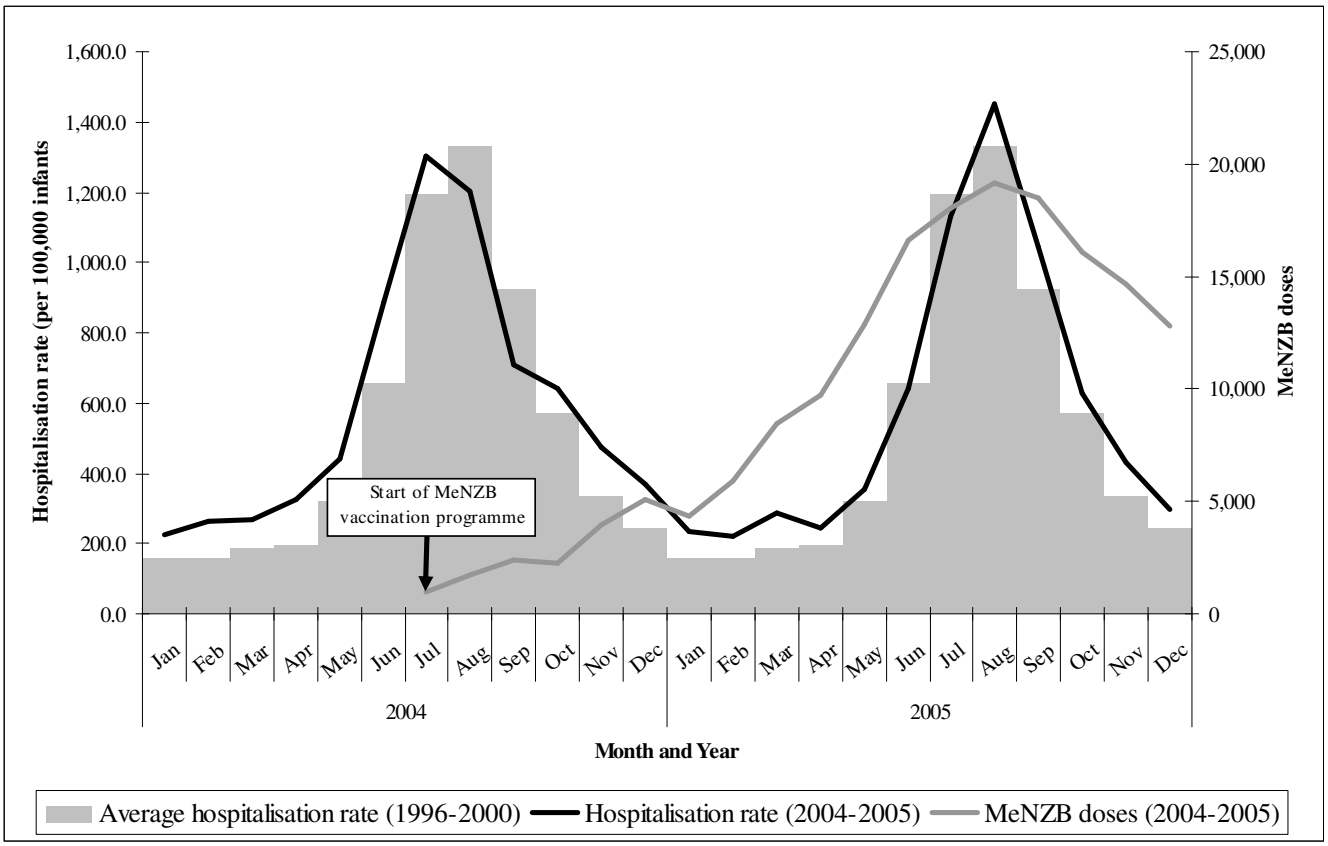


Table 1. Relative risk of bronchiolitis hospitalisation within 30 (or 60) days after vaccination with MeNZB™ compared with cases outside 30 (or 60) days after vaccination with MeNZB™ and non-vaccinees, infants aged 6 weeks through 11 months, by vaccine dose, New Zealand, 2005

Risk interval following vaccination	Dose(s) of vaccine	Number of cases within risk interval*,**	Number of cases outside risk interval†	Relative risk (95% CI)
30 days	Any/All	913	2302	0.73 (0.67–0.78)
	1	434	2781	0.92 (0.83–1.02)
	2	320	2895	0.81 (0.72–0.91)
	3	157	3058	0.53 (0.45–0.63)
60 days	Any/All	1479	1736	0.36 (0.33–0.38)
	1	703	2512	0.69 (0.63–0.75)
	2	508	2707	0.59 (0.54–0.65)
	3	263	2952	0.42 (0.37–0.48)

*Bronchiolitis admission occurring 1–30 (or 1–60) days after vaccination; **The sum of the dose-specific cases does not equal the number of cases for 'Any/All' doses because National Immunisation Register records indicated that a few bronchiolitis cases occurred after administration of a fourth MeNZB™ dose—in fact, a fourth MeNZB™ dose was not recommended for infants until January 2006; †Bronchiolitis admission occurring more than 30 (or 60) days after vaccination or in non-vaccinee.

Matched case control study—We identified 337 bronchiolitis cases that were admitted to hospital during 3 February 2005 through 31 December 2005 that met the case-definition and for which we could identify at least one matched control. These 337 cases were matched against 367 control subjects, with 313 case-control pairs matched 1:1, 19 groupings with a 1:2 matching ratio, four with a 1:3 ratio, and one with a 1:4 ratio. The point estimates for every odds ratio for cases vaccinated either 30, 14, or 7 days prior to hospital admission were less than 1.00 (Table 2), suggesting that there was no statistically significant increased risk of having been vaccinated within the specified time period prior to hospitalisation for bronchiolitis.

Table 2. Relative odds of vaccination with MeNZB™ 30 (or 14 or 7) days prior to hospitalisation for bronchiolitis compared with matched* controls, infants aged 6 weeks through 11 months, New Zealand, 2005

Hospital admission following vaccination	Number of cases**	Odds ratio (95% CI)
30 days	187	0.71 (0.49–1.03)
14 days	103	0.72 (0.47–1.11)
7 days	49	0.65 (0.34–1.24)

*Cases were matched to controls (up to 4 per case) by gender, DHB of residence, month of admission, year of admission, month of birth, and year of birth; **Bronchiolitis admission within specified number of days following vaccination.

Discussion

'Clusters' of disease are defined as "…an unusual aggregation, real or perceived, of health events that are grouped together in time and space."[8] Attempts to discern the existence and, if real, the cause(s) of such clusters are subject to a number of challenges; notably, the health events being investigated are often rare and increases in the number of these events tend to be small and occur over a long time period. Many (if not most) clusters occur simply by chance—a fact that can compromise the perceived credibility (and, hence, the acceptability) of the results of such investigations.

Indeed, based on our results, 'chance' is the most likely explanation for the occurrence of the six hospitalised bronchiolitis cases (five in MeNZB™ vaccinees) that were observed among participants in the Phase II clinical trials. The separate (and combined) results of our investigations strongly and consistently suggest that administration of the vaccine is not associated with an increased risk of hospitalisation for bronchiolitis. Furthermore, this finding is consistent with the fact that there has been no previously reported association between bronchiolitis and other outer membrane vesicle vaccines—or, for that matter, with any other vaccine.

Of course, none of these analyses are without their inherent weaknesses. The graphical comparison of the seasonal patterns in the incidence of hospitalised bronchiolitis cases among infants suggests that there has been little change between the pre- and post-MeNZB™ era. However, because this comparison is ecologic, it cannot adequately account for potentially important interactions of agent (i.e. the vaccine), host (i.e. vaccinees), and environment (e.g. socioeconomic or other factors

associated with the likelihood of being vaccinated and/or the risk of exposure to respiratory syncytial virus or other bronchiolitis-causing infections) that might affect the true relationship, if any, between the vaccine and bronchiolitis.

In contrast, the cohort analysis provides stronger evidence that there was no increased risk of hospital admission for bronchiolitis following vaccination. Of note, we included in this analysis all cases that occurred in 2005, so as to mitigate the possible confounding influence (or bias) caused by the observed seasonal variability in incidence. However, because we included only hospitalised bronchiolitis cases (i.e. representing cases at the more-severe end of the clinical spectrum), the generalisability of our results may have been limited somewhat. (This is a potential shortcoming of our case-control study, as well.)

Furthermore, although one of the strengths of this analytical approach derives from the fact that study participants contribute person-time-at-risk to both the vaccinated and non-vaccinated groups (thereby serving, in part, as their own comparison subjects), there may have been residual bias or confounding[9] for which data were not available in these datasets which were designed to serve primarily administrative (rather than research) needs.

The results of the matched case-control design are arguably the most robust, in that we were able to control at the level of each individual study participant for several important potential confounders. It is, however, possible that our attempts to simultaneously control for a variety of potential confounders may have resulted in 'overmatching'—i.e. inadvertently controlling for one or more factors that are, in fact, associated with the actual causal chain linking this vaccine with an increased risk of hospitalisation for bronchiolitis.

Nonetheless, in weighing the consistent, strong results from our separate and combined investigations against the foregoing methodological and data quality issues, we conclude that administration of this group B meningococcal vaccine is not associated with an increased risk of hospital admission due to bronchiolitis.

Competing interests: The New Zealand Ministry of Health was funded by Chiron Vaccines (now Novartis Vaccines and Diagnostics S.r.1) to undertake post-marketing safety monitoring of MeNZB.

Note: All authors participated in the above-described work as part of the Meningococcal Vaccine Strategy, New Zealand Ministry of Health, Wellington.

Author information: Paul Stehr-Green, Consulting Epidemiologist and Affiliate Associate Professor, Department of Epidemiology, University of Washington, Seattle, Washington, USA; Yvonne Galloway, Senior Adviser, Accident Compensation Corporation (ACC), Wellington; Charlotte Kieft, Data Analyst, ACC, Wellington; Anne McNicholas, Senior Adviser, New Zealand Ministry of Health, Wellington

Acknowledgments: Chiron Vaccines (now Novartis Vaccines and Diagnostics S.r.1) funded the New Zealand Ministry of Health to undertake the overall safety monitoring programme.

In addition, we gratefully acknowledge the contribution of Jane O'Hallahan (New Zealand Ministry of Health) in the investigations and the preparation of this manuscript.

Correspondence: Paul Stehr-Green (Consulting Epidemiologist and Affiliate Associate Professor). Email: pstehrgreen@att.net

References:

1. Milner AD. Ribavirin and acute bronchiolitis in infancy. BMJ. 1988;297:998–9.
2. Dawson KP, Mogridge RN. Acute bronchiolitis: a three year study. N Z Med J. 1989;102:528–9.
3. Pattemore PK. Infections of the respiratory tract in children. New Zealand Practice Nurse. 1992;December:30–5.
4. McNicholas A, Galloway Y, Stehr-Green P, et al. Post-marketing safety monitoring of a new group B meningococcal vaccine in New Zealand, 2004-2006. Hum Vaccin. 2007;3:196–204.
5. Chen RT. Special methodological issues in pharmacoepidemiology studies of vaccine safety. In: Strom BL, editor. Pharmacoepidemiology. 3rd ed. Chichester: John Wiley & Sons; 2000:707–32.
6. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. J Natl Cancer Inst. 1959;22:719–48.
7. Centers for Disease Control and Prevention. Guidelines for investigating clusters of health events. MMWR Recomm Rep. 1990;39(RR-11):1–23.
8. Glanz JM, McClure DL, Zu S, et al. Four different study designs to evaluate vaccine safety were equally validated with contrasting limitations. J Clin Epidemiol. 2006;59:808–18.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Meningococcal B: tell me everything you know and everything you don't know. New Zealanders' decision-making regarding an immunisation programme

Paul B Watson, Judy Yarwood, Kim Chenery

Abstract

Aims To describe parents' decision-making process and investigate what factors influenced decisions regarding children's participation in the MeNZB™ immunisation programme.

Method 10 parents who consented to their children receiving the vaccine, 10 parents who did not consent to immunise their child, and 1 parent who chose to immunise only 1 of their 2 children all participated in a semi-structured interview. A qualitative descriptive thematic analysis was thereafter undertaken.

Results Both groups of parents were influenced by similar factors and followed similar decision-making processes. Four non-linear phases were identified; a gut reaction, a trigger, reconnaissance, and risk analysis. Most parents expressed a degree of uncertainty about their decision.

Conclusion Parents had a largely unfulfilled desire for reliable, valid, and balanced information about the MeNZB vaccine. Health authorities balance of risk at a population level may not coincide with that of individual parents. This study suggests other factors are likely to influence decisions. Parents' generally negative view of official information provided in the MeNZB immunisation programme suggest that communication strategies may require rethinking for future programmes.

The success of the Ministry of Health's recent meningococcal B (MeNZB™) immunisation programme was dependent upon parents choosing to immunise their children. And a high level of vaccine uptake was a priority in preventing disease.[1]

Prior to the MeNZB campaign, literature indicated New Zealand parents preferred to visit their general practitioner for vaccinations.[2] Moreover, a significant number of parents had low confidence in vaccination and did not trust received government vaccination information.[3,4]

Several studies have identified variables that seem to influence vaccination uptake including the use of combination vaccines and access to information.[5,6] However, these studies insufficiently explain individual decision-making processes.

Relatively little qualitative research into parents' decision-making about childhood vaccination has been carried out.[7] This study aimed to explore how parents made decisions in relation to their child(ren)'s participation in the MeNZB immunisation programme and the factors that influenced their decisions.

Methods

For this qualitative descriptive study, recruitment was through advertisements in two free community newspapers delivered to Christchurch households. We sought families who consented, and who did not consent to have their child(ren) receive the meningococcal B immunisation.

Inclusion criteria required participants to be:

- A parent or guardian of at least 1 child aged between 6 weeks and 15 years old who had made a decision about whether their child(ren) received the MeNZB immunisation;
- English speaking and live in Christchurch.

Ethical approval was granted by the Upper South B Regional Ethics Committee and Christchurch Polytechnic Institute of Technology Ethics Committee. Data collection took place between November and December 2005. Semi structured interviews focused on parent's descriptions of their decision-making process. Additional questions prompted for details about sources and quality of information and other factors that underpinned decision-making.

All interviews, conducted at a location and time convenient to participants, were audiotaped and lasted between 45 to 60 minutes. Sociodemographic data including parental age and gender, ethnicity, education levels, income, family size, and their child(ren)'s previous immunisation history were also collected.

Transcripts of each audiotaped interview were initially coded line by line by two of the three authors. Codes focussed on actions and events in the parents' narratives. All three authors then constantly examined the emergent codes, grouped these into thematic categories and compared them across the two groups. Similarities and differences in the decision-making processes were then evaluated.

Results

Interviews were conducted with 21 parents, 10 who had consented to immunisation, 10 who had not, and 1 who had decided to immunise only 1 of their 2 children. Demographic data are presented in Table 1. The majority (18) of interviewees were mothers, 2 were both parents, and 1 was a father. Both groups represented a range of ethnicities and socioeconomic situations.

Of the parents who consented to the MeNZB vaccination, 90% had children who were fully immunised and 10% partially. Conversely, of the parents who did not consent, 40% had not previously immunised their children, and 30% only partially.

Despite reaching different decisions, the majority of parents followed a similar decision-making process consisting of four non-linear phases: *a gut reaction*, *a trigger*, *reconnaissance*, and *risk analysis.*

A gut reaction—Parents in both groups experienced a gut reaction. They typically described, often in the absence of any specific information about the vaccine, a decision they would probably make:

> We pretty much knew we would get it done when we heard about it *(Participant 1: immuniser)*

> Everyone around me was saying 'yes' and I was actually feeling like 'no' *(Participant 19: non-immuniser)*

Although two parents subsequently changed their mind, most remained committed to this initial decision. All parents expressed concern about the seriousness of meningococcal disease, however those parents who chose not to vaccinate believed there were other protective measures and perceived their child to be less susceptible:

> I have been a LaLeche League leader so to me breast feeding is the ultimate in protecting our children's immune system *(Participant 9: non-immuniser)*

Parents who chose not to immunise were also more concerned about vaccine safety and efficacy.

Table 1. Demographic characteristics of the study participants

Demographic characteristics[1]	Consented to MenzB (n=10)	Did not consent to MenzB (n=10)
Age of parent; mean (SD)	34.4 (5.5)	40 (6.8)
Children's immunisation status		
Fully immunised	9	3
Partially immunised	1	3
Not immunised	0	4
Ethnicity		
Māori	2	1
New Zealand European	7	8
Other	1	0
Cook Island Māori	0	1
Parent's highest qualification		
≥ Master's degree	1	0
Bachelor's degree	3	1
Other tertiary	3	3
Secondary school	3	6
Family income (NZ$)		
<24,999	1	2
25,000–54,999	4	4
>55,000	3	3
Not answered	2	1

Note: To ensure anonymity, the demographic characteristics of the one participant who decided to immunise one child and not the other have been withheld.

A trigger—Most parents identified a trigger, either a person, an event, or the media confirming local availability of the vaccine. The trigger for most of those with school-age children was a consent form sent home from school. Triggers varied for parents of pre-school children, some mentioned talking to or receiving notification about vaccine availability from their general practitioner. Several parents referred to the media as a trigger. Information disseminated by the media was approached with scepticism by most parents:

> I mean I'm not silly and I know that the media presents what they want. They want a newsworthy story that someone's going to watch so they are always going to promote the worst or best of something *(Participant 16: immuniser)*

Several parents were influenced in some way by Baby Charlotte's story. An emotional response served to change one parent's mind in favour of vaccination:

> I decided not to…I didn't know much about it and the immunisation has only been around or a wee bit not like the other immunisations that have been around for years, so I actually watched the television of that little girl who lost her arms and legs and you know I thought 'Yeah I might get them done' so I did *(Participant 6: immuniser)*

Others were more cynical and believed intense media coverage of Charlotte's story and her father's presence at public meetings to be a deliberate Ministry strategy designed to influence vaccine uptake:

> He [Peri Bisman] showed a lot of photos, a slide-show type thing…by the time he'd got to the end, it was like he was trying to brainwash you into having it done, not so much because it happened to his child but you really got the feeling that he was being paid by the Health Department to be there *(Participant 10: dual decision)*

Whatever the trigger, these were a cue to action and a reconnaissance phase.

Reconnaissance—Characterised by a 'hunt' for information about vaccine safety and efficacy, reconnaissance was undertaken in a variety of ways including reading medical and scientific articles, surfing the Internet, and both watching and reading populist media.

For many parents reconnaissance encompassed wanting to 'hear' a family member, friend or trusted health professional thoughts about vaccination. This information was influential for some parents in that it clarified information from other sources, confirmed their own thoughts, or gave them the confidence to act:

> The nurse at my kids' doctors, she's just brilliant. She's got little ones of her own so she's really up with it and I talked to her about her son getting immunised so that kind of helped out a bit too…it was more the side-effects, I thought yeah 'I can handle that'… just to know from someone that has had their kids done to actually know what happens *(Participant 6: immuniser)*

Parents wanted accurate, balanced, and referenced information from Ministry of Health officials and health professionals, however, most reported dissatisfaction and mistrust with 'official' information received:

> If they were able to say 'we haven't done it', just come out and say 'we haven't done it', be honest about it, but they couldn't be and I think that is why they had to make such a media deal of it because they didn't have the answers *(Participant 3: non-immuniser)*

Parents who consented to vaccinate, and those who declined, frequently described the Ministry of Health media publicity as 'scare mongering', 'controlling people through fear', 'fear driven', 'not balanced', and 'one-sided'. Even those with a more positive view made comments such as 'presenting the Government's received view' and 'needing in some way to twist it to force people to immmunise'.

Considerable dissatisfaction was expressed from both groups of parents with school-age children. Parents were concerned about the graphic nature of information given directly to children at school, in most cases without consent. This heightened fear amongst some children and parents felt it undermined their choice:

> They came home from school having seen the promo, they actually sent a video around the schools, they showed it to children as part of the health programme unbeknown to parents and they all came home within 2 days of each other saying, "Oh I've got to have this injection or we're going to get this horrible disease. So really faced with the fear of the children, I felt like I didn't have a choice *(Participant 16: immuniser)*

Concern was also voiced about the incentives used to encourage children to return consent forms within 3 days, and again parents felt under undue pressure to make a quick decision. Overall parental sentiment and need for information was summed up in the words of one parent who said:

> …just tell me everything you know and everything you don't know *(Participant 21: immuniser)*

Risk analysis—Balancing the risk of contracting meningococcal B versus the risks and benefits of the vaccine was interwoven with reconnaissance. Importantly for both

groups this process was taken seriously, was focused on individual children and was influenced by multiple factors relevant to each child, family experiences, and their context.

The perceived seriousness of meningococcal B and the perceived risk of contracting the disease were significant considerations for both sets of parents. Those who consented tended to have a strong perception of the seriousness of the disease particularly in relation to the diagnostic difficulties, the sudden onset and the fatal or disfiguring consequences. In consenting parents, the perceived seriousness of the disease was more dominant than the perceived risk of contracting the disease. Vaccine efficacy and safety issues seemed less of a concern.

In contrast, parents who declined immunisation may or may not acknowledge the seriousness of the disease but tended to perceive a lower risk of contracting the disease. Most did so by either denying the existence of an epidemic or stating that it was already in decline. Other reasons given included geographic location, ethnicity, socioeconomic status, health status, nutrition e.g. breastfeeding, and prevention measures such as not sharing drink bottles. For these parents, vaccine efficacy and safety issues were a dominant theme. They commonly referred to the lack of research, uncertainty about the number of vaccinations required to have an effect, and uncertainty about the duration of the coverage and the percentage of people protected by the vaccine.

Vaccine safety issues related to both short-term and long-term side-effects and the fact the vaccine only covered one strain of meningitis. However, perceptions were not always fixed, even after a decision had been made:

> She had no adverse effects to any of the jabs and that was another factor that if she had had a bad reaction to any of them I would have pulled her out of the programme *(Participant 12: immuniser)*

Having made a decision, the majority of parents continued to reflect on it. Most voiced a degree of uncertainty about their decision:

> 100% [confident] no but I'd say 90% we've done what we can to protect our children…hopefully there will be no side effects, no long term problems out of it *(Participant 15: immuniser)*

> It's a nerve-wracking decision all round, just making the decision about vaccination and I did at times feel like you're damned if you do and damned if you don't with this *(Participant 11: non-immuniser)*

Discussion

Our findings are similar to results from other qualitative studies suggesting parental beliefs about immunisation influence decisions.[8–10] Of the parents in our sample who previously believed in fully immunising their children, only 30% declined the MeNZB vaccine. Nevertheless our study has shown the decision to (or not to) vaccinate was far from routine. These results concur with a 1998 Australian study that found perceptions of susceptibility to disease and vaccine safety are dynamic.[8]

The biggest mass immunisation programme undertaken in New Zealand, MeNZB, confronted unique challenges. To affect the incidence of meningococcal disease in New Zealand, high levels of vaccine uptake were required, because immunisation with serogroup B meningococcal vaccines did not seem to reduce nasopharyngeal colonisation by the organism.[1]

National telephone research about the MeNZB vaccine conducted prior to roll-out showed that while disease awareness was high, perceptions of personal relevance was considerably lower.[11] Not surprisingly Ministry of Health publications and the mass media campaign surrounding the programme sought to address this issue by raising the perception of risk.

Statements in the Ministry fact sheets such as 'everybody in New Zealand is at high risk of contracting the disease' and 'the trials have found that the vaccine was safe' did little to raise individual perceptions of risk for most parents in this study or satisfy their concerns over vaccine safety and efficacy.

Indeed, recent New Zealand research indicates that information from official sources openly promoting immunisation is viewed by many parents as biased.[12] And in this study while parents who chose not to immunise were more likely to express a distrust of official information, those that did immunise were not prepared to take it at face value either.

Mass media campaigns to promote vaccination have been shown to be effective and the legitimacy of using stories and images of children affected by diseases to convey the seriousness of vaccine preventable diseases has been argued.[13,14] Media coverage of Charlotte's story heightened perceptions of risk and arguably was intended to illustrate the irrationality of choosing not to vaccinate. For a few parents in this study this strategy appeared to have the desired effect. However, our data also shows for other parents it had the opposite effect and may have undermined public trust in the Ministry of Health regardless of whether parents agreed with the immunisation or not.

Public trust is essential for immunisation programmes and should be protected.[15] Any undermining of trust could have negative consequences for future programmes.

School-based immunisation programmes have been shown to be effective.[16,17] Our data indicates both sets of parents had concerns about the graphic nature of information given directly to children, the short-time frame for return of consent forms, and the use of child focussed incentives.

Interestingly, prior to the meningococcal B programme the use of incentives in schools was reported as improving the rate of returned consent forms.[2] However, some parents in this study felt child focussed incentives, used in some schools to achieve a quick return of consent forms compromised their ability to make an informed decision.

