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This Issue in the Journal

Societal costs of obstructive sleep apnoea syndrome

Philippa H Gander, Guy Scott, Kara Mihaere, Helen Scott

Obstructive sleep apnoea syndrome (OSAS) is a treatable disorder in which people experience multiple breathing pauses during sleep. Untreated OSAS is associated with increased risk of other health problems and increased sleepiness, causing increased risk of accidents. This paper estimated the societal costs of OSAS among New Zealanders aged 30–59 yrs at \$40 million per year (range \$33–\$90 million). The analyses suggest that treatment is very cost-effective, by comparison with pharmaceutical treatments that the government already funds for other conditions. Thus, this economic analysis supports the case for improving the availability and accessibility of services for the diagnosis and treatment of OSAS.

Exploring knowledge and attitudes of taxi drivers with regard to obstructive sleep apnoea syndrome

Ridvan T Firestone, Philippa H Gander

Obstructive sleep apnoea syndrome (OSAS) is a medical condition where a sufferers' airway is repetitively blocked completely or partially during evening sleep. It results in fragmented sleep, daytime sleepiness, high risk of motor vehicle accidents, and poor cognitive functioning. There is research that suggests that OSAS may be highly prevalent among professional drivers. Our study examined taxi drivers' attitudes about symptoms of OSAS (i.e. excessive daytime sleepiness, having a large neck size as a proxy measure of Body Mass Index (BMI), snoring, and whether they stopped breathing at night (as observed by their bed partners) and how these views influenced their health and safety behaviours as professional passenger drivers. We found that there is a lack of knowledge about OSAS symptoms and how they are managed by the health professional, driver, and company managers. This lack of awareness has led to drivers avoiding addressing these issues due to fear of loss of employment and income. Clear guidelines for professional drivers, company managers, and healthcare professionals on the diagnosis and management of sleep disorders among drivers, and the potential consequences for driver licensing is needed.

Improved speech discrimination after cochlear implantation in the Southern Cochlear Implant Adult Programme

Justine Bradley, Philip Bird, Penny Monteath, Elisabeth Wells

The Southern Cochlear Implant Programme provides cochlear implant services for adults and children with severe to profound deafness in the lower North Island and South Island. This study looked at the ability of adult patients to understand speech following cochlear implantation. The results show a huge improvement in the ability to understand speech, which compares highly favorably with results throughout the

world. Older adults were able to benefit just as much as younger adults. The majority of the improvement occurred within six months of the people receiving their cochlear implants. Cochlear implantation can provide enormous improvements in hearing and quality of life for adults with severe to profound deafness.

Nasal fractures: patient satisfaction following closed reduction

Rachelle L Love

Nasal bone fracture can change the way the nose functions and looks. Surgery after nasal bone fracture aims to restore the nose to a satisfactory position. Results of surgery reported in the literature are mixed, with some authors advocating extensive surgery at the start in order to avoid the need for further surgery later on. This study demonstrates that manipulation of the nasal bones during a brief general anaesthetic is successful in restoring function and appearance in most patients and that few require further corrective surgery.

Self-dilation for refractory oesophageal strictures: an Auckland City Hospital study

Kenneth K S Wong, Dagmar Hendel

The oesophagus (gullet) may be narrowed as a result of injury and this may cause difficulty in swallowing. Traditionally, the area of narrowing can be re-expanded using a gastroscopy or video tube study but some patients need repeated procedures because of recurrent narrowing. The cost of repeated gastroscopies is expensive; patients can instead elect to use self-dilators which are specialised tubes that can be inserted by the patient through the oesophagus and are cost-effective as patients can do this without gastroscopy guidance. We report our experience of Auckland City Hospital patients using self-dilators and demonstrate that self-dilators are well-tolerated, easily administered and associated with minimal adverse outcomes. However, patients need proper education and ongoing support for this treatment to be effective.



Obstructive sleep apnoea syndrome

Jeffrey Garrett

Gander et al should be commended for the two informative studies on the obstructive sleep apnoea syndrome (OSAS) published in this edition of the *New Zealand Medical Journal*.^{1,2} Utilising a conservatively applied health economics analysis, they estimated a total annual societal cost of NZ\$40 million for untreated OSAS in the 30–60 year old age group. Acknowledging the imprecision of the measurement they have estimated the range to be \$33–90 million.

The findings are in keeping with previous international analyses,^{3,4} although more conservative than the *Wake Up Australia* study which estimated that sleep disorders affect 6% of Australians at a cost of AU\$10.3b/year.⁵

Gander and her team estimate that it costs NZ\$419 for not treating a patient versus \$389 incremental cost for the treatment of a patient. This makes OSAS treatment one of the most cost-effective therapies available within the health system. Indeed the estimated direct medical cost per Quality of Life Year (QALY) was \$94 compared with the average QALY cost of \$6865 for drug therapy paid by PHARMAC for all disorders.

Despite a large number of publications on OSAS by Gander and her team^{6–11} and which should have been sufficient to provide the basis for informing a national strategy on OSAS no such strategy exists. There remains no systematic approach to the investigation and management of sleep disorders and no public health funding allocated. What services are available, are fragmented and incomplete.

Funding is patchy and at the whim of an individual district health board (DHB). Consequently funding is often sought from other sources such as Work and Income NZ (WINZ) and Accident Compensation Corporation (ACC) by practitioners desperate to provide treatment for patients with a range of sleep disorders. Consequently there is no site on the Ministry of Health (MoH) website to inform either health professionals or the public on OSAS or on any of the large range of other sleep disorders. Even the MoH website publication on obesity¹⁰ makes no mention of OSAS despite the strong correlation between obesity and OSAS.¹¹ Indeed 70% of patients diagnosed with OSAS are obese.

The lack of a National Health Strategy for OSAS, has therefore led to a substantial variation in standards of healthcare delivery in New Zealand. In 2006, a review of all respiratory disorders in New Zealand revealed 5-fold variation in both the investigation and treatment of OSAS.¹²

The estimated number of sleep studies performed per year in New Zealand in 2006 totalled 50/100,000 compared with 282/100,000 in Australia and 427/100,000 in the US. The publication of these results in 2009 drew widespread interest from the media and an outcry from the respiratory community.

The incumbent Minister of Health, Tony Ryall, when interviewed, stated that he was concerned by the results and that he wished to meet with members of the Thoracic Society of Australia and New Zealand (TSANZ). However despite a number of subsequent requests to his office, no meeting has ever eventuated and no change in either the structure or delivery of New Zealand respiratory health services including OSAS has occurred.

It is therefore of no surprise that Gander's team found a lack of knowledge about the causes of sleepiness and OSAS among a cohort of taxi drivers selected for being at high risk of OSAS. Of equal concern was the apparent lack of knowledge amongst the taxi drivers' GPs about sleep-related disorders. Worse, those charged with making our roads safer (Accident Compensation Commission, National Road Safety Commission and The New Zealand Land Transport Agency) have inadequate structures in place to either screen or educate drivers working in high-risk industries.

Whilst the airline industry has invested heavily in the investigation and management of fatigue amongst its pilots and has adopted strategies impacted upon by researchers including Gander,⁹ this has not been the case on our roads as no effective educational or occupational screening programmes exist for drivers of heavy trucks and buses.

Excessive sleepiness contributes significantly to accidents both within vehicles and at work, and certain professional groups are at particular risk—e.g. truck drivers and public passenger service drivers (bus, taxi). Further, Māori and Pacific people are more likely to suffer from insomnia and OSAS than Europeans.⁶⁻⁸

Disparities in sleep problems between Māori and Europeans may impact on disparities in other health outcomes, acknowledging the increased risk of hypertension, ischaemic heart disease, stroke and possibly diabetes.¹⁵ The MoH and the DHBs both have the stated aim of reducing disparities in health outcomes between Māori and Europeans, yet substantially underfund a treatment that is not only cost-effective but which could contribute to reducing disparities in health outcomes.

It is important that New Zealand develops a National Strategy for the management of Sleep Disorders. Its time for New Zealanders and Health Authorities to wake up!

Competing interests: None.

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Adult cochlear implants in New Zealand—a chronic funding issue

Robert G Gunn

In 1987, when two deafened women received New Zealand's first cochlear implants, the expectation was that the devices would be little more than an aid to lip-reading. The procedure was new and seemed expensive, but for patients no longer able to communicate with their families, nor to work, nor to interact in society in general because they had become profoundly deaf, the potential was exciting. Steadily improving digital speech processing technology and implant design has since resulted in progressively improved hearing outcomes for implanted patients.

As Bradley et al¹ confirm, adult cochlear implant patients in New Zealand are now experiencing very high levels of speech perception, often very soon after their devices are switched on. Yet large numbers of adult patients continue to languish on a long waiting list for funding for their cochlear implant.

We are in an era of steady advancement in technology, costs and patient expectations, but also one of increasing financial constraints. As clinicians, we always want the very best for our patients, and of course, patients and their relatives want the best of treatment. Demand for services will always exceed supply. In the absence of a rational and explicit system in New Zealand to measure cost-effectiveness, one cannot blame those who have felt the need to resort to emotion-laden publicity campaigns in the media, to trigger what are in effect politically-motivated funding decisions.

Cheng and Niparko² conducted a meta-analysis of 9 studies of the cost-utility of cochlear implants, resulting in an assessment of the health utility of a profoundly deaf adult without a cochlear implant at 0.54 (95%CI 0.52–0.56), on a scale of 0 (death) to 10 (perfect health). After a cochlear implant, the health utility increased to 0.8 (95%CI 0.78–0.82), an increase of 0.26, or almost 50%. At the costings of the time (1999), this resulted in a cost-utility for unilateral implantation of US\$12,787 per Quality-Adjusted Life Year (QALY), which was well within the then-currently accepted range for medical and surgical interventions.

In the United Kingdom, the National Institute for Health and Clinical Excellence (NICE) was set up in 1999 in an attempt to rationalise the assessment of the cost-effectiveness of both pharmaceuticals and of interventional procedures. The organisation also issues guidelines to the Primary Care Trusts, through which funding flows, regarding the implementation of the NICE recommendations.³ The threshold for incremental cost-effectiveness used by the NICE organisation is £20,000 per QALY. While there has been criticism^{4,5} of the use of the QALY as a measure of cost-effectiveness, it is at least rational and transparent, as well as allowing comparison between interventions from different specialties.

NICE, in January 2009, issued the results of its assessment of cochlear implants in children and adults.⁶ For unilateral implantation of deafened adults, the cost utility of

£14,200 per QALY was well under the £20,000 per QALY threshold for funding, resulting in a guideline that cochlear implantation should be made available for these patients. Furthermore, their cost-utility assessment resulted in a guideline that *bilateral* implants should be made available for all profoundly deaf children, and for deaf adults with additional disabilities such as blindness. As a result of the NICE guidelines, deaf patients in the UK now receive their cochlear implants in a timely fashion.

In most other developed countries which rely on an insurance-based health system, cochlear implants are covered by health insurance. In Australia, cochlear implants are on the government's Approved Prosthesis list,⁷ and hence have to be funded by insurers.

How does our funding compare?

The early implants in this country were funded by the New Zealand Deafness Research Foundation as a research project. When the early results were so promising, administration of the New Zealand Cochlear Implant Programme moved to the National Audiology Centre, which resulted in its funding being channelled via the Disability Support Services section of Vote Health, which funds hearing aid subsidies, walking frames, house modifications, etc for the disabled. In the intervening 23 years, implant technology has steadily improved and cochlear implantation has become a mainstream management of profound deafness internationally, yet the funding stream for adult implants remains locked into the limited Disability Support Services vote. This separates it from all other forms of surgically-treated hearing loss, where the funding is channelled through the same Personal Health vote as funds all other surgical interventions.

While in recent years, funding increases have generally allowed deaf children to receive a unilateral implant in a timely fashion, the same cannot be said of adults who have become profoundly deaf.

Patients with other forms of surgically-remediable deafness, such as that due to middle ear pathology, are allocated priority using the same Clinical Priority Access Criteria (CPAC scoring) system as is used to prioritise all other otolaryngological procedures, which is based on degree and duration of symptoms, impact on quality of life, risk of complications etc.

Under these criteria, an adult who has become bilaterally profoundly deaf would score much more highly than a patient with, for example, a patient with a moderate unilateral or even moderate bilateral conductive hearing loss due to tympanic perforation or ossicular pathology.

The latter patients (quite rightly) are eligible for surgical intervention by tympanoplasty, stapedotomy etc to alleviate their hearing disability, whereas the patient who has become bilaterally *profoundly* deaf because of inner ear pathology, and for whom a cochlear implant is the only possible way to restore their hearing, is instead assessed by a completely different set of access criteria, with a much higher threshold. This has resulted in a growing waiting list, currently numbering 92 in the two programmes combined, with base funding for 20 per annum.

Funding cochlear implants via the disability vote because it also subsidises hearing aids for those with lesser degrees of hearing loss seems no more logical than funding joint replacement surgery from the same source just because it also pays for walking sticks and frames, or cataract surgery because it subsidises spectacles for children at school. Why should the disability of loss of hearing be treated differently from that of loss of mobility or loss of vision? Why should many of the most severely deaf patients not be allowed appropriate and demonstrably cost-effective surgery when patients with relatively modest levels of hearing loss, as well as other forms of disability, are readily funded for surgery to alleviate it? Why should the CPAC-qualifying patient who has become profoundly deaf not be allowed surgery within the maximum 6 month period mandated for other elective surgery?

For those with health insurance, the situation is only slightly better. Some insurers will consider covering the cost of the surgical procedure, but not that of the prosthesis itself, which is the largest expense. Various irrational arguments have been given, such as “the need for revision” (survival curves for current implants⁸ in fact show a long-term failure/revision rate of less than 3%, well below that of routinely-funded joint replacement prostheses⁹), “the need for on-going care” (annual audiology costs are low), and so on.

Why will a private health insurer fund a joint prosthesis when a natural joint has failed, a middle ear implant when an ossicle has failed, but not a demonstrably cost-effective cochlear prosthesis when the cochlea has failed?

We *have to* do better for our severely-to-profoundly deaf adult patients in this country.

Competing interests: None known.

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Societal costs of obstructive sleep apnoea syndrome

Philippa Gander, Guy Scott, Kara Mihaere, Helen Scott

Abstract

Aim To estimate the societal costs of obstructive sleep apnoea syndrome (OSAS) in New Zealand and develop a simulation tool to evaluate treatment options.

Method Treatment profiles, availability, uptake, and costs were based on services in the Wellington Region, and were used to develop a decision analytic model with micro costing of each potential outcome. Sensitivity analyses were conducted with 10,000 Monte Carlo simulations randomly varying each model parameter between high and low estimates.

Results Total annual societal costs of OSAS for New Zealanders aged 30–60 years were estimated at \$40 million (range \$33–\$90 million) or \$419 per case, with accidents being the major contributor. This included 58% direct medical, 13% direct non-medical, 25% indirect, and 3% intangible costs. The estimated incremental net cost of treating OSAS was \$389 per case treated (range \$338–\$427). The estimated incremental net direct medical cost per quality of life year (QALY) gained was \$94 (range \$56–\$310).

Conclusion The estimated incremental direct medical cost per QALY gained by OSAS treatment is well below the average QALY cost (\$6865) for drugs selected by PHARMAC to receive government subsidy for use in the healthcare system. Thus, the analysis strongly supports the cost effectiveness of OSAS treatment.

Obstructive sleep apnoea syndrome (OSAS) is a progressive disease that forms part of a spectrum of sleep-related breathing disorders. It is characterised by the occurrence of repetitive episodes of airflow reduction (hypopnoea) or cessation (apnoea) due to upper airway obstruction during sleep, accompanied by excessive daytime sleepiness.¹

Untreated OSAS is recognised as an independent risk factor for hypertension, cardiovascular disease, stroke, and motor vehicle accidents (due to excessive sleepiness).^{2,3} Evidence for OSAS as an independent risk factor for Type 2 diabetes is less clear. However evidence is converging from experimental sleep restriction studies, epidemiological studies, and intervention studies, to support the conclusion that OSAS exerts independent adverse effects on glucose regulation, through multiple mechanisms.⁴ Adults with undiagnosed OSAS are high users of health care services.⁵

In New Zealand, there are marked regional variations in funding for diagnosis and treatment of OSAS,^{6,7} which most commonly involves a device to maintain continuous positive airway pressure during sleep (a “CPAP machine”). In the age range 30–59 yrs, Māori are more likely than non-Māori to report OSAS symptoms and risk factors and to have OSAS.^{8–10} However, ethnicity is not an independent risk factor after controlling for body mass index (BMI) or neck circumference.^{8–10} There is also

evidence that Māori seen at sleep clinics have more severe OSAS than non-Māori, suggesting that there may be barriers for Māori in accessing specialist services.^{11,12}

The present study was undertaken to estimate the societal costs of OSAS among people aged 30–60 years, and to develop a simulation tool that could be used to evaluate treatment options, and to estimate the economic impact of OSAS on different population groups.

Methods

Study design—This study was undertaken in Wellington in 2005 and is a combination of cost of illness (COI), cost-benefit analysis (CBA), and cost utility analysis (CUA). The approach was based on an outcome tree and decision analytic model developed using three sets of information:

- The possible pathways of people with OSAS who seek treatment through the healthcare system, and of those who do not seek treatment, and the consequences of decisions made at each point in those pathways;
- The increased risk of accidents and comorbidities associated with untreated OSAS; and
- The costs associated with diagnosis and treatment versus not being diagnosed and/or successfully treated.

The prevalence of OSAS in the Wellington region was estimated to be 5.61% (95%CI 2.62–8.60%).^{9,10} The economic analysis was based on 1,692,260 people in the New Zealand population aged 30–59 years.¹³

Pathways through the healthcare system—The pathways in the outcome tree were based on services in the Wellington region (Drs Alister Neill and Angela Campbell, personal communication, 2005, unless otherwise referenced). The first point of contact with the healthcare system for a person seeking treatment for OSAS was a general practitioner (GP), who could recommend no further action, conservative therapies, or provide a referral to the respiratory medicine clinic at the Wellington public hospital. The number of patients who sought treatment for OSAS from their GP was unknown, and capped funding for treatment services beyond primary care made it impossible to estimate the proportion of OSAS sufferers who accessed such services. We have assumed that about 20% of people with OSAS sought treatment from their general practitioner,¹⁴ and of these 50% were referred on to the respiratory medicine clinic at Wellington Hospital.

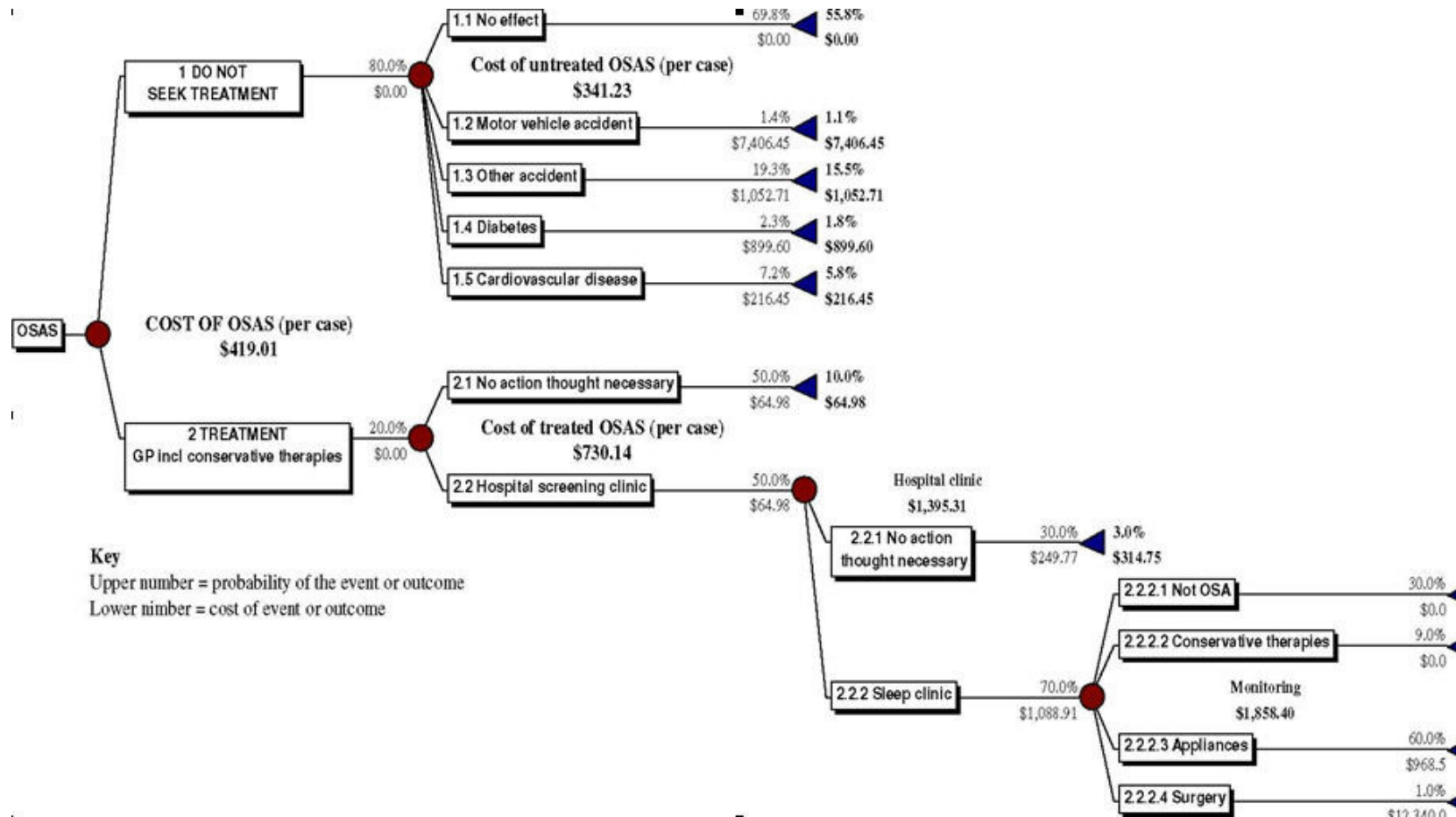
At the respiratory medicine clinic, patients were triaged based on a clinical evaluation that did not include sleep monitoring. This led to three possible outcomes: no further treatment recommended; referral back to their GP; or referral to the local specialist sleep clinic (WellSleep). At the time of this study, about 70% of patients seen at the respiratory medicine clinic for suspected OSAS were referred on to the sleep clinic for polysomnographic evaluation.

At the sleep clinic, patients underwent a clinical evaluation followed by an overnight polysomnographic sleep evaluation either in the clinic or at home. About 70% of patients evaluated had a diagnosis of OSAS confirmed. Treatment was initiated according to the severity of OSAS, and consistent with other aspects of the patient's health and life circumstances. An estimated 9% of patients seen at the sleep clinic were recommended conservative therapies for OSAS, 1% underwent surgical treatments, and the remaining 60% were treated with CPAP therapy or dental appliances.

The final version of the model (Figure 1) represents a simplification, with some very low probability outcome pathways excluded and others collapsed (for example, home-based and clinic-based polysomnographic monitoring are not considered separately). This was done in the interests of keeping a manageable level of complexity.

To account for uncertainties in the estimates of the proportion of patients following each trajectory, high and low probabilities were calculated as $\pm 25\%$ of the base case rate. For example, the base case estimate of the proportion of people with OSAS who sought GP treatment was 20%, with a low estimate of 15% and a high estimate of 25%.

Figure 1. The decision analytic model



Risk estimates—People with OSAS who did not seek treatment in the mainstream healthcare system were considered to be at increased risk for a number of adverse health and safety outcomes. Attributable fractions for each outcome were based on odds ratios from published meta-analyses of case-control studies, or from longitudinal studies. To account for uncertainties, base case and low and high estimates were calculated for each attributable fraction (Table 1).

Table 1: Attributable fractions for health and safety outcomes associated with untreated OSAS; base case (low estimate-high estimate)

Variables	MVAs	Other accidents	Diabetes	Cardiovascular Disease
OR for untreated OSAS	2.52 (1.83–3.45) ^a	2.2 (1.3–3.8) ^b	1.62 (0.67–3.65) ^c	2.0 (1.42–2.90) ^d
Risk without OSAS	0.09% (0.07–1.0%)	36.1% (27.7–43.8%)	4.0% (2.9%–8.5%)	8.6% (6.8–10.8%)
Risk with OSAS	2.3% (1.3–3.5%)	55.4% (33.3–74.8%)	6.3% (2.0–25.4%)	15.8% (9.4–26.0%)
OSAS-attributable fraction	1.37% (0.6–2.45%)	19.31% (5.55–30.95%)	2.31% (0.00–16.89%)	7.23% (2.60–15.20%)

MVAs=motor vehicle accidents.

^a Pooled OR (95%CI) from a meta-analysis of studies comparing MVA rates for people with and without OSAS.¹⁵

^b Independent OR (95%CI) for being involved in a workplace accident over a 10-year period, for men who reported snoring and workplace sleepiness at the start of the study, compared to those who did not. Adjusted for age, BMI, weight gain, years at work, and other workplace exposures.¹⁶

^c Independent OR (95%CI) for developing diabetes mellitus at 4-year follow-up, based on AHI at study start, (AHI≥15 compared to reference group AHI<5 at study start). Adjusted for age, sex, and body habitus.¹⁷

^d Independent ORs for the presence of hypertension at 4-year follow-up, based on AHI at study start, (reference group AHI=0 at study start). Low estimate is iOR for AHI=0.1–4.9, base case estimate is iOR for AHI=5–14.9, high estimate is iOR for AHI≥15. Adjusted for baseline hypertension status, BMI, neck and waist circumference, age, sex, and weekly use of alcohol and cigarettes.¹⁸ These values were used in the absence of reliable estimates of the increased risk of CVD associated with OSAS.

The incidence of motor vehicle accidents on the road, and of other accidents, was estimated from 2005 Accident Compensation Corporation claims,¹⁹ together with the national population data for 2005.¹³ The prevalence of diabetes was taken from the data for New Zealand adults in 2002/03.²⁰ The base case was taken as the average prevalence for males and females in the total population (4.1%). The high value was the average prevalence for Pacific people (10.0%) and the low value was the average prevalence for non-Pacific, non-Māori people (2.9%). The prevalence of cardiovascular disease (CVD) was derived from the data for New Zealand adults in 2003/03.²⁰ The base case was taken as the average prevalence for males and females in the total population (9.0%). The high value was the average prevalence for Māori (12.1%) and the low value was the average prevalence for Pacific people (6.9%).

Resource utilisations—At each node in the outcome tree, events occur and resources are consumed. For example, a person with diabetes may consult a general practitioner, have prescriptions dispensed, and incur loss of earnings and transport costs. These resource utilisations are summarised in Table 2.

Cost estimates—Costs were categorised as direct medical, direct non-medical, indirect, and intangible costs.²¹ Both human capital and willingness to pay approaches were used to place a value on a human life. In addition, we calculated a direct medical cost per quality of life year (QALY) gained if OSAS was successfully treated.²²

Only incremental costs (compared with the counterfactual) were included. For example, if a particular medical cost would have been incurred whether or not an accident happened, it was not included in the analyses. The timeframe was one year, so that discounting of costs and effects was not required. Unit cost estimates are summarised in Table 3 (excluding GST of 12.5%).

Table 2. Resource utilisations by event

Events →	1 Do not seek treatment					2 Treatment sought from general practitioner							
	No effect	Motor vehicle accident	Other accident	Diabetes	Cardiovascular disease	No action thought necessary	Referral to hospital clinic	Hospital clinic		Result of monitoring			
	1.1	1.2	1.3	1.4	1.5	2.1	2.2	2.2.1	2.2.2	Not OSA 2.2.2.1	Conservative therapies 2.2.2.2	Appliances 2.2.2.3	Surgery 2.2.2.4
Resources ↓	Units of resource utilised by event												
1 Direct medical													
1.1 General practitioner				2	2	1	1						
1.2 Hospital clinic consultation								1	1			2	2
1.3 Monitoring									1				
1.4 Appliances												1	
1.5 Surgery													1
1.6 Motor vehicle accident direct medical	1												
1.7 Other accidents direct medical			1										
1.8 Diabetes prescriptions				1									
1.9 Cardiovascular disease prescriptions					1								
2 Direct non-medical													
2.1 Transport private motor vehicle				2	2	1	1	1	2			2	2
2.2 Motor vehicle accident direct non-medical	1												
2.3 Other accidents direct non-medical			1										
3 Indirect													
3.1 Average hourly earnings				2	2	1	1	1	4			2	40
3.2 Motor vehicle accident indirect	1												
3.3 Other accidents indirect			1										
4 Intangible													
4.1 Motor vehicle accident intangible	1												
4.2 Other accidents intangible			1										

Table 3. Unit cost estimates

Cost item	Base case	Source
OSAS treatment direct medical		
General practitioner ^a	\$37.43	Average adult consultation fee ²³
Respiratory medicine clinic	\$222.22	Initial consultation fee for medical practitioner band III ²⁴
Sleep clinic diagnosis	\$768.89	Clinic average (Dr Angela Campbell, personal communication).
Appliances	\$468.97	Average cost to patient of CPAP device (some are subsidised; Dr Angela Campbell, personal communication, 2005)
Surgery ^b	\$11,029.20	AR-DRG v 5.0 (based on 20% tracheotomy, 80% maxillofacial surgery) ²⁵
Untreated OSAS direct medical		
Motor vehicle accident on the road	\$1,533.24	Average ACC payment for medical, hospital, public health acute, and dental services ^{19,26}
Other accident	\$469.47	Average ACC payment for all other types of accident ^{19,26}
Diabetes medications (per person per year)	\$769.63	12 prescriptions for control, 4 for monitoring glucose levels, plus prescription dispensing fees ²⁷
Cardiovascular disease medications (per person per year)	\$86.49	4 prescriptions, plus prescription dispensing fees ²⁷
OSAS treatment direct non-medical		
Private motor vehicle transport for treatment and diagnosis	\$6.20	Average reimbursement of 62 c/km for a 10 km round trip ²⁸
Untreated OSAS direct non-medical		
Motor vehicle accident	\$2,306.71	Average costs for vocational and social rehabilitation and conveyance ^{19,26}
Other accident	\$176.03	Average costs for vocational and social rehabilitation and conveyance ^{19,26}
Untreated OSAS indirect		
Production loss	\$21.35	Average hourly earnings ²⁹
Motor vehicle accident	\$2,735.01	Average income maintenance and independence allowances ^{19,26}
Other accident	\$380.76	Average income maintenance and independence allowances ^{19,26}
Untreated OSAS intangible		
Motor vehicle accident	\$831.48	Average weighted death benefit ^{19,26}
Other accident	\$26.45	Average weighted death benefit ^{19,26}

^aThe cost of counselling or conservative therapy was assumed to be included in the consultation costs for the GP or at the respiratory medicine clinic.

