THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

CONTENTS

This Issue in the Journal

4 A summary of the original articles featured in this issue

Editorials

6 New Zealand is not an island when it comes to global health policy engagement
Don Matheson, Belinda Loring

9 Sudden unexpected infant death—no more "stunned amazement"!
Nick Baker

13 Revised status of PSA testing in the early detection and treatment of prostate cancer
David S Lamb, Brett Delahunt, John N Nacey

Original Articles

16 Temporal trends and clinical characteristics of spontaneous intracerebral haemorrhage in the Waikato region of New Zealand: a hospital-based analysis
James Irwin, Peter Wright, Paul Reeve

26 Secondary prevention of vertebral fractures in a large New Zealand District Health Board
Katherine Bloomfield, Joe Singh

34 Emergency peripartum hysterectomy: a 10-year review in a tertiary obstetric hospital
Tze Yoong Wong

40 Outcomes of patients with untreated severe aortic stenosis in real-world practice
Suresh Perera, Namal Wijesinghe, Elene Ly, Gerard Devlin, Sanjeevan Pasupati

49 Review of 100 consecutive microvascular free flaps
Ryan Gao, Stanley Loo

57 Colonic self-expanding metal stents (SEMS) in acute large bowel obstruction
Mohammad I Khan, Adrian Claydon

Viewpoints

64 "Punching above its weight": why New Zealand must maintain leadership in global health
Judith McCool, Chris Bullen

69 Hypnosedative access and risk of harm
David B Menkes, Lucy M Shieffelbien, Mark Huthwaite

Clinical Correspondence

74 Colonoscopy—a rare cause of pancreatitis
Manar Khashram, Frank A Frizelle

77 Incarceration of an inguinal hernia post urinary catheterisation
Phil T Davey, Eunice J Minford

81 Medical image. An unusual cause of nonresponsive chronic dyspnoea
Prem P Gupta, Dipti Agarwal

84 Medical image. Xanthogranulomatous pyelonephritis
Habib U Rehman

Letters

86 Potential social and psychological consequences of the *Rena* incident: lessons from an international perspective
Sarb Johal, Ron Chambers, Susan Collins, Ian de Terte, Dianne Gardner, Bruce Glavovic, Lucy Johnston, A Nuray Karanci, Maureen F Mooney, Douglas Paton, David Johnston

90 End-of-term review of the New Zealand Government's response to climate change: a public health perspective
Nick Wilson, Ralph Chapman, Philippa Howden-Chapman

96 Banning pharmaceutical sponsorship: is ethical apartheid the right road ahead?
Lance Gravatt

98 Not for Resuscitation Orders—clarification is needed
John Tolliday, Hermione Denniss

99 Thames Hospital
Roger M Ridley-Smith

101 Playing with 'The Public Health'
Peter W Moller

104 Improving Emergency Department performance at Christchurch Hospital: an update
Michael W Ardagh

105 Eliminating tobacco point of sale displays: removing the retail detail from the devil
Janet Hoek, Anne Jones, Richard Edwards, Ninya Maubach, Julian Crane, Ben Youdan; for the ASPIRE2025 collaboration

110 Binge drinking is patterned by demographic and socioeconomic position in New Zealand: largest national survey to date
Santosh Jatrana, Kristie Carter, Sarah McKenzie, Nick Wilson

100 Years Ago in the NZMJ

116 Dominion Notes: Otaki Sanatorium

Consensus Statements

117 Consensus Statement on the Role of the Doctor in New Zealand
New Zealand Medical Association

Methuselah

121 Selected excerpts from Methuselah

Obituary

123 Ralph Marcus Lawson (Toby) Whitlock

Notice

125 NZMJ publication dates for 2012

Book review

126 Cleveland Clinic's Current Clinical Medicine (2nd edition; William D Carey)
Lutz Beckert

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

This Issue in the Journal

Temporal trends and clinical characteristics of spontaneous intracerebral haemorrhage in the Waikato region of New Zealand: a hospital-based analysis
James Irwin, Peter Wright, Paul Reeve

This study investigated intracerebral haemorrhage (ICH) or sudden bleeding within the brain detected in the hospitals of the Waikato region of New Zealand between 1999 and 2008. 653 episodes of ICH were identified. Observed ICH has increased in incidence in our hospitals over the past 10 years. Increasing availability of neuroimaging, increasing numbers of elderly, and increasing warfarin (a blood thinner used in heart conditions)-associated ICH were likely contributors to this observed increase. Radiological evidence of extension of intraventricular bleed, warfarin use, lobar location of bleed, and increasing age correlated with poorer survival. This data will be available for comparison with future studies to assess trends in incidence, patient characteristics and outcome in ICH.

Secondary prevention of vertebral fractures in a large New Zealand District Health Board
Katherine Bloomfield, Joe Singh

Osteoporosis is a common medical condition affecting many New Zealand older adults. Vertebral fractures are a common manifestation of osteoporosis and strongly predict future fractures. Medications proven to help prevent further fractures are available, yet this audit shows many patients with prior fractures presenting to Waitemata District Health Board were not adequately treated. This appeared particularly so for men with prior fractures.

Emergency peripartum hysterectomy: a 10-year review in a tertiary obstetric hospital
Tze Yoong Wong

According to the World Health Organization (WHO) postpartum haemorrhage (bleeding after a woman has just given birth) accounts for one-quarter of maternal deaths worldwide. There are occasions when an emergency peripartum hysterectomy (EPH) is necessary to remove the uterus, in order to control these haemorrhages. Although it is a life-saving procedure, this study found that EPH is itself associated with considerable maternal morbidity. This study also found that the most common indication for an EPH is abnormal placental implantation where the placenta is too close to the cervix or goes through the uterus lining, as a result of a previous caesarean delivery. It is hoped that this study will alert medical practitioners to this indication so that they will be more vigilant about the placental implantation in women with previous caesareans antenatally e.g. scans to determine placental site,

and the medical team and woman can prepare and plan ahead of the delivery if this problem arises.

Outcomes of patients with untreated severe aortic stenosis in real-world practice
Suresh Perera, Namal Wijesinghe, Elene Ly, Gerard Devlin, Sanjeevan Pasupati

Aortic stenosis once symptomatic can be debilitating and deadly. We attempted to assess the burden of this disease in New Zealand using Waikato Hospital as a reference centre. We found about half our patients referred for surgical aortic valve replacement are declined surgery. During our follow-up (34 months) 73% of patients were dead who were declined from surgery compared to 18% of operated patients. Also the surgically declined patients were significantly debilitated with symptoms leading to poor quality of life and required more hospital stay utilising the health dollar.

Review of 100 consecutive microvascular free flaps
Ryan Gao, Stanley Loo

Free flap surgery is an integral part of modern day plastic surgery. The operation involves the transfer of tissue, along with its blood supply from the original (donor) location to another (recipient) location. Free flaps are extremely versatile and they can be performed for a variety of reasons including reconstructing a breast following breast cancer surgery; coverage of a defect following tumour resections from the head and neck region and coverage of exposed bones following repair of fractures. After decades of refinement of surgical techniques and improved perioperative management, success rates of more than 90% have widely been reported in the international literature. This study showed that free flap reconstructions in our regional plastics and reconstruction centre have an excellent success rate (96%); and the results are comparable to international literature.

Colonic self-expanding metal stents (SEMS) in acute large bowel obstruction
Mohammad I Khan, Adrian Claydon

Colonic self-expanding metal stents are increasing used in the management of acute large bowel obstruction, both as a bridge to surgery and as a definitive palliative measure in patients unfit for surgery. We describe our experience from a New Zealand hospital and compare our data with that already published in literature. Our technical and clinical success rates were 90% and 88% respectively. The procedure was palliative in 15 patients and as a bridge to elective surgery in 13 cases. Procedure-related mortality was 7%. It was because of one early and one late perforation. The average length of stay post procedure was 2 days. Mean survival post stent insertion in the palliative group was 2.4 months and for those with a bridge to surgery was 14 months. Conclusion Our results support the data published from international centres in terms of deployment of SEMS in patients with acute large bowel obstruction, both as a bridge to surgery and as a definitive palliative measure.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

New Zealand is not an island when it comes to global health policy engagement

Don Matheson, Belinda Loring

The viewpoint article by McCool and Bullen in this edition of the *Journal* makes a compelling case for New Zealand's continued engagement in global health.[1] This engagement is not optional—the choice is between striving for policy coherence across different aspects of our health impact on other nations or continuing to segment the health, economic and environmental impacts of our international activities. New Zealand can contribute as well as learn from the experience of other health systems.

In health system performance comparisons across 7 nations in 2010,[2] New Zealand ranks 5th, down from 3rd equal with Australia in 2006.[3] We spend less than comparable nations; lead in terms of quality, coordinated and patient-centred care; but languish just ahead of the United States in terms of equity. This reinforces the importance of the recent health equity focus in this *Journal* and the NZMA,[4] and the need to keep health equity uppermost in the health sector's mind. Progressing health equity needs to strongly engage actors outside of the health sector, both nationally and internationally.

With economic recovery less than certain,[5] we need to take a closer look at how other nations are achieving good results for less.[6] Innovations are occurring in some countries, such as the Thailand Health Assembly[7] which would be valuable in the New Zealand context in developing a greater sense of direction for the sector by bringing together various actors and sectors involved in the social production of health, including groups often marginalized in policy making.

New Zealand's main contribution to global health derives from our farmers rather than our health workers. In a world that is becoming increasingly food insecure, we 'punch above our weight" in terms of food exports. Our biggest export,[8] dairy products, supplies 35% of the international dairy trade, and has possibly our biggest impact on global health. Our next biggest food export is meat products. A challenge for both these industries, and for New Zealand's international health reputation, is the extent to which they are able to supply much needed protein to the world, and at the same time avoid contributing to the advancing noncommunicable diseases (NCDs)[9] such as diabetes that have reached epidemic proportion in the Pacific, and are fast emerging as the major health challenge of the century, particularly for our growing markets in India and China.

In the past, New Zealand's trade in goods such as mutton flaps and tobacco have contributed to rising levels of NCDs in the Pacific.[10] Currently the dairy industry's largest export is concentrated or sweetened milk and cream. The free trade agreement with China[11] has boosted mutton flap sales to $170m in the last year. Both industries are making efforts to decrease fat content of their products, and develop new products that are more conductive to a healthy life. The net health impact of our food exports is unclear, but as a nation we need in future to play closer attention to making a positive

contribution to global food supplies, and not exacerbate recipient countries over nutrition problems.

Better policy coherence is required across our externally focused policies. New Zealand's current aid strategy of pursuing economic development as a core focus,[12] has the potential to further exacerbate NCD rates in the Pacific. This is not without consequence for New Zealand—New Zealand's health system is footing the bill for the consequences of the rising NCD epidemic, in visitors and migrants from the Pacific.[10] This lack of policy coherence means that New Zealand's trade and aid policies are undermining its own health budget.

Having a healthy, fair and stable region is not just good for others, it is in New Zealand's self-interest—in containing health care costs, and broader objectives such as regional security, environmental sustainability and economic prosperity. The health of New Zealanders faces real threat from a number of more global sources, more powerful and insidious than traditional infectious threats mentioned by McCool and Bullen.

Free trade agreements threaten to undermine PHARMAC[13] a key pillar of the New Zealand health sector's success. Having developed an agency that consistently negotiated lower prices for essential drugs than other nations, it has been singled out for elimination by Big Pharma, despite, or because of, its attractiveness to health systems worldwide. Our leadership in crafting the tobacco end game[14] will require strong global partners to withstand the inevitable legal challenges by Big Tobacco, a preamble to which is currently being played out in Australia as it attempts to pass plain packaging legislation.

Engagement in global health mechanisms is crucial for countries (especially small ones like New Zealand) to participate in decisions and negotiations with much larger players, including the private sector, to mitigate the harmful impacts and leverage positive contributions to health both for New Zealanders and others in our interconnected world.

New Zealand is inextricably linked to the rest of the globalised world, and participates constantly in a range of activities with direct implications for global health, through trade, agriculture, aid, and health policies. The decision on whether to engage is not up for debate—the decision is whether New Zealand engages coherently, explicitly and in a way that optimises the wellbeing of New Zealanders and of those around us.

Competing interests: None.

Author information: Don Matheson, Professor, Centre for Public Health Research, Massey University, Wellington; Belinda Loring, Senior Policy Officer, Global Action for Health Equity Network (HealthGAEN), Auckland

Correspondence: Don Matheson. Email: D.P.Matheson@massey.ac.nz

References:

1. McCool J, Bullen C. "Punching above its weight": why New Zealand must maintain leadership in global health. N Z Med J. 2011;124(1345). http://journal.nzma.org.nz/journal/124-1345/4935/content.pdf
2. Davis K, Schoen C, Stremikis K. Mirror on the Wall: How the Performance of the U.S. Health Care System Compares Internationally 2010. The Commonwealth Fund; 2010.

3. Davis K, Schoen C, Schoenbaum S, et al. Mirror, mirror on the wall: an international update on the comparative performance of Amercian health care. [Internet]. The Commonwealth Fund; 2007. http://www.commonwealthfund.org/usr_doc/1027_Davis_mirror_mirror_international_update_final.pdf
4. New Zealand Medical Association. Health equity position statement. N Z Med J. 2011;124(1330). http://journal.nzma.org.nz/journal/124-1330/4569/content.pdf
5. English B. Pre-election Economic and Fiscal Update 2011 from the Minister of Finance. Wellington: Minister of Finance; 2011.
6. Balabanova D, McKee M, Mills A. Good health at low cost' 25 years on. What makes a successful health system? London: London School of Hygiene & Tropical Medicine; 2011.
7. Rasanathan K, Posayanonda T, Birmingham M, Tangcharoensathien V. Innovation and participation for healthy public policy: the first National Health Assembly in Thailand. Health Expect [Internet]. 2011 Feb 1 [cited 2011 May 12]; http://www.ncbi.nlm.nih.gov/pubmed/21281413
8. New Zealand Trade & Enterprise. The New Zealand dairy industry [Internet]. 2010. http://cache.business.newzealand.com/vAmsd7A/media/103382/dairy-industry-fact-sheet.pdf
9. World Health Organization. Global Status Report on non-communicable diseases. Global Status Report on non-communicable diseases 2010. Geneva: World Health Organization; 2011.
10. Wyber R, Wilson N, Baker M. New Zealand's impact on health in the South Pacific: scope for improvement? N Z Med J. 2009;122(1291). http://journal.nzma.org.nz/journal/122-1291/3505/content.pdf
11. Beef and Lamb New Zealand. China FTA delivering for New Zealand sheep and beef farmers [Internet]. 2011 Aug 9 [cited 2011 Oct 28]. http://www.beeflambnz.com/main.cfm?id=31&nid=365
12. New Zealand Ministry of Foreign Affairs and Trade Aid Programme. International Development Policy Statement. Supporting sustainable development [Internet]. Wellington: New Zealand Ministry of Foreign Affairs and Trade; 2011. http://www.aid.govt.nz/what-we-do/Int_Dev_Policy_Statement_Supporting_Sustainable_Development.pdf
13. People's Health Movement Australia. The Trans Pacific Partnership Agreement: Implications for public health regulation and access to medicines [Internet]. 2011. http://phmoz.org/wiki/index.php?title=The_Trans_Pacific_Partnership_Agreement:_Implications_for_public_health_regulation_and_access_to_medicines
14. Edwards R, Russell M, Thomson G, et al. Daring to dream: reactions to tobacco endgame ideas among policy-makers, media and public health practitioners. BMC Public Health. 2011;11:580.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Sudden unexpected infant death—no more "stunned amazement"!

Nick Baker

The last 20 years have seen the dramatic reduction in the toll from Sudden Unexpected Death in Infancy (SUDI) in New Zealand from 200 to 60 deaths per annum. The reduction in mortality stems largely from the recognition that placing babies to sleep on their backs reduced the risk of death, as was highlighted in the New Zealand case control study in 1992.[1] It can be calculated that approximately 3000 infants have survived who would otherwise have died over these 20 years.

The fact that 60 deaths continue to occur annually is a major tragedy, however. Among the industrialised nations, New Zealand has the highest rate of death from SUDI.[2] The burden of this problem falls disproportionately in the Māori community and amongst families living in deprived circumstances.[3] The total mortality rate is 1.1 deaths per 1000 live births—the Māori rate is at 2.3 deaths per 1000 births while the rate for other ethnicity is 0.52 per 1000 births.[4]

Over the last few years, coroners rulings have increasingly stressed that many deaths from SUDI are "unnecessary and preventable" with intense focus falling on the hazards associated co-sleeping (when an infant and other individual/s adult, infant or child are sleeping together on a shared surface e.g. bed, couch, chair, floor, mat or other surface).[5,6]

> …Mothers and families are simply not getting the message and although loving their babies dearly and thinking they are doing the very best for them they are in fact unwittingly "killing" them. This is a very sad state of affairs and it seems to me, can only be rectified by education and on the ground assistance for mothers [Coroner Dr Wallace Bain].[5]

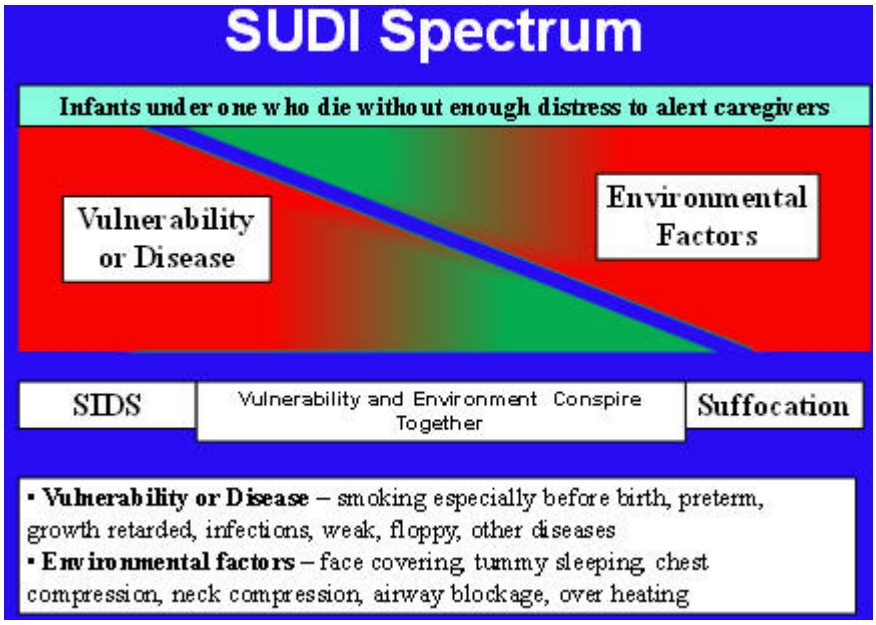
SUDI is an umbrella term used to describe a heterogeneous group of infants under age one who die without warning signs or distress sufficient to alert parents or caregivers. The term SUDI therefore relates to the experience from the viewpoint of parents or caregivers and allows preventive measures to target all the conditions within the group. The term encompasses a spectrum of cases ranging from those that remain unexplained following full investigation (SIDS) to cases which are fully explained (Figure 1). Between the two ends of the spectrum are cases where a pathologist or coroner is unclear to what extent the deaths are explained, and which tend to be called "unascertained."

Unexpected deaths where significant external forces are applied—e.g. motor vehicle crashes or assault are not included within the term SUDI. The explained SUDI group includes cases that are clearly attributable to factors which on their own alone, would be sufficient to cause death such as suffocation, strangulation or overwhelming infection. Typical circumstances of accidental suffocation include wedging or overlying.

Wedging occurs when a baby moves into a confined space such as a gap between wall and mattress, between mattresses, is entrapped in a faulty cot or amongst couch

cushions. With regard to overlaying, case review studies suggest that it does not require the whole body of another person to be on the baby. A limb, breast or even a small sibling can be sufficient to cause fatal compromise. In both these situations airway impairment can occur through face covering or neck flexing[7] and pressure on the chest can compromise breathing.[8]

Figure 1. Interaction between cause of death, vulnerability and environmental factors in sudden unexpected death in infancy



SUDI=Sudden Unexpected Death in Infancy, SIDS=Sudden Infant Death Syndrome.

A high proportion of infants that die from SIDS have "intrinsic vulnerabilities" such as tobacco exposure before birth, prematurity or growth retardation.[4] For some these intrinsic vulnerabilities are associated with death without any external factor or illness contributing. For the SUDI cases that are called unascertained, infant factors and environmental factors conspire together leading to an accumulation of risk which proves lethal. Whenever intrinsic vulnerabilities exist in an infant it is especially important that environmental factors which may compromise the infant are minimised. Any baby may, however, suffer suffocation or strangulation if placed in an unsafe sleeping environment.

Coroners only review cases where infants have died, and health professionals predominantly work with families whose infant care practices have not lead to death. It is no surprise that there are differences of opinion as to the best approach for SUDI prevention. Unfortunately polarisation of opinion can lead to confusion and failure to deliver clear messages to families. It is the responsibility of all professionals (health, legal, and social services) to deliver clear and consistent messages regarding SUDI prevention in order for us to learn lessons from these fatal cases and make a substantial reduction in New Zealand's unacceptable SUDI toll.

Coroners comment that on almost every occasion families have "stunned amazement" that the circumstances their baby died in were dangerous.[3,5] This represents a major failure of risk communication by health and other professionals. Families have a right to be informed that there are definite risks while co sleeping for all infants, especially under 3 months of age,[8] and greater risks for more vulnerable infants.

Families must have clear explanations and be supported in having a plan for sleeping arrangements tailored to their situation and infant needs which aims to minimise risks in the sleeping environment. If families decide to not follow the plan they must do so with their eyes open in full knowledge of the risks, just as people know swimming in deep turbulent water is never completely safe, and that there are some circumstances related to swimming skills or water conditions when the water must be avoided!

Information to reduce SUDI needs to be provided to families in the form of public health messages, antenatally, at birth and during postnatal and ongoing infant care. Health services need to model sleeping practices that reduce risks in all settings. When delivering information, messages need to be clear, consistent and take into account the health literacy and the reality of the lives lived by people from the deprived communities where risks cluster.

The low health literacy skills in some Māori and lower income households are likely to have a negative impact on the uptake of health messages and need to be recognised as communication techniques are developed.[9] In some Māori families, where SUDI occur, the mothers and family as a whole may be living in "survival mode", contending with multiple stressors and marginalised from wider health and social support networks. In this setting, simple provision of information is a poor mechanism for change; efforts are needed to support engagement with innovative and culturally appropriate behaviour modification approaches as well as addressing the determinants of deprivation.[10]

To reduce risk it is important that all infants have a smokefree place where they can sleep on their back, on a firm surface, with their faces clear in an arrangement where they cannot be trapped and nothing can accidentally move to cover their face or flex their neck. Even when an infant sleeps alone in a bed designed for an adult, there is at least a twenty-fold increased risk of suffocation compared with an infant in a cot.[11]

Furthermore, infants are also placed at increased risk when placed to sleep in faulty cots and cots with an inappropriately fitted mattress. New Zealand is developing some options to support safe sleep with the increasing use of Wahakura and Pepi Pods which are currently undergoing research evaluation. Work and Income NZ (WINZ) is also able to offer financial support for sleeping spaces for infants.[4]

The unexpected nature of SUDI can lead to the unfortunate acceptance that these deaths "just happen" and cannot be prevented, and leave families feeling disempowered. In fact a substantial proportion of SUDI are preventable. Health professionals have an absolute duty to ensure that families are never again left with "stunned amazement" if their infant dies in a setting of unsafe sleep.

Competing interests: None.

Author information: Nick Baker, Community Paediatrician, Nelson Marlborough District Health Board, Nelson Hospital, Nelson—and Chair, New Zealand Child and Youth Mortality Review Committee

Correspondence: Dr Nick Baker, Community Paediatrician, Nelson Hospital, Private Bag 18, Nelson, 7042, New Zealand. Fax: +64 (0)3 5461212; email: nick.baker@nmhs.govt.nz

References:

1. Mitchell EA, Taylor BJ, Ford RPK, et al. Four modifiable and other major risk factors for cot death: The New Zealand Study. J Paediatr Child Health. 1992;28(Suppl 1):S3–8.
2. Kinney HC, Thach BT. The Sudden Infant Death Syndrome. N Engl J Med 2009;361:795-805.
3. Craig E, Jackson C, Han DY, NZCYES Steering Committee. Monitoring the Health of New Zealand Children and Young People: Indicator Handbook. 2007. Auckland: Paediatric Society of New Zealand, New Zealand Child and Youth Epidemiology Service.
4. Child and Youth Mortality Review Committee, Te Rōpū Arotake Auau Mate o te Hunga Tamariki, Taiohi. 2009. Fifth Report to the Minister of Health: Reporting mortality 2002–2008. Wellington: Child and Youth Mortality Review Committee.
5. Bain W. Finding of Coroners Court Rotorua, 16 December 2010, Inquests into the Deaths of Babies F,G,H,I.
6. Evans GL. Report of the Coroners Court held at Wellington on 9 December 2008. Wellington, New Zealand: Coroners Court; 2009.
7. McIntosh CG, Tonkin SL, Gunn AJ. What is the mechanism of sudden infant deaths associated with co-sleeping? N Z Med J. 2009;122(1307). http://journal.nzma.org.nz/journal/122-1307/3905/content.pdf
8. Tappin D, Ecob R, Brooke H. Bedsharing, roomsharing, and sudden infant death syndrome in Scotland: A case-control study. J Pediatr. 2005;147:32–7.
9. Ministry of Health. Kōrero Mārama: Health Literacy and Māori Results from the 2006 Adult Literacy and Life Skills Survey. Wellington: Ministry of Health, 2010.
10. McManus V, Abel S, McCreanor T, Tipene-Leach D. Narratives of deprivation: Women's life stories around Maori sudden infant death syndrome. Social Science & Medicine. 2010;71:643e649.
11. Scheers NJ, Rutherford GW, Kemp JS. Where Should Infants Sleep? A Comparison of Risk for Suffocation of Infants Sleeping in Cribs, Adult Beds, and Other Sleeping Locations Pediatrics 2003;112;883-889.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

PSA testing in detection and treatment of prostate cancer

David S Lamb, Brett Delahunt, John N Nacey

After 2 years of deliberation, the Parliamentary Select Committee investigating the early detection and treatment of prostate cancer released its report on 27 July 2011.[1] The report encourages New Zealand men to seek up-to-date evidence-based information from their general practitioners about the advantages and disadvantages of screening for prostate cancer. The report also recommends that general practitioners routinely provide this information as part of the cardiovascular risk assessment performed on men aged 45 years. For men with known genetic risk factors for prostate cancer, the recommendation was that testing should be offered at an earlier age.

At first glance, the report might be taken as a simple statement defining the *status quo*, but this is not the case. Currently, only about 40% of the New Zealand male population over the age of the age of 50 years undergo regular prostate-specific antigen (PSA) testing for prostate cancer, which means that well over half of the male population is not being tested. This is a point of concern for two reasons; firstly, the Austrian Tyrol study suggests that the coverage of a PSA testing programme is more important for achieving population survival benefits than the intensity of the PSA testing.

The Austrian Tyrol study showed that testing of 86% of the population, many with a single PSA test, reduced prostate cancer mortality by 56%.[2] Secondly, data from New Zealand and Australia suggest that tested and untested groups of men have large ethnic and regional differences, indicating there are significant inequalities introduced by poor coverage. Māori men are tested less often than non-Māori,[3] and men who reside in country areas are tested less often than men who live in urban centres.[4]

The Select Committee report is now recommending that the key information on PSA testing should be provided to all men, so equitability is restored, and every man is able to make an informed decision on whether or not he wishes to be tested. The Select Committee is to be congratulated on their interpretation of the apparently conflicting results of the PSA-based screening trials reported to date. There are three basic requirements for a trial result to have any credibility.

Firstly, the randomisation process needs to be properly balanced, so that men in the screened arm should match those in the control arm. This was a fault of the Quebec trial[5] which was the first to report survival advantages for PSA-based screening.

Secondly, the follow up of men on screening trials for prostate cancer must extend over many years for a potential difference to be demonstrated. This is because the lead time from diagnosis by PSA based screening is 6–12 years.[6] The European Randomised Study of Screening for Prostate Cancer (ERSPC) was therefore designed to report *as soon as* a prostate cancer survival advantage of 20% was detected in the screened arm, which occurred after a median follow up of nine years.[7] The Göteborg randomised trial demonstrated that the survival advantage had increased to 40% by 14

years median follow up, and projections showed that the advantage was still increasing at that time.[8]

Thirdly, any randomised screening trial cannot draw from a population where PSA testing has already become common practice. This was a major fault in The Prostate, Lung Colorectal and Ovarian Cancer Screening Trial (PLCO) which reported no apparent survival benefit from screening, with a median follow up of 11.5 years.[9] However, the reason for this result was that in the three years prior to participation in the PLCO trial, 44% of men had had at least one PSA test, and by the sixth year of follow up, 52% of men on the control arm had had at least one PSA test after enrolling on the trial. Such contamination of the trial population reduces differences in PSA testing practices between the control and screened arms, and makes the result of the PLCO trial 'inconclusive' rather than 'negative'.

Once the flawed screening trials are removed from the equation, only the ERSPC and Göteborg trials remain, and the results of these trials give a consistent message. PSA testing *does* save lives,[10] and the number of lives saved increases as the time interval from the commencement of PSA testing becomes longer. This means that younger men are more likely to achieve a survival benefit from the early diagnosis and treatment of their prostate cancer than older men, due to an increased mortality from unrelated causes in the older age group.

The question will now be asked as to whether or not the survival advantages are large enough to justify the effort and the costs to the health services resulting from additional PSA testing. In addition to this the (albeit small) risks to men associated with prostatic biopsy need to be factored in. Of the approximately 600 men who die of prostate cancer each year in New Zealand, we estimate that about 400 will initially present with metastatic disease.

PSA testing has been shown to reduce the likelihood of presenting with metastases by 50 percent,[11] and as a consequence PSA testing has the potential to save up to 200 lives each year. Ultimately it is the decision of individual men to determine whether or not they agree with us and elect to be tested, just as every man has the right to decide whether or not the risks of the disease outweigh the risks of treatment, after prostate cancer is diagnosed.

Competing interests: None.

Author information: David Lamb, Associate Professor, Department of Pathology and Molecular Medicine, University of Otago, Wellington; Brett Delahunt, Professor, Department of Pathology and Molecular Medicine, University of Otago, Wellington; John N Nacey, Professor, Department of Surgery and Anaesthesia, University of Otago, Wellington.

Correspondence: Associate Professor David Lamb, Department of Pathology and Molecular Medicine, University of Otago, PO Box 7343, Wellington South 6242, New Zealand. Email: david.lamb@otago.ac.nz

References:

1. Parliamentary Select Committee Report on the early detection and treatment of prostate cancer. Released 27 July 2011

2. Oberaigner W, Horninger W, Klocker H, et al. Reduction of prostate cancer mortality in Tyrol, Austria, after introduction of prostate-specific antigen testing. Am J Epidemiol 2006;164:376-384. http://aje.oxfordjournals.org/content/164/4/376.long
3. Lamb DS, Bupha-Intr O, Bethwaite P, et al. Prostate Cancer – are ethnic minorities disadvantaged? Anticancer Res 2008;28:3891-3896.
4. Coory MD, Baade PD. Urban-rural differences in prostate cancer mortality, radical prostatectomy and prostate-specific antigen testing in Australia. Med J Aust 2005;182:112-115.
5. Labrie F, Candas B, Cusan L, et al. Screening decreases prostate cancer mortality: 11-year follow up of the 1988 Quebec prospective randomised controlled trial. Prostate 2004;59:311-318.
6. Draisma G, Boer R, Otto SJ, et al. Lead times and overdetection due to prostate-specific antigen screening: estimates from the European Randomised Study of Screening for Prostate Cancer. JNCI 2003;95:868-878.
7. Schroder FH, Hugosson J, Roobol MJ, et al. Screening and prostate-cancer mortality in a randomised European study. N Engl J Med 2009;360:1320-1328.
8. Hugosson J, Carlsson S, Aus G, et al. Mortality results from the Göteborg randomised population-based prostate-cancer screening trial. Lancet Oncology Published online July 1, 2010.
9. Andriole GL, Crawford ED, Grubb RL, et al. Mortality results from a randomized prostate-cancer screening trial. N Engl J Med 2009;360:1310-1319.
10. National Cancer Institute bulletin 13 July 2010. PSA screening substantially improves cancer-specific survival without the extent of over-diagnosis and treatment. National Cancer Institute: Bethesda, MD 20892-8322.
11. Aus G, Bergdahl S, Lodding P, et al. Prostate cancer screening decreases the absolute risk of being diagnosed with advanced prostate cancer – results from a prospective, population-based randomized controlled trial. Eur Urol 2007;51:659-664.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Temporal trends and clinical characteristics of spontaneous intracerebral haemorrhage in the Waikato region of New Zealand: a hospital-based analysis

James Irwin, Peter Wright, Paul Reeve

Abstract

Aims To determine the incidence, and any change in incidence, of spontaneous intracerebral haemorrhage (ICH) detected in the hospitals of the Waikato region of New Zealand (NZ) between 1999 and 2008. To analyse clinical and patient parameters, and to correlate these with outcome.

Methods A retrospective analysis was performed on patients presenting to Waikato and Thames Hospitals with ICH during the study period. Radiology reports, blood tests and the electronic clinical record were reviewed for each patient.

Results 653 episodes of ICH were identified. The average annual incidence per 100,000 per year was 17.4 (16.1–18.7, 95% confidence interval). This increased from an average of 14.4 (13.7–15.1) between 1999–2001 to 21.4 (20.6–22.2) between 2006–2008 (rate ratio 1.49, p<0.0001). 249 (38.1%) patients died within 30 days of their sentinel bleed. The presence of intraventricular extension of bleed on neuroimaging (Odds Ratio (OR) 6.18, p<0.001), warfarin use (OR 1.11, p=0.76), warfarin use *and* intraventricular extension of bleed (OR 23.8, p=0.014), lobar location of bleed (OR 1.88, p=0.001) and age (OR 1.16 for every 10-year increase in age, p=0.02) increased the likelihood of death within 30 days.

Conclusion Observed ICH has increased in incidence in our hospitals over the past 10 years. Increasing availability of neuroimaging, increasing numbers of elderly, and increasing warfarin associated ICH were likely contributors to this observed increase. Radiological evidence of extension of intraventricular bleed, warfarin use, lobar location of bleed, and increasing age correlated with poorer survival. This data will be available for comparison with future studies to assess trends in incidence, patient characteristics and outcome in ICH.

Intracerebral haemorrhage (ICH) is a significant cause of morbidity and mortality. Published community based incidence figures vary between 20–31 per 100,000 per year.[1,2] These patients make up 10–15% of all acute stroke events in Western populations.[3] Presenting symptoms include headache, focal neurological deficit, seizures and a decreased level of consciousness. Previous analyses have demonstrated that between 30–50% die within 30 days of their bleed.[1,2,4]

Survivors are often left dependent on others for care, and represent a significant drain on hospital and community resources.

Epidemiological studies have yielded uncertain conclusions regarding temporal trends in incidence of ICH.[1] Four population based stroke incidence studies in the past 30 years have analysed temporal trends in ICH incidence.[5–8] Crude incidence decreased

in Perth (Australia), while remaining stable in Dijon (France), Oxford (England) and in Finland. Anecdotally we felt incidence had been increasing in our hospital, and this hospital based retrospective analysis was performed to determine if this was true. The data was also analysed to identify parameters which correlated with any identified change in incidence, and parameters which correlated with 30-day survival.

The Waikato region of New Zealand has a population of approximately 375,000, and is serviced by Waikato Hospital in Hamilton and four smaller hospitals in peripheral centres. CT scanning is available in Waikato Hospital, and in one of the peripheral hospitals (Thames Hospital). All data regarding admission to these two hospitals and all radiology are recorded on the Waikato Hospital database.

Methods

An electronic search was performed on all hospital admissions entered on the IPM database of the Waikato District Health Board, for the 10 years between 1 December 1998 and 31 November 2008. Adults (age 15 or older) with an ICD-10 coded discharge diagnosis[9] of I61.0–I61.9 or I62.9 (all forms of intracerebral haemorrhage, isolated intraventricular haemorrhage and intracranial haemorrhage not specified) were captured. A search was also performed on the emergency department database for all patients who died in the emergency department during the study period.

All electronic CT and MRI reports of brain scans were reviewed for all patients. Patients with a neuroimaging report which documented a primary subarachnoid haemorrhage, subdural haemorrhage, extradural haemorrhage, traumatic intracranial haemorrhage, intracranial bleed associated with a neurosurgical procedure, an underlying malignancy or an arteriovenous malformation were excluded. Only patients with a spontaneous intracerebral haemorrhage or an isolated intraventricular haemorrhage were included in the dataset. Included patients had their age, sex, ethnicity, date of admission, date of discharge and discharge status (alive or dead) retrieved from the database.

The location of the bleed was recorded as being either lobar (in a cerebral hemisphere), deep (basal ganglia or brainstem), cerebellar or isolated intraventricular. If blood was identified in more than one location, the location where the reporting radiologist felt the primary bleed had occurred was recorded as the location of the bleed. Extension of bleed into the ventricular system was noted.

Evidence of warfarin use was sought in the electronic clinical record. The INR on admission, if performed, was recorded. Patients with an INR $\geq$1.5 had their paper clinical record retrieved and warfarin use was documented.