The Ministry of Health, in the context of a 14-year epidemic of group B meningococcal disease, initiated the MeNZB immunisation program. For parents in our study intricacies within the family context were just as (if not more) important in reaching a decision. Therefore parents' perceived balance of risk for their individual child may not coincide with the health authorities balance of risk at the population level.[18]

The parents we interviewed all followed a similar decision-making process, were influenced by similar factors and sought information about the MeNZB vaccine from a variety of sources.

Limitations of this study were its small sample size and single geographic location. Despite these limitations, a qualitative method allowed us to gather comprehensive information from parents.

Conclusion

Despite different outcomes, most parents experienced comparable influences, and followed similar decision-making processes, which have been identified in previous studies regarding parental decision-making related to childhood immunisations.

Risk analysis (one of four interwoven decision-making phases) demonstrated that whatever decision is made, parents do not undertake the process lightly. Indeed, decisions regarding their children's wellbeing were made with considerable thought, questioning, and discussion. The use of fear, however, to encourage immunisation in the context of this campaign was not received favourably and requires debate.

Furthermore, parents' generally negative view of communication strategies used at this time also has implications and deserves consideration for future mass immunisation programmes.

Competing interests: None.

Author information: Paul B Watson, Senior Lecturer, School of Nursing, Christchurch Polytechnic Institute of Technology (CPIT), Christchurch;
Judy Yarwood, Principal Lecturer, School of Nursing, CPIT, Christchurch;
Kim Chenery, Clinical Nurse Educator, Child Health Cluster, Christchurch Hospital, Canterbury District Health Board, Christchurch

Acknowledgements: Funding for the study was provided by the Christchurch Polytechnic Institute of Technology, Academic Research Committee. We also thank the parents who generously gave their time to tell us their experience of making decisions for their children about the MeNZB vaccine, as well as Lisa Phillips who transcribed the interviews.

Correspondence: Paul Watson, Senior Lecturer, School of Nursing, Christchurch Polytechnic Institute of Technology, PO Box 540, Christchurch; Fax: (03) 940 8019; email: watsonp@cpit.ac.nz

References:

1. Thomas M. Prevention of group B meningococcal disease by vaccination: A difficult task. N Z Med J. 2004;117(1200). http://www.nzma.org.nz/journal/117-1200/1016
2. Petousis-Harris H, Turner N, Soe B. Parent views on school based immunisation: A survey of parents of year 1 and 6 in three diverse Auckland schools. N Z Fam Physician. 2004;31:222–8.
3. Wroe AL, Turner N, Salkovskis PM. Understanding and predicting parental decisions about early childhood immunisations. Health Psychol. 2004;23:33–41.
4. Petousis-Harris H, Goodyear-Smith F, Godinet S, Turner N. Barriers to childhood immunisation among New Zealand mothers. N Z Fam Physician. 2002;29:396–401.
5. Cofi Delgi Atti ML, Rota MC, Bella A, Salmaso S. Do changes in policy affect vaccine coverage levles? Results of a national study to evaluate childhood vaccination coverage and reasons for missed vaccination in Italy. Vaccine. 2004;22:4351–7.
6. Gellin BG, Maibach EW, Marcuse EK. Do parents understand immunisations? A national telephone survey. Pediatrics. 2000;106:1097–102.
7. Hobson-West P. Understanding vaccination resistance: Moving beyond risk. Health Risk Soc. 2003;5:273–83.
8. Bond L, Nolan T, Pattison P, Carlin J. Vaccine preventable diseases and immunisations: a qualitative study of mothers' perceptions of severity, susceptibility, benefits and barriers. Aust N Z J Public Health. 1998;22:441–6.

9. White GE, Thomson AN. ‘As every good mother should’. Childhood immunisation in New Zealand: a qualitative study. Health and Social Care. 1995;3:73–82.
10. Meszaros J, Asch DA, Baron J, et al. Cognitive processes and the decisions of some parents to forgo pertussis vaccination for their children. J Clin Epidemiol. 1996;49:697–703.
11. Sexton, K, Lennon, D, Oster P, et al. Proceedings of the Meningococcal Vaccine Strategy World Health Organization Satellite Meeting, 10 March 2004, Auckland, New Zealand. N Z Med J. 2004;117(1200). http://www.nzma.org.nz/journal/117-1200/1026
12. Hamilton M, Corwin P, Gower S, Rogers S. Why do parents choose not to immunise their children? N Z Med J. 2004;117(1189). http://www.nzma.org.nz/journal/117-1189/768
13. Leask J, Chapman S. ‘The cold hard facts’ immunisation and vaccine preventable diseases in Australia’s newsprint media 1993-1998. Soc Sci Med. 2002;54:445–57.
14. Leask JA, Chapman S. An attempt to swindle nature: Press anti immunisation reportage, 1993-1997. Aust N Z J Public Health. 1998;22:17–26.
15. Verweij M, Dawson A. Ethical principles for collective immunisation programmes. Vaccine. 2004;22:3122–6.
16. Seid M, Simmes DR, Linton LS, et al. Correlates of vaccination for hepatitis B among adolescents: Results from a parent survey. Arch Pediatr Adolesc Med. 2001;155:921–6.
17. Mansoor O, Sarfati D, Durham G. Is confidence in immunisation declining? N Z Med J. 1998;111:300.
18. May T. Public communication, risk perception, and the viability of preventive vaccination against communicable diseases. Bioethics. 2005;19:407–21.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Evaluation of the Palliative Care Partnership: a New Zealand solution to the provision of integrated palliative care

Eileen McKinlay, Lynn McBain

Abstract

Aims This study reports an external evaluation of a funded model of integrated palliative care the *Palliative Care Partnership*. Care is delivered by a partnership between palliative care coordinators (augmented by specialist hospice clinicians), general practitioners, practice nurses, and supported by community district nurses. Mandatory induction clinician education and other support is a prerequisite.

Methods A mixed method approach including in-depth, semi-structured interviews with a purposeful sample of stakeholders and analysis of routinely collected data. The study was undertaken in the MidCentral District Health Board area.

Results All stakeholders report favourably on the model of care. Data analysis shows the majority of MidCentral general practitioners and many practice nurses have completed training and cared for at least one patient using the funding stream of up to $400 per patient. Clinicians report increased clinical confidence and satisfaction. Patients/family describe best practice palliative care delivery. Funder and management organisation report robust quality and funding procedures.

Conclusions The *Palliative Care Partnership* is an effective model of funded palliative care in primary care. It utilises the enhanced skills of primary and specialist clinicians to provide cost effective palliative care and is a model worthy of replication nationally and internationally.

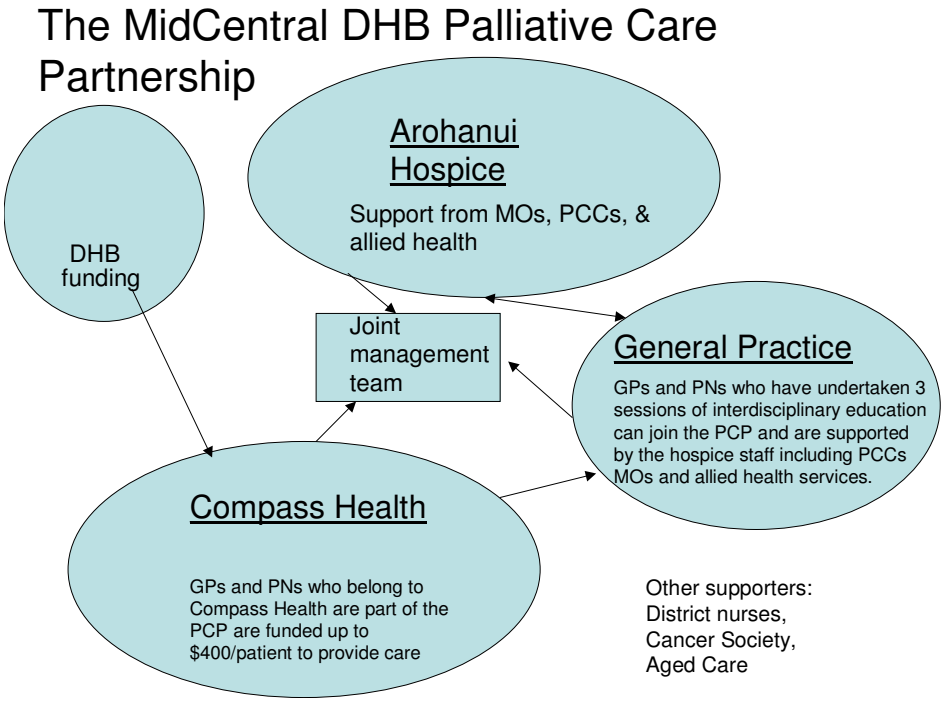
A basic human right at end-of-life is to receive quality palliative care. Ensuring that all who need palliative care receive it, challenges healthcare funders and providers.

The components of the *Palliative Care Partnership* (see Figure 1), have been fully described by Stewart et al.[1] In brief, as apposed to *specialist* palliative care services provided by hospices, the *Palliative Care Partnership* (PCP), funded by MidCentral District Health Board (MDHB) consists of *generalist* palliative care provided by a partnership of general practice teams (GPTs) (including general practitioner (GP) and practice nurse(s) (PN) from the same practice); specialist palliative care provided by registered nurse palliative care coordinators (PCCs) and other hospice staff; and health management services by Compass Health.

GPT attendance at induction and update interdisciplinary education provided by the Arohanui Hospice Education Unit and supply of a decision support manual are prerequisites to commencing PCP service provision.

Once formally accepted into the PCP, patients are assessed by PCCs, care-plans formulated and care given by the partners with the support of MDHB district nurses (DNs). A PCP management group provides oversight and quality assurance.

Figure 1. The MidCentral District Health Board Palliative Care Partnership



Methods

A mixed method evaluation approach was utilised.[2] Methods included qualitative interviews of a purposeful sample of stakeholders, analysis according to pre-determined evaluation questions of routinely collected quantitative data, and an audit of newly implemented 'shared' care-plans used by the PCP partners. The Central Ethics Committee approved this study in February 2006.

Interview schedules were developed in accordance with MDHB PCP contract specifications, tailored to participant group, and iterative according to participant involvement. The partner organisations committed to work closely with the evaluation team to ensure mutual understanding of the PCP and evaluation process.

Face to face or telephone interviews were undertaken in early 2006 and continued until data saturation was reached[3] with interviews audiotaped and transcribed.

Data from interviews were analysed by the research team using inductive thematic analysis,[4,5] identifying themes either held in common or disparate between those interviewed, and themes that coincided or were different from the literature. Member checking[6] for resonance was undertaking at the project midpoint by presentation and discussion of early findings. Data analysis was considered within the broad contract specification components and these components are used to report the results.

Results

Sixty-three people were interviewed either individually or in focus groups (see Table 1).

Table 1. Interviews undertaken

Data gathered	Number
Focus groups	6 GPT focus groups (and two additional telephone interviews) representing large and small practices in five areas in MDHB 1 PCC focus group
Face-to-face interviews	6 stakeholders (MDHB, Arohanui Hospice, Compass Health) 7 supporters (and one phone interview) including representation from MDHB, hospital palliative care team, Cancer Society, district and rural nurses, and an aged care residential facility team leader. 9 patients/family/friends
Hui	1 hui and one small group interview gaining viewpoints from Maori representatives

Referral management—Referrals to the PCP are made via a one-point entry system to Arohanui Hospice by a variety of health providers including GPTs; specialist palliative care services; secondary health services; DNs; Māori health providers (MHPs).

Referrers report a streamlined entry into the PCP with prompt assessment by a PCC. A small number of patients are declined entry to the PCP, typically the aged who also have a 'slow' terminal illness or those thought to be too early into the palliative care phase.

GPs felt early entry into the PCP maximised effective palliative care for patients, family and clinicians although PCP funding stakeholders have concerns over sustainability of early entry and longer care trajectory.

> I try and involve people as quickly as I can…there's really no point in me getting Arohanui involved if you've only got 3 days to die because you're not going to get any great benefit out of it (GPA Location 6)

Stakeholders feel individuals may still 'miss-out' on palliative care particularly those transient or not enrolled in general practice; Māori; have chronic illness or are still being 'actively' treated for a palliative illness.

> …what they do miss out is not so much about hospice care (it's all health care). If you start with deprivation you go out with deprivation…And there are some people who don't culturally identify with these services, like they just don't perceive they need them or want them or are scared of them (GPA Location 5)

Service provider—The evaluation confirmed GPTs in the PCP comprise GPs and PNs who hold current practicing certificates, having undertaken three sessions of mandatory and then update education. The majority of MDHB's GPs (n=56) belong to the PCP. All GPTs who have joined the PCP have cared for at least one patient.

Specialist palliative care assessment—A standardised assessment is undertaken by a PCC integrating "physical, spiritual, cultural, and psychosocial elements of the patient and family/whanau."[7]

Five PCCs employed by Arohanui Hospice work in defined geographical areas with particular GPTs, DNs and aged care providers. PCP partners report PCCs hold pivotal roles and as having close interpersonal relationships with referring partners.

GP clinicians spoke warmly of the individual PCC they work with, regarding them as close collaborators.

> …the good thing with the partnership is that I know (the PCC's) phone number by heart, I can phone her up any time I need to or want to. We know each other well enough now to know what our abilities are…(GPA Location 1)

And supporting provision of after-hours care.

> There's also an important issue about getting more comfortable to take on a palliative care patient now with this new contract. Because prior to it you felt isolated about providing after-hours cover (GPB Location 2)

PCCs also support aged residential care providers.

> …now they get in touch with us more, so that they're ringing us more often. Yes definitely now and the process is a lot more defined now—like before we were sort of maybe missing a few bits of information that probably now is quite vital…(aged care team leader)

Planning—PCCs develop a plan of care with patient and family which is a "…*living document to which all parties contribute and update*."[7] The care plan is retained in the home in a satchel of other information and is intended to be taken to medical appointments.

> I think having the folder. They can go home and think "Now what does that mean?" They'll ask. I think they are a lot more empowered…if they are too unwell and don't (want) to be bothered, quite often someone in the household will read it …(PNA Location 1)

A sample of 'shared care-plans' used by partners giving in-home care usually at end-of-life, were audited against criteria for completion of assessment, care-plans, goals for care and completion of progress notes. All records met the criteria for completion by both DN and PCC and one record for completion by DN, PCC, and GP.

Provision—In New Zealand, historically GPs, DNs, and families have undertaken provision of palliative care. In the last 30 years, hospices have played a major role in both advocating for and providing in-patient and community palliative care services. An unintended consequence sometimes expressed was of hospices taking-over the general practice palliative care role.

> …before this (the PCP) happened I tended to lose my patients as soon as they went into hospice. (Now) the ones I have, we were able to keep in touch (GPA location 4)

In contrast, the PCP aims to intentionally support general practice to provide early sustainable palliative care to patients and family often known for many years.

> I think the other thing that helps for us and for our patients is that they know we're involved and for a lot of our patients we are their doctor, whichever one of us. And whichever one, or one of the nurses, are their nurses…(GPB Location 2)

GPTs felt well supported by the content and manner in which the specialist advice was offered by the specialist hospice staff.

> I trust them implicitly…and it's very good that they are at the end of that phone, 24 hours per day. So if you have any doubt, and you think "Oh, I don't know about this", you can ring somebody (PNA Location 1).

From the inception of the project in mid 2004 until September 2006, there have been 585 patients in the PCP. Ethnicity recording is incomplete with numbers classified as 'unknown' or 'other'. Currently there are lower then MDHB rates of Māori, Pacific, and Asian.

Of the $400 per patient able to be claimed through free or subsidised consultations or home visits, between 2004 and September 2006 the average cost per patient was $152.52, with only 34 patients exceeding $350 of claims.

There have been 2191 claims on PCP funding with a range of between 1–25 claims per GP with 11 GPTs caring for more than 15 patients, 34 GPTs caring for 5 to 14 patients, and 12 GPTs caring for 1 patient. Most PCP patients had cancer (55%), or cardiovascular and respiratory conditions (9%) and the balance 'unknown' or 'other'. The most frequent reasons for making a claim (given that a GPT could claim for up to four items at one time) were medical consultation (28%), repeat prescription (29%), medication review (12%), symptom control (10%), and pain management (8%).

GPs appreciated being able to offer patients PCP-funded visits (CarePlus funding was being implemented during the study and funding interfaces yet to be addressed). Remaining patient barriers to PCP care noted were cost of transport and/or medication, particularly medication not currently PHARMAC subsidised.

GPs also felt being able to claim the PCP subsidy encouraged them to retain their patients when they needed palliative care.

> Its been nice as a GP not to be out of pocket for going to see people rather than basically doing freebies all the time…ok, cost does get in the way of clinical practice, there's is no doubt about that (GPA Location 1)

PNs involvement in the PCP has not entirely embedded. Whilst PNs were pleased to be included and valued the induction education, they did not always feel this preparation translated into day-to-day work and were reluctant to 'claim' for PCP activity.

> I can't say that I'm always that good at it (claiming) and there's often like phone ones that I will talk, well talk to someone on the phone about how they are going and unless someone says to me did you claim that or remind me (PNA Location 5)

Some PNs felt there were already enough people involved in providing palliative care whereas others described significant but often unarticulated roles including phone consultations to check-up on people, addressing or referring-on symptom control issues, accessing benefits, clarifying/reiterating medical information, organising scripts or DN support, finding equipment or smaller-size clothes and post bereavement support.

DNs hold a key role in the PCP although are not formal partners. Similar to general practice, they typically have longtitudinal relationships with patients and family often prior to referral into the PCP. DNs noted changes since the PCP began including an increased workload with more patients being cared for in the community and lines of communication changing from liaison with hospice staff to liaison with the GP and support from PCCs.

Monitoring and measurement—The PCP management team represents the different partner organisations. It operates under an annually reviewed Terms of Reference, with regular minuted meetings and receives and considers internal reports, audits, evaluations and routinely collected data and modifies business rules as necessary. The PCP management team comprised a stable and highly effective workgroup.

Commitment to the partnership has overcome previous perceived philosophical differences.

> I think it's really functional. There's a lot of openness. There's a lot of trust. And we do come to that table and we do have grievances and it can get a little bit testy at times. But we work

> through it and it doesn't go outside that room. We work through those issues (MIPA/Compass Health key stakeholders)

Each partner organisation has their own internal quality assurance and variation was noted between these. Areas not currently audited include patients declined entry into the PCP and the auditing of palliative care delivery in general practice.

Education for general practice teams—Attendance by a GP and PN(s) from the same practice as a team has fostered a sense of team work as well as achieving proven educational outcomes from interdisciplinary education[8] ensuring all partners were aware of current best practice palliative care.

GPs appreciated the update in symptom control, pharmacology knowledge and information about social and psychological services.

> …not something you probably learned about before…they had some really neat algorithms and things to help us treat different symptoms and conditions (GPB Location 4)

PNs valued the educational content, and that they were paid to attend and at the same rate as GPs.

> I really liked the fact that the GPs and the practice nurses did it together because of the stuff was very medical and it was really good to be in on that and to have more of an understanding …And then the flip side of that…there was a lot of nurse talk…Some of the GPs I thought gained more of an appreciation about perhaps the way we approach things (individual PN interview)

GPTs valued of the 'purple', reference manual and the researchers noticed that GPs shelved it close by their desk.

> …the other thing that's benefited has really being educated more about how to manage terminally ill people. There's a very good brochure booklet, the folder (purple folder) that we use. (GPA Location 6)

The experience of Māori receiving palliative care services—MDHB strategy has a strong focus on meeting the needs of Māori and in considering whether new services adequately redress existing inequality.[9] A hui (meeting) facilitated by the former Kaiwhakaruru Hauora Māori/Māori Health Advisor for MDHB resulted in discussion about barriers to health care and wider cancer care; only those relating to the PCP are reported here.

There are eight MHPs within the MDHB region.[10] Of these, two provide GP services and can offer PCP care. Being able to simultaneously enrol with a MHP and also with a GP practice may act both as a facilitator and a barrier to accessing PCP services.

The experience of patients/family/friends—Patients and family/friends each hold a different yet equally valid viewpoint complementing that of the health professional stakeholders.[11,12–14]

Current patients were very satisfied with care from the PCP. They appreciated the skill and human warmth of those involved (including family) and the frequent contact and assessment.

Patient difficulties included difficulty or delays in obtaining equipment or other services, financial concerns due to medication payments and duplication of services. Not everyone was aware of services accessible through the Cancer Society or Work and Income or Ministry of Health (transport allowance).

All but one family/friend were satisfied with past PCP care; the person not in agreement had had a family member being cared for in an aged care setting.

Family/friends suggested night-carer relief, more personal care assistance, financial and practical help in improving the home environment to assist care; and not all were aware of equipment and support available.

Discussion

Evaluation is a key component of health services research. It aims to describe and record change and what has led to change, establish whether there are linkages between theory and practice,[15] generate new knowledge and/or enable application of knowledge to other contexts.

Specialist palliative care teams are known to improve palliative care patient outcomes[16] but limited work has been undertaken to evaluate models of primary palliative care supported by specialist palliative care.[17–20] Research undertaken suggests services are cost-effective.[21,22] No similar New Zealand work has been undertaken although a recent New Zealand report highlights the need to do this.[23]

In the MDHB region, the PCP partnership grew from an almost simultaneous recognition by different stakeholder groups that there was potential for a different service delivery model appropriately utilising the skills of generalist and specialist clinicians and providing patient and family with seamless palliative care services from the time of referral.

The skill of implementing an integrated service is not just in meeting the service specifications but also ensuring there is effective collaboration between partners. The utility and functionality of the joint management group, utilisation of a care pathway[24] including induction education, use of PCCs , and reference manual with decision support has facilitated this. Care pathways use standardised information and are believed to minimise delays, facilitate and prioritise use of resources, specify anticipated treatment events, and build in quality processes, with the overall outcome being quality patient care.[15]

Across the country, MHPs hold DHB contracts for specific healthcare services,[10] for example to monitor diabetes. In this study contracts for specific care had the potential to result in silo-ed delivery. An example given was of a MHP nurse being funded to monitor a person's blood pressure in their home but not being funded to address the same person's palliative care needs.

People registered with a MHP without GP services and not concurrently enrolled with another general practice do not have straightforward access into the PCP (as a GP provider is a PCP requirement). Even those in a MHP with GP services are not eligible for subsidised medication.

Concerns noted in the Findings regarding equipment availability are not unique to the MDHB or the PCP. Third parties including 'needs assessment services' and occupational therapy review generally broker access to DHB-funded assistive equipment for any service including palliative care. This creates barriers both for clinicians in knowing what is available; whether the equipment is in stock and timing of supply and also for patients and family in knowing what equipment might be available and appropriate to ask for. In MDHB, when aware of need, PCCs actively advocate for equipment however can struggle to arrange supply or timely supply.

Limitations in this study include pragmatics of time, cost, and practical difficulty limited the number of stakeholder interviews particularly patients and families and

although a sampling framework identified key categories of people, not all opinions were canvassed.

Triangulation of results and use of routinely collected quantitative data aimed to minimise these effects similarly research protocol, and standard procedure for independent qualitative data analysis was rigorously maintained.

Data analysis shows the implementation of the PCP to be considered, staged, and supported by standardised mechanisms. Quality improvement mechanisms have been built-in at many stages and particularly through the joint management team processes. The external evaluation has made process improvement recommendations for each partner to be actioned by the PCP management quality assurance process.

In conclusion, the PCP is delivering comprehensive, holistic, and integrated palliative care incorporating both generalist and specialist palliative care skills to people and families/whānau and at a modest cost to the MDHB. The current service meets and exceeds current service specifications and has diversified beyond the original service brief. It is a model that should be considered for utilisation by other New Zealand and international health funders.

Competing interests: Eileen McKinlay and Lynn McBain convene a teaching programme for fourth year medical students called *Palliative Care in the Community*.

Author information: Eileen McKinlay, Lynn McBain; Senior Lecturers; Department of Primary Health Care and General Practice, Otago University, Wellington. (In addition, Eileen is a registered nurse and Lynn a general practitioner.)

Acknowledgements: We acknowledge the funding and support of the MDHB in undertaking this study and thank the partner organisations including Arohanui Hospice, Compass Health, and general practices in the region. We also thank patients/family/friends who took part in this study, in particular those who have since passed away.

Correspondence: Eileen McKinlay, Senior Lecturer in Primary Health Care, Department of Primary Health Care and General Practice, Wellington School of Medicine and Health Sciences, Box 7343, Wellington South. Fax: (04) 385 5539; email: eileen.mckinlay@otago.ac.nz

References:

1. Stewart B, Allen S, Keane B, et al. Palliative Care Partnership—a successful model of primary/secondary integration. N Z Med J. 2006;119(1242). http://www.nzma.org.nz/journal/119-1242/2235/
2. Patton M. Qualitative evaluation and research methods 2nd Edition. Newbury Park, California: Sage Publications; 1990.
3. Liamputtong Rice P, Ezzy D. Qualitative research methods. Melbourne: Oxford University Press; 1999.
4. Fitzpatrick R, Boulton M. Qualitative methods for assessing health care. In: Carter Y, Shaw S, Thomas C, eds. An introduction to qualitative methods for health professionals. London: The Royal College of General Practitioners; 1999:77–83.
5. Pope C, Ziebland S, Mays N. Analysing qualitative data. In: Pope C, Mays N, eds. Qualitative research in health care. London: BMJ Books; 1999.
6. Lincoln Y, Guba E. Naturalistic enquiry. Beverley Hills: Sage; 1985.
7. Palliative Care Partnership. Palliative Care Manual. Palmerston North: Arohanui Hospice and Compass Health; 2005.
8. Pullon S, Fry B. Interprofessional education in primary health care. Is it making a difference? J Interprofessional Care. 2005;19(6):569–79.