^b Surgical treatments for OSAS are changing, with tracheotomy becoming increasingly rare in this age group and gastric reduction becoming more common, particularly among patients who have private health insurance. Changes in the costings here do not make major differences to the total costings because of the very small proportion of patients receiving surgical treatments.

High and low values for each unit cost (except intangibles) were calculated as $\pm 25\%$ of the base case rate. The high estimates for intangible costs associated with accidents were calculated by multiplying the willingness to pay for a statistical life (\$2,830,000) ³⁰ by the proportion of accidents causing death (for motor vehicle accident high=\$28,239.93; for other accidents high=\$1,235.92). The low estimates for intangible costs associated with accidents were calculated by multiplying one year's average earnings (lost due to death) by the proportion of accidents causing death (for motor vehicle accident low=\$444.66; for other accidents low=\$19.46).

Sensitivity analysis—The structure outlined in Figure 1 was represented in a spreadsheet model and 10,000 Monte Carlo simulations were run using randomly generated values between the high and low estimates for each model parameter.³¹ Multiple linear regression was then used to evaluate the effects of each model parameter (independent variables) on the total direct and indirect costs, and the total costs calculated by the model.

Results

Table 4 summarises the estimated total base case societal costs of OSAS in New Zealand, for people aged 30–60 years.

Table 4. Total societal costs (base case) generated by OSAS (cost of illness)

Variables	Untreated (n=75,949)		Treated (n=18,987)		Total (n=94,936)	
	\$ per case	\$ total	\$ per case	\$ total	\$ per case	\$ total
Direct medical	142.84	10,848,785	649.63	12,334,604	244.20	23,183,190
Direct non-medical	66.79	5,072,686	14.12	268,049	56.26	5,340,735
Sub total direct	209.63	15,921,471	663.75	12,602,654	300.46	28,524,125
Indirect	115.09	8,741,271	66.40	1,260,719	105.36	10,001,990
Sub total direct + indirect	324.73	24,662,742	730.14	13,863,372	405.81	38,526,115
Intangible	16.50	1,253,360			13.20	1,253,360
Total cost	341.23	25,916,102	730.14	13,863,372	419.01	39,779,475

The incremental costs associated with untreated OSAS among people aged 30-60 years were estimated at \$25.9 million per annum (\$341 per case). The total costs of treatment were estimated at \$13.9 million per annum (\$730 per case). The total costs of OSAS were thus estimated at \$39.8 million per annum (averaging \$419 per case).

For 90% of the Monte Carlo simulations, the estimated total cost fell in the range \$32.9-89.8 million, with the top three cost determinants being the prevalence of OSAS, and the cost and incidence of motor vehicle accidents. Figure 2 illustrates the breakdown of total base case costs. Lost productivity was the largest contributor to indirect costs, while the 3% of intangible costs relate to loss of life. Costs associated with accidents (motor vehicle and other) contribute 59% of the estimated total costs.

Figure 2. Breakdown of total base case costs of OSAS

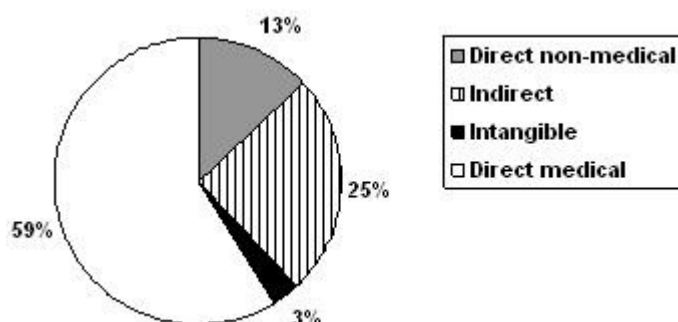


Table 5 shows the cost benefit and cost utility analysis for treating OSAS. It assumes a per case QALY gain of 5.4, with a low estimate of 0.10 and a high estimate of 8.00,^{22,32} and that 20% of people with OSAS are treated (low value 15%, high value 25%), for a total gain of 102,531 QALYs per year.

Table 5. Net incremental direct medical cost per QALY gained by OSAS treatment

Incremental cost/benefit/outcome	\$ per case	\$ total	cases
Costs incurred	649.63	12,334,604	18,987
Costs avoided	142.84	2,712,196	
Incremental cost per case treated	506.79	9,622,408	

Discussion

Previous studies that have estimated the societal costs of sleep disorders have generally taken a top-down approach.^{33,34} In contrast, the approach taken here using an outcome tree and decision analytic model yields a simulation tool that can be used to evaluate treatment options, and to estimate the economic impact of OSAS on different population groups. This is of particular interest, given the disproportionate burden of OSAS among Māori.⁸⁻⁹

For 2005, the estimated total annual societal costs of OSAS among New Zealanders aged 30–60 yrs was \$39.8 million, with 90% of the Monte Carlo simulations in the range \$32.9–\$89.8 million. Limitations of the data used to inform these cost estimates need to be considered.

OSAS prevalence is a key determinant of total costs and the base case prevalence used (5.6%) was higher than the estimated 4% in an Australian community study of men³⁵ and 2–4% in the Wisconsin Sleep Cohort.³⁶ Using much more restrictive criteria for the definition of OSAS (RDI \geq 5 plus ESS >10), we have recently estimated that the population prevalence of OSAS is 4.4% for Māori men, 4.1% for non-Māori men, 2.0% for Māori women, and 0.7% for non-Māori women.⁸ However, we expect that these estimates are very conservative. The Monte Carlo simulations in the present study included prevalence estimates of 2.6–8.6%.

Estimates of the increased risks of comorbidities and accidents associated with untreated OSAS are based, in the main, on studies that have focused on populations with severe OSAS. These are the only data available to inform these estimates and their applicability to the general New Zealand population is unknown. The focus on severe OSAS could have resulted in over-estimation of the costs associated with untreated OSAS in the present study.

On the other hand, a number of factors would have tended to make our estimates of the costs of untreated OSAS conservative. Medical costs would have been higher if hospital inpatient costs of cardiovascular disease and diabetes were included, and if a broader range of diseases had been included for which OSAS is a possible risk factor.

Our estimate of indirect costs included lost productivity for time off work but not absenteeism or low productivity while working.

Indirect and intangible cost estimates were based on ACC payments.¹⁹ However, the ACC database includes only those accidents for which claims were lodged, and which were judged as compensable under the scheme, so the incidence estimates are conservative and the costs represent the standards applied by the scheme, not necessarily all costs resulting from accidents.

The estimate of intangible costs is likely to be low because only those accidents causing death were quantified in dollar values. No attempt was made to quantify additional costs borne by family members as a result of living with an untreated OSAS sufferer (for example reduced productivity associated with having their sleep disturbed, or additional caregiving).

The outcome tree was based on patient trajectories in the Wellington region and may not be fully applicable to the variety of urban and rural settings in New Zealand, particularly since decisions on service provision are made at the level of district health boards, and services are not homogeneous nationwide.⁷ The profile of patients referred by GPs to the hospital screening clinic may be unrepresentative, since GPs who are better informed about OSAS may be more likely to make referrals.

The incremental direct medical cost per QALY gained by OSAS treatment was estimated to be \$94 (5th percentile \$56, 95th percentile \$310). From 1998–2005, decisions made by PHARMAC reflected an average cost per QALY gained of \$6865.³⁷ Thus, this economic analysis strongly supports the cost effectiveness of OSAS treatment by comparison with pharmaceutical treatments that the government already funds for other conditions.

A survey in late 2006 found that 12 of the 21 District Health Boards had a specified budget for the management of sleep-related breathing disorders, with the remaining 9 DHBs having referral pathways to other DHBs.⁷ The estimated number of sleep studies conducted (including laboratory-based and home-based polysomnographic studies and partial sleep studies without polysomnography), totalled 50/100,000 per year.

By comparison, for Australia the average was estimated at 282/100,000, for Canada 370/100,000 and for the USA 427/100,000.

Competing interests: None.

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Exploring knowledge and attitudes of taxi drivers with regard to obstructive sleep apnoea syndrome

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Abstract

Aim To examine the attitudes of taxi drivers towards symptoms of obstructive sleep apnoea syndrome (OSAS), and to determine whether these attitudes could influence their health and safety as a professional driver.

Method Qualitative research based on three focus groups conducted in Wellington, New Zealand. Participants were 27 taxi drivers who had a high pre-test risk for obstructive sleep apnoea. Assignment to focus groups was based on self-identification as being Māori and Pacific peoples, New Zealand European, or non-Māori and non-Pacific.

Results Participants described avoidance of health issues and dissatisfaction with their general practitioners. These attitudes were attributable to: (i) lack of knowledge, (ii) deliberate avoidance, and (iii) fear of loss of employment and income.

Conclusions The attitudes and level of knowledge of the focus group participants lead us to make the following recommendations. Drivers need systematic education about the effects of insufficient sleep and of OSAS on driving skills and safety. Taxi managers and drivers should cooperate to develop and implement safe driving policies to manage driver fatigue. Clear guidelines are needed for drivers, managers, and healthcare professionals on the diagnosis and treatment of sleep disorders among drivers, and their potential consequences for driver licensing.

In New Zealand, motor vehicle accidents (MVAs) account for over 30% of all deaths from external causes.¹ Alcohol, speed, and not wearing seat belts are commonly identified as causal factors,¹ however driver sleepiness and fatigue are estimated to contribute to 20% of all injury crashes.²

Obstructive sleep apnoea syndrome (OSAS) is a medical condition that is associated with elevated sleepiness and a heightened risk of MVAs.³⁻⁵ This condition is characterised by repetitive episodes of complete or partial upper airway obstruction that occur during sleep, and are usually terminated by brief arousals.⁶ Disease severity is defined by the number of apnoea (complete obstruction) and hypopnoea (partial obstruction) events per hour of sleep, as measured by the apnoea-hypopnoea index (AHI).⁶⁻⁸

The most common treatment for OSAS is nasal continuous positive airway pressure (CPAP), which splints the airway open during sleep and significantly improves daytime sleepiness.^{9,10} The profile of risk factors for OSAS suggests that prevalence may be high among professional drivers. These include being male, middle-older age, and having an elevated BMI, which may be exacerbated by the sedentary lifestyle of professional drivers.

Our recent study (n=243) estimated a high proportion (15%) of moderate to high risk of OSA among a sample of New Zealand-based taxi drivers.¹¹ In that study, our prediction equation was used to estimate the probability of having at least 15 respiratory disturbance events (apnoeas) per hour of sleep, using the respiratory disturbance index ($RDI \geq 15$). The equation included the following variables: being male, increasing age, increasing neck size, excessive daytime sleepiness ($ESS > 10$), snoring 'always', and reported observed apnoeas. These variables have been shown to be consistent predictors of OSA,¹² and thus it is considered to provide reliable estimates of *a priori* probability of OSA with 71–80% sensitivity and 81–86% specificity. This predictive model is in the process of being prospectively validated.

An Australian study of commercial vehicle drivers (n=2342) found 16% had OSAS.¹³ A recent study found an unexpectedly high prevalence (77.7%) of OSA (at least five respiratory disturbances per hour of sleep, $RD \geq 5$) among 153 Israeli professional drivers,¹⁴ of whom 47.1% were classified as sleepy, and 19% had severe sleepiness (classified as an average sleep latency on the multiple sleep latency test ≤ 5 minutes). Another study of 216 Hong Kong bus drivers found over 9% had OSAS, as defined by having an $RDI \geq 5$ and having excessive daytime sleepiness (Epworth Sleepiness Score ≥ 10).

There is minimal information available about the prevalence of sleep disorders among professional taxi drivers, who are at elevated risk for MVAs because of the extended time they spend driving, compared to non-professional drivers. In New Zealand, the taxi industry has recently undergone significant deregulation, which some industry participants believe has resulted in a progressive decline in drivers' working conditions and a surplus of taxi cabs and drivers.¹⁵ Some drivers maintain that this has forced them into working longer hours in order to earn a living.¹⁶

The increased MVA risk among untreated OSAS sufferers has drawn the attention of regulatory authorities in a number of countries, including New Zealand and Australia.^{17–19} The debate centres on whether OSAS sufferers should be allowed to hold certain categories of driving license, and on the conditions under which treated OSAS sufferers should be allowed to continue to drive. A major difficulty is predicting who is at elevated risk for MVAs, because the severity of sleep disordered breathing is not reliably correlated with measures of daytime sleepiness.

Risk assessment for the individual OSAS patient currently remains a clinical decision based on a combination of objective and subjective information.²⁰ Clinicians may (but are not obliged to) advise the chief medical advisor outlining a drivers' medical and driving circumstances, and make appropriate recommendations (e.g. temporarily suspend a drivers' license pending adequate treatment of OSAS).¹⁸

In New Zealand, access to specialist services for the diagnosis and treatment of OSAS is through referral from general practitioners (GPs), who are also typically responsible for follow-up evaluation of the adequacy of treatment, should this be imposed as a condition for driver licensing.

The aim of this research was to explore the attitudes of professional taxi drivers around OSAS symptoms. In particular, the study sought to better understand how drivers' attitudes influence their behaviour with regard to managing their health and safety as a professional driver.

Methods

Two local taxi companies distributed study packages to their drivers, which included a two-page questionnaire and a consent form for participation in a focus group. A total of 125 consent forms were returned. For each participant, questionnaire responses were used to derive a pre-test risk of OSA, defined as having an $RDI \geq 15$, based on a multivariate predictive model.¹¹ Recruitment continued until there were sufficient numbers in each focus group.

The groups were conducted at the Research School of Public Health, Massey University (New Zealand), in an environment that offered a neutral context for drivers to interact freely and with anonymity from their company management. The focus groups were co-facilitated by one of the named authors (RF) and a qualified medical physician. Each group lasted two hours. Nominal group technique was employed to minimise influence and tangential discussion.²¹

In line with this technique, participants were asked to write their responses to a set of semi-structured questions (*“Think of a time when you were working [as a taxi driver], and you felt really tired, not refreshed, and sleepy on the job, now consider these following questions.” How did you feel working in this situation? Were you worried about how you could perform as a taxi driver? If you were worried, what did you do about it?*).

Each participant’s responses were transcribed openly, and common ideas and different responses were identified, and used to prioritise topics and initiate group discussion. The focus group methodology is described in detail elsewhere.¹⁶ Table 1 describes the characteristics of each focus group. The “Other Ethnicity” group was self-identified, and included drivers from Asia, Fiji-Indian, and northern and eastern European countries.

Table 1. Focus group characteristics

Group	Number of men	Number of women	Age (years)	Taxi-driving experience (years)
New Zealand European	7	3	36–66	3–30
Māori/Pacific	6	1	46–64	1–27
Other ethnicity	8	0	40–64	4–37

Results

One major theme identified from the analysis of the focus group transcripts,¹⁶ and which is the focus of the analyses presented here, was characterised as “driver avoidance of health issues and dissatisfaction with doctors”. Within this major theme, three sub-themes were identified, namely: ignorance, avoidance, and personal fear.

Ignorance

Ignorance was defined as a lack of awareness about the underlying causes of sleepiness, and the potential risks of sleepy driving, and a lack of knowledge about the availability of treatment services for OSAS. It included both driver ignorance and ignorance among medical professionals. The following excerpts typify driver ignorance.

Driver 21 added: “I think most people [are] not aware that this is a problem. It’s just one of the things that happen! ...I think it’s just a lazy type of job you just sitting down—ya got nothing else to do [laughs.]”

Driver 22: “...my problem [i.e. OSAS] was identified with snoring. My wife complained to my doctor and she was the one that started this whole investigation off.”

Drivers were generally unaware that a symptom such as sleepiness could be an indicator of an underlying medical problem. Some blamed the sedentary nature of the job, which was perceived as inducing daytime sleepiness. This demonstrates ignorance of the physiological basis of sleepiness. As expected, drivers who had OSAS symptoms during sleep were unaware of them, until family members highlighted these issues.

Some drivers reported good relationships with their GPs and attended regular check-ups. However, their involvement in the focus groups had clearly heightened their awareness about OSAS symptoms and the dangers of sleepy driving, and in return the drivers expressed concerns about their GPs lack of screening for sleep complaints as part of their routine medical check-ups.

Driver 11: I see my doctor 4 times a year minimum ... he's never asked me that question [sleep related]...

Driver 14 added: Yeah the doctor never asks about sleep. Never!

Driver 16 said: ... [My doctor] might say to me, oh [it's] this time of the month, you haven't had a prostate for 12 months, we'll check you over...If I was sleepy, I'd soon tell him that's for sure. But that's a personal thing, I guess ...

From the comments, it appeared that doctors were generally more concerned with evaluating the more familiar medical issues that could affect a driver's license renewal, such as visual acuity, hypertension, and other medical conditions such as diabetes, but even these conditions were not routinely assessed according to some drivers. Another facet of this problem was that some drivers did not think to mention sleep-related issues as a health concern, because they did not identify them as a priority, as illustrated by the following example.

Driver 11: You don't even think to tell the doctor when you get there, you don't even think about it ... you worry about your eyesight and what not, and so forth, not the sleeping business!

The overall impression obtained from the discussion was that many of the drivers' GPs are not well informed about sleepiness or sleep complaints, nor do they routinely assess symptoms of OSAS.

Avoidance

The second sub-theme, avoidance, was defined as a conscious decision by drivers not to reveal health concerns to their GPs. The following discussion illustrates this.

Driver 17: You tell him [doctor], and then they'll probably give you another test or something else and that will delay your certificate of driving, and that's probably the worst thing...a delay of another week ... it might delay you the last certificate for fitness, you know to go and get your licence, you see what I mean?

Driver 15 added: ...yeah might open a can of worms

Driver 17 continued: I mean if the doctor tells you 'excuse me sir, you're a little bit overweight, try and lose some weight,' you don't want to say 'what about my sleeping disorder?' 'I feel a bit tired!' You don't want to open up, you know? And I suppose all the taxi drivers ... feel like that too.

The issues raised by these two drivers led others into a group consensus. The first issue is withholding information in order to protect their employment status. Both drivers described actively concealing any health problems that might impact on their being assessed as medically unfit to drive. This is understandable in the prevailing

context where there was no standard format for the fitness-to-drive screening of professional taxi drivers carried out by GPs.

The second issue relates to withholding information from the doctor for personal reasons. Driver 17 raised the point that drivers who are overweight, and know that they have a sleeping problem, would not seek help from the doctor because they would feel embarrassed. Other reasons for avoiding seeing the doctor included both the cost of the consultation and any medications prescribed, and the loss of income from being off the road to attend the appointment.

Personal fear

The third sub-theme was personal fear. This was characterised by drivers not wanting to believe that something is physically wrong, or being apprehensive about finding out about further or more serious health conditions that could compromise their ability to earn a living. A common element in this sub-theme was mistrust of other people's concerns about the driver's health.

Driver 17: ... I used to smoke until 3 years ago and my wife used to say 'you snore a lot you might as well you know try and stop smoking'. So three years ago I stopped but I'm still snoring [laughs with the group]. ... It's either your wife or somebody, your friend watching you while you're sleeping and they tell you that there's something wrong with your sleeping. But you never believe them because you don't know what you're doing while you're sleeping.

Fear of loss of income was a significant factor for the participants, who face considerable day-to-day variability in income and very limited alternatives for owner drivers if they get sick. One approach suggested was to seek additional information (e.g. on the internet) to assess the possible outcomes, before speaking to the doctor. This approach also avoids medical costs (e.g., medication, specialists, and laboratory tests).

Driver 24: If I knew I had apnoea and I was scared to go to the doctor because I might lose my license I might get some information first [like searching the internet or reading a book]. If apnoea was treatable then I'll go and seek help and treat it. If not, I'd probably shut up [laughs].

Taking time out from driving to look after personal needs was identified with loss of income, although it was recognised that failing to look after one's self could lead to larger health problems in the long term. Whether attributed to greed or need, this seemed to be accepted as part of the 'taxi culture', because of the competitive nature of the business.

Driver 20: I mean not everyone is hard fetched for cash. Some people are just like that you know. The first thing is to get another job ... maybe I'm wrong but that's how I look at it. A lot of people would do these things. They don't have any time for themselves. All they do is sit on the stand and work and work and work, and after a certain time and unless people decide no they have to make some times for themselves they have a problem and they got to go and see a doctor. They have to make the time you know.

The themes identified above reflect attitudes which may limit drivers' ability to recognise and act on OSAS symptoms or other sleep complaints. This raised the question of who they saw as being responsible for their health and safety. Some believed that their GPs were responsible, because they conduct the medical check-ups and provide a certificate, effectively declaring drivers as 'fit and safe' to drive, whilst others believed that the onus of responsibility lies heavily on the individual driver.

Discussion

This qualitative exploration of the attitudes of taxi drivers at high risk for OSA has highlighted a number of areas where strategies could be implemented to improve driver health and safety.

First, the lack of knowledge among drivers about the causes of sleepiness, including OSAS, and the associated driving risk, points to the need for better driver fatigue management education.

The National Road Safety Committee has developed an inter-agency *Driver Fatigue Strategy*.²² One of the deliverables in the strategy is a commitment to providing 'educative measures to assist drivers to modify their behaviour to reduce the incidence of driver fatigue'. In relation to commercial drivers, the Accident Compensation Commission is charged with workplace delivery of 'Managing Fatigue' training and raising awareness with heavy motor vehicle drivers. In addition, the New Zealand Land Transport Agency (NZLTA) is charged to ensure that the fatigue section of the "Your Safe Driving Policy" resource reflects the most up-to-date advice on managing fatigue.

From examining the NZLTA website in May 2009, advice for companies developing a "Safe Driving Policy" indicated that the policy must address driver fatigue, but fatigue management education was not discussed among the recommended strategies. Nor was it listed as a course to consider for driver training and education, although it was recommended that regular staff seminars or refresher meetings should cover fatigue as one of a number of listed topics.

With regard to the issues raised by the present study, it is not clear whether any of these measures will reach taxi drivers, most of whom either work as subcontractors or are self-employed, but pay dues to a taxi company that provides communication services and branding.

Another option would be to include driver fatigue management education as part of the approved course that drivers must undertake to get a P-endorsement licence to carry paying passengers. This approach means that the regulating authority takes responsibility for providing such education, but the responsibility remains with the individual drivers to apply this knowledge in their professional activity.

Fatigue management training has been shown to result in better knowledge levels and (self-reported) improvements in personal fatigue management strategies among professional tanker drivers.²³

Acknowledging the role of shift-work is important as well, particularly as sleep becomes displaced from its usual night position, and this can pose problems with neurobehavioral and cognitive performance.

Shift-workers tend to select their sleep-wake schedules because of their work commitments and this disrupts the synchronisation of internal sleep structure. This results in sleep deprivation, fragmented sleep, and complaints of excessive daytime sleepiness.²⁴ The issues around shift-work, sleep deprivation and other sleep disorders are a complex web of circadian, sleep and social factors, with each influencing the other and impacting on the ability to adapt and cope with daily pressures.²⁵ It is also

outside the scope of this study, but it is a necessary consideration in future research with professional taxi drivers.

Second, in the experience of these focus group participants, GPs did not routinely ask questions regarding sleepiness or OSAS symptoms, nor did they demonstrate knowledge or awareness of sleep problems in general, or the risks associated with sleepy driving. For example, some drivers were told that sleepiness was part of the normal aging process, or that they just needed to lose weight.¹⁶

These findings are consistent with previous research,^{26–28} which has also highlighted the need for continuing medical education programmes to up-skill GPs in sleep medicine. One of the deliverables in the national Driver Fatigue Strategy is to ensure that medical practitioners are aware of the effects of sleep deprivation and its contribution to driver fatigue.²²

The role of GPs is pivotal for managing taxi driver fatigue issues, because of their established rapport with their regular patients, especially since they act as a conduit for referral to specialist services for OSAS treatment and diagnosis, and because follow-up management of OSAS patients is often referred back to GPs. However, the relationship between taxi drivers and their GPs is complicated by the fact that GPs also have the responsibility for evaluating whether a driver is fit for work.

Both in the legally-required medical assessments for license renewal, and if significant health concerns are identified between the required assessments, a GP can notify the Chief Medical Advisor who may decide to suspend a driver's license. The drivers in the present study were acutely aware of this and admitted to actively concealing any health problems that might impact on their being assessed as medically fit to drive. Similar issues have been reported with other groups of professional drivers.^{13,14,27–29}

Clearer guidance from the NZLTA about the criteria for being considered medically unfit, and about returning to work subject to adequate treatment, could help diffuse some of these concerns, if drivers and their GPs knew about the criteria. Standardised forms for required medical assessments might also improve drivers' confidence and assist GPs in this process.

In the national Driver Fatigue Strategy, the NZLTA committed to reviewing the relevant sections of its 'Medical Aspects of Fitness to Drive' resource and related forms for medical practitioners, by December 2008. However, use of these resources is voluntary and it is unclear how many GPs are aware of them, or use them. Another possibility would be to have the fitness-to-drive medical assessments undertaken by trained occupational physicians who are not the driver's GP, as is the case for commercial airline pilots.

With regard to ongoing management of professional drivers with OSAS who are receiving treatment such as Continuous Positive Airway Pressure (CPAP), one approach used in the UK (Rosemary Gibson, personal communication, 2008) is to schedule an annual check-up at the sleep clinic, which includes downloading the data collected by the CPAP machine about the amount of time that it has been used. Threshold criteria for treatment compliance can be then used to trigger different actions. For example, if a driver's usage rate is at least 80%, then the driver's license can be renewed.

If the usage rate is between 50-80%, the driver is required to return for a further follow up at the sleep clinic in three months time. Lower rates of usage could trigger a referral back to the sleep specialist. This process could be reinforced by forwarding the names of non-compliant drivers to the regulator, in the interests of public safety. However, in New Zealand, funding for sleep apnoea services is directed at diagnosis and initial follow-up.

There is no funding for long-term follow-up of patients on CPAP due to limited resources (Dr Alister Neill, personal communication, 2008). It might have been interesting to explore the acceptability of this approach with drivers in the focus groups. However their lack of knowledge and experience with OSAS treatment would have rendered this discussion very hypothetical.

To improve the identification, diagnosis, and management of OSAS patients, additional research is needed to provide reliable screening tools for GPs, and to clarify measures to identify those individuals most likely to be at elevated risk for MVAs.²⁰ However, improvements in the identification and referral of patients with OSAS at the primary care level needs to be matched with an appropriate level and distribution of specialist services nationwide.³⁰

It would be also be useful to have a better understanding of the knowledge and awareness levels among GPs. The current study invited a random selection of GPs from the wider Wellington region to attend a focus group discussion on these issues. However, only one GP responded favourably, others were too busy to attend. At the time of this invitation, New Zealand GPs were preparing to roll out a national strategy of immunising infants against Meningococcal-B, which the responding GPs reported had higher priority.

Nevertheless, the role of GPs in managing professional drivers with OSAS is an important one. Further emphasis on the doctor's role to advocate for this particular group of patients is necessary, and this could be endorsed through professional development education workshops about sleep and sleep related disorders.

Implications for public health policy

The Ministry of Transport and its respective transport regulatory agencies, the Accident Compensation Corporation, and the Department of Labour are working together through the National Road Safety Committee's inter-agency strategy to combat driver fatigue. One of their key activities is raising awareness about the dangers of sleepy driving, for both professional drivers and private motorists. This can be expected to reduce the acceptability of professional drivers being sleepy at work, and increase the demand for healthcare services to manage chronic sleep disorders, including OSAS.

The Ministry of Health needs to become involved in strategic planning of healthcare services to meet these needs. Taxi company managers also need to be educated about, and enforce safe driving policies, so that individual drivers feel supported and are reminded about adhering to such policies.

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Improved speech discrimination after cochlear implantation in the Southern Cochlear Implant Adult Programme

Justine Bradley, Philip Bird, Penny Monteath, J Elisabeth Wells

Abstract

Aim To assess the rate and amount of improvement in speech discrimination scores following cochlear implant (CI) at the Southern Cochlear Implant Programme—Adult (SCIPA).

Methods A retrospective review of those implanted between 1999 and 2008 at SCIPA. We recorded type of implant, age of onset of deafness, age at implant, aetiology, and speech discrimination test (hearing in noise test, HINT) results pre-implant and over time post-implant.

Results The mean post-implant HINT score (auditory alone) for the 78 patients who had follow-up up to nine months was 74%, and at 18 months (52 patients) was 81%, compared with a mean pre-implant score of 15%. Performance plateaued at around 6 months post-implant. Age at operation was unrelated to outcome but earlier onset of deafness (and higher percentage of life deaf) gave a poorer outcome. Medium pre-implant scores were associated with lower post-implant scores than those with low or high pre-implant scores.

Conclusions Our results compare favourably with world-wide standards. Benefit in speech discrimination appears to plateau 6 months post-implant. A shorter duration of deafness or percentage of life spent deaf gives better results, but patients with very poor pre-implant functioning may in fact perform better at long term follow-up than those with slightly better pre-implant functioning.

Glossary of abbreviations

SCIP: Southern Cochlear Implant Programme

SCIPA: Southern Cochlear Implant Programme- Adult

HINT: Hearing in Noise Test (described in introduction)

HINT A: auditory cues alone

HINT AV: audiovisual cues

S/O: Switch- on of cochlear implant

BKB sentence test: Bench, Kowal and Bamford sentence lists—open-set sentence lists (i.e. response options not prescribed in advance) similar to HINT

CID sentence test: Central Institute for the Deaf everyday sentence lists- open-set sentence tests similar to HINT

K-CID sentence test: Korean version of the CID test

NVA speech recognition: Nederlands Vereniging Audiologie—Dutch open-set speech recognition tests consisting of monosyllabic (consonant-vowel-consonant) word lists

NU-6 words: Northwestern University Auditory Test No. 6: open-set lists of monosyllabic words

A cochlear implant (CI) is an electronic device which bypasses the auditory function of the outer, middle and inner ear and stimulates the cells of the spiral ganglion (and therefore the cochlear nerve) directly. Cochlear implants are indicated in severe and profound sensorineural hearing loss (SNHL) when hearing aids provide insufficient information for understanding speech.