The data was analysed using R.[10] Comparison of ICH rates between different time periods was performed using the *rateratio.test* package in R, assuming a Poisson distribution of ICH events. The trend in incidence was analysed comparing rates in 1999–2001 with 2006–2008. For each period weighted estimates of Waikato regional population counts were made from 1996, 2001 and 2006 census data.[11] Association between predictor variables and 30-day mortality was analysed using logistic regression using the *glm()* function (stats package, version 2.11.0) in R.

Possible variables for the regression model were selected using screening univariate analysis—for categorical variables using a chi squared test, and for continuous variables using univariate logistic regression analysis. Variables significantly correlated with 30-day mortality (p value<0.10) were added sequentially into a logistic regression model, and those with significant independent correlation with 30-day mortality were retained.

The data was analysed to confirm the absence of significant outlier values, and interactions between variables were searched for and included in the final model if significant. A covariance matrix was generated to identify significant colinearity between predictor variables. Odds ratios were calculated as the exponential of the β value of each predictor variable, and as $e^{\beta\,(\text{main term 1})} \times e^{\beta\,(\text{main term 2})} \times e^{\beta\,(\text{interaction term})}$ for interaction terms. All p values calculated were 2-tailed, and a value of <0.05 was considered to be statistically significant.

Statistical information regarding age, ethnicity and sex stratified population counts in the Waikato region were obtained from Statistics New Zealand, using 1996, 2001 and 2006 Census data.[11]

Results

653 episodes of ICH were recorded during the study period. 20 were recurrent episodes in patients who had already suffered an ICH episode during the study period, and 12 episodes were multifocal. The mean incidence of ICH was 17.4 per 100,000 persons per year. There was no significant difference in incidence between sexes.

The majority of ICH occurred in the NZ European ethnicity group (Table 1). Rates in Māori, Pacific Island and Asian populations were lower than population prevalence would predict.

Table 1. ICH incidence stratified by ethnicity

Ethnicity	Frequency of ICH	Waikato population by ethnicity*	Ethnicity-specific incidence (per 100,000 per year)
Asian	14	14,816	9.4
Māori	78	80,910	9.6
NZ European	500	305,180	16.4
Pacific Island	11	11,935	9.2
Other	20	–	–
Not recorded	30	–	–
Total	**653**	**375,408**	**17.4**

*Taken as a weighted average for 1998–2008 from 1996, 2001 and 2006 census figures.

Incidence of ICH increased during the study period (Figure 1), from 14.4 per 100,000 per year (95% CI 13.7–15.1) between 1999–2001, to 21.4 (20.6–22.2) between 2006–2008 (rate ratio 1.49, p<0.0001).

107 episodes of ICH occurred in patients taking warfarin, increasing from an average of 2.20 per 100,000 per year in 1999–2001, to 4.05 in 2006–2008 (RR 1.84, p=0.018, Figure 1).

Incidence increased with age (Table 2), and the majority of ICH occurred in a lobar location (Table 3). The mean age at presentation was 71.7 years for those of NZ European ethnicity, 62.4 for Māori, 64.0 for Pacific Island and 59.9 for Asian ethnicity.

Table 2. Age-specific incidence of ICH

Age	Frequency of ICH	Waikato age-specific population*	Age-adjusted incidence (per 100,000 per year)
15–44	38	159,468	2.4
45–54	45	47,759	9.4
55–64	107	34,775	30.8
65–74	163	24,994	65.2
75–84	223	14,488	153.9
>85	77	4282	179.8

*Taken as a weighted average for 1998–2008 from 1996, 2001 and 2006 Census figures.

Figure 1. Yearly frequency of ICH (blue=total, green=warfarin-associated)

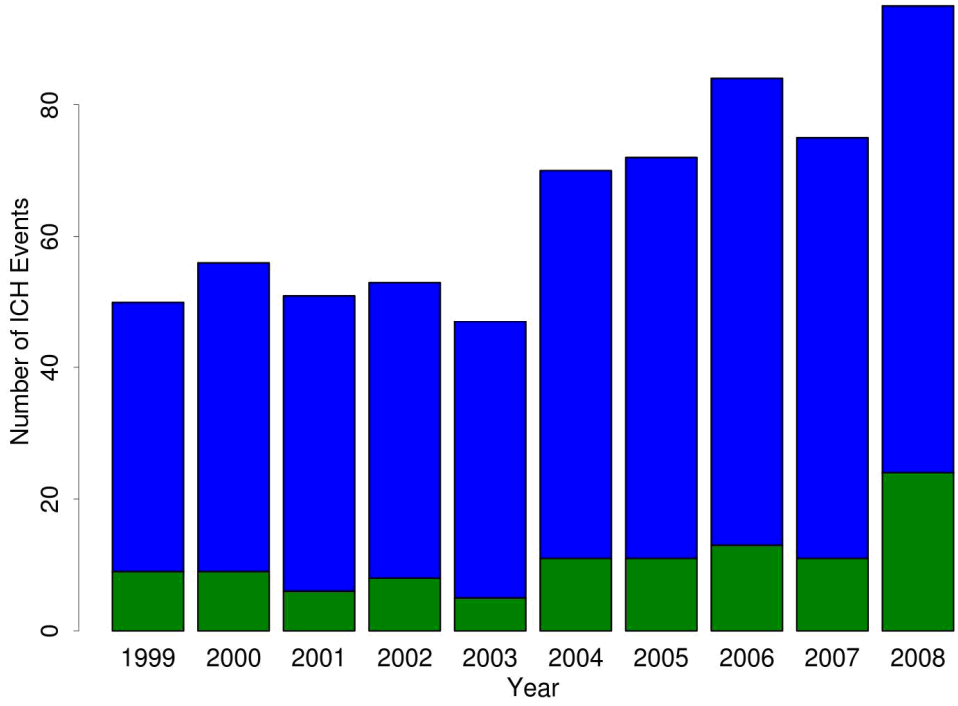


Table 3. Location of ICH

Location of ICH	Frequency	Percentage of all ICH episodes
Deep (brainstem/basal ganglia/deep white matter)	205	31.3%
Lobar	361	55.3%
Cerebellar	65	10.0%
Isolated intraventricular	10	1.5%
Not known (radiology not available)	12	1.8%
Total	**653**	**100%**

By univariate analysis, intraventricular extension of haemorrhage, warfarin use, age, lobar location of bleed and Māori ethnicity were significantly associated with 30-day mortality (Table 4).

The final logistic regressional model included intraventricular extension of haemorrhage, warfarin use, lobar location of bleed and age as independent predictor variables (Table 5). A significant interaction existed between warfarin use and intraventricular extension of bleed. For those not taking warfarin intraventricular extension of bleed was strongly predictive of 30-day mortality.

For those taking warfarin this effect was multiplied threefold, meaning warfarin users who had intraventricular extension of bleed on neuroimaging had 24 times the likelihood of death within 30 days compared with those who did not have intraventricular extension of bleed and were not taking warfarin.

Table 4. Univariate analysis of predictors of 30-day mortality

Variable	Odds Ratio	P-value
Ethnicity New Zealand European Māori	 1.0 0.55	 Reference 0.03
Age (per 10-year increment)	1.18	0.006
Male sex	1.31	0.10
Warfarin use	1.92	0.002
Location of bleed Basal ganglia/brainstem Lobar Cerebellar	 1.0 1.43 1.67	 Reference 0.05 0.08
Isolated intraventricular	2.20	0.22
Ventricular extension of bleed	5.96	<0.001

Table 5. Factors predictive of death within 30 days – final logistic regressional model

Predictor variable	β value	Odds Ratio*	P-value
Age (per 10 year increment)	0.15	1.16	0.022
Lobar location of bleed	0.631	1.88	0.001
Warfarin use, no ventricular extension of bleed	0.103	1.11	0.76
Ventricular extension of bleed, no warfarin use	1.82	6.18	<0.001
Warfarin use *and* ventricular extension of bleed	1.24	23.8	0.014

*Odds Ratio is equivalent to e^{β}.

For those taking warfarin, the median INR at the time of bleed was 2.7; 62% of patients had an INR≤3.

Discussion

This analysis demonstrates a 49% temporal increase in incidence of hospital observed ICH between 1999 and 2008 in the Waikato Region. It also shows ventricular extension of bleed, warfarin use, lobar location of bleed and age to be associated with increased 30-day mortality. A lower incidence of ICH in Pacific Island, Māori and Asian populations was noted.

Community based stroke incidence studies suggest ICH incidence over the past 20 years has either remained static or decreased.[1] Our data shows a significant increase in observed ICH in our region's hospitals. We have collected a set of ICH events detected in hospital, and therefore cannot extrapolate our observed trends in incidence

to the general population. However, in hospital we are clearly identifying and treating increasing numbers of patients with ICH.

We consider this increase may be the result of increasing use of neuroimaging to detect ICH, of increasing presentation of patients with ICH to hospital, or of truly increasing ICH incidence. The potential contribution of these three factors is considered below.

A proportion of patients suffering ICH in our region during the study period will have died before having had neuroimaging performed. Without a postmortem these patients would not have been diagnosed with ICH and would not have been included in this dataset. The proportion of ICH patients who did not receive neuroimaging may have been higher 10 years ago, when CT scanning was a scarcer resource. There has been a threefold increase in the number of brain CT scans performed on emergency department patients in Waikato and Thames hospitals over the study period (Personal Communication, Radiology Department, Waikato Hospital).

In a comparably resourced area, the Auckland Regional Community Stroke Study Group (ARCOS) recorded that the proportion of all stroke sufferers who underwent neuroimaging increased from 42% in 1991-92 to 88% in 2002-03.[12] In our study increasing performance of CT scanning has probably contributed to a perceived increase in ICH incidence during the study period.

There may have been a change in referral pattern for patients suffering acute neurological events over the study period; both to hospital from the community, and for neuroimaging when in hospital. Expectations of care for patients by general practitioners (GP's) and hospital specialists are influenced by availability of tests and treatments.

An increase in availability of CT scanning might be expected to alter GP referral patterns of patients suffering an acute neurological event, and also to alter acceptable indications for in-hospital neuroimaging requests. This could equally apply to patients with transient neurological symptoms as to semi-moribund patients, who in previous times may have remained at home with an expectation of death.

We also consider that factors exist which may have led to a true increase in ICH incidence. We have observed an increase in warfarin associated ICH, are aware of an increase in age of our population, and are aware of an increase in antiplatelet medication and HMG CoA reductase inhibitor (statin) use.

Warfarin use approximately doubles the risk of ICH.[13] Indications for long-term warfarin treatment include prophylaxis of cardiogenic thromboembolism in patients with atrial fibrillation or artificial heart valves, and prophylaxis of venous thromboembolism. Studies in the 1990s confirmed the benefit of treatment with warfarin to reduce the risk of stroke in patients with atrial fibrillation.[14-16]

The number of prescriptions for warfarin in the Waikato region has increased by a factor of 1.65 between 1999 and 2008 (Personal Communication, NZ Ministry of Health [NZMOH]). Whilst acknowledging that the temporal increase in warfarin associated ICH observed in this study is also subject to the confounding effect of changing neuroimaging practice and changing referral patterns, it may have contributed to the observed temporal increase in ICH.

There has been a 41% increase in those aged ≥75 in the Waikato region between 1996 and 2006, from 15,430 to 21,730.[11] This compares to a 9% increase in total population. Statistics NZ attribute this growth to an increase in life expectancy, and an increase in birth rate during the 1930's and 1940's. Forty six percent of all ICH events in our analysis occurred in those over the age of 75, and the observed increase in this population represents an increase in those most at risk of ICH.

There was an increase in the use of antiplatelet and statin medication over the study period, both of which are associated with increased risk of ICH.[17–19] Statin use in the Waikato region increased by a factor of 4.5 over the study period, from 20,000 prescriptions per year to 85,000 (Personal Communication – NZMOH).

Aspirin use, which is associated with a 40% increase in risk of ICH,[19] increased by a factor of 3.2 in the region over the study period, from 25,000 prescriptions per year to 81,000 (Personal Communication – NZMOH). Clopidogrel and dipyridamole have been regulated over this period by PHARMAC in New Zealand restricting their use,[20] and therefore are unlikely to have contributed significantly to the absolute increase in ICH observed in this study.

The average annual incidence of ICH in this study of 17.4 per 100,000 per year is comparable to published incidence figures of 20 - 30 per 100,000 per year.[1,2] Ethnicity specific incidence was lower in Māori, Pacific Island and Asian populations, who were also younger at presentation in comparison to NZ Europeans. Statistics NZ data show that during the study period only 0.9% of Māori, 0.8% of Pacific Islanders and 1.0% of Asians in the Waikato region were aged 75 or older, compared to 5.9% of the European population.[11] In Māori and Pacific Island populations this difference is related to a shorter life expectancy, while in the Asian population it is due to migration patterns (Asian immigrants in NZ are generally young and recently immigrated).[11] Asian populations have been shown to have a higher incidence of ICH,[21,22] a difference attributed to higher blood pressure.

The ARCOS stroke group demonstrated in a population based study a crude ICH incidence in Māori, Pacific Island and Asian peoples similar to that of NZ Europeans.[21] Our hospital based data demonstrated a lower crude incidence rate of ICH in these ethnic groups. The difference between our data and the ARCOS data is unlikely to be the result of younger age in Māori, Pacific Island and Asian ethnic groups, who were younger in both study populations.

We feel it is most likely to be due to the confounding effect of health treatment and health seeking patterns, which probably vary by ethnicity. Cultural, financial and language barriers alter access to healthcare, and disparities in health between Māori and non-Māori in NZ are known to be partly attributable to a difference in healthcare access.[23] The reduced ICH incidence we observed in Māori, Pacific Island and Asian ethnic groups may be a consequence of reduced healthcare access.

Intraventricular extension of bleed on neuroimaging, warfarin use, age and lobar bleed location were found to correlate with 30-day mortality. Ventricular extension is associated with more severe brain injury, and are also carries risk of causing obstructive hydrocephalus. Previous analyses have documented an increase in risk of mortality associated with intraventricular blood,[24] and in this analysis it was a strong predictor of 30-day mortality.

An interesting finding of this study was the interaction between warfarin use and intraventricular extension of blood noted on neuroimaging. Warfarin use increases risk of death in ICH by increasing haematoma volume and duration of bleeding.[25] In our study the presence of intraventricular blood increased the likelihood of death within 30 days for all patients.

For warfarin users this effect was multiplied by a factor of three, meaning the presence of intraventicular blood increased the likelihood of death within 30 days for warfarin users by a factor of 24, in comparison to those not taking warfarin and without intraventricular extension of their bleed. For those without intraventricular extension of bleed, warfarin use did not confer an increased likelihood of death within 30 days.

The prolongation of bleeding time effected by warfarin use appears to have had an especially marked detrimental effect for patients whose bleed had extended into their ventricles. A previous analysis has shown intraventricular extension of bleed to be predictive of 30-day mortality in warfarin users,[26] but the relative risk in comparison to non warfarin users has not been previously described.

As patients age the risk of dying due to ICH is known to increase,[24] and our analysis conforms to this finding. As people age there is a trend for them to have more medical co-morbidity, and less physiological reserve to overcome acute illness.

Brainstem location of bleed is associated with higher mortality in ICH, followed by basal ganglia then lobar location.[24,27] There is less space for haematoma expansion below the tentorium, and infratentorial bleeds may quickly cause a pressure effect on crucial brainstem structures. However, in our cohort lobar location of bleed was independently associated with increased 30-day mortality.

The retrospective nature of our study means the data was subject to a number of possible confounding factors. We acknowledge that as we have relied on ICD coding at discharge there is a risk of case ascertainment bias. To further investigate this we ran an additional query on the hospital database for ‘cerebral infarction’, ‘ICH’ and ‘stroke, not specified as haemorrhage or infarction’ as diagnoses, considering the proportion of ‘unspecified’ strokes as a surrogate marker of coding accuracy.

Unfortunately the data from before 2003 was not accessible but from 2003 to 2010 there was no appreciable change in the proportion of stroke events for which neither infarction or bleed was coded as the diagnosis. This would suggest coding practice has not significantly changed over the study period, and so is unlikely to have influenced observed ICH incidence. Secondly, obtaining radiological information from the clinical radiology report may have resulted in inaccurate haematoma identification and localization, and may have introduced interobserver variability.

We also did not include recognized factors predictive of 30-day mortality in ICH – haematoma volume and Glasgow Coma Score (GCS) on presentation.[24] Our identified predictor variables may have been correlated with these factors, meaning their association with 30-day mortality may not have been truly independent. Collection of bleed volume and GCS data would have required review of radiology films and paper clinical notes for each patient which was beyond the resources of our study group. However, three of the four predictor variables we have identified have previously been described as predictive of 30-day mortality in ICH.[24,28]

Although warfarin use has been described previously as a predictor of 30-day mortality in ICH, we are not aware of a description of the observed interaction between warfarin use and intraventricular extension. We believe there is a plausible biological explanation for why this interaction might exist and that it is likely to be a true predictor of 30-day mortality in patients suffering ICH.

Summary

Observed ICH has increased in incidence over the past 10 years in the hospitals of the Waikato region of NZ. Increasing availability of neuroimaging, increasing age and increasing use of warfarin were identified as potential contributing factors. Radiological evidence of intraventricular extension of bleed, the use of warfarin, lobar location of bleed and increasing age correlated with poorer survival. This data will be available for comparison with future studies to assess trends in incidence, patient characteristics and outcome in ICH.

Competing interests: None.

Author information: James Irwin, Medical Registrar; Peter Wright, Neurologist, Department of Neurology; Paul Reeve, General Physician, Department of General Medicine; Waikato Hospital, Hamilton

Acknowledgements: We thank Stephen Holmes (Clinical Audit Support Coordinator, Waikato Hospital) for his assistance.

Correspondence: James Irwin, Medical Registrar, Department of General Medicine, Waikato Hospital, Private Bag 3200, Hamilton 3240, New Zealand. Email: jazirwin@gmail.com

References:

1. Van Asch CJ, Luitse MJ, Rinkel GJ, et al. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. Lancet Neurol 2010;9:167-76.
2. Sacco S, Marini C, Toni D, et al; Incidence and 10 year survival of intracerebral haemorrhage in a population based registry. Stroke 2009;40:394-9.
3. Qureshi AI, Tuhrim S, Broderick JP, et al. Spontaneous intracerebral haemorrhage. N Engl J Med 2001;344:1450-60.
4. Anderson CS, Chakera TM, Stewart-Wynne EG, et al; Spectrum of primary intracerebral haemorrhage in Perth, Western Australia 1989-90: incidence and outcome. J Neurol Neurosurg Psychiatry 1994;57(8):936-40.
5. Islam MS, Anderson CS, Hankey GJ, et al. Trends in incidence and outcome of stroke in Perth, Western Australia during 1989 to 2001: the Perth community stroke study. Stroke 2008;39:776-82.
6. Lovelock CE, Molyneux AJ, Rothwell PM. Change in incidence and aetiology of intracerebral haemorrhage in Oxfordshire, UK, between 1981 and 2006: a population based study. Lancet Neurol 2007;6:487-93.
7. Benatru I, Rouaud O, Durier J, et al. Stable stroke incidence rates but improved case fatality in Dijon, France, from 1985 to 2004. Stroke 2006;37:1674-9.
8. Sivenius J, Tuomilehto J, Immonen-Raiha P, et al. Continuous 15 year decrease in incidence and mortality of stroke in Finland: the FINSTROKE study. Stroke;2004:35:420-5.
9. World Health Organization http://apps.who.int/classifications/apps/icd/icd10online

10. R Development Core Team (2008). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.R-project.org
11. Statistics New Zealand http://www.stats.govt.nz
12. Anderson CS, Carter KN, Hackett ML, et al. Trends in Stroke Incidence in Auckland, New Zealand, During 1981 to 2003. Stroke 2005;36;2087-93.
13. Hart RG, Benavente O, McBride R, et al. Antithrombotic Therapy To Prevent Stroke in Patients with Atrial Fibrillation: A Meta-Analysis. Ann Intern Med 1999;131:492-501.
14. Stroke Prevention in Atrial Fibrillation Investigators. Stroke Prevention in Atrial Fibrillation Study; Final results. Circulation 1991;84:527-39.
15. Stroke Prevention in Atrial Fibrillation Investigators. Warfarin versus aspirin for prevention of thromboembolism in atrial fibrillation: Stroke Prevention in Atrial Fibrillation II Study. Lancet 1994;343:687-91.
16. European Atrial Fibrillation Trial Study Group. Secondary prevention in non-rheumatic atrial fibrillation after transient ischemic attack or minor stroke. Lancet 1993;342:1255-62.
17. Iso H, Jacobs DR, Wentworth D, et al. Serum cholesterol levels and six-year mortality from stroke in 350,977 men screened for the multiple risk factor intervention trial. N Engl J Med 1989;320:904-10.
18. SPARCL investigators. High Dose Atorvastatin after Stroke or Transient Ischaemic Attack. N Engl J Med 2006;355:549-59.
19. He J, Whelton PK, Vu B, et al. Aspirin and risk of hemorrhagic stroke: a meta-analysis of randomized controlled trials. JAMA 1998;280(22):1930-5.
20. Pharmac NZ. http://www.pharmac.govt.nz
21. Feigin V, Carter KM, Hackett ML, et al. Ethnic disparities in incidence of stroke subtypes: Auckland Regional Community Stroke Study, 2002-2003. Lancet Neurol 2006;5:130-9.
22. Jiang B, Wang WZ, Chen H et al. Incidence and trends of stroke and its subtypes in China: Results from three large cities. Stroke 2006;37:63-5.
23. Ellison-Loschmann L, Pearce N. Improving Access to Health Care Among New Zealand's Māori Population. Am J Public Health. 2006 April; 96(4):612–7.
24. Hemphill JC 3rd, Bonovich DC, Besmertis L, et al. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. Stroke 2001;32:891-7.
25. Flaherty ML, Tao H, Haverbusch M, et al. Warfarin use leads to larger intracerebral hematomas. Neurology 2008;71:1084-9.
26. Zubkov A, Claassen DO, Rabinstein AA. Warfarin associated intraventricular hemorrhage. Neurol Res. 2007 Oct;29(7):661-3.
27. Rosenow F, Hojer C, Meyer-Lohmann C, et al. Spontaneous Intracerebral Haemorrhage. Prognostic factors in 896 cases. Acta Neurol Scand 1997;96:174–82.
28. Rosand J, Eckman MH, Knudsen KA, et al; The effect of warfarin and intensity of anticoagulation on outcome of intracerebral hemorrhage. Arch Intern Med 2004;164(8):880-4.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Secondary prevention of vertebral fractures in a large New Zealand District Health Board

Katherine Bloomfield, Joe Singh

Abstract

Introduction International data suggests osteoporotic vertebral fractures are undertreated. The aim of this audit was to identify treatment gaps in patients with known vertebral fractures at Waitemata District Health Board (WDHB).

Methods Retrospective review of patients admitted to WDHB from July 2006 to June 2007. Inclusion criteria were age over 65 years, admission to any service with a primary or secondary diagnosis of vertebral fracture. Exclusion criteria were fractures related to malignancy. Demographic data, details of vertebral fracture, and history of prior fractures were documented. Osteoporotic medications at admission and discharge were collected.

Results We analysed 154 patients. The mean age was 81.5 years and 101 (66%) were women. At discharge, 42 (27%) of patients were on no treatment and 51 (33%) were treated with calcium, vitamin D and a bisphosphonate. Men were significantly more likely to be on no treatment ($p<0.05$). Lack of treatment did not appear to be associated with age or frailty. Subgroups studied included patients with prior non-vertebral fractures, primary diagnosis of vertebral fracture and patients on corticosteroids with rates of no treatment of 20%, 21% and 16% respectively.

Conclusion Secondary treatment of vertebral fractures in patients admitted to WDHB is suboptimal. Men were particularly affected.

Osteoporosis is a costly disease with the total health cost of osteoporosis in New Zealand estimated to be over $1.5 billion per anum.[1] It is very common with 1 in 3 women and 1 in 5 men over the age of 50 years thought to eventually experience an osteoporotic fracture.[2–4] A prior fragility fracture is one of the most robust predictors of future fragility fractures; the presence of a vertebral fracture predicts both further vertebral and non vertebral fractures with up to 19% sustaining a further vertebral fracture within 1 year.[5]

In women, vertebral fractures are strong predictors of incident hip fractures, with the risk increasing with the number of vertebral deformities.[6] In older patients, functional impairment with vertebral fractures can be similar to that following a hip fracture, with significant pain and/or impairment in activities of daily living.[7–10] Several previous studies suggest vertebral fractures are associated with increased mortality similar to that seen following neck of femur fractures.[11–13]

International data has shown osteoporotic fractures are both underdiagnosed and undertreated.[14–17] Approximately only 30% of vertebral fractures come to clinical attention and only 2–10% require hospitalisation.[18] Over recent years work has been focused on appropriate secondary prevention of neck of femur (NOF) fractures, and

the introduction of orthogeriatric services across many New Zealand (NZ) District Health Boards (DHBs) has improved the management of NOF fractures.[19] We are aware however, that in their current form, many orthogeriatric services may not interact directly with patients with other fragility fractures such as vertebral or Colles' fractures.

Secondary prevention of patients with osteoporosis has level one evidence of support. The aim of this study was to investigate whether a treatment gap between best practice and actual clinical practice existed in our DHB.

Methods

We performed a retrospective chart review of patients admitted to Waitamata District Health Board, Auckland, NZ from July 2006 to June 2007. Electronic discharge summaries were reviewed and patients with evidence of vertebral fracture as either a primary or secondary discharge diagnosis were identified. Patients over the age of 65 and admitted to any service across the DHB were reviewed. Patients were excluded if fracture was secondary to malignancy. Demographic data, details of vertebral fracture, history of prior fractures, and medications (calcium and vitamin D supplementation, other osteoporotic medicines and steroids) at time of admission and discharge were collected. Discharge medications are reported in this article. Note was also made of any readmissions during the audit period for evidence of any further fractures or medication changes to osteoporosis regime.

Results

Demographic information—In total 163 patients with a primary or secondary diagnosis of vertebral fracture were identified. Nine patients were excluded due to bone metastases or multiple myeloma, leaving 154 patients. The mean age was 81.5 years with 101 (65.6%) women (see Table 1 and Figure 1 for further details).

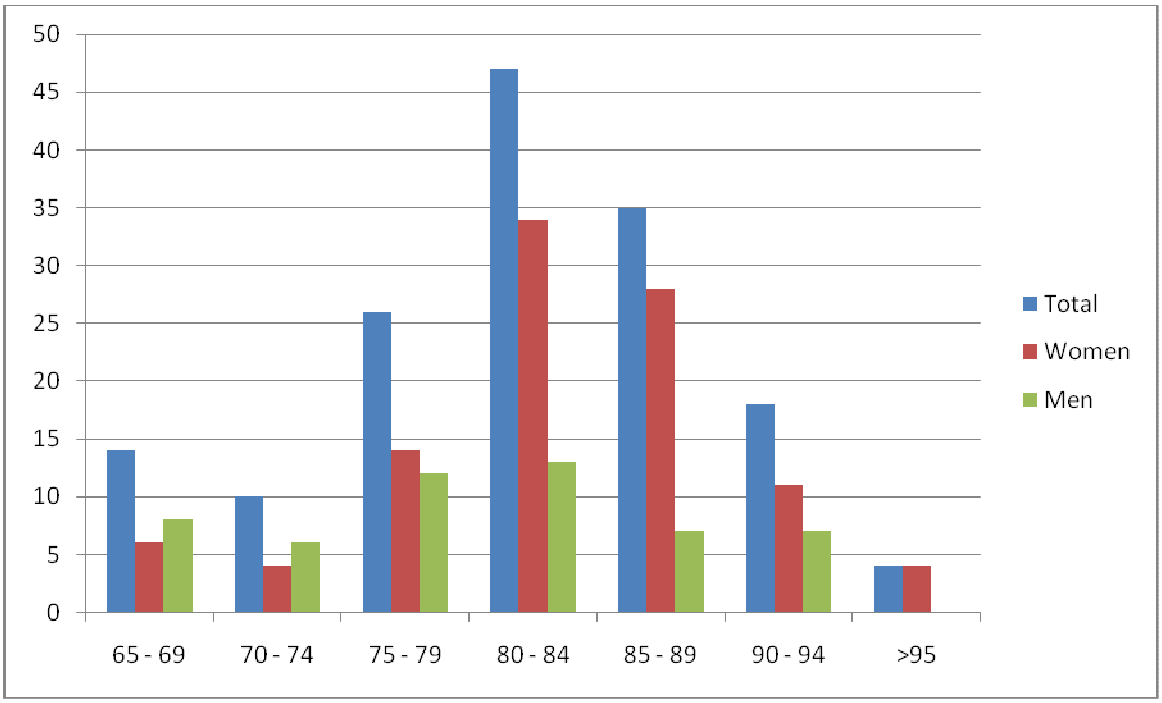
Table 1. Patient and fracture characteristics

Total population		**154**
Women	Total Mean age Range	101 (65.6%) 83.2 years 66 – 97 years
Men	Total Mean age Range	53 (34.4%) 79.8 years 67 – 93 years
Service of admission	General medicine Older people's health General surgery Orthopaedics Emergency medicine	117 (76%) 19 (12%) 2 (1%) 10 (6%) 6 (4%)
Additional non-vertebral fracture	Total Neck of femur Pelvic Other Lower limb Colles' Other upper limb Chest wall	54 patients(35%) 24 fractures 14 fractures 6 fractures 4 fractures 10 fractures 15 fractures
More than one vertebral fracture		101 (66%)
New/primary diagnosis vertebral fracture		28 (18%)
Corticosteroids		31 (20%)
Readmission with subsequent fracture		8 (5%)

Patients were predominantly admitted through the General Medical service (76%), with 12% admitted through Older Peoples health services and the remaining 12% through the Emergency Department, orthopaedics or general surgery.

As shown in Table 1, two-thirds of patients had more than one documented vertebral fracture and over one-third had a history of additional non-vertebral fractures. Ninety-one percent of vertebral fractures were either thoracic or lumbar, 6% cervical and with 4% not documented in hospital records.

Figure 1. Age distribution



Treatment rates in the overall group—One-third of patients (51) were documented as being treated with a combination of calcium, vitamin D and some form of bisphosphonate (Table 2).

Table 2. Treatment rates in overall patients and subgroups

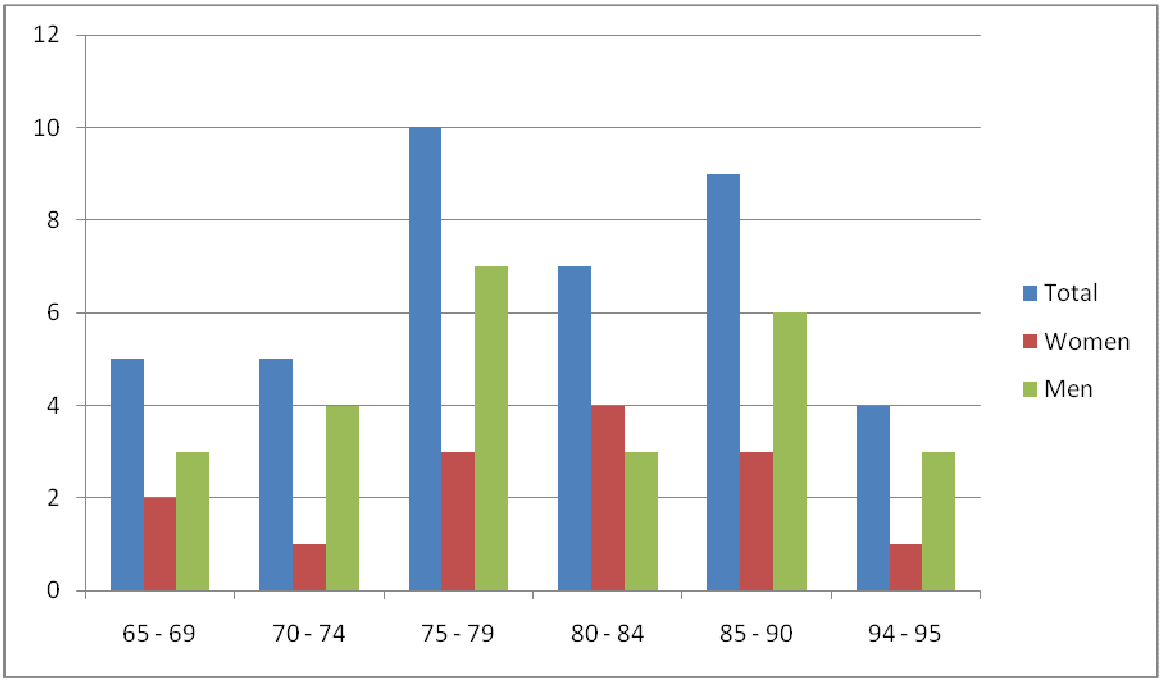
Variables	Ca/VitD/BP combination	none	BP	Ca	Vit D
Total group	51 (33%)	42 (27%)	73 (47%)	93 (60%)	83 (54%)
Additional non vertebral fracture	25 (46%)	11(20%)	31 (57%)	37 (69%)	34 (63%)
New/primary diagnosis	9 (32%)	6 (21%)	15 (54%)	19 (68%)	15 (54%)
Corticosteroids	16 (52%)	5 (16%)	19 (61%)	25 (81%)	23 (74%)

Ca=calcium supplementation, VitD=Vitamin D supplementation, BP=bisphosphonate.

Seventy-three patients (47%) were treated with a bisphosphonate alone. In comparison 42 patients (27%) were on no treatment for osteoporosis (vitamin/mineral supplementation or antiresorptive treatment). No patients were prescribed other forms of osteoporotic treatment, such as hormone replacement therapy.

There was a statistically insignificant trend that patients treated with all three modalities were more likely to be women. Conversely there was a significant likelihood that patients receiving no medications for osteoporosis were more likely to be male ($p<0.05$). The age range for the 42 patients not on any treatment for osteoporosis is similar to the spread in the total population (Figure 2).

Figure 2. Age distribution in patients on no treatment



Three out of the 42 patients on no treatment were discharged to a private hospital setting with the remaining discharged home or to rest home level of care. Thirty two of these 42 patients (76%) were seen through the general medical service with the remaining patients admitted and discharged through orthopaedics (7 patients), general surgery (2 patients) and the emergency department (1 patient).

When looked at as a percentage of patients admitted through each service with a vertebral fracture, this represents 27% general medical admissions, 70% of orthopaedic admissions, 100% of general surgical admissions and 17% of emergency department admissions with vertebral fracture.

Treatment rates in subgroups—Previous non-vertebral fractures: Over one third of patients (54, 35%) had documentation of a total of 73 prior non-vertebral fracture in addition to documented vertebral fracture (Table 1). These were predominantly neck of femur, pelvic and chest wall fractures. Nine patients had documentation of more

than two prior non vertebral fractures. In this subgroup of 54 patients 46% were on calcium, vitamin D and bisphosphonate treatment, and 20% were receiving none of these medications. Over half of the patients (57%) were receiving bisphosphonate treatment (Table 2).

New diagnosis of vertebral fracture: 28 (18%) patients were admitted with a new diagnosis of vertebral fracture, with 23 (15%) of these being the primary reason for admission to hospital, and the remaining 5 being older fractures identified on chest X-ray films. These patients were again predominantly admitted through the general medical service (22/28). At time of discharge 6 of these patients (21%) were not on any form of treatment, where as 9 (32%) were on full combination treatment, and 15 (54%) were documented to be receiving bisphosphonates (Table 2).

Treatment with corticosteroids**:** 31 patients (20%) were documented as being treated with corticosteroids. The main indication for steroid treatment was chronic obstructive pulmonary disease (21 patients) and the age range in this group was similar to the overall group with a range of 66 to 94 years of age. In this subgroup of 31 patients 5 patients (16%) were on no treatment, with 16 patients (52%) on full combination treatment and 19 (62%) receiving bisphosphonates (Table 2).

Discussion

To our knowledge this is the first published audit on the secondary prevention of vertebral fractures in a large general hospital in NZ. The recommended practice in NZ at the time of this audit for patients with known vertebral fractures included adequate calcium intake of 1–1.5 g per day, adequate vitamin D supplementation of 800–1000 IU per day and use of antiresorptive medications, of which bisphosphonates are first line treatment.[20]

Patients in this audit were, as expected, predominantly women and of an old age, however the younger-old were still well represented in numbers. Overall the group had a high fracture burden as evidenced by multiple vertebral and non vertebral fractures. Results clearly show inadequate rates of treatment for this important condition with 53% of patients not prescribed bisphosphonate treatment. Unfortunately there was no documentation of why patients were not receiving bisphosphonates, such as contraindications or adverse effects in any of these patients, or of patients referred to endocrine or geriatrician lead osteoporosis clinics which are available at this DHB.

It is possible that some patients receive adequate calcium and vitamin D from dietary and other sources without requiring supplementation, however given the age range of this population this is likely to be the case in only a very small number of patients, if any. In which case adequate documentation of calcium intake or vitamin D levels should have been recorded by physicians in order to highlight these issues had been addressed and considered. This was not the case in any patients. It is also possible that treatment with calcium or vitamin D supplementation may not have been included in discharge papers despite the patient actually taking them.

This study was conducted before the safety of calcium supplementation was questioned. Given the intermittent nature of treatment with bisphosphonates, it is also possible that this was occasionally not properly documented, which may mean

treatment rates are underestimated in this study. However we believe a treatment gap still exists for vertebral fracture secondary prevention.