9. Ministry of Health. A health equity assessment tool (equity lens) for examining inequalities in health; 2004. http://www.moh.govt.nz/moh.nsf/ea6005dc347e7bd44c2566a40079ae6f/24474c7464606a5acc25700b0009d6f8?OpenDocument
10. New Zealand Government. Maori Health Providers; 2006. http://www.maorihealth.govt.nz/moh.nsf/menuma/Maori+Health+Providers
11. Gysels M, Hughes R, Aspinal F, et al. What methods do stakeholders prefer for feeding back performance data: a qualitative study in palliative care. Int J Qual Health Care. 2004;17(5):375–81.
12. de Raeve L. Ethical considerations in palliative care research. Palliative Medicine. 1994;8:292–305.
13. Corner J. Is there a research paradigm for palliative care? Palliative Medicine. 1996;10:201–8.
14. Aranda S. Conducting research with the dying: ethical considerations and experience. Int J Palliative Nursing. 1995;1(1):41–7.
15. Atwal A, Caldwell K. Do multidisciplinary integrated care pathways improve interprofessional collaboration. Scand J Caring Sci. 2002;16:360–7.
16. Hearn J, Higginson I. Do specialist palliative care teams improve outcomes for cancer patients? A systematic literature review. Pall Med. 1998;12:317–32.
17. Buikstra E, Pearce S, Hegney D, Fallon T. SEAM- Improving the quality of palliative care in regional Toowoomba, Queensland: lessons learned. Rural and Remote Health. 2006;6(1):415.
18. Goldschmidt D, Groenvold M, Johnsen AT, et al. Cooperating with a palliative home-care team: expectations and evaluations of GPs and district nurses. Pall Med. 2005;19:241–50.
19. Thomas M, Quinn C. Palliative care: rapid redesign to support systemwide quality improvement. J Healthcare Quality. 2002;24(1):25–9.
20. Taylor A. Improving practice with the Liverpool Care Pathway. Nursing Times. 2005;101(35):36–7.
21. Finlay I, Higginson I, Goodwin D, et al. Palliative care in hospital, hospice, at home: results from a systematic review. Annals of Oncology. 2002;13 Supp(4):257–64.
22. Setoyama O. Issues in implementation of palliative care- need for a redefinition of the outcome of palliative care. Japanese J Can Chemotherapy. 2000;27(9):1390–5.
23. Palliative Care Advisory Committee. Palliative Care in New Zealand Speaking with one clear voice: a national organisation for palliative care [letter]. Wellington; 2006. http://www.nurse.org.nz/misc/misc_palliative_care.htm
24. Kelsey S. Managing care pathways. Manag Dev Health Care. 2005;4(1):50–5.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Tobacco smoking prevalence in Pacific Island countries and territories: a review

Kumanan Rasanathan, Colin F Tukuitonga

Abstract

Aim To comprehensively review adult and youth smoking prevalence data in Pacific Island countries and territories (PICT).

Methods MEDLINE search for period 1986–2006 and search of World Health Organization and Centres for Disease Control and Prevention databases.

Results Smoking prevalence in PICT ranges from 22%–57% (males) and from 0.6%–51% (females). All PICT male populations (except Palau) report higher rates than in Australia and New Zealand.

Nauru, Tokelau, French Polynesia, New Caledonia, and Kiribati report high rates of female smoking. Youth rates of smoking range from 3%–68% (although unavailable for many PICT).

Palau, Northern Mariana Islands, Guam, Cook Islands, and American Samoa report very high levels of youth smoking in both males and females. Smoking prevalence appears to have decreased in the last 30 years in male populations with a variable picture in female populations.

Conclusions PICT continue to show high levels of smoking prevalence, with youth smoking rates particularly concerning. There is a need for more robust and systematic collection and publication of smoking prevalence data in PICT, especially youth data, but this should not delay urgently required action to reduce tobacco use in PICT. The Framework Convention on Tobacco Control provides a powerful tool, but its provisions should be implemented rapidly, particularly increased tobacco taxation.

The global burden of disease attributable to tobacco use continues to increase, particularly in low and middle income countries.[1] Unlike other low and middle income countries in Asia and Africa, most Pacific Island Countries and Territories (PICT) have long been in the epidemiological transition, with non-communicable disease comprising the majority of the disease burden.[2,3]

Cardiovascular disease is the major cause of mortality in PICT, as in other low and middle income countries,[4–6] and rates of cancer also appear to be increasing. Recent estimates of the population attributable fraction of cardiovascular disease in different PICT due to smoking range, in male populations, from 11% in Palau to 33% in Kiribati, and in female populations, from 0.4% in Federated States of Micronesia (FSM) to 32% in PNG.[7] Tobacco use is arguably thus the most important modifiable risk factor for disease in PICT.

Despite this, there is a lack of robust information about trends in the prevalence of tobacco smoking in PICT. The availability and quality of data varies greatly. Whilst estimates for smoking prevalence in PICT are available in World Health Organization

(WHO) publications,[8] these publications do not necessarily include the most up-to-date sources of information and some estimates date back to the 1980s. The only review of smoking rates in PICT evident in the peer-reviewed journal literature appeared in 1986 and this paper reported on only eight PICT.[9]

This paper aims to comprehensively review the literature to report on the current smoking prevalence in both adult and youth populations in the 22 PICT (shown in Table 1) served by the Secretariat of the Pacific Community (SPC) and included in the Western Pacific Region of the WHO. This paper also attempts to consider trends in tobacco use in PICT over the last 30 years and what these trends suggest for future health promotion efforts in the Pacific region to decrease the burden of disease related to smoking.

It should be noted that while this paper concentrates on smoking, chewing tobacco and other forms of tobacco use are important in some PICT, for example in Palau.[10]

Table 1. Pacific Island countries and territories*

American Samoa	Northern Mariana Islands
Cook Islands	Palau
Federated States of Micronesia (FSM)	Papua New Guinea
Fiji	Pitcairn Islands
French Polynesia	Samoa
Guam	Solomon Islands
Kiribati	Tokelau
Marshall Islands	Tonga
Nauru	Tuvalu
New Caledonia	Vanuatu
Niue	Wallis and Futuna

*As served by the Secretariat of the Pacific Community

Methods

The MEDLINE database was searched for the period January 1986 to December 2006, using the search terms ('tobacco' or 'smoking') and ('Pacific' or 'Oceania' or the names of the 22 PICT). The WHO's Infobase (http://www.who.int/ncd_surveillance/infobase/web/InfoBaseCommon/) and Tobacco-Free Initiative (http://www.who.int/tobacco/en/index.html) databases were also searched along with the Centres for Disease Control and Prevention (CDC)'s Youth Risk Behaviours Surveillance System (http://www.cdc.gov/healthyyouth/yrbs/index.htm) and Global Youth Tobacco Survey (http://www.cdc.gov/Tobacco/global/GYTS.htm).

Unpublished reports were found within the WHO and CDC databases and also sought by communication with tobacco control personnel in the region. The aim was to find all estimates of smoking prevalence in PICT that used random sampling techniques such as to provide nationally representative estimates that could be used to compare between PICT (for adults, smoking was defined as daily use, and for youth, smoking was defined as current use).

The most recent such estimate was used to assess smoking prevalence for each PICT in adult and youth populations, with estimates prior to 1990 not considered for inclusion as a current estimate. Time trends in tobacco use in the Pacific were considered by comparing current national estimates in Cook Islands, Niue, Fiji, Kiribati, Nauru, New Caledonia, and Samoa with those reported in the

aforementioned 1986 paper[9] which derived estimates from surveys conducted between 1975 and 1981. Prevalence estimates were also obtained from Australia and New Zealand for comparison.

Results

The MEDLINE search yielded 10 relevant papers.[9,11–19] Searches of the WHO and CDC databases and bibliographies identified a further 15 relevant documents and resources.[8,20–33] The surveys identified used differing methodologies and were undertaken over a range of years. As such, there are limitations to their comparability between different PICT and between different timepoints within the same state. However, the surveys utilised all aimed to produce nationally representative estimates.

Adult smoking prevalence

Post-1990 estimates for smoking prevalence were identified for American Samoa (survey year 2004),[33] Cook Islands (2004),[33] FSM (but limited to Kosrae, 1994),[14] Fiji (2002),[33] French Polynesia (1995),[8] Guam (2003),[17] Kiribati (1999),[8] Nauru (2004),[33] New Caledonia (1992),[8] Niue (2002),[29] Palau (1998),[22] Papua New Guinea (1990),[8] Samoa (2004),[33] Tokelau (2005),[33] Tonga (1998),[15] Vanuatu (1998),[21] and Wallis and Futuna (1996).[8] These estimates are summarised in Figure 1 along with comparisons from Australia (2001)[34] and New Zealand (2006).[35] Suitable estimates were not found for Marshall Islands, Northern Mariana Islands, Pitcairn Island, Solomon Islands, and Tuvalu.

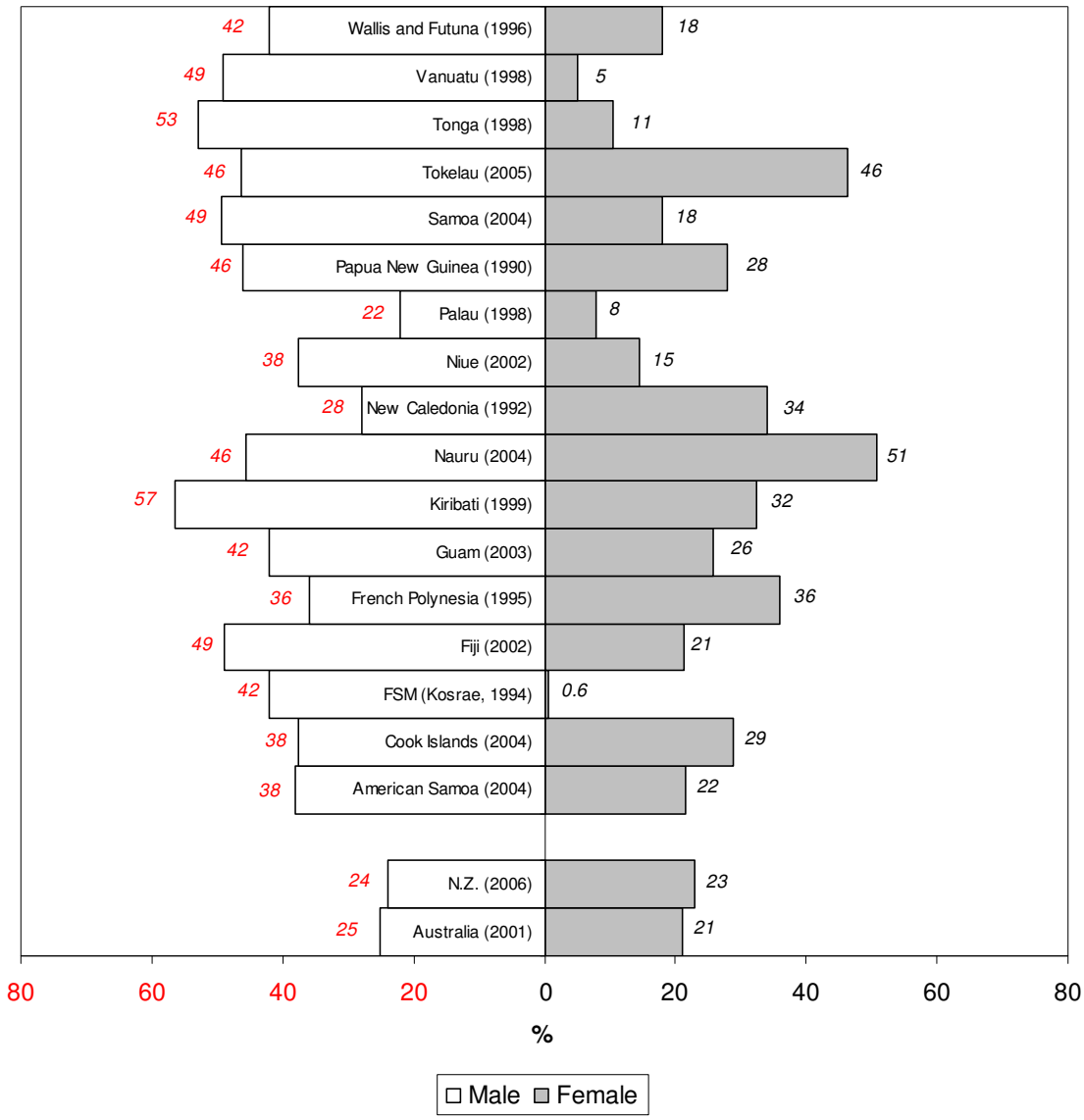
Survey definitions of adult varied. WHO STEPS data (discussed further below) was the source of estimates for American Samoa, Cook Islands, Fiji, Nauru, Samoa, and Tokelau. These surveys defined adult as 25–65 years old. The adult estimates for FSM were derived from 20–85 year olds; for French Polynesia, Niue, Papua New Guinea and Wallis and Futuna, from those 15 years and over; for Guam, 18 years and over; for Kiribati, 16 years and over; and for New Caledonia, Palau, Tonga and Vanuatu, from those aged 20 years and over. The estimates for smoking prevalence in Australia included those aged 18 years and over, and for New Zealand, those aged 15 years and over.

Adult smoking prevalence varied from 0.6% in FSM (Kosrae) women and 5% in Vanuatu women to 53% in Tongan men, 57% in Kiribati men, and 51% in Nauru women. Despite this great variation, in general, more men in PICT smoked than women with the exception of New Caledonia and Nauru, where more women smoked than men, and Tokelau and French Polynesia, where there was no gender difference.

Reported estimates of male smoking prevalence in PICT were all higher than recent estimates from Australia and New Zealand, with the exception of Palau. However, many PICT reported lower levels of male smoking prevalence than the age-standardised estimate of 41.3% reported in male Pacific peoples in New Zealand.[35]

There was much greater variation in female smoking prevalence estimates in PICT than in male prevalence estimates. Many PICT had lower levels of reported smoking in women than in New Zealand and Australia, in particular, FSM (Kosrae), Vanuatu, Palau, and Tonga.

Figure 1. Adult smoking prevalence in the Pacific[8,14,15,17,21,22,29,33–35]



In contrast, PICT such as Nauru, Tokelau, French Polynesia, New Caledonia, and Kiribati reported much higher levels of smoking for females than in New Zealand and Australian women. Most PICT reported lower prevalence of smoking in women than the age-standardised estimate of 33.8% in Pacific women in New Zealand.[35]

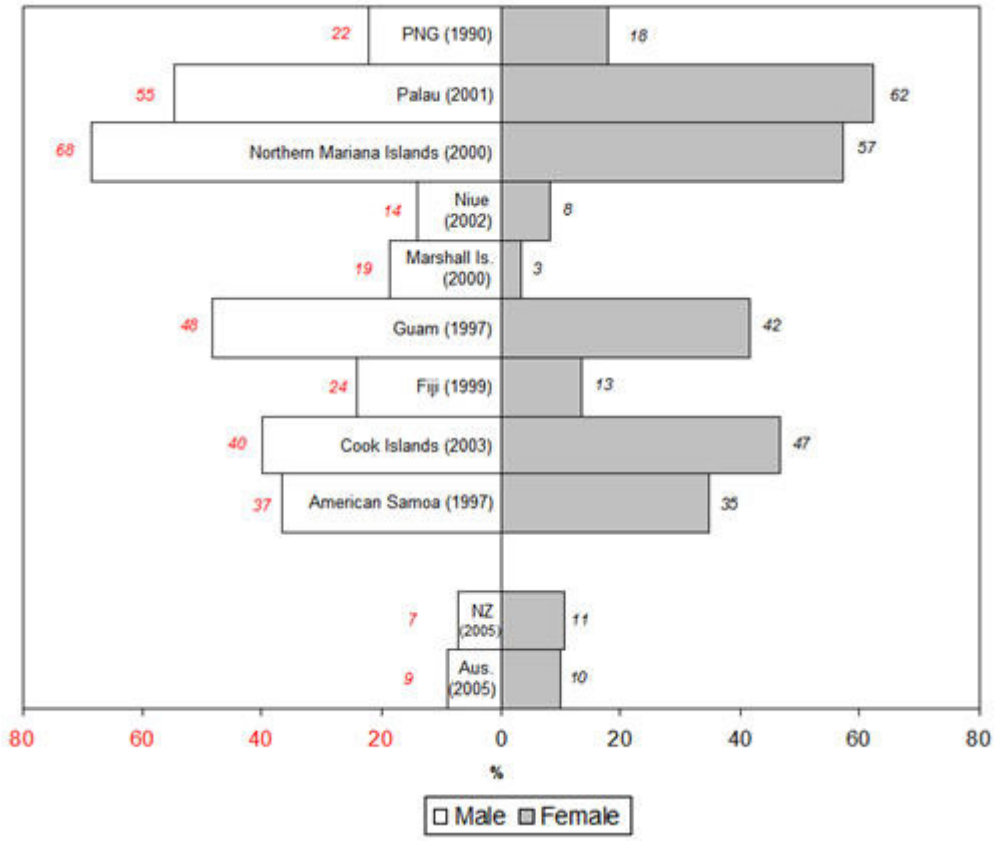
Age-stratified estimates were available for Fiji,[27] Niue, Palau, Samoa, Tonga (in a previous study[20] differing to the one used for the most recent country estimate), Nauru (again in a previous study[30]), and Vanuatu.

In general, there were not major differences between age groups, except for lower levels of smoking in elderly age groups (>65 years) in Nauru, Niue, and Tonga.

Youth smoking prevalence

Recent estimates for youth smoking rates were available for fewer PICT than for adult smoking levels. Post-1990 estimates were available for only American Samoa (survey year 1997),[31] Cook Islands (2003),[28] Fiji (1999),[23] Guam (1997),[32] Marshall Islands (2000),[18] Niue (2002),[29] Northern Mariana Islands (2000),[24] Palau (2001),[26] and Papua New Guinea (1990).[8] These estimates are summarised in Figure 2 along with youth smoking rates in New Zealand (2005)[35] and Australia (2005).[36] Estimates were also available for FSM but these were not stratified by gender but rather undertaken separately for the islands of Kosrae (41.2% current smoking prevalence in 13–15 year old students) and Pohnpei (35.1% prevalence in 13-15 year old students).[25]

Figure 2. Youth smoking prevalence in the Pacific[8,18,23,24,26,28,29,31,32,35,36]



Most of the youth prevalence rates identified were derived from the Global Youth Tobacco Survey (developed by CDC and WHO) and focused on adolescents aged 13 to 15 years old or from the Youth Risk Behaviours Surveillance System (also CDC) for American territories which covered 13–17 year old students.

The Marshall Islands' estimate covered 11–17 year old students, the Niue estimate was derived from 15–17 year old adolescents, and the Papua New Guinea estimate included 10–15 year old young people. The comparison estimates for Australia included 12–17 year olds students and for New Zealand, 14–15 year old students.

Due to this variability, the youth estimates are less comparable between PICT than the adult estimates for smoking prevalence described above.

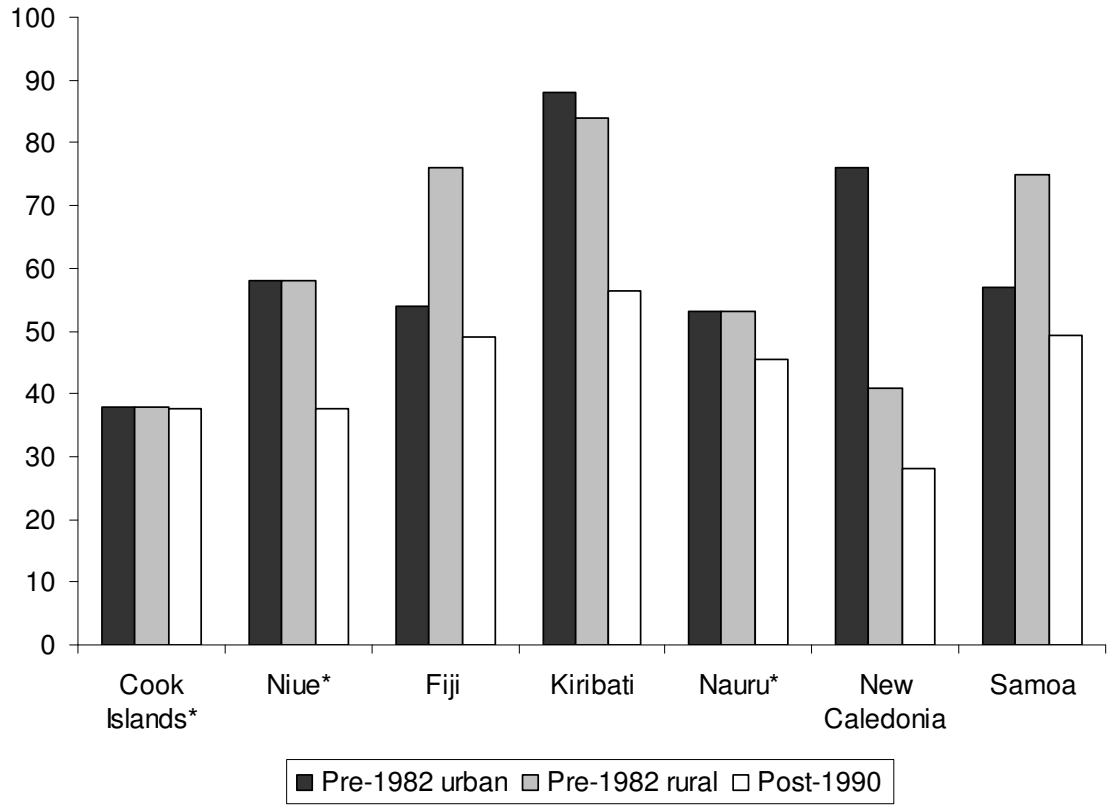
Palau, Northern Mariana Islands, Guam, Cook Islands, and American Samoa reported very high rates of youth smoking in both males and females, significantly higher than estimates for young people in New Zealand and Australia. These estimates were also higher than those reported for young Pacific people in New Zealand—10.2% for males and 14.5% for females.[35]

All PICT with youth estimates showed similar rates for male and female young people with the exception of Marshall Islands and Fiji, which reported much higher rates in young males.

Time trends in smoking prevalence

Comparisons of recent national estimates in Cook Islands, Niue, Fiji, Kiribati, Nauru, New Caledonia, and Samoa with estimates from 1975–1981 reported by Tuomilehto et al[9] are presented for adult male populations in Figure 3 and adult female populations in Figure 4. The older estimates included as adult those aged 20 years and over, except for the Nauru estimate which included those aged 15 years and over. Older estimates of youth smoking prevalence were not identified.

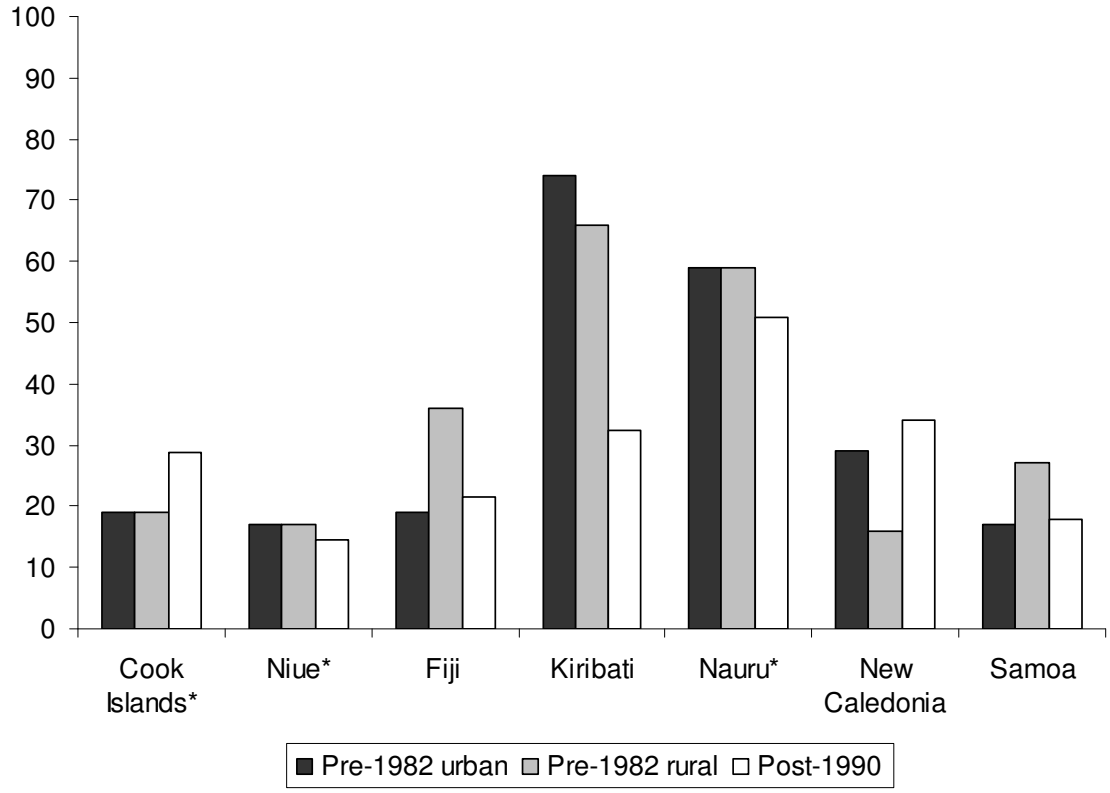
Figure 3. Adult male smoking prevalence (%) time trend[8,9,29,33]



*For Cook Islands, Niue, and Nauru, a single pre-1982 estimate was identified and thus this figure is used for both urban and rural populations.