Cochlear implantation was first performed in New Zealand in 1986.¹ An initially small national programme was based in Auckland. Cochlear implants were first performed in Christchurch in 1998. The Southern Cochlear Implant Programme (SCIP) was established in 2003. Located in Christchurch it serves the South Island and lower North Island. It is divided into separate Adult (SCIPA) and paediatric programmes. The SCIPA comprises two surgeons, two audiologists and a rehabilitationist. Potential candidates are referred to the programme, principally by audiologists and otolaryngologists, and undergo assessment by all three components of the CI team. Currently 80% of referrals are found to be suitable candidates.

The key audiologic test performed is aided speech discrimination, details of which are provided below. This testing aims to determine how well the candidates can utilise their aided hearing to understand speech. The same testing processes are used post-implant to assess the effectiveness of the CI.

Previous research has indicated that there are many factors contributing to the outcome of CI including age, duration of deafness/age at onset of deafness, anatomic issues as determined by radiology, other handicaps, speech and language abilities, functional hearing, family and social support, expectations/motivation, educational setting, communication mode, availability of support services, and the intensity of post-implant rehabilitation therapy.^{15,4}

Many studies have shown a significant negative correlation with duration of deafness,^{4,7,9,11,12} and a positive correlation with age at onset of deafness, hearing aid use, and progressive hearing loss.⁷ In particular, hearing loss prior to development of speech and language inhibits its development and also reduces the effectiveness of cochlear implantation later on.

There are differing findings in the literature regarding the time after CI that maximal benefit is obtained. Some studies indicate that this is reached as early as 3 months,¹⁰ others suggest it is as late as 2 years.⁴ This is an important factor to be able to counsel patients about before and after their CI, so they will have some idea of their likely long-term outcome as they carry out their rehabilitation.

In 2005 our Programme introduced the Nucleus® Freedom™ (Cochlear Ltd, Lane Cove, New South Wales, Australia) device which superseded the older technology from the same company of the Nucleus 22 and the later Nucleus 24 cochlear implants. Anecdotally we thought our patients were experiencing more rapid improvements in speech discrimination and slightly higher levels of peak performance, but wished to accurately investigate this.

The objective of this study was to assess the outcome with respect to improvement in speech discrimination test results after CI in patients enrolled at SCIPA, and to determine how long after a CI there is a plateau in performance. We also aimed to assess the effect of duration of deafness and percentage of life spent deaf on results, and the effects of a new CI device.

Methods

A retrospective review of all files of patients receiving follow-up in Christchurch for CI between 1999 and Dec 2008 was undertaken. Exclusion criteria were patients under age 18 (these would be under the Paediatric Programme), and patients with no or inadequate pre-implant data. Some patients had CI performed by the Southern Cochlear Implant Programme – Adult, then moved to the Northern Cochlear Implant Programme (NCIP) for follow-up. Most of these were included, as follow-up data was sent from the NCIP. Some patients had received their CI in NCIP and then moved to the SCIPA for follow-up. These were included if we had a copy of their pre-implant data sent from NCIP.

Files were obtained and the following were recorded: age, sex, type of implant: Nucleus® Freedom™ versus older devices – Nucleus 22 and Nucleus 24, age of onset of deafness, age at implant, percentage of life spent deaf (calculated from above), cause of deafness where known, and speech discrimination test results. We originally intended to measure both age at onset of hearing loss and age when hearing aids were first fitted, along with which sides were aided.

Due to lack of consistent information we were only able to estimate age of onset of deafness with any degree of accuracy. Speech discrimination was measured using the hearing in noise test (HINT), which is a standardized test of speech recognition using sentences delivered in a sound field of 55 dB HL sound pressure level. In determining cochlear implant candidacy, HINT is performed without background noise, despite its name. HINT consists of 25 equivalent 10-sentence lists, two lists are used at each assessment.

The tests are played on a DVD with a speaker/amplifier and are tested both with audiovisual (AV) cues (watching the presenter read the lists whilst listening) and with only auditory (A) cues (visual display turned off). The patient is scored for the percentage of words correctly identified on the lists. This is carried out pre-implant with their hearing aid on, at switch-on (s/o) of CI, then at 1 month, 3 months, 6 months, 9 months, 12 months, 18 months and 3 years post-implant where available.

The following were assessed: improvement in HINT A and AV over time post-CI, overall improvement at 9, 12 and 18 months, characteristics of patients lost to follow-up or with missing data, relationship between final outcome and duration of deafness, percentage of life spent deaf and type of implant used.

Statistical methods: All analyses were carried out using SAS 9.1. To investigate the time course of recovery up to 9 months after an implant, measures A and AV were analysed separately using repeated measures analysis of variance (PROC ANOVA) with time as the repeated measure (pre-implant, s/o, 1 month, 3 months, 6 months and 9 months), and a between subjects factor based on division of the patients into approximately three equal-sized groups based on their pre-operation scores.

The Huynh-Feldt adjustment for non-sphericity was used for repeated measures sources of variance. Chi-square tests and t-tests were used to compare those who had complete data up to 9 months and those who did not on pre-implant variables. Prediction of long-term outcome used data from 18 months if available and otherwise the 12 month values were used. Including 12 month values in this way increased the number of A outcome observations from 40 to 46 and the number of AV observations from 37 to 44.

Prediction of outcome was carried out using one way analysis of variance (PROC GLM). Because of ceiling effects, particularly for AV, Welch's test was used if Levene's test for the homogeneity of variance was significant with $\alpha=0.10$, although this precaution mostly made little difference.

Results

A total of 171 patients were on the cochlear implant rehabilitation files at SCIPA. Of this total, 57 were excluded for the following reasons: 11 had been implanted too recently for analysis (2 months or less), 12 had transferred from the Paediatric Programme so did not apply to our study, 17 had been implanted prior to 1997 so speech discrimination tests other than HINT had been used for their pre-implant and post-implant assessments, nine had no follow-up notes available, four were non-users

(discussed later), and four did not have pre-implant speech discrimination tests available.

A total of 114 patients were then left for analysis. However, among this group there was much missing data. Only 55 had complete data for A and 53 for AV. Comparison of those with complete data and the remaining patients showed no significant differences in any of the background characteristics or of pre-implant functioning.

We used complete data only for the analyses as imputation of data was deemed inappropriate due to the ceiling effect of the results. This was to ensure internal validity, i.e. comparing people across time for the same people. Pre-implant characteristics are shown in Tables 1–3, and the pre-implant scores on HINT are shown in Tables 4 and 5.

Table 1. Characteristics of those with a cochlear implant between 1999 and 2008

Variables	Number assessed	Mean (SD)	Range
Age (years)	113	50 (15.3)	20–83
Age (years) of onset of deafness	111	21 (19.4)	1–63
Duration of deafness (years)	111	30 (14.5)	1–63
Percentage of life deaf	111	64% (30.5)	2–99
HINT pre-implant A	112	15 (16.8)	0–65
HINT pre-implant AV	111	67 (23.9)	0–100

Table 2. Mean HINT AV scores (% correct) at pre-implant, switch-on (s/o), and 1–9 months post-implant: 53 patients

Variables	Mean HINT AV score	SD	Range
Pre-implant	66	24	0-100
s/o	82	17	32-100
1 month	93	10	67-100
3 months	96	9	57-100
6 months	96	6	74-100
9 months	98	4	79-100

Table 3. Mean HINT A scores (% correct) among those with complete data up to 9 months by grouping based on pre-implant scores (55 patients)

HINT A scores pre-implant	Number	Mean pre-implant score (SD)	Mean s/o score (SD)	Mean score at 1 month (SD)	Mean score at 3 months (SD)	Mean score at 6 months (SD)	Mean score at 9 months (SD)
0	18	0 (0)	33 (27)	60 (27)	71 (29)	77 (27)	83 (21)
1–15	17	6 (4)	23 (28)	44 (31)	53 (33)	65 (31)	69 (29)
16+	20	34 (12)	46 (29)	69 (29)	79 (22)	82 (24)	83 (25)
Total	55	14 (17)	34 (29)	58 (30)	68 (30)	75 (28)	79 (25)

Table 4. Mean HINT AV scores (% correct) among those with complete data up to 9 months by grouping based on pre-implant scores (53 patients)

HINT AV scores pre-implant	Number	Mean pre-implant score (SD)	Mean s/o score (SD)	Mean score at 1 month (SD)	Mean score at 3 months (SD)	Mean score at 6 months (SD)	Mean score at 9 months (SD)
0-60	18	39 (20)	75 (20)	93 (7)	94 (10)	96 (7)	97 (5)
61-78	17	69 (5)	80 (19)	92 (11)	93 (11)	96 (4)	98 (4)
79+	18	90 (7)	90 (9)	92 (11)	98 (4)	97 (6)	98 (4)
Total	53	66 (25)	82 (17)	93 (10)	95 (9)	96 (6)	98 (4)

There was significant heterogeneity in aetiology. The recorded causes included idiopathic, genetic, maternal rubella, meningitis, otosclerosis, endolymphatic hydrops and large vestibular aqueduct syndrome.

The mean post-implant HINT scores at nine months were 74% for A only (78 patients, range 11–100%), and 96% for AV (74 patients, range 62–100%). The mean scores at 18 months were 81% for A only (52 patients, range 14–100) and 98% for AV (50 patients, range 70–100). Improvement over time following CI was assessed by comparing HINT scores pre-implant with those at follow-up appointments, for both A and AV (Tables 6 and 7). As mentioned earlier, this was only possible for 55 patients for A and 53 patients for AV due to lack of complete data on the remaining patients.

Table 5. Age at operation vs longterm HINT A and AV scores: 58 patients

Age (years) at operation (number of patients)	Mean longterm A score (% correct) (SD)	Mean longterm AV score (% correct) (SD)
20–44 (22)	75 (28)	96 (7)
45–59 (19)	79 (23)	97 (5)
60+ (17)	83 (16)	99 (2)
p-values	0.46	0.33

Table 6. Age of onset vs longterm HINT A and AV scores: 56 patients

Age (years) of onset (number of patients)	Mean longterm A score (% correct) (SD)	Mean longterm AV score (% correct) (SD)
<3 (13)	63 (28)	95 (8)
3-19 (17)	79 (27)	98 (5)
20+ (26)	86 (14)	99 (2)
p-values	0.01	0.07

Table 7. Percentage of life deaf vs longterm HINT A and AV scores: 56 patients

Percentage of life deaf (number of patients)	Mean longterm A score (% correct) (SD)	Mean longterm AV score (% correct) (SD)
<50% (19)	88 (12)	99 (2)
51-89% (22)	80 (25)	98 (4)
90%+ (15)	64 (26)	95 (8)
p-values	0.006	0.11

We also assessed improvement by initial scores (for both A and AV) divided into three groups: Table 8 (A) and Table 9 (AV).

Table 8. Implant model (Nucleus Freedom vs Nucleus 22 and 24) vs longterm HINT A and AV scores: 58 patients

Implant (number of patients)	Mean longterm A score (% correct) (SD)	Mean longterm AV score (% correct) (SD)
Freedom (21)	89 (14)	99 (3)
Nucleus 22 & 24 (37)	73 (26)	97 (6)
p-value	0.01	0.15

Table 9. Pre-implant HINT A score vs longterm A and AV scores (% correct): 58 patients

Pre-implant A score (number of patients)	Mean longterm A score (SD)	Mean longterm AV score (SD)
0 (18)	82 (20)	99 (2)
1–15 (18)	67 (26)	95 (7)
16+ (22)	86 (20)	98 (5)
p-value	0.02	0.06

Predicting longer term outcome: only 52 patients had 18 month follow-up for A and 50 for AV. However by adding in 12 month data for those missing 18 month data, the numbers were increased to 58 and 57 respectively. The effect of age at operation, age of onset of deafness, and percentage of life spent deaf were assessed in terms of A and AV scores at long-term follow-up.

Age at operation was unrelated to outcome but the earlier the onset of deafness (and higher percentage of life deaf), the poorer the outcome, as would be expected (see Tables 5–7). There was no outcome difference between the sexes but patients with Nucleus Freedom implants did better at long-term follow-up than those with older implants (see Table 8). In terms of pre-implant functioning, medium pre-implant levels of A (scores of 1–15%) were associated with lower post-implant A scores, as was also found when looking at recovery up to 9 months.

Pre-implant AV scores had no significant effect on long-term outcome (see Tables 9–10). Multivariate analysis was not possible due to the small numbers of patients. The ceiling effect for AV was such that there was little ability to predict this outcome as it was mostly very good with means all over 90%.

Table 10. Pre-implant HINT AV score vs longterm A and AV scores (% correct): 57 patients

Pre-implant AV score (number of patients)	Mean longterm A score (SD)	Mean longterm AV score (SD)
0-60 (16)	84 (19)	99 (8)
61-78 (17)	72 (26)	98 (3)
79+ (24)	80 (24)	97 (5)
p-value	0.38	0.75

We looked at the highest and lowest performers within the group of 114 patients. We defined the very high performers as a post-implant HINT A score of at least 95% (at most recent assessment), and low performers as postoperative HINT A score of less than 60%. There were 43 high performers and 20 low performers out of the 114 patients.

Overall for the low performers the percentage of life spent deaf was more than 50% except for one patient, and for the high performers about half (22) of the patients had spent more than 50% of their life deaf while 21 of the patients had spent less than 50% of their life deaf.

For the high performers, 15 out of the 43 had pre-implant HINT A scores of more than 30. Nine of these were at least 25 years old at onset of deafness and had mainly progressive aetiology (e.g. otosclerosis), the other three being younger at onset but also progressive causes, and three with congenital deafness. A further 15 with lower pre-implant scores were at least 25 years old at onset and mainly progressive causes and had spent less than a third of their life deaf. One further patient had a congenital hearing loss that was made worse by otosclerosis over time.

There were 13 patients who would not have been expected to be high performers, with congenital deafness, implanted between ages 20-43 and pre-implant HINT A scores ranging from 0 to 30%.

Of the 20 lowest performers, 14 had congenital deafness, 11 with pre-implant HINT A scores of less than 15%, and 19 of whom were older than 35 at implant. There were two older patients: one aged 72 with progressive hearing loss and duration of deafness of 36 years, pre-implant HINT A score of 9%, and one aged 79 with endolymphatic hydrops as likely aetiology, duration of deafness of 49 years, and pre-implant HINT A score of 14%.

The remaining four low performers were aged between 49 and 55 and had differing background characteristics: a 55 year old with unknown aetiology, duration of deafness of 47 years and pre-implant HINT A score of 18, a 53 year old with chronic middle ear disease, duration of deafness of 48 years and pre-implant HINT A 18, a 52 year old with otosclerosis, duration of deafness of 32 years and pre-implant HINT A score of 55, and a 49 year old with chronic middle ear disease, duration of deafness of 4 years and pre-implant HINT A score of 0.

Only four out of 20 of the lowest performers' scores had not improved post-implant compared with pre-implant measurements. The remaining lowest performers had still improved significantly, despite having a low post-implant score compared with the

average for the group. The mean post-implant score for HINT A for the 20 lowest performers was 33 (range 14-56), compared with a mean pre-implant score of 15 (range 0-55).

Four patients are now not using their cochlear implants. One patient was found to be a malingerer, the patient is able to converse on the telephone without a hearing aid or cochlear implant. The second patient has superficial siderosis of the CNS, presumably with a large neural component towards his hearing loss meaning the device is ineffective. The third patient had a right petrous apex chondrosarcoma treated with surgery and radiotherapy and a left acoustic neuroma treated with radiotherapy. He could not perceive any sound from his right CI despite pre-implant testing suggesting a functioning auditory nerve on the right. The final patient had many psychosocial issues and significant negative thought processes which were thought to be the main cause of failure.

Discussion

Our medium-to-long term results after CI in adults compare favourably with others in the literature. This can be difficult as most studies have used different speech perception tests, so direct comparisons can not be made, however, the difference between pre-implant and post-implant scores can still be seen.

Our patient group was defined as adults receiving a CI, and includes those with prelingual and postlingual deafness, which can make it more difficult to draw comparisons. However our result of a mean of 74% correct HINT (A) score at 9 months and 81% at 18 months compared with a mean pre-implant score of 15% compares well even with studies of adults with postlingual deafness only (who would be expected to have a better result).

A Manchester study of 34 patients with postlingual deafness aged at least 65 years having a CI between 1989 and 2002 (with a mean duration of deafness of 11 years) had similar results to our study with a BKB sentence test mean score of 73% at 9 months (compared with a mean of 0 pre-implant), despite a shorter duration of deafness.² A Minnesota group of 33 patients over the age of 18 (average 52 years) with postlingual deafness who had CI before 2002 achieved a mean CID sentence test score of 54% at one year compared with 14% pre-implant.³

A Korean group of 13 postlingually deaf patients (average duration of deafness 9 years) with CI between 1988-1998 had a mean K-CID sentence test score of 52% at 12 months (pre-implant scores were not recorded).⁴ A study of 37 postlingually deaf adults with CI between 1989-1997 in the Netherlands obtained an average score in NVA open-set speech recognition of 36% at very long-term follow-up (6 years or more after CI).⁵

A Tennessee study of 27 patients over age 50 with postlingual deafness, with CI before 2004 had mean HINT score of 5.4% pre-implant, and 66% at mean of 4 years post-implant.⁶ A study of 33 postlingually deaf patients in the Netherlands implanted before 1994 obtained mean scores on speech perception testing (test not specified) of 28% at 12 months compared with 0% pre-implant.⁸

A Swedish study of patients aged over 16 (mean 50 years) and mean duration of deafness of 15 years with CI before 1994 had a mean open set spondee word score of

20% at one year (0% pre-implant).⁹ A study of 89 postlingually deaf CI patients in Antwerp with average age 58 years showed a mean post-implant NVA phoneme score of 47%-68% (differing means depending on differential age groups) at 12 months compared with 5-7% pre-implant.¹³

Studies have shown a significant increase in health-related quality of life following CI, which correlates with results in speech perception tests.¹² A quality-of-life study conducted by our programme has shown significant differences between implanted patients and patients on the waiting list for CI and also in the quality-of-life of their significant others.¹⁴

Patients with lower scores on speech perception can still obtain the benefits of being able to hear environmental sounds (e.g. a dripping tap, the doorbell, improvement in driving awareness etc), all four of the patients whose speech discrimination did not improve did receive some benefit from improved hearing of environmental sounds with their implant.

As previously mentioned there are many factors which affect the success or otherwise of cochlear implantation. In this study we have clearly shown that patients with a longer duration of deafness and a higher proportion of their lives with deafness, had significantly poorer results with cochlear implantation. This is consistent with previous findings in the literature^{4,7,9,11,12}.

The vast majority of these people still had very significant improvement in speech discrimination. Although long periods of deafness without hearing aids also contribute to poor results, our retrospective review was not able to adequately capture this data.

The age of the patient at implantation did not affect their outcome. Although just over 30% of our patients were aged over 60, the vast majority were under 80 years of age at the time of implantation. The literature confirms somewhat poorer results of cochlear implantation over the age of 80. We would expect that, in time, a higher proportion of our patients may be in the older age group.

Our impression of improved performance with the new Freedom device was confirmed with a mean auditory alone (A) score of 89% versus 73% with the older devices. This result could be somewhat confounded by the increased rehabilitation support available to our patients after 2004.

It is always disappointing when expensive technology is not used. We are undertaking an audit of our assessment processes to try to avoid inappropriate implants in those patients who are psychologically not able to benefit from the technology.

Our patient with superficial siderosis and another patient from the Northern Cochlear Implant Programme of New Zealand have been reported in the literature as the first two failures of CI in this condition.¹⁶ We have subsequently successfully implanted a patient with this debilitating condition.¹⁷

An interesting finding was that in terms of pre-implant functioning, the “middle group” of patients with pre-implant A values of 1-15% had a lower mean final score at 9 months than those with pre-implant scores of 0% (and those with higher pre-implant scores, as expected). This has also been noted in the literature.⁹ We have no explanation for this finding.

In terms of the time at which near-maximal benefit is obtained, we appeared to have a near plateau at 6 months, with mean HINT A scores of 75% compared with 79% at 9 months and 81% at 18 months. For AV scores, this was difficult to assess due to the ceiling effect (very high scores near 100%) which restricts the opportunity to observe changes. This is probably why AV scores peak faster.

This “plateau” at 6 months is in general consistent with findings from previous research. The Manchester study previously mentioned,² found that most benefit occurred during the first 9 months. The Netherlands study of 33 patients had a plateau at 6 months,⁸ and the larger Netherlands study,¹³ found a peak at 6-12 months. An Iowa study of adults with postlingual deafness reached a plateau in speech discrimination 6-9 months after CI.

Differing results were from the Korean study,⁴ which did not obtain a plateau until 2 years. A Californian study of 46 adults implanted before 1994 with a mean age of 53 years and duration of deafness between 2-69% of lives achieved at least 80% of their 12-month performance levels on open-set CID sentences at the first visit at 3 months, but for a more difficult test (NU-6 words), most improvement occurred in the first 6 months.¹⁰

There was much missing data which meant we used a smaller sample than initially intended. This is not entirely unexpected as many of the patients do not live locally and have to travel long distanced for follow-up. We noted that patients who had implants in 2004 were far more likely to have incomplete follow-up. This is likely to be because there was a changeover of rehabilitationist that year.

To assess for exclusion bias, we analysed background characteristics of the patients who were not used due to missed data and found there was no difference. Also, it can often be the case that the patients who do not attend for follow-up are actually performing better and feel that they do not need any further rehabilitation.

In conclusion, our study has enabled us to inform patients presenting for CI at SCIPA that our results compare very favourably with world-wide standards, that maximal benefit in terms of speech recognition appears to occur at around 6 months post-implant. Patients with a shorter duration of deafness or percentage of life spent deaf do better on average, but that patients with very poor pre-implant functioning may in fact perform better at long-term follow-up than those with slightly better pre-implant functioning.

Competing interests: None known.

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Nasal fractures: patient satisfaction following closed reduction

Rachelle L Love

Abstract

Aim Nasal fractures are commonly treated by primary closed reduction. However, studies suggest this produces unacceptable functional and aesthetic results. Many patients require revision surgery. We aimed to assess patient satisfaction following closed reduction of nasal fractures.

Methods Retrospective chart review of patients with nasal fractures treated by closed reduction at the Wellington Regional Plastic, Maxillofacial and Burns Unit, New Zealand over a 2-year period was undertaken. Digital manipulation under general anaesthetic was performed. Patients were followed-up by telephone with a structured interview.

Results Of 116 consecutive patients, 74 (65%) were successfully contacted. 65 (88%) were satisfied with functional outcome, and 64 (86%) with aesthetic outcome. Of the 34 patients reporting incomplete correction, 12 (35%) would consider revision surgery.

Conclusion Patients treated with closed reduction of nasal fracture performed under general anaesthetic with digital manipulation reported high levels of satisfaction with functional and aesthetic outcomes. Revision rate is low.

The nose is the central and dominant feature of the face and forms an important aesthetic unit.¹ The nasal bone is the most commonly fractured facial bone.² The force required to fracture the nose is less than that for any other facial bone.³ Even mild trauma can cause obstruction, discomfort and decreased olfaction.

Primary closed reduction is the mainstay of treatment for nasal fracture although unacceptable functional and aesthetic results have been reported.⁴⁻⁷ Poor results stem specifically from the failure to recognise septal fracture⁸ and the limited mobility of the nasal bones within the skin envelope.¹ This is generally compounded by lack of technical expertise and limited resources. Revision surgery occurs in up to 50% of patients.^{3,4,9}

A previous study from our unit compared the results of digital reduction of nasal fractures under general anaesthetic with instrumental technique under local anaesthetic.¹⁰ The current study evaluated patient satisfaction following closed reduction of nasal fractures and identified reasons for patient dissatisfaction.

Method

The charts of consecutive patients treated for nasal fracture in a tertiary referral centre between June 2004 and July 2006 were retrospectively reviewed. Children under 17 years of age and patients treated with other facial fractures were excluded.

Closed nasal reduction was performed under a brief general anaesthetic as a day procedure. Reduction of the nasal bones and septum was achieved by insertion of the little finger into the nares and countered by external digital manipulation using the opposite hand. No instrumentation was used and intranasal bleeding rarely occurred. A small moulded Plaster of Paris splint was applied over the dorsum of the nose for 5 days. The patients were routinely followed up by their General Practitioner.

Demographic data, mechanism of injury, airway obstruction and clinical deformity were noted subjectively and objectively. A telephone survey with a structured questionnaire was conducted. Patients were asked to determine if the function and appearance of their nose was worse or the same following closed reduction. They were asked to assess their overall satisfaction using a scale of 1 (very poor) to 10 (excellent). Patients who reported incomplete correction were asked whether they would consider revision surgery. Reasons for refusing revision were identified.

Results

161 consecutive patients who underwent closed nasal reduction were identified. 116 fulfilled inclusion criteria. 74 patients (65%) were successfully contacted for the telephone survey. Follow-up was carried out a minimum of 6 months after closed nasal reduction.

48 patients (65%) were men. Patients were aged 17–83 (average 22) years. Most fractured noses in men were the result of sports (20/48) and assault (19/48). Half of the fractures sustained through sports were from rugby. Other causes were cricket, basketball and soccer. Falls (12/26) were the dominant cause in women.

Patients presented to our unit on an average 4.3 (range 0–16) days following injury and were treated on average 1.2 (range 0–11) days later. 62 (84%) of patients received their operation on the same day as they were seen in clinic.

65 (88%) patients were satisfied with functional outcome and 64 (86%) were satisfied with the aesthetic outcome of their procedure.

Of the 34 patients (46%) with incomplete correction, 12/34 (35%) would consider revision surgery. One patient (3%) had already had revision.

Two principle reasons for declining surgical revision were identified by patients. There was a lack of confidence in consequent improvement, and a reluctance to tolerate the subsequent rehabilitation time. No patients refused revision because of the risk of surgery itself.

Discussion

Closed reduction of nasal fractures is the accepted treatment in most Otolaryngology and Plastic Surgery Units as most Clinicians attempt to balance good long-term results with minimally invasive methods of reduction.¹¹ However, acceptable results are not universal in the literature, with as few as 50% of patients confirming satisfactory results in some studies.^{4,6,8,12} Many patients seek revision.

38% of patients in our study presented exclusively with aesthetic concerns, about three times as many as those with purely functional concerns. This reflects Fernandes¹ observation that the aesthetic component of nasal bone fracture is a stronger incentive to seek medical attention. It is difficult to assess whether nasal fractures cause more aesthetic than functional complaints or whether functional deformity is better tolerated. Likewise, Hung et al¹³ report that pre-existing nasal symptoms not related to the fracture can adversely hamper the patient's perception of a good outcome.

Early studies indicate that prompt manipulation increases the likelihood of acceptable results¹⁴⁻¹⁷ and should be performed when the swelling resolves at 3–10 days of injury.¹⁸ In this study, closed reduction was carried out on average 5.5 days following injury. Most patients received their surgery on the same day that they were seen in clinic.

Digital manipulation of nasal bone fractures is not a commonly used technique, but has the advantage of minimising mucosal damage and nasal haemorrhage due to instrumentation.¹⁹ Satisfactory outcomes have been reported with this technique,^{10,19} but there will be some patients in whom a complete reduction is unable to be achieved.

The issue of reporting of poor results is complex and has not been adequately dealt with in the literature. It appears that patients tolerate poor functional and aesthetic outcomes, describing these as “worse, but satisfactory”. In this and other studies a significant number of patients with poor results refused revision. This may reflect an unwillingness to undergo a second general anesthetic,^{1,13} and is the basis for the suggestion that patients be offered primary septorhinoplasty in the first instance,¹ especially where septal deformity is recognised pre-reduction.

Most patients in our study refused revision. None declined because of the risk of anaesthetic. They cited a lack of confidence in consequent improvement, and a reluctance to tolerate the rehabilitation time.

Patients were contacted at least 6 months postoperatively and this may impact on patients’ reporting of outcomes. It is generally accepted that the result of treatment cannot be evaluated until one or 2 years after treatment, since trauma as well as the consequent surgery may lead to secondary deformity.²⁰ Longer follow-up of our patients would give more accurate results.

Functional and aesthetic results of primary closed reduction of adult nasal fractures using digital manipulation under GA are satisfactory and lead to a low revision rate.

Competing interests: None

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Self-dilation for refractory oesophageal strictures: an Auckland City Hospital study

Kenneth K S Wong, Dagmar Hendel

Abstract

Aim To determine the demographics, indications and long-term outcomes of patients using self-dilators for refractory oesophageal strictures.

Methods Patients with oesophageal strictures who performed self-dilation were analysed retrospectively. Patients who were still alive were also interviewed via telephone.

Results A total of 8 patients were analysed and 2 patients had since died. Most patients were in the 20–29 age group (n=4) when they first attempted self-dilation. The most common cause for oesophageal strictures was ingestion of corrosive (n=5). Each patient underwent on average 20 endoscopies (including endoscopic dilations) before attempting self-dilations. 1 patient developed oesophageal perforation during endoscopic dilation. 3 patients were still using self-dilators at the time of analysis. All the patients were only using lubricants (K-Y jelly) and none required topical anaesthetic such as Xylocaine throat spray.

Conclusions Oesophageal self-dilators were well-tolerated and complications were rare. By reducing the need for endoscopies, they are potentially cost-effective. However, patients must receive proper education before they are able to administer this treatment with confidence.

Patients with refractory oesophageal strictures who require regular dilations can be managed in the community with long-term self-dilation programmes¹. The efficacy of self-dilation in patients within the Auckland region has yet to be analysed. We present the demographics, indications and long-term outcomes of patients using self-dilators for refractory oesophageal strictures.

Methods

This is a retrospective study of all patients who performed self-dilation for oesophageal strictures between 1996 and 2007 who were managed at the Gastroenterology Department of Auckland City Hospital. Patients were identified from the Endoscribe™ software and also from the investigator's database. Surviving patients were followed-up via telephone with a questionnaire. The questions asked in the questionnaire were

1. "Are you still using self-dilators?"
2. "If yes, how often are you using self-dilators?"
3. "If not, when did you stop and how often had you been using them?"
4. "What sort of difficulty, if any, did you experience while using self-dilators?"

We define an oesophageal stricture as an anatomic restriction due to cicatricial luminal compromise or fibrosis that results in the clinical symptom of dysphagia without endoscopic evidence of inflammation². A stricture is considered refractory by some authors if there is failure to remediate the anatomic problem to a diameter of 42 French over 5 sessions at 2-week intervals².