Although rates of treatment were improved in the subgroups studied compared to the overall group, particularly in patients prescribed corticosteroids, we are still failing many of these patients. The rates of secondary prevention in our study are somewhat better than similar reports from Australia. Teede et al published results from a multisite study of minimal trauma fractures presenting to emergency departments in 2003 to 2005 which also demonstrated poor use of calcium, vitamin D and bisphosphonates.[21]

Interestingly male patients were significantly more likely to receive no secondary prevention than female patients There is likely a perception that osteoporosis is a disease of old women, however as clearly has been demonstrated both younger and male patients are afflicted with this disease. This is particularly concerning, especially when considering the increased mortality rates seen in men compared to women after hip fractures (one year mortality rates of up to 38% in men compared to up to 28% in women)[11-13]. This is an issue that we believe needs further education.

We also looked at possible influences on lack of treatment such as age, frailty and service of admission. All medical treatments should be individualised, with medicines prescribed when a positive risk/benefit ratio exists. Therefore in some cases very old patients or those with a perceived short survival span would not necessarily benefit from treatment. Given the spread of age across this subgroup of patients however, we do not believe age influenced prescribing in these cases. This is in contrast to a previous study were older patients were less likely to receive adequate treatment[22]. We used discharge destination as a surrogate marker of frailty in this audit. Again, we do not believe perceived frailty was a significant influence.

Although most patients were seen through the general medical services, a patient seen through one of the surgical services was less likely to receive appropriate medical treatment. Surgical colleagues may feel uncomfortable prescribing these medications and should be given appropriate referral points of contact to address this issue. Changes to the way orthogeriatrics is delivered at WDHB may help capture the small number of patients that are admitted under orthopaedics with vertebral fractures. Unfortunately however, the greatest patient numbers were seen through the general medical service.

This service at WDHB is a busy acute service and it is likely that most cases of secondary prevention were neglected due to significant service demands. We remain disappointed however, as we believe that secondary prevention of osteoporosis is as important as secondary prevention of other diseases and we suspect patients with ischaemic heart disease have greater rates of appropriate treatment in the same DHB. In other words greater awareness and education is necessary to improve secondary prevention of osteoporosis across the DHB.

Great strides have been made over recent years in improving the rates of osteoporosis treatment post hip fractures with the instigation of orthogeriatric services across many District Health Boards in NZ. Local unpublished audits in the Auckland region have demonstrated improved rates of secondary prevention in neck of femur fractures over

recent years. However a large group of patients with other fragility fractures may clearly be missed by these services depending on how they are structured.

Given the cost of osteoporosis both financially to the health service and in terms of morbidity and mortality, we need to educate medical staff about the importance of all osteoporotic fractures and the readily available secondary prevention measures that do actually make a difference.

In addition, DHBs need to consider other avenues to capture this large group of patients. At Waitemata DHB, the position of a multiservice osteoporosis/fracture liaison nurse may help to improve the gap between best practice and actual clinical practice at our DHB.

Competing interests: None.

Author information: Katherine Bloomfield, Freemasons' Senior Lecturer and Geriatrician, Department of Geriatric Medicine, University of Auckland and Waitemata District Health Board, Auckland; Joe Singh, Geriatrician, Department of Geriatric Medicine, Waitemata District Health Board, Auckland

Correspondence: Katherine Bloomfield, Department of Geriatric Medicine, University of Auckland and Waitemata District Health Board, 120 Shakespeare Road, PO Box 93 503 Takapuna, North Shore 0740, Auckland, New Zealand. Fax: +64 (0)9 4427166; email: Katherine.bloomfield@waitematadhb.govt.nz

References:

1. Brown P, McNeil R, Radwan E, Willingale J. Osteoporosis New Zealand report: The burden of Osteoporosis in New Zealand: 2007-2020. October 2007. Available at www.bones.org.nz
2. Melton LJ, Atkinson EJ, O'Conner MK, et al. Bone density and fracture risk in men. J Bone Miner Res 1998;13(12)1915-23.
3. Melton LJ, Chrischilles EA, Cooper C, et al. How many women have osteoporosis. J Bone Miner Res 2005;20(5):886-92.
4. Kanis JA, Johnell O, Oden A, et al. Longterm risk of osteoporosis in Malmo. Osteopor Int 2000;11(8):669-74.
5. Lindsay R, Silverman SL, Cooper C, et al. Risk of new vertebral fractures in the year following a fracture. JAMA 2001;28593(3):320-3.
6. Black DM, Arden NK, Palermo L, et al. Prevalent vertebral deformities predict hip fracture and new vertebral deformity but not wrist fracture. Study of Osteoporotic Fractures Research Group. J Bone Miner Res 1999;14(5):821-8.
7. Greendale GA, DeAmicis TA, Bucur A, et al. A prospective study of the effect of fracture on measured physical performance: results from the MacArthur study. J Am Geriatr Soc 2000;48(5):546-9.
8. Papaioannou A, Watts NB, Kendler DL, et al. Diagnosis and management of vertebral fractures in elderly adults. Am J Med 2002; 113(3):220-8.
9. Huang C, Ross PD, Wasnich RD. Vertebral fractures and other predictors of physical impairment of health care utilisation. Arch Int Med 1996;156(21):2469-75.
10. Rapado A. General management of vertebral fractures. Bone 1996;18(3 Supple):191S-196S.
11. Bouza C, Lopez T, Palma M, Amate JM. Hospitalised osteoporotic vertebral fractures in Spain: analysis of the national hospital discharge registry. Osteoporos Int 2007;18(5):649-657.
12. Ismail AA, O'Neill TW, Cooper C, et al. Mortality associated with vertebral deformities in men and women: results from the European Prospective Osteoporosis Study (EPOS). Osteoporos Int 1998;8(3):291-7.

13. Kanis JA, Oden A, Johnell O, et al. Excess mortality after hospitalisation for vertebral fractures. Osteoporos Int 2004;15(2):108-12.
14. Solomon DH, Finkelstein JS, Katz JN, et al. Underuse of osteoporosis medications in elderly patients with fractures. Am J Med 2003;115(5):398-400.
15. Solomon DH, Brookhart MA, Gandhi TK, et al. Adherence with osteoporosis practice guidelines: a multilevel analysis of patient, physician and practice setting characteristics. Am J Med 2004;117(12):919-24.
16. Neuner JM, Zimmer JK, Hamel MB. Diagnosis and treatment of osteoporosis in patients with vertebral compression fractures. J Am Geriatr Soc 2003;51(4):483-91.
17. Finnern HW, Sykes DP. The hospital cost of vertebral fractures in the EU: estimates using national datasets. Osteoporos Int 2003;14(5):429-36
18. Cooper C, Atkinson EJ, O'Fallon WM, Melton LJ. Incidence of clinically diagnosed vertebral fractures: a population-based study in Rochester, Minnesota 1985-1989. Bone Miner Res 1992;7(2):221-7.
19. Sidwell AI, Wilkinson TJ, Hanger HC. Secondary prevention of fractures in older people: evaluation of a protocol for the investigation and treatment of osteopososis. Int Med J 2004;34(3):129
20. Osteoporosis New Zealand. www.bones.org.nz
21. Teede HJ, Jayasuriya IA, Gilfillan CP. Fracture prevention strategies in patients presenting to Australian hospitals with minimal trauma fractures: a major treatment gap. Int Med J 2007;37(10):674.
22. Andrade SE, Majumdar SR, Chan A, et al. Low frequency of treatment of osteoporosis among post-menopausal women following a fracture. Arch Int Med 2003;163:2052-57.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Emergency peripartum hysterectomy: a 10-year review in a tertiary obstetric hospital

Tze Yoong Wong

Abstract

Aim To evaluate the incidence, indications and complications associated with emergency peripartum hysterectomy (EPH) performed at Christchurch Women's Hospital, New Zealand.

Methods A retrospective case series analysis of EPH from 2000–2009. Cases were identified using the hospital's computerised database. Those medical records were reviewed. EPH was defined as one performed for major postpartum haemorrhage unresponsive to other treatment within 24 hours of delivery.

Results Nineteen EPH cases were identified among 47,520 deliveries, giving an incidence of 0.4 per 1000 deliveries. The indications were invasive placental adhesion—accreta, increta, percreta (63%), uterine atony (16%), placenta praevia (10.5%) and uterine tear with atony (10.5%). All cases of abnormal placentation in this study had previous caesareans or curettages. A significant association between previous uterine surgery and abnormal placentation was shown (p=0.02), especially those with previous caesarean (p=0.003). No maternal or perinatal mortality was recorded. Maternal morbidity was prevalent, including eight disseminated intravascular coagulopathies, seven intensive care, three bladder injuries, two re-explorations, one respiratory failure and one pulmonary embolism.

Conclusion Invasive placental adhesion is the major indication for EPH. This study demonstrates an association between the presence of scarred uteri as a result of previous uterine surgery, and abnormal placentation.

Postpartum haemorrhage (PPH) is one of the major causes of maternal morbidity and mortality. It encompasses both primary and secondary forms. A widely used definition for primary PPH is blood loss of 500 ml or more from the genital tract within 24 hours after birth,[1] as proposed by the World Health Organization (WHO) in 1990. Secondary PPH occurs between 24 hours and 12 weeks postnatally.[2,3] Major PPH is classified as bleeding of 1000 ml or more.[3]

The WHO estimates that PPH accounts for nearly one-quarter of all maternal deaths worldwide.[4,5] In New Zealand, three maternal deaths related to PPH were reported during the 2006–2008 triennium, in the Fourth Report of the Perinatal and Maternal Mortality Review Committee.[6] The report also found that alongside pre-eclampsia, PPH was the second highest direct cause of maternal death after amniotic fluid embolism.[6] Whilst in the 2006–2008 report of the UK Confidential Enquiries into Maternal Deaths, PPH claimed five maternal lives.[7]

Caesarean delivery has a long history but was often associated with extremely poor outcomes. In 1876, Italian obstetrician Eduardo Porro was the first to describe a

successful caesarean hysterectomy to reduce haemorrhage.[8] In modern obstetric practice, emergency peripartum hysterectomy (EPH) is a life-saving procedure to control massive haemorrhage when medical treatment and conservative surgery have failed. It includes both caesarean hysterectomy and hysterectomy following vaginal birth. The incidence in developed countries ranges from 0.2 to 5 per 1000 deliveries.[9] Despite its life-saving capacity, EPH is, however, associated with high morbidity and mortality, and negatively affects women's future fertility. Consequently, these crucial disadvantages need to be reflected upon when considering treatment.

The purpose of this study was to evaluate the incidence, indications and complications associated with EPH performed in a tertiary obstetric centre in a developed country.

Methods

This study was a retrospective case series analysis of EPH performed at Christchurch Women's Hospital, New Zealand, in a 10-year period between 1 January 2000 and 31 December 2009.

All delivery data for the study period was downloaded from the hospital's computerised database into Microsoft® Access® software. The data was further limited by the presence of The International Statistical Classification of Disease and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM) procedural code of 35653 (abdominal hysterectomy) within the same admission as the birth.

The inclusion criteria were an EPH performed for major postpartum haemorrhage unresponsive to other treatment within 24 hours of vaginal or caesarean delivery, and a pregnancy of at least 24 weeks' gestation. This study also included cases of planned caesarean hysterectomy that subsequently required emergency operation due to antepartum haemorrhage or early labour.

The exclusion criterion was elective caesarean hysterectomy performed for antenatal diagnosis of abnormal placentation or gynaecological conditions such as malignancy.

All medical records of identified cases were reviewed. A standard proforma was used to collect data, which consisted of clinical variables such as basic demographics, parity, gestational details, previous uterine surgery, mode of delivery, type of hysterectomy, indication and outcomes. The information was then collated and analysed.

Statistical analysis was performed using the Open Source Epidemiologic Statistics for Public Health (OpenEpi) v2.3.1 software. Dichotomous variables were analysed using the Fisher's exact test (two-tail), with odds ratios (OR), 95% confidence intervals (CI) and p-values calculated for statistical significance. A p-value of less than 0.05 was considered statistically significant.

Results

During the 10-year study period, there were a total of 47,520 deliveries at Christchurch Women's Hospital including 33,570 (71%) vaginal and 13,950 (29%) caesarean deliveries. Nineteen cases of EPH were identified, which gave an incidence rate of 0.4 per 1000 deliveries. Of the 19 EPHs, 18 women (95%) had caesarean hysterectomy (1.3 hysterectomies per 1000 caesarean sections) and 1 woman (5%) had hysterectomy following vaginal delivery (0.03 hysterectomies per 1000 vaginal deliveries).

The median maternal age was 35.5 years (range 25 to 44 years). The median gestational age at birth was 37 weeks (range 28 to 41 weeks).

The operative notes and the histology reports of the uterus and placenta were used to determine the final reason for the hysterectomy. The indications were invasive placental adhesion—accreta, increta, percreta (63%), uterine atony (16%), placenta praevia (10.5%) and uterine tear and atony (10.5%) (Table 1).

Abnormal placentation, including invasive placental adhesion and placenta praevia, were therefore present in more than 70% of the cases. Of the 12 invasive placental adhesions, four (33%) were placenta accreta, five (42%) were increta and three (25%) were percreta.

EPH was performed in two primiparous (10.5%) and 17 multiparous (89.5%) women. The most common indication for EPH in multiparas was morbidly adherent placenta, while the sole reason for primiparas was uterine atony (Table 1).

Table 1. Indications for emergency peripartum hysterectomy and comparison between primiparas and multiparas

Indications	n (%)	Primipara	Multipara
Invasive placental adhesion (accreta, increta, percreta)	12 (63%)	0	12 (70%)
With praevia	10		
Without praevia	2		
Uterine atony	3 (16%)	2 (100%)	1 (6%)
Placenta praevia only	2 (10.5%)	0	2 (12%)
Uterine tear and atony	2 (10.5%)	0	2 (12%)
Total	**19 (100%)**	**2 (100%)**	**17 (100%)**

Sixteen women (84%) in the study group had a previous history of uterine surgery, either caesarean or curettage, and in particular, 15 of those (94%) had at least one caesarean section. Fourteen of the 16 cases (87.5%) that underwent previous uterine surgery were associated with abnormal placentation. There was a significant association between previous uterine surgery and abnormal placentation (p=0.02), especially those with previous caesarean (p=0.003).

The results of this study showed that multiparity, abnormal placentation and previous uterine surgery, particularly prior caesarean, were associated with EPH.

There was no reported maternal or perinatal mortality; however, maternal morbidity was not uncommon. Eight women had disseminated intravascular coagulopathy (42%), seven required intensive care (37%), three had bladder injury (16%), two returned to theatre for further operation (10.5%), one had respiratory failure requiring ventilation (5%) and one had pulmonary embolism (5%). Blood transfusion was necessary in all women. Other associated complications included eight febrile morbidities (42%), five wound infections (26%), four postoperative ileus (21%) and one pneumonia (5%).

Ten total hysterectomies and nine subtotal hysterectomies were performed. More maternal complications were observed in the total hysterectomy group. Although the differences in outcomes between women undergoing the two different types of hysterectomy were not statistically significant (Table 2), there was a non-significant trend for those women with a total hysterectomy to be more likely to develop disseminated intravascular coagulopathy.

Table 2. Comparison of outcomes between total and subtotal hysterectomies

	Total	Subtotal	OR	95% CI	P value
No. of cases	**10**	**9**			
Disseminated intravascular coagulopathy	6 (60%)	2 (22%)	5.25	0.70–39.47	0.23
Intensive care	4 (40%)	3 (33%)	1.33	0.20–8.71	>0.99
Bladder injury	2 (20%)	1 (11%)	2.00	0.15–26.73	>0.99
Re-exploration	1 (10%)	1 (11%)	0.89	0.05–16.66	>0.99

Discussion

The incidence of EPH in this study was 0.4 per 1000 deliveries and fell within the range reported by a recent systematic review, Rossi et al 2010, which is 0.2 to 5 per 1000 deliveries.[9] The rate shown in this study is also comparable with those reported by other tertiary obstetric centres in developed countries, such as 0.56 in Baskett 2003 (Canada) and 0.36 in Smith et al 2007 (United Kingdom).[10,11]

Compared with vaginal delivery, the incidence of EPH was approximately 40-fold higher in caesarean delivery. Previous studies have shown a 9.5 to 20-fold increase in incidence among women who delivered by caesarean section.[9] This difference could be due to the presence of routine second trimester anomaly scan in New Zealand that concurrently assesses the placental site. This enables early detection and subsequent follow up of abnormal placental implantation, thus preventing the possibility of undiagnosed cases having vaginal birth.

Early studies found that uterine atony and uterine rupture were the more common causative factors of EPH than placental disorders.[12,13] Recent articles have described invasive placental adhesion as the leading indication for EPH,[9–11,14,15] which is also supported by the findings of this study. This is most likely a consequence of recent year increases in the number of caesarean sections and uterine curettages, and improved treatment of uterine atony with prostaglandins.[9,14] In addition, the introduction of newer conservative surgical haemostatic measures for control of atonic PPH, for instance, intrauterine balloon tamponade and B-Lynch brace suture, may have played a role. In 2006, B-Lynch asserted that over 1300 of his sutures had been performed worldwide with success in all but seven cases.[16] This highlights its efficacy to prevent complications and sequelae of PPH.

The risk of morbidly adherent placentation increases proportionally with the number of caesarean deliveries or curettages.[14] All cases of abnormal placentation in this study had a history of uterine surgery. This demonstrates a clear association between the presence of scarred uteri as a result of previous uterine surgery, and abnormal placentation in subsequent pregnancies. It is foreseeable that this will continue to be an issue due to the increasing rate of caesarean delivery worldwide. To that end, caesarean section should only be performed for valid clinical reasons.

Similar to other series, the results of this study show that EPH is associated with considerable maternal morbidity.[9–11,14,15] The rate of urological complication following EPH of 16% is comparable with the 18% reported by Habek et al 2007 but higher than the 6% noted by Smith et al 2007.[11,15]

The prevalence of urological injury may be due to the distortion of the lower uterine segment and pelvic anatomy caused by the invasive placental adhesion and praevia.[9] Disseminated intravascular coagulopathy and infections were common in this review. This observation stresses the importance of prompt availability of blood products and early haematology involvement. Surgeons should also consider the use of postoperative prophylactic antibiotics to reduce the risk of infection.

Recent literature suggests that subtotal hysterectomy is preferred in emergency peripartum situations.[11,14,17] Instability of the maternal condition means that it is expedient to adopt a less complex and faster operation. Subtotal hysterectomy is generally safer and provides a lower risk of urinary tract injury.[10,11]

In contrast, total hysterectomy requires further distal dissection of the vascular plexus, bladder base and pelvic floor,[16] where visceral injuries, increased blood loss and longer operating time are more likely to occur. Nevertheless, it is important to emphasise that the choice should depend on the condition, timing and surgical accessibility of the hysterectomy.[16]

Subtotal hysterectomy is more appropriate in situations of uterine atony, bleeding in the uterine body, and non-fully dilated cervix where it can be easily identified.[9,18] Total hysterectomy is, however, preferred in cases of bleeding from the lower segment secondary to abnormal placentation.[10]

The management of uncontrolled PPH represents a challenge for many obstetricians as it may lead to an EPH, which is a stressful procedure associated with high maternal morbidity and the loss of future fertility in women of reproductive age. Every obstetric unit should therefore have a guideline or algorithm for the management of PPH.

Obstetricians should be familiar with the different surgical techniques that allow for preservation of the uterus, with hysterectomy being the last resort. Notwithstanding that, women with haemodynamic instability and life-threatening haemorrhage should proceed to early hysterectomy to avoid the risk of increased morbidity and blood loss, instead of incurring delays by attempting various conservative surgical methods to control massive PPH. In cases where an EPH is inevitable, the skilled assistance of an experienced gynaecologist colleague should be considered at the earliest opportunity to enhance the outcome.[16]

Conclusion

Although EPH is a life-saving procedure, it is associated with maternal morbidity and loss of future fertility. This review documents invasive placental adhesion as the major indication for EPH. Additionally, previous uterine surgery resulting in scarred uteri with subsequent abnormal placentation is a significant association with EPH.

Competing interests: None.

Author information: Tze Yoong Wong, Registrar, Department of Obstetrics and Gynaecology, Middlemore Hospital, Auckland.

Acknowledgements: The author acknowledges the assistance of Philip Lalor (Information Analyst, Christchurch Women's Hospital) and Irene Zeng (Biostatistician, Centre for Clinical Research and Effective Practice, Middlemore Hospital).

Correspondence: Tze Yoong Wong. Email: tzeywong@gmail.com

References:

1. World Health Organization. The Prevention and Management of Postpartum Haemorrhage. Report of a Technical Working Group. WHO/MCH/90.7. Geneva: WHO, 1990.
2. World Health Organization. WHO guidelines for the management of postpartum haemorrhage and retained placenta. Geneva WHO 2009.
3. Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum haemorrhage. Green-top guideline No.52. London: RCOG, 2009.
4. World Health Organization. Maternal mortality fact sheet. WHO/MPS/08.12. Geneva: WHO, 2008.
5. World Health Organization. WHO recommendations for the prevention of postpartum haemorrhage. WHO/MPS/07.06. Geneva WHO 2007.
6. PMMRC 2010. Perinatal and maternal mortality in New Zealand 2008: Fourth report to the Minister of Health July 2009 to June 2010. Wellington: Ministry of Health, 2010.
7. Centre for Maternal and Child Enquiries (CMACE). Saving Mothers' Lives: reviewing maternal deaths to make motherhood safer: 2006–08. The Eighth Report on Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG 2011;118(Suppl. 1):1–203.
8. Todman DH. Eduardo Porro (1842-1902) and the development of caesarean section: a reappraisal. The Internet Journal of Gynecology and Obstetrics. 2007;7(2).
9. Rossi AC, Lee RH, Chmait RH. Emergency postpartum hysterectomy for uncontrolled postpartum bleeding: a systematic review. Obstet Gynecol. 2010;115:637-44.
10. Baskett TF. Emergency obstetric hysterectomy. J Obstet Gynaecol. 2003;23:353-5.
11. Smith J, Mousa HA. Peripartum hysterectomy for primary postpartum haemorrhage: incidence and maternal morbidity. J Obstet Gynaecol. 2007;27:44-7.
12. Clark SL, Yeh SY, Phelan JP, et al. Emergency hysterectomy for obstetric hemorrhage. Obstet Gynecol. 1984;64:376-80.
13. Chestnut DH, Eden RD, Gall SA, Parker RT. Peripartum hysterectomy: a review of cesarean and postpartum hysterectomy. Obstet Gynecol. 1985;65:365-70.
14. Kastner ES, Figueroa R, Garry D, Maulik D. Emergency peripartum hysterectomy: experience at a community teaching hospital. Obstet Gynecol. 2002;99:971-5.
15. Habek D, Becareviç R. Emergency peripartum hysterectomy in a tertiary obstetric center: 8-year evaluation. Fetal Diagn Ther. 2007;22:139-42.
16. B-Lynch C, Whitelaw N. The surgical management of post partum haemorrhage. Fetal and Maternal Medicine Review. 2006;17:105-23.
17. Roopnarinesingh R, Fay L, McKenna P. A 27-year review of obstetric hysterectomy. J Obstet Gynaecol. 2003;23:252-4.
18. Bruinse HW, Metz GCH, Kwee A. Surgical treatment of postpartum haemorrhage. International Congress Series. 2005;1279:369-75.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Outcomes of patients with untreated severe aortic stenosis in real-world practice

Suresh Perera, Namal Wijesinghe, Elene Ly, Gerard Devlin, Sanjeevan Pasupati

Abstract

Background Surgical aortic valve replacement remains the gold standard of the treatment of severe symptomatic aortic stenosis but is often not considered due to excessive risk factors and comorbidities especially in elderly patients. We describe the burden of untreated severe aortic stenosis at a tertiary care hospital in New Zealand.

Method Consecutive patients with severe aortic stenosis presented between January-December, 2005 were studied retrospectively. Outcome assessment included mortality, hospital stay and on going symptoms (angina >CCS class II, dyspnoea >NYHA class II and syncope).

Results A total of 105 patients with severe aortic stenosis were identified (mean age 76±13 years, 51% men). Patients were divided into 3 groups according to the management strategy. (Group 1: Not referred for surgery as asymptomatic (n=25), Group 2: Declined for surgery (n=41), Group 3: Accepted for surgery (n=39)). Median follow-up was 34 months (interquartile range: 16–36 months). All-cause mortality in Group 1, Group 2 and Group 3 were 36%, 73% and 18% respectively while hospital days per 100 patient-years were 3.5, 10.1 and 6.4 and symptoms on last follow-up were 0%, 64% and 0% respectively. Almost half of symptomatic patients (Group 2 versus 3) were denied valve surgery due to comorbidities. Symptomatic patients had a significant mortality ($p<0.0001$) benefit with less hospitalisations ($p<0.0001$) post surgery.

Conclusions Untreated symptomatic severe aortic stenosis is associated with a poor prognosis and significant morbidity. For symptomatic patients with severe aortic stenosis who are denied surgery, alternative therapies such as transcatheter aortic valve implantation could be a viable option.

Aortic stenosis accounts for the vast majority of aortic valve disease, with a prevalence of approximately 1–2% among people over 65 years and 4% among people over 85 years.[1] The aetiology is mainly calcific stenosis due to senile degenerative valve disease. Management of aortic stenosis has been tailored based on the severity of the condition, presence of comorbidities, age of the patient and their operative risk. Usually, treatment is only needed when aortic stenosis is severe and patients are symptomatic.

Aortic stenosis is the commonest acquired valvular lesion with a prevalence of 1–2% in the over 65s.[1] Calcific aortic stenosis, the commonest cause of aortic stenosis, shares the predisposing factors of coronary artery disease: age, male sex and hypercholesterolaemia.[2]

The onset of symptoms in patients with aortic stenosis is a poor prognostic indicator without valve replacement. More than half the patient will die within the next 12–18 months of symptom onset, unless the aortic valve is replaced reliving the afterload on the ventricle.[2] In addition, quality of life is adversely affected with frequent hospital admissions during the remaining years of lives.

The management of the asymptomatic patient with severe aortic stenosis is less straightforward. Asymptomatic patients have an almost normal life expectancy without valve replacement with <1% incidence of sudden cardiac death[3] precluding the need for aortic valve surgery. In patients in whom surgery is considered necessary, prompt aortic valve replacement (AVR) can return age-corrected prognosis to that of a normal population.[4,5]

Procedural mortality for isolated aortic valve replacement in the surgically accepted group is low (<4%) and long-term results are excellent. Worldwide, however, over 30% of patients with severe symptomatic AS are not accepted for surgery due to comorbidities or are not indeed referred for surgery.[6–11]

The emergence of transcatheter valve implantation techniques offers opportunities to expand treatment options to these patients. It is essential, therefore, in the assessment of this new technology, to understand the burden of untreated disease, not only for clinicians but also for the health administrators.

We describe our attempts to define the burden of aortic stenosis in a large tertiary care centre in New Zealand with the intent to improve the care of patients with aortic stenosis in our region with a better understanding of patient demographics and impact of the disease on not only mortality but also morbidity and cost to the health care system.

This study evaluates the burden of untreated severe aortic stenosis at Waikato Hospital.

The study aims are to:

- Assess the outcome of patients with severe aortic stenosis who are managed with the surgical and medical option.
- Improve the management of severe aortic stenosis by understanding the patients' demographics.
- Assess the impact on the patients' quality of life.
- Assess the impact to the health care system.
- Assess the impact on the survival of the patient.

Methods

All consecutive patients, with severe aortic stenosis who presented to the Echocardiography Laboratory at Waikato Hospital, New Zealand between 1 January 2005 and 31 December 2009, were identified retrospectively.

Definition of severe aortic stenosis—Echocardiography criteria for diagnosing severe aortic stenosis applied according to ACC/AHA guidelines[3].

We used at least one of the following parameters:

- Aortic valve area <1 cm^2.
- Maximum velocity across aortic valve >4 m/s.
- Mean pressure gradient across aortic valve >40 mmHg.

If there was any discrepancies with these parameters, the Dimensionless Index was used to confirm severe aortic stenosis

Long-term outcome data was obtained by review of medical records and telephone contact with patients and their primary care physicians. All of the patients identified with severe aortic stenosis (106 patients) were divided into 3 groups according to the management strategy:

- Group 1: Asymptomatic aortic stenosis.
- Group 2: Symptomatic aortic stenosis not treated surgically.
- Group 3: Symptomatic aortic stenosis treated surgically

The history of symptomology was assessed by the cardiologist and asymptomatic patients were followed 6–12monthly if felt they were operable in the future. All symptomatic patients were discussed at the combined meeting with cardiac surgeons and cardiologist for eligibility for surgery.

The mortality, number of days spent in hospital per year and symptomatic status (angina >CCS class II, dyspnoea >NYHA class II, syncope) during follow-up were compared between these three groups.

Statistical analysis—Continuous variables were presented as the mean value±SD. These variables were compared, in between the groups, by using Student t-test. Discrete variables including the outcome measures of severe aortic stenosis were compared using Chi-squared test (double classification) with Yates correction. Event-free survival was plotted using Kaplan-Meir curves (censored data).

Results

A total of 105 patients with severe aortic stenosis were identified (mean age 76±13 years, 51% men) over the 12-month study period. The majority (76% (80/105)) were symptomatic with aortic stenosis. The median follow-ups in Group 1, Group 2 and Group 3 were 35 months (interquartile range: 27–37 months), 24 months (interquartile range: 11–36 months) and 36 months (interquartile range: 32-37 months) respectively. The baseline demographics of these groups are shown in Table 1. Clinical endpoints are shown in Table 2. At 36-month follow-up patients with symptomatic severe aortic stenosis treated surgically with aortic valve replacement had an excellent survival outcome compared to symptomatic patients who were declined for surgery (82% versus 27%, $p<0.0001$) (Figure 1). Of interest, mortality was also increased in patients with asymptomatic aortic stenosis compared to mortality of patients with symptomatic aortic stenosis treated surgically with aortic valve replacement (36% versus 18%, $p<0.001$). Unfortunately, the precise cause of death in-group 1 is unknown as the majority (8 out of 9 patients) died in the community.

Table 1. Baseline demographic data

Baseline characteristics	Group 1 (n=25)	Group 2 (n=41)	Group 3 (n=39)
Mean age (± SD)	81.7±14.4	83.4±7.6	66.1±12.2
Gender: Female Male	 44% 56%	 56% 44%	 44% 57%
Prior MI	22%	41%	17%
Coronary artery disease	34%	59%	50%
Previous PCI	4%	10%	17%
Previous CABG	0%	13%	26%
Prior stroke	20%	15%	2%
Prior TIA	16%	15%	7%
Peripheral vascular disease	16%	18%	12%
COPD	24%	31%	7%
Atrial fibrillation	40%	49%	43%
Diabetes Mellitus	20%	21%	24%
Hypertension	64%	69%	60%
Smoking: Current smoker Ex smoker	 12% 28%	 3% 44%	 2% 36%
Logistic Euro Score	16%	26%	10%
-<10%	32%	19%	68%
-10%-20%	36%	19%	17%
->20%	32%	62%	15%
NYHA class I-II	100%	64%	60%
NYHA class III-IV	0%	36%	40%
CCS class I-II	100%	90%	83%
CCS class III-IV	0%	10%	17%
Syncope	8%	15%	10%

PCI=Percutaneous coronary intervention, CABG=Coronary artery bypass surgery, TIA=Transient ischemic accident, COPD=Chronic obstructive pulmonary disease, NYHA=New York Heart Association, CCS=Canadian Cardiovascular Society.

Table 2. Outcomes of patient groups during follow-up

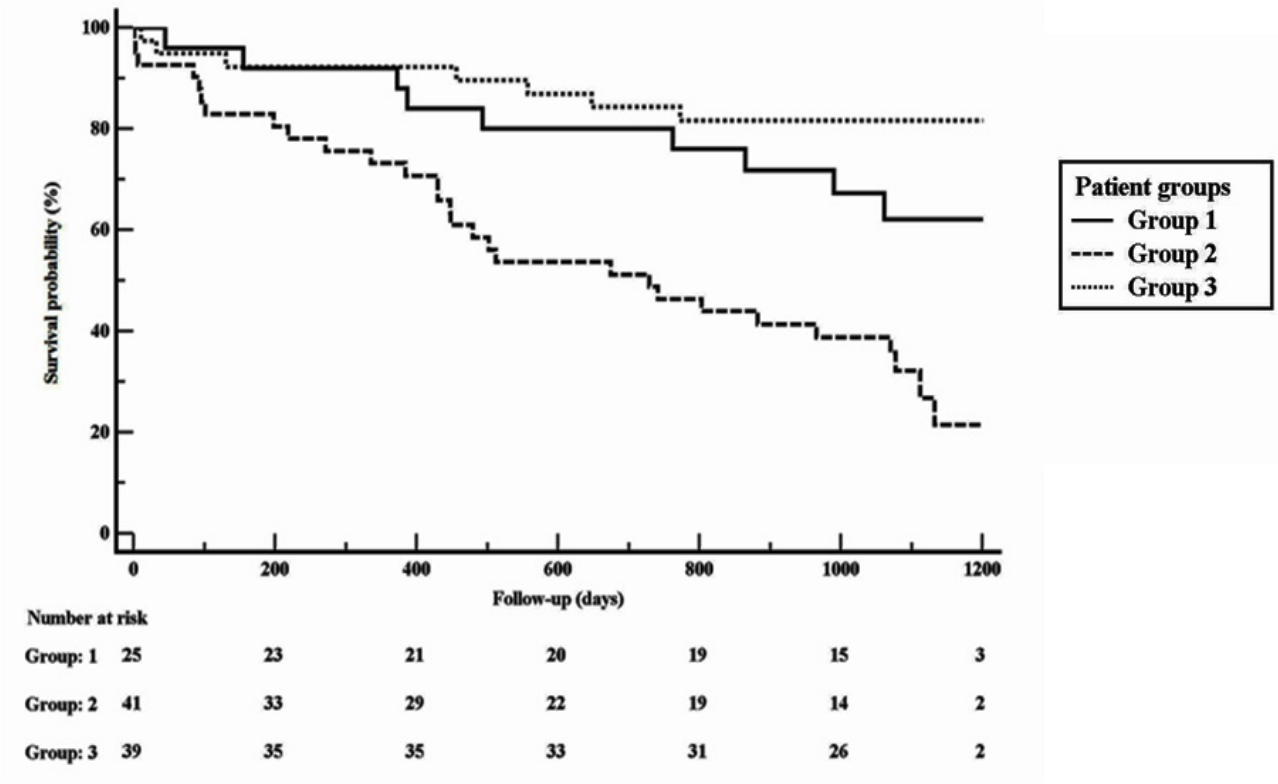
Group	Number of patients	All-cause mortality	Hospital days per 100 pt-years	Symptoms at follow-up
Group 1	25 (24%)	9 (36%)	3.5	0%
Group 2	41 (39%)	30 (73%)	10.1	64%
Group 3	39 (37%)	7 (18%)	6.4	0%

Patients with symptomatic severe aortic stenosis managed conservatively (Group 2) had significantly more recurrent hospitalisation and time in hospital related to cardiovascular causes compared to patients who underwent surgical valve replacement (10.1 versus 6.4 days/100 patient-years, p<0.0001).

Similarly, 2 of 3 symptomatic patients who were managed conservatively (Group 2) had on going symptoms, (greater than CCS class II angina or NYHA class II

dyspnoea or syncope). Asymptomatic patients and patients with aortic valve replacement had remained free from significant valve related symptoms during follow-up.

Figure 1. Kaplan-Meier survival of the 3 patient groups



Limitations—As our study was a retrospective analysis and most death in our cohorts occurred in the community there were major limitations in finding the actual cause of death. This limits our ability to define the actual valve related mortality.

Discussion

Aortic stenosis is a common valvular heart disease in the western world. It is a disease that is increasing in prevalence as the population ages. The onset of symptoms in the patient with severe aortic stenosis is a poor prognostic sign. These patients should be closely monitored and should be treated with aortic valve replacement if appropriate.

While surgical aortic valve replacement offers excellent result for patients with symptomatic severe aortic stenosis, those who are not considered as suitable surgical candidates have poor survival.[2,12–14]

Almost half of the symptomatic patients in our study were declined for aortic valve replacement surgery. This was mainly due to presumedhigh-risk involved with open-heart surgery because of their multiple comorbidities and age. The mean logistic EuroScore in this group was significantly high compared to a mean logistic EuroScore of the symptomatic patients treated surgically (26.1±16.0 versus 10.4±12.3,

p<0.0001). This implies that non-surgical patients had more co-morbidity and were of higher risk compared to the surgical patients. In addition, they were much older compared to the surgical group (mean age: 83.4±7.6 versus 66.1±12.2, p<0.0001).

Hence in our study as patients who were declined for surgery could have been done so appropriately due to multiple comorbidities which could have lead to an early mortality, we cannot assess the actual valve related mortality due to this decision.