There are significant difficulties in comparing estimates in this way due to the differing methodologies employed in these surveys. However, smoking rates appeared to have decreased in most male populations. Time trends for female populations were variable with increasing rates noted in Cook Island and New Caledonian women.

Figure 4. Adult female smoking prevalence (%) time trend[8,9,29,33]



*For Cook Islands, Niue, and Nauru, a single pre-1982 estimate was identified and thus this figure is used for both urban and rural populations.

Discussion

PICT continue to show very high levels of tobacco smoking, with the exception of a few female PICT populations. Whilst there is some evidence that adult male smoking rates in PICT have decreased in the last 30 years, smoking rates remain high and there are concerning levels of smoking in youth populations.

Overall, rates of smoking in PICT are higher than those seen in neighbouring high income countries such as Australia and New Zealand, although similar to prevalence for Pacific peoples in New Zealand.

These findings are concerning given the high burden of cardiovascular disease in PICT and increasing rates of cancer. In the context of struggling health systems,

action to combat tobacco use is thus a major priority to improve the health of populations in PICT.

There is a lack of comprehensiveness and rigour to some of the surveys included above from which estimates are derived. As such, some estimates may not be representative for all parts of the nations they derive from. In some PICT, it is very difficult to carry out genuinely national surveys given the multiple islands that these states contain. Furthermore, there are vast differences between populations within many PICT and thus these national figures may mask similar difference in tobacco use. However, this criticism potentially applies to national figures for all countries.

A more pressing concern is the age of some of the estimates, and the hitherto lack of regular monitoring of smoking prevalence using consistent methodology. As such, it is difficult to be sure of the time trends in smoking in PICT and the apparent decrease seen in adult male smoking prevalence should not be cause for complacency given the high rates of youth smoking and the latency of much smoking-related harm.

Many of these concerns about the availability and quality of smoking prevalence data in PICT will be addressed by the implementation and publication of WHO's STEPS survey for non-communicable disease risk factors, which includes tobacco use.[37]

STEPS surveys have already been carried out in many PICT. STEPS data for tobacco use in adults is available for Fiji, American Samoa, Samoa, Tokelau, Nauru, and Cook Islands, and these estimates are used above. However, currently Fiji is the only PICT to have published a final report based on the STEPS survey results.

Data has also been collected in Marshall Islands, FSM, Vanuatu, Tonga, Solomon Islands, and Kiribati[33] though it is unclear when results will be available. As such, the availability and comparability of smoking prevalence data in PICT should improve greatly in the medium term. It is thus imperative that all PICT publish and disseminate results of STEPS surveys and continue to undertake smoking surveys using STEPS methodology at regular intervals to enable robust monitoring of smoking rates to inform tobacco control efforts.

Despite the reservations about the estimates reviewed in this paper, all of the reported surveys employed randomised sampling aimed at producing a representative estimate and/or have been recognised by WHO as providing a national estimate. Whilst there should be some caution exercised in interpreting differences between PICT, due to the differing methodologies used, the estimates provide the best indication available of tobacco smoking prevalence in these states.

Most importantly, given the large magnitude of smoking prevalence in many PICT, concerns about methodology should not obscure recognition of the scope of the continuing threat that tobacco poses to public health in these countries and territories and of the need for further action.

All PICT eligible to do so have become parties to the Framework Convention on Tobacco Control (FCTC)[38] and have pledged to make progress on legislation and policy consistent with the FCTC at the meetings of Pacific Health Ministers in Tonga in 2003 and Samoa in 2005.[39,40] This is encouraging but PICT need to make urgent progress on action to reduce tobacco use in their populations.

In particular, tobacco taxation remains an underutilised tool with rates of tax often much lower than in countries such as Australia and New Zealand. Indeed, tobacco taxation has been partially responsible for the decline in tobacco use in many high income countries such as New Zealand[41] and it has been suggested that tobacco

taxation is even more effective in low and middle income countries, judging from Papua New Guinea data.[42]

The findings reviewed here thus suggest that PICT should proceed with measures such as taxation and other tobacco control legislation and policy, as detailed in the FCTC, at the same time as they carry out improved monitoring of tobacco smoking rates in their states.

The importance of implementing STEPS monitoring of tobacco use will lie not in establishing the problem of tobacco use but rather in evaluating the effectiveness of tobacco control measures. Without such immediate action, the burden of disease from cardiovascular disease and cancer in PICT is likely to continue to increase at great cost to the peoples of the Pacific.

Competing interests: None.

Author information: Kumanan Rasanathan, Honorary Senior Lecturer; Colin F Tukuitonga, Associate Professor; Section of Pacific and International Health, School of Population Health, University of Auckland, Auckland

Correspondence: Dr Kumanan Rasanathan, Section of Pacific and International Health, School of Population Health, University of Auckland, Private Bag 92019, Auckland, New Zealand. Fax: +64 (0)9 373 7624; email: k.rasanathan@auckland.ac.nz

References:

1. Jha P, Chaloupka FJ, eds. Tobacco control in developing countries. Oxford: Oxford University Press; 2000.
2. Marshall M. The second fatal impact: cigarette smoking, chronic disease, and the epidemiological transition in Oceania. Soc Sci Med. 1991;33:1327–42.
3. Taylor R, Lewis ND, Levy S. Societies in transition: mortality patterns in Pacific Island populations. Int J Epidemiol. 1989;18:634–46.
4. Gaziano TA. Reducing the growing burden of cardiovascular disease in the developing world. Health Aff. 2007;26:13–24.
5. Woodward M, Reid MA. Cardiovascular disease in the Asia-Pacific region: challenges for health research and policy. Med J Aust. 2003;179:71–2.
6. Collins VR, Dowse GK, Cabealawa S, et al. High mortality from cardiovascular disease and analysis of risk factors in Indian and Melanesian Fijians. Int J Epidemiol. 1996;25:59–69.
7. Martiniuk AL, Lee CM, Lam TH, et al. The fraction of ischaemic heart disease and stroke attributable to smoking in the WHO Western Pacific and South-East Asian regions. Tob Control. 2006;15:181–8.
8. Tobacco-Free Initiative Western Pacific Region. Country profiles on tobacco or health 2000. Manila: World Health Organization, Western Pacific Regional Office; 2000.
9. Tuomilehto J, Zimmet P, Taylor R, et al. Smoking rates in Pacific islands. Bull World Health Organ. 1986;64:447–56.
10. Lew R, Tanjasiri SP. Slowing the epidemic of tobacco use among Asian Americans and Pacific Islanders. Am J Public Health. 2003;93:764–8.
11. Li N, Tuomilehto J, Dowse G, et al. Prevalence of coronary heart disease indicated by electrocardiogram abnormalities and risk factors in developing countries. J Clin Epidemiol. 1994;47:599–611.
12. Le Marchand L, Hankin JH, Bach F, et al. An ecological study of diet and lung cancer in the South Pacific. Int J Cancer. 1995;63:18–23.
13. McGarvey ST. Cardiovascular disease (CVD) risk factors in Samoa and American Samoa, 1990-95. Pac Health Dialog. 2001;8:157–62.

14. Shmulewitz D, Auerbach SB, Lehner T, et al. Epidemiology and factor analysis of obesity, type II diabetes, hypertension, and dyslipidemia (syndrome X) on the Island of Kosrae, Federated States of Micronesia. Hum Hered. 2001;51:8–19.
15. Colagiuri S, Colagiuri R, Na'ati S, et al. The prevalence of diabetes in the kingdom of Tonga. Diabetes Care. 2002;25:1378–83.
16. Hiawalyer G. Smoking prevalence among young people in Papua New Guinea. Pac Health Dialog. 2002;9:209–13.
17. Centres for Disease Control and Prevention. State-specific prevalence of current cigarette smoking among adults—United States, 2003. MMWR Morb Mortal Wkly Rep. 2004;53:1035–7.
18. Chen TH, Ou AC, Haberle H, et al. Smoking rates and risk factors among youth in the Republic of the Marshall Islands: results of a school survey. Pac Health Dialog. 2004;11:107–13.
19. Mishra SI, Osann K, Luce PH. Prevalence and predictors of smoking behavior among Samoans in three geographical regions. Ethn Dis. 2005;15:305–15.
20. Woodward A, Newland H, Kinahoi M. Smoking in the Kingdom of Tonga: report from a national survey. Tob Control. 1994;3:41–64.
21. Carlot-Tary M, Hughes M, Hughes MC. Vanuatu non-communicable disease survey report. Noumea: Multipress; 1998.
22. Futterman A, Lyman A. Palau Substance Abuse Needs Assessment (SANA). Koror: Palau Ministry of Health; 1998.
23. Centres for Disease Control and Prevention. Fiji Global Youth Tobacco Survey (GYTS) fact sheet. Atlanta: Centres for Disease Control and Prevention; 2000.
24. Centres for Disease Control and Prevention. Commonwealth of Northern Mariana Islands Youth Tobacco Survey (YTS) fact sheet. Atlanta: Centres for Disease Control and Prevention; 2000.
25. Centres for Disease Control and Prevention. Federated States of Micronesia Global Youth Tobacco Survey (GYTS) fact sheet. Atlanta: Centres for Disease Control and Prevention; 2002.
26. Hansen L, Whipps VR, Lyman A. Palau Youth Tobacco Survey 2001. Atlanta: Centres for Disease Control and Prevention; 2002.
27. Pryor J, Cornelius M, Decourten M, et al. Fiji NCD STEPS report (draft) V4.9. Manila: World Health Organization, Western Pacific Region; 2002.
28. Centres for Disease Control and Prevention. Cook Islands Global Youth Tobacco Survey (GYTS) fact sheet. Atlanta: Centres for Disease Control and Prevention; 2003.
29. Laugesen M. Mission report on tobacco from Niue, 2003. http://www.who.int/ncd_surveillance/infobase/web/InfoBasePolicyMaker/Reports/reportViewer.aspx?surveyCode=101172a1&UN_Code=570&rptCode=ALL&dm=8
30. Shaw J. Unpublished data for Nauru; 2003. http://www.who.int/ncd_surveillance/infobase/web/InfoBasePolicyMaker/Reports/reportViewer.aspx?surveyCode=101168a1&UN_Code=520&rptCode=ALL&dm=8
31. Centres for Disease Control and Prevention. Youth Risk Behaviour Surveillance System: youth online: comprehensive results: American Samoa; 2006. http://apps.nccd.cdc.gov/yrbss/QuestYearTable.asp?cat=2&quest=Q30&loc=AS&year=Trend
32. Centres for Disease Control and Prevention. Youth Risk Behaviour Surveillance System: youth online: comprehensive results: Guam; 2006. http://apps.nccd.cdc.gov/yrbss/QuestYearTable.asp?cat=2&quest=Q30&loc=GU&year=Trend
33. World Health Organization, Secretariat of the Pacific Community. Prevention and control of noncommunicable diseases; 2007. http://www.wpro.who.int/NR/rdonlyres/08DD967F-C8B4-41CC-BC64-0C3153573C7C/0/NCD.pdf
34. White V, Hill D, Siahpush M, Bobevski I. How has the prevalence of cigarette smoking changed among Australian adults? Trends in smoking prevalence between 1980 and 2001. Tob Control. 2003;12(suppl ii):ii67–ii74.

35. Ministry of Health. Tobacco trends 2006: monitoring tobacco use in New Zealand. Wellington: Ministry of Health, 2006.
36. White V, Hayman J. Smoking behaviours of Australian secondary students in 2005. Melbourne: Cancer Council Victoria; 2006. http://www.nationaldrugstrategy.gov.au/internet/drugstrategy/publishing.nsf/Content/E1B70590AD4EF56DCA257225000EDCE9/$File/mono59.pdf
37. World Health Organization. STEPwise approach to chronic disease risk factor surveillance (STEPS); 2007. http://www.who.int/chp/steps/riskfactor/en/index.html
38. World Health Organization. Provisional summary record of the seventh meeting, fifty-seventh session of the regional committee of the Western Pacific region of the World Health Organization; 2006. http://www.wpro.who.int/NR/rdonlyres/CCED2785-95E9-4E16-B4A3-7C7BC667D97B/0/SR7.pdf
39. World Health Organization, Secretariat of the Pacific Community. Tonga Commitment to promote healthy lifestyles and supportive environment. Geneva: World Health Organization; 2003.
40. World Health Organization, Secretariat of the Pacific Community. Samoa commitment: achieving health islands: conclusions and recommendations. Geneva: World Health Organization; 2005.
41. Wilson N, Thomson G. Tobacco tax as a health protecting policy: a brief review of the New Zealand evidence. N Z Med J. 2005;118(1213). http://www.nzma.org.nz/journal/118-1213/1403
42. Chapman S, Richardson J. Tobacco excise and declining tobacco consumption: the case of Papua New Guinea. Am J Public Health. 1990;80:537-40. http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1404661

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Emerging modalities in dysphagia rehabilitation: neuromuscular electrical stimulation

Maggie-Lee Huckabee, Sebastian Doeltgen

Abstract

Aim The aim of this review article is to advise the New Zealand medical community about the application of neuromuscular electrical stimulation (NMES) as a treatment for pharyngeal swallowing impairment (dysphagia). NMES in this field of rehabilitation medicine has quickly emerged as a widely used method overseas but has been accompanied by significant controversy.

Methods Basic information is provided about the physiologic background of electrical stimulation. The literature reviewed in this manuscript was derived through a computer-assisted search using the biomedical database Medline to identify all relevant articles published until from the initiation of the databases up to January 2007. The reviewers used the following search strategy: [(deglutition disorders OR dysphagia) AND (neuromuscular electrical stimulation OR NMES)]. In addition, the technique of reference tracing was used and very recently published studies known to the authors but not yet included in the database systems were included.

Summary This review elucidates not only the substantive potential benefit of this treatment, but also potential key concerns for patient safety and long term outcome. The discussion within the clinical and research communities, especially around the commercially available VitalStim™ stimulator, is objectively explained.

Swallowing impairment (dysphagia) represents a substantial health issue in New Zealand and can be a clinical consequence of a broad spectrum of health conditions across the lifespan, including prematurity, developmental disability, traumatic brain injury, head and neck cancer, neurodegenerative disorder, and stroke. A closer investigation of only one aetiology suggests that the outcomes of this disorder are substantial.

The New Zealand Ministry of Health 2002/3 health survey documents that approximately 1 in 50 adults will suffer a cerebrovascular event (stroke) in their lifetime, with approximately 2724 new stroke events documented for the year 2002–3.[1] Of these, 40%–70% will present with dysphagia,[2] thereby affecting between 1–2000 new individuals per year—up to 44% of these will have persistent, chronic dysphagia and are at high risk for aspiration.[2]

In a study of medical outcomes, Smithard[3] reports that patients with dysphagia present significantly higher risks of chest infection and poorer nutritional state than stroke patients without dysphagia. The presence of dysphagia was also associated with significantly increased risk of death, disability, length of hospital stay, and institutional care.

When other factors were taken into account, dysphagia remained as an independent predictor of mortality. However, data also suggest that focused management of

swallowing impairment can reduce the burden of disease.[4] As such, rehabilitation practitioners have devised a number of behavioural strategies to alter biomechanical features of swallowing pathophysiology.[5–7]

There is increasing interest regarding the application of neuromuscular electrical stimulation (NMES) to rehabilitation practices for patients with dysphagia subsequent not only to stroke, but other structural, neurological, developmental, and functional conditions. It is the intent of this review article to inform the New Zealand medical community of basic definitions for NMES and to summarise relevant research, thus allowing for informed choices when approached by patients or clinicians with an interest in this therapeutic modality.

Definition and electrophysiological background

Neuromuscular electrical stimulation (NMES) is defined as "the external control of innervated, but paretic or paralytic, muscles by electrical stimulation of the corresponding intact peripheral nerves."[8] This is achieved through the carefully regulated administration of pulsed electrical current to nerves, myoneural junctions or muscles.[9] The therapeutic benefit of electrical stimulation is a consequence of skeletal muscle contraction and subsequent effects on strength, reaction time, and stamina.[10]

The current administered during electrical stimulation changes the ionic composition of the neural or muscular cell membrane and triggers transmission of a motor unit action potential with a subsequent motor response. The conduction of an action potential and the chemical synaptic transmission created by electrical stimulation involves the same processes of neurosecretion and chemoreception as a naturally occurring excitation. However, it differs from physiologic muscle activity in the ordering of muscle fibre recruitment, the synchronicity of individual motor units, and the intensity of stimuli required to produce these changes. These are important distinctions when considering the complex patterned motor event of pharyngeal swallowing.

Application of neuromuscular electrical stimulation in rehabilitation medicine

The literature in physical medicine and rehabilitation reports numerous applications for electrical stimulation: increasing muscle strength and range of motion, correcting contractures from spasticity, increasing sensory awareness and volitional muscle control, and decreasing antagonistic spasticity.[11]

Research has documented favourable effects of NMES on muscle function, cortical muscle representation, corticobulbar excitability, or motor recovery and thus supports the use of NMES in physical rehabilitation medicine.[12–15] Other research has reported no direct benefits from NMES, for example as a training method for elbow strength or as a treatment approach for motor recovery of gait in patients with stroke.[16,17]

This discrepancy in research outcomes highlights the need for both systematic investigation of rehabilitative effectiveness and clearer definitions of the scope of application for this modality.

Risks and contraindications

Electrical stimulators introduce active current into biologic tissue and at much higher intensity levels than endogenous current; thus, there are potential risks to patient safety. NMES is reported to be contraindicated in patients with demand type pacemakers, superficial metal implants or orthotics, skin breakdown, cancer, history of cardiac or seizure disorder, impaired peripheral nerve conduction systems, and pregnancy.[8] In addition to the general risks of NMES, there may be potential specific contraindications to the use of this technology in the head and neck region, as would be the logical application in swallowing.

In 1985, the USA Food and Drug Administration (FDA) required several warning labels on neuromuscular electrical stimulation devices that clearly raise concerns for the use of this device in swallowing rehabilitation. One of these states:

> Severe spasm of the laryngeal and pharyngeal muscles may occur when the electrodes are positioned over the neck or mouth. The contractions may be strong enough to close the airway or cause difficulty in breathing (FDA, 1985)[18]

These early warnings were based not on empirical research but on a logical concern for patient safety given the proximity of major arteries supplying the brain and of cranial nerves influencing respiratory function.

On 6 June 2001, the FDA granted approval for the use of a specific device, the VitalStim,™ for use in swallowing rehabilitation.[19] However, this approval also came with warnings:

> The long-term effects of chronic electrical stimulation are unknown. Stimulation should not be applied over the carotid sinus nerves. If electrodes are placed improperly and the unit is not used with the recommended frequency, intensity, and purse, it may cause laryngeal or pharyngeal spasm[19]

Many clinical researchers and basic scientists are investigating the safely and efficacy of this technology for swallowing rehabilitation. The quality of subsequent publications is variable and thus results require careful scrutiny before implementing this modality into clinical work.

The following review is based on a computer-assisted search using the biomedical database Medline to identify all relevant articles published until from the initiation of the databases up to January 2007.

The reviewers used the following search strategy: [(deglutition disorders OR dysphagia) AND (neuromuscular electrical stimulation OR NMES)]. In addition, the technique of reference tracing was used and very recently published studies known to the authors but not yet included in the database systems were included. It should be noted that at this stage of clinical development, there are no 'A' Level randomised, double blind, placebo-controlled trials supporting this modality.

A chronology of the literature in swallowing

Park et al[20] investigated the effect of oral electrical stimulation in 4 stroke patients with chronic dysphagia presenting the physiologic abnormality of "delayed swallowing reflex."

Oral stimulation was carried out using stimulation applied to the posterior soft palate through a custom designed palatal prosthesis. Stimulation parameters were set with a

duration of 200 μsec, repeated at 1-second intervals and intensity at the patient's individual pain tolerance.

In this limited sample, NMES did not facilitate more timely onset of swallowing, although half of the patients demonstration decreased penetration/aspiration. Although stimulation parameters were carefully explained, no clear justification was provided for the selection of these parameters and no comparison treatment was provided. However, this initial work suggested a positive effect of this modality on at least some biomechanical features of swallowing.

Freed and colleagues, who are responsible for development of the VitalStim™ device, investigated the clinical effects of NMES on 110 stroke patients with swallowing disorders; time post onset was not specified;[11] 83 patients were enrolled in an electrical stimulation (ES) group while 36 patients received what they considered to be a 'standard' treatment, that of thermal tactile stimulation (TTS). Randomisation for treatment group assignment was not applied.

Sixty-minute treatments was administered by the primary investigator daily for inpatients and 3 times per week for outpatients until the participants achieved a swallowing function score of at least 5 out of 6, or progress plateaud. Pre- and post-treatment videofluoroscopic swallowing studies (VFSS) were administered by the primary investigator; however, outcomes were based on a non-standardized scale based on the ability to safely swallow different food consistencies, rather than biomechanical performance.

On the surface, results of this study were promising. 98% of ES patients improved in some way, compared to 69% of TTS patients. Although the results generate considerable excitement and suggest a positive outcome, the design of this study limits the validity of the results in several ways. No justification or experimental control of stimulation parameters was undertaken or reported.

TTS is problematic as a comparison treatment. This poorly understood technique, consisting of providing a cold stimulus to the anterior faucial arches, has not withstood the rigours of empirical research. Outcome measures were based on a non-validated rating scale and ratings were assigned only by the primary investigator, who also provided the treatment.

Further, an unspecified number of patients in the NMES group received concomitant dilatation of the upper oesophageal sphincter, which is an accepted treatment in its' own right. These flaws corrode the validity of the positive results and illustrate the need to interpret the available research with caution.

Leelamanit et al provided synchronized electrical stimulation to 23 patients with moderate-to-severe dysphagia, specifically those diagnosed with "reduced laryngeal elevation."[22] Time post onset ranged from 3 to 12 months. NMES was provided through surface electrodes to the thyrohyoid muscles and presented at a frequency of 60 Hz, with an amplitude of 100 V for 3–30 treatments of 4 hours per day until they demonstrated improved swallow. Treatment outcomes were rated by the primary investigator based on a patient's ability to swallow more than 3 ml water without aspiration, adequate oral intake with weight gain, and improved laryngeal elevation during VFSS.

Twenty patients demonstrated clinical improvement, whereas 3 patients had no improvement; 6 patients relapsed on follow-up at 2 to 9 months, but regained benefits with another round of treatment. No control group was utilised in this project and, as

with the prior study, outcomes measures were by the primary investigator with no control for rater bias.

Burnett and colleagues followed with an investigation of self-triggered NMES on electromyographic activity of the mylo- and thyrohoid muscles in 9 healthy adults.[23] Electrical stimulation was synchronized with swallowing behaviour and delivered through hooked-wire electrodes at a frequency of 30 Hz and at the highest comfortable intensity level.

Rather than relying of subjective measures of treatment outcome, objective measures of muscle activity were calculated to document treatment effects. Analysis of the data revealed no significant change in amplitude or duration of muscle activity after self-triggered, synchronised electrical stimulation.

Five very recent manuscripts have attempted to more critically evaluate NMES specifically using the VitalStim™ device. Suiter et al[24] evaluated the influence of stimulation using this device on submental muscle activity in normal controls. An AB/BA treatment design revealed that 7 of 8 subjects exhibited no significant gains in myoelectric activity of the submental muscle group following 10 hours of NMES treatment using the treatment protocol described by Freed et al;[21] 2 subjects withdrew from the study due to mild skin irritations after treatment.