In practice, however, we did not strictly adhere to this - strictures were considered refractory in our department if endoscopic dilatations were required once a week. With refractory oesophageal strictures our aim was to dilate to 51–54 French via endoscopy and then to proceed to self-dilation at 48–51 French using Maloney dilators. There was no set criteria when to consider self-dilation. In general, patients still needing endoscopic dilations after 3 months would be considered candidates for self-dilation.

After a series of endoscopic dilations, the first self-dilation attempt would be made on the same day immediately after an endoscopic dilation under conscious sedation with midazolam (0.5–1mg) and fentanyl (50–75 mcg). The patients would then have supervised self-dilations for the next 6 days without sedation. Local anaesthetic throat spray was often used for the first 1-2 self-dilation attempts only. They would proceed to self-dilation at home when the nurses were satisfied with their safety and competence.

Results

A total of eight patients were identified using self-dilators during this study period. Two patients had since died.

Most patients were in the 20–29 age group (n=4) when they first attempted self-dilation. Two patients were in the 40–49 age group and another two were in the 70–79 age group.

Five of the eight patients were males. Four patients were from the Auckland District Health Board, two from Counties-Manukau District Health Board, one from Waitemata District Health Board and one from Northland District Health Board.

With regards to the aetiology of strictures, the most common cause was ingestion of corrosive (n=5) followed by mucosal irritation from long-term nasogastric tube placement (n=2) and radiotherapy-induced fibrosis (n=1).

On average, each patient underwent 20 endoscopies (including endoscopic dilations) before attempting self-dilations. The number of endoscopies performed for each patient ranged from 7 to 39.

The highest number performed before self-dilation was attempted was on one patient who had 39 gastroscopies for a severe stricture involving the upper to lower mid oesophagus. The least number of endoscopies performed prior to self-dilation was seven and this patient only had mild narrowing with a length of 1cm on initial endoscopy. Other patients had at least moderate strictures. There appeared to be a correlation between the severity of the strictures and the number of endoscopic dilations needed.

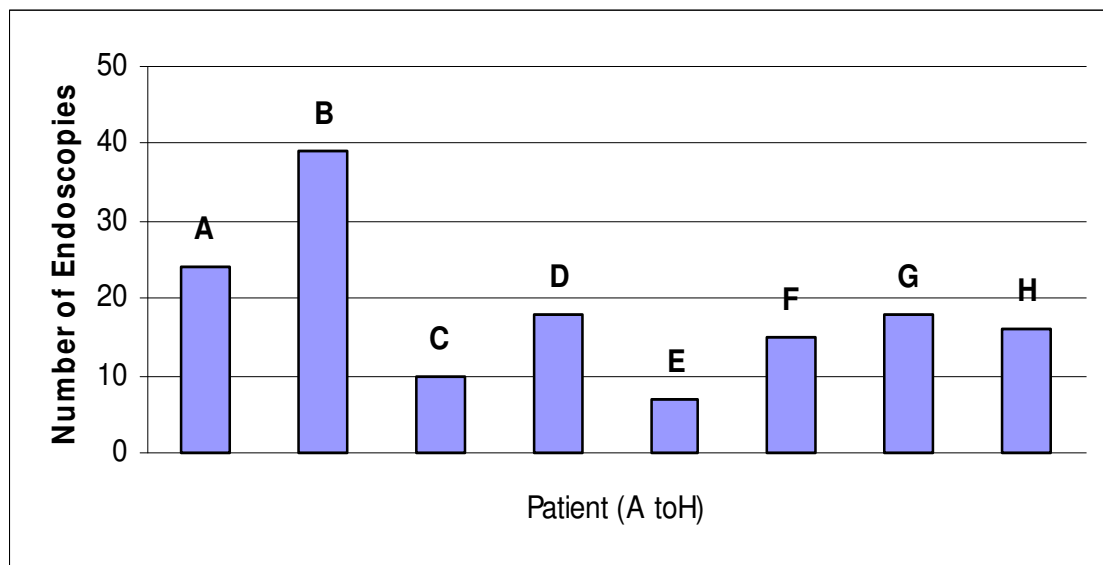
Only one patient developed oesophageal perforation but this happened during endoscopic dilation. This was the same patient who underwent the highest number of gastroscopies in this study. Nevertheless, he was able to perform self-dilation without further complications. There were no other complications reported amongst other patients. One patient died of bowel obstruction from colorectal cancer and the death was not directly related to oesophageal dilation. Another patient died from indeterminate causes.

At the time of this analysis in January 2008, there were six surviving patients. One patient was lost to follow-up. Of the remaining five patients that could be contacted, the average duration of continuous usage of self-dilation was 48.6 months (range 5 months to 82 months). Three patients were still using self-dilators.

The frequency of usage was from 3–4 times a week in two patients, once a week in two patients and approximately 1–2 times a year in one patient “as required”.

Three of the patients reported no difficulties inserting the self-dilators. One patient described psychological hesitancy in using the device, stating that it felt “unnatural” but there was no actual physical impediment to using the dilator. Another patient who had radiotherapy-induced oesophageal stricture complained of difficulty passing the dilator down far enough and was experiencing nausea and early satiety after using the instrument. This patient had only started using self-dilators in August 2007 and this was the most recent commencement of use among all the patients.

Figure 1. Number of previous endoscopies prior to self dilatation



All the patients were only using lubricants (K-Y jelly) and none required topical anaesthetic such as Xylocaine throat spray.

To date, none of the patients had surgical interventions for their oesophageal strictures.

Discussion

This study has shown that oesophageal self-dilators were remarkably well-tolerated and complications rarely occurred. The single patient who developed oesophageal perforation while having endoscopic dilatation had no further complications while using self-dilators. With practice, patients reported easy passage of the instrument and none reported local pain even without the use of topical analgesics.

Maloney dilators accomplish their result from the radial push transmitted to the stricture by the tapered portion of the tube as it passes through the narrowed area.³ Most patients obtain relief after dilation, but, as a case series demonstrates, 63% will develop recurrent dysphagia requiring repeat dilation.⁴

Uncomplicated strictures can be effectively dilated with blind passage of progressively larger Maloney dilators under the guidance of the 'rule of three' which states that no more than three successively larger dilator should be passed after initial resistance is met.⁵

The complications of dilation range from bleeding and perforation to bacteraemia, of which bacteraemia is thought to be the most common complication but is of little clinical significance.⁶ Perforation of the oesophagus is the most feared complication but the incidence is low, approximating 0.3% to 0.5% per procedure,⁷ and correlates directly with more complex strictures.⁸

The safety of self-dilators among patients is reflected in another study which analysed 51 patients with corrosive oesophageal strictures.¹ Of the 51 patients, 6 (11.8%) developed mediastinitis with initial endoscopic dilatation but no complication occurred when they commenced self-dilation.

The most common cause for oesophageal strictures in our study was corrosive ingestion. In one study of 239 patients who ingested corrosives, 65% of patients went on to develop oesophageal stenosis of which 59.3% were classified as moderate and 23% were severe.⁹ Less common causes for oesophageal strictures as highlighted in our study are mucosal irritation from nasogastric tubes and radiotherapy-induced fibrosis.

Oesophageal stents are an option to treat oesophageal strictures but we have not used them in this setting. Self-expanding metal stents are not suitable for benign strictures as they are difficult to remove after placement due to embedment into the oesophageal wall.¹⁰ In addition, they are associated with significant morbidity including stent migration, recurrent strictures, fistula formation, bleeding and death.¹¹ These stents are not FDA-approved for benign strictures.¹²

More recently, retrievable stents have been used for benign oesophageal strictures but they were also limited by stent migration, this being the most common complication, occurring 27% (range 7–57%) of the time in a meta-analysis.¹³

Self-dilation is cost-beneficial as it reduces the need for endoscopies and hospitalization. The reimbursement for a single gastroscopy session at Auckland City Hospital is \$814.48 while an oesophageal dilator costs \$500.00 and this can be re-used by the patient.

It is also safer as no sedation is required. It can be administered at anytime and this gives patients greater autonomy. Nevertheless, proper education must be given to patients before they can administer this therapy with confidence. The role of nurses is important in this regard in providing training, reassurance and support.

Competing interests: None.

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Getting tangled in red tape: the challenges of doing clinical research in New Zealand

Sarah Loveday, Ed Mitchell

Abstract

Regulations and the number of forms that require completion remain a major frustration for researchers attempting to establish a clinical research project. It is essential that clinical research is of the highest standard and abides by ethical principles. However, the duplication of information required and lack of co-ordination between national and local ethics and research committees is a hindrance to conducting clinical research. This publication highlights the pitfalls in our current system with a case study, and suggests changes to the ethical review process that may aid researchers in establishing clinical research.

New Zealand has recently appointed a chief science advisor to the Prime Minister, with an aim to actively promote science and research in order to help stimulate economic growth. However, a fundamental threat to research may impede scientific progress and impact economic growth.

Scientist Dr William Rolleston has spoken out against the “onerous and unnecessary regulations” that stifle research in New Zealand.¹ For new researchers a major frustration in establishing a clinical research project is the number of repetitive forms that require completion.

This over-regulation of clinical research is not a new phenomenon nor is it specific to New Zealand. A number of recent articles have been published bemoaning the overregulation of medical research in England and the United States of America.²⁻⁴

While clinical research needs to be of the highest standard with reasonable checks in place the duplication of information required and lack of co-ordination between services is a hindrance in conducting clinical research. This article will highlight the pitfalls in our current system with a case study.

Case study

In June 2008 funding was successfully obtained for the author (SL) to complete a research project as part of a Fellowship in Developmental Paediatrics. The project was a cross-sectional observational study of body composition in children with Down syndrome.

All children were seen at Starship Children’s Hospital, the paediatric service within the Auckland District Health Board (ADHB). An application for ethical approval from the regional ethics committee (using the national ethics application form) was sent in October 2008 and obtained in November 2008 with minimal changes required to the participant information sheet and consent form.

In addition to the national ethics application form, a number of forms were required in order to use an examination and interview room in the hospital. In order to apply to use a room, the project needed to be registered with the ADHB Research Office. The application form for this registration (ADHB Research Project Registration Form) required duplicate information to that in the National Ethics Application Form.

Once the project was registered with the hospital research office, a further form was required to grant use of a room for research purposes (Children's Research Centre Application Form). However, prior to gaining approval to use a room, the project required approval from the hospital Māori Research Review Committee (MRRC).

In order to identify children for the study, a search of the hospital laboratory database of abnormal karyotypes was required. Two separate forms were required to use this database. A number of minor changes were requested from the ADHB research office. Full consent from ADHB was gained in December 2008.

Table 1. Application process for research approval

Form	Approval process	Number of pages
1	Starship Foundation Research Fellowship Application Form	4
2	National Ethics Application: Main form Unable to give informed consent form Patient information sheet Consent form	17 2 4 1
3	NRL Application Form to get ratification for using DXA	1
4	Locality Assessment for ADHB	3
5	Locality Assessment for University of Auckland	3
6	Application Form for Approval of a Research Project at ADHB	5
7	Children's Research Centre Application Form	2
8	ADHB Māori Review Committee Approval	(Send forms 1–3, 5, 6)
9	Request Form for Access to Hospital Laboratory Database (LabPlus)	1
10	Acceptance of Services for Data Retrieval Provided by LabPlus	1
11	Locality Assessment for CMDHB	3
12	CMDHB Research Application	2
13	CMDHB Māori Research Review Committee Application Form	3
14	Locality Assessment for WDHB	3
15	WDHB Māori Research Review Committee Approval Form	(Send forms 1, 2, and ADHB approval)
16	WDHB Knowledge Centre Approval of Research Application Form	Online application

Abbreviations: ADHB – Auckland District Health Board; CMDHB – Counties Manukau District Health Board; DXA – dual energy X-ray absorbiometry; NRL – National Radiation Laboratory; WDHB – Waitemata District Health Board.

The study required enrolment of children living in areas of Auckland outside of the ADHB area, although all children were seen only at Starship Children's Hospital. Locality assessment forms were completed for the other two district health boards in Auckland, Counties Manukau District Health Board (CMDHB) and Waitemata District Health Board (WDHB). This required several forms including CMDHB Research Application, CMDHB MRRC Application Form, CMDHB Budget Form,

Knowledge Centre Approval of Research Application Form and WDHB MRRC Approval Form.

As the author was not an employee of CMDHB or WDHB, a credentialing form needed to be completed for both sites, even though all that the study required was to enrol children living in the CMDHB and WDHB areas. At each stage each research committee required slightly different modifications to either the participant information sheet or the consent form.

Final ethical approval to enrol children living in the wider Auckland area was obtained in March 2009 5 months after the initial application was made. The application process required 16 separate steps, with considerable duplication of information required on the forms (Table 1).

History of health and disability ethics in New Zealand

The Medical Research Council was established in 1950.⁵ Applications to conduct research required applicants to sign an ethical agreement in order to obtain funding. Over time ethical standards were developed and published and a committee on ethics in research was established in 1984. The Auckland hospital ethics committee was established in 1971 with considerable debate at its inception as to the necessity of ethical surveillance.⁶

A turning point in New Zealand's history of research ethics was the 1987 Cartwright Inquiry of the treatment of cervical cancer at National Women's Hospital. Following this ethical review was seen as not only important but a necessity and ethical standards "must be applied rigorously to research and treatment protocols on behalf of patients".⁵

The Health Research Council was established with an ethics committee under the Health Research Council Act of 1990. Regional health authorities were required to establish ethics committees or to contract ethics committees to provide ethical review by 1993. Further health reforms lead to minor changes in the inter-relationship between the ethics committees and the HRC.

The New Zealand Public Health and Disability Act of 2000 established a National Ethics Committee. Currently New Zealand has seven Health and Disability Ethics Committees of which six are regional. Applications to conduct research are submitted either to a regional committee or to the multiregional committee as appropriate. District Health Boards (DHBs) retain Research Review Committees to advise the clinical board of local research initiatives and contracts.

Principles of ethical review

The primary role of ethical review of research in New Zealand is two-fold: firstly to prevent unacceptable risk of harm to participants, and secondly to ensure that all participants are aware of what their participation will involve and have given informed consent.⁷ The underlying principles of ethical review are respect of persons, informed consent, privacy and confidentiality, validity of research proposal, minimisation of harm, justice, and social and cultural responsibility.

Respect of persons encompasses the recognition of the beliefs, autonomy and privacy of individuals while protecting those who have reduced competence. Informed

consent requires adequate information to be given which is understandable to the participants and that consent is voluntary without coercion.

Privacy and confidentiality are essential for the protection and promotion of human dignity and wellbeing. It is important that research is methodologically valid to ensure it is worthwhile and participants are not unduly exposed to risks entailed in research.

The ethical principle of justice requires the burdens and benefits of research to be fairly distributed over a population without discrimination. In addition ethical review in New Zealand upholds respect for the social and cultural sensitivity of the study population. The National Ethics Committee form encompasses all of these principles.

Improving systems

Having multiple forms to address the same principles has given rise to considerable red tape in the process of gaining approval to conduct clinical research. This duplication threatens to overwhelm researchers with paper work, and may delay the final approval by several months as described in our case study.

Several improvements could be made in our current ethical approval process with no threat to appropriate clinical standards or the principles governing ethical review.

Firstly, the amount of duplication required could be mitigated by each DHB research committee accepting the national ethics application form instead of requiring duplication of all the same information in a DHB-specific form. If additional information is required, then only this should be included in a DHB-specific form.

Similarly, a national Māori Research Review Committee form should be developed which would be accepted in all sites. Conducting research in Auckland is a unique experience within New Zealand as patients are divided into three DHB areas. However, for the purposes of research Auckland should be treated as one area.

In addition, a more common sense approach needs to be taken when looking at ethical consent for new research. A cross-sectional observational study in which participants simply undergo anthropometric measurements is very different from a study in which participants are exposed to a novel medication or intervention.

Our current system makes no allowance for the potential harm to participants and the same numbers of forms are required for both. If, in New Zealand, we are serious about stimulating clinical research, then the current impediments of over-regulation must be addressed.

Competing interests: None.

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MRI imaging of the inner ear for Meniere's disease

Jeremy Hornibrook, Mark Coates, Tony Goh, Philip Bird

Until now, imaging of the inner ear has been by computed tomography (CT) scanning which can delineate its bony borders, defects and congenital abnormalities. Because of their size, imaging of internal structures has not been possible. Therefore much inner ear pathology has been elucidated from post-mortem histology.

It is 70 years since temporal bone histology demonstrated that in Meniere's disease the fundamental abnormality is an excess of fluid in the endolymphatic compartment, called endolymphatic hydrops. The official definition of "definite" Meniere's disease is attacks of vertigo accompanied by documented fluctuating hearing and/or aural fullness in the affected ear, whereas "certain" Meniere's disease requires a post-mortem to prove the hydrops.¹

In animal models it had been shown, with long scanning times, that intratympanic delivery of gadolinium selectively enhances perilymph, delineating it from endolymph.² In humans, medical resonance imaging (MRI) inner ear studies have been limited by the spatial resolution of 1.5 Tesla scanners. Intratympanic gadolinium was shown to enter the human ear on 1.5 Tesla scanner in 2005.³

Newer scanners with greater magnetic strength and improved image sequencing have made ultrastructural detail achievable. On 3 Tesla scans, human endolymphatic hydrops has been clearly demonstrated.⁴⁻⁷ The dosage, timing of administration and safety have been established, and a grading system has been suggested.⁸

Patients

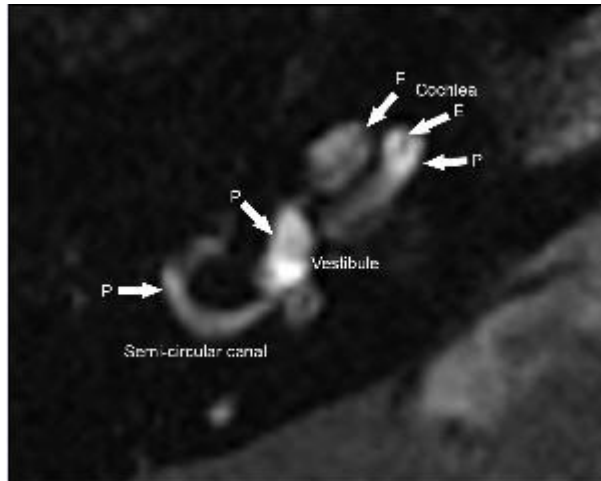
MRI inner ear scanning was conducted on two patients: (1) an 80-year-old male with vertigo attacks and mild right ear hearing loss, experiencing no aural symptoms and not fulfilling the AAOHNS criteria for Meniere's disease; and (2) a 46-year-old male with a 2-year ear history of vertigo attacks accompanied by progressive hearing loss, tinnitus and aural fullness in the right ear fulfilling the criteria for Meniere's disease.

Twenty-four hours before the scan, multihance gadolinium 1.6 ml in 10 ml saline was introduced to the right middle ear through a small myringotomy (and replenished 4 to 5 times, a total of 1.0–1.5 ml over 45 minutes with the patient lying to the opposite side).

Images were obtained on a 3 Tesla Magnet (General Electric HDX). Two inversion recovery sequences were obtained with inversion times of T1 1000 (endolymph) and 2500 (perilymph) in addition to routine 3D Fiesta imaging of the inner ear/IAMs.

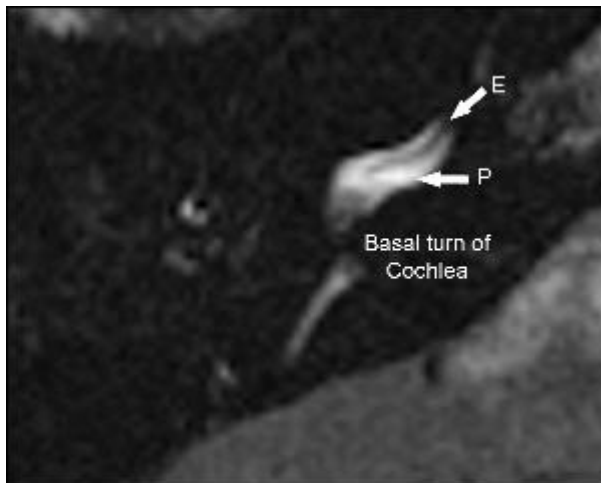
Patient 1. Normal inner ear: no hydrops

Figure 1. Perilymph sequence. The cochlea, vestibule and one semicircular canal are labelled.



P=perilymph, E=endolymph.

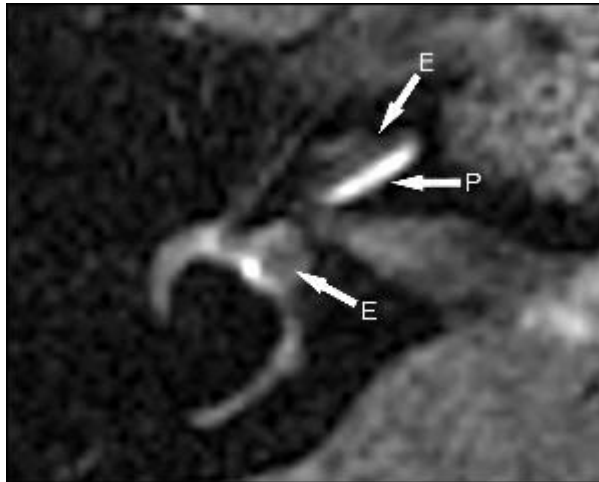
Figure 2. Perilymph sequence. Basal turn of the cochlea, with normal endolymphatic compartment; no hydrops.



P=perilymph, E=endolymph.

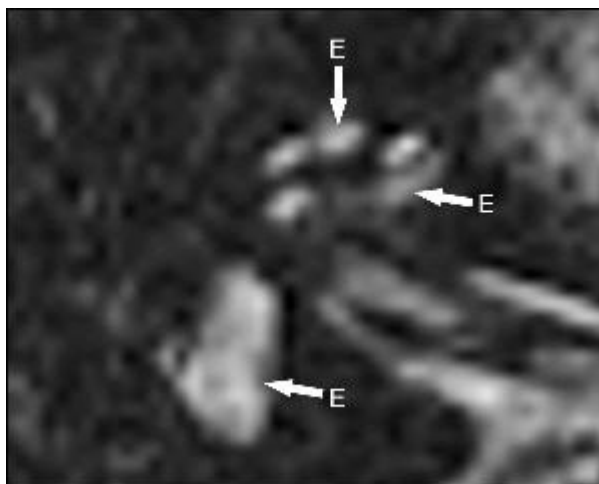
Patient 2. Meniere's disease: significant hydrops

Figure 3. Perilymph sequence. Significant enlargement [33–50%] of the endolymphatic compartment in the cochlea; in the vestibule and semicircular canal endolymphatic hydrops [>50%] has displaced almost all perilymph.



P=perilymph, E=endolymph.

Figure 4. Endolymph sequence. Enlargement of the endolymphatic compartment in the cochlea; endolymph fills the vestibule.



E=endolymph.

Discussion

In these two subjects, MRI inner ear scanning was normal in Patient 1 and clearly showed endolymphatic hydrops in Patient 2 with a history fulfilling the AAOHNS criteria for a diagnosis of “definite” Meniere's disease.¹

The recently proposed hydrops grading system⁸ is simple: none, mild, significant. In the vestibule an endolymph/perilymph ratio of one-third=none; one-third to one-half=mild; and >50%=significant. In the cochlea, no Resisner's membrane displacement=none; Resisner's membrane displacement with the area of the endolymphatic compartment not exceeding the area of the scala vestibuli (perilymph)=mild; the endolymphatic space exceeds the area of the scala vestibuli (perilymph)=significant.

Early human studies established that intratympanically administered gadolinium enhances cochlear perilymph within 4 hours⁶ and the perilymph in all areas by 24 hours.³ In three patients scanned at 6 days the gadolinium and had almost disappeared.³ In animal studies a 8-fold dilution of gadolinium had no adverse affects on the stria vascularis⁹, and there have been no reports of it causing hearing loss or aggravating tinnitus.

In Japan, Nakashima et al¹⁰ have used MRI imaging to study 73 patients with inner ear diseases including Meniere's disease, idiopathic sudden sensorineural hearing loss, and fluctuating hearing loss without vertigo. They used 3D-real IRI (a 3-dimensional technique) MRI which gives clearer visualisation of the perilymph space than the 2-dimensional technique used in New Zealand.

MRI scanning of the inner ear is an exciting new development in the diagnosis of inner ear conditions. Correlation of symptoms with imaging should significantly contribute to the understanding of inner ear diseases. For example, recurrent non-positional attacks of vertigo similar to Meniere's disease without hearing loss may be due to endolymphatic hydrops, or have an entirely different pathology.

Conversely, fluctuating hearing loss, tinnitus and aural fullness often occur without vertigo. Do these people have endolymphatic hydrops confined to the cochlea? Although patients with unilateral inner ear symptoms usually receive MRI scanning to exclude vestibular schwannoma/acoustic neuroma, both this and intratympanic gadolinium are mildly invasive.

Rigorous clinical research will be required to delineate the role of this new technology in the management of Meniere's disease and other inner ear conditions.

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Bilateral superior canal dehiscence syndrome

Jeremy Hornibrook, David O'Neill-Kerr, Latham Berry, Grant Carroll

Superior canal dehiscence (SCD) syndrome is a newly recognised condition¹⁻⁴ where dehiscence of bone over the superior semicircular canal can lead to unusual auditory and vestibular symptoms and signs.

In the 1880s, Ewald demonstrated that pressure applied to surgically fenestrated canals in pigeons could cause a nystagmus in the plane of the stimulated canal. This has long been recognised as a possible complication of cholesteotoma eroding bone over the horizontal canal accounting for horizontal nystagmus induced by applying alternating pressure to the external ear canal, called Hennebert's sign.

In 1929, Tullio⁵ showed that in dogs with surgically fenestrated superior canals loud sounds could effect a nystagmus in the plane of the canal. Also, in rabbits and pigeons with intact labyrinths loud sounds could induce vestibular responses with disconjugate rotation of the eyeballs, tilting of the head and leg flexion. These vestibular responses are now called the Tullio phenomenon.

More recent electrophysiological studies in humans showed that a myogenic response to a loud click stimulus occurs in the sternomastoid muscle.⁶ The response—named the vestibular-evoked myogenic potential (VEMP)—is generated by the saccule and is non-hearing dependent. Ears affected with the Tullio phenomenon have an abnormally low VEMP threshold.⁷

Case report

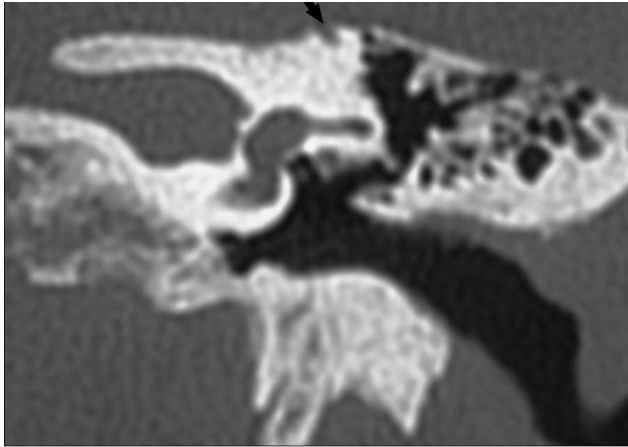
A 58-year-old female presented complaining of intermittent dizziness, oscillopsia and aural symptoms. For 2 years there had been pulsatile tinnitus in both ears, particularly the right ear. More recently stooping, nose-blowing or a sneeze would elicit brief vertigo and oscillopsia. Also she had noticed that when walking on firm ground she could hear her footsteps: "my footsteps just echo inside my body".

On examination ear drums, hearing and acoustic reflexes were normal. There was no spontaneous nystagmus. Vestibulo-ocular reflexes were normal. When the patient performed a valsalva manoeuvre a brief down beat and slightly right-torsional nystagmus occurred. This was reproduced and recorded in infrared light by a right eye camera (Figure 1 – mpeg video-clip accessible at <http://www.nzma.org.nz/journal/123-1321/4307/video.mpg>).

Further provocative tests for Tullio phenomena were done. However, straining against a closed glottis, alternating pressure in the external ear canal, or a 0.5kHz tone at 90dB and a 1kHz tone at 120dB did not induce symptoms or nystagmus.

CT scans with 0.625m slices in the coronal plane showed a bony dehiscence of both superior semicircular canals (Figure 2). MRI scans imply an abnormal absence of bone between the membranous portion of the superior canals and the overlying dura (dark space), in comparison with a subject with a normal bony covering (Figure 3).

Figure 2. Coronal CT scan right ear. Arrow indicates absence of bone over the superior canal.



Coronal CT scan left ear. Arrow indicates absence of bone over the superior canal.

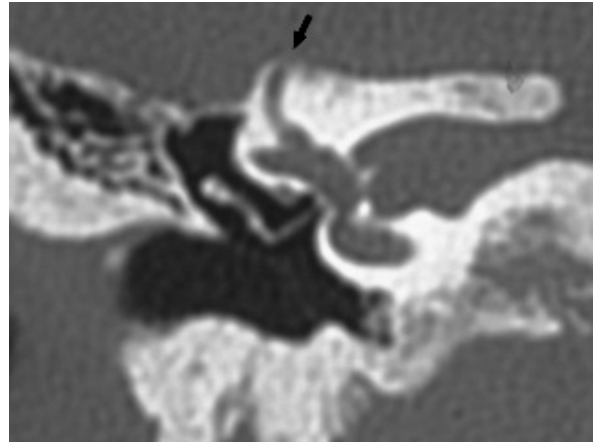
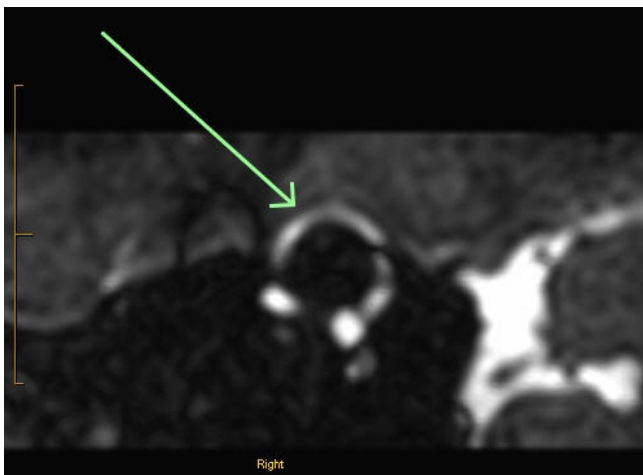
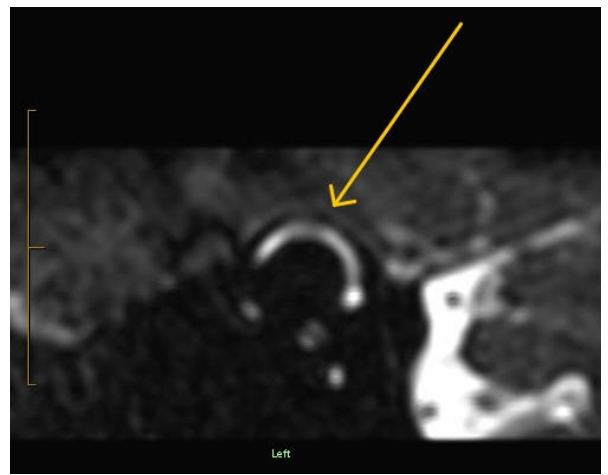


Figure 3. Right coronal MRI scan. Arrow indicates the closeness of dura to superior canal perilymph.