Usually, symptomatic aortic stenosis patients, who are declined for aortic valve replacement surgery due to excessive risk, have a poor survival. In our study, their survival was as low as 27% at 36 months, which is consistent with the published literature. In a retrospective study of 144 symptomatic patients, survival at 3 years was 87% in 125 patients who underwent valve replacement compared to 21% in 19 patients who were managed conservatively.[15]

In our study, symptomatic patients treated surgically had not only a significant reduction in mortality (18% versus 73%, p value<0.001) but also in days spent in hospital for cardiac causes (6.4 versus 10.1 days/ 100 patient-years, p<0.0001) compared to those were declined for surgery. Although randomised trials comparing surgery to continued conservative therapy for severe symptomatic aortic stenosis have not been performed, observational studies have found that aortic valve replacement surgery in this setting is almost always followed by symptomatic improvement and a substantial increase in survival.[4,5,16]

There have been major advances in cardiac surgery, which affect the consideration of surgical treatment in elderly patients who in the past may not have been surgical candidates.[17] Among patients who survive the surgery and perioperative period, the level of function, quality of life and survival are the same as in a general population of age-matched subjects.[5,18-21] As a result, the classic view that surgery should be considered only for elderly patients in excellent general condition is being challenged as higher success rates for isolated aortic valve replacement is obtained in patients with comorbidities with pre existing good functional level.[3,22–24]

An unexpected finding of our study was that 1 in 3 of asymptomatic patients dying during the follow-up. Our study is also limited by being unable to define the exact cause of death in this group especially where the majority were elderly. Objective defining of the symptom status with a treadmill may have better categorise this group. Although exercise testing is contraindicated in patients with symptomatic aortic stenosis, it has been shown to be safe in asymptomatic patients and can identify the presence of exercise-induced haemodynamic compromise, which is a relative indication for elective surgery.[3,25–27]

High-risk echocardiographic features (presence of moderate to severe valvular calcification, a rapid progression in the aortic jet velocity (0.3 m/sec within one year) and a high aortic jet velocity >4.5 m/sec), advance age (>50 years) and raised plasma B-type natriuretic peptide concentration may help to identify the patients who are likely to rapidly progress to symptomatic state needing aortic valve surgery or dying from a cardiac cause.[8,26–30]For symptomatic patients who are declined surgery, alternative therapies such as transcatheter aortic valve implantation may be an option.[31–34] This is a promising approach[35–39] that is now being used at many highly

specialised centres around the world for specific patients (elderly, surgically declined symptomatic aortic stenosis population).

Randomised study data using the Edwards SAPIEN™ valve (PARTNER NCT00530894) will be available in 2010 that looks at outcomes against surgery in the high-risk population and also in patients declined from surgery. Data on cost effectiveness and long-term durability of this valve will allow wider application of this program.

Conclusion

Untreated symptomatic severe aortic stenosis is associated with a poor prognosis and significant morbidity while aortic valve replacement surgery results in improved survival and quality of life. A significant number of patients with symptomatic aortic stenosis are declined surgical aortic valve replacement due to comorbidities and new therapies such as transcatheter aortic valve implantation may be of value as alternative treatment options to manage these patients. Asymptomatic patients with severe aortic stenosis should be carefully monitored objectively for development of symptoms and early referral for surgery should be recommended.

Competing interests: None.

Author information: Suresh Perera, Namal Wijesinghe, Elene Ly, Gerard Devlin, Sanjeevan Pasupati, Cardiologists, Department of Cardiology, Waikato Hospital, Hamilton

Correspondence: Dr Sanjeevan Pasupati, Department of Cardiology, Waikato Hospital, Private Bag 3200, Hamilton 3240, New Zealand. Email: drspasupati@gmail.com

References:

1. Otto CM. Aortic stenosis—listen to the patient, look at the valve. N Engl J Med 2000;343:652-4.
2. Ross J Jr. Braunwald E. Aortic stenosis. Circulation 1968;38:61-7.
3. Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing Committee to Revise the 1998 guidelines for the management of patients with valvular heart disease) developed in collaboration with the Society of Cardiovascular Anesthesiologists endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons. J Am Coll Cardiol 2006;48:e1-148.
4. Lindblom D, Lindblom U, Qvist J, Lundstrom H. Long-term relative survival rates after heart valve replacement. J Am Coll Cardiol 1990;15:566-73.
5. Gilbert T, Orr W, Banning AP. Surgery for aortic stenosis in severely symptomatic patients older than 80 years: experience in a single UK centre. Heart 1999;82:138-42.
6. Bouma BJ, van Den Brink RB, van Der Meulen JH, et al. To operate or not on elderly patients with aortic stenosis: the decision and its consequences. Heart 1999;82:143-8.
7. Iung B, Cachier A, Baron G, et al. Decision-making in elderly patients with severe aortic stenosis: why are so many denied surgery? Eur Heart J 2005;26:2714-20.
8. Pellikka PA, Sarano ME, Nishimura RA, et al. Outcome of 622 adults with asymptomatic, hemodynamically significant aortic stenosis during prolonged follow-up. Circulation 2005;111:3290-5.

9. Charlson E, Legedza AT, Hamel MB. Decision-making and outcomes in severe symptomatic aortic stenosis. J Heart Valve Dis 2006;15:312-21.
10. Bach DS, Cimino N, Deeb GM. Unoperated patients with severe aortic stenosis. J Am Coll Cardiol 2007;50:2018-9.
11. Abdul-Hamid AR, Mulley GP. Why do so few older people with aortic stenosis have valve replacement surgery? Age Ageing 1999;28:261-4.
12. Carabello BA. Timing of valve replacement in aortic stenosis. Moving closer to perfection. Circulation 1997;95:2241-3.
13. Carabello BA. Ventricular function in aortic stenosis: how low can you go? J Am Coll Cardiol 2002;39:1364-5.
14. Carabello BA, Crawford FA, Jr. Valvular heart disease. N Engl J Med 1997;337:32-41.
15. Schwarz F, Baumann P, Manthey J, et al. The effect of aortic valve replacement on survival. Circulation 1982;66:1105-10.
16. Kouchoukos NT, Davila-Roman VG, Spray TL, et al. Replacement of the aortic root with a pulmonary autograft in children and young adults with aortic-valve disease. N Engl J Med 1994;330:1-6.
17. Gammie JS, Brown JW, Brown JM, et al. Aortic valve bypass for the high-risk patient with aortic stenosis. Ann Thorac Surg 2006;81:1605-10.
18. Olsson M, Granstrom L, Lindblom D, et al. Aortic valve replacement in octogenarians with aortic stenosis: a case-control study. J Am Coll Cardiol 1992;20:1512-6.
19. Shapira OM, Kelleher RM, Zelingher J, et al. Prognosis and quality of life after valve surgery in patients older than 75 years. Chest 1997;112:885-94.
20. Kolh P, Kerzmann A, Lahaye L, et al. Cardiac surgery in octogenarians; peri-operative outcome and long-term results. Eur Heart J 2001;22:1235-43.
21. Kolh P, Lahaye L, Gerard P, Limet R. Aortic valve replacement in the octogenarians: perioperative outcome and clinical follow-up. Eur J Cardiothorac Surg 1999;16:68-73.
22. Asimakopoulos G, Edwards MB, Taylor KM. Aortic valve replacement in patients 80 years of age and older: survival and cause of death based on 1100 cases: collective results from the UK Heart Valve Registry. Circulation 1997;96:3403-8.
23. Bramstedt KA. Aortic valve replacement in the elderly: frequently indicated yet frequently denied. Gerontology 2003;49:46-9.
24. Langanay T, De Latour B, Ligier K, et al. Surgery for aortic stenosis in octogenarians: influence of coronary disease and other comorbidities on hospital mortality. J Heart Valve Dis 2004;13:545-52; discussion 552-3.
25. Linderholm H, Osterman G, Teien D. Detection of coronary artery disease by means of exercise ECG in patients with aortic stenosis. Acta Med Scand 1985;218:181-8.
26. Amato MC, Moffa PJ, Werner KE, Ramires JA. Treatment decision in asymptomatic aortic valve stenosis: role of exercise testing. Heart 2001;86:381-6.
27. Otto CM, Burwash IG, Legget ME, et al. Prospective study of asymptomatic valvular aortic stenosis. Clinical, echocardiographic, and exercise predictors of outcome. Circulation 1997;95:2262-70.
28. Rosenhek R, Binder T, Porenta G, et al. Predictors of outcome in severe, asymptomatic aortic stenosis. N Engl J Med 2000;343:611-7.
29. Gerber IL, Stewart RA, Legget ME, et al. Increased plasma natriuretic peptide levels reflect symptom onset in aortic stenosis. Circulation 2003;107:1884-90.
30. Lim P, Monin JL, Monchi M, et al. Predictors of outcome in patients with severe aortic stenosis and normal left ventricular function: role of B-type natriuretic peptide. Eur Heart J 2004;25:2048-53.
31. Cribier A, Eltchaninoff H, Bash A, et al. Percutaneous transcatheter implantation of an aortic valve prosthesis for calcific aortic stenosis: first human case description. Circulation 2002;106:3006-8.

32. Webb JG, Pasupati S, Humphries K, et al. Percutaneous transarterial aortic valve replacement in selected high-risk patients with aortic stenosis. Circulation 2007;116:755-63.
33. Grube E, Laborde JC, Gerckens U, et al. Percutaneous implantation of the CoreValve self-expanding valve prosthesis in high-risk patients with aortic valve disease: the Siegburg first-in-man study. Circulation 2006;114:1616-24.
34. Lichtenstein SV, Cheung A, Ye J, et al. Transapical Transcatheter Aortic Valve Implantation in Humans. Initial Clinical Experience. Circulation 2006.
35. Webb JG. Percutaneous aortic valve replacement. Curr Cardiol Rep 2008;10:104-9.
36. Webb JG, Altwegg L, Boone RH, et al. Transcatheter aortic valve implantation: impact on clinical and valve-related outcomes. Circulation 2009;119:3009-16.
37. Piazza N, Grube E, Gerckens U, et al. Procedural and 30-day outcomes following transcatheter aortic valve implantation using the third generation (18 Fr) corevalve revalving system: results from the multicentre, expanded evaluation registry 1-year following CE mark approval. EuroIntervention 2008;4:242-9.
38. Grube E, Buellesfeld L, Mueller R, et al. Progress and Current Status of Percutaneous Aortic Valve Replacement: Results of Three Device Generations of the CoreValve Revalving System. Circ Cardiovasc Intervent. 2008;1:167-175.
39. Ye J, Cheung A, Lichtenstein SV, et al. Six-month outcome of transapical transcatheter aortic valve implantation in the initial seven patients. Eur J Cardiothorac Surg 2007;31:16-21.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Review of 100 consecutive microvascular free flaps

Ryan Gao, Stanley Loo

Abstract

Aim To analyse the outcome of microvascular free flap reconstructions in Middlemore Hospital (South Auckland, New Zealand).

Method 100 consecutive free flap reconstructions from January 2004 to April 2010 were identified from the Middlemore Hospital Theatre Coding List. Basic patient demographics and indication for surgery along with free flap types were recorded and outcomes were analysed.

Results The free flap success rate was 96%. There were 21 short term complications without any perioperative mortality. The most common complication was flap infection (7/21) followed by vascular thrombosis (6/21 venous and 1/21 arterial). Other complications included partial ischaemic flap (3/21), haematoma (2/21), venous congestion (1/21) and partial wound dehiscence (1/21).

Fourteen flaps needed salvage procedures in the operating theatre including eight cases for re-anastomosis of vessels. The overall successful salvage rate was 71% resulting in four failures. The successful salvage rate following re-anastomosis of vessels was 63%.

Conclusion Overall success and salvage rates for free flap reconstructions at our plastics and reconstruction centre are comparable to that of international literature. Diligent postoperative monitoring and early return to theatre for re-exploration is the key to ensuring maximal free flap success.

After decades of refinement of surgical techniques and improved perioperative management, free flaps have become accepted as the procedure of choice for reconstructing many complex wound defects. Success rates exceeding 90% have consistently been reported in the international literature.[1–4] Nevertheless, complications from free flap reconstructions inevitably occur and flap failure can be disastrous for both the patient and the surgical team involved.

Herein, we report the outcome of 100 consecutive free flap reconstructions from our regional plastics and reconstruction centre.

Method

100 consecutive free flap reconstructions from January 2004 to April 2010 were identified from the Middlemore Hospital Theatre Coding List. Patients who were identified as having undergone a ‘free flap’ operation or any subtype of free tissue transfer [e.g. latissimus dorsi (LD), serratus anterior (SA), anterolateral thigh (ALT) etc] were included in the study.

Basic patient demographics were recorded as well as indication for surgery and type of free flap. Adverse outcomes were recorded as free flap failure or other complications including infection, venous congestion, dehiscence, haematoma formation, and vascular thrombosis.

Results

100 consecutive free flap reconstructions were performed on 96 patients by 17 consultant plastic surgeons during the study period. One patient received two LD flaps following bilateral forefoot amputations as a result of meningococcal septicaemia. Another patient received two free flaps (LD and SA) following Gustilo-Anderson type IIIB injuries to both his legs.[5] Two patients underwent successful repeat free flaps following failure of the first operation. There were 34 females and 62 males. The median age at the time of surgery was 43 years (range 3–79 years). The most common indication for free flap reconstruction was limb trauma (60%), followed by tumours (13%), infections (11%), mastectomies (10%), burns (3%) and iatrogenic causes (3%) (Table 1).

Table 1. Indications for free flaps versus complications

Indications	No.	Complica-tions	Thrombosis	Infection	Ischaemia	Haematoma	Venous congestion	Dehiscence
Limb trauma	60							
MVA	38	8	4 (1†)	3	1‡			
Non MVA	22	5	1	2	1‡	1‡		
Tumours	13	3						
Limb	7	1	1†			1†	1†	
Head and neck	5	0	1					
Trunk	1							
Infection/necrosis	11							
Chronic ulcer	5	1		1				
Osteomyelitis	3							
Necrotising fasciitis	2							
Gangrene	1	1						1‡
Mastectomy	10	0						
Burns	3	2		1	1‡			
Iatrogenic	3	0						
Redo Flap	2							
Postoperative wound	1							
Total	**100**	**21**	**7**	**7**	**3**	**2**	**1**	**1**

† Associated with flap failure; ‡ Associated with partial flap failure; MVA=Motor vehicle accident.

Ten different types of free flaps were utilised during the study period (Table 2). Three types of free flaps (LD, ALT and transverse rectus abdominus myocutaneous [TRAM]) contributed to 65% of the total cases. Flap failures occurred independently compared to the types of flaps used. Specifically, there was one failure from each of the LD, TRAM, gracilis and fibular flaps. The LD was the most widely utilised flap accounting for 26% of the total cases. Half of the TRAM flaps (10/20) were performed for breast reconstruction following mastectomies. The use of ALT flaps proved to be extremely safe in this series; of the 19 ALT flaps utilised, only three complications occurred (two infections and one partial flap failure). On the contrary, complications from gracilis and fibular flaps were high, 3/9 and 2/6 respectively, including one failure from each of these two types of flaps.

Table 2. Free flap types versus complications

Flap type	No.	Complica-tions	Thrombosis	Flap infection	Ischaemia	Haematoma	Venous congestion	Dehiscence
LD	26	6		4	1‡	1†		
TRAM	20	3	1†	1	1‡			
ALT	19	3		2				1‡
Radial forearm	11	3	3					
Gracilis	9	3	2 (1†)			1‡		
Fibular	6	2	1				1†	
Parascapular	1	1			1‡			
Others	8	0						
Lateral forearm	4							
Serratus anterior	2							
Iliac crest	2							
Total	**100**	**21**	**7**	**7**	**3**	**2**	**1**	**1**

† Associated with flap failure; ‡ Associated with partial flap failure; LD=Latissimus dorsi; TRAM=Transverse rectus abdominus myocutaneous; ALT=Anterolateral thigh.

Overall, 21 complications occurred without any perioperative mortality. All seven cases of postoperative infections responded well to intravenous antibiotics. Fourteen flaps were taken back to the operating theatre for further procedures, of which, ten (71%) were successfully salvaged (Table 3). Eight flaps required redo of vascular anastomosis (seven venous and one arterial).

Five out of the eight (63%) redo anastomosis were successful, with the help of medicinal leeches in two cases. Five flaps required debridement of partial ischaemic tissue. The resulting defects were able to be primarily closed in three cases. Split-thickness skin graft was used for soft tissue coverage following debridement of a LD and a gracilis flap. The reasons for the partial flap failures were not accurately recorded for all five cases. However, it was evident from the clinical documents that haematoma formation and wound dehiscence were to blame for partial failure of the gracilis and the ALT flaps respectively.

Table 3. Free flaps taken back to theatre

Flap type	No.	Flaps taken back to theatre	Redo venous anastomosis	Redo arterial anastomosis	Debridement of partial ischaemic tissue	Unsuccessful evacuation of haematoma
LD	26	2			1‡ + SSG	1†
TRAM	20	2	1†		1‡	
ALT	19	1			1‡	
Radial forearm	11	3	3 (2xLeech)			
Gracilis	9	3	1	1†	1‡ + SSG	
Fibular	6	2	2 (1†)			
Parascapular	1	1			1‡	
Total	**92**	**14**	**7**	**1**	**5**	**1**

† Associated with flap failure; ‡ Associated with partial flap failure; Leech: medicinal leech therapy; SSG=Split-thickness skin graft; LD=Latissimus dorsi; TRAM=Transverse rectus abdominus myocutaneous; ALT=Anterolateral thigh.

Discussion

Free flap failure is a dreaded complication which plagues even the most experienced microvascular surgeons. The repercussions of flap failure include poor functional and cosmetic outcomes for the patient along with devastating psychological impact on both the patient and the surgical team involved.

Studies which have investigated the causes of flap failures have shown that vascular compromise is the leading cause of flap failures in the immediate postoperative period. Furthermore, venous thrombosis occurs much more frequently than arterial occlusions.[6-10] On the other hand, flaps that fail after 48 hours were mostly due to mechanical stress around the anastomosis. Factors that predispose to mechanical stress around the anastomosis include haematoma formation, physical kinks in the vascular loop, poor flap design or inappropriate in-setting during the time of surgery.[7]

In our series of 100 consecutive free flaps, a total of four flap failures occurred (Table 4). Three flaps failed as a result of vascular thrombosis, including two venous and one arterial thrombus formation. The fourth case failed as a result of mechanical stress around the anastomosis caused by a large haematoma.

Table 4. Flap failures

Cases	No. 1	No. 2	No. 3	No. 4
Flap type	Gracilis	TRAM	Fibular	LD
Age/gender	47yo/M	49yo/F	49yo/M	73yo/F
Indication	Trauma	Tumour	Tumour	Tumour
Causes of failure	Arterial thrombus	Venous thrombus	Venous thrombus	Haematoma
Identifiable factors Attributable to failure	Arterial thrombus	Prolonged operation	Convoluted anastomosis	Mechanical obstruction
Outcome	Redo flap	Redo flap	Resection	Resection

LD=Latissimus dorsi; TRAM=Transverse rectus abdominus myocutaneous.

It is worth noting that the most common indication for free flap coverage in our series was limb trauma (60% of total cases). This is reflective of the fact that our plastics unit is part of a large tertiary trauma centre. Thirteen complications arose from this cohort of patients including one flap failure.

The flap failure occurred in a 47-year-old patient who sustained a Gustilo-Anderson type IIIB distal tibia fracture as a result of a motor vehicle accident.[5] He underwent an uncomplicated intramedullary nailing of the fracture and subsequently proceeded to have a free gracilis flap for coverage of the resulting pre-tibial soft tissue defect.

Two hours postoperatively, it was noted that the flap became ischaemic and the patient returned to theatre for exploration. A small arterial thrombus was identified approximately 1cm distal to the anastomosis. Despite thrombecotomy, the gracilis flap failed to survive and the patient underwent a successful free LD flap 5 days later. Arterial thrombus is known to be associated with a poor flap outcome even with prompt salvage attempts. In Nakatuska's review of 2372 free flaps for head and neck

reconstruction following cancer resection, arterial thrombectomy was successful in only 15% of cases.[11]

Free flap coverage for soft tissue defects following tumour resection accounted for 13% of the total cases performed during the study period. A disproportionally high number of complications occurred in this cohort of patients including three failures. Venous thrombosis was accountable for two of the failures and the final flap failure was caused by a large haematoma causing mechanical stress around the anastomosis.

The second free flap failure in our series occurred in a patient who underwent resection of a high grade leiomyosarcoma from her thigh with immediate TRAM flap reconstruction. The patient had an extended procedure with total operative time of more than 12 hours. Strong Doppler signals were recorded during and shortly after the operation. However, two hours postoperatively the flap was found to be ischaemic and upon exploration in the operating theatre, a venous thrombus was evacuated distal to the anastomosis.

The salvage procedure proved to be futile with the flap again becoming ischaemic several hours later requiring complete resection. The patient subsequently underwent a successful LD flap 2 weeks later. Prolonged operation time of more than 10 hours has been shown to be a significant risk factor for poor flap outcome including failure.[12-15] Another predictor of flap outcome has been shown to be the number of operating surgeons in theatre.[16,17] Intuitively, longer operations with more surgeons often equate to higher degree of surgical complexity, greater intraoperative fluid shifts, greater risks of infections and longer ischaemia period.[12]

Extended ischaemia time increases the risk of anoxic injury as a result of anaerobic metabolism and the accumulation of inflammatory mediators. In addition, following re-establishment of vascular flow following prolonged ischaemia; the incidence of reperfusion injury also increases markedly with resultant deleterious effect on flap survival.[12] Furthermore, surgeon fatigue associated with extended operating time may potentially affect proper tissue handling, vessel preparation and anastomosis, flap insetting and skin closure.

The third flap failure occurred in a patient who underwent reconstruction of his humerus with a free fibular flap. The patient originally had resection of an aggressive fibromatosis from his left humerus with intercalary allograft reconstruction and plating. The allograft was complicated by wound infections requiring multiple debridements, long term antibiotics and ultimately removal of the allograft and insertion of cement spacer.

Two years after his primary resection, the patient proceeded to have a free fibular flap to reconstruct his humerus. Approximately 4 hours after the operation, the flap was found to show signs of venous congestion. Upon urgent exploration in the operating theatre, the arterial anastomosis was found to be patent and functional. However, a very convoluted venous anastomosis was identified with evidence of venous congestion. The excessive redundancy in the venous anastomosis was excised and the ends re-anastomosed.

Unfortunately, the flap failed to survive and the patient eventually underwent excision of his fibular flap and an en bloc upper humeral interscapulothoracic resection. The nature of this retrospective review precluded accurate assessment of the reason why

there was such a convoluted venous anastomosis. Nevertheless, better flap design with particular attention paid to the venous anastomosis and more careful in-setting of the flap may have made a difference in the outcome of this fibular flap.

The final flap failure occurred in a 73-year-old lady with a history of large malignant fibrous histiocytoma over her thigh involving all four quadriceps muscles. She underwent wide local excision with neoadjuvant radiotherapy and a delayed LD flap for coverage of the soft tissue defect a month later. Approximately 4 hours postoperatively; the flap appeared to be congested with poor Doppler signals. Upon urgent exploration, a large haematoma was identified deep to the flap with evidence of diffuse bleeding points around the anastomosis. The anastomosis was found to be patent and functional without any evidence of thrombosis. The haematoma was evacuated and the bleeding vessels cauterised.

Unfortunately, the flap continued to show signs of worsening congestion and the patient returned to theatre 6 hours later for repeat exploration. Intraoperatively it was noted that there were generalised ooze from the wound with more focal bleeding vessels which were again cauterised. The flap was re-inset and the wound closed. Fourty-eight hours later, the flap was found to be necrotic and was excised. It is postulated that the persistent wound ooze and microvascular bleeding may have been related to the harmful effects of neoadjuvant radiotherapy on local tissues and vasculature.

An in vitro comparative study of biopsies from irradiated versus unirradiated arteries in 147 patients undergoing free flap surgery showed that radiation caused significant intimal thickening.[18] The authors also noticed an increase in proteoglycan deposition and inflammatory cell content in the irradiated vessels. Whilst some studies have echoed the increased risk of recipient site complications in post-irradiated wound beds,[13,17,19] the majority of published literature thus far has failed to show any statistically significant effect of radiotherapy on the overall rate of free flap complications or length of hospital stay.[8,12,20–23]

In their retrospective review of 185 consecutive free flap reconstructions, Clark et al concluded that surgeons should not be deterred from performing free tissue transfer for reconstruction in post-irradiated wounds.[17]

Conclusion

Free flaps are safe and reliable reconstructive options particularly for complex defects. The present study showed that free flap reconstructions in our regional plastics and reconstruction centre have excellent success and salvage rates and the results are comparable to international literature. It must be re-iterated that full institutional support in the form of allocation of theatre time, personnel and training of allied health staff to effectively monitor postoperative patients is essential to ensure free flap success.

Competing interests: None.

Author information: Ryan Gao, House Officer; Stanley Loo, Consultant Plastics and Reconstructive Surgeon; Middlemore Hospital, Auckland

Correspondence: Ryan Gao, Department of Plastics and Reconstructive Surgery, Middlemore Hospital, Hospital Road, Otahuhu, Auckland, New Zealand. Fax: +64 (0)9 8496238; email: ygao921@gmail.com

References:

1. Kruse AL, Luebbers HT, Gratz KW, Obwegeser JA. Factors influencing survival of free-flap in reconstruction for cancer of the head and neck: a literature review. Microsurgery. 2010;30:242-8.
2. Khouri RK. Free flap surgery. The second decade. Clin Plast Surg. 1992;19:757-61.
3. Stephan B, Schenk JF, Nemeh A, Pindur G. The use of antithrombotic agents in microvascular surgery. Clin Hemorheol Microcirc. 2009;43:51-6.
4. Rasheed T, Lewis HG, Gordon DJ. A review of 100 consecutive free tissue transfers. Ulster Med J. 2000;69:14-8.
5. Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 1976;58:453-8.
6. Hidalgo DA, Disa JJ, Cordeiro PG, Hu QY. A review of 716 consecutive free flaps for oncologic surgical defects: refinement in donor-site selection and technique. Plast Reconstr Surg. 1998;102:722-32.
7. Novakovic D, Patel RS, Goldstein DP, Gullane PJ. Salvage of failed free flaps used in head and neck reconstruction. Head Neck Oncol. 2009;1:33.
8. Kroll SS, Schusterman MA, Reece GP, et al. Choice of flap and incidence of free flap success. Plast Reconstr Surg. 1996;98:459-63.
9. Miyasaka M, Ichikawa K, Nishimura M, et al. Salvage operations of free tissue transfer following internal jugular venous thrombosis: a review of 4 cases. Microsurgery. 2005;25:191-5.
10. Brown JS, Devine JC, Magennis P, et al. Factors that influence the outcome of salvage in free tissue transfer. Br J Oral Maxillofac Surg. 2003;41:16-20.
11. Nakatsuka T, Harii K, Asato H, et al. Analytic review of 2372 free flap transfers for head and neck reconstruction following cancer resection. J Reconstr Microsurg. 2003;19:363-8.
12. Pattani KM, Byrne P, Boahene K, Richmon J. What makes a good flap go bad? A critical analysis of the literature of intraoperative factors related to free flap failure. Laryngoscope. 2010;120:717-23.
13. Singh B, Cordeiro PG, Santamaria E, et al. Factors associated with complications in microvascular reconstruction of head and neck defects. Plast Reconstr Surg. 1999;103:403-11.
14. Rosenberg AJ, Van Cann EM, van der Bilt A, et al. A prospective study on prognostic factors for free-flap reconstructions of head and neck defects. Int J Oral Maxillofac Surg. 2009;38:666-70.
15. Finical SJ, Doubek WG, Yugueros P, Johnson CH. The fate of free flaps used to reconstruct defects in recurrent head and neck cancers. Plast Reconstr Surg. 2001;107:1363-6.
16. Haughey BH, Wilson E, Kluwe L, et al. Free flap reconstruction of the head and neck: Analysis of 241 cases. Otolaryng Head Neck. 2001;125:10-7.
17. Clark JR, McCluskey SA, Hall F, et al. Predictors of morbidity following free flap reconstruction for cancer of the head and neck. Head Neck. 2007;29:1090-101.
18. Russell NS, Hoving S, Heeneman S, et al. Novel insights into pathological changes in muscular arteries of radiotherapy patients. Radiother Oncol. 2009;92:477-83.
19. Pohlenz P, Blessmann M, Heiland M, et al. Postoperative complications in 202 cases of microvascular head and neck reconstruction. J Craniomaxillofac Surg. 2007;35:311-5.
20. Choi S, Schwartz DL, Farwell DG, et al. Radiation therapy does not impact local complication rates after free flap reconstruction for head and neck cancer. Arch Otolaryngol Head Neck Surg. 2004;130:1308-12.

21. Smolka W, Iizuka T. Surgical reconstruction of maxilla and midface: clinical outcome and factors relating to postoperative complications. J Craniomaxillofac Surg. 2005;33:1-7.
22. Ryan MW, Hochman M. Length of stay after free flap reconstruction of the head and neck. Laryngoscope. 2000;110:210-6.
23. Bengtson BP, Schusterman MA, Baldwin BJ, et al. Influence of prior radiotherapy on the development of postoperative complications and success of free tissue transfers in head and neck cancer reconstruction. Am J Surg. 1993;166:326-30.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Colonic self-expanding metal stents (SEMS) in acute large bowel obstruction

Mohammad I Khan, Adrian Claydon

Abstract

Aim Colonic SEMS are increasing used in the management of acute large bowel obstruction, both as a bridge to surgery and as a definitive palliative measure in patients unfit for surgery. We describe our experience from a New Zealand hospital and compare our data with that already published in literature.

Methods In this retrospective 4-year study, data was collected from the case notes of 28 consecutive patients with acute large bowel obstruction referred for colonic SEMS. Uncovered Boston Scientific colonic SEMS were placed endoscopically under fluoroscopic guidance. Technical success was considered as correct placement of stent after deployment and clinical success as the passage of flatus and faeces after stent insertion. Data was analysed using descriptive statistics.

Results Our technical and clinical success rates were 90% and 88% respectively. The procedure was palliative in 15 patients and as a bridge to elective surgery in 13 cases. Procedure-related mortality was 7%. It was because of one early and one late perforation. The average length of stay post procedure was 2 days. Mean survival post stent insertion in the palliative group was 2.4 months and for those with a bridge to surgery was 14 months.

Conclusion Our results support the data published from international centres in terms of deployment of SEMS in patients with acute large bowel obstruction, both as a bridge to surgery and as a definitive palliative measure.

Large bowel obstruction is a surgical emergency. It is most commonly caused by colorectal cancer and rarely by benign strictures and extrinsic compression of the colon.[1] Presently, the standard of care for such patients is either a two-step diverting colostomy with subsequent colonic resection or a one-step resection with primary anastomosis with or without on-table lavage, especially in right-sided tumours where surgery is considered to be curative.

Permanent colostomy is performed in incurable, advanced cancers. Insertion of a self expanding metal stent (SEMS) is now a well established alternative to emergency surgery. It can be used both as a bridge to elective surgery, allowing time for optimization of preoperative care or as a definitive procedure in cases of locally unresectable tumours or in patients with comorbidities and therefore at high risk for surgery.

SEMS, therefore, avoids emergency surgery which has a high mortality and complication rate. Colostomy care also entails increased costs and a lower quality of life.[2] However, SEMS are costly (about $ 2000/stent) and are also associated with

procedure-related complications including bleeding, perforation, stent occlusion and migration.[3]

Over the last decade, there has been increasing international literature on the use of SEMS in acute large bowel obstruction. To date, New Zealand data has been limited to a single study which compared SEMS with traditional surgery in patients with metastatic tumours.[4] We describe our own experience in a New Zealand unit with SEMS, both as a palliative procedure in inoperable cases and as a bridge to elective surgery.

Patients and Methods

This is a retrospective study of 28 consecutive patients (12 males, 16 females) referred for colonic SEMS since November 2006 to our endoscopy unit. The mean age of the patients was 72 years with an age range from 31 to 92 years. All patients had CT of the abdomen and pelvis prior to stenting.

Twenty-four patients had clinical and radiographic evidence of colonic obstruction at the time of referral (abdominal pain, dilated loops on imaging), while the remaining four patients had either radiographic (two patients) or endoscopic evidence (two patients) of bowel obstruction with no visible lumen. Seven patients had right-sided obstruction (hepatic flexure; 3, transverse colon; 4) while the rest were left-sided obstructions. Two of the patients with right-sided tumours had stent insertion as a bridge to surgery. Both were referred from a peripheral hospital. Initial surgery was postponed as both patients had nutritional depletion, renal impairment, and in one case, urosepsis.

The mean length of the obstructing lesion, as identified on the CT scan was 4.8cm (range; 2–10cm). As shown in Table 1, thirteen patients were referred as a bridge to elective surgery. Fifteen patients were referred for palliative stenting because of locally unresectable lesions or the patients were either considered unfit for surgery (one patient) or declined surgery (two patients). These included one patient with extrinsic rectosigmoid compression because of metastatic ovarian tumour and one patient with diverticular stricture.

Table 1. Summary statistics of patients referred for palliative stenting and stenting as a bridge to surgery

Variables	Bridge to surgery	Palliative stenting
Patient No	15	13
Male : Female ratio	5:8	7:8
Right-sided lesions (*n*)	2	5
Left-sided lesions (*n*)	13	8
Mean tumour length (cm)	4	6
Survival		
Alive (*n*)	9	1
Dead (*n*)	6	12
Mean hospital stay (days)	2	2

SEMS were placed by two endoscopists during the study. An endoscopic technique was used under fluoroscopic guidance as seen in Figures 1 and 2.

Figure 1. Endoscopic placement of stent in a patient with primary colonic neoplasm

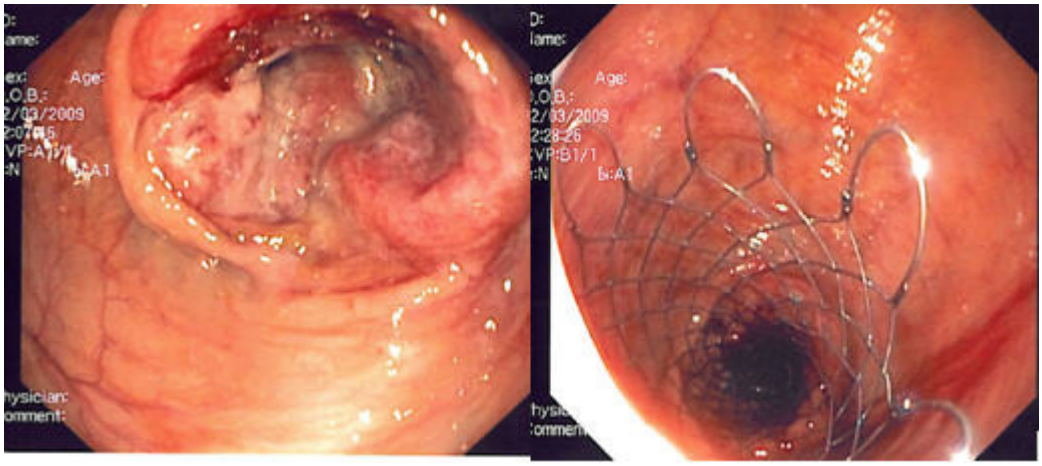
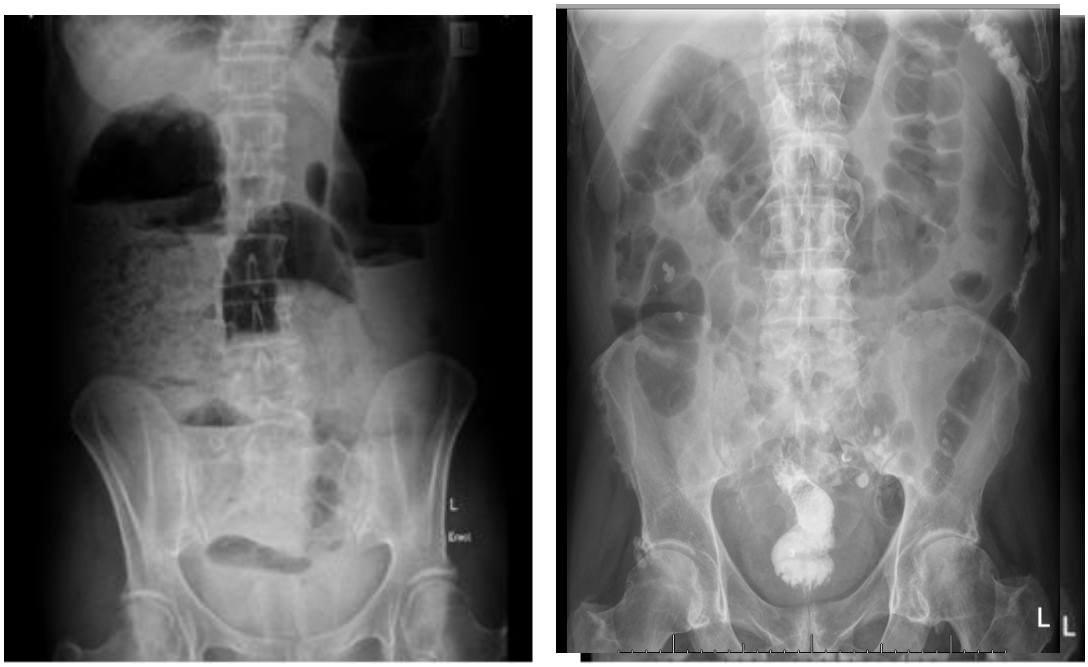


Figure 2. Plain radiographs pre and post deployment of SEMS in a patient with acute large bowel obstruction.



Intravenous midazolam and fentanyl were used for conscious sedation. Olympus therapeutic colonoscopes were used for all procedures. A therapeutic Olympus gastroscope was used in left-sided obstructions if the colonoscopy failed to reach the obstruction site. All cases were non-traversable with the scope. Routine balloon dilatation of the lesion before stenting was not performed.