Humbert and colleagues at the National Institutes of Health (NIH) evaluated the effects of stimulation provided through 10 different surface electrode placements on hyolaryngeal movement in normal individuals at rest and during swallowing.[25] Maximum tolerated stimulation was provided using the protocol described by Freed et al.[21] Blind ratings of VFSS were evaluated using the NIH-Swallowing Safety Scale and specific biomechanical measures of the larynx and hyoid at rest and during swallowing under the conditions and stimulation and no stimulation.

In summary, significant hyolaryngeal descent occurred with stimulation at rest, and reduced hyolaryngeal elevation occurred during swallowing; both movements are antagonistic to functional swallowing. Stimulated swallows were also judged to be ‘less safe’ than nonstimulated swallows.

These studies have documented an absence of change or potential worsening of biomechanical function in unimpaired physiology, however, one could argue that similar effects may not be evident in patients with impaired physiology. Therefore, the same NIH research group evaluated the effectiveness of the VitalStim™ device in a population of patients with chronic pharyngeal phase dysphagia.[26] Time post onset of dysphagia was 6 months or more.

Treatment was provided according to the stimulation parameters advocated by the manufacturer of the device. VFSS was completed to allow for blinded measurement of hyoid movement and subglottic air column position during no-stim (no current induced); low-stim level (lowest intensity level, at which a participant felt a “tingling” sensation); and high-stim level (highest tolerable intensity without discomfort) conditions.

In the primary result, 8 of 10 participants demonstrated hyoid depression of up to 5–10 mm during stimulation of the muscles at rest, a movement trajectory antagonistic to swallowing. In the second result, low levels of stimulation (providing sensory input only) resulted in no improvement in aspiration or penetration but a slight improvement on the NIH Swallowing Safety Scale. Higher levels of stimulation, which would facilitate muscle contraction, had no effect on aspiration or penetration.

Overall the authors conclude that because of interference with hyolaryngeal excursion, "before such a tool is used in therapy, improved understanding of its immediate effects should be gained in the presence of specific types of swallowing difficulties before it is applied widely…"(p9).

Blumenfeld et al undertook at retrospective study of 40 consecutive patients who underwent traditional dysphagia therapy (a combination of therapeutic exercise, diet texture modifications and compensatory manoeuvres) compared to 40 prospective patients who underwent electrical stimulation therapy according to the VitalStim™ treatment paradigm.[27]

Patients were assigned a functional swallowing score, based on the non-validated scale used by Freed et al;[21] no control was provided for rater bias. In summary, patients who underwent electrical stimulation demonstrated significantly greater improvement than those who received traditional therapy.

Important to the interpretation of this study is that the traditional patients were evaluated retrospectively and the stimulated patients evaluated prospectively. Thus an overall advancement in clinical methods between the ensuing years may contribute to outcome differences.

Kiger et al also used a comparison of retrospective to prospective patients in their investigation of VitalStim™ therapy compared to traditional swallowing therapy.[28] Twenty-two patients with swallowing disorders were divided into two groups: the retrospective group received traditional dysphagia therapy while the prospective group received VitalStim™ therapy. Pre- and post-treatment VFSS or fibreoptic endoscopic evaluation of swallowing (FEES) were used to evaluate swallowing function based on a 7-point ordinal rating scale that described the patients' biomechanical swallowing functions as well as their ability to swallow different food consistencies.

The traditional treatment group improved more in the oral phase than the VitalStim™ group. No significant differences in post treatment outcomes for the pharyngeal phase, diet consistency tolerated, and oral intake measures were identified between the two groups.

Careful review of the current literature on the effectiveness of VitalStim™ therapy exposes an interesting observation. Generally, studies using non-blinded subjective outcome measures based on non-validated rating scales, reported potential success of the VitalStim™ treatment.[21,22,27] If, however, blinded and more objective measures such as myoelectric activity,[23,24] hyoid movement,[25,26] or biomechanics[28] were defined as outcome measures, then no positive effect was reported.

A careful evaluation of methods was undertaken by a research group in Manchester, UK to systematically investigated the effects of NMES of the pharynx and the faucial pillars, respectively.[29,30] Both studies documented that optimal stimulation parameters increased corticobulbar excitability and, importantly, were specific to the site of stimulation; i.e. optimal stimulation frequency for the pharynx (5 Hz) was different from that for the faucial pillars (0.2 Hz). Most importantly, certain stimulation parameters were found to be inhibitory to neural function underlying swallowing.

In the Power et al study, corticobulbar inhibition correlated with radiographically documented evidence of swallowing impairment in normal research participants.[30] Documentation of adverse effects urges the clinician to respect the obvious risks associated with NMES as a treatment in swallowing rehabilitation. The paucity of

research in this area and the subsequent lack of evidence for the effectiveness and safety of this treatment pose a major obstacle in justifying the use of this treatment in swallowing rehabilitation.

Summary

The application of neuromuscular electrical stimulation in the management of the dysphagic patient is a hotly debated topic internationally—it has divided the community of clinicians and researchers into two camps with very little "shades of grey" between.

There are vehement supporters of the technique and its clinical effectiveness that may not be willing to carefully review the literature with critical eye; conversely, there are equally staunch critics that perhaps are not receptive to the emergence of this new treatment modality.

In a chapter on neuromuscular electrical stimulation, Alon comments that:

> The present disarray, and the natural tendency to accept nonscientific, subjective and commercially motivated claims….may threaten the substantive potential that electrical stimulation can offer as an objective clinical modality[10]

This comment was offered in reference to NMES applications in physiotherapy but certainly rings true for swallowing rehabilitation as well.

In response to the emerging controversy, a position statement regarding the use of NMES in swallowing rehabilitation was ratified by the New Zealand Speech Therapists Association at the Annual General Meeting in April 2007. This statement concludes as follows:

> Based on available published literature and the ethical guidelines that govern clinical practice, it is thus the position of the New Zealand Speech-Language Therapists Association that application of this treatment modality in swallowing rehabilitation cannot be supported by empirical evidence, has the under-evaluated potential to cause harm, and does not meet the expectations for evidence-based practice. Application of this technique in the patient population is considered premature and should therefore not be utilised in the treatment of swallowing disorders until further evidence is available[31]

John Basmajian, MD, a physiatrist (a physician specialising in physical medicine and rehabilitation) in Canada and one of the premier researchers in motor potentials and rehabilitation, has posed a cautionary comment regarding rehabilitation practices. He comments, "Probably half of what we do in rehab is useless or harmful. Unfortunately I don't know which half that is" (Basmajian JV, Personal Communication, 1996).

It behooves us to struggle with the painstakingly slow progress of research to figure this out. Our early years of research and clinical practice related to dysphagia management have necessarily focused heavily on physiologic definition and diagnosis. As we begin to elucidate the nature of the disorder, we now are in a position to attend more heartily to treatment issues. In this endeavour, our best intentions to provide innovative care need to be balanced with judiciousness and a critical eye.

Competing interests: None.

Note: Significant sections of this review article form the basis for a position paper ratified by the New Zealand Speech Therapists' Association on the application of NMES in swallowing rehabilitation.

Author information: Maggie-Lee Huckabee, Senior Lecturer (Dysphagia and Neuroscience), Department of Communication Disorders at University of Canterbury and the Van der Veer Institute for Parkinson's and Brain Research, Christchurch; Sebastian Doeltgen, Doctoral Student, Department of Communication Disorders at the University of Canterbury and the Van der Veer Institute for Parkinson's and Brain Research, Christchurch

Correspondence: Maggie-Lee Huckabee, Van der Veer Institute for Parkinson's and Brain Research, 66 Stewart St, Christchurch. Fax: (03) 364 2760;
email: maggie-lee.huckabee@canterbury.ac.nz

References:

1. Ministry of Health. A Portrait of Health: Key results of the 2002/03 New Zealand Health Survey. Wellington: Ministry of Health; 2004.
2. Marik PE, Kaplan D. Aspiration pneumonia and dysphagia in the elderly. Chest. 2003;124:328–36.
3. Smithard DG, O'Neill PA, Parks C, Morris J. Complications and outcome after acute stroke. Does dysphagia matter? Stroke. 1996;27(7):1200–4.
4. Carnaby G, Hankey GJ, Pizzi J. Behavioural intervention for dysphagia in acute stroke: a randomised controlled trial. Lancet Neurol. 2006;5(1):31–7.
5. Langmore SE, Miller RM. Behavioural treatment for adults with oropharyngeal dysphagia. Arch Phys Med Rehab. 1994;75(10):1154–62.
6. Miller RM, Langmore SE. Treatment efficacy for adults with oropharyngeal dysphagia. Arch Phys Med Rehabil. 1994;75(11):1256–62.
7. Huckabee ML, Pelletier CA. Management of Adult Neurogenic Dysphagia. San Diego: Singular Publishing Group; 1999.
8. Baker LL, McNeal DR, Benton LA, et al. NeuroMuscular Electrical Stimulation: A Practical Guide. 3rd Edition. Downey, CA: Los Amigos Research and Education Institute, Inc; 1993.
9. Ragnarsson KT. The physiologic aspects and clinical application of functional electrical stimulation in rehabilitation. In: Downey JA, Myers SJ, Gonzalez EG, Lieberman JS (eds). The Physiological Basis of Rehabilitation Medicine. 2nd Edition. Boston, MA: Butterworth-Heinemann; 1994, p573–97.
10. Alon G. Principles of electrical stimulation. In: Nelson RM, Currier DP (eds). Clinical Electrotherapy. 2nd Edition. Norwalk, CT: Appleton & Lange; 1991, p35–101.
11. Kasman G. Surface EMG and Biofeedback in Physical Therapy, non-published course materials. Lake Forest, IL; 1994.
12. Glanz M, Klawansky S, Stason W, et al. Functional electrostimulation on poststroke rehabilitation: a meta-analysis of the randomized controlled trials. Arch Phys Med Rehabil. 1996;77:549–53.
13. Sullivan JE, Hedman LD. A home program of sensory and neuromuscular electrical stimulation with upper-limb task practice in a patient 5 years after stroke. Physical Therapy. 2004;84:1045–54.
14. Thompson AK, Stein RB. Short/term effects of functional electrical stimulation on motor-evoked potentials in ankle flexor and extensor muscles. Experimental Brain Research. 2004;159:491–500.
15. Kimberley TJ, Scott ML, Auerbach EJ, et al. Electrical stimulation driving functional improvements and cortical changes in subjects with stroke. Experimental Brain Research. 2004;154:450–60.
16. Holcomb WR. Effect of training with neuromuscular electrical stimulation on elbow flexor strength. Journal of Sports Science and Medicine. 2006:5;276–81.
17. Yavuzer G, Geler-Kulcu D, Sonel-Tur B, et al. Neuromuscular electric stimulation effect on lower-extremity motor recovery and gait kinematics of patients with stroke: a randomized controlled trial. Arch Phys Med Rehabil. 2006;87:536–40.

18. Department of Health and Human Services, Food and Drug Administration. Electrical Muscle Stimulator (EMS) Labeling Indications, Contraindications, Warnings, etc. Rockville, MD; 1985.
19. U.S. Food and Drug Administration (FDA) 510 (k) summary. Dysphagia Treatment Device; 6 Jun 2001. http://www.fda.gov/cdrh/pdf/k002410.pdf
20. Park CL, O'Neill PA, Martin DF. A pilot study of oral electrical stimulation on swallow function following stroke: an innovative technique. Dysphagia. 1997;12:161–6.
21. Freed ML, Freed L, Chatburn RL, Christian M. Electrical stimulation for swallowing disorders caused by stroke. Respiratory Care. 2001;46(5):466–74.
22. Leelaminit V, Limsakul C, Geater A. Synchronized electrical stimulation in treating pharyngeal dysphagia. Laryngoscope. 2002;112:2204–10.
23. Burnett T, Mann EA, Stoklosa JB, Ludlow CL. Self-triggered electrical stimulation during swallowing. Journal of Neurophysiology. 2005;94:4011–18.
24. Suiter DM, Leder SB, Ruark JL. Effects of neuromuscular electrical stimulation on submental muscle activity. Dysphagia. 2006; DOI: 10.1007/s00455-005-9010-7.
25. Humbert IA, Poletto CJ, Saxon KG, et al. The effect of surface electrical stimulation on hyolaryngeal movement in normal individuals at rest and during swallowing. J Appl Physiol. 2006;101:1657–63.
26. Ludlow CL, Humbert I, Saxon K, et al. Effects of surface electrical stimulation both at rest and during swallowing in chronicpharyngeal dysphagia. Dysphagia. 2006; DOI: 10.1007/s00455-006-9029-4.
27. Blumenfeld L, Hahn Y, LePage A, et al. Transcutaneous electrical stimulation versus traditional dysphagia therapy: a nonconcurrent cohort study. Otolaryngology- Head and Neck Surgery. 2006;135:754–7.
28. Kiger M, Brown CS, Watkins L. Dysphagia Management: An analysis of patient outcome using VitalStim™ therapy compared to traditional swallow therapy. Dysphagia. 2006; DOI: 10.1007/s00455-006-9056-1.
29. Fraser C, Power M, Hamdy S, et al. Driving plasticity in the human adult motor cortex is associated with improved motor function after brain injury. Neuron. 2002;34:831–40.
30. Power M, Fraser C, Hobson A et al. Changes in pharyngeal corticobulbar excitability and swallowing behavior after oral stimulation. American Journal of Physiology – Gastrointestinal and Liver Physiology. 2003;286:45–50.
31. Huckabee ML, Doeltgen S. Position Paper on Neuromuscular Electrical Stimulation in Swallowing Rehabilitation. New Zealand Speech Therapists Association, April 2007.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Nitrofurantoin-induced interstitial lung disease

Daniel Chou-yen Lin, Hasan Bhally

Long-term use of nitrofurantoin for urinary tract infection prophylaxis can cause serious pulmonary side effects as the following case illustrates.

Case report

A previously healthy 67-year-old woman presented to our emergency department in February 2007 with progressive dyspnoea for 2 weeks. This was preceded by 6 weeks of persistent dry cough. .She had no fever, wheezing, chest pain, or sputum production. She was a 20 pack-year ex-smoker.

She had no previous exposure to tuberculosis or industrial chemicals. However, she suffered from recurrent urinary tract infections which were diagnosed mostly from her symptoms of cystitis. In 2002 she was prescribed nitrofurantoin 50 mg daily for prophylaxis in the community.

On examination she appeared anxious and dyspnoeic. She was afebrile and normotensive with respiratory rate of 24 per minute and oxygen saturation was 94% on room air. Respiratory examination revealed dullness on percussion at both bases and widespread fine inspiratory crackles throughout both lungs. Arterial blood gas showed hypoxia with pO_2 8.9 kpa and respiratory alkalosis with PH 7.53 , and pCO_2 3.9 kpa on room air. Chest X ray (Figure 1) showed diffuse interstitial infiltrates.

Figure 1. Chest X-ray on admission

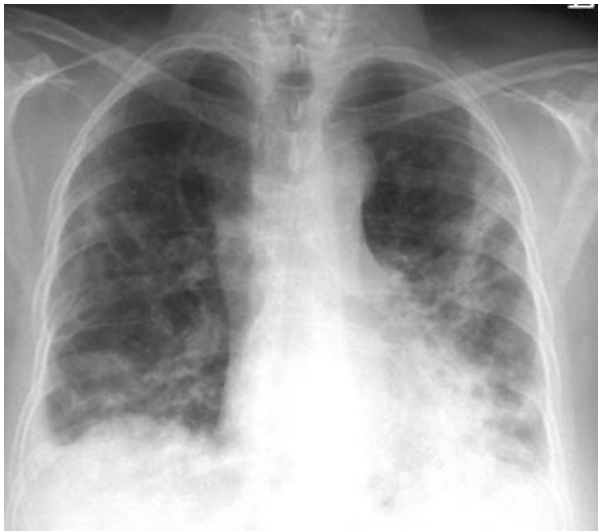
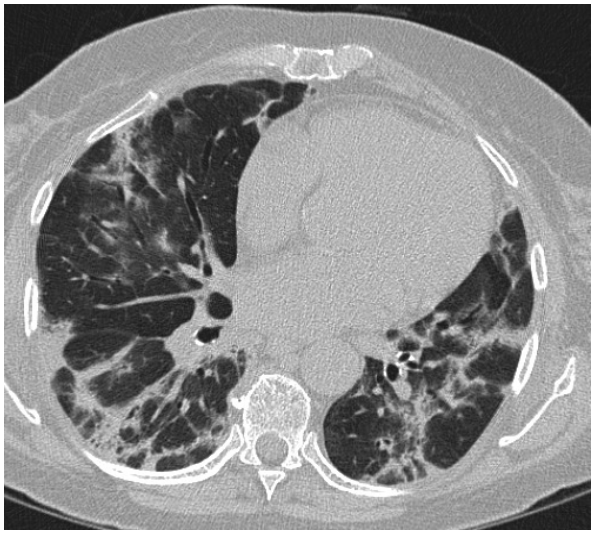


Figure 2. High-resolution computed tomography (HRCT) on admission

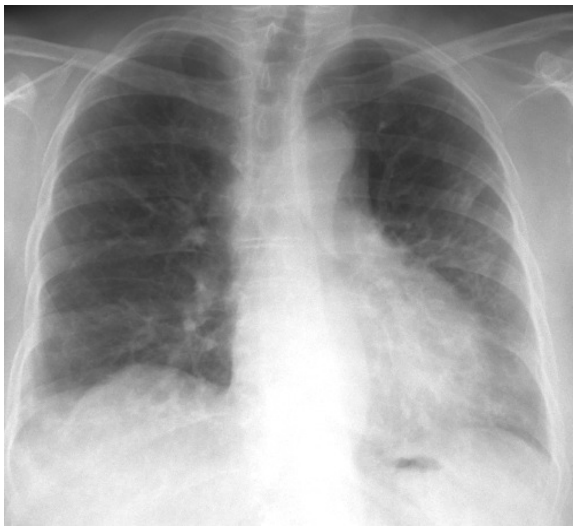


Initial treatment with roxithromycin was initiated. However, subsequent spirometry revealed severely restrictive lung disease with FEV_1 0.88 (53% predicted) and FVC 0.95 (47% predicted).

HRCT showed widespread ground-glass appearance in both lungs with organising pneumonia at both bases. There was no evidence of intestitial pneumonitis or fibrosis (Figure 2). This was felt to be characteristic of drug-induced lung disease. A diagnosis of nitrofurantoin-induced interstitial lung disease (NIILD) was made.

Nitrofurantoin was subsequently stopped and prednisone treatment at 40 mg OD was initiated. She had a prolonged hospital course and was eventually discharged with home oxygen. After 2 months, her symptoms had improved dramatically and a follow-up chest X-ray also showed marked improvement (Figure 3).

Figure 3. Chest X-ray 2 months after nitrofurantoin withdrawal



Discussion

Nitrofurantoin is a synthetic nitrofuran antimicrobial agent used for more than 50 years in urinary tract infections (UTIs).[1] It has been widely used for UTI prophylaxis especially for older females. Long-term use is known to cause serious adverse effects including pulmonary and hepatic toxicity, renal failure, and peripheral neuropathy.[1–3]

NIILD was first described in 1957 and it was found to have acute and chronic forms.[1] The former is a hypersensitivity reaction causing pneumonitis.[6] The latter is caused by the chronic pulmonary injury and inflammation resulting in organising pneumonia and fibrosis.[1,4] This is more common in older female exposed to nitrofurantoin for more than 6 months.[1–3]

Clinically, patients with chronic NIILD mostly present with cough and progressive dyspnoea for more than 3–4 weeks. It may be associated with chest pain, lethargy, or myalgia.[3] Most have inspiratory crackles on chest examination. Arterial blood gases frequently show hypoxia and the inflammatory markers may be normal or mildly elevated.[3]

Chest X-rays always reveal diffuse interstitial infiltrates throughout both lung fields. HRCT typically shows diffuse infiltrate with ground-glass appearance and organising pneumonia.[2–4]

NIILD has been reported from a number of countries.

In New Zealand, MEDSAFE in 2002 reported a 67-year-old female with NIILD died from hypoxia 3 months after diagnosis. And the Centre of Adverse Reaction Monitoring (CARM) Database revealed that 34% of the nitrofurantoin adverse reactions are associated with pulmonary damage.[1]

In Australia, Adverse Drug Reactions Advisory Committee (ADRAC) reported 46 nitrofurantoin-induced pulmonary reactions from 1995 to 2004; 87% of them were related to chronic nitrofuraintoin use (NIILD) with exposure between 8 months to 16 years. They all have evidence of interstitial pneumonitis or pulmonary infiltrates on radiological images. Elderly females were more commonly affected. Twelve cases had significant recovery but some patients had persistent lung damage; two patients died.[2]

Reports from USA revealed similar results. Mayo Clinic 2005 reviewed 18 patients with NIILD 1997–2002. Most of them were older females receiving nitrofurantoin with variable dose. They developed symptoms as soon as 10 months after exposure and symptoms persisted for 4 months on average before diagnosis. Half of them showed restrictive pattern on spirometry, but all had characteristic radiological features. After discontinuation of nitrofuraintoin, most showed symptomatic improvement after 2 months although most showed radiological improvement only after 36 months.[3,5]

The role of steroids in treating NIILD is unknown due to lack of study and this relatively rare disease.

In summary, chronic NIILD occurs primarily in older women with long-term use of nitrofurantoin. However, any patient on nitrofurantoin can potentially develop this disease if the exposure is 6 months or more. Their symptoms can be severe and disabling, and it is potentially lethal if the medication is not stopped quickly. Typical symptoms and signs, careful drug history, and radiological findings are important for diagnosis in early stages.

Although chronic NIILD is rare, it is recommended that nitrofurantoin should only be used in a proven and uncomplicated urinary infection, and the duration should not exceed 6 months when used for prophylactic purposes.[1] It also recommended withdrawing nitrofurantoin at the first sign of pulmonary damage. Most patients improve after discontinuation of nitrofurantoin after few months both with or without use of steroids.[5]

Author information: Daniel Chou-yen Lin, General Medical Registrar; Hasan Bhally, General Physician; North Shore Hospital, Auckland

Correspondence: Daniel Chou-yen Lin, General Medical Registrar, North Shore Hospital, 51A Francis Street, Takapuna, Auckland. email: dan_din2000@hotmail.com

References:

1. Pulmonary reactions with nitrofurantoin (May 2002). Prescriber Update (MEDSAFE). 2002;23(2)24–5. http://www.medsafe.govt.nz/profs/PUarticles/nitrofurant.htm
2. Pulmonary toxicity with long-term nitrofurantoin. Australian Adverse Drug Reactions Bulletin. 2004;23(4). http://www.tga.gov.au/adr/aadrb/aadr0408.htm

3. Chronic nitrofurantoin-induced lung disease. Mayo Clin Proc. 2005;80(10):1298–302.
4. Fawcett IW, Ibrahim NBN. BOOP associated with nitrofurantoin. Thorax. 2001;56:161.
5. Sheehan RE, Wells AU, Milne DG. Nitrofurantoin-induced lung disease: two cases demonstrating resolution of apparently irreversible CT abnormalities. Journal of Computer Assisted Tomography. 2000;24:259–61.
6. Cameron C, Bayliff CD, Patterson NAM. Nitrofurantoin-associated acute pulmonary toxicity. Can J Hosp Pharm. 1999;52:83–6.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Vacuum assisted closure devices in the elderly—a word of caution

Tejaswi Kandula, Justin A Roake, David R Lewis

Vacuum assisted wound closure (VAC) or topical negative pressure (TNP) therapy has been used for over a decade to facilitate the healing of several different types of wounds.[1,2] Successful use of this technique has been described in the treatment of chronic wounds; extensive, acute soft-tissue injuries; contaminated wounds; sternal infections and mediastinitis following cardiac surgery; full and partial thickness abdominal wall defects; perineal wounds; various types of orthopaedic trauma; burns; and diabetic foot disease.[1,2]

The reported complication rate of VAC is low and frequently not serious—ranging from minor bleeding and infection to delayed wound healing.[1,2] Occasional fatal or near fatal complications like sepsis, toxic shock syndrome, and exsanguinating haemorrhage have, however, been documented,[1,2] and as the use of VAC dressings becomes more prevalent, more complications may become apparent.

While following manufacturer's instructions for the use of VAC will serve to minimise these complications, the following case report highlights a simple problem with potentially serious consequences.

Case report

A 74-year-old lady (Mrs X) was referred to the vascular surgical team at Christchurch Hospital for severe ischaemic rest pain affecting her right foot. Following review of her arterial imaging (Figure 1), a right axillo-femoral bypass operation was carried out.