Left coronal MRI scan. Arrow indicates the closeness of dura to superior canal perilymph.

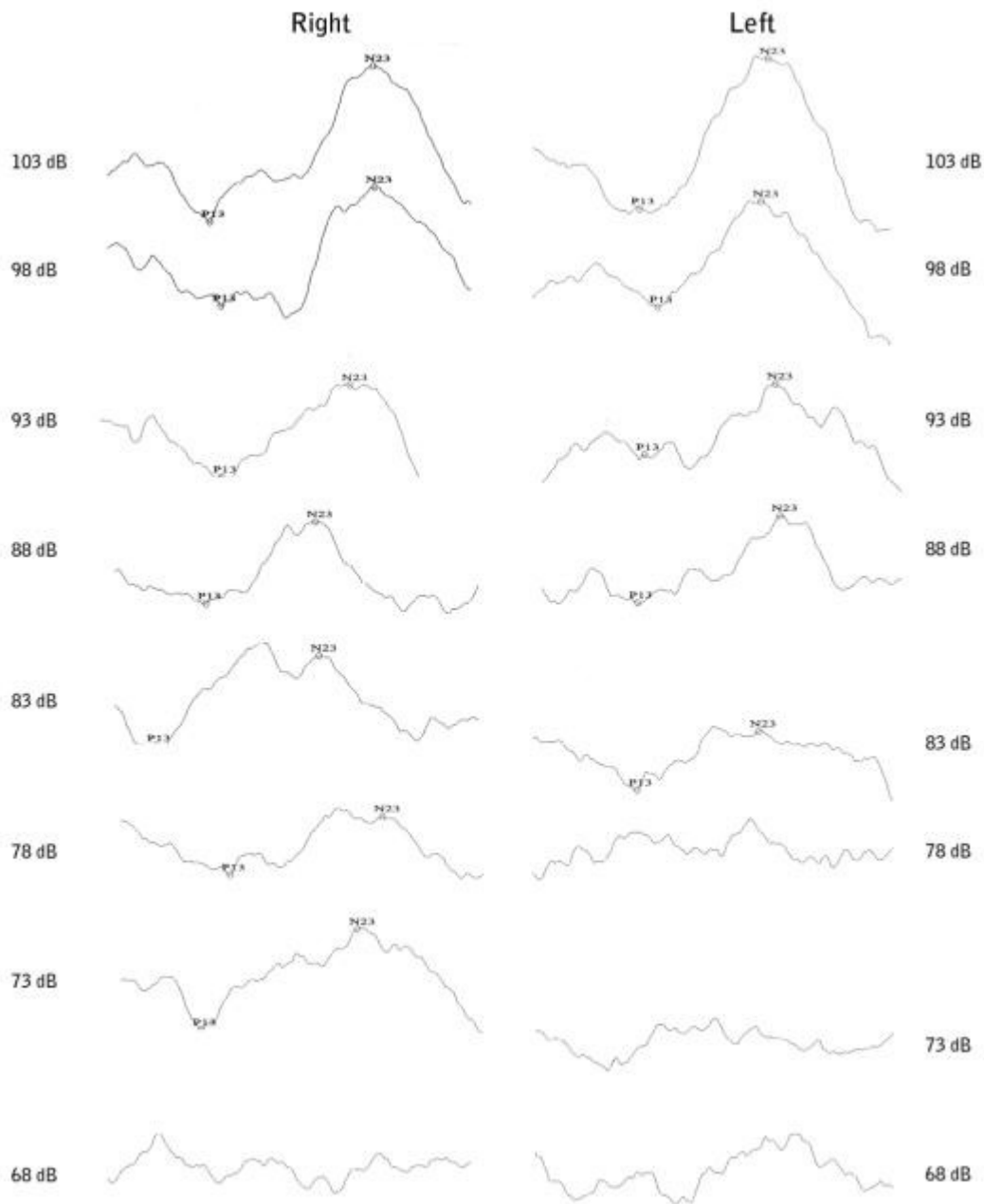


Right coronal MRI scan of a normal ear. Arrow indicates bone separating dura from the superior canal.



Cervical VEMPS were measured with surface electrodes over the sternum and each flexed sternomastoid muscle, using 0.1 msec clicks from a headphone in 5 dB descending steps. On the right the threshold for a clear N13/P23 wave-form was 73dB, and 83 dB on the left, both significantly below the normal (96 ± 5 dB)⁸ (Figure 4).

Figure 4. Cervical VEMPS



In this patient there is both CT scanning and VEMPS evidence of bilateral SCD syndrome.

Discussion

Bony dehiscence over the superior canal can result in vestibular or auditory symptoms and signs, or both. The reasons for the differences are not known, but the dehiscence creates a so-called “mobile third window” effect. The vestibular symptoms are vertigo and oscillopsia.

A valsalva manoeuvre with closed nostrils, positive pressure in the ear canal cause an ampullofugal deflection (excitatory) of the superior canal cupula. In contrast—a valsalva manoeuvre against a closed glottis—bilateral jugular venous compression and a negative pressure in the ear canal can cause an ampullopetal (inhibitory) cupula deflection. The auditory symptoms can be autophony (hearing one’s own voice loudly in the ear), hypersensitivity for bone-conducted sounds, a blocked ear or pulsatile tinnitus.

In a microscopic study of 1000 temporal bones sectioned vertically in the plane of the superior canal, 0.5% had a complete dehiscence and 1.4% had bone so thin it could appear dehiscent even on high resolution CT scanning.⁹ Conventional CT scans are displayed in the axial and coronal planes. False positive dehisces can be reduced with 0.5-mm-collimated helical scans with reformation of the images in the plane of the superior canal.^{10,11}

The onset of SCD symptoms is typically in adulthood when, presumably, abnormally thin bone over the canal is disrupted by trauma or eroded by overlying structures. In most patients (as in this report) the symptoms are mild, and are prevented by avoiding the stimuli that cause them. In rare cases with disabling symptoms (sound-induced vertigo, pulsatile oscillopsia) surgical treatment is justified, either by closure of the defect with fascia and bone cement¹² or by superior canal plugging.¹³

In summary, SCD syndrome can cause unusual auditory and vestibular symptoms. Elicitation of nystagmus by a valsalva manoeuvre, sound stimulation or external canal pressure and a CT scan implying dehiscence are strongly suggestive, but a reduced VEMP threshold is required for certain diagnosis.

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Inadvertent swallowing of toothbrush

Dinesh Lal

A 15-year-old physically and mentally healthy Caucasian female presented to Middlemore Hospital's Emergency Department (Auckland, New Zealand) after swallowing a standard 19cm toothbrush. She was running up some steps with the toothbrush in her mouth when she suddenly tripped and fell pushing most of the toothbrush into her oesophagus.

She immediately started choking and her younger brother came to help. Part of the toothbrush was still in the mouth but with apparently a very strong gag reflex she swallowed this down before it could be pulled out. On arrival in Emergency Department she was well, apart from describing a sensation of the toothbrush churning around in her stomach

An abdominal X-ray could not visualise the brush as it was radio-opaque. She underwent a flexible gastroscopy under general anaesthesia. Minimal erythema of interarytenoid notch was seen on the left side.

The toothbrush was located in the stomach with the head end lying proximally buried into the gastric mucosa. The very proximal end of the head of the toothbrush was grabbed with a snare and pulled out with the gastroscope via the oropharynx. Moderate traction force was applied to negotiate the esophago-oro-pharyngeal angle due to the length of the toothbrush.

A relook endoscopy did not reveal any signs of trauma. A repeat chest X-ray did not reveal any pneumomediastinum and she was discharged home the same night a few hours later.

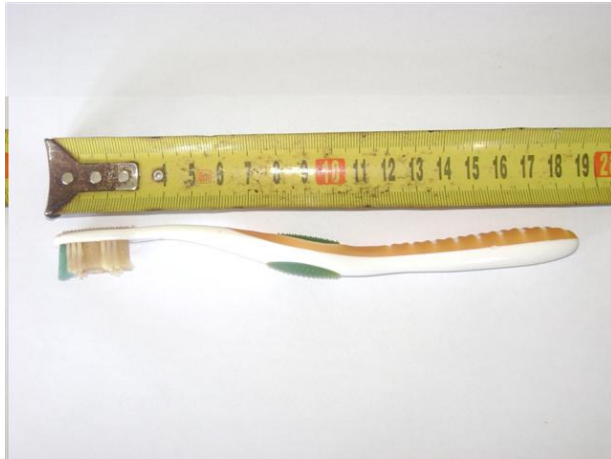
Discussion

It is generally recommended that objects longer than 6–10cm should be removed as they will have difficulty in passing the duodenal sweep.^{1–3} Using an overtube in assisting removal can be helpful by grabbing the object with a snare, manoeuvring into the overtube and withdrawing the entire apparatus with foreign body, overtube and endoscope in one motion.⁴ However in this patient an overtube was not used.

Use of an overtube itself can cause oesophageal mucosal damage especially in a younger patient. The ends of the toothbrush were relatively smooth and the shaft relatively bendable. The greatest resistance encountered was at the oropharyngo-oesophageal angle and this can be minimised by hyperextending the neck when the brush reaches this level.

In summary, walking or running around with a toothbrush in the mouth is potentially dangerous. A similar swallowed toothbrush (Figure 1) can be fairly safely removed via flexible gastroscopy under general anaesthetic without the aid of an overtube.

Figure 1. Toothbrush removed from stomach



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Poland syndrome: rare presentation in two cases

Hayrettin Gocmen, Yücel Akkas, Selim Doganay

Abstract

Poland syndrome was first described in 1840 by Alfred Poland while still a medical student and the other components of the syndrome were described at London Guy's Hospital following the dissection of a cadaver's hand, which had hypoplasia and syndactyly. The incidence of Poland syndrome has been reported to be 1 in 30,000 live births. In the present case report, two Poland syndrome patients with ipsilateral hypomastia and a reduction in the axillary/pectoral hairs diagnosed during adulthood are presented; one patient was affected on the left side and had widespread café au lait spots, and the other patient had respiratory dysfunction due to multiple rib anomalies..

Poland syndrome was first described in 1840 by Alfred Poland while still a medical student and the other components of the syndrome were described at London Guy's Hospital following the dissection of a cadaver's hand, which had hypoplasia and syndactyly.¹

The main component of this anomaly is absence of the pectoralis major muscle.² This may also be accompanied by the absence or hypoplasia of the pectoralis minor, serratus anterior, latissimus dorsi, and deltoid muscles, and hypoplasia of the breast and the absence of nipples in girls. Rarer associations include rib defects, scoliosis, dextrocardia, renal hypoplasia, leukaemia, and Mobius syndrome.³ Some studies have reported the female-to-male ratio to be 1:3,⁴ whereas other studies have suggested that both genders are affected equally.⁵

The incidence of Poland syndrome has been reported to be 1 in 30,000 live births;⁶ Poland syndrome affects the right side in 67%–75% of cases.^{4,7} In the present case report, two Poland syndrome patients with ipsilateral hypomastia and reduction in the axillary/pectoral hairs diagnosed during adulthood are presented; one patient was affected on the left side and had widespread café au lait spots, and the other patient had respiratory dysfunction due to multiple rib anomalies.

Case report

Case 1—A 25-year-old male admitted with a complaint of congenital chest deformity involving the left side. The patient complained of a burning sensation on the left side of his chest and lack of strength in the left arm on extension. The physical examination revealed absence of the pectoral muscles. The physical examination also revealed café au lait spots on his chest (Figure 1).

Reduction of hairs in the left axillary region and on the left side of the chest was noted compared to the contralateral side. There was no oligosyndactyly (OS). Strength was 5/5 in the right arm and 4/5 in the left arm. Pectoral muscles in the left hemithorax were not observed on the thoracic computed tomography (CT) scan (Figure 2). There were no concomitant rib anomalies.

Figure 1. Pectoral muscles are absent on the left side (short arrow) and café au lait spot are seen on his right chest (long arrow)

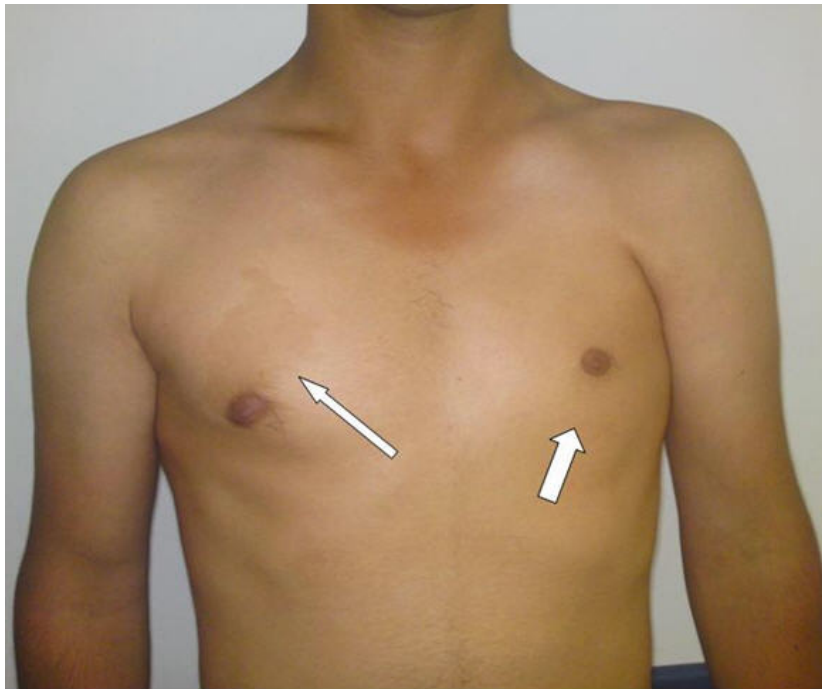
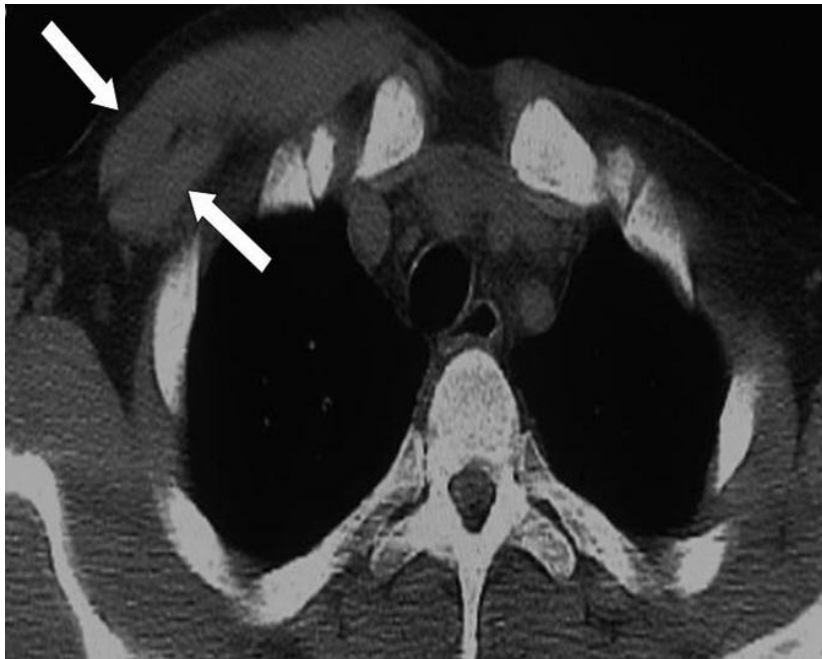


Figure 2. Axial plain CT scan shows pectoral muscles on the right chest (arrows) but pectoral muscles are absent on the left side



The abdominal ultrasonography and Doppler imaging of the neck were free of any pathologic findings. The results of the respiratory function tests were within normal limits (forced vital capacity [FVC]: 5.37 L 95%; forced expiratory volume1 [FEV1]: 4.82 L 102%; FEV1/FVC: 90%). The patient had two sisters and no disease-related abnormalities were reported in the family history

Case 2—A 21-year-old male admitted with complaints of a deformity of the right side of the chest and dyspnoea on exertion. The patient complained of lack of strength in the right arm and right shoulder. The physical examination revealed absence of the pectoral muscles on the right side and a marked volume loss within the right hemithorax (Figure 3). The patient had two sisters and no disease-related abnormalities were reported in the family history. There was hair reduction on the right side of the chest and in the axillary region.

Figure 3. The pectoral muscles on the right side are absent. There was hair reduction on the right side of the chest. The right breast was hypoplastic



The right breast was hypoplastic and there was no OS. The strength of the right and left arm was determined to be 3–4/5 and 5/5, respectively. Pectoral muscles of the right hemithorax were not observed on thoracic CT scan (Figure 4A). There were rib anomalies involving two levels and loss of volume within the right hemithorax (Figure 4B, 4C).

Figure 4A. Axial plain CT scan shows pectoral muscles on the left chest (arrow) but pectoral muscles are absent on the right side

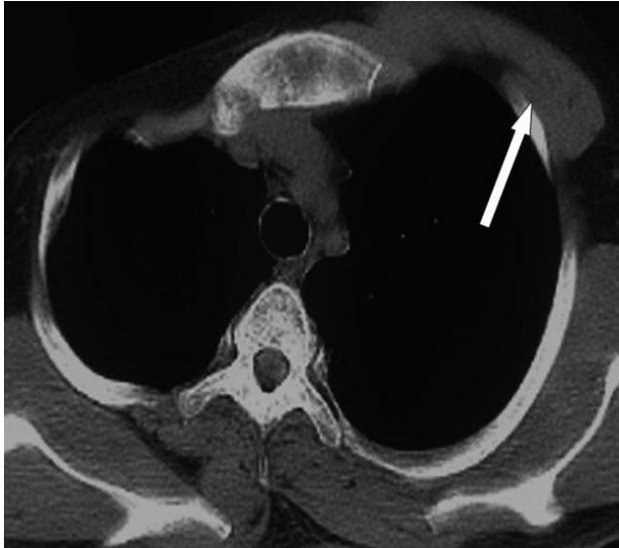


Figure 4B. Plain chest radiography shows rib anomalies (arrows) and loss of volume within the right hemithorax

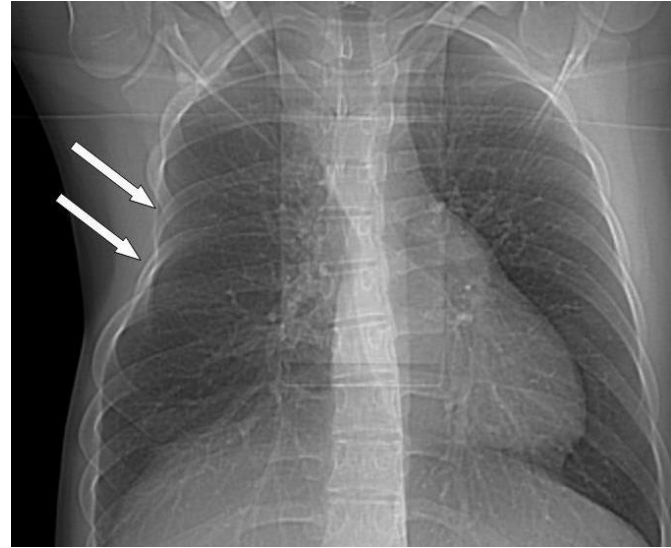
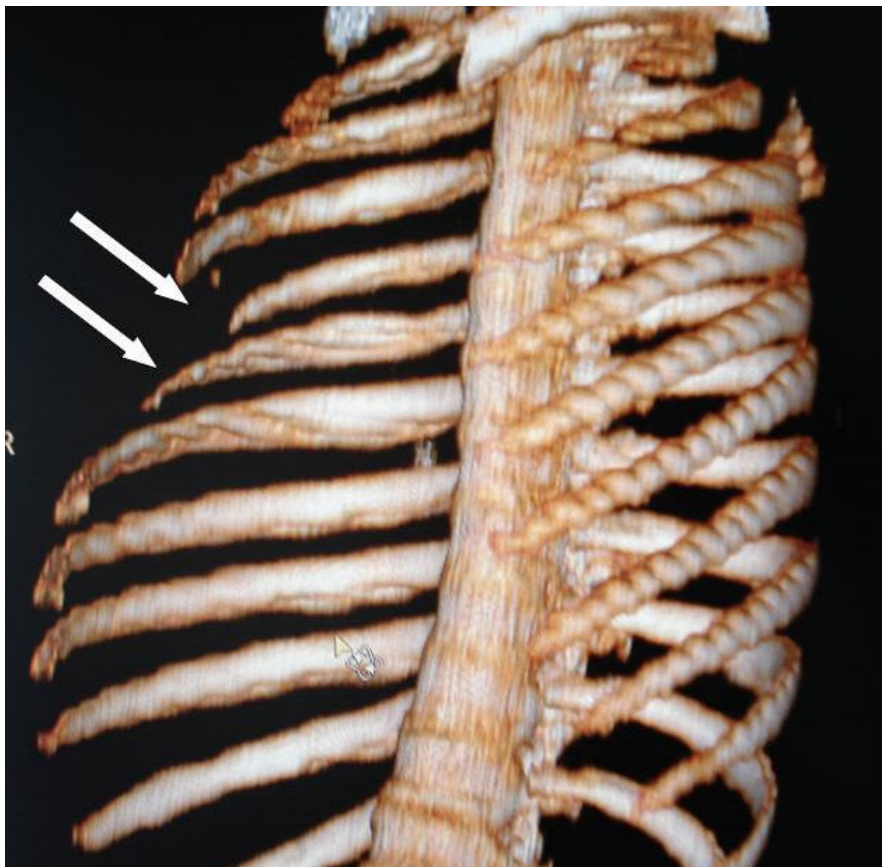


Figure 4C. On volume rendering three-dimensional computed tomography (3D-CT) there were rib anomalies (arrows)



The abdominal ultrasonography and Doppler imaging of the neck were free of any pathologic findings. The results of the respiratory function tests revealed mild restrictive respiratory insufficiency (FVC: 3.39 L 67%; FEV1: 2.88 L 63%; FEV1/FVC: 74%).

Discussion

Poland syndrome, as first described by Alfred Poland in a 26-year-old male, most often occurs sporadically.^{2,8} Some authors have suggested that Poland syndrome is a genetic disease which is inherited with an autosomal dominant pattern.⁹ In fact, Poland syndrome has even been reported in more than one member of a family and referred to as the Familial Poland syndrome⁹. In contrast, Stevens et al⁶ have reported the presence of Poland syndrome in one of two monozygotic twins; thus, there was no purely genetic transmission. Poland syndrome was not noted in the family members of our cases.

Although the disease pathology underlying Poland syndrome is not well understood, various hypotheses have been advanced. The most widely agreed-upon hypothesis is a decrease in blood flow during the intrauterine period due to development of malformations or spasm of the brachiocephalic arterial structures following mutation of the upper extremities while budding from the chest wall in the 6th–7th weeks of pregnancy.

Blood flow disruption in the subclavian artery is known to be a cause of upper extremity injury, while injuries of the pectoralis major muscle, breast, and the other chest wall structures has been reported to be due to an effect on the internal thoracic artery.^{4,10,11}

In addition to anomalies of the pectoralis major muscle, which is associated with this mechanism, syndactyly of the fingers, ipsilateral nipple anomalies (hypoplastic, aplastic, or inverted nipples), ipsilateral radius and ulnar anomalies (hypoplasia and aplasia), and anomalies of the ribs, are also rarely observed.

Ipsilateral breast hypoplasia and reduction in axillary and chest hairs was observed in both of our cases; however, there were no syndactyly or forearm anomalies. Nonetheless rib anomalies at two levels that led to ipsilateral volume loss in the right hemithorax, was observed in the second case.

A rare association between Poland syndrome with other organ system-related symptoms including microcephaly, cerebral atrophy, disorders in myelination, situs inversus or dextrocardia, hemivertebra, gastroschisis, paralysis of the cranial nerve or mental retardation, psychosocial retardation, hypospadias, and urinary system anomalies have also been reported⁶. No pathologic findings were observed on the abdominal ultrasonography and Doppler imaging of the neck in either of our cases. There was no dextrocardia and no psychiatric or neurologic complaints.

Absence of the pectoralis major muscle is usually unilateral and almost always observed on the right side. Our first case had involvement of the left side, which has been reported as a rare condition; whereas in our second case, the defect was on the right side.

In 1998, Karnak et al¹³ replaced this generally accepted opinion by publishing the first case of a 6-year-old girl who was described as having bilateral Poland syndrome anomalies due to absence of the pectoralis major muscles, symmetric chest wall deformities, and bilateral arm anomalies, together with hypoplasia of the breast tissues and nipples.

In spite of Poland syndrome generally causes aesthetic problems, surgical intervention has tried for only functional aims. In this context, many attempts at surgical intervention have been performed for problems, especially the forearm and fingers. Defects of the chest wall do not generally require treatment.

No cases of Poland syndrome associated with respiratory dysfunction verified with spirometry have been reported. Normal spirometric respiratory function was observed in a case with Poland syndrome reported by Deniz;¹⁴ however, mild decreases in the maximum inspiratory pressure (MIP) and maximum expiratory pressure (MEP) were detected. In our second case, there was volume loss in the ipsilateral hemithorax and restrictive respiratory insufficiency, accompanied by anomalies of two ribs.

Our cases admitted with complaints of loss of strength in the ipsilateral shoulder and arms. The physical examination revealed loss of strength of the affected side. Mysnysk et al¹⁵ have reported the loss of 20% and 29% of horizontal strength following measurement with a Cybex dynamometer in a study involving two professional wrestlers with Poland syndrome. Quantitative evaluation with electromyography (EMG) was also planned in our cases; however, the patients declined testing.

Endocrine anomalies, melanosis, and an increase in the incidence of benign and malignant tumors may also be observed in Poland syndrome. Although the most commonly encountered malignant tumors are lymphoreticular tumours, such as leukemia and lymphoma, childhood solid tumours, such as neuroblastoma and Wilms' tumours, may also be observed¹⁶.

A case of a 12-year-old girl who admitted with amenorrhea and absence of right breast enlargement has been reported¹⁷. No endocrinologic or oncologic problems were reported in our two cases.

The present report of two cases presented with left-sided involvement and rib anomalies with respiratory dysfunction, accompanied by café au lait spots is of clinical importance due to the rare occurrence of Poland Syndrome with an incidence of 1/30,000.

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The Annual Meeting at Auckland. The President's Address

Ladies and Gentlemen,—

The inaugural meeting of the annual conference of the British Medical Association is a fitting occasion for a public gathering; since of all professions the medical one is the most intimately bound up with public interests, with questions of social advancement and the amelioration of conditions of living, with the welfare and happiness of the individual, and with the maintenance and prolongation of the period of usefulness of each unit of society. The day when disease and its effects were looked upon as a visitation of God or the gods is happily a thing of the past.

To-day the public mind has been aroused to the knowledge that, broadly speaking, disease is the result of unsuitable and unsanitary surroundings; it appreciates that the ravages of typhoid, plague, and other deadly infectious diseases are not best avoided by this or that particular line of treatment when once the disease has broken out, but by proper sanitation, by a scientific system of drainage, and the maintenance of proper air spaces about individual homes and the conservation of suitable large open areas and public recreation grounds, and I have no hesitation in saying that the serious outbreak of typhoid fever which occurred in this town two years ago and which taxed your hospital resources to the utmost, and which cost the community the lives of many useful citizens, including those of several nurses who contracted the disease in the discharge of their duties in nursing the sufferers, was a very grave, reflection on a city like Auckland, which can find money to build bridges and Town Halls whilst yet it has no proper system of drainage and whilst the sanitation of many parts of the city and Suburbs is a disgrace to any civilised community.

The desirability and usefulness of bridge and Town Hall building I in no sense decry, but I do say that desirable as they may be, their public usefulness is not for one moment to be compared with that of a proper system of drainage, and this later ought to be pushed on with, and every effort and all available financial energy directed towards its completion at the earliest possible date. The mental and physical-well-being of the individual members of the community is a matter of very pressing moment to the City and the State, since every departure from such condition becomes either directly or indirectly a charge upon and inconvenience to the community.

Every healthy man or woman is or ought to be an asset of the State, hence the necessity for the preservation of individual health and the prevention and cure of disease by all means in our power. To this end all over the country in all big centres, and also in many ridiculously small ones, we have hospitals established. With the increase of population these have increased in size, and we constantly hear complaints of the expense of these institutions to the ratepayer and taxpayer. This, Ladies and Gentlemen, is frequently a very short-sighted point of view. In thickly-populated centres disease tends to increase in a ratio greater than the actual increase in numbers, hence hospital expenditure may be expected to increase in geometrical rather than in arithmetical progression, and whilst I urge as strongly as anyone the necessity for the economic management of these institutions according to all the most strictly business-

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like and commercial methods, yet in no case should efficiency be sacrificed to economy, and it is very necessary to bear in mind that a sick man or a dead man belongs to the debit and a healthy one to the credit side of the State balance sheet.

Let us have our large hospitals equipped in every respect with all modern convenience and appliances which have been proved useful in the treatment of disease, the community will save in the end.