A super stiff Boston Scientific 0.035 guidewire was passed through the stricture and then a canula was passed over the guidewire for radiological contrast to assess the length of the lesion and colonic anatomy immediately proximal to the lesion. The canula was then withdrawn and a metallic stent was passed over the wire, through the biopsy channel of the scope and positioned across the stricture.

The position of the released stent was checked both endoscopically and fluoroscopically before the endoscope was withdrawn. The stent used were mainly the Boston Scientific uncovered SEMS of different lengths and diameters were deployed depending upon the tumour length and site. The shortest stent used was 60mm and the longest one was 120mm. The widest diameter stent used was 25mm.

The procedure was considered technically successful if the SEMS was correctly placed across the stricture as determined by fluoro-imaging and endoscopy. Clinical success was considered to be the return of flatus and bowel movement.[5]

Descriptive statistics were used to analyse the data.

Results

Twenty-five patients had technically successful stent placement (90 %). Three of them required balloon dilatation of either the proximal or distal end of the stent after placement due defective expansion of the stents. One patient had a technical failure as the obstruction site was not reached and the patient was sent for emergency surgery. One patient had clinical failure after technically successful stent deployment and had emergency surgery. One patient had defective stent opening and after failure of balloon dilatation required the insertion of a second stent. One patient had immediate procedure-related perforation and died on the second day of stent placement. The three technical failure cases were from the group referred for palliative stenting.

One patient had delayed perforation 6 weeks after the placement of stent. One patient had significant haemorrhage post stenting requiring transfusion but settled on conservative treatment. Two patients had stent migration, one with diverticular stricture and another one with malignant rectal stricture one week post stent deployment. They went for elective surgery. One patient had two stents inserted to cover a long malignant stricture. Another patient had a second stent insertion after the first one migrated distally.

The mean length of hospital stay for successful stenting was 2 days (mean 1–7 days). Four cases were done as day cases and twelve were discharged the day after the procedure. Two patients died as inpatients after the procedure, one with perforation and another one with clinical failure after stent deployment. One patient had an inpatient surgery after the successful deployment of stent.

Ten patients are still alive post stenting. All except one of them had stenting as a bridge to surgery as shown in Table 1. The mean survival, to date, for them is 14 months.. Those who died after clinically successful placement of stent had a mean survival of 2.7 months (range; one week – six months).

As shown in Table 1, only one patient in the palliative group is still alive. He had resectable tumour at the time of diagnosis but surgery was precluded because of medical comorbidities . Only two patients had a survival of less than a month after successful deployment of stent.

Discussion

The first reported case of colonic SEMS was by Dohmoto *et al* in 1991 as palliative treatment in a case of malignant stenosis of the rectum.[6] Since then it has been increasingly used as a therapeutic modality not only in primary colorectal cancers but also for extrinsic and benign causes of large bowel obstruction.[1,7]

Colonic SEMS may be used as a primary form of palliation in advanced and unresectible tumours or as a bridge to elective surgery in patients presenting with acute large bowel obstruction. Colonic SEMS compares favourably with emergency surgery in terms of mortality and morbidity, length of hospital stay, cost-effectiveness and quality of life.

We use a combined endoscopic and fluoroscopic method in all of our patients as compared to a pure radiological method employed in some centres.[8] In literature the technical and clinical success rate is around ninety percent which are comparable to our study.[9]

Three of our patients (10%) had defective expansion of either the proximal or distal end of the stent as picked by a follow up X-ray on the second day of the procedure. They were either successfully balloon dilated (two cases) or re-stented (one case).

Seven of our patients (25%) had either a hepatic flexure tumour (3 cases) or a transverse colon lesion (4 cases). Although, the literature suggests more technical difficulties in proximal colonic lesions all of our right-sided stents were a technical and clinical success.[10] Also, once the lesion was reached we had successful guidewire cannulation of the stricture in all cases. This is better than those reported from other centres.[11]

We think our higher cannulation rate may be related to the use of sphincterotomes for tumours which are present on colonic bends and may be difficult to cannulate with a straight cannula or a wire. The angulation of a sphincterotome can be controlled in a graded manner and the tip directed to the axis of the tumour.

We employed uncovered stents in all of our cases except one as they less likely to migrate.[12] Two of our patients had stent migration after technically correct placement and one had distal migration after misplacement (10 %). Inappropriate patient selection as those with low grade obstruction and chemotherapy given post stent insertion with shrinkage of tumour mass are other causes of stent migration in literature.[13]

Obstruction of SEMS with tumour in growth and over growth can happen and is commonly treated with placement of a second stent through the first stent.[9,14] We have not encountered them in our study.

Bleeding is rare in SEMS placement.[15] One of our patient developed significant bleeding requiring transfusion but it settled on conservative management. Perforation is more common when pre-dilatation of the stricture is performed.[9] We did not routinely pre-dilate but had one case of early and one case of late perforation. Both cases did not have rescue surgery and died with comfort care. Our study, therefore, has a procedure-related mortality of seven percent.

SEMS are more cost effective when compared with emergency surgery.[16] Our study with a mean hospital stay of 2 days is comparable with short hospital stay following successful placement of SEMS. This is also supported by the previous New Zealand study which showed significantly reduced mean hospital stay in the stented patients.[4] We recommend SEMS deployment as a day-stay procedure in cases of uncomplicated SEMS insertion.

There is a clear role of SEMS as a definitive procedure in locally unresectable tumours presenting with acute large bowel obstruction. However, its role, as a bridge to surgery, in patients with a potentially curative disease is still debatable, with conflicting reports in literature. Clearly, the higher morbidity and mortality rates of emergency surgery, especially in left-side tumours, has to be balanced against the potential stent-related complications. Perhaps, the local expertise in each hospital and tumour characteristics (left versus right-sided tumours) should dictate the choice of intervention.

The limitation of our study was the retrospective design of the study. Data collected from the review of case notes has a potential for under-reporting of the complication rates.

Competing interests: None.

Author information: Mohammad I Khan, Adrian Claydon, Gastroenterologists, Tauranga Public Hospital, Tauranga

Correspondence: Dr Mohammad I Khan, Gastroenterologist, Tauranga Public Hospital, Private Bag 12024, Cameron Road, Tauranga, New Zealand. Email: imran.khan@bopdhb.govt.nz

References:

1. Arnell T, Stamos MJ, Takahashi P, et al. Colonic stents in colorectal obstruction. Am Surg. 1998;64:986-8.
2. Londono-Schimmer EE, Leong AP, Phillips RK. Life table analysis of stomal complications following colostomy. Dis Colon Rectum. 1994;37:916-920.
3. Athreya S, Moss J, Urquhart G, et al. Colorectal stenting for colonic obstruction: the indications, complications, effectiveness and outcome – 5-year review. Eur J Radiol. 2006;60(1):91-94.
4. Frizelle FA, Carne P, Robertson, G. Colonic stents. ANZ J Surg. 2003:73(Suppl. A17–CR23).
5. Syn WK, Patel M, Ahmed MM. Metallic stents in large bowel obstruction: experience in a District General Hospital. Colorectal Disease. 2004;7:22-26.
6. Dohmoto M. New method: endoscopic implantation of rectal stent in palliative treatment of malignant stenosis. Endosc Dig. 1991;3:1507-12.
7. Carter J, Valmedre S, Dalrymple C, et al. Management of large bowel obstruction in advanced ovarian cancer with intraluminal stents. Gynaecol Oncol. 2002;84:176-9.
8. Camunez F, Echenageusia A, Simo G, et al. Malignant colorectal obstruction treated by means of self expandable metal stents: effectiveness before surgery and in palliation. Radiology. 2000;216:492-7.
9. Khot UP, Lang AW, Murali K, Parker MC. Systemic review of the efficacy and safety of colorectal stents. Br J Surg. 2002;89:1096-102.
10. Keymling M. Colorectal stenting. Endoscopy. 2003;35:234-8.
11. Mainer A, De Gregorio MA, Tejero E, et al. Acute colorectal obstruction: treatment with self expandable metallic stents before scheduled surgery – results of a multicentre study. Radiology 1999;216:492-7.
12. Choo YW, Do YS, Suh SW, et al. Malignant colorectal obstruction: treatment with flexible covered stent. Radiol.1998;206:415-21.
13. Lopera JE, Ferral H, Wholey M, et al. Treatment of colonic obstruction with metal stents; indications, technique and complications. Am J Roentgenol. 1997;169:1285-90.
14. Baron TH, Dean YA, Yates MR, et al. Expandable metal stents for the treatment of colonic obstruction: technical outcomes. Gastrointest Endosc. 1998;47:277-85.

15. De Gregorio MA, Mainar A, Tejero E, et al. Acute colorectal obstruction: stent placement for palliative treatment – results of a multicentre study. Radiology. 1998;209:117-20.
16. Osman HS, Rashid HI, Sathananthan N, Parker MC. The cost effectiveness of self-expanding metal stents in the management of malignant left-sided large bowel obstruction. Colorectal Dis. 2000;2:233-7.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

"Punching above its weight": why New Zealand must maintain leadership in global health

Judith McCool, Chris Bullen

Abstract

As a small island nation, with a population of only 4.4 million, and geographically isolated from the centres of global power, New Zealand could be seen as of marginal relevance to the global health agenda. This paper argues that New Zealand has been and should remain a player in global health, even if current fiscal constraints may suggest otherwise. Involvement fits with our responsibilities and commitments in the Pacific region and our wider interests, including ethical international trade, security, global alliances and the fundamental protection of health

In this paper, we examine how, to the contrary, New Zealand has played a key role in influencing global health policy. In a time of fiscal constraint, where it might be tempting to reduce government-level involvement, we argue that small nations such as New Zealand can, and should, continue to exert influence where the opportunities arise.

New Zealand is a signatory to eight international human rights conventions or treaties including the first global health treaty, the World Health Organization (WHO) Framework Convention on Tobacco Control (FCTC). By signing and ratifying these instruments, successive governments have committed New Zealand to making progress to improve the rights and social outcomes, including health, of our people. Indeed, it is the often uncomfortable evidence of the adverse impacts of social and economic inequalities in health within our own populations that has underpinned the New Zealand position on key issues on the global arena.[1,2]

New Zealand has also played a key role in providing assistance to improve the health of its regional neighbours. This role has never been more relevant or acutely defined since the events of 2009, including the H1N1 pandemic, the economic crisis and global food crisis, and natural disasters.[3,4]

The current government gives strong priority to economic development and trade, but neither are achievable for any country without good health.[5]

International context

Globalisation involves the transfer and transgression of goods, services, images and ideas, impacting in diverse ways on health and health equity. As a member of the World Trade Organization, New Zealand has actively pursued globalisation through free trade agreements with larger economies. This has placed pressure on smaller trade partners to accelerate the introduction of trade liberalisation policies. However, such developments can lead to dramatic changes in patterns of work, income, lifestyle behaviours and ultimately in patterns of disease and injury.[3]

Adverse health impacts are expressed most acutely in settings which are the most poorly resourced to introduce regulatory safeguards. The nations of the Pacific region, for example, face an overwhelming increase in the prevalence of chronic non-communicable diseases (NCDs) such as heart disease, strokes, asthma and diabetes, almost entirely attributable to changes in diet, in tobacco and alcohol consumption and sedentary behaviour associated with urbanisation and globalisation.[6]

On the other hand, globalisation offers benefits, such as increased access to global export markets, generic pharmaceuticals and other useful goods and services. Indeed, globalisation has resulted in considerable income gains within those countries positioned to take advantage. However, there is also evidence that the economic benefits from globalisation disproportionately favour the already well-off.[4] The costs of such inequity are yet to be fully accounted for.

Good global health governance is vital to protect people from the risks posed by globalisation. Global health institutions such as the WHO have a mandate to formulate and promulgate health protecting policies to member states. Examples of such policies include the International Health Regulations, which focus on global communicable disease control[7] and the FCTC[8] discussed earlier.

International health security issues have received greater prominence as public health officials make preparations in anticipation of future pandemic diseases.[7] Severe acute respiratory syndrome (SARS) and more recently Influenza A (H1N1) have reinforced the validity of these concerns to human health in New Zealand and the need for New Zealand to be involved in global health policy and action.[9]

As a member of the WHO Executive Board from 2007–2010, New Zealand was able to advocate on behalf of the nations of the Pacific region to ensure that the issues most critical to the region featured on the global health agenda. In September 2011 New Zealand hosted the Pacific Islands Forum and in the same month, a New Zealand perspective on NCDs was heard at the United Nations Summit in New York. This was an extraordinary opportunity for New Zealand to have a voice on the current status and options for effective global action on NCD.[10]

Benefits for New Zealand

But what are the benefits for New Zealand to be part of a global health decision-making forum?

First, there is the power of presence: being at the table makes good sense when critical decisions are made that will directly and indirectly impact health through association with allied economies. Active participation at the World Health Assembly means that New Zealand can strengthen and support the Western Pacific Region's position on a range of critical health issues, particularly those that have direct consequences for the region, for example, climate change. Without presence, there is no voice and no influence on proposed actions.

Second, there are intangible but perhaps more sustainable benefits of global health investment for New Zealand. Since the WHO was established in April 1948 (following earlier initiatives in 1946), there has been a fundamental agreement on the collective benefits of international (cross-border) health security. The focus on international security remains as critical as ever.

For example, the International Health Regulations[11] adopted at the 2005 World Health Assembly were the result of a global agreement to upgrade the existing regulations that pre-date the threat of pervasive human health hazards. New Zealand played a critical role in supporting the introduction of the new regulations which were devised as a mechanism for surveillance and control of diseases or organisms which could threaten human health.[12] The introduction of international law is logical and sensible in the context of a globalised world which is attempting to achieve goals including the Millennium Development Goals,[13] reduce chronic non-communicable disease[10] and mitigate the impact of climate change.[14]

Current and future challenges

Consistent with WHO priorities for the Western Pacific Region, New Zealand's agenda for global health has been primarily focused on five areas: health systems strengthening, primary care, NCDs, improving aid harmonisation and global governance.[15] These areas were prioritised as being of critical importance to a region struggling with persistent preventable communicable disease as well as NCD and a depleted health care system. The outbreak of H1N1 in April 2009 prompted an immediate response from the New Zealand government to contribute to the global and regional response.[9] The previous outbreak of SARS was the warm-up for this new virus that threatened to spread swiftly.

However, we argue that a broader perspective on health and a continued interest in the welfare of our neighbours is needed in future. Since 1946, there has been a growing recognition towards recognising that health is not merely the absence of disease (or war), but the well-being within all communities.[2] Increasingly, health status is understood as a reflection of upstream factors, most of which lie outside the health sector (including trade, politics and workforce issues).[16]

What, for example, is the role of countries like New Zealand in negotiating workforce development and the push/pull factors which impact on vulnerable Pacific nations? The Pacific region is highly vulnerable to the "pull" of their health workforce to New Zealand and Australia where the income and living conditions are perceived to be an improvement but which has significant capacity issues for the local health services.

New Zealand is noted for "punching above its weight" on the global health stage, for example, through its acknowledged leadership on tobacco control.[17] New Zealand has a reputation for constructive participation, negotiation and skilful diplomacy at WHO and UN meetings. We support New Zealand governments in continuing to do this. It is neither practical nor ethical to view health as merely a concern within countries' own national borders. Acceptance of the responsibilities that accompany being in a globally connected world is a mark of New Zealand's willingness to play its part, as a relatively well-resourced leader in our region.

Lee Jong-Wook, Director-General of the WHO from2003–2006, stated "a world torn apart by gross health inequalities is in serious trouble ...the global health community can do much to reduce the suffering and death among vulnerable groups".[18] In 2008, the UK Government released a bold strategy, 'Health is Global'. That strategy set out to: "first, do no harm" through evaluating the impact of global trade agreements on global health and promoting outcomes that support the Millennium Development Goals.

Aside from worthy motives based on concerns for social justice and moral responsibility, the UK government clearly saw a number of benefits arising from its stance, including international trade, security and global alliances and the fundamental protection of health within the UK by "tackling the health challenges that begin outside [their] borders".[9]

Reducing health disparities, and reducing health care costs and economic degradation associated with health inequities, is one very powerful motivating force behind the rise and rise of global health investment—from both public and private accounts. Health inequities are one of the most significant threats within the Pacific region. As noted there is a rapidly escalating epidemic of chronic NCDs, environmental effects from climate change loom large and new and re-emerging infectious diseases threaten. More broadly, the region's economic status as a whole relies upon resource-rich nations to boost overall economic productivity which in the small Pacific islands is waning.[20] New Zealand and Australia both play a significant role as do China, Taiwan, Japan and Korea.

New Zealand is well positioned to support the Pacific region on these matters, through participation at the global health level. There is also a case based on enlightened self-interest. There are positive externalities for New Zealand if we contribute building healthy global policy in the Pacific and beyond. These extend beyond the impacts on export trade on the Pacific, with the sustainable management of fisheries, the environment and regional security all having direct implications for New Zealand.

Minister of Foreign Affairs and Trade, Hon Murray McCully stated in a speech to the Centre of Strategic and International Studies in May 2011: "*Our economic prospects are deeply intertwined with those of the rapidly growing countries of Asia. Yet both our makeup and our geography give us an increasing involvement in and responsibility for the future stability and security of the Pacific. I often refer to the former as our zone of opportunity and the latter as our zone of responsibility*".[21] The message is clear New Zealand is committed to the Pacific region.

However, it is the ways this commitment is expressed in terms of scope, policy and practice that is of concern. In our view New Zealand should ensure strategies are not only relevant to the people of the region but should incorporate a health determinants perspective and continue to build on our well-deserved reputation as a good global citizen and regional leader. We should not and cannot afford to do otherwise.

Competing interests: None.

Author information: Judith McCool, Senior Lecturer, Global Health; Chris Bullen, Director, Clinical Trials Research Unit; School of Population Health, University of Auckland

Correspondence: Dr Judith McCool, Global Health, School of Population Health, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand. Email: j.mccool@auckland.ac.nz

References:

1. Tobias M, Blakely T, Matheson D, et al. Changing trends in indigenous inequalities in mortality: lessons from New Zealand. Int J Epidemiol. 2009;38(6):1711-1722. http://ije.oxfordjournals.org/content/38/6/1711.abstract

2. Crampton P, Hoek J, Beaglehole R. Leadership for health: developing a canny nanny state. N Z Med J 2011;124(1329):66-72. http://journal.nzma.org.nz/journal/124-1329/4528/content.pdf
3. Fa'alili-Fidow J. Ensuring economic, health and social wellbeing for Papua New Guinea through trade. Asia Pacific Journal of Public Health, 2011;23(1):79-85.
4. Schrecker T, Labonte R, De Volgli R. Globalisation and health: the need for a global vision. Lancet 2008;372(9650):1670-1676.
5. Wyber R, Wilson N, Baker M. New Zealand's impact on health in the South Pacific: scope for improvement. N Z Med J. 2009;122(1291). http://journal.nzma.org.nz/journal/122-1291/3505/content.pdf
6. Lower T, Nauan B, Abel M, et al. Curbing the tide – Non-communicable disease in the Pacific. Pac Health Dialog 2005;12(2):61-64.
7. Baker M, Forsyth A. The new International Health Regulations: a revolutionary change in global health security. N Z Med J 2007;120(1267). http://journal.nzma.org.nz/journal/120-1267/2872/content.pdf
8. WHO. Framework Convention on Tobacco Control. Geneva, World Health Organization, 2003.
9. Jennings L. Influenza A(H1N1)09: another public health risk to New Zealand. N Z Med J 2009. 22(1298). http://journal.nzma.org.nz/journal/122-1298/3700/content.pdf
10. Beaglehole R, Bonita R, Horton R, et al. Priority actions for the non-communicable disease crisis. The Lancet 2011;377(9775):1438-1447.
11. WHO. International Health Regulations. Geneva, World Health Organization, 2005.
12. WHO. Medium Term Strategic Plan 2008-2013 (amended draft), 2009.
13. United Nations Development Programme. Millennium Development Goals. 2000 [cited 2011 23 May 2011]; Available from: http://www.un.org/millenniumgoals/
14. McMichael AJ, Neira M, R Bertollini, et al. Climate change: a time of need and opportunity for the health sector. The Lancet, 2009;374(9707):2123-2125.
15. Edgar W. Global Health; why New Zealand needs to be involved. In Otago International Health Research Network Conference, Dunedin, 2010:
16. Marmot M. Working through the issues of global governance for health. The Lancet 2009;374(9697):1231-1232.
17. Edwards R, Thomson G, Wilson N, et al. After the smoke has cleared: evaluation of the impact of a new national smoke-free law in New Zealand. Tob Control 2008;17(e2).
18. Jong-Wook L. Global health improvement and WHO: shaping the future. The Lancet, 2003;362(9401): 2083-2088.
19. Primarolo D, Malloch-Brown M, Lewis I. Health is global: a UK government strategy for 2008-13. The Lancet 2009;373:443-444.
20. Bank AD. Pacific Economic Monitor. In Pacific Monitor, Bank AD, Editor, 2011.
21. McCulley M. Speech to the Centre of Stragetic and International Studies. Wellington, New Zealand, 2011.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Hypnosedative access and risk of harm

David B Menkes, Lucy M Shieffelbien, Mark Huthwaite

Abstract

Aim To review PHARMAC's decision, effective 1 September 2010, to remove the 1-month restriction on funded prescription of hypnotics and anxiolytics.

Method We consider the evidence for an association between access to these medicines and risk of harm.

Results Prescription volumes and reported harms have both increased over the last decade in New Zealand; available studies and clinical experience suggest a causal link. Preliminary data collected since PHARMAC's funding change suggest an exacerbation of the problem.

Conclusion The decision to relax funding restrictions on hypnosedatives is expected to increase drug-related harms in a sub-population of users. Improved pharmacovigilance could inform policy regarding these agents.

Based on a recommendation from the Pharmacology and Therapeutics Advisory Committee (PTAC), the New Zealand drugs purchasing agency PHARMAC has decided to lift a longstanding funding restriction on hypnotics and anxiolytics. As of 1 September 2010, the 1-month reimbursement limit per prescription was lifted to 3 months, in accord with most other funded medicines.[1]

As is customary for its funding and access decisions, PHARMAC invited submissions on this proposed policy change. The Royal Australian and New Zealand College of Psychiatrists, together with a number of addiction specialists, expressed concern that removal of the monthly funding restriction would likely increase the availability, overuse, and abuse of these compounds. However realistic these concerns might have been, PHARMAC decided to proceed with the change anyway.

PHARMAC's reasons for lifting the reimbursement restriction include bringing funding policy in line with prescribing regulations, as determined by the New Zealand government's drugs regulator MedSafe, which has always allowed 3-month prescriptions but required monthly dispensing of the Class C medicines in question.

PHARMAC's argument is technically correct, but seems to disregard the valuable role that its historical funding restriction has played in prompting regular clinical review and thereby arguably limiting supply and overuse of these drugs. While it is uncertain how much drug-related harm this 'anomalous' policy may have prevented over the years, it seems plausible that harms are set to increase with the new, easier access to these medicines.

Overseas evidence indicates that prescribing restrictions can dramatically reduce benzodiazepine supply and related problems[2] but there is as yet little direct evidence that relaxing restrictions has the opposite effect. Moreover, PTAC and others have

argued that PHARMAC's historical reimbursement restriction has in some cases compromised the ability of competent prescribers to use these drugs effectively.

Benzodiazepines are class C5 controlled drugs in New Zealand (www.medsafe.govt.nz/profs/class/classification.asp), with common adverse effects including psychomotor and cognitive impairment and corresponding risk of accidents and falls.[3–5]

Less common but serious side-effects include mood disorder, disinhibition, suicide and violence, particularly in combination with alcohol or other central nervous system depressants.[6–8] Because of a tendency to induce pharmacodynamic tolerance, their effectiveness as hypnotics and anxiolytics wanes with repeated administration and longer-term use is generally contraindicated.[9]

Tolerance to benzodiazepines, together with a euphoriant effect in many individuals, confers further risks in terms of abuse and dependence.[10–13] These problems extend to clonazepam and clobazam, used mainly as anticonvulsants, and to the chemically distinct benzodiazepine agonist zopiclone[14,15] but not to buspirone, which is marketed as an anxiolytic but has no street value or abuse potential.[16,17]

Outcome studies indicate an increased risk of inappropriate benzodiazepine use and of adverse outcomes when prescribing is not regularly reviewed; this appears particularly important during the first 4–6 weeks after initiation.[18–21] Prescribing guidelines in many countries, including NZ, recommend short term prescribing wherever possible; the decision to continue beyond 2–4 weeks should be well documented and reviewed at regular intervals.[20,22–26]

From 2002 to 2010, PHARMAC data indicate that prescriptions for hypnosedatives have increased steadily—an average of 7% annually, with comparable increases in the anxiolytic and hypnotic subgroups (5.5% and 8.0% respectively). The overall growth in prescription volume is driven largely by two agents: zopiclone (13.4% average annual increase) and lorazepam (9.4% increase). Zopiclone is now by far the most frequently prescribed hypnotic in New Zealand with over 560,000 prescriptions during the year ending June 2010. What effect have these increases had on rates of problems and what further effect might be expected from PHARMAC's reimbursement policy change?

Unfortunately, existing pharmacovigilance systems in New Zealand are fragmented and do not allow reliable and comprehensive detection of such harms. One opportunity for 'joined-up' detection, the Chemical Injury Surveillance System was launched in 2001, but has yet to be developed in this role regarding prescription medicines, and remains limited to certain regions of the country. As a consequence, the best available current indices are indirect, notably notifications to the National Poisons Centre (www.poisons.co.nz/); these provide a limited index of one set of harms that can result from prescription of hypnosedatives.

Other important categories of harm, notably cases presenting to hospitals, are inconsistently recorded and very few notified to the Poisons Centre or the Centre for Adverse Reactions monitoring (http://carm.otago.ac.nz/). Referrals to alcohol and drug services also provide an indication of problems, but because of regional variation in funding and access cannot be used to estimate problem rates, let alone changes in these over time.

Even though an underestimate of overall harms, Poisons Centre notifications thus provide an important index of prescription drug problems across New Zealand. Calls to the Centre are carefully recorded and can be analyzed by drug, intent, age, and other variables. In addition to self-poisoning, these include non-intentional events including the important and frequent category 'child exploratory'.

With regard to hypnosedatives, the data indicate an upward trend since 2002 for both anxiolytic and hypnotic subgroups, with average annual increases of 8.9% and 7.2% respectively. It is notable that these figures are of the same magnitude and appear to parallel the growth in prescription volumes described above. Over the seven years to 30 June 2009, a total of 2707 calls related to hypnosedative drugs were recorded by the Poisons Centre; during the same period 5,919,693 prescriptions were recorded. The overall crude rate of 45.7 notifications per 100,000 prescriptions is similar for anxiolytics (47.3) and hypnotics (45.1).

More compelling from the standpoint of causality is the observation of a similar rank order of drugs with regard to prescription volume and poisoning notifications (zopiclone followed by lorazepam and diazepam).

In summary, available data indicate a continuing rise in both hypnosedative prescribing and one set of drug-related harms in New Zealand. The parallel growth in these two measures strongly suggests, but does not prove, a causal link between them. Nonetheless, such a link is plausible, in line with available research, and consistent with clinical experience here and overseas.

In light of this, PHARMAC's decision to lift the 1-month funding restriction for these drugs seems likely to aggravate an already serious set of problems. Funded repeat prescriptions beyond 1 month inevitably means that clinical review is less likely to occur, and ongoing supply correspondingly more likely. Preliminary trends observed since PHARMAC's policy adjustment include, for example, no change in the continuing 8–10% annual increase in the number of patients prescribed zopiclone (183,000 in the year ending 30 June 2011) but a rather larger 18% jump in the number of subsidised tablets dispensed (20,232,000) [N.B. the latter figure over-estimates actual supply increases since an unknown number of patients had previously paid for repeats that would not be counted in PHARMAC data prior to September 2010]. More worrying is a corresponding 25% increase (from 210 to 263) in Poison Centre notifications regarding zopiclone over 12 months since the change.

A further increase in problems can be expected, especially in the surprisingly large group with mental disorder and a comorbid tendency to substance misuse.[27] These individuals both supply and consume hypnosedatives available 'on the street', and it can reasonably be expected that illicit New Zealand supplies—already identified as a problem[28]—will grow as a consequence of PHARMAC's funding change. Further difficulty is particularly anticipated from patients who obtain prescriptions from more than one doctor.

PHARMAC has made it clear that its reimbursement policy has now been brought into line with Medsafe's prescribing regulations for hypnosedatives, and it is up to the latter authority to review and amend these as required. The question then becomes: how much evidence of harm will Medsafe require to do this? Unfortunately, available

pharmacovigilance systems are insufficient to fully define the extent of the problem and its expected escalation with the recent change in funding policy.

Should Medsafe develop vigilance systems and await definitive evidence, or act now on the limited signals available? The fact that 3 months' funded supply of hypnosedatives is now available from a single prescription constitutes, we believe, an unnecessary and avoidable risk to public health.

Competing interests: None.

Author information: David B Menkes, Associate Professor, Waikato Clinical School, University of Auckland, Hamilton; Lucy M Shieffelbien, National Poisons Centre, Preventive and Social Medicine, University of Otago, Dunedin; Mark Huthwaite, Senior Lecturer in Psychological Medicine, School of Medicine, University of Otago, Wellington

Acknowledgements: We thank PHARMAC for access to data and Drs Gavin Cape, Paul Glue, Rose Neild and Geoff Robinson for discussions.

Correspondence: David Menkes, Waikato Clinical School, Private Bag 3200, Hamilton 3240, New Zealand. Fax: +64 (0)7 8398712; email: david.menkes@waikatodhb.health.nz

References:

1. Pharmaceutical Management Agency. New Zealand Pharmaceutical Schedule – Update effective 1 September 2010. Wellington: New Zealand Government; 2010.
2. Brahams D. Benzodiazepine overprescribing: successful initiative in New York State. Lancet. 1990;336:1372-3.
3. Glass J, Lanctôt KL, Herrmann N, et al. Sedative hypnotics in older people with insomnia: meta-analysis of risks and benefits. BMJ. 2005;331:1169-73.
4. Golombok S, Moodley P, Lader M. Cognitive impairment in long-term benzodiazepine users. Psychol Med. 1988;18:365-74.
5. Tata PR, Rollings J, Collins M, et al. Lack of cognitive recovery following withdrawal from long-term benzodiazepine use. Psychol Med. 1994;24:203-13.
6. Mallon L, Broman J-E, Hetta J. Is usage of hypnotics associated with mortality? Sleep Med. 2009;10:279-86.
7. Neutel CI, Patten SB. Risk of suicide attempts after benzodiazepine and/or antidepressant use. Epidemiol. 1997;7:568-74.
8. Menkes DB. Triazolam-induced nocturnal bingeing with amnesia. Aust N Z J Psychiatry. 1992;26:320-1.
9. Menkes DB. Hypnosedatives and anxiolytics. In: Dukes MNG, editor. Meyler's Side Effects of Drugs. 14th ed. Amsterdam: Elsevier; 2000. p. 121-38.
10. Committee on the Safety of Medicines. Benzodiazepines, dependence and withdrawal symptoms. Current problems. 1988;21:1-2.
11. Lader M. Benzodiazepines – The Opium of the masses? Neuroscience. 1978;3:159 65.
12. Maletzky BM, Klotter J. Addiction to Diazepam. Int J Addiction. 1976;11:95-115.
13. Schweizer E, Rickels K. Benzodiazepine dependence and withdrawal: a review of the syndrome and its clinical management. Acta Psychiatr Scand (Suppl.) 1998;89:1455-9.
14. Bannan N, Rooney S, O'Connor J. Zopiclone misuse: an update from Dublin. Drug Alc Rev. 2007;26:83-5.
15. Hajak G, Müller WE, Wittchen HU, et al. Abuse and dependence potential for the non-benzodiazepine hypnotics zolpidem and zopiclone: a review of case reports and epidemiological data. Addiction. 2003;98:1371-8.

16. Cole JO, Hecht Orzack M, et al. Assessment of the abuse liability of buspirone in recreational sedative users. J Clin Psychiatry. 1982;43:69-74.
17. Lader M. Assessing the potential for buspirone dependence or abuse and effects of its withdrawal. Am J Med. 1987;82(5, Suppl. 1).
18. Ghosh Nodiyal A, Ahuja N. Audit of pattern of benzodiazepine prescribing in a community mental health team in England. Eur Psychiat. 2009;24(Suppl. 1):S543.
19. Huthwaite M. An Audit of Benzodiazepine Prescribing. Quality in Health Care Conference; 1995 April; South Africa.
20. Lader M, Russell J. Guidelines for the prevention and treatment of benzodiazepine dependence: summary of a report from the Mental Health Foundation. Addiction. 1993;88:1707-8.
21. Naidoo K, Bohra M, Mathurine O. Mother's little helper: prescribing practice of anxiolytics and hypnotics in a community mental health team. Eur Neuropsychopharmacol. 2009;19(Suppl. 3):S605-S6.
22. American Psychiatric Association. Task force report on benzodiazepine dependence, toxicity, and abuse. Washington: American Psychiatric Association. 1990.
23. Chief Medical Officer. Benzodiazepines warning. A communication to all doctors from the Chief Medical Officer. CMO's Update. 2004;37(January 2004).
24. Committee on Review of Medicines UK. Systematic review of the benzodiazepines. British Medical Journal. 1980;2:719-20.
25. Royal Australian and New Zealand College of Psychiatrists. College Guidelines for the Use of Benzodiazepines. . RANZCP Practice Guidelines #5. 1991.
26. Royal College of Psychiatrists. Benzodiazepines: Risks, Benefits or Dependence. A Re-Evaluation. R C Psych Council Report. 1997;CR 59.
27. Clark RE, Xie H, Brunette MF. Benzodiazepine prescription practices and substance abuse in persons with severe mental illness. J Clin Psychiatry. 2004;65:151-5.
28. Ministerial Committee on Drug Policy. National Drug Policy 2007–2012. Wellington: New Zealand Government; 2007. p. 33.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Colonoscopy—a rare cause of pancreatitis

Manar Khashram, Frank A Frizelle

Colonoscopy is a common procedure for investigation for colonic pathology either on the basis of symptoms or screening for abnormality. Complications are uncommon—but iatrogenic, harmful to patients, and potentially the subject to complaints. It is important to have a good understanding of what the complications are, how often they occur and how they occur. There are a few well recognised complications such as perforation, bleeding and postpolypectomy syndrome, however there are also a number of less well recognised complications reported such as infection, splenic rupture, having the snare caught on a large polyp, benign pnemoperitioneum, diverticulitis, appendicitis. In this report we outline another rare complication, that of post colonoscopy pancreatitis.

Case report

A 77-year-old otherwise fit man underwent a colonoscopy to investigate a change in bowel habit. His normal medications included aspirin 100 mg once daily and simvastatin 20 mg nocte for primary cardiovascular prevention. He had no known drug allergies. He was a non-smoker and had a minimal alcohol intake. The procedure itself was uncomplicated and no tissue sampling was undertaken. He was sedated for the procedure with 100 mcg of fentanyl and 5 mg of midazolam intravenously.

The patient developed an acute onset of central abdominal pain 4 hours post procedure and presented to our acute surgical service. On physical examination, the patient had a pulse of 72 beats per minute, blood pressure of 130/70 mmHg and a temperature of 37°C. His abdomen was tender on palpation centrally but did not exhibit signs of peritonitis.

Investigations revealed an amylase 1258 U/L, lipase 4248 U/L, C-reactive protein (CRP) 11 nmol/L, gamma-glutamyl transferase 542 U/L, aspartate aminotransferase 484 U/L, alanine aminotransferase 460 U/L, and alkaline phosphatase 92 U/L. Calcium, phosphate, albumin and protein levels were normal and fasting cholesterol was 5.3 mmol/L.

Radiological investigations included a transabdominal ultrasound showed a normal biliary system. A magnetic retrograde pancreatic cholangiogram (MRCP) showed inflammation of the pancreas (Figure 1). A computed tomography (CT) on day 4 revealed a thickened pancreas consistent with inflammation and extensive mesenteric stranding and fluid collections (Figure 2).

He was managed with intravenous fluids, analgesia and clear fluid diet escalating to normal diet over the initial days. The amylase and lipase returned to normal levels and the CRP peaked at 272 nmol/L on day 4. He was discharged on day 12 post presentation in stable condition, the amylase was 40 U/L, lipase was 85 (8–78) U/L and CRP 96 nmol/L. On 3-month follow up, the patient has returned to his normal activities and had no further episodes of abdominal pain.