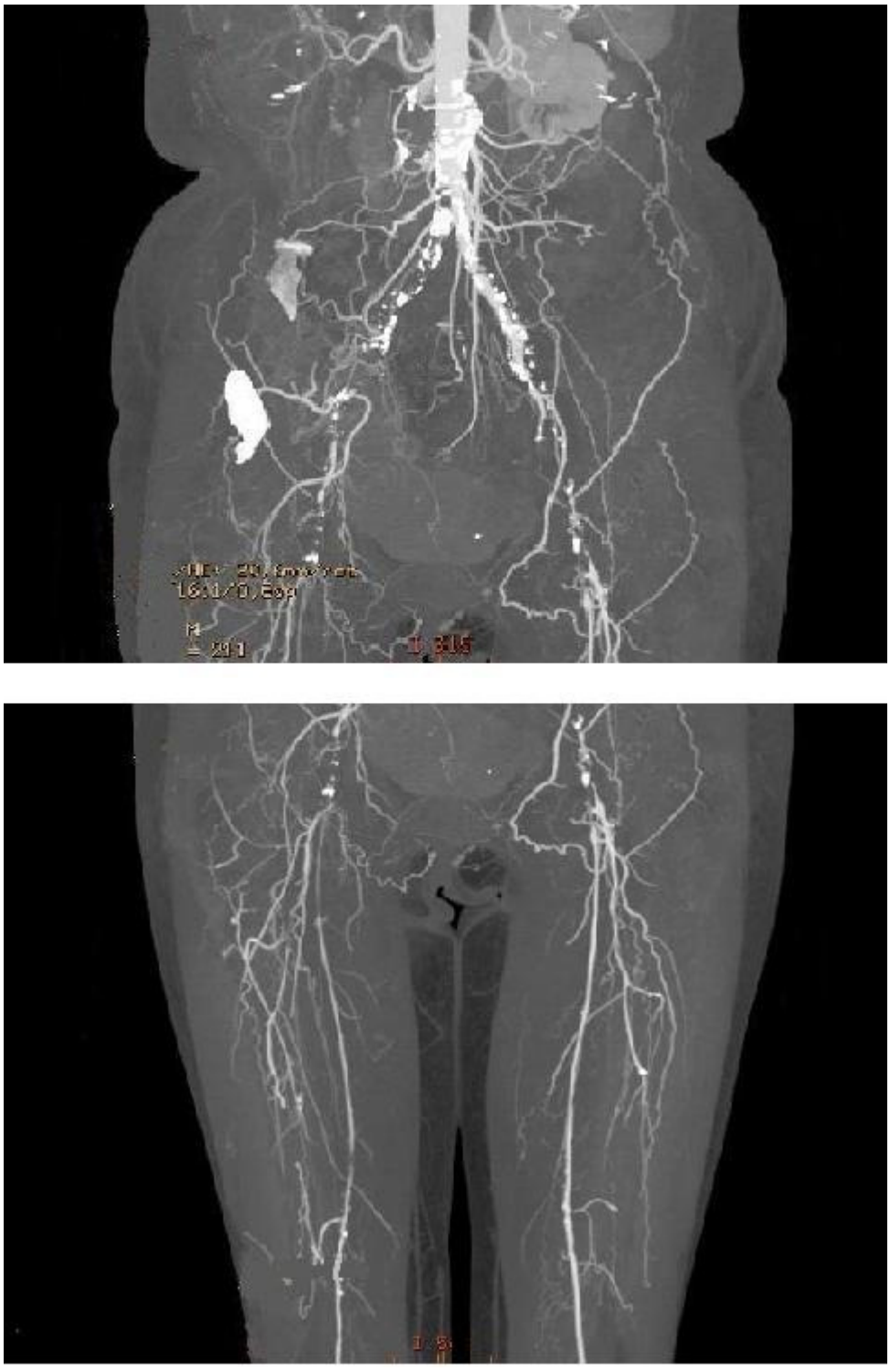
Unfortunately, the bypass graft thrombosed 3 months after the initial attempt at revascularisation. Although thrombectomy led to successful revascularisation, the patient developed necrotic right third and fourth toes that required amputation.

Because of the risk of infection and further necrosis, the amputation wound was not closed. TNP therapy was applied to the wound in an attempt to optimise healing. Postoperative improvement was steady and Mrs X was transferred for rehabilitation.

During rehabilitation, the patient had an unfortunate accident. The VAC device was next to her, with the tubing trailing on the ground from the device to her foot. She tripped over the tubing as she got up to meet a visitor and sustained a fall. There were no nursing staff present with her at the time.

The neck of her right femur was fractured and a total hip joint replacement was required. She suffered from acute confusion postoperatively but eventually recovered from her orthopaedic surgery and was discharged home nearly 5 months after her initial admission. Clinic follow-up 6 weeks post toe amputation revealed a fully healed wound and the patient was discharged from further routine follow-up with the vascular service.

Figure 1. CT angiogram—series of reconstructed images showing occlusion of the right common iliac, external iliac, common femoral, and proximal superficial femoral arteries



Discussion

VAC devices have become progressively more compact, light-weight, and easy to handle. They incorporate several safety features designed to minimise complications.[2] In spite of these obvious improvements, the devices are attached to the patient via tubes and therefore pose a risk of tripping and falls in the elderly patient.

In the elderly, hip fractures from a fall are common and well-documented. Morbidity and mortality data from the United States suggests an incidence of 800–900 hip fractures per 100,000 population per year and an incidence of 20–50 fatalities per 100,000 population per year resulting from falls.[3]

Elderly people who sustain a hip fracture suffer a very high morbidity and mortality. One in five people die in the first year after a hip fracture, and one in four elderly people require a higher level of long-term care.[4] People with three or more comorbidities preoperatively have a 2.5 times higher risk of death at 30 days and also a significantly higher risk for developing complications.[5]

The vascular surgical service at our hospital treats a high proportion of older patients with a heavy atherosclerotic burden and extensive comorbidities. The prevalence of smoking is high. Coexisting diabetes, cardiovascular disease, cerebrovascular disease, respiratory disease, and renal disease is not uncommon. Our patients therefore carry a very high risk of morbidity and mortality in the event of a hip fracture.

The nature of wounds treated by vascular surgeons means that VAC devices are frequently used. While the risk of tripping over the tubing might seem obvious and self evident, it is a risk that is easily overlooked. It is important to remember that falls in the elderly can lead to severe disability or even death and simple precautions to avoid falls would be worthwhile.

When applying VAC therapy, both the patient and their carers should be educated about hazard of falls and should be made conversant with techniques aimed at maximising safety. VAC therapy has revolutionised wound care in many branches of medicine and the authors do not feel it is contraindicated in elderly patients but would advise vigilance regarding patient and carer education.

Author information: Tejaswi Kandula, House Officer; Justin A Roake, Professor of Surgery; David R Lewis, Vascular Surgeon; Christchurch Vascular Group, Department of Vascular Surgery, Christchurch Hospital, Christchurch

Correspondence: Mr David R Lewis, Vascular Surgeon, Christchurch Hospital, Private Bag 4710, Christchurch. Fax: (03) 364 0352; email: david.lewis@cdhb.govt.nz

References:

1. Pham CT, Middleton PF, Maddern GJ. The safety and efficacy of topical negative pressure in non-healing wounds: a systematic review. J Wound Care. 2006;15:240–50.
2. Argenta LC, Morykwas MJ, Marks MW, et al. Vacuum assisted closure: state of clinic art. Plast Reconstr Surg. 2006;117 (Suppl 1):127–42.
3. Centers for Disease Control and Prevention. Fatalities and injuries from falls among older adults – United States, 1993-2003 and 2001-2005. Morb Mortal Wkly Rep. 2006;55:1221–4.
4. Gillespie WJ. Extracts from "Clinical Evidence"– hip fracture. BMJ. 2001;322:968–75.
5. Roche JJW, Wenn RT, Sahota O, Moran CG. Effect of comorbidities and postoperative complications on mortality after hip fracture in elderly people: prospective observational cohort study. BMJ. 2005;331:1374–6.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Did Janet Frame have high-functioning autism?

Sarah Abrahamson

Abstract

Janet Frame (1924–2004) was one of New Zealand's most well-known authors and unusual personalities. Her formal psychiatric diagnosis, however, has not been clear. Some have suggested that she was simply "different". This paper proposes that there is a name for her difference: high-functioning autism. The features of this condition apparent on analysis of her autobiographies are examined.

Janet Frame was an interesting example of what may be achieved by those with strong autistic features. It is to be hoped that current and future generations of New Zealanders with autism spectrum disorders are recognised early and given appropriate advice.

Janet Frame, the New Zealand author (1924–2004) was a complex figure. She spent many years in mental institutions, with a diagnosis of schizophrenia, which was later disproven. No alternate diagnosis has been widely canvassed. I propose that she had definite signs of another diagnosis: high-functioning autism, also known as Autistic Disorder.

The text regarded by experts in this field as the definitive guide to high-functioning autism spectrum disorders (ASDs)[1], and the DSM-IV TR criteria for Autistic Disorder[2] have been used as sources of comparison with Janet's symptoms and signs. Her combined autobiographical works (*To the Is-land, An Angel at My Table*, and *The Envoy from Mirror City*[3]) have been examined, and her autistic features discussed.

High-functioning autism and Asperger's disorder

There are a number of different terms for high-functioning autism spectrum disorders (ASDs), also known as Pervasive Developmental Disorders; the term "high-functioning" indicating an IQ of greater than 70.

Terms in common use include high-functioning autism, Autistic Disorder, Asperger's Disorder, and Asperger's Syndrome. High-functioning autism is partly differentiated from Asperger's Disorder or Syndrome by the possible presence of significantly delayed language development.

The key symptoms and signs of high-functioning autism are described in Text Box 1.

Table 1. High-functioning autism in adults

Key features

- Impairment in social and high-level communication skills
- Impairment in development of normal peer relationships
- A special interest which is abnormal in intensity and focus

Other common features

- A delay in language development
- Impairment in imagination
- A need for strict routine
- Impairment in recognition of faces
- Over- or under-sensitivity to sensory stimuli
- Difficulty using eye contact appropriately
- Motor clumsiness
- Impairment in perception of own and other's emotions
- Impairment in appropriate expression and control of emotions

The autobiographies

To the Is-land (1982)

In the first volume, Janet described having significant childhood language difficulties, and very likely a language delay. She mispronounced many words. She also could not remember when she learnt to read, having done so very early. This may indicate that she had hyperlexia, an advanced ability in word recognition with relatively poor comprehension, which occurs frequently in those with autism.

The title of her first autobiographical volume, *To the Is-land*, reflected her preference for the written form of words to the spoken; she insisted on mispronouncing "Island" as it was spelt, and took some persuading that the "s" was silent.

From an early age, Janet developed a strong interest in poetry, which was to become a lifelong interest. This appears to have been sufficiently intense to be considered an autistic "special interest". She did not feel that she had imagination, in common with many people with autism. To compensate for this deficiency she analysed poetry to determine how she could seem more imaginative and artistic. She was also talented in maths, common in autism.

Janet struggled socially at school, as do many with autism. She described being picked last for activities, and had only one close friend in her school years. She interacted with her brother and sisters, but describes her happiest times as those she spent alone, sitting on a hill, or with animals. The animals in Janet's life have a significant role in her books: in common with many people with autism she found it easier to be with animals than with people.[4]

Janet's social and communication difficulties do not appear to be have been shared by any of her siblings, who she described as being average children. Her father, however, seemed to have autistic tendencies. He had little understanding of the humour in everyday life. He also had great difficulty expressing appropriate emotions; for example, when trying to express fondness and concern for his wife, it came out as ridicule. Janet came to understand her father more than her siblings, and forgave him for what was perceived by others as a lack of caring.

An Angel at My Table (1984)

Janet's difficulties in interacting with people in adult life are a major theme in her autobiographies. She described being afraid of even going into her University Student Union in Dunedin. She made no friends at Teacher's Training College, and was unable to socialise with her fellow teachers on school placements.

Being unable to form a useful relationship with anyone in her student years, Janet drifted into counselling sessions with her psychology tutor. She was thankful that someone took an interest in her, especially a young man. She made every effort to continue this attention, even reading about schizophrenia and fabricating some of the symptoms to ensure she had his attention.

This situation unfortunately resulted in an admission to the psychiatric hospital in Dunedin. The doctors here concluded that she did not have a mental illness. When she was about to be discharged home, however, she became agitated with her mother and lost her temper, dreading going home to the stresses of her family: such outbursts are common in those with autism when under stress. This was seen as a sign of madness, and she was committed to Seacliff Hospital.

Janet's situation deteriorated from here. She had only a precarious grasp of normal social interactions, and these deteriorated in the company of those with mental illness and intellectual disabilities. She was taken away from her lifelong special interest of literature, thus adding to her loss of sense of self. She appears to have become depressed during this time, and taken on many of the dysfunctional behaviours of those around her.

During this time, her doctors had arranged a pre-frontal leucotomy, a form of psychiatric surgery in use at the time for certain conditions. This surgery was prevented only by her winning a literary award for one of her books.

Janet was eventually discharged from hospital with the stigma of having schizophrenia. Her family, who were her only supports, treated her differently from before, and she found it difficult to relate to them. She subsequently moved to Christchurch, where her only way to get help with the stresses of everyday life was to present to a psychiatric institution, where she was again admitted. By the end of her years in hospital, she has spent 9 years of her life as a psychiatric inpatient.

Eventually Janet moved to Auckland, where she was positively influenced by another author, Frank Sargeson. He introduced her to the lifestyle of a writer and to non-threatening social contact with colleagues, where she could stay in the background if she did not feel comfortable speaking. Frank encouraged Janet to apply for a scholarship to go to the UK.

The Envoy from Mirror City (1985)

Janet's experiences in the UK and Europe were largely positive. She was admitted electively to a prestigious psychiatric hospital, the Maudsley, where she was able to speak at length to sympathetic doctors. Her doctors helped Janet to understand that she did not have schizophrenia. They were not able to provide her with any other label, which left Janet feeling lost, as she was sure by this stage that she was different from other people. Her doctors were, however, able to help her determine her differences, and to accept them without feeling that she should be the same as others.

One doctor advised her that she should live alone, and not socialise if she didn't feel like it; a common way for autistic people to lead a happier lifestyle.

During her time in the UK and Europe, Janet formed several significant, but short-term, relationships. She moved to Spain, and formed a friendship with an American artist, based on their mutual artistic experience. She also developed a romantic relationship for the first time, at aged 33, with another young American, Bernard, who shared her interest in poetry. He was not as enthusiastic in the relationship as her, and he returned to the US.

Janet also formed friendships with the locals in Spain and Italy. She found that they were able to forgive her social inadequacies, partly because they appreciated her making the effort to speak their language, and because her social difficulties were hidden among the language and cultural differences.

Returning to London after her time in Spain and Italy, Janet continued her platonic relationship with an Irish man, Patrick, who also appeared to have autistic tendencies. He expressed his fondness for Janet by sending her food parcels, buying her useful objects and attempting to control her life rather than in words. He did not attempt to start a romantic relationship, but seems to have simply appreciated the company of a like-minded person. He reminded her of her father, who also had autistic tendencies. However, Janet did not especially like Patrick, and when he attempted to become more controlling she ended the friendship.

Eventually Janet settled into a more autistic, solitary life, spending most of her time alone writing. On her return to New Zealand after 7 years overseas she had become such a loner that she had to ask a neighbour she barely knew to see her onto the boat, having no close friends.

By this stage, Janet had reached a point of self-acceptance. She was able to face her family and home country with the confidence of knowing that she was different, not mad. She became one of New Zealand's most prominent writers, and was internationally acclaimed.

Differential diagnoses

Asperger's Disorder,[5] another category of ASD, should also be considered as a possible cause of Janet's symptoms. There is only one measurable difference between Asperger's Disorder and high-functioning autism: in Asperger's Disorder there must be normal or near-normal language development.

There is currently debate among clinicians and researchers in this field regarding whether autism and Asperger's Disorder are separate conditions or varieties of the same condition. In Janet's case, while her early language development was certainly

impaired, we cannot be certain which of these two categories she would have most clearly fit into if ASDs had been widely known during her early years.

The other possible diagnoses to be considered are personality disorders, particularly Schizotypal Personality Disorder.[6] This condition shares a number of features with ASDs, and it is certainly conceivable that someone could fit the criteria for Schizotypal Personality Disorder while still having an underlying ASD. The exact relationship between this and other personality disorders and ASDs is not yet known: further research may reveal a substantial overlap in these conditions.

Conclusion

Janet Frame fits within the diagnostic criteria for high-functioning autism, based on an analysis of her autobiographies. It is hoped that this paper will stimulate doctors and other health professionals to consider the diagnosis of autism, rather than suggest purely a mood, psychotic, or personality disorder, in patients presenting with signs similar to Janet Frame. It is also to be hoped that people with these symptoms are given appropriate advice, as those treating Janet were eventually able to provide, to help them to achieve their full potential.

Competing interests: None.

Author information: Sarah Abrahamson, Rehabilitation Physician, The Queen Elizabeth Centre, Ballarat, Victoria, Australia

Correspondence: Sarah Abrahamson, Rehabilitation Physician, The Queen Elizabeth Centre, 102 Ascot Street South, Ballarat, Victoria 3350, Australia. Fax. +61 3 53203661; email: sjabrahamson@yahoo.com

References:

1. Attwood T. The Compete Guide to Asperger's Syndrome. London: Jessica Kingsley Publishers; 2006.
2. American Psychiatric Association. 299.00 Autistic Disorder. In: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000:70–5.
3. Frame J. Janet Frame: the Complete Autobiography. London: The Women's Press; 1990.
4. Grandin T. Animals in Translation—Using the Mysteries of Autism to Decode Animal Behaviour. New York: Scribner; 2005.
5. American Psychiatric Association. 299.80 Asperger's Disorder. In: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000:80–4.
6. American Psychiatric Association. 301.22 Schizotypal Personality Disorder. In: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000:697–701.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Two cases of perforated gastric ulcer: operation—recovery (case 1)

Case report written by P Clennell Fenwick—M. B. London, F.R. C.S. Bd. (Late) Surgeon to Christchurch Hospital, N. Z—and published in N Z Med J 1908;6(25):9–13

CASE I.—R. W., male, aged 44 years, was admitted into Christchurch Hospital on June 7th.

He had had symptoms of gastric trouble for the last 4 years, accompanied by vomiting and diarrhoea. During the last few months he has been carefully dieted. He was seen in the Out-patient Department yesterday afternoon, and then complained of pain in the stomach and attacks of vomiting. The abdomen at this time was examined, and was noted as "very hard."

This afternoon he was suddenly attacked with intense pain, and vomited. He had had a light breakfast and very little lunch. There was no history of haematemesis or melaena.

On admission, the patient is very cold and pale, and collapsed.

The abdomen is rigid, and pain is felt on pressure over the gastric area. Temperature, 96°F; pulse, 102. The legs are drawn up.

A sixth grain of morphia was given, and I was called—to see the case. At 8 p.m. I incised in the right rectus line and immediately exposed a clear cut opening into the stomach the size of a threepenny piece. The opening was on the anterior surface of the stomach and close to the Pylorus.

The opening was quickly closed by Lemberts, sutures infolding the edges and reinforced by two more, superficial Lembert sutures.

An indiarubber drainage tube was inserted into the right flank and a second one into the supra-pubic region.

The temperature rose immediately and reached 101 by 2 a.m.

There was no pain, and patient looked well next day. He passed flatus 36 hours after operation, and 48 hours after operation the temperature was again normal, and never rose above this.

On June 11th. Peptonoids were given every hour, and in 10 days he was taking light diet without any trouble.

Immediately after operation he was given one pint of saline per rectum, and this was repeated every two hours for three days.

He left the hospital in July.

On September 27th. he was re-admitted on my advice to undergo Gastro-Jejunostomy. This was performed by my colleague Dr. Acland, owing to my disablement by a poisoned hand.

The incision was in the middle line, and very few adhesions were found round the first operation scar.

At present date, November 2nd., he is convalescent and looking very well.

In this case I was fortunate in finding the perforation immediately under my incision, and in getting the case so soon after the accident—about five hours only had elapsed between the perforation and the operation. The effect of the frequent rectal injections of saline appeared most beneficial.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

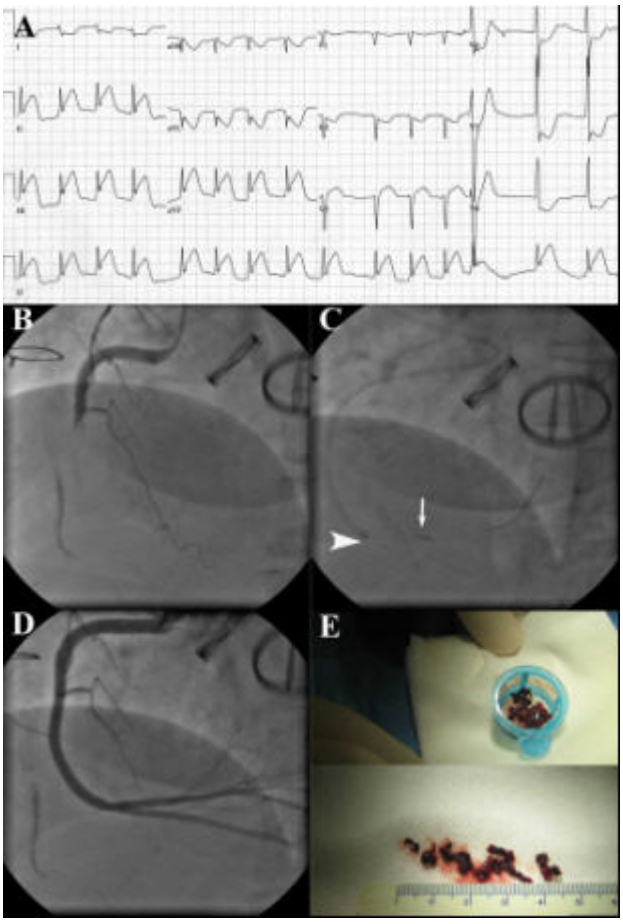
Transcatheter treatment of large coronary embolism

Suwatchai Pornratanarangsi, Peter N Ruygrok, Mark WI Webster

A 66-year-old woman with a history of rheumatic heart disease, chronic atrial fibrillation, and ATS (ATS Medical Inc., Minneapolis, MN, USA) mitral and aortic prosthetic valves presented with an acute onset of severe central chest pain.

She had been taking warfarin regularly with an INR of 2.5 at presentation. Her ECG showed atrial fibrillation with 3 mm ST segment elevation in leads II, III, and aVF plus ST segment depression in leads I, aVL, and V4-V6 (panel A).

Preliminary angiography revealed a thrombotic mid-vessel occlusion of the right coronary artery (panel B) and a normal left coronary artery. No improvement of coronary flow was achieved after performing multiple balloon dilatations. Therefore, a combination of deep engagement of the 6F right Judkins guiding catheter to distal vessel (arrow head), withdrawal of the expanded filter-based distal protection device (arrow) as the retrieval catheter (Fogarty manoeuvre), and aspiration into the guiding catheter lumen was performed (panel C).



After three passes, complete evacuation of thrombus from the vessel was achieved (panel D) with relief of chest pain. No atherosclerotic lesion was identified and coronary embolism was considered the most likely diagnosis. A large amount of thrombotic debris was aspirated and captured in the filter basket (panel E). The remainder of her hospital admission was uneventful.

Author information: Suwatchai Pornratanarangsi, Interventional Cardiology Fellow; Peter N Ruygrok, Cardiologist; Mark WI Webster, Cardiologist; Green Lane Cardiovascular Service, Auckland City Hospital, Auckland

Corresponding author: Mark Webster, Green Lane Cardiovascular Service, Auckland City Hospital, Private Bag 92 024, Auckland 1001. Fax (09) 307 4950; email: MWebster@adhb.govt.nz

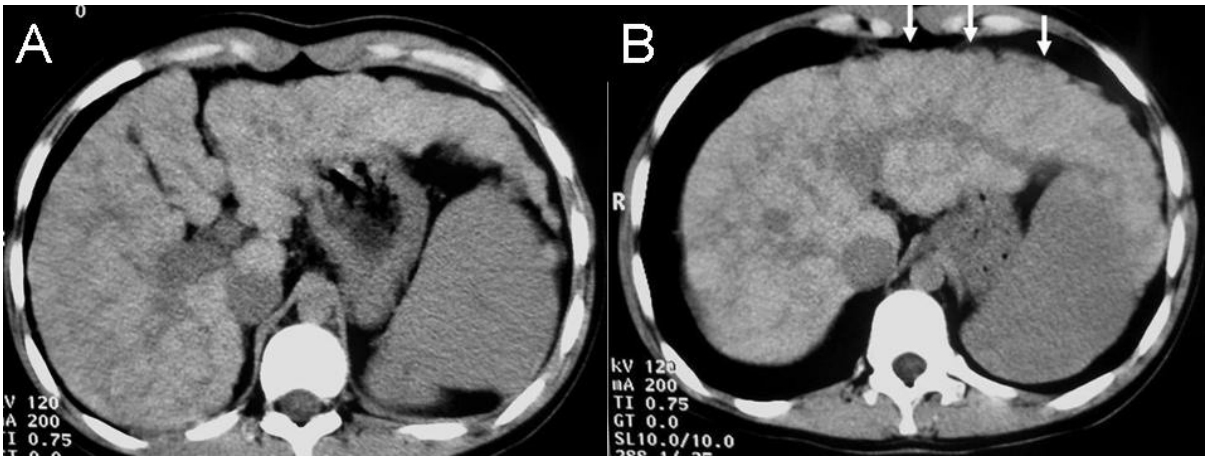
THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Hepatic Wilson's disease

Guner Sonmez, Ersin Ozturk

An 11-year-old boy was admitted to the hospital with jaundice, abdominal swelling, vomiting, and abdominal pain. A noncontrast computed tomography (CT) scan of the abdomen revealed hepatomegaly and increased hepatic attenuation (Figure 1A). The contours of the liver were irregular (Figure 1B). The diagnosis of Wilson's disease was established on the basis of blood, urine, and eye tests, and a liver biopsy.