I have said that Hospitals are established in some ridiculously small districts, and this is, I think, a unwise and wasteful procedure. No small hospital can be thoroughly well equipped except at extravagant rates, since if equipped for all emergencies large sums will be wasted in appliances which may never be used or used so seldom that when needed they are out of order and those in charge of them have never has opportunity for becoming expert in their use. County hospitals should be receiving stations for the urgently ill and for accident cases so severe that they unfit the sufferers for travelling to a large centre. Other cases of serious disease should be drafted at the expense of the small districts to a hospital in a large centre, only in a large centre can every convenience be economically provided, and only in a large centre can the experience of disease be developed to its full extent and the patient get the advantage of expert skill.

The Mental Hospitals also are becoming an ever-increasing charge on the community, and whilst we are all agreed that the care of the mentally afflicted is as much the duty of the community as the care of the bodily ill, and that such cases should be treated on the most modern and humanitarian lines, yet the increasing ratio of mental disease is becoming so alarming a problem that sooner or later serious steps will have to be taken to check this increase, and for myself I hope the day is not far distant when the State will forbid marriage of individuals who are or have been the subject of disease which is likely to prove hereditary, and will rigidly enforce the permanent segregation of individuals the subjects of chronic or relapsed mental disease, the personal liberty of such tends to increase of criminality and mental disease and must ultimately become a serious menace to the State.

How many people, I wonder, ever contemplate the immense commercial value of the recent advances in the science of medicine. For example, the discovery by Manson and Ross after years of research of the fact that malaria is always carried by a certain species of mosquito, which can easily be exterminated ; later the discovery of the causes of other deadly tropical diseases, for example, Sleeping Sickness. Consider what this means—that huge areas of the globe up to now uninhabitable, areas rich in minerals, vegetation and general productiveness, will be thrown open to civilization, and the immense potential riches made available for mankind.

In conclusion, Ladies and Gentlemen, let me say that the medical profession, though individually inclined rather to Conservatism, is the most Liberal of all. None so ready to take advantage of any and every discovery in any branch of science in which there appears the least chance of benefit in the treatment prevention of disease, and let me remind you that both individually and collectively the medical profession have done

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and are doing more to enhance the welfare of the individual and to aid the
general progress of social advancement that you are sometimes inclined to credit them
with.





Proceedings of the 203rd Scientific Meeting of the Otago Medical School Research Society, Thursday 15 July 2010

The predictive relationship between activity change and functional disability in acute low back pain: A prospective cohort study. P Hendrick¹, L Hale¹, M Bell², S Milosavljevic¹, D Hurley-Osing³, S McDonough⁴, D Baxter¹. ¹Centre for Physiotherapy Research, School of Physiotherapy, University of Otago, ²Department of Preventive and Social Medicine, Dunedin School of Medicine, University of Otago, ³School of Physiotherapy and Performance Science, University College Dublin, Ireland, ⁴Health & Rehabilitation Sciences Research Institute, School of Health Sciences, University of Ulster, Northern Ireland.

Activity advice and prescription are commonly used in the management of low back pain (LBP). However, no research has assessed whether objective measurements of physical activity predict outcome, recovery and course of LBP.

One hundred and one patients with acute LBP were recruited. Activity levels at baseline and 3 months were measured with an RT3 triaxial accelerometer and the Seven Day Physical Activity Recall (7d-PAR) Questionnaire (n = 83). Habitual activity levels prior to the onset of LBP and at 1 year were measured with Baecke Physical Activity Questionnaire (BPAQ). Each participant completed the following LBP outcomes: Roland Morris Disability Questionnaire (RMDQ), Visual Analogue Scale (VAS) pain measurement and a “simple” activity question, detailing resumption of “normal” activities (Y/N), at baseline and 3 months. At 1 year seventy seven participants returned completed VAS, RMDQ, BPAQ and modified Nordic LBP Questionnaires.

There was no significant change in activity levels measured with the RT3 ($P = 0.56$) or the 7d-PAR ($P = 0.43$) from baseline to 3 months or BPAQ activity levels from baseline to 1 year ($P = 0.70$). Objective physical activity change (RT3) did not predict RMDQ change in multivariate analysis at 3 months ($P = 0.84$) or 1 year ($P = 0.77$). A patient report of a return to full “normal” activities at 3 months ($P < 0.0001$) predicted RMDQ change at 3 months. A higher VAS score at 3 months ($P = 0.02$) predicted chronic LBP (Nordic LBP Questionnaire) at 1 year.

None of the objective or questionnaire activity measures at baseline, 3 months or 1 year predicted functional recovery in acute LBP. These results question the role of activity in LBP recovery and stress the importance of the patient’s perception of activity ‘normalisation’ in recovery from acute LBP.

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Effectiveness of a sleep hygiene programme, developed with youths, on sleep hygiene, daytime somnolence, sleep quality and body mass index. E Tan, B Galland, P Cleland, C Lobb. Department of Women's and Children's Health, Dunedin School of Medicine, University of Otago, Dunedin.

Childhood sleep disturbance, often due to poor sleep hygiene (or habits), can negatively impact on daytime functioning. Although effective, few sleep hygiene interventions target youth specifically and none, as we know, have been developed in consultation with youth. We aim to evaluate the effectiveness of a novel 20 week F.E.R.R.E.T (an acronym for Food, Emotions, Routine, Restrict, Environment and Timing) sleep programme, developed in consultation with 21 youths, in improving sleep, sleep hygiene and daytime functioning.

Youths (aged 10-18 years) with self-identified sleep problems (e.g. initiating and/or maintaining sleep) were recruited and the programme delivered by one researcher. An education pack, ongoing telephone and outpatient support was provided. Participants completed the Pediatric Daytime Sleepiness Scale (PDSS), Adolescent Sleep Hygiene Scale (ASHS) and Pittsburgh Sleep Quality Index (PSQI) twice before (5 and 1 week) and three times after intervention (6, 12 and 20 weeks). Body mass index (BMI) Z-scores were also obtained.

Thirty-three youths (mean age 12.9 years; M/F = 1.2) enrolled and retention was 100%. We found significant improvements in daytime sleepiness, sleep hygiene and sleep quality. PDSS scores (mean = 16.69) improved (-4.87, 95% confidence interval or CI -6.45 to -3.29; $P < 0.001$) as did ASHS scores (mean = 4.72) post-intervention (0.20, CI 0.07 to 0.32; $P = 0.002$). PQSI scores (mean = 7.75) also improved (-3.16, CI -3.90 to -2.42; $P < 0.001$) after intervention. BMI Z-scores (mean = 0.8) decreased significantly post-intervention (-0.13, CI -0.20 to -0.05; $P = 0.001$), despite no height change.

The F.E.R.R.E.T sleep programme is effective in improving sleep and daytime sleepiness, and might prove feasible for weight management as seen in the BMI reduction - we will now target obese youth, a group which often experience disturbed sleep. The consultation process might have accounted for the positive outcomes and high retention.

Direct regulation of *c-Myc* by cohesin. J Rhodes, F Bentley, J Horsfield. Department of Pathology, Dunedin School of Medicine, University of Otago, Dunedin.

Cohesin is a multi-protein complex that plays an essential role in sister chromatid cohesion. Recently it has emerged that cohesin also functions in regulating gene expression, sometimes in combination with CCCTC-binding factor (CTCF). Our work has revealed that cohesin positively regulates the *c-Myc* oncogene. In zebrafish, loss of cohesin subunits *rad21* and *smc3* reduced the transcription of *myca* (zebrafish *c-Myc*), indicating that cohesin is required for normal *myca* expression. Cohesin

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regulation of *c-Myc* is conserved in *Drosophila*, mouse and human. We aim to understand the mechanism by which cohesin regulates *myca* expression.

Chromatin immunoprecipitation (ChIP) performed on 1-day-old zebrafish embryos revealed that the Rad21 subunit of cohesin binds to the transcriptional start site of *myca* (4.4 ± 0.5 , fold enrichment relative to site where no Rad21 binding is predicted \pm SEM) and an upstream CTCF binding site (8.9 ± 1.9). Although CTCF has been shown to recruit cohesin, Rad21 still bound these sites in *ctcf* knockdown embryos in which *myca* expression was unaltered. Greater enrichment of Rad21 was detected at the upstream site in the absence of *ctcf* ($P = 0.0098$, two sample *t*-test, $n = 5$).

To determine if the Rad21 binding site upstream of *myca* affects chromatin structure at the *myca* locus, ChIP was performed using antibodies specific for active and repressive histone modifications. From 10 kb upstream of *myca* to the start of the gene, there was no difference between the chromatin structure of *rad21* mutants and wild type embryos. However, repressive chromatin marks (reduced acetylated H3K9 and increased trimethylated H3K27) were observed at the transcriptional start site of *myca* in *rad21* mutant embryos.

In conclusion, the regulation of *myca* by cohesin is direct and appears to be independent of CTCF. Down-regulation of *myca* transcription by cohesin depletion is associated with repressive histone marks at the *myca* gene.

A model system for heterologous expression of cysteine-rich secretory proteins, antigen 5 and pathogenesis-related group 1 proteins in *E. coli*. A Nguyen¹, T Milne², B Monk², J Tyndall¹. ¹National School of Pharmacy, ²Department of Oral Sciences, Faculty of Dentistry, University of Otago, Dunedin.

The Cysteine-rich secretory proteins, Antigen 5 and Pathogenesis-related 1 proteins (CAP) superfamily appears ubiquitous in eukaryotes. Most CAP proteins are multidomain proteins rich in disulfide bonds. Some CAP proteins are proposed to have essential roles in mammalian reproduction, others have been linked to gliomas and prostate cancer, but their biochemical functions are poorly understood. Tex31, a protein isolated from the venom of *Conus textile* (Cloth of gold cone), is the only CAP member identified with an enzymatic activity. In an effort to further understand the structure and function of CAP proteins, we have expressed the pathogenesis-related domain of Tex31 (PRD_{Tex31}) in soluble form.

PRD_{Tex31}, the N-terminal truncated PRD (NdeI_{PRD}Tex31) and the disulfide mutant constructs of NdeI_{PRD}Tex31 were heterologously expressed in *E. coli* strain DH5 α using maltose-binding protein (MBP) as a fusion partner and purified using amylose-affinity chromatography and size-exclusion chromatography (SEC). A significant fraction of each Tex31 chimera (~80%) was affinity-purified. Disulfide bonds in the PRD_{Tex31} constructs caused the formation of aggregated soluble complexes. Ablation of a specific disulfide bond in recombinant NdeI_{PRD}Tex31 constructs plus the addition of the mild detergent n-dodecyl- β -D-maltoside together with sonication prior to SEC eliminated small heat-shock proteins IbpA/B from the preparation and 50% of the MBP-NdeI_{PRD}Tex31 was recovered in soluble, monodisperse form.

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Crystallisation of MBP-NdeI PRD Tex31 using hanging-drop vapour-diffusion gave non-diffracting, 10 – 30 µm diameter crystals within 6 weeks.

Optimisation of crystallisation conditions for MBP-NdeI PRD Tex31 chimeras is needed to obtain crystals that produce suitable X-ray diffraction data.

This expression protocol can now be applied to other members of this protein superfamily in an effort to understand their function.

Permanent spatial memory deficits after bilateral vestibular deafferentation and the effects of cannabinoids. J-H Baek, Y Zheng, C Darlington, P Smith. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Previous studies of rats with bilateral vestibular deafferentation (BVD) have demonstrated spatial memory deficits. In this study, we investigated whether rats exhibited spatial memory deficits at 14 months following BVD and whether these deficits could be exacerbated by the administration of cannabinoid (CB) drugs.

Twenty-eight adult rats were divided into 4 groups: 1) sham surgery + vehicle (n = 8); 2) sham surgery + the CB₁/CB₂ receptor agonist WIN55,212-2 (WIN) (n = 7); 3) BVD + vehicle (n = 6); and 4) BVD + WIN (n = 7). WIN, at a dose of 1 or 2 mg/kg/day, or vehicle was administered (s.c.) on days 1 – 10 and 11 – 20 (respectively), 30 min before the rats performed in a foraging task to test spatial memory.

Unexpectedly, the BVD animals were impaired in using the visual cues in the light during the probe trial, where the home location was changed to a novel position. Sham animals made significantly more visits to the old home compared to the BVD animals ($P = 0.02$, ANOVA). In the dark, when visual cues were unavailable, BVD animals were unable to use self movement cues to remember the correct home location ('homing'), which was demonstrated by their significantly longer homing distance and time (both $P = 0.000$, ANOVA). Whereas the sham animals had a clear orientation toward the correct home ($P < 1 \times 10^{-12}$, Rayleigh's test), BVD animals did not. However, WIN at 2 mg/kg/day significantly reduced the homing time ($P = 0.000$, ANOVA) and number of errors ($P = 0.004$, ANOVA) of BVD animals.

These results suggest that at 14 months post-BVD, the animals are not only impaired in a spatial memory task when visual cues are unavailable, but even when they are available. The involvement of the CB system is more complicated than expected.

Lessons for patient safety in primary care from the 'no-fault' treatment injury database. K Wallis, S Dovey. Department of General Practice and Rural Health, Dunedin School of Medicine, University of Otago, Dunedin.

Legislative reforms in 2005 removed 'error' from medical injury compensation in New Zealand and provided for the creation of a unique 'no-fault' treatment injury database. This study aimed to analyse the first four years of data from the 'treatment

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injury' claims database to identify targets for improvement in patient safety in primary care.

There were 6007 primary care treatment injury claims lodged and 3853 were accepted (64%). Most claims were 'minor' (83%), but there were 729 'major' (12%), 244 'serious' with 'the potential to result in death or permanent loss of function' (4%), and 58 'sentinel' 'resulting in death or major permanent loss of function' (1%). 'Serious' and 'sentinel' events are reported to the Director General of Health and sometimes also the Medical Council or Medsafe. Medication caused most injuries (45%) and most 'serious' and 'sentinel' events (62%). Injuries included haemorrhage from warfarin (13), renal failure (7) and gastric bleeds (5) from anti-inflammatory drugs, strokes (4), thromboembolism (3) from oral contraceptives, avascular necrosis from steroids (7) and pulmonary fibrosis from nitrofurantoin (5). 'Injections and vaccinations' caused 12% of injuries and 11% of 'serious' and 'sentinel' events. Delay / failure to diagnose caused few injuries overall (2%) but a disproportionate number of 'serious' and 'sentinel' events (15%) for missed cancer (15), infection (6), testicular torsion (5), and fatal cardiac conditions (4). Spinal / neck manipulation by chiropractors and physiotherapists caused 3% of injuries and 2% of the 'serious' and 'sentinel' events including strokes (4). Ear syringing caused 3% of injuries including 78 perforated eardrums.

Analysis of the treatment injury database identifies targets for improvement in patient safety in primary care. Information about the type, prevalence and impact of injury can be used to guide injury prevention initiatives and educate practitioners about the dangers and pitfalls in practice and also to inform 'consent' discussions with patients.



Overtime—a risk factor for coronary heart disease (CHD)?

The epidemiologists in this study observe that CHD and overtime work are both common features in developed countries. They speculate on their relationship in this prospective study of 6014 British civil servants (70% men) aged 39–61 yrs over 11 years. Their conclusion was that 3–4 hr overtime work per day was associated with 1.60-fold increased risk of incident CHD compared with employees with no overtime work. Adjustment for all 21 cardiovascular risk factors measured made little difference to these estimates. The overall increased risk was 1.67 for fatal cardiovascular events and non-fatal myocardial infarction.

Convincing? Maybe—an editorial writer points out that the blood pressure risk factor issue is uncertain as only one reading of blood pressure was taken at the outset and no further readings were taken. He also points out that no information is available about whether the participants were taking cardio-protective medication. These points might confound interpretation of the results. As an aside it is slightly amusing that although the researchers have called their study the Whitehall Study, the majority of the authors are based outside England (3 Finland, 1 France and 3 England).

European Heart Journal 2010;31:1737-44 & 1672-3.

Age adjusted D-dimer cut-off value in the elderly?

The D-dimer assay is commonly used as a screening test for the presence of thromboembolic disease. The usual cut-off value is $<500\mu\text{g/l}$ (500ng/ml)—i.e. thromboembolism unlikely below the cut-off. However, as we have noted in the past (NZMJ 12/10/07) there is evidence that D-dimer levels are higher in the elderly and it has been suggested that the threshold should be raised in the elderly.

This retrospective cohort study from Europe involves 3000 patients who were suspected of having thromboembolic disease. All had investigation for pulmonary embolism consisting of a clinical probability calculation, a D-dimer test, and, finally, computed tomography or leg venous compression ultrasonography, or both. The diagnosis was correlated with the D-dimer levels and the age of the patient by decade—i.e. 50s, 60s, etc. They derived a formula for the cut-off age—patient age $\times 10$ ug/L. So the cut-off age for a 75 yr old would be 750. They believe that the new cut-off value, combined with clinical probability assessment, greatly increased the proportion of older patients in whom pulmonary embolism could be excluded without reducing safely. Now we need a prospective study to validate their proposition.

BMJ 2010;340:c1475.

The family history in the medical record

All medical students are told of the importance of the family history of their patients. This study reports on the quality of the family histories recorded in the short-stay

medical unit admission notes in an Australian teaching hospital (The Royal Perth). The short-stay unit is for general medical patients who are expected to stay 3 days or less. 300 case notes were randomly selected and reviewed. The family history was well done in 48 (16%), insufficiently done in 31 (10.3%) and not recorded at all in 221 (73.7%). There was a trend to more comprehensive family histories in younger patients and those with chest pain. Not good. Why? Perhaps the short-stay ward situation; perhaps it may reflect local issues in undergraduate education in West Australia. Could a high work load be relevant? We can presume that this study will provoke some soul searching.

MJA 2010;192:682-4.

Reduced retinopathy progression in Type 2 diabetes

The ACCORD (Action to Control Cardiovascular Risk in Diabetes) Study group are primarily interested in cardiovascular events in Type 2 diabetes but are also interested in the reduction of retinopathy progression. In this paper they report on a sub-group of 2856 diabetic patients who were randomised to receive either intensive or standard treatment for glycemia (target glycated hemoglobin level, <6.0% or 7.0 to 7.9%, respectively) and also for dyslipidemia (160 mg daily of fenofibrate plus simvastatin or placebo plus simvastatin) or for systolic blood-pressure control (target, <120 or <140 mm Hg). At 4 yrs there was significantly less retinopathy progression in the intensive glycemia treatment group (7.3% vs 10.4%).

The fenofibrate plus simvastatin group also had a significantly less progression rate in their retinopathy (6.5% vs 10.2%). There was no reduction in progression in those whose hypertension was more stringently controlled. We will wait with interest longer follow up reports.

N Engl J Med 2010;363:233-44.

Ambulatory blood pressure monitoring (ABPM) in the community

Blood pressure (BP) recordings in the surgery or clinic have many failings—the main fault is that they are a one off snapshot and may be unrepresentative of the BP during 24 hrs. Other faults include different sphygmomanometers, different staff recording the BP and the white-coat effect. Labile and intermittent high or low BPs may evade detection. This review article provides evidence for the added value provided by ABPM. Several papers support this view including one meta-analysis involving >7000 patients followed over a median time of 9.5 yrs—ABPM better than conventional reading in predicting cardiovascular events.

Apparently the American Heart Association and the American Society of Hypertension favour ABPM. The equipment—a cuff, a small monitor weighing less than one pound attached to a belt and a connecting tube. Great, but there must be a hitch. Yes, two in fact. Some patients find they are uncomfortable and disturb their sleep. More important they are expensive and many health insurers in the USA will not reimburse their use.

Southern Medical Journal 2010;103:447-52.



Reassessing Cartwright—understanding the factual record

I hope that my delayed entry into the public controversy concerning the Cartwright Inquiry may help clarify some of the many points raised in the 30 July 2010 issue of the *New Zealand Medical Journal*.

The nature and role of the ‘1984 paper’

A number of assumptions and assertions concerning this paper and its role demand elucidation.

A retrospective study—Professor Linda Bryder stated in her book¹ and at a seminar at the University of London on 16 June 2010 that Sandra Coney has ‘admitted’ that the authors of the 1987 original Metro article^{2(pp.47–64)}, Sandra Coney and I, had not understood the retrospective nature of the 1984 analysis.^{1 (p.35)}

This is a misrepresentation. Coney explained in her book³ that, on first reading the 1984 paper in 1985, it was not clear who was responsible for the clinical management of the patients whose cases were reported there. However, her book went on to describe how, having undertaken extensive research, including interviewing all the authors of that paper, and having seen the internal hospital memoranda concerned, we were quite clear, well before writing the Metro article in 1987, that it was a retrospective analysis. Coney was describing our research process not making an ‘admission’ of error.^{3(p.17)}

The validity of the Cartwright Findings is independent of the 1984 paper—It has been asserted by both Professor Bryder^{1(p.35)} and Helen Overton⁴ that the Cartwright Inquiry made the same alleged error. Close reading of the Cartwright Report shows that this is incorrect.

The 1984 paper was not central to the Inquiry process or its findings. The Inquiry had access to thousands of original clinical case notes of women treated at National Women’s Hospital during the relevant period. These case histories provided the critical evidential base of the Inquiry and showed a most significant gap between what various Parties said, especially Dr Green, and what had actually been performed on patients.

The important role of the patient case notes

Parties to the Inquiry had full access to this case evidence on condition of preserving the anonymity of individual patients. These cases were the subject of intensive cross examination of expert witnesses. Case material also provided much of the content presented in the three submissions by Phillida Bunkle, Sandra Coney and Dr Forbes Williams^{5–7} as well of many other Parties.

I believe it was because this case evidence was so compelling that none of the Parties appealed the Inquiry findings.

Unfortunately, researchers who were not involved in the judicial process do not have access to this crucial evidence. This places severe limitations on attempts to reassess the evidential base of the Inquiry.

Professor Bryder seeks to overcome these limitations, by:

- Reference to the 2 case histories published in the appendix to the Cartwright Report,^{10(pp.268–286)}
- Analysis of the crucial cross examination of international expert witnesses about these cases, and
- Use of evidence from patients either in letters or presented to the Inquiry.^{1(pp.4,48-52,53-54,57-59,61-65,132,134-135)}

In the absence of access to the case notes this procedure is, however, weak. The Inquiry identified 131 cases similar to the two contained in the Appendices and it is only by considering the detailed facts of those notes that the crucial issue of treatment of curative intent can be illuminated. Moreover, it is difficult to decipher the import of the cross examination without access to the clinical records the witnesses referred to.

Finally many of the letters from women were solicited by counsel for Dr Green, Professor Bonham and the University of Auckland. They were often from women who did not yet know what had happened to them; most were not subject to cross examination and, hence, rate as inferior to evidence based on case histories or expert testimony that was subject to cross-examination by Parties of all persuasions.

Professor Bryder also quotes from the cross examination of two patients who gave evidence publicly. For example, she quotes from one woman who was very satisfied with her treatment and appreciative of Dr Green's care.^{1(p.49)} This case, however, demonstrates the difficulties of evaluating such evidence without access to the patient's case notes.

What the judge, counsel and Parties were aware of, but the patient and Professor Bryder were not, is that the patient had been repeatedly observed as a research subject without treatment for many years while CIS spread throughout her vagina. Rather than support the view that Dr Green provided excellent care, this patient's statements demonstrated how uninformed she was and how seriously her trust in National Women's Hospital was misplaced.

The significance of the Cartwright Inquiry as a judicial inquiry.

It is important to clarify that the Cartwright inquiry was a *judicial* inquiry with status, process and rules of evidence equivalent to those of the High Court. It examined the second-hand evidence of the 1984 paper but only accepted its findings in so far as they were corroborated by its own evidence base as interrogated by international experts.

The appropriate appeal of findings of such an inquiry is via an application to the High Court for a Judicial Review by participating Parties. Presumably, had counsel for aggrieved Parties judged that there had been weaknesses in the evidence or the process of its evaluation, they would have recommended an appeal. None did so.

An attempted application for Judicial Review by a member of the public friendly with Dr Green was struck out, in part on the grounds that the applicant had no standing with the Inquiry, was not familiar with the issues, and because it was considered that it was wrong for an unconnected person to launch an appeal when the Parties themselves had not chosen to do so.⁸

Evidence in a judicial process—It is important to understand that the Inquiry independently examined this body of original evidence. The team of medical advisors, which consisted in Professor Eric McKay, a gynaecologist from Australia, Dr, later Professor Dame Linda Holloway, a pathologist, and Dr, later Professor, Charlotte Paul an epidemiologist.

The team of medical advisors were officers of the inquiry not witnesses. They did not give evidence. They advised the judge.

It also follows from the judicial status of the Inquiry that when its findings are contested in the media the judge cannot defend herself. (Imagine the consequences if every court decision was publicly contested by the judge.) It has, therefore, been appropriate that members of the medical advisory team, Professors Holloway and Paul, who are familiar with the evidence, have played a role explaining the findings of the Inquiry.

Definitions of ‘conventional treatment’

Professor Sir Iain Chalmers criticises Professor Paul for not providing the Inquiry with a definition of ‘conventional treatment’.⁹ It is not the role of the medical advisory team to give evidence; they are not witnesses and are not cross examined. A judge relies on the advice of experts who can be cross-examined. These expert witnesses are responsible for reviewing the published evidence (of much of which they were the authors) in giving their opinions. The judge noted that the experts’ advice was ‘derived from an examination of medical literature, a review of research projects and personal experience in practice’.^{10(p.106)} Counsel representing Parties of all interests participated in cross examining these experts.

The judge concluded that: ‘the appropriate treatment of CIS, if invasive cancer is to be avoided, is to remove the lesion. The patient must then be monitored so that further treatment can be offered if there is persisting disease or a recurrence...’.^{10(p.106)}

The judge also found that: ‘All overseas authorities were agreed that since the mid-1950’s the aim in treating a patient with a diagnosed cancer precursor, including CIS, has been to eradicate the disease. The method of treatment has always depended on the available skills and equipment, but the aim remains unchanged....’.^{10(p.107)}

The definition of ‘treatment’ is eradication of the lesion rather than a protocol of specific interventions. None of the experts thought, however, that diagnostic wedge or punch biopsies were ‘treatment’ even though such biopsies very occasionally have the effect of eradicating lesions. ‘Treatment’, thus, implies curative intent not just a particular procedure.^{10(p.104)}

A series of propositions flows logically from this definition:

- Whatever eradication procedure was used, whether the more usual cone biopsies or the less common hysterectomy or other forms of excision, follow-up was necessary to ensure complete eradication, with more extensive removal if abnormal cells continued to be detected.
- Those patients who continued to have positive smears, sometimes for years, without attempts to remove the abnormality can be considered to have been inadequately treated.
- Procedures that were not directed at removing abnormal tissue were not 'treatments of curative intent'.
- 'Treatments of curative intent' can be distinguished from those procedures which are not, according to whether their purpose was to remove abnormal tissue or not.
- Counting the number of surgical procedures each patient eventually had does not show that no women with CIS was untreated^{4:1}. In some cases, intention changed. Long periods of interventions with no curative intent, were followed by drastic procedures to eradicate malignancy.^{1(p.149)}

Case notes, the original research proposal¹⁰⁽⁴⁻⁷⁰⁾ and his many publications showed that Dr Green, was following some women who had had only diagnostic biopsies. For instance, in 1970, he described following '75 patients with untreated or incompletely treated CIS'.¹¹

Further, these were not the only form of non- or inadequate treatment; patients with cervical micro-invasion, vaginal and or vulval CIS, and other abnormalities of the genital tract were also involved.¹⁰⁽²³²⁻²³³⁾ Professor Joe Jordan, an expert witness, for example, noted that there was 'another group where a definitive diagnosis of microinvasive carcinoma was made and ignored'.^{12(p.28)} The judge subsequently found evidence of 'cases where microinvasive carcinoma has not been treated with even the least radical procedure'.^{10(p.113)}

Finally the definition of treatment identifies that for many women their *CIS* was not treated. It is true that they may *eventually* have had extensive surgical procedures, after diagnoses of microinvasive or invasive cancer had developed. But even then there were frequently delays of years.

Thus, it is not the case, as Professor Bryder claims, that there is no evidence of non-treatment or that it is impossible to distinguish groups which differ by treatment.

The support of Professor A Cochrane

Professor Chalmers is concerned that there is inadequate recognition of Professor Cochrane's support for randomised trials to settle the issues involved.⁹

The Inquiry found evidence that such trials were considered unethical even at the time. Evidence to the Inquiry showed that Dr Green cited Professor Cochrane's support in his internal memorandum of 1973 justifying his research.^{10(p.82)} In his own evidence to the Inquiry, Dr Green also testified, in two places, that Professor Cochrane supported the ethics of his research.^{10(pp.77,79)} He neglected to say, however,

that when he and Professor Cochrane had applied to the Medical Research Council in the UK for support for a randomisation of Green's practice, it was rejected as unethical. Under cross examination Dr Green eventually conceded that he was aware of this.^{10(p.82)}

The judge concluded that 'This is one occasion when I cannot accept that there was an oversight or memory loss on Dr Green's part'.^{10(p.82)} The judge also concluded that the issue should have been followed up by the hospital since the MRC's refusal would have prompted them to reconsider the ethical legitimacy of Dr Green's activities because 'the validity of the 1966 trial would have appeared far more questionable'.^{10(p.82)}

The construction of Professor Bryder's evidence

Professor Chalmers concludes his article by citing, with approval, the conclusion of Professor Bryder's third chapter. This passage reads:

'What then was the conventional treatment' that the patients at National Women's were apparently denied by Herb Green? According to Cartwright it was not hysterectomy which had already been rejected throughout the world as a routine response to CIS in favour of cone biopsy or local excision by the 1960's. Yet many gynaecologists still believed that hysterectomy was the appropriate response to the problem, including star witness to the Inquiry Ralph Richart. A significant minority of gynaecologists was questioning the appropriateness of hysterectomy and cone biopsy, both of which were far from benign procedures. Kolstad might have queried Green's clinical decisions, but he was the first to admit that there were no clear cut answers. Jordan might also have been critical of Green's approach, but he did acknowledge the 'dilemmas' in deciding appropriate treatment for asymptomatic women when the treatment options themselves carried a 'high morbidity'. Jeffcoate recommended cone biopsy only when smears repeatedly continued indicative of malignancy'.^{9(p.111)}

It is quite understandable that without access to the case history evidence Professor Chalmers could accept this summary at face value especially as he is not familiar with the archival record. However, this passage encapsulates a number of problems with Professor Bryder's study. Professor Jordan, for example, is quite clear. He said of some of the women whose clinical notes he reviewed 'the patients, in fact, were not treated. I think that's the point, not even inadequately. They weren't treated'.¹²

One of the most serious concerns is that Professor Bryder sometimes misconstrues critical passages. For example, she uses the quotation concerning Jordan's 'dilemma' five times to suggest that other clinicians sympathized with Dr Green's position.^{1 (pp. 40,50,51,55,149)} In fact, Jordan made two references to clinical dilemmas during his cross examination concerning the terrible fate of women whose CIS had been merely observed while it spread throughout the vagina and in some cases other areas of the genital track.^{13 (pp. 2-5)} These women had the highest mortality. By the time of the Inquiry, 7 of the women who developed vaginal invasion had died of the disease.^{10 (p. 233)} At least 15 of the 19 women identified as having CIS of the vagina, had a previous history of cervical abnormalities. 13 of these 19 developed invasion^{10 (pp. 232-233)} and it was the difficulty in treating these women that posed the dilemma to which Jordan referred.^{13 (pp. 2-5)}

The 'dilemma' Jordan referred to in the passage quoted by Professor Bryder, was a discussion about the decision to be made about patient 60/64 in 1981. The decision was difficult because treatment at this late stage entailed the excision of the vagina

and possibly other genital organs with a very high risk of damage to bladder and colon.^{13 (p.4)}

The sentence in Jordan's statement which occurs immediately before that quoted by Professor Bryder,^{1(p. 40)} but which she omits, could not be more explicit. He said, '*I think that some definitive treatment to the vaginal vault lesion should have been instituted in the early 1960's, and at the latest in October 1965, when the vaginal vault biopsy confirmed the presence of severe dysplasia.*'^{13(p.4)} The full text of Jordan's evidence to the Inquiry, thus, makes clear his view, that the 'dilemma' was created by the more than twenty years of prevarication about diagnosis and delays in treatment. Jordan was extensively cross examined concerning these cases and he is quite clear that the predicament was created by non treatment and delay.¹² This is forthright professional criticism not sympathy, as Professor Bryder would have us believe.