Figure 1. MRI showing high-signal intensity abnormality resembling haemorrhagic fluid (arrow)

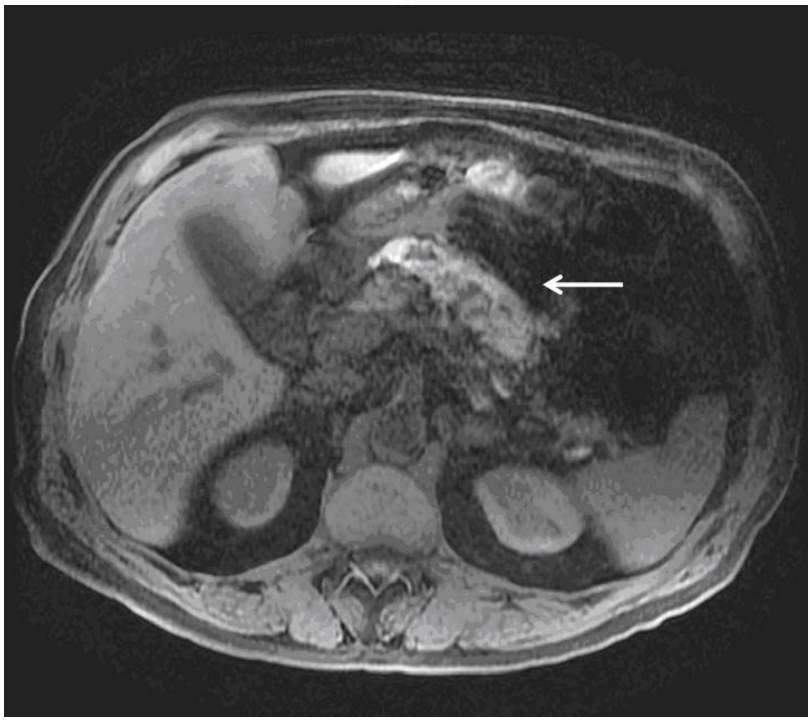
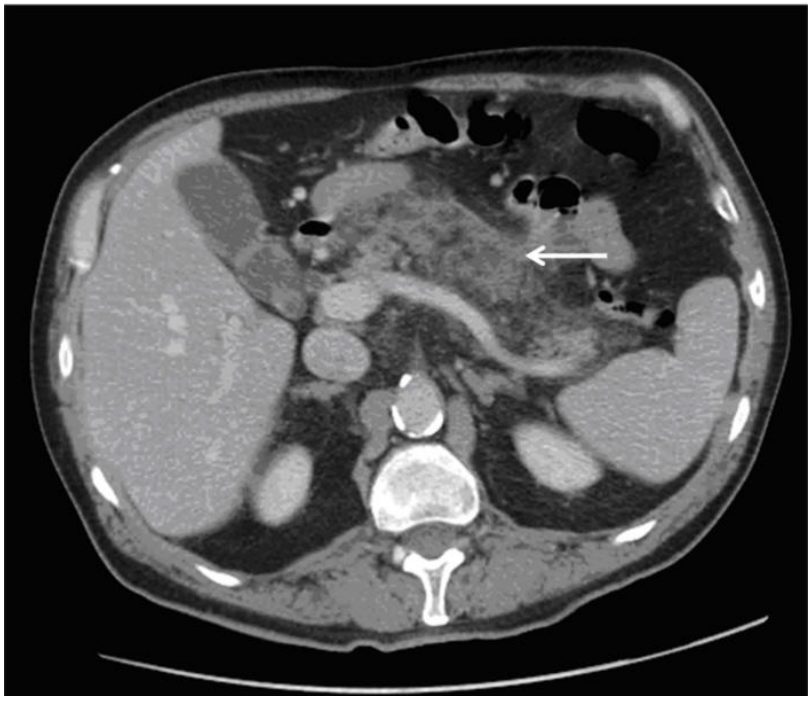


Figure 2. CT on day 4 showing pancreatitis with adjacent stranding and small fluid collections (arrow)



Discussion

Serious complications following colonoscopy are well documented in the literature. The risk of bleeding increases when tissue sampling is performed and the risk of colonic perforation is low, generally quoted in the <1% figures. Other serious but rare complications of colonoscopy includes splenic trauma and sepsis.

The two common aetiologies causing approximately 80% of acute pancreatitis are gallstone migration and alcohol abuse.[1] The remaining causes include metabolic derangement, medications, anatomical anomalies and trauma.

A literature search revealed only two case reports of patients pancreatitis following colonoscopy.[2,3] In both these cases, the procedure was reported to be difficult around the splenic flexure and multiple attempts to insert the endoscope were made. This was not the case with our patient.

Given the pancreas appearance on the CT and MRI being consistent of haemorrhagic likely to be due to the trauma. The proposed hypothesis is mechanical trauma to the body and tail of the pancreas caused by the mechanical entry of the endoscope and possibly air insufflation around the splenic flexure and transverse colon.

It is very unlikely that the simvastatin was the cause of the pancreatitis as the patient has been on treatment for more than 3 months. Statin-induced pancreatitis occurs rarely[4] and most of the published case studies reported a short duration from the time the medication was started to the pancreatitis. In addition, the patient was re-challenged with simvastatin during the admission and his symptoms did not worsen.

Abdominal pain post colonoscopy is a relatively common symptom and is frequently benign. Patients presenting to emergency services should have investigations to rule out perforation. Once perforation has been ruled out and depending on the clinical presentation, pancreatitis caused by the procedure should be suspected.

With the increase use of endoscopy in diagnostic and therapeutic colonic conditions, clinicians should be aware of such complications to initiate early diagnosis and treatment.

Author information: Manar Khashram, Surgical Registrar; Frank A Frizelle, Colorectal Surgeon; Department of General Surgery, Christchurch Hospital, Christchurch

Correspondence: Dr Manar Khashram, Department of Surgery, Christchurch Hospital, PO Box 4345, Christchurch 8014, New Zealand. Fax: +64 (0)3 3640352; email: manar.khashram@gmail.com

References:

1. Frossard JL, Steer ML, Pastor CM. Acute pancreatitis. Lancet 2008;371:143–52.
2. Thomas AW, Mitre RJ. Acute pancreatitis as a complication of colonoscopy. J Clin Gastroenterol 1994;19:177-8.
3. Ko HH, Jamieson T, Bressler B. Acute pancreatitis and ileus postcolonoscopy. Can J Gastroenterol 2009;23(8):551-553.
4. Nitsche CJ, Jamieson N, Lerch MM, Mayerle JV. Drug induced pancreatitis. Best Prac Res Clin Gastroenterol 2010;24:143-55.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Incarceration of an inguinal hernia post urinary catheterisation

Phil T Davey, Eunice J Minford

Case report

An 83-year-old gentleman presented to the emergency department complaining of lower abdominal pain extending into his back since the previous day. The attending clinician was concerned that the gentleman was in acute urinary retention, with a palpable bladder and inserted a 16Ch Foley urinary catheter. Clear urine was obtained.

The patient was referred surgically for investigation and admission. The patient had a past medical history of ischaemic heart disease, with a previous myocardial infarction, and a degree of congestive cardiac failure as well as being in atrial fibrillation. He also suffered from Paget's disease. His previous surgical history included bilateral inguinal hernia repairs and a transurethral resection (TUR) of the prostate for benign hyperplasia.

The admitting surgical team noted on examination a tense, tender, irreducible lump in the right inguinal region beneath the old scar. The patient was able to inform the team that he had a recurrence of his inguinal hernia on the right side, but elective surgery had been declined due to his extensive co-morbidities.

The provisional diagnosis of an incarcerated, recurrent inguinal hernia was made. In light of his abdominal pain extending into the back, as well as concerns regarding the patient's fitness for surgery due to extensive comorbidities, a CT scan was organised, prior to surgical exploration of the right groin.

CT scanning of the abdomen and pelvis revealed the tip and the inflation balloon of the urinary catheter to be present in the right inguinal hernia sac, with part of the bladder (Figures 1 & 2).There was no evidence of large or small bowel in the hernia. A wedge compression fracture of T11 vertebral body was identified which accounted for his back pain, as well as sacral changes in keeping with Paget's disease.

The catheter was immediately deflated by the surgical team and withdrawn out of the hernia, but not removed. The lump instantly disappeared and the discomfort in the inguinal region settled. The gentleman's symptoms improved, the catheter was removed and he was passing urine comfortably. Elective repair of the hernia was deemed inappropriate due to his extensive comorbidities. His back pain settled with analgesia and he was subsequently discharged home.

Figure 1. Sagittal view showing the Foley catheter and the urinary bladder extend out through the inguinal region, as well as the wedge fracture of the T11 vertebra

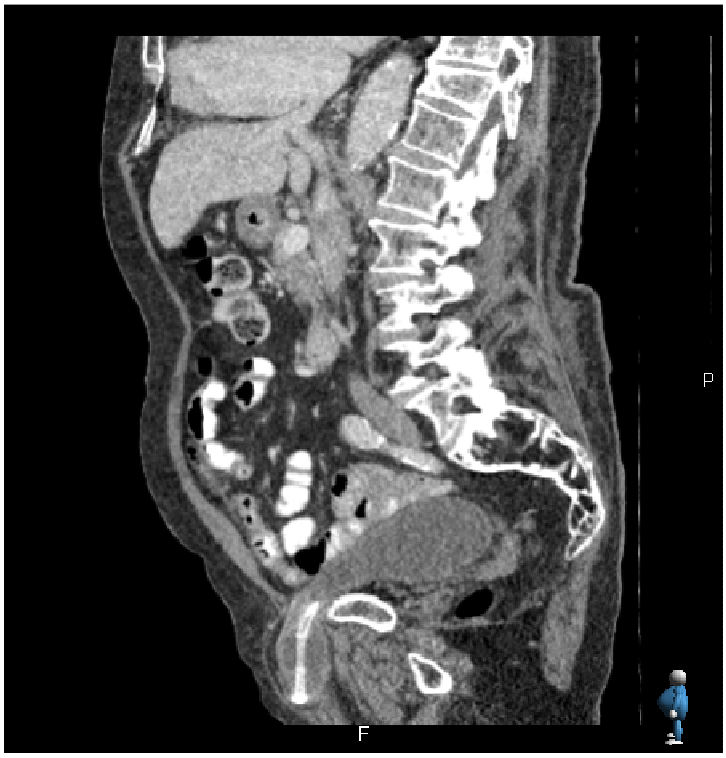
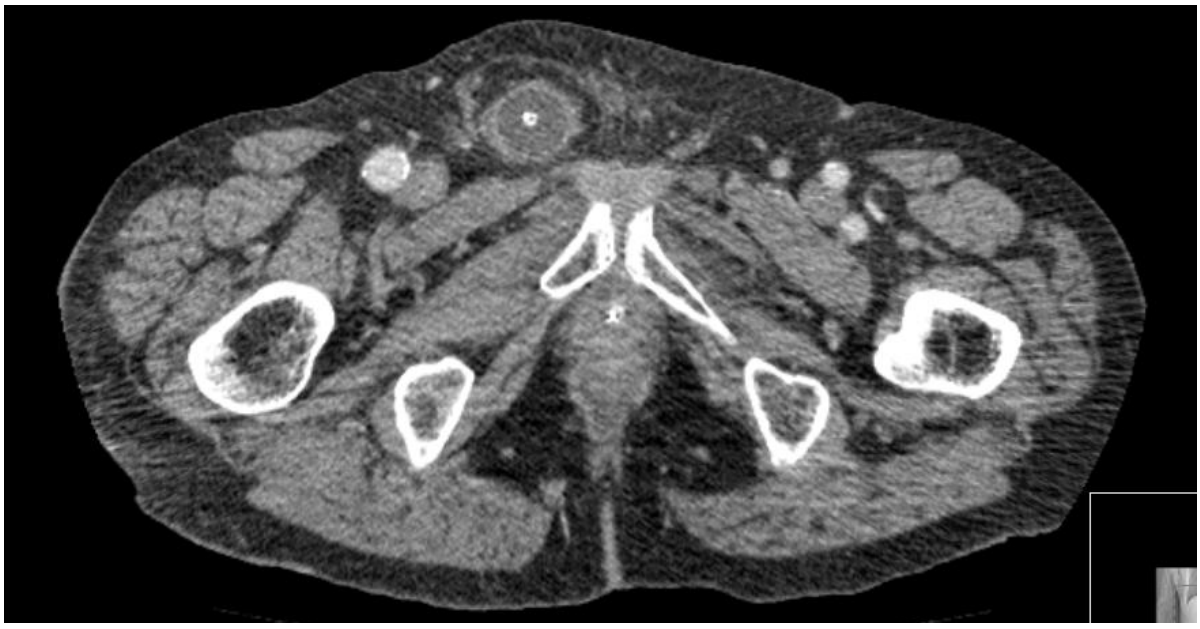


Figure 2. Transverse view showing the balloon of the Foley catheter in the right inguinal hernia



Discussion

Inguinal hernias can contain a wide variety of intra-abdominal organs, such as the ovaries, fallopian tubes, appendix, as well as large and small bowel.[1] Occasionally herniae present with unusual intra-abdominal pathology, such as metastatic peritoneal disease[2], pseudomyxoma peritonei syndrome[3] and also adenocarcinoma of the bowel[4]. There are many well described eponyms for inguinal hernias, such as Amyand's hernia containing the appendix, or Littre's hernia containing a Meckel's diverticulum.

Sliding inguinal hernias involving the bladder are relatively rare, with estimates ranging between 1-4% of all inguinal herniae.[5,6] Large herniations of the bladder into the scrotal sac have been termed 'scrotal cystocoeles'.[7] The literature reveals one case report of a male with a scrotal mass who needed to manually compress his scrotum in order to void.[8]

Many factors can contribute to the presence of the urinary bladder in a hernia, including urinary outlet obstruction causing chronic bladder distension and contact of the bladder wall with the hernial orifices as well as loss of bladder tone[9]. These are all features that may have been present in this patient as he had previously undergone a TUR of prostate for obstructive symptoms.

This case demonstrates the need to be vigilant when placing a urinary catheter in an individual with a history of groin hernias and to be suspicious of an acute hernia in catheterised patients. If we had proceeded to surgical exploration on clinical findings alone, the patient would have undergone an arguably unnecessary urgent operation and been exposed to significant anaesthetic risks given his poor cardiac status. It also highlights the potential benefit that CT scanning can provide in cases with an unusual clinical history and presentation.

Author information: Phil T Davey, Specialist Registrar General Surgery, Department of General Surgery; Eunice J Minford, Consultant General Surgeon; Antrim Area Hospital, Antrim, Northern Ireland

Correspondence: Phil T Davey, Department of General Surgery, Antrim Area Hospital, Bush Road, Antrim BT41 2RL, UK. Email: phildavey@doctors.net.uk

References:

1. Gurer A, Ozdogan M, Ozlem N, et al. Uncommon content in groin hernia sac. Hernia. 2006;10(2):152-5.
2. Nicholson CP, Donohue JH, Thompson GB, Lewis JE. A study of metastatic cancer found during inguinal hernia repair. Cancer. 1992;69:3008–11.
3. Esquivel J, Sugarbaker PH. Clinical presentation of the pseudomyxoma peritonei syndrome. Br J Surg. 2000;87(10):1414-8.
4. Boormans JL, Hesp WL, Teune TM, Plaisier PW. Carcinoma of the sigmoid presenting as a right inguinal hernia. Hernia. 2006;10(1):93-6.
5. Curry NS. Hernias of the urinary tract. In: Pollack HM, McClennan BL. Clinical urography, 3rd ed. Philadelphia, PA: Saunders, 2000:2981–2991.
6. Bisharat M, O'Donnell ME, Thompson T et al. Complications of inguinoscrotal bladder hernias: a case series. Hernia. 2009;13(1):81-4.
7. Levine B. Scrotal cystocele. JAMA 1951;147:1439–41.

8. Bjurlin MA, Delaurentis DA, Jordan MD, Richter HM 3rd. Clinical and radiographic findings of a sliding inguinoscrotal hernia containing the urinary bladder. Hernia. 2010;14(6):635-8.
9. Bacigalupo LE, Bertolotto M, Barbiera F et al. Imaging of Urinary Bladder Hernias. AJR. 2005;184:546–551.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

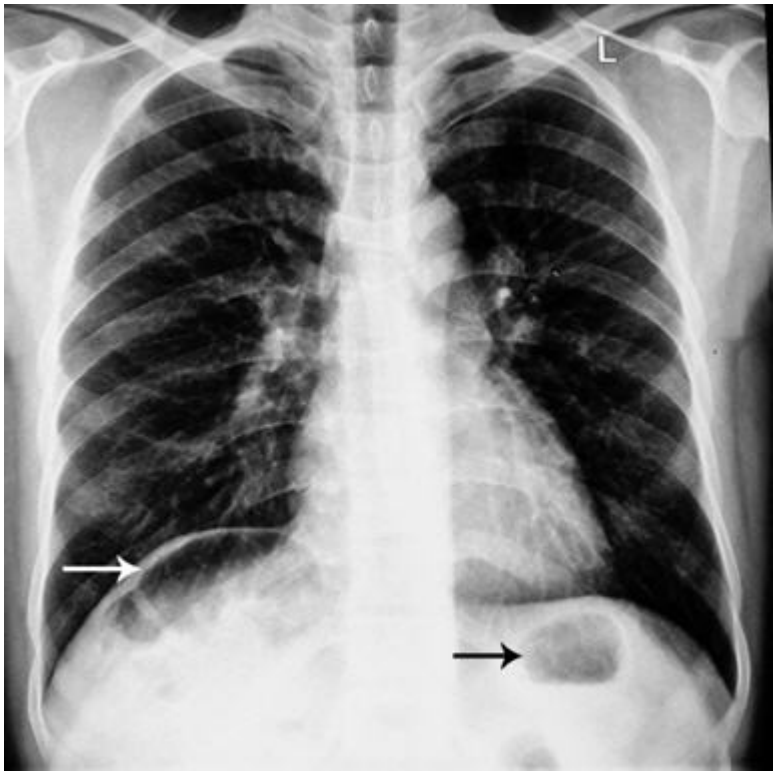
An unusual cause of nonresponsive chronic dyspnoea

Prem P Gupta, Dipti Agarwal

Clinical

A 38-year-old male, a smoker (28 pack years), was referred to our Institute with history of chronic dyspnoea having poor response to inhaled bronchodilators. Erect chest radiographs were performed and the posteroanterior view is shown (Figure 1).

Figure 1. Chest radiograph posteroanterior view, showing lucency (white arrow) under right hemidiaphragm consistent with loop of colon interposed between right hemidiaphragm and liver. Over left side, air-fluid level in stomach below left hemidiaphragm is distinctly visible (black arrow).



What is the diagnosis?

Answer

The findings of gas under the right hemidiaphragm with haustral markings are suggestive of a colonic loop interposed between right hemidiaphragm and liver—the findings are described as *Chilaiditi's sign*. As the patient was also having clinical symptoms, a diagnosis of *Chilaiditi syndrome* was made.

Discussion

Chilaiditi's sign was initially described by a Greek radiologist Demetrius Chilaiditi while working in Vienna in 1910. Chilaiditi sign is characterised by the transposition of a loop of large intestine (usually hepatic flexure of transverse colon) in between the right hemidiaphragm and the liver. It is often detected incidentally on a plain abdominal radiograph or chest radiograph.[1] The sign may be seen permanently or intermittently. Chilaiditi sign associated with clinical symptoms like shortness of breath,[2] abdominal pain,[3] and torsion of the bowel[4] is described as *Chilaiditi syndrome.*

The exact cause for this disorder remains elusive. This is seen in around 0.1–0.25% of chest radiographs; more often in males and almost always in adults though isolated reports in children are present.[2] The association with chronic obstructive pulmonary disease, cirrhosis, ascites etc. has been described. Other factors predisposing to it may include absence of normal suspensory ligaments of the transverse colon, abnormality of the falciform ligament, paralysis or eventration of the right hemidiaphragm, aerophagia and redundant colon, as might be seen with chronic constipation or in bedridden individuals. It can also be associated with relative atrophy of the medial segment of the left lobe of the liver.

This anatomical variant is sometimes mistaken for pneumoperitoneum and may lead to unnecessary surgical interventions. The presence of haustral folds can accurately establish that the air beneath the diaphragm is contained within large bowel. Left lateral decubitus abdominal films may help when confusion is not resolved.

In most instances, the treatment is largely conservative and may comprise of bed-rest, fluid supplementation, nasogastric decompression, enemas, high-fibre diets and stool softeners.[6]

Author information: Prem P Gupta, Professor, Respiratory Medicine; Dipti Agarwal, Assistant Professor, Physiology; Postgraduate Institute of Medical Sciences, University of Health Sciences, Rohtak, India

Correspondence: Professor Prem Parkash Gupta, 9J/17, Medical Campus, PGIMS, Rohtak, India, PIN-124001. Email: gparkas@yahoo.co.in

References:

1. Saber AA, Boros MJ. Chilaiditi's syndrome: what should every surgeon know? Am Surg. 2005;71:261–3.
2. Keles S, Artac H, Reisli I, et al. Chilaiditi syndrome as a cause of respiratory distress. Eur J Pediatr. 2006;165:367–9.
3. Glatter RD, April RS, Miskovitz P, Neistadt LD. Severe recurrent abdominal pain: an anatomical variant of Chilaiditi's syndrome. MedGenMed. 2007;9: 67.

4. Plorde JJ, Raker EJ. Transverse colon volvulus and associated Chilaiditi's syndrome: case report and literature review. Am J Gastroenterol. 1996;91:2613–6.
5. Sanyal K, Sabanathan K. Air below the right diaphragm: Chilaiditi sign. Emerg Med J. 2008;25;300.
6. Risaliti A, De Anna D, Terrosu G, et al. Chilaiditi's syndrome as a surgical and nonsurgical problem. Surg J Gynecol Obstet. 1993;176:55–8.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Xanthogranulomatous pyelonephritis

Habib U Rehman

Clinical

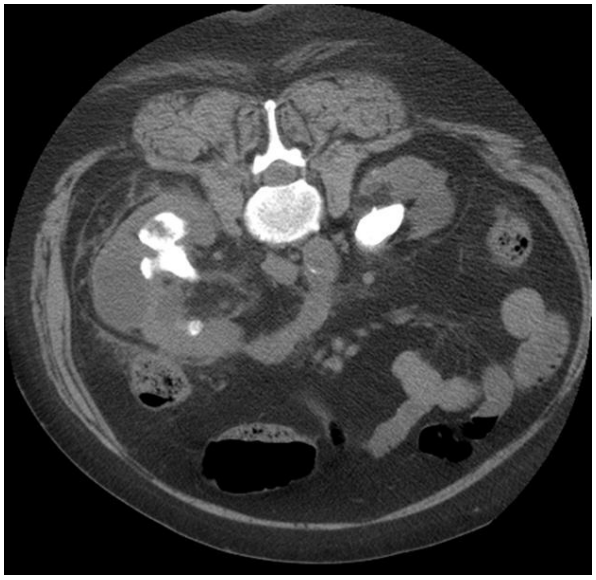
A 60-year-old woman was admitted with history of nausea, vomiting, lethargy and dizziness of 1 week's duration. She denied any history of dysuria, polyuria or abdominal pain. She had no previous history of renal calculi or urinary tract infection.

Following blood results were obtained: sodium 130 mmol/L (135–145), potassium 7.6 mmol/L (3.5–5.0), chloride 104 mmol/L (98–110), anion gap 7 mmol/L (10–20), urea 76.6 mmol/L (3.0–7.1), and creatinine 1421 µmol/L (60–130). Arterial blood gas analysis revealed a pH of 7.14 (7.35–7.45), pCO_2 of 22 mmHg (33–45), pO_2 of 133 mmHg and a base excess of -20.3 whilst inhaling oxygen at 3 L/min.

Urinanalysis showed large amounts of blood and was positive for nitrite and Ca500. Renal ultrasound showed a large lobulated staghorn calculus in the right kidney and replacement of the right renal parenchyma with multiple round hypoechoic lesions. The left kidney had similar appearances however the calcification in the renal pelvis was smaller measuring 1.4 cm. The round hypoechoic lesions also appeared smaller and somewhat less numerous. *Escherichia coli* grew in urine culture. Ciprofloxacin was prescribed.

Computerised tomographic (CT) scan showed bilateral obstructive staghorn calculi. Left kidney was atrophic and both kidneys were largely replaced by circular communicating low density fluid collections (see Figure 1).

Figure 1



Discussion

Xanthogranulomatous pyelonephritis (XGP) is a rare chronic pyelonephritis caused by previous urinary tract obstruction and recurrent infection. The renal parenchyma is destroyed and replaced by lipid laden foamy macrophages. Vast majority of patients are middle-aged women and present with fever, flank or abdominal pain, anorexia, weight loss and lower urinary tract symptoms.[1] In 90% of cases a single kidney is diffusely affected.

Involvement of both kidneys is exceedingly rare.[2] *Escherichia coli* and *Proteus mirabilis* are the most common organisms.[3] Diagnosis is usually suggested by CT scan findings, which typically shows renal enlargement, calcification filling the renal pelvis, and replacement of the renal parenchyma by multiple hypodense areas.[4]

Treatment usually involves antibiotics and total or partial nephrectomy. Nephrectomy is usually done for advanced disease. Focal or bilateral XGP should be treated with antibiotics particularly if the patient is not a surgical candidate because of other comorbid conditions. Antibiotics are also required pre- and postoperatively in surgical patients.

Author information: Habib U Rehman, Clinical Assistant Professor, Department of Medicine, Regina Qu'Appelle Health Region, Regina General Hospital, Regina, Saskatchewan, Canada

Correspondence: Dr HU Rehman, Clinical Assistant Professor, Department of Medicine, Regina Qu'Appelle Health Region, Regina General Hospital, 1440 – 14th Avenue, Regina, SK, S4P 0W5, Canada. Email: habib31@sasktel.net

References:

1. Korkes F, Favoretto RL, Bróglio M, et al. Xanthogranulomatous pyelonephritis: Clinical experience with 41 cases. Urology 2008;71:178-80.
2. Perez LM, Thrasher JB, Andeson EE. Successful management of bilateral xanthogranulomatous pyelonephritis by bilateral partial nephrectomy. J Urol 1993;149:100-2.
3. Malek RS, Elder JS. Xanthogranulomatous pyelonephritis: a critical analysis of 26 cases and of the literature. J Urol 1978;119:589-93.
4. Loffroy R, Guiu B, Watfa J, et al. Xanthogranulomatous pyelonephritis in adults: clinical and radiological findings in diffuse and focal forms. Clin Radiol 2007;62:884-90.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Potential social and psychological consequences of the *Rena* incident: lessons from an international perspective

The grounding of the *Rena* on the Astrolabe Reef in the Bay of Plenty and the consequences of the oil-spill are of great concern to local communities, and throughout the rest of New Zealand. International research on the environmental effects of similar maritime incidents—e.g. the *Sea Empress* in Milford Haven, Wales, UK in 1996[1] enables helps us to form a view on the possible resulting psychosocial concerns and consequences.

The *Sea Empress* is a useful case comparison as it released 480t of heavy fuel oil into the waters of southwest Wales when grounded, as well as 72,000t of blended crude oil. This heavy fuel oil is similar to the load carried by the *Rena*, and reacts quite differently to water than lighter crude oils. Lighter fractions of oil tend to form a sheen on water which evaporates relatively quickly. However, heavy fuel oil reacts very differently, by forming large, thick globules that become difficult to clear up, especially in colder waters.

Unlike most natural disasters, industrial or technological disasters such as this tend to create chronic uncertainty over an extended period, especially concerning health effects, economic impacts, extent of ecological damage and recovery, issues of fair and just compensation, sociocultural recovery, explanation and closure. Psychological stress may be heightened for individuals and communities who are more vulnerable due to their connections to threatened or damaged resources.[2] This increased stress may show itself in everyday life, including disruption to daily routines, family life , work, and future plans. For some family members after the *Exxon Valdez* oil spill in 1989, this showed itself over time through increased drug and alcohol usage, elevated levels of domestic violence, feelings of helplessness, betrayal, anger, anxiety, and depression, as well as acute physical symptoms such as sore eyes, sore throat and self-reported headaches.[3,4]

As well as uncertainty, technological disasters also tend to highlight issues associated with exposure to hazard. Thus, interventions focusing on protecting the health of individuals being exposed to hazard become critical in response to these incidents. The hazard exposure risks should be carefully and clearly articulated. Training and appropriate protective equipment should be issued, and protective behaviours encouraged whilst balancing the need to harness the often considerable community impetus to contribute to their own protection and recovery.

Perceived breach of trust is likely to be a critical factor in dealing with the aftermath of the Rena incident. The concept of recreancy is defined as, "the failure of experts or specialized organizations to execute proper responsibilities to the broader collectivity with which they have been implicitly or explicitly trusted".[5] This relates to institutions entrusted to protect the public, to control technology, and respond to crises.

Society generally believes that technology should and can be controlled, and a failure to do so can erode this trust. Often, technological disasters have a 'primary responsible

party'[2]—even though other organisations may share some responsibility—that provides focus for blame and anger, as well as frustration, hostility, and calls for compensation. This is a live process for the *Rena* clean-up and recovery that may have consequences for levels of confidence in social institutions, and may also affect how risks concerning further technological projects may be viewed (e.g. offshore oil exploration).

A belief that there has been a failure to protect the public can create a perception of increased, uncontrolled risk and threat to personal and economic security, as what was perceived to be safe and controlled is revealed not to be so. Individuals and communities may reconsider the status of what was previously thought to be safe as well as the worth of those perceived as culpable in permitting the risk to become uncontrolled, and may also become anxious and wary about heightened levels of perceived threat.

Issues concerning insurance liabilities and compensation after technological disasters have in some cases been tied up for years after the event, which creates another significant layer of uncertainty. Longitudinal evidence seems to indicate that much of chronic stress, anxiety and disruption as result of *Exxon Valdez* disaster were a byproduct of prolonged litigation.[6] Prompt resolution of issues regarding insurance cover and other possible litigation for affected communities will be critical in moving things forward, whilst balancing the need to protect blamed individuals from community anger.

Recovery from the *Rena* incident depends very much upon how it continues to unfold. Like southwest Wales and the effects of the *Sea Empress* incident, the Bay of Plenty is a region of remarkable conservation and tourism interest, and supports diverse fishery and renewable resource industries. If the effects of the oil spill are longer lasting, there is potential for disruption of social capital for example, through migration from the affected region due to lack of economic opportunities, or fears for health.

The nature and extent of impacts may be tied to how the incident continues to affect both domestic and international tourism, based on attractive beaches and recreational boating and fishing activities, as well as local communities' ability to make use of these amenities and the farming of renewable resources. Perceptions of tourists and local communities that the beaches and water are safe will be pivotal in ensuring renewal of traditional economic activities in the area, as well as reassuring potential consumers of seafood products. Trusted intermediaries will be needed to provide assurance about potential health impacts including air and water quality, consequences of the impact of heavy fuel oil and dispersants on the local ecology, and seafood safety to verify that standards have been met. Strong customary and spiritual ties with land and sea may result in a possibly greater and longer lasting economic impact as well as other consequences.

Taking a broader perspective, the *Rena* incident has the potential to place strain upon budgets designed to meet need for local everyday services (e.g., increase in police calls, council expenditure, resolving community conflicts, increased call upon health resources), and to further stretch national resources at time of fiscal challenge. Furthermore, the *Rena* incident occurs at a challenging time for New Zealand, with considerable resource allocated to recovering from the Canterbury earthquakes. Wider

community perceptions may be that New Zealand has had its fair share of disasters over past months. Yet it is clear that an opportunity for communities to demonstrate strengths and resilience is being firmly grasped in response to the *Rena* grounding.

Volunteering to help in the acute phase clean up is commendable as long as people are enabled to participate in this while protecting themselves and others from needless exposure to risk in their eagerness to assist. Dissatisfaction with a perceived lack of action by authorities led to individuals taking matters into the own hands and beginning to clean up without appropriate clothing of safety instruction. Rapid coordinated action and good communication are critical in empowering communities to take appropriate, safe action. Longer-term planning also needs urgent consideration as the initial groundswell of interest moves on to other issues.

In sum, organisations tasked with leading the response to, and recovery from the *Rena* incident would be well served in attending to the different kinds of community response after a technological disaster. The rapid adoption of appropriate health protection measures, a transparent process aiming for the prompt resolution of insurance and litigation concerns, and the rebuilding of trust are likely to significantly influence long-term outcomes.

Associate Professor Sarb Johal
Chair of the Psychosocial Recovery Advisory Group, Joint Centre for Disaster Research, Massey University / GNS Science, Wellington

Ron Chambers
Clinical Psychology Professional Advisor & Consultant Clinical Psychologist, Anxiety Disorders Unit, Specialist Mental Health Services, Canterbury District Health Board, Christchurch

Susan Collins
Research Officer, Joint Centre for Disaster Research, Massey University / GNS Science, Wellington

Ian de Terte
School of Psychology, Massey University, Wellington

Dr Dianne Gardner
School of Psychology, Massey University, Auckland

Professor Bruce Glavovic
EQC Chair in Natural Hazards Planning; Associate Director of the Joint Centre for Disaster Reasearch, Palmerston North

Professor Lucy Johnston
Dean of Postgraduate Research, Canterbury University, Christchurch

Professor A Nuray Karanci
Department of Psychology, Middle East Technical University, Turkey

Maureen F. Mooney
Research Officer, Join Centre for Disaster Research, Massey University / GNS Science, Wellington

Professor Douglas Paton
School of Psychology, University of Tasmania, Australia

Professor David Johnston
Director, Join Centre for Disaster Research, Massey University / GNS Science, Wellington

References:

1. Law RJ, Kelly C. The Impact of the "Sea Empress" oil spill. Aquat. Living Resour. 2004;17, 389-394.
2. Gill DG, Picou JS, Ritchie LA. The *Exxon Valdez* and BP oil spills: A comparison of initial social and psychological impacts. American Behavioral Scientist. 2011;August 5, 0002764211408585, first published on August 5, 2011 doi:10.1177/0002764211408585
3. Lyons RA, Temple JMF, Evans D, et al. Acute health effects of the *Sea Empress* oil spill. J Epidemiol Community Health. 1999;53:306-310.
4. Picou JS, Marshall BK, Gill DA. (). Disaster, litigation and the corrosive community. Social Forces 2004;82(4):1493-1522.
5. Freudenburg WR. The "risk society" reconsidered: Recreancy, the division of labor, and risks to the social fabric. In Cohen, MJ. (Ed.), Risk in the modern age: Social theory, science and environmental decision-making. New York, NY: St Martin's Press; 2000. p. 107-122.
6. Picou JS, Martin CG. Long-term impacts of the *Exxon Valdez* oil spill: Patterns of social disruption and psychological stress seventeen years after the disaster. Report prepared for the National Science Foundation. Mobile: University of South Alabama; 2007.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

End-of-term review of the New Zealand Government's response to climate change: a public health perspective

Near the end of each electoral term seems an appropriate time to review the New Zealand Government's response to major threats to health. Climate change is a very serious problem[1,2] and is an important threat to international health[3] as well as health in New Zealand (NZ).[4,5] Actions to both mitigate climate change and to start to adapt to a more disrupted future climate are becoming critical. Such measures can be conceptualised as forms of "catastrophe insurance".[6]

We therefore searched government websites (as per other work[5]), and examined media releases by the "Minister for Climate Change Issues" covering the January 2009 to mid-October 2011 period.[7] An analysis of particularly significant new responses and progress in what we consider are the top five domains for action, is shown in Table 1 below.

Table 1. Evaluation of new responses by the New Zealand Government in the 2009 to 2011 electoral term that relate to climate change

Key domain (prioritised)	New responses (reducing greenhouse gas emissions and adaptation)	Summary around progress
Contributing to international action	Despite some positive work on agricultural greenhouse gas abatement internationally,[8] the Government has not taken a leadership role in pushing forward international treaties on controlling greenhouse gases (GHGs), committing to stronger emissions reductions, or transferring appropriate technology to developing countries. While current international arrangements have major limitations,[9] the NZ Government has *not*: Adopted an appropriate or ambitious goal for long-term GHG emission reduction. Instead, it has adopted a "50% by 2050" goal which is inconsistent with the scientific evidence on the scale of emission reductions that well-off countries need to make to contain climate change risks.[10 11] Explored the promotion of alternatives to slow-moving international arrangements (e.g., the alternative of having "carbon clubs" involving a small number of countries which are major emitters[9] or which can potentially apply taxes at the oil wellhead[12]); Explored direct relationships with major developing country emitters (e.g. as per Norway working with Indonesia and Brazil to reduce deforestation[13]). Advocated strongly for other OECD countries to eliminate their fossil fuel subsidies (worth tens of billions of dollars[14]). Indeed, work in these areas will probably be impeded by the marked downsizing of the workforce in the Ministries of the Environment and Foreign Affairs.[15] Nevertheless, a small plus is NZ support for renewable energy projects in the Pacific (e.g., Tonga[16]).	Very limited. Useful initiative in agricultural GHGs, but weak overall emissions target for 2050 adopted.
Giving price signals to the market	NZ's carbon price under the ETS is currently only NZ$12.50 per tonne of CO_2. This can only be described as inadequate, given that the 'social cost' of carbon is more likely to be in the hundreds of dollars.[17] Even though NZ's emission trading scheme (ETS) at inception was weak,[18] the Government passed legislation in 2009 to weaken it further. The	Mixed – some ongoing support for insulating homes, but backwards movement on the ETS

Key domain (prioritised)	New responses (reducing greenhouse gas emissions and adaptation)	Summary around progress
	International Energy Agency (IEA) noted in 2010 that "there is no guarantee that the NZ ETS, with all its administrative burdens, will result in absolute emissions reductions".[19] The Parliamentary Commissioner for the Environment's submission to the 2011 ETS review noted that NZ's emissions are on course to exceed by 30% its pledged level for 2020 (of 10% to 20% reductions).[20] In 2011, the Government signalled that it will further slow or postpone the entry of agriculture into the ETS, and slow the rate at which the ETS price steps up for sectors already included.[21 22] Nevertheless, a positive aspect of having some form of ETS is that it may be making it easier for the Australian Government to pass a law for a carbon tax in 2012, and move towards its own ETS and related equity policies.[23] The cooperation between the countries on such schemes is also promising.[24] The Government cancelled a planned fuel tax increase in 2011,[25] and government resources allocated to support fossil fuel exploration have been substantially increased.[26] In terms of housing, however, the Government has continued to support subsidies on insulating homes through its "Warm Up New Zealand" programme, which has now been delivered to over 100,000 homes. This has been shown to achieve health gains in NZ[27] and is highly cost-beneficial.[28] Other subsidies are however, very minor (e.g., to solar water heating, electric cars and for biofuels).	
Supporting domestic R&D (e.g., renewable energy)	The Government has continued to support research and development (R&D) on reducing agricultural methane and nitrous oxide.[29 30] But as far as we can ascertain, there has been no signal from government for research funders to specifically prioritise funding in the areas of: renewable energy, and energy efficiency. For example, the Ministry of Science and Innovation's Statement of Intent 2011-2014 does not mention renewable energy, energy efficiency or climate change.[31] Nevertheless, this Ministry has signalled some helpful investments in research areas such as "Hazards and Infrastructure", e.g. in "better urban design and development, and resilient infrastructure".[32] None of the Health Research Council investment signals relate to the health impacts of climate change or to related issues around sustainability.[33] Only one grant (out of 19) for technology development in 2011 was energy efficiency related (energy-efficient light bulbs).[34]	Limited, some potential
Supportive regulation and policy development	A national policy statement on renewable electricity generation was introduced, giving greater weight to the national interest in meeting the government's 90% renewable electricity target. However, there has been no substantive progress on strengthening regulations for: improved energy efficiency of housing stock and appliances; and vehicle advertising (which generally lack information on efficiency and emissions[35]). Work on a "regulated vehicle fuel economy standard" (in progress prior to the election) was abandoned in August 2009.[36] There was also little evident progress on national-level regulations to reduce urban sprawl (work was folded into reform work on the Resource Management Act) and a moratorium on new thermal power stations (introduced by the previous government), was lifted.[26]	Some support given to renewable energy.
Supportive infra-structure investment	The Government is investing in public transport improvements (e.g., rail services in Wellington and Auckland[37]) which might favour long-term emissions reductions. There has also been additional government support for the development of recreational cycleways, and for two pilot local initiatives to improve cycling and walking.[38] Nevertheless, the budget for encouraging walking and cycling has been trimmed. Overall, the focus on and budget for public transport, cycling and walking is very modest compared to funding for roading in recent government agency plans.[39]	Mixed – good on some aspects of infrastructure, but problematic roading focus

Key domain (prioritised)	New responses (reducing greenhouse gas emissions and adaptation)	Summary around progress
	Furthermore, the IEA in 2010 described the growth in the transport sector as the country's "biggest energy-saving challenge", and described policies for the sector as "vague" and unclear.[19] One development that may indirectly lower emissions is the Government's major investment in ultra-fast broadband infrastructure[40] (e.g., with recent roll-out in various regions). The rebuilding necessary after the Christchurch earthquake has provided an opportunity for replacement buildings to be built to the higher current Building Code,[41] but has not been taken as an opportunity to retrofit existing housing to a higher energy standard as this is considered non-allowable "betterment".	