Figure 1A **Figure 1B**



Wilson's disease (hepatolenticular degeneration) is a rare autosomal recessive inherited disease. The accumulation of copper in tissues, mainly in the liver, brain, kidney, and cornea is the primary cause of the disease. The hepatic manifestations usually begin before the age of 20 years.

CT findings of Wilson's disease include diffuse hepatomegali, contour irregularity, increased hepatic attenuation, and multiple small nodules within the liver. Once diagnosis is firmly confirmed, treatment should start as soon as possible.

Penicillamine is the drug of choice but oral zinc is being increasingly used as maintenance therapy. Prednisone is usually required to treat reactions associated with penicillamine discontinuation. As acute liver injury may not respond to medical therapy, liver transplantation may be required.

Author information: Guner Sonmez, Ersin Ozturk; Department of Radiology, GATA Haydarpasa Teaching Hospital, Istanbul, Turkey

Correspondence: Guner Sonmez, Assistant Professor, GATA Haydarpasa Egitim Hastanesi, Radyoloji Servisi, 34668, Uskudar, Istanbul, Turkey. Fax: +90 216 3304388; email: gunersonmez@hotmail.com

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

A case of ectasia

Onur Sildiroglu, Guner Sonmez, Cinar Basekim, Ersin Ozturk, Hakan Mutlu, Esref Kizilkaya

A 25-year-old man was admitted to hospital with right upper quadrant pain. His family history was unremarkable. Laboratory studies were within normal limits.

Ultrasound and computed tomography (CT) of the abdomen were performed (Figures 1 and 2). These showed communicating cystic ectasia of the biliary tree (Figures 1 and 2, white arrow heads) and polycystic kidney disease (PKD) (Figure 2, white arrows).

Figure 1. Abdominal Doppler ultrasonography

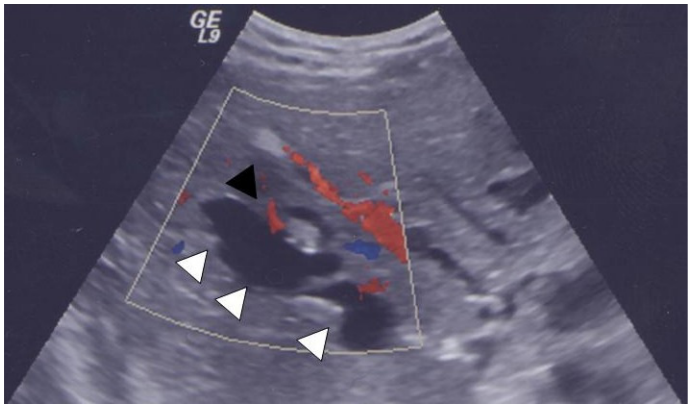
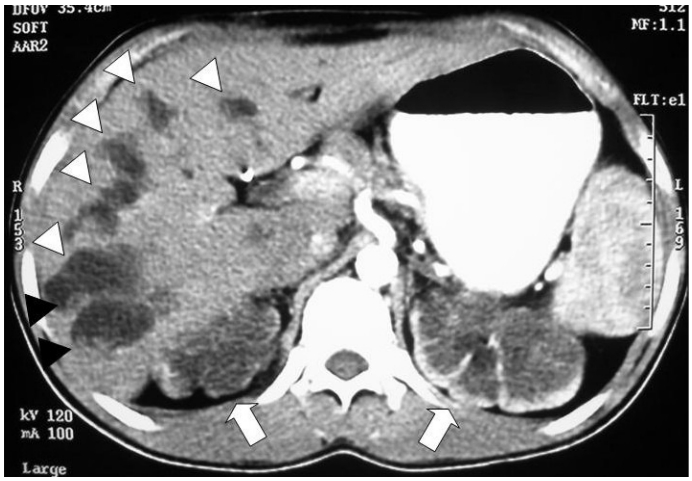


Figure 2. CT scan of the abdomen



What is the diagnosis?

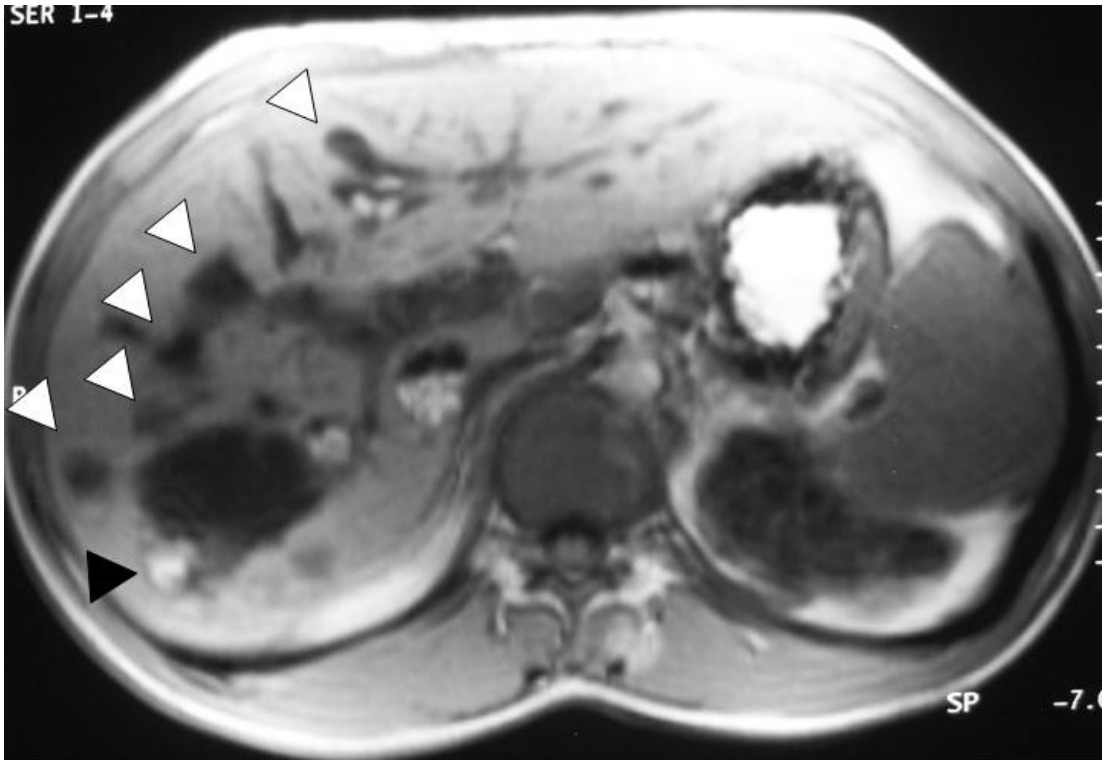
Diagnosis: *Caroli's disease.*

Discussion

Caroli's disease consists of saccular ectasia of the intrahepatic bile ducts. Right upper quadrant pain and fever are the major clinical symptoms. Jaundice can be seen in rare cases. Complications include pyogenic cholangitis, hepatic abscess, intrahepatic biliary stasis, and calculi. Malignant transformation such as cholangioma, which is generally cholangiocarcinoma, is a rarely seen complication.

Ultrasound, CT, and MRI (Figure 3 below) can be used for diagnosis of Caroli's disease. The MRI may show dilated intrahepatic biliary ducts and the 'intraluminal portal vein sign' or 'central dot sign' that represents a vascular focus within the dilated bile ducts (black arrow head on Figure 3).

Figure 3. Axial contrast-enhanced T1-weighted MR image shows cystic dilated intrahepatic bile ducts (white arrow head).and intraluminal portal vein (black arrow head)



Author information: Onur Sildiroglu, Guner Sonmez, Cinar Basekim, Ersin Ozturk, Hakan Mutlu, Esref Kizilkaya; GATA Haydarpasa Teaching Hospital, Department of Radiology, Istanbul, Turkey

Correspondence: Onur Sildiroglu MD, GATA Haydarpasa Teaching Hospital, Department of Radiology, Istanbul, Turkey. Fax: +90 216 5422808; email: sildiroglu@yahoo.com

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

A new twist for drug cost watchdogs

We know a fair amount about PHARMAC and some think it is unique. But it is not. Australia has its PBC (Pharmaceutical Benefits Advisory Committee), Germany has Iqwig (the Institute of Quality and Efficiency in Healthcare), and the UK has NICE (the National Institute for Health and Clinical Excellence). Each of these parlies with the drug companies for cheaper drug prices. NICE has come up with a new scheme with one of the pharmaceutical giants over a very expensive drug.

Janssen-Cilag will charge the NHS for bortezomib (Velcade), its new drug for multiple myeloma, only if the patients show a complete or partial response. And the drug company will rebate the full £25,000 (€37,000; US$50,000) cost of bortezomib for those who do not respond when treated in line with the drug's indication.

Nice one NICE, but read the small print carefully.

BMJ 2007;335:122–3

Any (good) new treatments for hot flushes?

Most women approaching menopause will have hot flushes. Whilst symptoms are transitory in many women, others have long-lasting flushes that interfere with their quality of life. They can be associated with other bothersome symptoms, such as depression, nervousness, agitation, insomnia, and inability to concentrate.

The cause may be fluctuating oestrogen levels, hence the usually useful results with oestrogen therapy. However, long-term oestrogen is associated with an increased risk of heart disease, thromboembolic disease and stroke, and breast cancer.

This review examines alternative treatments including transdermal 17-β-oestradiol, gabapentin, SSRI or SNRI (paroxetine, venalfaxine, citalopram), clonidine, red-clover isoflavones, and soy isoflavones.

The transdermal oestradiol was slightly better than oral oestrogen and progestagen tablets, but all of the rest were no better than placebo, which incidentally had a 50% success rate. So oestrogen remains the gold standard, but should be used sparingly, after discussion with the patient.

Lancet 2007;369:2062–4

Prevention of stroke and heart attacks

Atherosclerotic arterial disease is associated with an increased risk of myocardial infarction, stroke, and death from cardiovascular causes. Antiplatelet drugs reduce this risk. In those with atrial fibrillation, anticoagulation has been proven to reduce the risk of stroke. However in those with sinus rhythm, the role of oral anticoagulant agents in the prevention of cardiovascular complications in patients with arterial disease is unclear.

This multicentre international randomised trial should put the issue to rest. It demonstrates that the combination of an oral anticoagulant and antiplatelet therapy was not more effective than antiplatelet therapy alone in preventing major cardiovascular complications and was associated with an increase in life-threatening bleeding.

An editorial commends the conclusion.

N Engl J Med 2007;357:217–27 & 293–6

D-dimer concentration increases with age

Thromboembolic disease features an increase in D-dimer levels in the blood. Hence, the D-dimer assay is used as an exclusion test in the assessment of suspected venous thromboembolic disease; patients with a negative result have a low probability of thrombosis. Unfortunately levels are also elevated in other situations, including inflammation—so a raised D-dimer is not diagnostic of thromboembolism.

After reviewing the D-dimer results from 6631 unselected patients, the authors of this paper (from Auckland) conclude that age is also relevant. They conclude that the D-dimer test has little clinical value as an exclusion test for venous thromboembolism (VTE) when used in patients more than 80 years old as less than 5% of patients in this group have a negative D-dimer result.

It would also be helpful if the test were done only in appropriate circumstances—to try and rule out thromboembolism.

Internal Medicine Journal 2007;37:607–13

The demise of nursing (in the UK)—next and last instalment

Two previous abstracts (*NZMJ* 18/5/07 & 20/7/07) have appeared on this topic and I promise that this will be the last. A surgeon who has worked in UK hospitals for 25 years reminisces wistfully—"gone are the days when the sister on the ward was the conductor of the orchestra of patient care, who along with his/her staff knew everything about every patient."

He observes that because of poor nursing remuneration many senior nurses were attracted to better paid management positions—"relieved from front line duties, handed an office, a computer, and mountains of paperwork. It is high time for the nursing strategy to move back to basics, which is the care of the sick by the bedside."

Hear, hear!

Journal of the Royal Society of Medicine 2007;100:303

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Economics can be good for health: need for rational policy without the influence of vested interests

New Zealand Government's position on climate change—New Zealand has a proud history of leading the world in social changes, including universal suffrage, social welfare, and nuclear-free policies. Unfortunately, New Zealand has recently been lagging in addressing a key global issue: climate change.[1] A recent government proposal to control greenhouse gas emissions[2] is both overdue and inadequate. In comparison, Finland introduced a carbon tax in 1990, followed by similar moves in other OECD countries.[1,3]

The overall logic of the proposed greenhouse gas emissions trading system appears sound. However, the Government is exempting the agricultural sector (which accounts for around half of greenhouse emissions) until the year 2013.[2] Even then it plans to give this sector 90% of emissions credits for free,[2] rather than the fairer approach of selling or auctioning them. Is this a rational policy or the expression of vested interests?

The "agriculture exemption" disregards the critical "polluter pays" principle and transfers wealth to a sector riding high on record dairy prices. New Zealand's emissions trading policies should be efficient, fair, and enacted without further delay given the threat posed by climate change to the environment and global public health. Prompt action will also help to turn the rhetoric around New Zealand's "clean green" image into a reality that can be genuinely used for promoting in-bound tourism and agricultural exports.

The systematic failure of the New Zealand Government to develop rational policy on economic policy levers to address health issues, is also evident in other areas, including: tobacco, alcohol, and food. Is it a coincidence that there are powerful vested interests in these areas?

Price signals around tobacco—Overwhelming scientific evidence supports tobacco taxation as one of the most effective tobacco control interventions,[4,5] including in New Zealand.[6–8] The government's current national tobacco control plan,[9] identifies price as a key policy intervention, but there has been no above-inflation increase in tobacco tax since the year 2000. With wage growth above inflation, and smokers switching to cheaper roll-your-own cigarettes, the price signal is being eroded.

The government's refusal to dedicate tobacco tax revenue to control activities, including helping smokers to quit, is an additional concern. This is despite the international evidence that dedicated taxes are an effective approach to ensuring sustainable funding of tobacco control activities and that such taxes are far more popular with the public and hence more politically palatable.[10]

Price signals around alcohol—As for tobacco, there is overwhelming scientific evidence that alcohol excise tax policy is an effective intervention for reducing harm from alcohol.[11,12] But where is the alcohol pricing policy that rationally attempts to reduce the enormous harm from alcohol in this country (a net 26,000 disability-

adjusted life years lost per year[13]) while still allowing moderate patterns of social consumption?

There is also no published policy work for New Zealand to explore the impact of differential alcohol excise taxes, so that those forms of alcoholic beverage that are the most hazardous are taxed at higher rates (e.g. beer versus wine for binge drinking[14]).

Price signals for healthy food choices—Market forces have led to the cheapest foods being the most obesogenic,[15] undermining public health efforts to address obesity. The obvious strategy to address the obesity and diabetes epidemics in New Zealand is to tax high-energy nutrient-poor foods and reduce taxation and/or subsidise healthier foods.

It is not surprising that previous suggestions along these lines have raised vociferous objections by the food industry. Could this be the reason that a Select Committee Inquiry dismissed price signals in a brief paragraph of its 49-page report?[16] That paragraph reflects a gross failure of policy analysis from any rational perspective.

While there are undoubtedly some complexities with taxes on unhealthy foods, such as trade-offs between high saturated fat and high salt foods,[17] the potential value of removing GST on, or providing vouchers for, fruit and vegetables to low income consumers would be an obvious area to start exploring.[18]

What needs to be done?—We consider that the lack of the rational use of economic instruments by the Government to protect the environment and public health is at least partly explained by the intense resistance from powerful agribusiness, processed food, tobacco, and alcohol industries. On the other hand, ineffective measures such as education campaigns are easy to support, and create the appearance of action, but have had limited impacts compared to price signals.

To help the politicians and officials to use appropriate policies we need a "system upgrade" in the current economic system and political culture:[19]

- Firstly, we need to find ways to communicate successfully that economic instruments (i.e. emissions trading, targeted taxes, and incentives) are legitimate and highly effective ways of achieving desirable policy objectives such as better diet, reduced tobacco use, and reduced greenhouse emissions and consumption; and as such should be welcomed.
- Secondly, we need to constrain corporate influence over the policymaking process, including covert lobbying and donations to political parties.
- Thirdly, we need to create public institutions (such as independent Trusts[19] and Tobacco Control Authorities[20]) that are free from corporate influence and political meddling, and which have real power to introduce and implement effective policy decisions[19] in the interests of health, the environment, and social justice.

As with many other developed countries, New Zealand already has a Reserve Bank that is designed to be free from political meddling—and operates in this way most of the time. PHARMAC has also managed to successfully constrain the growth of the national pharmaceutical budget with minimal political interference and the capacity to fight off multiple legal attacks by industry.

Such organisational arrangements and the other changes we suggest above are needed to protect public health and the environment. These should still be accompanied by

efforts to maximise (for more routine goods and services) the successful entrepreneurial aspects of market-based economies that are of proven efficiency.[21]

Competing interests: The first two authors have previously worked for NGOs and the Ministry of Health on tobacco control issues. Similarly, for nutrition issues for the first author (NW).

Nick Wilson, Richard Edwards
Department of Public Health
University of Otago
Wellington
(nick.wilson@otago.ac.nz)

Osman Mansoor
Public Health Physician
Wellington

References:

1. Wilson N, Melhuish M. Addressing the threat of climate change: is New Zealand lagging behind? N Z Med J. 2007;120(1251). http://www.nzma.org.nz/journal/120-1251/2479/
2. Parker D. A New Zealand Emissions Trading Scheme [Speech notes, 20 September]. Wellington: New Zealand Government; 2007. http://www.scoop.co.nz/stories/PA0709/S00345.htm
3. US EPA. The United States experience with economic incentives for pollution control (11.1.5.2. Energy/carbon taxes, August 1997). Washington DC: US Environmental Protection Agency, 1997. http://yosemite.epa.gov/EE/Epalib/incent.nsf/c484aff385a753cd85256c2c0057ce35/0483a144da8fa434852564f7004f3e68!OpenDocument
4. Hopkins DP, Briss PA, Ricard CJ, et al. Reviews of evidence regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. Am J Prev Med. 2001;20:16–66.
5. Jha P, Chaloupka FJ. Curbing the Epidemic: Government and the economics of tobacco control. Washington DC: The World Bank; 1999.
6. Wilson N, Thomson G. Tobacco tax as a health protecting policy: a brief review of the New Zealand evidence. N Z Med J. 2005;118(1213). http://www.nzma.org.nz/journal/118-1213/1403
7. Wilson N, Thomson G, Tobias M, et al. How much downside? Quantifying the relative harm from tobacco taxation. J Epidemiol Community Health. 2004;58:451–4.
8. O'Dea D. Tobacco taxation in New Zealand. Presentation at the Seminar: "Death and Taxes, future directions for tobacco taxation". Aotea Centre, Auckland, 3 September. 2007.
9. Ministry of Health. Clearing the smoke. A five-year plan for tobacco control in New Zealand (2004-2009). Wellington: Ministry of Health; 2004. http://www.moh.govt.nz/moh.nsf/0/AAFC588B348744B9CC256F39006EB29E/$File/clearingthesmoke.pdf
10. Thomson G. Dedicated tobacco taxes – Experiences and arguments. Presentation at the Seminar: "Death and Taxes, future directions for tobacco taxation". Aotea Centre, Auckland, 3 September. 2007.
11. Chaloupka FJ, Grossman M, Saffer H. The effects of price on alcohol consumption and alcohol-related problems. Alcohol Res Health. 2002;26:22–34.
12. World Health Organization. Global Status Report: Alcohol Policy. Geneva: World Health Organization, 2004. http://www.who.int/substance_abuse/publications/alcohol/en/index.html
13. Connor J, Broad J, Rehm J, et al. The burden of death, disease, and disability due to alcohol in New Zealand. N Z Med J. 2005;118(1213). http://www.nzma.org.nz/journal/118-1213/1412

14. Naimi T, Brewer R, Miller J, et al. What Do Binge Drinkers Drink? Implications for Alcohol Control Policy. Am J Prev Med. 2007 33:188–93.
15. Drewnowski A. The real contribution of added sugars and fats to obesity. Epidemiol Rev. 2007;29:160–71.
16. Health Select Committee. Inquiry into Obesity and Type II Diabetes in New Zealand. Wellington: Forty-eighth Parliament, 2007. http://www.parliament.nz/NR/rdonlyres/47F52D0D-0132-42EF-A297-6AB08980C0EA/61821/DBSCH_SCR_3868_5335.pdf
17. Mytton O, Gray A, Rayner M, et al. Could targeted food taxes improve health? J Epidemiol Community Health. 2007;61:689–94. Abstract at http://jech.bmj.com/cgi/content/abstract/61/8/689
18. Wilson N, Mansoor O. The best recipe? Combining food taxes with subsidies on healthy foods [e-letter]. J Epidemiol Community Health. 2007;(28 August). http://jech.bmj.com/cgi/eletters/61/8/689#1348
19. Barnes P. Capitalism 3.0: A guide to reclaiming the commons. San Francisco: Berrett-Koehler, 2006.
20. Thomson G, Wilson N, Crane J. Rethinking the regulatory framework for tobacco control in New Zealand. N Z Med J. 2005;118(1213). http://www.nzma.org.nz/journal/118-1213/1405
21. Baumol W, Litan R, Schramm C. Good capitalism, bad capitalism, and the economics of growth and prosperity. New Haven: Yale University Press, 2007.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Tobacco smoking was dramatically reduced among New Zealand health care workers between 1963 and 1996, but what happened after that?

Health care workers (HCW) have an important and multifaceted role to play in tobacco control. At an individual level, they can help educate the population; at a community level they can initiate, motivate, and support government policy measures; and at a societal level they can influence larger and more permanent changes in national tobacco control strategies and help promote global tobacco control efforts.[1]

HCW also serve as role models for healthy behaviour, and the example they set in public is critical, because patients and the general community will undoubtedly be influenced by any health promoting activities they adopt. Despite this fact, tobacco use had become an ingrained habit in most Western countries by the early 20th century and many HCW smoked.

By the 1950s and 1960s, however, an increasing body of evidence linking tobacco use and disease began appearing in the literature, which in turn, led to a sustained decline in the smoking habits of most countries. From an international perspective, New Zealand has historically displayed some of the lowest smoking prevalence rates among both its doctors[2] and nurses.[3]

The first published study of tobacco use among New Zealand HCW was a postal survey undertaken by Cedric Gardiner and C N Derek Taylor on behalf of the Department of Health.[4]

In March 1963, all registered medical practitioners in the country were posted a simple, two-page smoking questionnaire. A total of 2623 surveys were returned from 3022 questionnaires posted, including 50 late returns, allowed a response rate of 88%. Among them, 76% reported that they had been smokers at some point in their lives, 37% had since quit, and 39% were current smokers. Of the smokers, 64% smoked cigarettes only, 26% smoked pipes or cigars and cigarettes, and 8% smoked pipes only.[4]

In October 1972, a smoking questionnaire was posted to 3776 doctors on the New Zealand medical register,[5] to which 3113 (83%) replied.[6] Among the respondents, 32% were current smokers (35% of males and 15% of females), 29% were ex-smokers, and 39% had never smoked. At this time, it was noted by the authors that cigarette smoking had clearly decreased between the 1963 and 1972 surveys.[5] No information about cigar or pipe smoking was sought during the survey. Interestingly however, 30% of the male doctors' wives smoked cigarettes, compared to only 21% of the male doctors, themselves.[6]

Table 1. Smoking surveys previously conducted among New Zealand health care workers (HCW)

Year of Study	HCW Profession	Current Smoker*			Previous Smoker*	Never Smoked*	Data Source	Ref. **
		All	Male	Female				
1996	Doctors	5%	5%	5%	19%	76%	1996 Census	10
	Nurses	18%	27%	18%	27%	55%		
1981	Doctors	15%	15%	13%	27%	58%	1981 Census	9
	Nurses	31%	39%	31%	19%	49%		
1976	Doctors	20%	20%	17%	33%	48%	1976 Census	7,8
	Nurses	37%	49%	36%	14%	49%		
1972	Doctors	32%	35%	15%	29%	39%	Survey	5,6
1963	Doctors	39%	41%	29%	37%	24%	Survey	4

* Figures rounded to the nearest whole number

** Reference number as listed in this manuscript

A unique opportunity arose in 1976, when New Zealand became one of the first countries in the World to introduce a smoking question on their national census form. As a result, considerable research on tobacco use among both doctors and nurses was subsequently undertaken by Sir David Hay and colleagues between 1976 and 1998,[7–10] while at the Princess Margaret Hospital and the National Heart Foundation.