Conclusion

In conclusion, I would like to emphasise that in contributing to this discussion I do not want to leave the impression that I consider any reconsideration of the Cartwright Report to be undesirable. On the contrary, we should always be prepared to objectively reassess its conclusions and recommendations in the light of new knowledge. An objective reassessment would be of far more value to New Zealand women than the current dispute.

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A patient's response to recent criticisms of the findings in the report of the Cervical Cancer Inquiry 1988

In light of the articles and letters published recently in the New Zealand Medical Journal relating to the Unfortunate Experiment, and Professor Linda Bryder's book *A History of the 'Unfortunate Experiment' at National Women's Hospital*, it seems timely and only just, that as one of the patients of National Women's throughout the years concerned, I should present my perspective. I was part of Professor Green's experimental group which becomes obvious when my clinical notes are read. I am the woman who was known as 'Ruth' during the course of the Inquiry. As a party to the Inquiry I received copies of all submissions and transcripts.

I agree with Professor Frank Frizelle that '...it is important...not to forget the real issues...' Patient consent to be part of 'research', 'trials', experiments' – call them what you will, was not obtained during the years in which Professor Green was carrying out his work at the hospital and his research was not confined to carcinoma in situ of the cervix. The progressions of carcinoma in situ of the vagina and of the vulva were also followed. Many women were harmed by these programmes and some died. Like Frizelle, I agree that the system was at fault, which is why there was an apology from the School of Medicine when the affair had drawn to an end, though not from the hospital. Professor Bonham who was Head of Obstetrics and Gynaecology, and Chairman of the Hospital Medical Committee at the hospital during that time, was found guilty of disgraceful conduct by the Medical Council who, in the aftermath, conducted their own inquiry.

There are opinions coming from some who know little about the affair, and have little knowledge or understanding of *The Cartwright Report* and recently produced scientific papers. For their sakes and for those who have not been fortunate enough to read the relevant literature on this topic, I will present my case history; I will also make some comments arising from the literature the Journal has presented to the public, in particular, the writings of Professor Linda Bryder, in the journal and also in her book. There are a number of errors in her book and in her recent editorial. She has corrected two of them in her book in regard to my personal history, but the major ones dealing with her interpretation of my medical history remain unchanged, and her belief that there was no experiment at National Women's is incorrect.

In order to clarify matters it is essential that I present a summary of my clinical notes with my own comments.

In brief:

- August 1964—Admitted for cone biopsy following an A3 smear.
- Green came to see me in the ward and said he was now not going to perform the biopsy. I was discharged after a colposcopy.
- March 1965—Colposcopy and punch biopsy. Path report = carcinoma in situ of the cervix.
- In a letter Green wrote to my GP on my discharge in 1979, he stated that I had had a cone biopsy in 1965. This is incorrect. I did not have a cone biopsy until 1971.

2.4.65 to 6.11.69

- Five smears = 'Cells suggestive of but not conclusive for malignancy.
- One smear = Cells strongly suggestive of malignancy.
- Four smears = Cells conclusive for malignancy.

23.3.70 Colposcopy clinic. Smear = Cells conclusive for malignancy.

24.3.70 Admitted. Wedge biopsies comprised of three sections.

1. Histology: Carcinoma in situ of the cervix.
2. Carcinoma in situ of the cervix.
3. Carcinoma of the cervix with microinvasion.

Discharged.

In 1970, a paper written by Green appeared in *The Australian and New Zealand Journal of Obstetrics and Gynaecology*.

Green wrote:

...The only way to settle finally the problem of what happens to in situ cancer is to follow indefinitely patients with diagnosed but untreated lesions...A group of 27 women (up to December 1967) are being followed without "treatment"...after an initial diagnosis of CIS has been established by biopsy.

21.5.70 to 7.1.71

One smear = Cells strongly suggestive of malignancy

Two smears = Cells conclusive for malignancy

14.2.71 Re-admitted

EUA Cone biopsy of the cervix

Histology: Carcinoma in situ with microinvasion of the cervix.

Discharged.

22.4.71–15.10.71

Two smears = Cells strongly suggestive of malignancy

23.11.71 Re-admitted

E.U.A. Wedge biopsy of the cervix

Histology Report: Carcinoma in situ of the cervix.

Discharged.

10.3.72–11.12.75

One smear = Atypical cells but no evidence of malignancy

Two smears = Cells suggestive of but not conclusive for malignancy

Two smears = Grade 3

In the *New Zealand Medical Journal* October 1974, Green had written:

...This series of 750 cases of in situ cancer, and the following of 96 of them with positive cytology for at least two years, represents the nearest approach yet to the classical method of deciding such an issue as the change or not from one state to another – the randomised controlled trial. It has not been randomised and it is not well controlled, but at least it has been prospective.

Bryder writes in her editorial that in a published oration to the 1990 General Scientific Meeting of the Royal Australasian College of Surgeons in Wellington, Sir Graham Liggins commented on the fact that the 1984 McIndoe article on which 'the cervical cancer inquiry was based, was misinterpreted by the authors of the Metro article and by the judge'. This misinterpretation according to Bryder consisted of regarding it as a prospective study rather than a retrospective study. Bryder's view does not equate with what Green published in the *New Zealand Medical Journal* October

1974.Green's study was prospective and McIndoe's article was a retrospective report of Green's study material.

27.1.76 E.U.A Ring biopsy of cervix. D&C.

Path report = Curettings: Sections show fragments of endocervical tissue and a few portions of carcinoma in situ of the cervix without stroma.

Biopsy cervix = Carcinoma of the cervix – Excision appears incomplete

The overall thrust of Bryder's argument is that Green was not performing an experiment. How then does she explain the conflicting management policies described by Green below?

In April 1968 Green's lecture notes to doctors state: 'Conventional treatment of CIS comprises cone biopsy excision' and that women with 'follow-up doubtful or positive smears require further biopsy excision or even hysterectomy'.

In 1969 Green describes a group of patients diagnosed and treated (sic) by punch biopsy that were given no further treatment and followed with persistent positive cytology.

Surely one group received conventional treatment and the other group were in an experiment of which I was part.

8.4.76–27.9.79 The five smears I had during that time were Grade 1.

This is taken by Linda Bryder as an indication that I had been well treated. It is also where Bryder's 'analysis' of my case breaks down completely as does her criticism of the expert Professor Per Kolstad's evidence.

Kolstad described my case as 'terrifying mismanagement'. Bryder makes the statement that '...under questioning at the Inquiry Kolstad admitted that his source of information on this (my clinical history) was primarily the *Metro* article.' In fact, cross examined by David Collins, counsel for Green, Kolstad made it very clear that his submission had been based on the *Metro* article but that before taking the stand he had read my file and stated that '...I have read it through and it seems to be well up to what was presented in the *Metro* article.' He reaffirmed adamantly what he had said earlier. For Bryder to state that his comment re 'terrifying management' related to one case only, namely mine, is extremely misleading. He studied other files such as those of Mrs T, 62W/10, Mrs L, 64W/214, Mrs R, 66W/74, Mrs C, 69W/63. In each case he used similar terms; 'mismanagement', 'severe mismanagement', 'horrified' by the 'mismanagement' and 'another example of terrifying mismanagement'. Under cross examination he was adamant that the women concerned had not had the appropriate treatment.

What Bryder overlooks is that in my case, the scar tissue caused by multiple biopsies had led to cervical stenosis. The defoliating cells from the endocervix are not available when the smear is taken. The condition also causes severe dysmenorrhoea. At the Inquiry, the dangers of this condition in regard to negative smears were described by Per Kolstad and also by Dr Colin Laverty, a gynaecological histopathologist and cytologist from Sydney who had been called by the Commission. Laverty said '...problems of increased age and previous conisation (often multiple)

must have affected the reliability of cytology in the group of patients which is the subject of this inquiry’.

Assisting Counsel Lowell Goddard pointed out during her final summing up, that this very danger had been spoken of in a paper by Kreiger and McCormack, which Green had used in 1966.

All this information was available to Bryder in the transcripts of the Inquiry and in my own book, both of which she has read. And there is little point quoting specialists who say that a patient need have only three negative smears before discharge as opposed to my five. Good specialists would not make such a decision when the patient was suffering from cervical stenosis and smears could be unreliable, especially when the last histological report of that patient indicated that some form of carcinoma was still present.

Green described the condition in regard to the dysmenorrhoea in my notes when he wrote: ‘On examination the explanation is fairly obvious – the vaginal cervix has now almost disappeared and the external os is so narrowed as to be very difficult to pick up. A No 2 dilator could not be introduced ...she may need admission for a dilatation.’ I was obviously suffering from cervical stenosis still and this should have set alarm bells ringing.

This E.U.A. and Dilatation of Cervix took place on 5.4.77.

The Path Report following this procedure reads as follows: ‘Curettings: L.M.P. Sections show Fragments of carcinoma **devoid of underlying stroma**, probably carcinoma in situ.’

There was nothing equivocal about this report as Bryder states. Malignant cells were still present. That is obvious, and overall my file may indicate ‘follow-up’, but not the ‘careful follow-up’ that Bryder would have us believe. In the light of no definitive, conventional or adequate treatment, any thoughtful person would wonder just what was being followed up. Professor Iain Chalmers complains that no one has provided him with a definition of ‘conventional treatment’. As a practising gynaecologist in the sixties and seventies he should know what conventional treatment involved at that time. If he cannot remember then it has been defined in this response.

When Collins suggested to Kolstad that ‘...there was one equivocal pathological report of probably carcinoma in situ, correct?’ Kolstad’s reply was, ‘Yes. That is not equivocal, that is quite certain that there was something there that could be called carcinoma. They did not have underlying stroma therefore they couldn’t tell if this was an invasive cancer. That is the result of this report and that was on 5th of April 1977.’

Collins asked Kolstad if the pathologist could have reservations about the distinct possibility of it being carcinoma in situ when he uses the words ‘probably carcinoma in situ’, Kolstad’s reply was, ‘Yes, because he had carcinomatous tissue with no underlying stroma, but was a little suspicious that this could also be an invasive lesion and if I had that report, I would at once have done a conization.’

Finally in response to Collins’ insistence of the importance of the negative smears, Kolstad says, ‘I have tried to tell you over and over again now, that 5th April 1977 there was a positive histopathological report...’ It is clear that Koldstad did know

about the five negative smears and that he did not consider them evidence that the disease was cured.

8.9.77 Return to clinic.

Green wrote in my file that the cervix was still somewhat stenosed and commented that 'The histology report is somewhat surprising. Smear taken. See in one year.' Smear Grade 1.

27.9.79 Green wrote 'Findings as before i.e. atrophic cervix...I do not think any further follow-up is indicated.' Smear Grade 1.

This despite the persistent stenosis.

I was discharged from the hospital with no further treatment.

As Bryder says, '...the categorisation of smears was not a precise science.' The concept of false negative smears was not unknown in the sixties and seventies. All the more reason in my case, and the cases of other women, to not disregard a negative smear result after years of positive ones, and biopsies that repeatedly showed the present of, at best, carcinoma in situ, and especially so when stenosis had occurred.

1985. Smear test carried out by my GP returned a result of Grade 3. This time I decided to go to a private specialist. I had not moved between the public and private sectors of the health service prior to this as Bryder alleges in her book. (p 193)

Following a biopsy and examination at Brightside Hospital I was admitted to National Women's and on 28.10.85 I had a further examination under anaesthetic, biopsy of cervix and insertion of caesium after-loaders. The cancer was classified as Stage 1B, and the path report revealed the curettings showing invasive squamous carcinoma of the cervix, as did the biopsy of the cervix. On 17.12.85 I was subjected to a Wertheim's hysterectomy and pelvic lymphadenectomy. I have had no recurrence of the disease and see myself extremely lucky when I consider what happened to some other remarkable women that I came to know.

When I first met with Graeme Overton and he told me that I had had positive smears during my years at National Women's, I was shocked and extremely upset that I had never been told this. Overton's comment was that Green was 'conservative' in his treatment. In a letter he wrote to the Medical Superintendent prior to my radiation and surgery for cancer, Overton's summary of my case ended with '...further biopsy in 1977 showing carcinoma in situ'. This shows Bryder as incorrect again.

There are other points Bryder makes that must be addressed.

Bryder notes that Archie Cochrane supported Green's research but she omits to observe that when the two of them made an application to the British Medical Research Council for funding to continue Green's work, it was declined as unethical. This was conceded by Green at the Inquiry. Bryder is misleading again.

As to the debate about how many of the women died as a result of this research programme, one would be one too many, but statistical and scientific research has shown that there were more than that. Those of us who were part of the experiments, and those who lost dear ones, are outraged that a lay person now tells us that all we went through was for our own good. There is no sensitivity shown to these people.

Women suffered from the research into the study of the natural history of carcinoma in situ of the cervix, and of the vulva and vagina, but there was also the trial known as the R-series. This was another of Green's trials and was approved by the Hospital Medical Committee in 1972. It was a randomised trial to gauge which was more effective, caesium treatment followed by surgery, or caesium treatment followed by radiotherapy. The patient was examined under anaesthetic in the theatre. If suitable for surgery the randomness was decided by the toss of a coin while the patient was still anaesthetised. Later the patient would be told that it had been decided that 'this' particular treatment was best for her. I know a woman who has been suffering since 1975/76 as her organs slowly deteriorate; a result of the radiotherapy she received when included in this trial after it had been ascertained that she was suitable for surgery. She has had over thirty years of misery.

The few matters I have raised relate largely to my own involvement in the National Women's experiments. Any thinking person must wonder how many other errors there are.

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THE NEW ZEALAND MEDICAL JOURNAL

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A response to Dr Paul Patten

Paul Patten has jumped to the wrong conclusion in his 30 July 2010 letter published in the *NZMJ*. My reference to 'the particular relationship ... with gynaecologists whose work she was researching' was not a reference to Professor Mantell and Dr Baird, as a close reading will show and as I have already acknowledged to Dr Baird.

Professor Skegg wrote his own chapter in *The Cartwright Papers*. So did I. To condemn each of these chapters because we work in the same university is an odd basis for response. I suggest that it is better to read the chapters and make up one's own mind.

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Comment on the book review of *The Cartwright Papers* in the NZMJ

I was interested in the 30 July 2010 issue of the NZMJ, both as a medical historian for the past three decades and as Professor Bryder's partner for the past 20 years.

I have no problem in declaring my personal interest in this matter, but I think it was unfortunate, to say the least, that the *Journal* commissioned Professor Jenny Connor to review *The Cartwright Papers*. Professor Connor is a departmental colleague of Professor Charlotte Paul and they co-taught the Otago PUBH 721 course in Advanced Epidemiology in 2010. Given Professor's Paul central role in the repeated attacks on Professor Bryder and her contributions to *The Cartwright Papers*, it would have been preferable to seek a reviewer from another sphere.

Professor Connor clearly allies herself with Professor Paul and her co-authors, claiming, with regard to Professor's Bryder's book, that 'The mistakes, misunderstandings and mischief in the scientific material and the failure of process in her research are laid bare.' However she provides not a shred of evidence to support this allegation, stating only that 'The second set of essays comprises three responses to Linda Bryder's book by Professor Barbara Brookes, a medical historian at the University of Otago, Charlotte Paul, and Sandra Coney.' Readers are entitled to expect a more balanced appraisal.

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A retrospective study: response to Dr McCredie

Dr McCredie's response has not succeeded in allaying my concerns about her and her colleagues' retrospective study of the so-called 'unethical' practice at National Women's Hospital.¹ As a guideline to accepted practice at the time I would expect them to cite a contemporary authoritative medical textbook. Instead they use as their 'textbook' reference a popular general history of obstetrics and gynaecology from ancient to modern times.

When this book was reviewed in the *Bulletin of the History of Medicine* in 1997 the reviewer, wary of the attempt at such a broad sweep, described the book as 'shallow' and warned readers of the need to contextualize the evidence offered up by the authors. I do not accept this as an authoritative source.

McCredie tells us that in her view 'good intentions' on the part of Green 'are not enough'. This suggests that she now accepts that Green approached his patients with good intentions, while still contending that her own retrospective study (conducted more than twenty years after Green retired) showed that his management had harmed some women. The same could be said of many medical treatments of the past, where it was later found that they caused harm despite good intentions at the time. Medical history is full of such examples.

McCredie's citation of the informed consent section of the 1964 Declaration of Helsinki omits a crucial qualifier to the relevant quotation. Her version reads "... the doctor should obtain the patient's freely given consent after the patient has been given a full explanation" but the original states: "*If at all possible, consistent with patient psychology, the doctor should obtain the patient's freely given consent after the patient has been given a full explanation.*" At that time it was regarded as ethical for doctors to exercise their own judgment as to how much information to pass on to patients. See pages 67–71 of my book for further discussion.²

In her final sentence McCredie cites 'unpublished data' as her source to explain that cone biopsies were not carried out with curative intent but for diagnostic purposes. Such a vague reference alone is not worthy of serious academic debate—it is not enough to tell readers that we must accept the authors' findings on trust.

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A response to Ms Sandercock and Dr Burls regarding the methods used in the analysis for our first paper ‘Natural history of cervical neoplasia and risk of invasive cancer in women diagnosed with cervical intraepithelial neoplasia 3’

We published two papers reporting our findings from a review of the medical records of women diagnosed with cervical intraepithelial neoplasia 3 (CIN3) at National Women’s Hospital in 1955–76.^{1,2} The papers’ aims were quite different; so too were the methods, in particular the criteria identifying ‘treatment groups’ for the analyses, as explained below.

Sandercock and Burls wrote:³ ‘It is difficult to follow exactly what this paper [referring to *Lancet Oncology* 2008,¹ whose title is given above] was trying to prove, but as a means to demonstrate that conservative treatment led to worse outcomes, the methods are wholly inadequate.’ However, the aim of this paper (stated in the Summary and Introduction) was descriptive, namely to estimate the risk of cancer of the cervix or vaginal vault in two quite different circumstances: (i) when CIN3 had not been eradicated (‘inadequate treatment’); and (ii) when CIN3 had been eradicated on every occasion its presence had been indicated by positive cytology (‘adequate treatment’).

We drew attention to the contrast between these estimates which, not surprisingly, were very different but we made no formal comparison. Nor did we use the term ‘conservative treatment’; it is ambiguous, particularly in this context.

For the first aim of the *Lancet Oncology* paper, it was necessary to define a group of women in whom CIN3 persisted after the initial treatment, irrespective of the type of procedure. In practice, early follow-up cytology is the only way to identify these women. In this ‘inadequate treatment’ group, women were censored in the analysis whenever they had been treated ‘adequately’. Although post-procedure cytology could be considered an outcome for clinical purposes, it was not an outcome for this analysis—invasive cancer was the outcome.

Sandercock and Burls³ questioned our inclusion, in the ‘inadequate treatment’ group, of 4 women who developed cancer within 2 years of CIN3 diagnosis but who had no informative follow-up cytology, on the grounds that an outcome (cancer) had been used to categorise their treatment as ‘inadequate’. Before making the decision to include these women, we had considered what was likely to have been the status of their CIN3.

The three possibilities were: (i) that CIN3 had been completely removed by the initial treatment, followed by the appearance of *de novo* CIN3 which had progressed to invasive cancer in less than two years; (ii) that CIN3 had been incompletely removed, in which case our inclusion of these women was correct; or (iii) that invasive cancer was present at the time of CIN3 diagnosis but was not detected because of the limited nature of the biopsy. Given the short timeframe before the appearance of cancer, the

second or third possibilities were more likely. Whatever the circumstances, each of these women would have had CIN3 for part, if not all, of the less than 2 year period between their initial treatment and diagnosis of invasive cancer. Since our objective was to estimate the risk of progression from CIN3 to cancer, we were more likely to get an estimate closer to the true risk by including, rather than omitting, these 4 women.

It should be noted that the third possibility represents a risk inherent in the design of Dr Green's clinical study and for which the undertaking that 'if at any stage concern was felt for the safety of the patient, a cone biopsy would be performed',⁴ proved to be no safeguard.

The aim of our second paper in the *ANZJOG*² was to describe the 'medical experience' of women diagnosed at National Women's Hospital with CIN3, in particular during 1965–74 which were the main years of Dr Green's clinical study. For this analysis, women were grouped according to the type of initial treatment (defined as the most extensive surgical procedure in the 6 months after CIN3 diagnosis), irrespective of post-treatment cytology or subsequent treatment. It was here, in which the initial surgical management alone determined the groups for analysis, that we showed a clear difference in the risk of cancer attributable to withholding or delaying treatment with curative intent.

In this respect, I do not accept Dr Graeme Overton's interpretation⁵ of the numbers in McIndoe et al.⁶ Dr Overton used 'principal initial treatment' and 'initial treatment' for what McIndoe et al designated 'definitive management' or 'management' and which included 'later cone biopsy' and 'later total hysterectomy' (Tables 1 and 4).

McIndoe et al use the term 'initial treatment' only once (p 455, col 2, para 1). In this paragraph, of the 14 women in group 2 (i.e. those with continuing positive cytology) whose 'initial treatment' was said to be cone biopsy, 4 only had the cone biopsy 'later' (between 2 and 8 years after the original diagnosis of CIN3; Figures 2 and 3). They also stated (p 455, col 1, para 2) that all but 4 of the 131 group 2 women had a 'further biopsy' (range 1-19, median 6 years after the initial biopsy) to establish the final diagnosis. Some, but not all, of these 'further biopsies' were the same as the 'later' treatments in Tables 1 and 4.

No information was given in the tables about the duration of the delay between the first and later procedures. However, unpublished data from our analyses show that, for half of the women who were diagnosed with CIN3 in 1965–74, whose initial treatment was punch or wedge biopsy and who had positive cytology in the following 6–24 months, the first more extensive procedure (ring or cone biopsy, or hysterectomy), was delayed for more than 5 years. These subsequent procedures were probably equivalent to the 'later' treatments of McIndoe et al. Thus we have evidence, some indirect, that many 'later' treatments in Tables 1 and 4 of McIndoe et al were delayed several years after diagnosis of CIN3.

In Table 4 of McIndoe et al,⁶ a higher proportion of women in group 2 received cone biopsy or hysterectomy only as 'later' treatment or not at all (51%) than was the case for women in group 1, who did not have continuing positive cytology (22%), a finding in accord with our *ANZJOG* paper.²

Dr Green took a risk by delaying eradication of precancerous disease—the women in his clinical study paid a price.

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Intravenous antibiotics

In the NZMJ Digest for August 2010, I have read Professor Mike Ardagh's article: *How to achieve New Zealand's shorter stays in emergency departments health target*. [eNZMJ 11 June 2010;123(1316): <http://www.nzma.org.nz/journal/123-1316/4152/content.pdf>]

The wording of this title is clumsy, but if you read on you will see what he means. How do you keep people away from Emergency Departments (EDs), and how do you manage them better if they come regardless? He remarks that "ED overcrowding is causing death and other harms in this country."

Ardagh takes the case of a man with pneumonia who needs IV antibiotics. He reveals a series of delays in the handling of the case within the hospital that are inimical to good patient care, and that take up too much time and too many resources. At the end of a paragraph that consists of questions for which Professor Ardagh seeks answers, he asks why intravenous antibiotics could not be delivered in the community.

If he really wants to know, I can help him with that one by describing the management of such a case as it applies here in Wellington, where private medical care cannot compete with the superior organisation of IV therapy to be obtained in the Short Stay Ward at Wellington Public Hospital.

I have for many years had a damaged limb that is very susceptible to attacks of streptococcal cellulitis. I need penicillin at those times, and I need it fast. Sometimes I can abort an attack with oral floxapen; other times I need it to be given intravenously. Some years ago, when I developed an attack, I got one intravenous shot from a GP plus the \$100 bill for the house call. That helped get me right. Recently, when I fell ill again, my GP directed me to the ED at Wellington Hospital. It was a long wait to be seen, but once I had got through the swing-doors, it was all go. A nurse got a needle into a vein, and the IV floxapen was begun.

I got a total of four spaced injections, each delivered right on time. At GP house-call prices, excluding the concealed subsidies paid to GPs, and adjusting for inflation, I got \$500 worth of IV penicillin therapy, two meals, and a bed for the night. I was back home in 24 hours, pulling a chair up to the table, and preparing a thank-you note to the staff of the Short Stay Ward.

In a lot of medical debate, the accent is on what we ought to be paying doctors, with no discussion at all on the ability of the patients or the taxpayers to pay them. Nobody who gets sick needs to be told where they can get it all for free, and, if I get another bad attack of streptococcal cellulitis, I'll know where to go. My GP is in complete agreement.

Roger M Ridley-Smith
Retired GP
Wellington



Long-term benefit of increasing the prominence of a quitline number on cigarette packaging: 3 years of Quitline call data

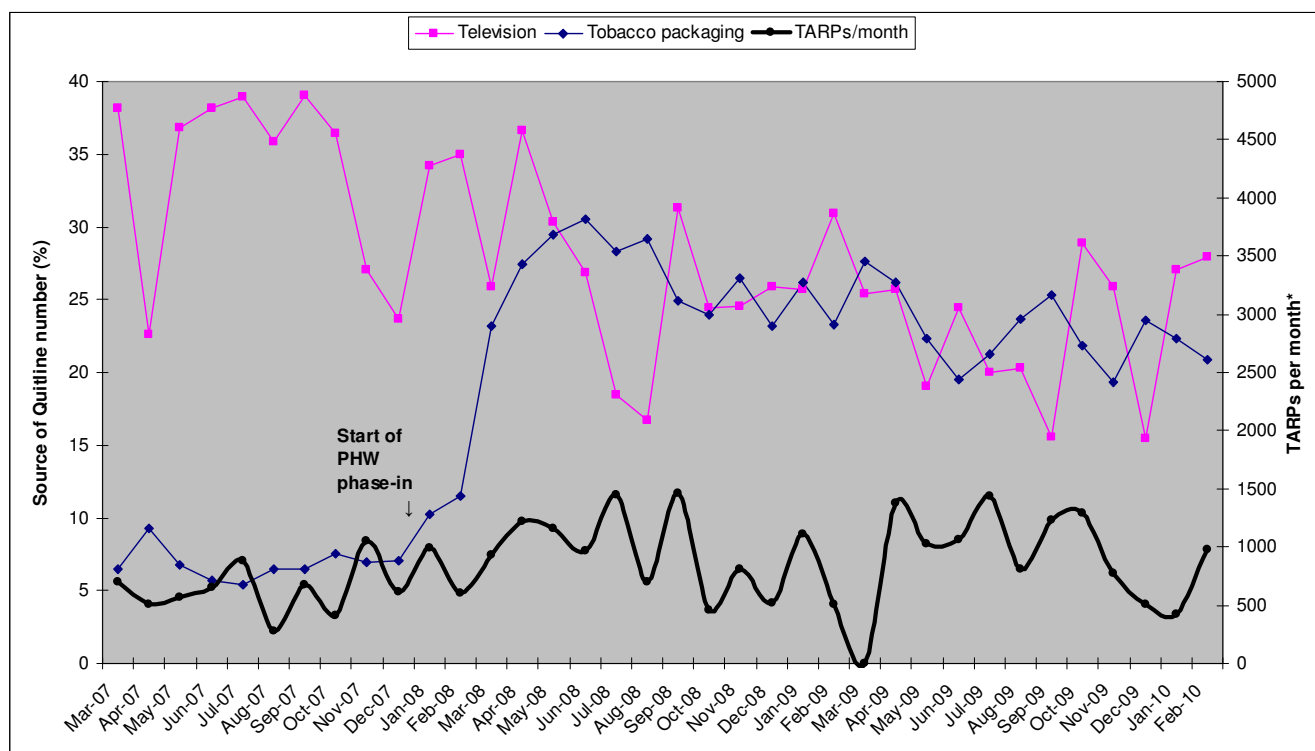
In 2008 the law required tobacco packaging in New Zealand to include pictorial health warnings (PHWs) and the national Quitline number.¹ Previously, text-only health warnings provided a telephone number, but did not explicitly link this to the “Quitline”. Research indicated that New Zealand smokers became more aware of the Quitline number on packs since PHWs were introduced,^{2,3} and there was an immediate increase in the proportion of new callers who registered with the Quitline following the introduction of PHWs.⁴ We investigated whether Quitline callers’ use of packaging to source the Quitline number continued beyond the initial introduction of PHWs.

Methods—The national free-phone Quitline service in New Zealand routinely collects data on where new callers sourced the Quitline number (i.e., a standardised question asked of all new callers). The Quitline service provided us with data on the proportion of new callers who reported obtaining the Quitline number from cigarettes packaging before and after the introduction of PHWs (i.e. for the three-year period March 2007 to February 2010). These data were compared to the proportion of callers who cited television advertising as the source of this number (which was the major source at the start of the study period). We reviewed monthly “target audience rating points” (TARPs) data on the reach and frequency of television advertising for smoking cessation advertisements (most of them showed the Quitline number).