In summary, in this last electoral term there appears to have been little substantive progress by the current government on reducing greenhouse gas emissions (via work internationally or domestically), despite government targets (2020 and 2050) requiring material action. Government responses towards *adapting* to climate change impacts seem to be even more deficient (hardly more than some guidance documents[42]). This lack of attention may be considered to be very serious given the potential size of the climate change threat – to public health and for the whole of society. It can also be considered economically wasteful in that the New Zealand economy is placed at increased risk of having to make a more abrupt and disorderly transition in the future. Also if other nations react to this lack of response by imposing carbon tariffs on New Zealand exports, this could also have serious economic consequences given the economy's dependence on trade.

A more detailed review of the New Zealand Government's performance in the area would encompass many other aspects (some of which we have discussed elsewhere[43] [44]). A more extensive review could also make comparisons with the previous government. We briefly note, however, that the previous government also made relatively poor progress in most of the areas tabulated above, albeit with more action on public transport and a stronger version of the ETS.

A reason for the lack of progress by serial New Zealand governments in addressing climate change may reflect concern around the costs of action and also a lack of appreciation of the co-benefits to health.[45] There is also the role of vested commercial interests in influencing policy (as can be seen with the country's food sector[46]), and a strong focus by politicians on crises e.g., most recently the global financial crisis and the Christchurch earthquake. Nevertheless, these are not sufficient reasons for inaction, given the serious and potentially catastrophic nature of climate change.

Nick Wilson,[1*] Ralph Chapman,[2] Philippa Howden-Chapman[1]

1. Department of Public Health, University of Otago, Wellington, New Zealand
2. Environmental Studies, Victoria University of Wellington, Wellington, New Zealand

*Correspondence: nick.wilson@otago.ac.nz

References:

1. Committee on Stabilization Targets for Atmospheric Greenhouse Gas Concentrations; National Research Council. Climate stabilization targets: Emissions, concentrations, and impacts over decades to millennia. Washington, DC: National Academies Press; 2010. Available from: http://www.nap.edu/openbook.php?record_id=12877&page=R1
2. IPCC. Climate Change 2007: Impacts, Adaptation and Vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge, UK: Cambridge University Press, 2007. http://www.ipcc.ch/ipccreports/ar4-wg2.htm
3. McMichael AJ, Lindgren E. Climate change: present and future risks to health, and necessary responses. J Intern Med. 2011;270:401-13.
4. Metcalfe S, Woodward A, Macmillan A, et al. Why New Zealand must rapidly halve its greenhouse gas emissions. N Z Med J. 2009;122:72-95.
5. Wilson N, Slaney D, Baker M, et al. Climate change and infectious diseases in New Zealand: A brief review and tentative research agenda. Rev Environmental Health 2011;26(2):93-99. http://www.reference-global.com/doi/abs/10.1515/REVEH.2011.013
6. Aldred J. Ethics and climate change cost-benefit analysis: Stern and after. New Political Economy. 2009;14:469-488.
7. Smith N. Hon Dr Nick Smith: Releases [3 December 2008 to 21 September 2011]. http://www.beehive.govt.nz/minister/nick-smith/release
8. Global Research Alliance on Agricultural Greenhouse Gas Research. http://www.globalresearchalliance.org/
9. Victor D. Global warming gridlock: Creating more effective strategies for protecting the planet. Cambridge, UK: Cambridge University Press, 2011.
10. Chapman R, Boston J. 'Gazetting New Zealand's 2050 Emissions Target': Submission on New Zealand's 2050 greenhouse gas emissions target. Wellington: Victoria University of Wellington, 2011.
11. Anderson K, Bows A. Beyond 'dangerous' climate change: emission scenarios for a new world. Philos Transact A Math Phys Eng Sci. 2011;369:20-44.
12. Davis S, Peters G, Caldeira K. The supply chain of CO2 emissions. Proc Natl Acad Sci U S A. 2011;[E-publication 17 October 17]. doi:10.1073/pnas.1107409108
13. Ministry of the Environment. The Government of Norway's International Climate and Forest Initiative. http://www.regjeringen.no/en/dep/md/Selected-topics/climate/the-government-of-norways-international-.html?id=548491
14. Vidal J. World Bank: ditch fossil fuel subsidies to address climate change. The Guardian. 2011;(21 September). http://www.guardian.co.uk/environment/2011/sep/21/world-bank-fossil-fuel-subsidies?newsfeed=true
15. Vance A. 'Timebomb' set as public service jobs are slashed. Dominion Post. 2011;(10 September):A5.
16. APNZ. $8m for Tongan solar power plant. N Z Herald. 2011;(6 September). http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=10749747
17. Ackerman F, Stanton E. Climate risks and carbon prices: Revising the social cost of carbon. Portland: Economics for Equity and the Environment, 2011.
18. Bertram G, Terry S. The carbon challenge: New Zealand's emissions trading scheme. Wellington: Bridget Williams Books, 2010.
19. International Energy Agency (IEA). Energy Policies of IEA Countries: 2010 Review: New Zealand. Paris: OECD/IEA, 2010.
20. Wright J. Emissions Trading Scheme Review. Wellington: Parliamentary Commissioner for the Environment, 2011.
21. Emissions Trading Scheme Review Panel. Doing New Zealand's fair share. Emissions Trading Scheme Review 2011: Final Report. Wellington: Ministry for the Environment, 2011.

http://www.climatechange.govt.nz/emissions-trading-scheme/ets-review-2011/review-report.pdf

22. Fallow B. Farmers off hook in ETS review. N Z Herald. 2011;(19 September). http://www.nzherald.co.nz/politics/news/article.cfm?c_id=280&objectid=10752678
23. Howden-Chapman PL, Chapman RB, Capon AG, et al. Carbon pricing is a health protection policy. Med J Aust.2011;195:311-2.
24. Smith N, Groser T. Progress made on trans-Tasman carbon market [Media Release, 2 August 2011]. http://www.beehive.govt.nz/release/progress-made-trans-tasman-carbon-market
25. Joyce S. This year's fuel tax increase cancelled [Media release, 26 April 2011]. http://www.beehive.govt.nz/release/year%E2%80%99s-fuel-tax-increase-cancelled-0
26. Brownlee G. Opening Address to the New Zealand Petroleum Conference. [Presentation, 20 September 2010]. http://www.beehive.govt.nz/speech/opening-address-new-zealand-petroleum-conference
27. Howden-Chapman P, Matheson A, Crane J, et al. Effect of insulating existing houses on health inequality: cluster randomised study in the community. BMJ. 2007;334:460.
28. Chapman R, Howden-Chapman P, Viggers H, et al. Retrofitting houses with insulation: a cost-benefit analysis of a randomised community trial. J Epidemiol Community Health. 2009;63:271-7.
29. Pastoral Greenhouse Gas Research Consortium (PGgRc). Research. http://www.pggrc.co.nz/Research/tabid/40/Default.aspx
30. Ministry for the Environment. New Zealand's Fifth National Communication under the United Nations Framework Convention on Climate Change. Wellington: Ministry for the Environment, 2009.
31. Ministry of Science and Innovation. Statement of Intent 2011-2014. Wellington: Ministry of Science and Innovation, 2011. http://www.msi.govt.nz/sites/all/files/u4/MSI%20SOI%202011-14.pdf
32. Ministry of Science and Innovation. Ministerial notice for Hazards and Infrastructure Research. Wellington: Ministry of Science and Innovation, 2011. http://www.msi.govt.nz/funding/upcoming/hazards
33. Health Research Council. Projects. http://www.hrc.govt.nz/funding-opportunities/researcher-initiated-proposals/projects
34. Ministry of Science and Innovation (MSI). Technology Development Grant 2011 - Overview of recipients. http://www.msi.govt.nz/business/tdg/2011
35. Wilson N, Maher A, Thomson G, et al. Vehicle emissions and consumer information in car advertisements. Environ Health. 2008;7:14.
36. Joyce S. Govt won't proceed with fuel economy standard [Media release, 28 August 2009]. http://www.beehive.govt.nz/release/govt-won%E2%80%99t-proceed-fuel-economy-standard
37. Joyce S. Auckland to get 50% more electric trains [Media release, 1 September 2011]. http://www.beehive.govt.nz/release/auckland-get-50-more-electric-trains
38. NZ Transport Agency (NZTA). Walking and cycling model communities announced. Wellington: NZTA, 2010. http://www.nzta.govt.nz/about/media/releases/725/news.html
39. Ministry of Transport. Government Policy Statement on Land Transport Funding. Wellington: Ministry of Transport, 2011. http://www.transport.govt.nz/ourwork/KeyStrategiesandPlans/Documents/GPS-July-2011.pdf
40. Crown Fibre Holdings Limited. Ultra-fast broadband. http://www.crownfibre.govt.nz/ultra-fast-broadband.aspx
41. Howden-Chapman P, Chapman P, Abrahamse W, et al. Christchurch's Regeneration: research and science-based insights Wellington: New Zealand Centre for Sustainable Cities, 2011.
42. Smith N. New guidance for councils on preparing for climate change. [Media release, 7 May 2010]. http://www.beehive.govt.nz/release/new-guidance-councils-preparing-climate-change
43. Wilson N, Chapman R, Howden-Chapman P. New Zealand Government response to climate change: largely fogged up? N Z Med J. 2009;122(1303):111-3.

44. Howden-Chapman PL, Chapman RB, Capon AG, et al. Carbon pricing is a health protection policy. Med J Aust. 2011;195:311-2.
45. Wilson N, Chapman R, Clover D, et al. A farsighted energy strategy would benefit health in New Zealand. N Z Med J.123(1322):92-5.
46. Jenkin GL, Signal L, Thomson G. Framing obesity: the framing contest between industry and public health at the New Zealand inquiry into obesity. Obes Rev. 2011;[E-publication 13 August].

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Banning pharmaceutical sponsorship: is ethical apartheid the right road ahead?

We read with a heavy heart the letter by the New Zealand Medical Students' Association (NZMSA) in the 14 October 2011 edition of the *NZMJ* titled "A policy of no pharmaceutical sponsorship: a case for health equity." Before responding to the points made in the letter though, I must declare my conflict of interest in that I am an employee of a multinational pharmaceutical company. However, it is not in my professional capacity that I wish to respond, but as a Kiwi and father.

NZMSA set out an argument for disengagement from what they describe as the pernicious influence of the pharmaceutical industry. The segregation policy which they espouse has the connotation of ethical apartheid where you are prejudged based not on one's actions but on who employs you. The pharmaceutical industry has room to improve in terms of ethical engagement based on the common good of patients, but this ought not to be a beauty pageant where we endeavour to separate the self righteous team from those of supposed moral turpitude.

Lest we forget the skeletons in the medical profession's ethical wardrobe: the cases of sexual impropriety with vulnerable patients;[2] fraud (28 prosecutions between 1994 and 2002);[3] tax avoidance;[4] and misrepresentation of clinical results,[5] to name but a few.

My own son, Zachary, a fourth-year medical student, died of meningococcal septicaemia in 2009. After more than 2 years of fighting for truth and justice, the Auckland District Health Board finally offered a public apology for Zachary's substandard care and for failing to investigate and openly disclose. This is sadly one of many cases of medical misadventure annually.

We are all stakeholders in the health system with something valuable to offer. We all make ethical (and other) errors of judgement from time to time. Together we can be stronger but ethical apartheid will only weaken us all. As the good book says: before you take the splint out of your neighbour's eye first take the log out of your own.

The NZMSA appears to also imply that the existence of a state monopsony purchaser such as PHARMAC is an important tool in achieving national health equity. Unfortunately this hypothesis is unreferenced. PHARMAC's statutory obligation to provide the most benefit for the greatest number within a fixed resource is the epitomy of utilitarianism. It has long been recognised that not only is utilitarianism not dependent upon equity but indeed is inconsistent with it.[6]

Lance Gravatt
Marketing Company President
AstraZeneca Limited
Auckland

References:

1. Chao PP. A policy of no pharmaceutical industry sponsorship: a case for health equity [letter]. N Z Med J 124(1344). http://journal.nzma.org.nz/journal/124-1344/4916/content.pdf
2. Doctor admits sexual misconduct with patient. http://www.voxy.co.nz/national/doctor-admits-sexual-misconduct-patient/5/25438
3. Auckland doctor found guilty of fraud. http://www.scoop.co.nz/stories/GE0203/S00041.htm
4. Supreme Court decision creates “new playing field” for tax avoidance. http://business.scoop.co.nz/2011/08/24/tax-case-decision-creates-%E2%80%9Cnew-playing-field%E2%80%9D-for-avoidance/
5. Retracted autism study an “elaborate fraud”, British journal finds. http://edition.cnn.com/2011/HEALTH/01/05/autism.vaccines/index.html
6. Utilitarianism and Horizontal Equity: the Case for Random Taxation. http://papers.ssrn.com/sol3/papers.cfm?abstract_id=349091

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Not for Resuscitation Orders—clarification is needed

Debate surrounding decisions not to attempt cardiopulmonary resuscitation (CPR) continues to inflame opinion and attract controversy. This is illustrated by the case of Janet Tracey where issues surrounding communication have led to the family taking legal action against Addenbrooke's Hospital, Cambridge, United Kingdom.[1]

A recent survey of medical, surgical and orthopaedic medical and nursing staff demonstrated that Do Not Attempt Resuscitation (DNAR) orders may be interpreted as limiting other forms of care in addition to CPR . This was particularly true in more junior members of staff.[2]

We believe that hospitals and institutions caring for an elderly population should be using the terminology Do Not Attempt CardioPulmonary Resuscitation (DNA-CPR) on documents indicating CPR is not appropriate. This is a move which has been endorsed by the National Health Service (NHS) in the United Kingdom in an attempt to aid communication with staff, patients and relatives.

There is a need for good communication and clarity regarding issues surrounding end-of-life care. The use of the term DNA-CPR is more specific than DNAR and should be the preferred term for use in patient records and order forms.

John Tolliday and Hermione Denniss
Medical Registrars at Hutt Valley Hospital, Lower Hutt, New Zealand

References:

1. Meikle J. Addenbrooke's and Andrew Lansley sued over 'do not resuscitate' rule. The Guardian. 26th August 2011. Available at http://www.guardian.co.uk/society/2011/aug/26/addenbrookes-andrew-lansley-sued-resuscitate [Accessed 31/10/11]
2. Stewart M, Baldry C. The over-interpretation of DNAR. Clinical Governance: An International Journal 2011;16(2):119-128.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Thames Hospital

If you have half an hour to spare, go to www.waikatodhb.govt.nz and roam around the cluttered website "Waikato District Health Board – Caring for You." It will be of particular interest if you happen to live in the area and you are one of the 365,730 people that this body looks after. The figures supplied are getting better all the time and it is moving into more and more new fields. The Board has 6,000 employees; it had 4,800 in 2001. The budget this year is $1.1 billion dollars, over double that of ten years ago.

The Annual Report confirms that the Board last year arranged 142,368 mental health community visits, and about the same number of district nurse community visits. There were thousands of outpatient visits, but we note the 20,724 "did not attends," and they are estimated to have cost the Board $3.11 million.

Click on "Meet the Team." This site has nothing to do with doctors or nurses, but there you will learn that "effective communication is central to good management practice and crucial to building and maintaining public confidence." The Media and Communications Team has provided some photographs of themselves. There appear to be sixteen of them, doing everything from patients' records to graphic design and desk-top publishing.

There is a page somewhere headed "Health Professionals," and it is "aimed at GPs and other health professionals."

Click on "Thames Hospital," run by the same Board. This secondary hospital gets plenty of attention, and that is hardly surprising, because, apart from the contented state of the maternity services provided in the Thames-Coromandel region, the evidence on the website suggests that the provision of emergency medical services is a perpetual source of anxiety.

Consider this. The population normally served is 44,000. Over the summer months it swells to 200,000. Thames Hospital copes with a staggering 13,000 ED attendances per year, and that number is increasing by ten percent per annum. Of these attendances, one-third are made by persons over sixty-five years of age.

"The hospital is staffed by four general physicians, three general surgeons and a visiting geriatrician supported by registrars, and medical officers/senior house officers. There are two anaesthetists and Emergency Department (ED) doctors who are specifically trained. Medical officers/senior house officers cover low risk inpatient surgical patients during the weekend. A physician is on site or on call at all times. ED doctors are also on site 24/7."

Surgeons and anaesthetists are not available weekends. The Hospital is 100 Km from the Waikato Hospital and the time for the journey is given as ninety minutes.

The site informs us that six general practitioners in the town will, in rotation, work a six-hour shift in ED on Saturdays to relieve the strain. You can pick up on a helpful

Question and Answer section regarding this involvement of the GPs in the hospital. One of the questions reads as follows;

Q. What if you don't want to be seen by a GP?

A. If you choose not to be seen by a GP you will experience a longer wait for an ED doctor.

After a few hours wait for attention, any GP can begin to look good, but in the Emergency Department, "best practices and lean thinking guide practice improvement and change."

Although there are no photographs, or even any mention, of the individual doctors in the employ of the Waikato DHB, there are photographs of the Board members, and of the administration team, and, when you go to the Thames Hospital section of the site, you are invited to "check out our wonderful team of midwives," some of whom have graciously agreed to be photographed.

Twelve midwives are listed. These include Jane, for whom "birth is a miracle," and Sandy, home birth specialist. They all appear to have the use of a brand new Birthing Unit, which is equipped with a birthing pool, birthing slings and birthing balls.

Deliveries in the Unit total about 100 per annum, i.e. two a week. The midwives are at pains to book and manage only low-risk patients, and, as Lead Maternity Carers, they can make all their own decisions.

If, on balance, you would rather see a GP for your medical problems, you should go to www.finda.co.nz , and type in "Thames Medical Centre." If you want to know who works there, you will have to phone up and ask.

Roger M Ridley-Smith
Retired GP
Wellington, New Zealand

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Playing with 'The Public Health'

When it comes to determining public policy, the Director for Environmental Protection and the acting Director of Public Health state that governments must adopt the 'weight of evidence' approach.[1] This method would presumably weigh positive effects, negative effects and potential effects. The likelihood of each of them and their significance are important. If a factor has serious impact and the evidence in relationship to it is strong then the justification for taking action is also stronger. Even so, Karl Popper suggested that because of the uncertainties inherent in broad public policy it is better to make changes incrementally rather than by heroic actions.

He was particularly concerned about unexpected outcomes which could make conditions worse as a result of government action.

We have such as situation in relation to air pollution in New Zealand. Several papers in this journal[2–4] have raised questions about the government's assessment of the risk from PM_{10}. The response from the government has been to produce an expensive document "The National Air Quality Compliance Strategy to Meet the PM_{10} Standard"[5] and a users' guide[6]. These set out the requirements which must be met by regional councils. They include: Prohibition of new solid-fuel open fires from September 2012, replacement of older wood burners, monitoring of ambient PM_{10} by councils, council controls to prevent breaches of the PM_{10} standard, annual compliance reviews by the Ministry for the Environment, possible intervention by the Minister.

The fundamental objection to the Ministry's regulations is their assertion that PM_{10} is the main agent in the increase in deaths in the winter. In this, the Ministry confuses association with causation. By contrast, the U.S. Environmental Protection Agency (E.P.A.) in 2006 revoked an annual PM_{10} standard[7] due to the "lack of evidence linking health problems to long-term exposure to coarse particle pollution (PM_{10})". It has retained a 24-hour standard of 150 $\mu g/m^3$. This level, three times the New Zealand standard, should not be exceeded more than once per year, on average, over 3 years.

The E.P.A. is more concerned about $PM_{2.5}$. These fine particles which may form in the atmosphere from ozone, oxides of nitrogen, sulphur dioxide, volatile hydrocarbons, oxygen and water, by photochemical reactions, can gain access to the small airways in the lung. The significant pathogenicity of these substances is well accepted. They arise from motor vehicle exhausts, the burning of coal and oil, and industrial pollutants.

Why has the New Zealand Government taken such an extreme view over PM_{10} when the E.P.A. has largely discarded it as a relevant measure? The Ministry for the Environment has stated "air-pollution from all sources is estimated to cause more than 1600 premature deaths, 930 hospitalisations and 2.6 million restricted activity days in urban areas in New Zealand every year. The majority of these health effects are from PM_{10} emissions."[5] These claims are a misuse of the statistics.[2,3]

The Minister stated in his foreword: "The challenge for government-both local and central-is how to COMPEL people to take action to improve the quality of their air."[5] So we have a theory and measure which the U.S. has discarded, bolstered by faulty statistics, with a threat of compulsion to comply. As well as this, in their response to the air pollution articles in the *NZMJ*, Currie and Hunt[1] suggested that the articles "offer unsubstantiated criticism of the government's national environmental standards for air quality" and should have first been submitted to either the Ministry for the Environment or the Ministry of Health "to ensure an objective assessment and avoid obvious errors which might mislead the reader"!

Hoare[2] and Palmer[3] have assessed the methodology and statistical analyses used in studies of PM_{10} for some years and through submissions to the administrators, to various Ministry-initiated enquiries, to the local regional Council, and to elected representatives, have tried to persuade the Ministry to review its stance. Their efforts have resulted in no perceptible change in the Ministry's position. Given the authoritarian attitudes exhibited in the above paragraph this is not surprising. The situation raises doubts about the propriety of public consultations in relation to air pollution over the last few years. None of this speaks well for 'The Open Society' in New Zealand.

The PM_{10} programme is a waste of money for individuals, ratepayers and the taxpayer. As it is based on a flawed assumption it results in a false direction to air pollution activities. As well as this, it steps beyond control of a proposed environmental pollutant into the field of social engineering by interfering in the private life of individuals and their friends in their own homes. A civil authority should not do this without overwhelming justification.

We were warned in 2005 by the electricity provider[8] that the programme would lead to an increase in the cost of electricity because of the need for increased generation and line capacity. Because of a high prevalence of fuel poverty in the South Island (spending more than10% of disposable income on heating) this could have disastrous repercussions. Furthermore, this consequence was clearly forecast and was not unforeseen. Did the Ministry for the Environment take it into consideration? During the Christchurch earthquake the weather, fortunately, was mild. If it had not been, given the shift from wood-burning to electricity or gas dictated by Environment Canterbury, the Hospital could have been inundated with elderly people and infants with chest infections. A warning about the continuing risk to electricity security has come from the Institute of Professional Engineers giving further concern over the shift to electricity for domestic heating.

In New Zealand, increased mortality in the winter is one of the worst in the world. The most plausible reason for this, biologically, is exposure to the cold resulting in viral and then bacterial chest infections. It can also trigger myocardial infarction in the susceptible. It is known that average temperatures in our houses are well below the World Health Organization-recommended minimum of 18°C.[9] We need to keep our houses warmer without adding to pathogenic air-pollution or adding to carbon emissions. Given the limitations on other renewable sources, the burning of dry wood fills this role, is affordable and provides a secure heating if the electricity supply fails.

As Howden-Chapman et al[9] have pointed out we need to take a broader view of the factors that influence our air quality and health. Better insulation of housing is very

important and the Ministry for the Environment has taken an effective lead in this. The issues surrounding which sources of energy to use for domestic heating need urgent reappraisal. Because the chemicals SO_2, NO_2, O_3 and volatile hydrocarbons are so important, transport planning and the location of industry and control of its effluents are critical.

The National Air Quality Compliance Strategy to Meet the PM_{10} Standard should be suspended. The administrators who developed this strategy should explain why they have continued to incriminate PM_{10} as the cause of excess deaths in the winter, when the data only show an association, and the U.S. E.P.A. has discarded the theory. They would also need to refute the papers which point out that it is a misuse of the statistics to attribute specific numbers of deaths to PM_{10}.

A group with a broader perspective now needs to examine the issues. Any proposed action or regulation should be judged on its potential to enhance clean air, security of domestic heating, and affordability by individuals, ratepayers, taxpayers and industry. In the longer term, the implications for town planning, transport planning and provision, and industrial development are most important. Whilst the benefits of some initiatives may take time to become manifest, the detection of any unexpected outcome demands on-going monitoring from the beginning. The issues are complex and have broad implications for the future of our cities

Peter W Moller
Rheumatologist
Christchurch, New Zealand
peter.moller@xtra.co.nz

References:

1. Currie K, Hunt D Government response to air pollution articles in NZMJ. N Z Med J. 2011; 124 (1335) http://www.nzmj.com/journal/124-1335/4694/content.pdf
2. Hoare JL. Limitations of the scientific basis for the management of air quality in urban New Zealand. N Z Med J. 2011; 124 (1330) http://www.nzmj.com/journal/124-1330/4558/content.pdf
3. Palmer P, Mann JD How toxic are fine particles emitted from home fires in Christchurch, New Zealand? N Z Med J. 2011; 124 (1330) http://www.nzmj.com/journal/124-1330/4565/content.pdf
4. Moller P Excess winter mortality, wood fires and the uncertainties associated with air pollutants. N Z Med J. 2011; 124 (1330) http://www.nzmj.com/journal/124-1330/4562/content.pdf
5. The National Air Quality Compliance Strategy to Meet the PM_{10} Standard. N.Z. Government ME 1064. ISBN 978-0-478-37250-2 (electronic).
6. Users' Guide National Environmental Standards. N.Z. Government ME 1068. ISBN 978-0-478-37254-0 (electronic).
7. PM Standards Revision – 2006. U.S. Environmental Protection Agency. http://www.epa.gov/oar/particlepollution/naaqsrev2006.html
8. Presentation to Environment Canterbury 'Proposed Canterbury Natural Resources Regional Plan – Air Quality' 2005.
9. Howden-Chapman P, Hales S, Chapman R, Shaw C Improving Air Quality: co-benefits for the urban system. Presentation to the Clean Air Society of Australia and New Zealand, August 2011, Auckland.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Improving Emergency Department performance at Christchurch Hospital: an update

Our paper 'Project RED—a successful methodology for improving emergency department performance,' was recently published in the *Journal*.[1] It was accompanied by an editorial which correctly pointed out that the 'Shorter Stays' target is a whole health system target, not an emergency department target.[2]

Our paper used two performance measures as indicators of progress. The first was waiting time for triage category 2 patients. At the time of submission of our paper 77% of triage 2 patients were being seen within 10 minutes, against a target of 80%. Our second performance measure was the percentage of patients with a total Emergency Department length of stay under 6 hours. At the time of submission our performance against this measure was 90.5%, against a target of 95%. The editorial reiterated comments in our paper that performance against the 6-hour length of stay target, known as the shorter stays in the emergency department health target, was very dependent on the ability of other parts of the system, particularly hospital inpatient services, to take patients from the emergency department.

While Project RED was necessarily an emergency department-centred project, it contributed to (and was in the context of a) growing commitment to whole of system reforms. Both Project RED and this growing commitment have seen the past several months with over 90% of triage 2 patients seen within 10 minutes and over 95% of emergency department patients with a total emergency department length of stay of less than 6 hours.

In addition to a confidence that patient care has improved in general, I have no doubt that the improved processes and enhanced relationships consequent to these and related activities over the last few years provided a strong foundation for the major internal and external incident responses to the Christchurch earthquakes.

Mike Ardagh
Professor of Emergency Medicine, University of Otago, Christchurch and Emergency Medicine Physician, Christchurch Hospital

References:

1. Ardagh MW. Pitchford AM. Esson A. et al. Project RED – a successful methodology for improving emergency department performance. N Z Med J 14 October 2011;124(1344). http://journal.nzma.org.nz/journal/124-1344/4905/
2. Jones P. The 'Shorter Stays' target is a whole health system target, not an emergency department target [editorial]. N Z Med J 114 October 2011;124(1344). http://journal.nzma.org.nz/journal/124-1344/4902/

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Eliminating tobacco point of sale displays: removing the retail detail from the devil

Background—The research initiative for the 2025 countdown to end of tobacco in New Zealand has well and truly begun with the ASPIRE[1] inaugural symposium, which brought together nearly 90 delegates on 29 September 2011 to consider removal of tobacco retail displays.

The Smokefree Environments (Controls and Enforcement) Amendment Act 2011 (SECEA Act), which Parliament passed by 117 votes to 3 in July 2011, requires the removal of tobacco retail displays. In so doing, the legislation responds to an increasing volume of evidence documenting the adverse effect these displays have. First outlined comprehensively in a systematic review,[2] subsequent work has further documented how displays increase the risk of experimentation among children,[3–5] promote impulse purchase[6,7] and induce lapsing among former smokers and quitters.[8,9] There is also evidence from Ireland that removal of point of sale displays has a range of positive effects,[10] and do not result in the disastrous economic impacts predicted by the tobacco industry and other opponents.[11]

Yet, while the new ACT reflects a substantial evidence base, its effectiveness will depend largely on the regulations developed to implement its provisions. To explore implementation questions, examine new opportunities, and learn from others' experiences, the ASPIRE2025 collaboration (Research for a tobacco-free Aotearoa; http://aspire2025.org.nz/) recently held a seminar examining how various Australian states and territories had implemented measures requiring the removal of tobacco retail displays. Anne Jones, CEO of ASH Australia, provided insights into regulations implemented in Australia and their outcomes, following which policy makers, NGO representatives, and researchers outlined issues arising from their perspectives. To stimulate discussion around these questions, we summarise key elements of the seminar below.

Implementation: timing, inclusiveness and specificity—Australian jurisdictions varied in the timing requirements they set for retailers; some set earlier dates for general retailers but allowed 'specialist tobacconists' a longer phase-in period. The net effect was a proliferation of such specialists, which diluted the initial impact of the measure. New Zealand should follow the all-inclusive approach taken by the Northern Territory, whose legislation applied to all tobacco retailers equally.

Duty-free tobacco sales have attracted increasing research attention[12,13] and the new Act should apply equally to in-bound and out-bound stores, thus avoiding the situation that occurred in New South Wales, where the regulation was implemented initially for out-bound displays while a legal challenge delayed application of the policy to in-bound flights for 12 months. This anomaly resulted in in-coming passengers being exposed to very large POS displays regulators.

Retail storage varies in Australia; some retailers have products under the counter while others use cupboards behind the counter. Cupboards behind the counter present

strong visual cues and also require retailers to turn their backs on customers, something retailers believe represents a security risk.[14,15] For these reasons, we strongly suggest the regulations require all tobacco products to be stored in under the counter drawers, which provide no overt visual cues and offer retailers a safer and more secure storage option

What is left at point of sale?—Smaller retailers have argued that removing tobacco displays may confuse smokers and predispose them towards shopping in larger stores, which they may consider more likely to stock tobacco products.[14,16] The Act responds to this concern by allowing retailers to display a sign indicating that tobacco products are available; the Regulations will define the content, size and position of these signs.

We suggest these signs should be limited to the size of a business card, akin to the stickers used by debit card companies to indicate a retailer accepts a specific card. Further research is required to test whether retailers who choose to display an availability sign should also be required to feature a large graphic health warning or a Quit sign in a prominent position.

Depending on the jurisdiction, Australian retailers may feature up to three different types of signage: a price ticket, price board and price list. Retailers use price tickets to identify the products stored in alphabetical order and obscure tobacco products that may not be displayed (such as products at additional points of sale). States allowing price tickets prescribe the size, colour, content and font size of information on these.[17] Price boards may detail tobacco brands available; variants; pack sizes and prices (including discounts, which Victoria allows).[18] Price lists are not visible within the store but refer to pages detailing the brands and variants available, and the cost of these; where regulators have permitted price lists, retailers may only provide these on request. Table 1 below outlines the current situation in Australian States and Territories.

Some Australian states also allow retailers to display price boards, lists, or both; given the use of these to promote brands and bonus packs, we suggest New Zealand regulators allow only price tickets with no visible price board or, failing this, a price list, to be shown only on request. If the regulations allow price boards or lists, we recommend these should also feature a graphic health warning that covers at least 50 percent of the page surface. In the case of multi-page price lists, we recommend each page feature a different warning to ensure smokers are exposed to as many different warning messages as possible, should they ask to peruse a price list.

Given the variety of policies implemented, it is imperative that New Zealand's regulations reflect best practice and research findings, and that a full evaluation plan is also implemented. The tobacco industry and its front groups have consistently opposed legislation to remove POS displays by arguing these have failed in other jurisdictions.[14] Evidence from marketing theory suggests decreasing the visibility and accessibility of a product will reduce its salience, perceived normality, and use,[19] making it highly unlikely these policies would have no effect. However, without evidence from evaluations to test the industry's claims, policy makers considering removing POS marketing may be swayed by industry arguments and defer passing legislation.

Table 1: Price tickets/Price boards/Labelling after POS display ban – Australian State/Territory legislation (at July 2011)

Jurisdiction	Price tickets – display	Price Boards	Price list	Other labels
ACT	✓(can be located 'below or next to blocked out smoking products' – could be inside or outside cupboard)	X	X (Staff barcode sheet permitted)	
NSW	✓ (must be attached to a sales unit)	✓ (Board or price tickets not both, most retailers use price board)	X	
NT	X	✓	✓ (Can be produced on customer request)	✓ (Small label visible to retailer only, no price info)
QLD – TBC – Bill currently before QLD Parliament; new regulations not yet drafted.	✓ (Bill says must be 'fixed at the place' where tobacco products are kept – could be in or outside unit)	X	X	
SA	✓ (can be located inside or outside cupboard or both)	✓	X	
TAS	X	✓	X	✓ (Can use price ticket inside cupboard; must only be visible to retailer)
VIC	X	✓	X	✓ (Dept advises ok as long as does not amount to a tobacco advertisement)
WA	✓ (can be located inside or outside cupboard)	✓	✓ (Can be produced on customer request)	X

Source: Table courtesy of Anne Jones, ASH Australia; information supplied originally by Quit Victoria

New opportunities in the retail sector: looking to the future—The SECEA Act represents an important advance in tobacco control, but achieving the tobacco free Aotearoa 2025 goal will require additional policy measures and interventions. The SECEA Act does not introduce retailer licensing and thus misses opportunities to create more protective environments. Implementing a retailer licensing or registration scheme would enable greater monitoring of outlets selling tobacco, facilitate communication with tobacco retailers, and enable the implementation of further measures. Licensing arrangements would assist reputable retailers by making those who currently breach sales regulations more accountable.

Licensing could lead to a number of potentially useful developments: For example, all retailers might receive training to ensure they understood the legal requirements of the new law and regulations as well as the toxicity and addictiveness of tobacco products. Licensing could stipulate that only people aged 18 or over should sell tobacco (thus aligning the sales and purchase age requirements). It should also require retailers to provide cessation advice and information to smokers each time they purchase tobacco,

thus ensuring cessation messages are delivered and repeated at the point of every sale, an intervention likely to promote quit attempts.