In the first of these studies, 97% of the sample responded, of whom 4089 were doctors[7] and 27,323 were nurses.[8] Among them, 20% of doctors (males: 20%, females: 17%) reported themselves to be regular smokers, while the corresponding rate among nurses was 37% (males: 49%, females: 36%). Smoking information was restricted to cigarette consumption only.

In 1981, smoking data from the national census was again analysed for doctors and nurses.[9] Of the 4937 respondents working in the medical profession, 15% were current smokers, 27% ex-smokers, and 58% who had never smoked. Of the 30,720 nurses surveyed, 31% were current smokers, 19% ex-smokers, and 49% who had never smoked.[9]

By the 1996 New Zealand population census (in which 7335 doctors and 30,507 nurses participated), the rate of cigarette smokers in the medical profession had fallen to an all time low of 5% (males and females), while among nurses it was now 18% (27% for males and 18% for females).[10]

Following the retirement of Sir David Hay in the 1990s, detailed analysis of tobacco smoking rates among New Zealand HCW effectively ceased.

While the five original studies clearly reveal a sustained decline in smoking rates among HCW in this country, no contemporary data is currently available, despite the fact that a national census was conducted in 2001, and again 2006.

The authors of this letter, therefore, feel it is essential that HCW smoking data from these subsequent national surveys be analysed and publicly disseminated as soon as possible, so that the results may be used by tobacco control advocates in both New Zealand and beyond.

Derek R Smith
Associate Professor
International Centre for Research Promotion and Informatics
National Institute of Occupational Safety and Health
Kawasaki, Japan

Peter A Leggat
Professor
Anton Breinl Centre for Public Health and Tropical Medicine
James Cook University
Townsville, Australia

References:

1. World Health Organization (WHO). The role of health professionals in tobacco control. Geneva: WHO; 2005.
2. Smith DR, Leggat PA. An international review of tobacco smoking in the medical profession: 1974-2004. BMC Public Health. 2007;7:115.
3. Smith DR, Leggat PA. An international review of tobacco smoking research in the nursing profession, 1976-2006. J Res Nurs. 2007;12:165–81.
4. Gardiner CE, Taylor CND. Smoking habits of New Zealand doctors: A survey of the smoking habits of medical practitioners in New Zealand. N Z Department of Health, Wellington, 1964.
5. Christmas BW, Hay DR. The smoking habits of New Zealand doctors: a review after ten years. N Z Med J. 1976;83:391–4. http://www.nzma.org.nz/journal/120-1263/2758/1976.tiff
6. Hay DR, Christmas BW. The smoking habits of women doctors and doctors' wives in New Zealand. Prev Med. 1976;5:78–88.
7. Hay DR. Cigarette smoking by New Zealand doctors: results from the 1976 population census. N Z Med J. 1980;91:285–8.
8. Hay DR. The smoking habits of nurses in New Zealand: results from the 1976 population census. N Z Med J. 1980;92:391-3.
9. Hay DR. Intercensal trends in cigarette smoking by New Zealand doctors and nurses. N Z Med J. 1984;97:253–5.
10. Hay DR. Cigarette smoking by New Zealand doctors and nurses: results from the 1996 population census. N Z Med J 1998;111:102–4. http://www.nzma.org.nz/journal/120-1263/2758/1996.tiff

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Media frenzy

It used to be said that "the French have a word for it." They still do. The word I am thinking of is *médiatisation,* best translated as "intense media exposure." The adjective is *médiatique,* and describes what happens when the media get hold of a story they want you to hear.

I came across these words in a French monthly magazine, *Esprit.* The issue for January 2007 contained articles on the health services in France, some written by doctors. They confirm that the situation is the same everywhere, with the media watching for the bad news.

Briefly, the French have a cost explosion, serious levels of debt, and a maldistribution of doctors and specialists. They are confronting the elimination of small and under-performing hospitals, the geriatric bulge, end-of-life care issues, unnecessary resuscitation, doctor-patient relationships, the surge towards casualty departments, tensions in public-private relationships, the collapse of services in rural areas, medical discipline, feminisation of the workforce, a reduced number of working hours and working years, no house-calls in Paris, and so on.

In discussing contentious end-of-life issues, one author talked about *médiatisation réductrice.* This nicely describes what happens when the media, not content with hammering something, beat it out of shape. When almost unrecognisable, it does not support proper debate.

On 26 September 2007, Tony Ryall, the Opposition Spokesperson on Health, and one of a team of five that includes two MPs who are medically qualified, released a discussion paper on health entitled *Better, sooner, more convenient.* It runs to 45 pages (http://www.national.org.nz/files/__0_0_HEALTH_lowres.pdf). Simultaneously, Mr Ryall announced that a National government would get rid of the present system of capping the fees charged by general practitioners on top of the subsidies they receive from the government.

The media fell on the doctors' fees like wolves, and they were not very nice to Tony Ryall. A telecaster warned (twice) of "pain" under a National government if GP fees were deregulated. That was the *médiatisation* bit. By contrast, the contents of the report on health aroused little interest.

To give you the flavour of Mr Ryall's report, here are some quotations from it.

> Walk-in access at general practice should be more widely available to provide choice and faster care of patients.
>
> Health workforce numbers can be boosted by…reducing personal taxes to make it more attractive for health professionals to stay in New Zealand.
>
> Concerns are often raised that specialists working in both the public and private sector have an incentive to under-perform in the public hospitals in order to increase more lucrative private work.
>
> A tax rebate for private health insurance could cost an estimated $200 to $250 million.

Current spending of around 13 billion dollars per annum will infallibly lead to tax increases as demands rise. 24,000 actual and potential taxpayers left the country in a

12-month period, and most wage demands in the health sector are met with adjustments for inflation that merely drive inflation faster. Over 90% of the population want all hospital services to remain free of charge, and 100% of politicians know that.

The National Party is in a hopeless bind over health costs. There is nothing they can do to bring them down. In the meantime, the authors of the report say that they would like to hear from you. There are some boxes for you to tick.

Roger M Ridley-Smith
Retired GP
Wellington

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Defining vitamin D deficiency

In the 21 September 2007 issue of the *New Zealand Medical Journal*, Livesey et al conclude that most Christchurch people are vitamin D deficient most of the time based upon a criteria for vitamin D deficiency as a serum 25-hydroxyvitamin D (25OHD) <75 nmol/L.[1]

In an accompanying editorial, Scragg and Bartley state that "defining vitamin D deficiency as a 25OHD level below 50 nmol/L is clearly not supported by the current evidence, optimum health occurs at much higher levels than this;" and conclude that optimum vitamin D status occurs with 25OHD levels >80 nmol/L.[2]

We have previously argued that there is no strong evidence to support the use of such high thresholds for vitamin D deficiency[3] and that the widely used definitions of vitamin D deficiency (25OHD <25 nmol/L) and vitamin D insufficiency (25OHD < 50 nmol/L) remain appropriate.[4]

A recent panel of experts was not able to achieve a consensus definition of vitamin D sufficiency with recommendations ranging from 50–80 nmol/L,[5] while other authors have suggested an even broader range from 25 to >100 nmol/L.[5,6] The recommendations at the upper end of this range are based upon the measurement of surrogate endpoints such bone density or muscle strength in observational and cross-sectional studies. Such studies are potentially subject to confounding by frailty because people with poorer health are likely to spend less time outdoors, have less sun exposure, and have lower 25OHD levels than their healthy peers (rather than low vitamin D levels *causing* ill health).

In addition, people leading sedentary lives are at increased risk of obesity, and increased fat mass is inversely associated with 25OHD levels.[7,8] This association may confound the reported relationships between low vitamin D status and conditions such as diabetes, ischaemic heart disease, hypertension, and cancer that occur more commonly in obesity.[9] Confounding by health status can be powerful, as evidenced by the disparate results of randomised controlled trials and observational studies of postmenopausal hormone replacement therapy.

In contrast to the cross-sectional studies, intervention studies with clinically relevant endpoints such as fractures tend to give lower estimates of optimal 25OHD levels. For example, in a meta-regression analysis, the achieved 25OHD level associated with a reduction in all non-vertebral fractures was <50 nmol/L and for hip fractures was about 65 nmol/L,[10] in agreement with four interventional studies suggesting that serum PTH is not further suppressed by increasing 25OHD levels above 40–60 nmol/L.[6, 11-13]

In order to achieve year-round serum 25OHD levels >75–80 nmol/L, vitamin D supplementation of most, if not all, of the population would be required. We suggest that implementation of population-based strategies to achieve such vitamin D levels are premature in the absence of clear evidence of benefit (and safety). Indeed, currently there is no clinical trial evidence that increasing vitamin D levels impacts favourably on non-skeletal outcomes. At present, the only convincing clinical trial

evidence for beneficial skeletal effects of vitamin D supplementation is in institutionalised elderly women co-treated with calcium supplements.[14]

Mark J Bolland
Research Fellow

Andrew Grey
Associate Professor of Medicine

Tim Cundy
Professor of Medicine

Ian R Reid
Professor of Medicine

Department of Medicine
University of Auckland
Auckland

References:

1. Livesey J, Elder P, Ellis MJ, et al. Seasonal variation in vitamin D levels in the Canterbury, New Zealand population in relation to available UV radiation. N Z Med J. 2007;120(1262). http://www.nzma.org.nz/journal/120-1262/2733
2. Scragg R, Bartley J. Vitamin D—how do we define deficiency and what can we do about it in New Zealand? N Z Med J. 2007;120(1262). http://www.nzma.org.nz/journal/120-1262/2735
3. Bolland MJ, Grey AB, Reid IR. Vitamin D sufficiency. Osteoporos Int. 2007;18:835–6.
4. Lips P. Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocr Rev. 2001;22:477–501.
5. Dawson-Hughes B, Heaney RP, Holick MF, et al. Estimates of optimal vitamin D status. Osteoporos Int. 2005;16:713–6.
6. Aloia JF, Talwar SA, Pollack S, et al. Optimal vitamin D status and serum parathyroid hormone concentrations in African American women. Am J Clin Nutr. 2006;84:602–9.
7. Lucas JA, Bolland MJ, Grey AB, et al. Determinants of vitamin D status in older women living in a subtropical climate. Osteoporos Int. 2005;16:1641–8.
8. Bolland MJ, Grey AB, Ames RW, et al. Determinants of vitamin D status in older men living in a subtropical climate. Osteoporos Int. 2006;17:1742–8.
9. Field AE, Coakley EH, Must A, et al. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. Arch Int Med. 2001;161:1581–6.
10. Bischoff-Ferrari HA, Willett WC, et al. Fracture prevention with vitamin D supplementation: a meta-analysis of randomized controlled trials. JAMA. 2005;293:2257–64.
11. Malabanan A, Veronikis IE, Holick MF. Redefining vitamin D insufficiency. Lancet. 1998;351:805–6.
12. Lips P, Duong T, Oleksik A, et al. A global study of vitamin D status and parathyroid function in postmenopausal women with osteoporosis: baseline data from the multiple outcomes of raloxifene evaluation clinical trial. J Clin Endocrinol Metab. 2001;86:1212–21.
13. Patel R, Collins D, Bullock S, et al. The effect of season and vitamin D supplementation on bone mineral density in healthy women: a double-masked crossover study. Osteoporos Int. 2001;12:319–25.
14. Chapuy MC, Arlot ME, Duboeuf F, et al. Vitamin D3 and calcium to prevent hip fractures in the elderly women. N Engl J Med. 1992;327:1637–42.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Alan Caselberg

Born Dannevirke 4 June 1926; died Wellington 29 August 2007; aged 81.
M.B.Ch.B., F.R.N.Z.C.G.P., Dip. Obst.

Alan was the second son of Herbert and Jenny Caselberg, of Wellington. Herbert received the CBE in 1963.

Alan had his early schooling in Hawera before moving to Wellington. He attended Wellington College, Wellesley College, and Otago Boys' High School during the war. After leaving school he initially attended Otago University to begin his medical studies but transferred to Queens University Belfast, Northern Ireland. While there he flew Tiger Moth aeroplanes as a member of the Queens University Air Squadron. He returned to New Zealand in 1950, completing his degree in 1953. He spent his first professional years as a House Surgeon at Wellington and Hutt Hospitals.

In 1955 Alan met Pauline Rothwell and they were married later that year. 1956 was a professionally rewarding year spent as Medical Registrar at Palmerston North Hospital, followed by 6 months doing obstetrics at St Helens Hospital in Christchurch.

In 1957 Alan went to Levin as a locum. Three doctors practicing independently in Levin at that time—Ed Petersen, Hamish Neale, and Peter McKinlay—decided to band together and they invited Alan to join them as a partner.

Theirs was the first group medical practice in a country town in New Zealand. There was no hospital in Levin, so they had to deal with all sorts of emergencies, including accidents on the roads. They set up their own little casualty department, and, after a couple of years, one of the radiologists from Palmerston North put in an X-ray machine. It was this general medicine that Alan found most enjoyable. Tararua Medical Centre was unique in its day, stimulating much thought and providing the template for many others to use.

These years were very happy ones for the partners and their families. Alan said, "Country general practice was very busy, rewarding, and emotional, but tremendously satisfying." However, in 1978, his wife Pauline died suddenly at the age of 46. Alan had to cope with this sad loss, and the demands of general practice, whilst his children were still young.

In 1984, Alan spent 3 months as a Medical Officer at the Turoa skifield. He left Levin in 1987 after 30 years, and in 1988 began work as Senior Medical Officer at the Casualty Department at Wellington Hospital. Over the next 20 years, he kept up active medical practice in a variety of locums.

A colleague, Prof Graham Harley CBE, recently said of him, "Alan had a wonderful personality; it was such that whoever he met never forgot him."

In recent years he travelled widely. He went to Belfast (Northern Ireland), the United States, Guyana, Canada, and China, while renewing friendships with colleagues, friends, and relatives.

Alan was diagnosed with prostate cancer 7 years ago, but remained active until February 2007—always planning some new adventure. He underwent radiotherapy, and other oncology treatment, and, latterly, supportive palliative care from his family and the Mary Potter Hospice in Wellington.

He is survived by his children Andrew Caselberg, Jane Ball, and Belinda Henshaw (all of Wellington) plus three grandchildren.

Alan's family supplied this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Geoffrey James Taine

Orthopaedic Surgeon (1918–2007)

Geoff Taine died recently in Napier, his home for 53 years. He arrived in 1954 as the first and sole orthopaedic surgeon in Hawke's Bay, and proceeded to establish the specialty in this region, one of the few outside a metropolitan centre at that time.

The primary influences in his life were outlined in a memoir he wrote for his family: education at King's College in Auckland, where a strong work ethic was established; and the importance of family given the support required for education during the Depression. Otago Medical School and Knox College were followed by early exposure to orthopaedics, but a career was put on hold by army service in Italy. He relished the friendships made and the contact with people from a wide range of backgrounds.

Post-war specialisation involved a self-motivated series of jobs in the UK, in the absence of a formal training programme as currently exists. A dedication to succeed led to a Fellowship after several attempts, and hence to his consultant appointment, no doubt a daunting prospect in an era where the specialty was in its infancy in New Zealand.

Thirty years of public service followed, initially permanently on call, managing the effects of a range of conditions not so commonly seen today: polio, TB, and chronic osteomyelitis. All were dealt with on the principle of "to do ones best for the patient with what was available." His personal return was "to make a positive difference in the lives of the patients."

Other surgeons followed, and the region did not miss out on the introduction of new developments such as joint replacements and arthroscopy. Dedicated children's clinics, a longtime CCS involvement, and working trips to the Cook Islands, all show a dedication to the less fortunate.

Other interests were pursued with typical enthusiasm; family history research produced several articles and a monograph. Sailing provided a welcome escape from the phone (pre-cellphones!) and gardening was also a longtime interest (in several properties). A gradual retirement was accompanied by a series of canine companions.

Geoff bore his final years of declining physical health with characteristic stoicism and humour, and died at home aged 89. He leaves to Hawke's Bay a legacy of an orthopaedic service built on service and dedication, and to his family, memories of a full and active life.

Geoff's son, Bill Taine, wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

George Richard (Dick) Laurenson

15 August 1929 – 28 August 2007

"A life of achievement, duty, and service. A man of humanity and vision." These words penned by his wife Bronwen admirably describe Dick who passed away after a long illness.

Dick was born on the West Coast, the son of an engineer. The family moved to Wellington when he was a baby, as his father had been appointed Commissioner of Transport. He attended Kelburn School, and then Wellington College. After leaving school he joined the Public Works as a cadet draughtsman. Dick planned a career as an engineer, however possibly inspired by his medical uncle he changed to medicine. After an intermediate year at Victoria he went to Dunedin and graduated M.B.,Ch.B., in 1954. In the meantime he had met Suzanne (Sue) and had his first two children.

He returned to Wellington Hospital for his resident training. Three years later he started a general practice in Epuni to raise funds to train overseas. Over the next 5 years, this practice, including obstetrics and anaesthetics, became very busy.

In 1963 he sailed to Britain with Sue and five children. Registrar posts at The West Middlesex and The Royal National Orthopaedic Hospitals led to him achieving FRCS (London) in 1964.

Returning to New Zealand, now with six children, he worked at Wellington Hospital before being asked to establish the Hutt Orthopaedic Unit, with Graeme Smaill in 1967. He sat and passed the FRACS in 1970.

I met him in 1972 when we returned from Britain, and joined him at Hutt Hospital, and also in private practice. He could not have been a better friend, colleague, and mentor. He was a people person, very compassionate, and always ready to listen to a patient. His explanations of prognosis and treatments were clear and concise. He was a staunch ally, but if he disagreed he was a formidable foe.

He was passionate in his beliefs. Patients were often surprised when they opened their back door, to see their surgeon Dick Laurenson campaigning for the Labour Party.

Other interests included tramping, and Rotary.

Dick supported and helped institute podiatry training in New Zealand. He was instrumental in gaining acceptance for podiatrists to use local anaesthetic for minor procedures.

He supported the Wellington Physically Disabled Association, was on the committee from 1970 to 1978, and created a life member in 1980. Dick was the team doctor during a sporting trip to Japan in 1975.

He served terms on various New Zealand Orthopaedic Association committees including the executive, the Accident Compensation Subcommittee, and was also a delegate to the Chiropodists Board.

In 1980 he returned to Wellington Hospital, accepting the post of Director of Medical Services. After several administration restructurings he took early retirement in 1992, to become involved and work on the family orchard and farm in Otaki.

In retirement he enjoyed travelling (safaris in Africa, a tour of China, and regular trips to Europe and the United Kingdom). He also indulged his love of music, particularly opera.

Dick was farewelled by his friends at Old St Paul's on Saturday 1 September. He is survived by his second wife Bronwen and his six children and their families.

Chris Bossley (Orthopaedic Surgeon) wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Toxicology handbook

Lindsay Murray, Frank Daly, Mark Little, Mike Cadogan (editors). Published by Churchill Livingstone (Elsevier), December 2006. ISBN: 9780729537896. Contains 468 pages. Price AUD$69.95

This 443-page, pocket-sized, handbook is a useful addition to the genre of clinical toxicology reference sources. Compiled by four Australian consultant emergency physicians and/or specialist clinical toxicologists, its succinct style and firmly practical focus should be an asset in the emergency care environment. The authors draw on their experience as clinicians, consultants, and teachers to identify the key matters for emphasis in the diagnosis and management of acute poisonings.

The first chapter discusses approaches to emergency resuscitation and investigations, decontamination, supportive care, ongoing monitoring, enhanced elimination, and antidotes. The importance of risk assessment to guide management is stressed, but so too is the need for continual review, given potential "data" uncertainties (including ingestion time and dose), and the dynamic nature of acute poisoning.

The second chapter includes outlines on various poisoning syndromes and their distinguishing features; withdrawal syndromes; assessment and interpretation of acid-base and osmolar gap disorders; as well as brief sections on special considerations with poisonings in the elderly, young, and in pregnancy. The utility of the (12-lead) ECG is also discussed, in evaluation of the considerable number of pharmacological agents with significant effects on cardiac ion channels in overdose.

There are two chapters on specific toxins (~200 pages) and antidotes. They feature brief sections on handy tips, pitfalls, and controversies. These include warnings of rapid or delayed timeframes of symptom development, the limitations of specific investigations (e.g. pulse oximetry) in certain poisonings, cautions against under-dosing of some antidotes, tips to minimise acetylcysteine dosing errors, specific roles of controversial antidotes, and many more. The Appendices include useful nomograms and illustrative ECGs.

The final two chapters, on envenomings and antivenoms, discuss some spiders and venomous marine creatures, and Australian snakes.

This handbook is highly recommended.

Michael Beasley
Medical Toxicologist
National Poisons Centre
University of Otago, Dunedin

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Joint and soft tissue injection (4th edition): injecting with confidence

Trevor Silver. Published by Radcliffe Publishing (Oxford, UK), 2007. ISBN-10: 1846191904. Contains 128 pages. Price £27.95

The book is a comprehensive guide to intra-articular and soft tissue injection. While it is primarily aimed at general practitioners it would also be extremely useful to hospital-based doctors, in-particular junior staff. Appropriate knowledge of functional anatomy and accurate diagnosis are highlighted as prerequisites to joint injection. The layout is clear, with large simple photographs and diagrams.

The opening Chapter gives a brief review of the frequency of musculoskeletal problems and the relative lack of evidence base for joint injections. The following section provides general guidelines addressing frequently asked questions such as frequency of joint injection, contraindications, and post-injection advice.

Interestingly the author comments that Medical Defence organisations advise members to wear sterile gloves when undertaking joint injections. Despite this, all the photographs in the book reveal ungloved hands. The subsequent chapters deal with specific regions and outline common conditions for which steroid injections are used as well as injection approaches. The final Chapter is an excellent overview of musculoskeletal imaging techniques and how they may best be utilised.

While detailed instructions are given about needle size, it is unfortunate that doses of steroid to be injected are quoted in ml rather than mg. In some cases the type of steroid used is stated and therefore the dose can be calculated based on the standardised vials as quoted in the opening chapter. However, in many instances the type of steroid is not stated at all, leaving uncertainty about the exact dose required.

Readers should be aware of the different preparations available here in New Zealand, namely methylprednisolone acetate (Depo-medrol) 40 mg/ml and 10 mg/ml, and triamcinolone acetonide (Kenacort) 40 mg/ml.

In summary, this book is an extremely useful guide to joint and soft tissue injection techniques. It is well worth having as a reference book at general practices and departments where joint injection is undertaken.

Lisa Stamp
Senior Lecturer and Rheumatologist
University of Otago, Christchurch

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1263 ISSN 1175 8716

Oxford handbook of emergency medicine (3rd edition)

Jonathan Wyatt, Robin Illingworth, Michael Clancy, Colin Robertson, Colin Graham. Published by Oxford University Press, December 2006. ISBN 9780199206070. Contains 768 pages. Price £24.95

The primary target audience of this handbook is the population of junior doctors who staff British Accident and Emergency Departments. For this audience it is an excellent book. It is pocket-sized and with a plastic cover (like all the Oxford Handbooks) making it a useful 'on the job' reference. Although pocket-sized, it is comprehensive, with over 700 pages of guidance about most problems a junior doctor in an Accident and Emergency Department might face.

Australasian Emergency Departments tend to have a greater degree of senior supervision of junior medical staff (with some exceptions) and many have registrars training for the fellowship of the Australasian College for Emergency Medicine. There are other differences in practice between Australasian 'EDs' and British 'A and Es'. There are some differences in drugs used (for example, intranasal diamorphine for paediatric analgesia is commonly used in British A and Es) and in units of laboratory results (for example, Kpa rather than mmHg for arterial blood gas results). These, and a few other minor cultural differences in Emergency Medicine are the only opportunity to criticise this otherwise excellent book as a resource for New Zealand doctors.

The Handbook begins with a page of Golden Rules of Emergency Medicine representing a distillation of considerable wisdom. Then follow about 40 pages of 'General Approach' including advice ranging from 'how to cope as a junior doctor' to responding at a road traffic crash.

The next section is titled 'Life Threatening Emergencies' and includes the 2006 revised resuscitation guidelines from the British Resuscitation Council. There are some minor differences from the New Zealand Resuscitation Guidelines, although of little significance.

The remainder of the handbook has sections about clinical conditions. This edition is appropriately current with inclusion of a number of clinical decision tools gaining popularity in emergency departments, (such as the 'ABCD' score for transient ischaemic attack, the CURB 65 score for community acquired pneumonia and the Modified Wells Score for deep venous thrombosis), and discussion of SARS and Avian Influenza.

The clinical advice is good, generally with good basic pre-clinical and clinical content but stopping short of 'intensive care' or 'specialist' interventions. The emphasis is to seek early advice or referral. As such, it is pitched at a good level for medical students, house officers, senior house officers, and junior registrars. More would be expected above this level of training.

Michael W Ardagh
Professor of Emergency Medicine Sciences
University of Otago, Christchurch