Two-thirds of smokers purchase tobacco from dairies but only 20% of smokers used NRT to assist their last quit attempt; of these, 75% obtained NRT from a pharmacy using a Quitcard.[20] The Maori Affairs Select Committee Inquiry into the Tobacco Industry Report recommended: *"That nicotine replacement therapies be required to be sold everywhere tobacco is sold, thereby ensuring smokers can choose a safe option whenever they crave nicotine"*.[21] Policy makers now have a golden opportunity to implement this recommendation, require retailer licensing, and create a retail environment that facilitates quitting rather than maintains smokers' addiction. Tobacco retailers potentially represent one of the best sources of regular quit advice; given smokers frequent interactions with retailers, linking traditional tobacco supply outlets to NRT provision and Quitline follow-up could represent a major cessation force.

Other measures could include additional protections, such as restricting entry to outlets selling tobacco to people aged 18 or over, a measure that would further decrease children's exposure to tobacco, help prevent young people taking up smoking, and drive increases in quit attempts. Measures to control the location, proximity to schools, opening hours, and density to tobacco retailers in lower decile areas may also be warranted. Licensing would enable communities to have a stronger input into tobacco retailing locations within their area, while local authorities could set a cap on the licences issued and ensure tobacco retailing was not concentrated in areas of high deprivation.

Perhaps most importantly, licensing would clearly signal that, although tobacco retains its status as an anomalous 'legal' product, it is nevertheless a toxic and abnormal product; and that selling tobacco is not a right but a restricted service contingent on meeting certain proscribed standards.

Despite the excellent progress the SECEA Act represents, the ASPIRE2025 symposium reminds us that the task is not yet complete. Not only do the regulations accompanying the Act need to avoid problems experienced in Australia, but retail environments themselves require further analysis so we can avail ourselves of the opportunities they present to promote cessation.

Janet Hoek, Anne Jones, Richard Edwards, Ninya Maubach, Julian Crane,
Ben Youdan; for the ASPIRE2025 collaboration

References:

1. ASPIRE2025. Point of Sale and Beyond: A Research and Policy Seminar. Wellington, 2011.
2. Paynter J, Edwards R. The impact of tobacco promotion at the point of sale: A systematic review. Nicotine & Tobacco Research 2009;11(1):25-35.
3. Paynter J, Edwards R, Schluter PJ, McDuff I. Point of sale tobacco displays and smoking among 14-15 year olds in New Zealand: a cross-sectional study. Tob. Control 2009;18(4):268-74.
4. Thomson G, Hoek J, Edwards R, Gifford H. Evidence and arguments on tobacco retail displays: marketing an addictive drug to children? N Z Med. J. 2008;121(1276):87-98.

5. Henriksen L, Schleicher NC, Feighery EC, Fortmann SP. A longitudinal study of exposure to retail cigarette advertising and smoking initiation. Pediatrics 2010;126(2):232-8.
6. Carter OBJ, Mills BW, Donovan RJ. The effect of retail cigarette pack displays on unplanned purchases: results from immediate postpurchase interviews. Tob. Control 2009;18(3):218.
7. Burton S, Clark L, Jackson K. The association between seeing retail displays of tobacco and tobacco smoking and purchase: findings from a diary style survey. Addiction 2011.
8. Hoek J, Gifford H, Pirikahu G, et al. How do tobacco retail displays affect cessation attempts? Findings from a qualitative study. Tob. Control 2010;19(4):334.
9. Germain D, McCarthy M, Wakefield M. Smoker sensitivity to retail tobacco displays and quitting: a cohort study. Addiction 2010;105(1):159-63.
10. McNeill A, Lewis S, Quinn C, et al. Evaluation of the removal of point-of-sale tobacco displays in Ireland. Tob. Control 2011;20(2):137.
11. Quinn C, Lewis S, Edwards R, McNeill A. Economic evaluation of the removal of tobacco promotional displays in Ireland. Tob. Control 2011;20(2):151.
12. Vardavas C, Seidenberg A, Connolly G. Regulating duty free sales and tobacco advertising in airports: a call for action. Tobacco induced diseases 2011;9(1):1-3.
13. Wilson N, Thomson G, Edwards R, Peace J. Estimating missed government tax revenue from foreign tobacco: survey of discarded cigarette packs. Tobacco Control 2009;18(5):416-18.
14. Association of Community Retailers. Submission to the Health Select Committee on the Smoke-free Environments (Controls and Enforcement) Amendment Bill, 2011.
15. Hoek J, Vaudrey R, Gendall P, et al. Tobacco retail displays: a comparison of industry arguments and retailers' experiences. Tobacco Control 2011.
16. Association of Community Retailers. Submission to Ministry of Health's Proposal to Ban Tobacco Retail Displays in New Zealand: Association of Community Retailers, 2010.
17. City Health Information. http://www.melbourne.vic.gov.au/enterprisemelbourne/industries/healthcare/Documents/Tobacco_Retailing.pdf Melbourne, UD.
18. State Government of Victoria Department of Health. Tobacco retailers: Price board requirements: State Government of Victoria, 2010.
19. Hoek J, Jones SC. Regulation, public health and social marketing: a behaviour change trinity. Journal of Social Marketing 2011;1(1):32-32-44.
20. Ministry of Health. Tobacco Use in New Zealand: Key findings from the 2009 New Zealand Tobacco Use Survey. In: Health Mo, editor. Wellington: Ministry of Health 2010.
21. Māori Affairs Committee. Inquiry into the tobacco industry in Aotearoa and the consequences of tobacco use for Māori. Wellington: New Zealand Parliament, 2010.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Binge drinking is patterned by demographic and socioeconomic position in New Zealand: largest national survey to date

There is good evidence that episodes of heavy alcohol consumption on single occasions ("binge drinking") and not simply the total average consumption, play a key role in determining alcohol-related harm in a population.[1-3] In New Zealand (NZ), the role of hazardous alcohol consumption in contributing a large morbidity and mortality burden is also well documented.[2 4-8] To add to the understanding of the NZ situation, we report here on the largest NZ survey to date on alcohol usage.

Methods—We used data from Wave 3 (2004/05; SoFIE w1-7 data Version 1) of the longitudinal Survey of Family, Income and Employment (SoFIE), a survey of the adult NZ population that is described in detail elsewhere.[9] But to summarise, the survey involves a nationally-representative sample with annual (computer-assisted) interviewing conducted in the home.

A total of 19,255 adults (85% of the original sample) responded to the health questionnaire at Wave 3. Of these (18,520; 96%) provided information on alcohol use. During the Wave 3 health module participants who had had a drink containing alcohol (one standard drink was defined as a can or small bottle of beer; a small glass of wine; a single nip of spirits) in the last 12 months were asked how many days in the last four weeks they had drank alcohol (i.e. frequency); and how many drinks containing alcohol they had consumed on a typical day when they were drinking. Participants were also specifically asked whether they had ever had more than eight (for men) or six (for women) standard drinks on one occasion and if so how many occasions in the last 4 weeks.

The frequency of binge drinking (defined here as drinking more than eight (for men) or six (for women) standard drinks on one drinking occasion) in those who reported drinking in the past 12 months was categorised as: never binge, binge monthly, binge two times per month, binge weekly and daily or almost daily binge drinking. Socioeconomic measures included employment status, highest reported education, area deprivation and individual-level deprivation (all measured at Wave 3). Although data are not weighted to represent the NZ population, however the SoFIE sample (at Wave 3) has similar age, sex and ethnicity distribution to the NZ population.

Table 1. Demographic and socioeconomic characteristics of the SoFIE-Health study population with regard to any alcohol use and binge drinking pattern*

Variables	Number of respondents	Never drink	Drink but never binge	Binge monthly	Binge twice per month	Binge weekly	Bingedaily /almost daily
	N	row %	row %	row %	row %	row %	row %
All	18520	18.3	60.1	7.7	4.5	5.5	3.8
Sex							
Women	9990	21.6	62.3	6.7	3.4	3.9	2.2
Men	8530	14.5	57.6	8.9	5.9	7.4	5.7
Age group (years)							
15–24	2980	21.0	43.5	10.4	6.9	10.7	7.4
25–34	2600	13.7	52.5	13.3	7.5	8.5	4.6
35–44	3660	14.3	60.2	10.1	5.5	6.0	4.0
45–54	3420	17.1	64.0	7.0	3.9	4.5	3.5
55–64	2705	17.4	69.9	4.6	2.6	3.1	2.2
65+	3155	26.3	69.4	1.4	1.1	0.8	1.1
Ethnicity							
NZ European	14060	12.8	65.8	7.7	4.7	5.4	3.7
Māori	2250	22.9	44.9	12.0	5.8	8.7	6.0
Pacific	825	55.2	29.1	5.5	3.0	4.8	2.4
Asian	960	51.6	43.8	2.1	1.0	1.0	0.5
Other	420	31.0	52.4	4.8	3.6	4.8	3.6
Marital status							
Divorced/widowed/separated	3215	22.1	61.1	6.1	3.4	4.0	3.1
Married	9560	17.3	67.0	6.4	3.4	3.7	2.2
Never married	5735	17.9	48.0	10.8	7.1	9.5	6.7
Maximum education qualification							
Degree or higher	2610	14.6	66.9	7.5	4.2	4.6	2.3
Post school vocational	6285	13.2	63.9	8.6	4.8	5.7	3.8
School qualification	4160	14.2	56.7	9.9	6.4	7.8	5.2
No qualification	4635	27.6	55.4	5.5	3.2	4.3	3.9
Labour market activity							
Working	11990	11.7	61.4	9.7	5.6	7.0	4.5
Unemployed	350	21.4	51.4	10.0	4.3	7.1	4.3
Inactive	6170	30.9	58.2	3.8	2.4	2.5	2.2
Deprivation (small area)							
NZDepQ1 (least deprived)	3805	11.0	68.1	7.8	4.5	5.7	2.9
NZDepQ2	3760	13.4	66.1	7.0	4.8	5.2	3.5
NZDepQ3	3395	17.2	59.9	8.7	5.2	5.3	3.5
NZDepQ4	3940	20.2	58.4	7.4	3.9	6.0	4.2
NZDepQ5 (most deprived)	3620	29.8	47.4	7.7	4.4	5.7	4.8
Deprivation – individual level (NZiDep)							
0 measure of deprivation	13340	15.9	63.7	7.3	4.5	5.2	3.4
1 measure	2785	23.2	52.6	8.8	4.8	6.5	4.1
2 measures	1105	23.5	50.7	8.6	4.5	7.2	5.4
≥3 measures	1275	28.6	47.5	9.0	4.3	5.5	5.1

Notes: * For the past 12 months, results are unweighted and unadjusted (see main text for details); All numbers of respondents presented in this paper are random rounded to the nearest multiple of five, with a minimum value of 5, as per Statistics New Zealand protocol. Row percentages may not always add up to 100% because of rounding or missing values.

Results & Discussion—Table 1 presents the demographic and socioeconomic characteristics of the study sample in the five categories of binge drinking and never drinkers. Notable findings are that:

- Binge drinking was more common in men than women (monthly, weekly and daily levels). Women were more likely to abstain.
- Binge drinking was particularly frequent in the 15–24 age-group. There was a decline in binge drinking (across all frequencies) with increasing age.
- All non-NZ European ethnic groups reported more abstention than NZ European. Correspondingly non-NZ European ethnic groups reported lower levels of drinking, in particular "never binge" in the last 12 months than the NZ European group. Māori respondents reported high levels of binge drinking at the monthly, weekly and daily levels compared to other ethnic groups.
- Binge drinking was more common in those reporting being "never married", compared to the other relationship categories.
- Frequency of binge drinking generally increased as educational level declined. This was a consistent pattern for the first three educational categories listed in the table (for monthly, weekly and daily levels), but the pattern was more mixed for when considering the "no qualification" group. The pattern by work status was also mixed, but with some indication of a higher frequency of daily binge drinking in the working vs unemployed groups.
- The patterns for both area and individual-level deprivation were generally mixed. Nevertheless, there was a clear gradient for increasing daily binge drinking frequency with each increment in greater area deprivation. There was a similar pattern for individual-level deprivation.

These patterns are generally consistent with previous (albeit smaller) NZ national surveys. For example, a national survey in 2007/08 found that most (61.6%) NZ drinkers consumed more than six (for men) or four (for woman) standard drinks at least once in the previous year, with 12.6% consuming similar amounts in a single drinking occasion at least weekly in the previous year.[10] It reported that: "youth, Maori men and women, Pacific men, and people living in more deprived neighbourhoods were more likely to drink higher amounts than recommended, to engage in risky drinking behaviours, and to experience more harm due to alcohol use". A similar survey in 2004 also reported that 14.7% of adults consumed large amounts of alcohol at least once a week (as per the level of standard drinks in the 2007/08 survey).[11] Hazardous drinking patterns were more common for men, young adults, and Māori vs non-Māori.

The 2006/07 NZ Health Survey reported a hazardous drinking pattern for 21.1% of adults, with relatively more hazardous patterns for: men, 18-24 year olds, Māori, Pacific peoples, and with increasing area deprivation (statistically significant for both sexes).[12] An earlier national health survey reported a lower hazardous drinking pattern for 17.2% of adults. There were relatively more hazardous patterns for: men, 15-24 year olds and Māori (a gradient for increasing area deprivation was suggestive, but not statistically significant).[13] The earlier national health survey in 1996/97 reported

similar levels of hazardous drinking in adults, but there was evidence for an increase since this time for Māori men.[12]

The role of these demographic and socioeconomic factors in causal pathways that result in binge drinking are not detailed here. Such information may follow from additional analyses of subsequent waves of SoFIE-Health data. Nevertheless, for policy-makers who wish to address the hazardous drinking situation in NZ, it is likely that successful control measures will tend to reduce gender, ethnic and socioeconomic inequalities in health.

NZ has a good evidence base upon which to improve alcohol control in the form of a major Law Commission Report[14] and a large body of local research (some summarised recently[15]). A subsequent Select Committee process occurred and a Report for Parliament produced, but it is unclear what will happen with this process given the failure of legislative action prior to the 2011 election. Furthermore, key components of the Law Commission's recommendations were missing from the Select Committee's Report (e.g., higher alcohol taxes, lower drink driving levels, and major restrictions on marketing). Criticisms of the limited response by the Government have been published,[16] [15] and the lack of action contrasts with high public support for improved policies around access to alcohol and enforcement of alcohol-related laws.[17] In particular, the lack of action on higher alcohol pricing is not consistent with the evidence for this being the most effective[18] and cost-effective intervention to reduce alcohol-related harm.[19] [20]

In summary, this large national survey extends previous research to indicate that binge drinking is relatively prevalent in the NZ population and affects all population groups. That said, hazardous drinking patterns are particularly prevalent in: men, young adults, Māori, and (to a variable extent) higher deprivation groups. So improved policy-making for alcohol control will benefit the whole of society and may also contribute to reducing gender, ethnic and socioeconomic inequalities in health in this country.

Competing interests: Nil.

Acknowledgements: SoFIE-Health is primarily funded by the Health Research Council of NZ as part of the University of Otago's Health Inequalities Research Programme. Establishment funding was also received from the University of Otago, Accident Compensation Corporation of NZ (ACC), and the Alcohol Advisory Council (ALAC). We thank Professor Jennie Connor (University of Otago) for her helpful comments on a draft.

Statistics New Zealand Security Statement: Access to the data used in this study was provided by Statistics New Zealand in a secure environment designed to give effect to the confidentiality provisions of the Statistics Act, 1975. The results in this study and any errors contained therein are those of the author, not Statistics New Zealand.

Santosh Jatrana[1], Kristie Carter[2*], Sarah McKenzie[2], Nick Wilson[2]

1. Alfred Deakin Research Institute, Deakin University, Victoria, Australia
2. Department of Public Health, University of Otago, Wellington, New Zealand

* Email for correspondance: kristie.carter@otago.ac.nz

References:

1. Bobak M, Room R, Pikhart H, et al. Contribution of drinking patterns to differences in rates of alcohol related problems between three urban populations J Epidemiol Comm Health 2004;58:238-42.
2. Connor J, Broad J, Rehm J, et al. The burden of death, disease, and disability due to alcohol in New Zealand. NZ Med J 2005;118(1213):1412.
3. Rehm J, Gmel G, Sempos CT, Trevisan M. Alcohol related morbidity and mortality. Alcohol Research and Health 2003;27:39-51.
4. Connor J, You R, Casswell S. Alcohol-related harm to others: a survey of physical and sexual assault in New Zealand. NZ Med J 2009;122(1303):10-20.
5. Huckle T, Huakau J. Young People and Drinking in New Zealand 2004. Auckland: Centre for Social and Health Outcomes Research and Evaluation and Te Ropu Whariki, Massey University, 2005.
6. Huckle T, Pledger M, Casswell S. Trends in alcohol-related harms and offences in a liberalized alcohol environment. Addiction 2006;101(2):232-40.
7. Kypri K, Paschall MJ, Langley J, et al. Drinking and Alcohol-Related Harm Among New Zealand University Students: Findings From a National Web-Based Survey. Alcoholism: Clinical and Experimental Research 2009;33(2):307-14.
8. Wilson N, Imlach Gunasekara F, Thomson G. The benefits and harms of alcohol use in New Zealand: what politicians might consider. NZ Med J 2011;124(1336):85-9.
9. Carter K, Cronin M, Blakely T, et al. Cohort profile: Survey of Families, Income and Employment (SoFIE) and Health Extension (SoFIE-health). Int J Epidemiol 2009;39(3):653-59.
10. Ministry of Health. Alcohol Use in New Zealand: Key results of the 2007/08 New Zealand Alcohol and Drug Use Survey. Wellington: Ministry of Health, 2009.
11. Ministry of Health. Alcohol Use in New Zealand: Analysis of the 2004 New Zealand Health Behaviours Survey – Alcohol Use. Wellington: Ministry of Health. http://www.moh.govt.nz/moh.nsf/indexmh/alcohol-use-in-new-zealand-2004, 2007.
12. Ministry of Health. A Portrait of Health: Key Results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health. http://www.moh.govt.nz/moh.nsf/indexmh/portrait-of-health, 2008.
13. Ministry of Health. A Portrait of Health: Key results of the 2002/03 New Zealand Health Survey. Wellington: Ministry of Health. http://www.moh.govt.nz/moh.nsf/pagesmh/3333, 2004.
14. New Zealand Law Commission. Alcohol in Our Lives: Curbing the Harm (NZLC R114). Wellington: New Zealand Law Commission. http://www.lawcom.govt.nz/project/review-regulatory-framework-sale-and-supply-liquor/publication/report/2010/alcohol-our-lives, 2010.
15. Wilson N, Imlach Gunasekara F. National alcohol plans. New Zealand's alcohol plan is less than "half hearted". BMJ 2011;342:d2147.
16. Kypri K, Maclennan B, Langley JD, Connor JL. The Alcohol Reform Bill: more tinkering than reform in response to the New Zealand public's demand for better liquor laws. Drug Alcohol Rev; 2011;30(4):428-33.
17. Maclennan B, Kypri K, Langley J, Room R. Public sentiment towards alcohol and local government alcohol policies in New Zealand. Int J Drug Policy 2011;[E-publication 8 July].
18. Wagenaar AC, Tobler AL, Komro KA. Effects of alcohol tax and price policies on morbidity and mortality: a systematic review. Am J Public Health 2010;100(11):2270-8.
19. Cobiac L, Vos T, Doran C, Wallace A. Cost-effectiveness of interventions to prevent alcohol-related disease and injury in Australia. Addiction 2009;104:1646-55.

20. NICE (National Institute for Health and Clinical Excellence). Alcohol-use disorders: preventing the development of hazardous and harmful drinking (NICE public health guidance 24) London: NICE. http://www.nice.org.uk/nicemedia/live/13001/48984/48984.pdf, 2010.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Dominion Notes: Otaki Sanatorium

Published in NZMJ May 1912:11(42):136.

Serious doubts as to whether the Otaki Sanatorium is doing what is claimed for it were expressed by Mr. B. R. Gardener at the meeting of the Charitable Aid Board in March.

Mr. Gardener thought that the board should be informed of the condition of the patients who passed through the sanatorium. At present all that was known about the institution was that certain patients were discharged from time to time as "able to work." Members of the board were informed that these people were able to work when they left the sanatorium, but they know nothing of how the patients fared later.

Only two or three patients had entered the institution from his district, and all of them had died. It was just possible that the treatment received by the patients was not having the good results that were claimed for it, and, in view of the fact that £30,000 to £40,000, had been spent on the place, the board had a right to know more about the patients after they left it.

Each patient entering the sanatorium cost about £150, if proper account was taken of capital cost of buildings, or about £90, omitting this. He did not believe that the sanatorium was doing the good work that members had been led to believe. He was not sure, either, whether the institution was being conducted as it ought to be. One patient had been in it for twenty months, and others had been in it for various periods, ranging down to six months. He thought that it was intended that it should be an educational sanatorium rather than a hospital. He understood that patients were to be treated there for three months or so, and that they were then to be sent away to treat themselves by the methods they had learned in the sanatorium. He would move:

> "That the names and last addresses of those patients that have been treated and discharged from the Otaki Sanatorium be forwarded to the Health Department with a request that their inspectors make inquiries as to the present condition of those that have left the sanatorium, and report to the board at a future date."

Mr. W. Tompsitt seconded the motion.

Mr. Van Staveren pointed out that the motion could do no good. The board had had the very same question under consideration eighteen months ago, and it had then been made clear that the board had no power to do more than recommend the matter to the Government for consideration. He hoped that the time would come soon when there would be legislation to deal with the position, but now there was none, and it was, therefore, useless to pass the motion.

The motion was carried.

NZMJ Note: A response to Mr Gardner's opinion is published at http://paperspast.natlib.govt.nz/cgi-bin/paperspast?a=d&d=EP19130905.2.159

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Consensus Statement on the Role of the Doctor in New Zealand

Preamble

In an environment of changing societal expectations, workforce pressures, significant scientific and technological advancements, including the increasing influence of the internet, the medical profession has developed this Consensus Statement that defines our aspirations as to the role of the doctor in 21st Century New Zealand and reaffirms our commitment to patients and the community.

Key statements

- Doctors regularly take ultimate responsibility for medical decisions and diagnoses in situations of complexity and uncertainty, drawing on scientific knowledge and principles, clinical experience, and well developed judgement.
- Doctors accept their ethical responsibilities to act in the best interests of their patients, and the population as a whole, and undertake this in a caring, compassionate, competent, and trustworthy manner.
- Doctors work in partnership with patients in the delivery of their healthcare and serve as advisors and interpreters in the pursuit of optimal health outcomes using evidence-based medicine and in accordance with available resources.
- Doctors work effectively as leaders. As members of healthcare teams, doctors recognise and respect skills and attributes of other practitioners.
- Doctors are advocates for improved population health and health equity for all people.
- Doctors are committed to the spirit and principles of The Treaty of Waitangi, particularly as it relates to the attainment of health equity for Māori.
- Doctors have diverse roles, within and outside of the health sector, in the promotion and maintenance of both individual and population health.
- Doctors accept responsibility for maintaining the high standards of the medical profession to uphold the trust placed in them by patients and the community, and demonstrate this through adherence to relevant declarations including the New Zealand Medical Association Code of Ethics and the Code of Health and Disability Services Consumers Rights.

Doctors as scientists

Doctors have the ability to access, interpret and assimilate new knowledge critically, have strong intellectual skills and grasp of scientific principles, and are capable of effectively managing uncertainty, ambiguity and complexity. They have the capacity

to work out solutions from first principles when patterns do not fit, and the ability to work outside guidelines when circumstances demand.

Doctors use scientific tools and techniques, including audit and research, to develop new knowledge.

Doctors as health professionals

Doctors share attributes with many health professionals that include listening and communication skills, the ability to work as part of a team, non-judgmental behaviour, compassion and integrity which combine to merit the trust of patients and whānau (extended family). They have the ability to assess patients' healthcare needs taking into account personal and social circumstances, culture and beliefs.

Doctors are trained to:

- Integrate information from a variety of sources in order to make decisions or reach diagnoses.
- Provide medical and/or surgical interventions, including the prescription of medicines, in both elective and emergency situations.
- Practise specific clinical skills such as the art of history taking and physical examination.
- Identify and minimise risk and harm.
- Identify and advise on appropriate tests, treatment options (including non-intervention) or preventative measures, and explain and discuss any associated risks, benefits and uncertainties.
- Support patients in understanding their condition and empower them to make informed decisions.
- Assist patients and whānau to decide when supportive care is preferable to intervention, including in relation to end-of-life decisions..

Doctors recognise the importance of maintaining their own health and are committed to supporting each other in achieving this.

Doctors' breadth and depth of training enables them to provide oversight of patient care in both acute and longer term care settings. This breadth and depth of training also enables doctors to move between a variety of roles in their daily practice and throughout their careers.

Doctors as leaders

Doctors embrace the concepts of clinical leadership and clinical governance, and are well suited to leadership roles within healthcare teams and the health sector more broadly.

Doctors exhibit leadership in making day-to-day clinical decisions based on using their medical knowledge to assess the impact, risk and likely outcome of decisions. Further, it is the role of doctors as leaders to apply their skills in the development of policy, strategy, service design, and clinical processes. As leaders, doctors have a

responsibility for ensuring patient safety and monitoring both individual and service level outcomes.

Doctors have a key role in providing higher level sector leadership, including in leading and facilitating change. Some doctors will further utilise their skills in formal leadership or management roles at various levels.

Doctors as health advocates

Doctors uphold the primacy of the individual patient:doctor relationship, with the requirement to advocate for the patient and advise about all treatment options. Doctors also appreciate the needs of their patients in the context of the wider health needs of the population. Where the capacity to treat is growing but resources are finite, doctors, as critical decision makers with responsibility for allocation of significant health resources, have a duty to use those resources wisely, and to engage in constructive debate about such use. As significant resources themselves, they are committed to ensuring their own and others' skills and knowledge are deployed to best effect. When appropriate, doctors use their influence to advocate for increased resources to improve health outcomes for their patients and populations.

Doctors have a role in the promotion of population health, including ongoing efforts to achieve health equity. Some doctors will take an increased focus on the health of the population through formal roles in health education or promotion, service improvement, public health and/or health advocacy. This commitment is to the health of all New Zealanders, but it exists alongside a professional responsibility for the health of individuals and communities throughout the world.

Doctors as teachers and learners

Doctors are committed to the education of current and future generations of medical practitioners, with the apprenticeship model central to this education. In addition, doctors recognise the importance of new methods of learning, such as simulation based learning, as vital adjuncts.

Doctors are also involved in the education of other medical and health practitioners as well as their patients, whānau and communities.

Doctors accept their responsibility to continue personal education and other professional development activities throughout their careers.

Doctors interact with colleagues in clinical practice, quality improvement and educational settings, are involved in supporting each others' practice, and in identifying and remediating each others' performance. Through these collaborative endeavours, doctors strive to ensure that they and their colleagues are fit to practise.

Doctors as members of the healthcare team and the broader health sector

Doctors accept responsibility to positively influence the culture and the environment in which they work. They achieve this through exhibiting behaviours that are nurturing, supportive and respectful, and which enable individuals and teams to

flourish and enjoy their work, and through advocacy for safe and health promoting workplaces and health care settings.

Doctors are committed to excellence in healthcare both in their individual performance as well as contributing to the systems essential to delivering quality care.

Doctors possess the ability to work effectively as members of healthcare teams, recognising and respecting skills and attributes of other practitioners and of patients and whānau.

This Statement has been developed through extensive consultation over a 6-month period that began with a 2-day symposium, at which over 80 leaders of the profession and other key players in the health sector expressed their vision as to the role of the doctor in 21st Century New Zealand. The signatories recognise that the role of the doctor will continue to evolve and offer this Statement to assist both the profession and policy makers to attract, train, and retain the most appropriate medical workforce for our needs.

It is endorsed by: The New Zealand Medical Association (NZMA), Cardiac Society of Australia and New Zealand (NZ Branch), Royal College of Pathologists of Australasia, Royal Australian New Zealand College of Radiologists, Royal Australian New Zealand College of Obstetrics and Gynaecologists, Council of Medical Colleges, New Zealand College of Public Health Medicine, Australasian College for Emergency Medicine, Australian and New Zealand College of Anaesthetists, New Zealand Rural General Practice Network, Royal New Zealand College of General Practitioners.

Acknowledgements: The NZMA acknowledges the particular contributions of Dr Andrew Old, Professor Harvey White and Dr Peter Foley who led the development of the written Statement and ensured that it accurately reflected the consensus view of the stakeholders involved.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Unnecessary hospitalisations of nursing home residents

This study, which is funded by the US National Institute of Aging, evaluates such hospitalisation in the USA. The authors start with the proposition that admission to an acute hospital in the last years of life can be burdensome and potentially of limited clinical benefit for patients with advanced cognitive and functional impairment. They suggest that such hospital admissions have the potential for fragmentation of care, changes in the management of chronic diseases, duplication of diagnostic workups, and medical errors. They defined such admissions as burdensome if they occurred in the last 3 years of life, if there was a lack of continuity in nursing homes after hospitalisation in the last 90 days of life, or if there were multiple hospitalisations in the last 90 days of life.

They concluded that 19% of 474,829 nursing home residents had at least one burdensome admission. They believe that such patients would be better served by remaining in their nursing home.

N Engl J Med 2011;365:1212–21.

Long-term follow up of adjuvant tamoxifen in the management of early breast cancer

This meta-analysis reviews 20 trials comparing outcomes in the management of early breast cancer with or without tamoxifen. There were over 10,000 women with oestrogen receptor (ER)-positive disease and they report that 5 years of tamoxifen substantially reduced recurrence rates over 10 years (Relative Risk 0.53) and reduced breast cancer mortality by about a third throughout the first 15 years of follow-up.

Overall non-breast-cancer mortality was little affected, despite small absolute increases in thromboembolic and uterine cancer mortality (both only in women older than 55 years), so all-cause mortality was substantially reduced. In ER-negative disease, tamoxifen had little or no effect on breast cancer recurrence or mortality.

So tamoxifen still plays an important role bearing in mind that aromatase inhibitors are ineffective in women whose ovaries are still functioning.

Lancet 2011;378:771–84.

Medical journals and conflicts of interests

The editor of the *BMJ* announced in August that the journal would no longer commission editorials and clinical reviews from authors with ties to industry. The issue was obviously conflicts of interest and the policy has prompted controversy. Nine letters, including one from a current editor of the *American Family Physician* (*AFP*), one from a former editor of the *BMJ*, and one from a former editor of the *NEJM* appear in a recent *BMJ* issue. The *AFP* and *NEJM* editors strongly endorse the new policy, seven oppose it, and one is undecided.

Your scribe is also undecided but notes one or two compelling arguments against the ban. For example readers might feel more confident about the assessment of a new drug in a clinical review article if the author, as a result of ties with industry, has direct experience with it—for example, as investigator in preregistration clinical trials or as adviser on its clinical development. On the other hand a completely unbiased reviewer might be ill-informed on the topic, lessening the merits of his/her review.

Interestingly we note that the *NEJM* had a similar ban in the 1990s but later reverted to "declaration of conflicting interest".

BMJ 2011;343:493–95.

Is there a place for adenoidectomy in children with recurrent upper respiratory infections?

In pre-antibiotic days, removal of the tonsils and adenoids was an almost mandatory procedure in infants as it was believed that such surgery would prevent recurrent upper respiratory tract infections. Whether it was or is efficacious is a moot point but the practice continues. This randomised controlled trial compared a strategy of adenoidectomy with or without myringotomy with a strategy of initial watchful waiting. The outcomes measured over 2 years were the number of upper respirator tract infections and middle ear complaints with fever.

There was no significant difference between the two cohorts. The children in the watch group who later had adenoidectomy also received no benefit from their surgery. The researchers conclude that immediate adenoidectomy confers no clinical benefits over initial watchful waiting as the prevalence of infections decreases equally over time in both groups.

BMJ 2011;343:d5154.

Polypharmacy in the elderly

It is generally acknowledged that complex polypharmacy heightens the risk of serious adverse drug events in the elderly. The authors of this paper point out that although frailer, older people are usually excluded from randomised controlled trials, prescribers often persist with evidence-based prescribing in end-of-life older patients. They suggest that lipid-lowering drugs are almost always inappropriate in end-of-life as their evidence-based benefits occur in a 5–10 year timeframe in a younger population.

Likewise, ACE-inhibitors and angiotensin receptor blockers to prevent diabetic nephropathy or to reduce mortality from heart failure are of little value when a patient's life expectancy is severely curtailed as a result of other irreversible disorders. They also point out that medication intended for primary or secondary disease prevention are inappropriate in the frail elderly. Their trial conclusion is that the focus in prescribing for the elderly should be oligopharmacy.

Age and Ageing 2011;40:419–22.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Ralph Marcus Lawson (Toby) Whitlock

1934–2011

Dr Toby Whitlock, clinical physiologist and medical leader, grew up in Wellington and attended Victoria University before undertaking his medical training in Dunedin.

In 1960 he relocated to Auckland for his 6th year and house surgeon years during which time he nurtured an interest in the new specialty of intensive care.

After a year in general practice in Mangakino to work off his Health Department bursary, he took up the position of the first intensive care registrar in Auckland.

There Toby developed an interest in instrumentation, with monitors, respirators and other gadgets becoming a significant part of his life.

Such was his interest that he interrupted his MRACP preparation to complete a postgraduate degree in Medical Engineering at Imperial College in London

After a crash course in respiratory physiology at Hammersmith Hospital, Toby returned to a temporary post as "Acting Physician in Clinical Physiology" at Green Lane Hospital with Dr Edward Harris. The position became permanent after he gained his FRACP in 1978.

Along with clinical work Toby devoted a substantial amount of time to very productive and rewarding cardiorespiratory research and implementation of the findings into clinical practice, working with Ed Harris, Eve Seelye, Brian Barratt-Boyes and others. His major contributions were in the fields of computerised instrumentation, pacemaking and statistics.

Toby was a tireless worker, a truly caring individual and also a leader. He accepted the role of as "Acting Medical Superintendent" for 3 months in 1987. His tenure extended to nearly 10 years until the position was disestablished. He served on many committees including the Green Lane Research and Educational Fund (which he chaired for 16 years), Auckland Hospital and Citizens Trust Board, and Auckland ethics committees.

Most regard Toby's greatest contribution to medicine to have been the mentoring and support of others—colleagues, technologists and registrars to further their education and achieve their potential. He took great joy in seeing them advance in their careers. Through his encouragement and facilitation, many clinical tasks such as respiratory function testing, Holter monitoring, echocardiography and cardiac perfusion (all originally performed by doctors) became the domain of technologists.

Toby was a father figure for the staff of his beloved Physiology Department and he genuinely cared about each and every one of them. He had a talent for remembering details about individuals such as their birthday or where they came from. He also had a great wit and a sense of fun and was voted Man of the Year by Physiology Department staff many times, before being awarded the unprecedented “Man of the Year Forever” title.

Toby Whitlock loved life and loved his family. He is survived by Valerie, his wife of 52 years, 3 sons, a daughter and 9 grandchildren

Peter Ruygrok (Consultant Cardiologist, Auckland City Hospital) wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

NZMJ publication dates for 2012

Please see the following table for upcoming *NZMJ* publication dates through to the end of 2012.

Upcoming publication dates and themes (updated occasionally) can also be accessed at http://journal.nzma.org.nz/journal/themesheet.pdf via the *How to contribute* link on the left side of the *NZMJ* website.

Year	Month	Date	Theme
2011	November	4	
		25	
	December	16	
2012	January	20	
	February	10	
		24	Cancer
	March	9	
		30	
	April	20	
	May	11	Respiratory disease
		25	
	June	8	
		29	
	July	28	
	August	10	Surgery
		24	
	September	7	
		21	
	October	12	
		26	
	November	9	
		23	
	December	14	

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Cleveland Clinic's Current Clinical Medicine (2nd edition)

William D Carey. Published by Saunders (Elsevier Australia), 2010.
ISBN: 978141606643. Contains 1376 pages. Price AU$109.80 (special online price)

Competition is good for business and a new medical text book certainly focuses the mind. The authors of second edition of the Cleveland Current Clinical Medicine have successfully applied new ideas by creating this hybrid online and hard-copy textbook.

Written by staff employed at the Cleveland Clinic it maintains a cohesive and compact format. It is certainly readable cover to cover for medical students or health professionals preparing for medical examinations.

The current text consistently sees itself as only one part of the hard-copy/online framework. Each chapter refers to online resources, international guidelines, and is also available on the publisher's website. All chapters are short—3 to 4 pages on average—focussed and the book is comprehensive by covering several social topics, dermatology, psychiatry and preventive medicine.

Compared to one of the standard text at our institutions, *Kumar and Clarke's Clinical Medicine*, it appears to be more conservative in its use of colours, diagrams and 'ask the author' hints. Some may find this less distracting. The structure of chapters is in strict alphabetical order and naturally based on American classification of diseases. For example, Asthma is found in Chapter 1 under Allergy and Immunology, Venous Thromboembolic disease is found in Chapter 2 under Cardiology, whereas most other respiratory topics are found in the Pulmonary chapter. This chapter includes lung cancer, although several other cancers are under chapter haematology and oncology. This of course is of no relevance if one does indeed use the search engine of the online version.

Compared to the online module Up-to-date, the online version of this textbook is slow, and scrolling of the text is difficult as every section of a chapter is loaded independently. However, context is well integrated to the hard bound copy and it is part of purchase of the price and doesn't need to be purchased separately.

Altogether, this is a comprehensive resource which provides an alternative for students and other health professionals to standard texts. I can recommend this medical text book for learning, revision and as reference standard.

Lutz Beckert
Respiratory Physician, Respiratory Medicine
Christchurch