Results and discussion—During the 12-month pre-PHWs period (March 2007 to February 2008), 7.5% and 34.9% of new callers (out of n=19,558 total callers), cited tobacco packaging and television advertising respectively as their source for the Quitline number (Figure 1). However, in the first full year of the new PHWs (March 2008 to February 2009), the proportions reporting tobacco packaging as the source increased to 26.4% and television advertising declined to 27.1% (out of n=20,152 total callers). The same pattern was still evident in the subsequent 12-month period at 22.9% and 23.3% respectively (n=18,309 for the period March 2009 to February 2010).

The proportions of Māori and Pacific callers citing tobacco packaging were similar to that for the overall caller population (i.e., for the last of the three time periods: 21.3% for Māori, 25.9% for Pacific and 23.2% for European/Other callers).

Figure 1. Trends in the two major sources of the Quitline number for smokers calling the New Zealand Quitline the first time (March 2007 to February 2010) showing the introduction point for new pictorial health warnings (i.e., with the Quitline number more clearly shown)



* Target audience rating points (TARPs) are a measure of the audience exposure to televised smoking cessation advertisements (most of them showed the Quitline number).

These findings suggest that promoting the Quitline number more clearly on tobacco packaging increases its long-term salience for smokers of different ethnic groups in New Zealand. In terms of stimulating quitline calls, these New Zealand data are consistent with the experience in Australia,⁵ Brazil,⁶ and Singapore,⁷ where such calls also increased after PHWs featured a quitline number. Similar experiences have also been reported when new text-warnings featured a quitline number in the Netherlands⁸ and the United Kingdom.⁹

Interestingly, the results observed in New Zealand occurred despite the relatively small size of both the Quitline numbers on the packaging i.e. the one in the text and the one superimposed on the picture (see photographs of the warnings¹⁰ and a comparison with the larger quitline number on Australian packs²).

Furthermore, the current PHWs arguably suffer from visual clutter and other suboptimal design features. Improving the warning design, including increasing the size of the front-of-pack PHW and placing the Quitline number on the front of tobacco packages, could further facilitate smokers' use of this zero-cost means of promoting cessation. Such steps could be taken when New Zealand next upgrades its

PHWs so that they better match state-of-the-art developments in terms of size, impact and design simplicity (e.g. as per recent PHWs from Brazil¹¹).

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Competing interests: Although we do not consider it a competing interest, for the sake of full transparency we note that one of us (JL), previously worked as a researcher for the organisation running the Quitline (The Quit Group). However, this is a government-funded non-profit organisation.

Acknowledgements: We thank The Quit Group (who run the Quitline on contract to the Ministry of Health) for the data. Funding support was from the Health Research Council of New Zealand (grant 06/453 for the ITC Project).

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Is New Zealand lagging behind other OECD countries in measures to reduce alcohol-related harm?

The need to improve measures to reduce alcohol consumption and hazardous drinking in New Zealand has become particularly topical in New Zealand following the release of a report by the Law Commission¹ and Government's response to this report, announced on 23 August 2010. This concern is appropriate given the high burden of harm to health (and particularly for Māori health)² in New Zealand. Indeed, alcohol is second only to tobacco as a cause of lost disability-adjusted-life-years (DALYs) in high-income countries according to recent global burden of disease work by the World Health Organization (WHO).³

The public and taxpayers should particularly welcome measures which aim to reduce alcohol-related harm given that some of the interventions may be cost saving to government (e.g. alcohol taxation and advertising restrictions)^{4,5} or at least be relatively cost-effective.⁶ The measures that may be implemented include increasing the age of alcohol purchase at off licences (including supermarkets) to 20 years, banning the sale of premixed drinks with high alcohol content that appeal to youth, and giving more power to local communities to influence the location, density and opening hours of alcohol outlets. However, the Government has missed opportunities to reduce harm from alcohol by delaying or ruling out the introduction of evidence-based measures such as lowering the legal blood alcohol limit for all drivers, introducing restrictions on alcohol advertising, promotion and sponsorship and increasing alcohol taxation.

Methods—To put the current New Zealand discussions into a wider context, we examined how current policies compare with other OECD countries. Data were obtained from the WHO Global Information System on Alcohol and Health (GISAH) (see <http://apps.who.int/globalatlas/default.asp>) and the World Health Organization Global Status Report on Alcohol Policy.⁷ For comparison purposes we used data only from OECD countries with fairly complete data on key indicators.

Results and discussion—Table 1 shows alcohol policies in 19 OECD countries for which data are available. Besides New Zealand, only three other countries in this table have a high blood alcohol limit of 80mg for drivers, although in the UK a report by NICE recently recommended lowering the limit to 50mg.⁸ In Canada, all provinces except Montreal had a limit of 50mg up until this year (2010). As might be expected, opposition to the change was intense, with the bar industry in Quebec being quoted as concerned at dropping beer sales and bar closures.⁹ The Nordic countries (Finland, Norway, Sweden, Iceland) have historically had stronger alcohol policies, although their inclusion into the European Union has meant freer access to alcohol and higher alcohol consumption,¹⁰ although deaths from liver cirrhosis (except in Finland) remain much lower than the rest of Europe.¹¹

Table 2 shows restrictions on advertising, promotion and sponsorship of alcohol in 19 OECD countries, many of which have introduced voluntary and/or statutory

regulation of advertising. Here we rank New Zealand as having the weakest restrictions—except for Belgium.

Table 1. Age limits for serving alcohol, blood alcohol level driving limits and off license restrictions in 19 OECD countries (2008 data)

OECD country	Blood alcohol level limit (in mgs†) for all drivers	Age limit for on premise alcohol purchase*	Age limit for off licence alcohol purchase*	Off licence restrictions on alcohol sales‡ by outlet density
Austria	50	16	16	No
Australia	50	18	18	No
Belgium	50	16 (18)	0 (18)	No
Canada	80	18	18	No
Denmark	50	16	16	No
Finland	50	18	18 (20)	Yes (s, w)
France	50	16 (18)	16	Yes (w, b)
Germany	50	16 (18)	16	No
Iceland	50	20	20	Yes (s, w, b)
Ireland	80	18	18	No
Italy	50	16	0	No
Netherlands	50	16 (18)	16 (18)	No
New Zealand	80	18	18	No
Norway	50	18 (20)	18 (20)	Yes (s, w, b)
Portugal	50	16	16	No
Spain	50	16	16	No
Sweden	20	18	18 (20 + wine)	No
Switzerland	50	16 (18)	16 (18)	No
United Kingdom	80	16 (18)	18	-

Data from World Health Organization Global Information System on Alcohol and Health (GISAH) (“-” means data not given)

*Age for service of spirits given in brackets if different to that for beer and wine; †Blood alcohol is the amount of alcohol present in a 100mL sample of blood, therefore 50mg is 0.05g of alcohol in 100mL (also 0.05% or 50mg/dL); ‡ Sales of beer (b) wine (w) and/or spirits (s);

A causal link between alcohol advertising and consumption is hotly contested but advertising does influence the drinking patterns and attitudes of young people¹² and advertising restrictions are widely considered to be one strand in a range of measures that can reduce alcohol-related harms.¹³

Table 3 shows excise taxes for beer, wine and spirits in 2004 (on countries for which data are available). New Zealand has below average taxation rates compared to many other OECD countries, particularly for its preferred national beverage, beer.

This analysis is very brief and many additional details would improve the quality of such international comparisons. Nevertheless, the results indicate that New Zealand is lagging behind the OECD laws on most of a range of evidence-based measures to reduce the harm caused by alcohol consumption.

Table 2. Restrictions on advertising, promotion and sponsorship in 19 OECD countries (2008) and ordered by regulatory intensity (authors' judgement)

OECD country	Restrictions on beer billboard ads	Restrictions on beer ads at point of sale	Restrictions on beer ads in print media	Restrictions on beer product placement on national TV	Restrictions on national TV beer ads	Restrictions on bar promotions (free alcohol)*	Restrictions on sponsorship of sports
Sweden	Total ban	Partial statutory restriction	Total ban				
Norway	Total ban			No restrictions	Total ban		
Iceland	Total ban					Partial statutory restriction (w); no restrictions (other)	Partial statutory restriction
France	No restrictions	Partial statutory restriction		Total ban		Total ban (w); no restriction (other)	Total ban
Switzerland	Partial statutory restriction	No restriction	Partial statutory restriction	Total ban		Partial statutory restriction; total ban (bc s; s)	Partial statutory restriction (b,w); total ban (s)
United Kingdom	Partial statutory restriction			Total ban	-	Partial statutory restriction	Voluntary/self-regulated
Finland	Partial statutory restriction					Partial statutory restriction (bc b; w); total ban (other)	Partial statutory restriction (b,w); total ban (s)
Italy	Partial statutory restriction			By voluntary agreement	Partial statutory restriction		
Canada	Partial statutory restriction			No restrictions	Partial statutory restriction	No restrictions	
Australia	Voluntary/self-regulated	No restrictions	Voluntary/self-regulated	No restrictions	Total ban	Partial statutory restriction	No restrictions
Spain	Partial statutory restriction		Voluntary/self-regulated	No restrictions	Partial statutory restriction	No restrictions	
Denmark	Voluntary/self-regulated			Total ban	Partial statutory restriction	No restrictions	

OECD country	Restrictions on beer billboard ads	Restrictions on beer ads at point of sale	Restrictions on beer ads in print media	Restrictions on beer product placement on national TV	Restrictions on national TV beer ads	Restrictions on bar promotions (free alcohol)*	Restrictions on sponsorship of sports
Germany	Voluntary/self-regulated			Total ban	Partial statutory restriction	No restrictions	
Portugal	No restrictions			Partial statutory restriction		Partial statutory restriction; no restrictions (bc)	Partial statutory restriction
Ireland	Voluntary/self-regulated			Partial statutory restriction		Voluntary/self-regulated (w); no restrictions (bc); partial statutory restriction (s,b)	Voluntary/self-regulated
Austria	No restrictions			Partial statutory restriction	No restrictions		
Netherlands	Voluntary/self-regulated			No restrictions	Voluntary/self-regulated		
New Zealand	No restrictions					Partial statutory restriction; no restrictions (bc)	Voluntary/self-regulated
Belgium	No restrictions						

Data from World Health Organization Global Information System on Alcohol and Health (GISAH) (“-” means data not given).

*Free alcohol includes beer, spirits, wine and below-cost beer, spirits and wine; bc = below cost; b=beer, w=wine, s=spirits.

Table 3. Taxes on beer, wine and spirits (2008), ordered from highest overall average tax to lowest

OECD country	Tax as a percentage of retail price		
	Beer	Wine	Spirits
Finland	47.7	37.3	59.9
Iceland	40.1	35.4	52.7
Norway	14.5	42.1	71
Belgium	23.9	33	53.5
New Zealand	59.4	12.8	33.8
Sweden	11.7	34.6	50.1
Ireland	21.5	25.7	44
Denmark	31.9	15.4	42
Netherlands	25	16.8	45.4
Switzerland	46	0	38.7
United Kingdom	7.7	42.2	11.9
Australia	38.1	0	15.7
Hungary	21.6	0	28.4
France	3.8	1.2	22.5
Portugal	0.4	0	24.9
Austria	13.9	0	10
Canada	3.2	2.1	12.7

Data from World Health Organization Global Information System on Alcohol and Health (GISAH).

A recent report to the European Commission on evidence-based policies that would be effective and cost-effective in reducing social, economic and health harms from alcohol included:

- Lowering blood alcohol limits for driving,
- Increasing alcohol taxes,
- Reducing the volume of alcohol advertising in all media (acknowledging that self-regulation was not effective),
- Restrictions to alcohol sales (acknowledging that these were only effective if adequately enforced), and
- Encouraging brief advice interventions in primary care.¹³

Some steps towards achieving these policies have been made by the recent announcement of the New Zealand Government to review the liquor laws, but much more can be done to better protect public health from alcohol-related harm.

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Randal Forbes Elliott

KBE GCStJ (12 Oct 1922 – 20 Jul 2010)

It could be said of Sir Randal Elliott that he was born into distinction: his father, James Sands Elliott (later Sir James) was the first house surgeon appointed to Wellington Hospital and, when he came to enter practice as a general-practitioner surgeon, he secured a plot of land adjacent to his own father's Presbyterian church in Kent Terrace, where he had his architect friend Gray Young design him a house with consulting rooms attached.



In this house, in which Randal grew up, prominent figures from James Elliott's wide circle contrasted with the residents of Te Aro flat for whose out-of-hours visits there was a speaking tube at the front door. It provided a stimulating environment. The house has also, in recent years, become the New Zealand headquarters of the Royal Australasian College of Surgeons. Unlike his two brothers, half a generation older, who attended Wellington College (their father's old school) Randal was educated at Wanganui Collegiate School, then at the Otago University Medical School, graduating at the end of 1946. In later life he would be one of the most prominent of Wanganui Old Boys.

He travelled to Britain after his house jobs in Wellington, and promptly acquired the Diploma in Ophthalmology; he held registrar posts at Moorfields, University College Hospital and the London and, when he returned to New Zealand in 1953, he brought with him the English FRCS in Ophthalmology, complementing this in the same year with the Australasian Fellowship.

During this period, too, he had married Pauline Young; they had six daughters and one son and, as the children grew up, Pauline was able to return to nursing duties as one of two unusually qualified staff nurses (Lady Beattie and Lady Elliott) who added a certain cachet to the orthopaedic ward of Wellington Hospital.

Randal was appointed to the visiting staff of Wellington Hospital in 1954; he would become head of his department and of the combined hospital staff, and a prominent figure in the establishment of the clinical school in Wellington. He was president of the New Zealand Ophthalmological Society in 1972, and served his term as an examiner in ophthalmology for the College.

He was made OBE in 1975 and knighted as KBE 2 years later. At this stage of his career he was widely touted in the media as a prospective governor-general.

He was a leading figure in the Order of St John, serving as Chancellor 1980–86 and emulating his father in reaching the highest rank as Bailiff Grand Cross in 1987. Two years later he was senior surgeon and warden of the Ophthalmic Hospital of the Order in Jerusalem, where he applied his administrative skills to a much-needed reform of the institution.

Like his father (who edited the *New Zealand Medical Journal* for many years) he was prominent in the affairs of the New Zealand Medical Association under its various titles, culminating in his appointment as chairman of Council 1971–73, when he saw off an attempt to set up a rival organisation, and President in 1977.

He became a member of the Wellington Club in 1955, was president 1986–90 and subsequently a trustee.

He was active in good works, leading surgical teams to the Pacific and South-East Asia in his role as an RNZAF medical officer, and reaching the rank of Group Captain.

His experience of eye injuries resulting from road accidents made him an effective lobbyist for the use of safety glass in windscreens, a pioneer in the use of seat belts and an authoritative figure in the field of road safety.

Tall and spare, he enjoyed the outdoors (he listed his recreations as yachting, ski-mountaineering, tramping, kayaking and fishing) and it was tragic that the onset of Parkinsonism deprived him of these pleasures, even as his increasing deafness limited his opportunities for the conversational exchanges in which he excelled. Pauline's death a year ago came as a severe blow to him.

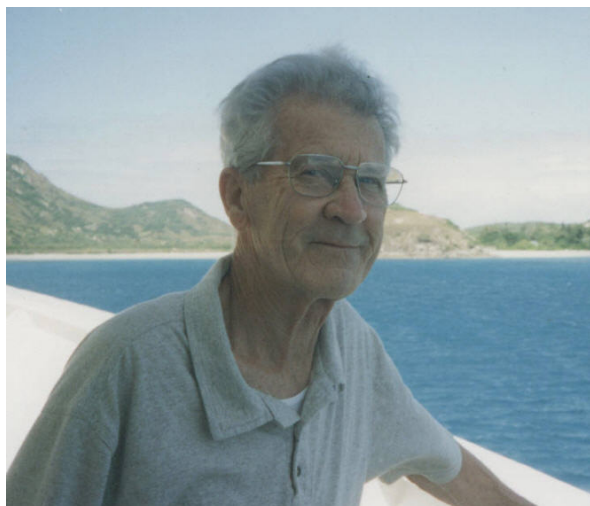
Wyn Beasley (Surgeon/Writer, Wellington) wrote this obituary.



Desmond Denis O'Sullivan

MB BCH BAO MD (NUI) DCH (London)

Desmond Denis O'Sullivan died peacefully at home on 27 July 2010.



Born in Dublin Ireland in 1918, he was the fourth child in a family of five. One son died in infancy and the three remaining sons all became doctors.

As a child Des lived for a time in Phoenix Park Dublin, a 2000-acre piece of real estate in the city, which houses the President of the Republic, some embassies, police houses and caters for practically every sport. His father was in charge of training recruits for the mounted police. It was an ideal place for running the horses each morning.

Des was educated at the Christian Brothers College in Dublin and did tertiary studies at the National University of Ireland, Dublin, completing his MB BCH BAO in 1942.

After finishing his studies he went to England where he worked at Crumpsall Hospital before doing locums at Blakely Manchester and Keswick in the Lake District. The latter practice belonged to a Dr Wakefield who had gone with the British Expedition to Mt Everest—his second attempt. In the setting of the Peak District Des quickly became a devotee of climbing. It was the aspiration of all climbers to climb 12 peaks in a day.

Another locum in Sussex brought his visit to England to a close. At this point in time Des returned to NUI to do a Masters Degree which he received in 1947.

Shortly after, his brother Jack saw a job vacancy for a doctor to join the NZ J-Force in Japan. Des was a successful applicant and after being kitted out in London, Captain O'Sullivan flew in a Sunderland flying boat to Japan. This flight took 2 weeks. Instead of returning to London at the end of his term, he persuaded the authorities that the New Zealand troops might need a doctor on their voyage home. So he accompanied the soldiers on their return journey.

Des joined J-Force doctors at Wanganui Hospital before doing locums in the South Island. In the North Island he took up locums at Cambridge, Wellsford and Brown's Bay, finally buying the Brown's Bay practice on the North Shore.

Early in 1955 Des returned to England, this time to do a Postgraduate Degree in Child Health (London). On his return to New Zealand he fully intended to sell up and return to Ireland, but it was then he met a teacher, Anne Mooney, who became his wife in 1957.

Over the years his sporting interests were rugby, tennis, golf, sailing, snow-skiing, water-skiing, and walking. Groups which gave him great pleasure were Rotary (a Paul Harris Fellow) U3A and ESOL. He was passionate about history, literature and travel to distant lands.

Des lived a long life, in fact he is somewhat of a modern miracle when you consider he suffered his first heart attack in 1979. A testament to both the standard of medical care he received and in no small part to his own medical expertise.

He is survived by wife Anne, daughter Maura, sons Donal, Joseph and Gerard.

Anne O'Sullivan wrote this obituary (since Des outlived most of his contemporaries this was compiled from his memoirs).



James (Jim) Ainslie Begg

The Begg clan of Southland-Otago has produced many doctors. Jim Begg was a prominent and respected psychiatrist with a keen sense of his Scottish heritage. He was medical superintendent of Christchurch's Sunnyside Hospital during the transformation of mental illness treatments worldwide. He died recently, aged 89.



While always seeking, testing and implementing new ideas in psychiatry, Begg clung fondly to his farming background at Wyndham, near Invercargill, and Caledonian culture.

He was born in Dunedin and attended Columba College, when boys were still admitted at what is now a girls' school. His secondary schooling was at John McGlashan College and he studied medicine at Otago University, graduating in 1946.

As a fifth-year student, he was posted to Greymouth as chief medical officer. Son Evan wonders if this was penance for choosing medical studies instead of going to World War II.

He married Nonie Leggatt, of Nelson, in 1947. They had six children but daughter Shona died in 2001.

Begg started work as a hospital doctor and a GP in Wellington, Hawera and Epsom, in the UK. He studied psychiatry in Edinburgh for 18 months and began work in that speciality at Nelson's Ngawhatu Hospital.

He became principal psychiatrist at Seacliff-Cherry Farm, near Dunedin, in 1960. This position involved consulting work at Dunedin, Balclutha and Kew (Invercargill) hospitals and lecturing at Otago University. His final move was to Christchurch in 1965, as principal psychiatrist at Sunnyside, where he served for 20 years until his retirement. He became medical superintendent and was visiting consultant to Ashburton and Timaru hospitals.

Begg's career spanned a time of great change in the treatment of psychiatric illness. He was at the forefront of change, developing systems for patients to play a part in their own management. He introduced the "therapeutic community" approach, mixing age groups and sexes in educational, recreational and social activities, including art and music.

He expanded outpatient, group therapy and day-ward services, using increased community and volunteer assistance. His work in forensic psychiatry through the 1970s led to consulting work with the Justice Department, involving him in big police investigations. He led the adolescent unit in the early 1980s and remained involved with it for several years after his retirement at age 65.

Further study in the US in 1973 helped him handle the changes that came with the move to discharge hospital patients and the establishment of community-based care and rehabilitation systems. He was on several committees involved with the changes and was chairman of the Psychogeriatric Advisers Committee.

His working life was one of long hours and little time for leisure. Daughter Alison says her father gave time to patients, who visited him at home. However, he always managed to fit in family time. He took the family on many camping and caravanning trips and passed on his love of the outdoors.

He made up for his busy working years in retirement, as an avid outdoors man, music lover and traveller, brimming with the joy of living and socialising. Daughter Bronwen remembers family camping and boating excursions at Lake Rotoiti, around New Year. Her father continued to go there in later years. His tramping and climbing feats, such as scaling Mt Robert at 87, would have daunted many young people.

Evan says that after Nonie died in 1991, “Dad revved up and lived life even faster”. He launched into tramping and skiing “with great gusto”. He joined many choirs and through that, met his second wife, Jessie. They married in 1998.

Always he played the bagpipes. Begg loved to get back to his Scottish roots, donning his kilt and piping in the haggis. This he did at the 90th reunion of Knox College, his Otago hall of residence. He played at many functions. For many years, and even at 89, he piped in the performers at the annual Doctors’ Concert. He played the organ, too, often doing so at hospital Christmas services.

Begg maintained close affinity with the family farm, Wyndham Station, in Southland. He was passionate to see it remain in family hands.

Evan says his father’s “special characteristics were impeccable honesty and integrity, never saying anything nasty about anyone, and amazing positivity. He always enjoyed a joke. Above all, he was hugely enthusiastic about everything. He was a larger-than-life character.”

James Ainslie Begg, born Dunedin, 21 August 1920; died Christchurch 24 June, 2010. Pre-deceased by wife Nonie and daughter Shona; survived by wife Jessie; sons Evan and Robert; daughters Bronwen, Alison and Hilary; and his grandchildren.

Mike Crean wrote this obituary; it first appeared in The Press newspaper (Christchurch).



John Joseph Horan

Doctor John Horan was called to a high mountain rescue on the morning of his wedding day.



He joined alpine guides and police officers to rescue a climber and recover a body, before getting to the church on time for marriage to Merle Halliburton in 1951.

Mountain rescues were all in a day's work for the self-effacing doctor. Though no outdoors man, he was involved in many climbs in his medical career—winched from helicopters on to glaciers and snow-covered ridges, knowing at any moment the chopper might have to leave and he would need to climb down to safety.

Not all rescues were in the mountains. He was winched on to a Taiwanese fishing boat in Lyttelton Harbour, because of a typhoid outbreak on board.

He made mercy flights to the Chatham Islands for women in difficult labour. He brought a Russian seaman with a severed arm off a ship in the Southern Ocean to Christchurch, where a plastic surgeon successfully re-attached the arm.

Horan was committed to care. He had a gift for making patients feel at ease. He never charged a patient, relying on government subsidies. He said he could not focus on a person's needs if he was considering how much to charge. He never employed a practice nurse, insisting a GP should perform all medical duties. (Merle worked as his receptionist.)

In death, he gave his body to science. He was grateful to have learned on human bodies at Otago University Medical School and wanted to give something back.

He attended St Albans Primary School, Shirley Intermediate and Christchurch Boys' High. His mother encouraged him in music and he gained "letters" in violin while at school. He was a lifetime music lover and played in orchestras and with the New Zealand Army Band.

Horan graduated from medical school in 1948 and worked as a house surgeon in Ashburton, where he met Merle. He took on the sole-charge medical practice in Whataroa, South Westland, to fulfil his government bursary commitment. Before the road south of Fox Glacier was complete, he flew once a month to Haast for day clinics.

After two years in South Westland, Horan returned to medical school as a demonstrator in anatomy. He then moved to Granity, in Buller, for a year. From

1954–71 he was in general practice in Ashburton. For much of that time, he was also visiting physician to Ashburton Hospital.

He learned to fly with the Ashburton Aero Club. Merle says: “He just wanted to do it. Anything he wanted to do, he did. He was quietly determined.”

He joined the air force reserve in 1967. The air force appointed him to provide medical cover to Prince Charles and Princess Anne for their 1970 stay at Mt Peel Station in South Canterbury.

He moved to Burnham military camp as senior medical officer in 1971, then went back to Otago and gained the diploma in public health. He was appointed deputy medical officer of health in Christchurch and medical officer of health in Palmerston North, with oversight for Whanganui.

Merle says he disliked office work and longed for closer contact with people. He became a lecturer in community and preventive medicine at the Christchurch Clinical School and enjoyed working with students.

However, involvement in administration for the Department of Health and North Canterbury Hospital Board irked him. His hankering for another taste of military life drew him back to Burnham in 1977. He became senior medical officer of health for the army and assistant director of military services, with the air force rank of wing commander, while continuing as part-time lecturer and examiner for Christchurch Clinical School.

The next year he was appointed base medical officer at Wigram Air Base, in addition to his duties with the army. He was appointed also honorary physician and surgeon to then Governor-General Sir Keith Holyoake.

Horan was involved in development of air force ambulance flights for Canterbury hospitals and the promotion of life- support apparatus for use on planes. He made many trips in air force Friendship planes and Iroquois helicopters on mercy missions. He supervised the use of neonatal incubators that his team had developed for recharging by aircraft generators.

He was a foundation member of the NZ College of Community Medicine, in 1980.

Retirement from the air force in 1988 brought little rest for Horan. He continued to serve on the aged people’ care and advisory group of Presbyterian Support Services. He was adviser on war pensions in Canterbury and Westland for the Department of Social Welfare.

He and Merle regularly prepared and served meals at the Christchurch City Mission, on a Masonic Lodge roster. He wrote articles and papers for the Medical Association.

Music was his great love and he was an excellent violinist. He enjoyed touring with the NZ Army Band by invitation, and playing with the band and in solo items.

Merle says he was a lovely, humble and caring man.

John Joseph Horan, born Christchurch, 10 December 1922; died Rangiora, 22 April 2010. Survived by wife Merle.

Mike Crean wrote this obituary; it first appeared in *The Press* newspaper (Christchurch).



Cases in Pre-hospital and Retrieval Medicine

Dan Ellis and Matt Hooper. Published by [Churchill Livingstone \(Elsevier Australia\)](#), Dec 2009. ISBN 9780729538848. Contains 200 pages. Price AU\$71.96 (online price at Elsevier Australia)

Cases in Pre-hospital and Retrieval Medicine has been written by two experienced flight doctors who have worked for helicopter retrieval services in the UK, Australia and Israel.



Retrieval medicine is a specialised field and weaves together skills shared with pre-hospital care as well as trauma, intensive care, aviation, wilderness and emergency medicine. There has been a need for a coherent text in this area of medicine.

The format is case studies with discussion. The field of retrieval medicine is not suited to a didactic chapter by chapter text. In practice, missions consist of a large portion of planning, preparation and protocol with the rest being troubleshooting and creative problem solving.

The narrative formats of the cases demonstrate how challenges in the field are met and overcome.

Section A has cases that are focused on general pre-hospital assessment and stabilization. Section B dwells more on problems related to retrieval. It goes into depth on the preparation and stabilization of the patient prior to transfer, and also covers some difficult ethical and administrative issues. The last section has special cases - scenarios such as in-flight medical emergency on commercial flights, EMS planning for mass gathering events and aircraft issues. The appendix is a collection of pictorial guides on practical procedures, checklists and mnemonics. One could argue that more detail could be added in some of the discussions but I believe the authors have struck the correct balance. Appropriate references and further reading are listed for each case.

The audience is doctors working in pre-hospital and retrieval medicine especially those from the disciplines of Intensive Care and Emergency Medicine. Paramedics and flight nurses wishing to upskill will find this an excellent read. The book is beautifully presented and clearly set out. There are excellent colour photos to set-up each scenario. This is not a handbook—it is a study guide. The breadth of material covered is essentially the core curriculum for Retrieval Medicine but is so accessible that it will be useful to a wider audience.

Paul Gee

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THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association

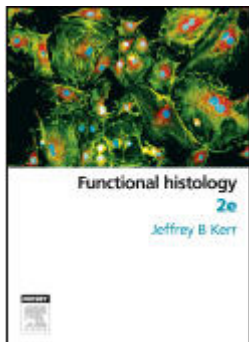


Functional Histology (2nd edition)

Jeffrey B Kerr. Published by [Mosby \(Elsevier Australia\)](#), 2010.

ISBN 9780729538374. Contains 512 pages. Price AU\$112.50 (online price at Elsevier Australia)

This is an informative and beautiful book. It is well organised with an appealing layout of the text and illustrations.



The illustrations are truly outstanding and include many hundreds of photomicrographs, electron micrographs and diagrams as well as some well-chosen gross anatomy photographs. They are beautifully reproduced and clearly labelled, and have excellent captions.

The text is very readable and concise with clear and interesting explanations of complex topics. The author employs helpful analogies to bring to life the complexities of cell and tissue structures and their functions.

The careful juxtaposition of the text with the related illustrations greatly facilitates comprehension and concentration.

In the preface, the author outlines the scope of the book and the target audiences, which seem to include students and practitioners in the health professions, as well as those involved in biomedical research. A quick check of reviews available on the internet shows that it appears to be highly regarded as a histology textbook by undergraduate students in the health sciences. I expect that many medical practitioners would enjoy this book, not only for the stunning photographs, but also because it provides a very accessible review and update of normal structure and function of human cells, tissues and organ systems. It would be an excellent resource for aspects of continuing medical education.

My only criticism, which is a minor one, is that the author has included at the conclusion of his discussion of each organ system a very brief discussion of important and common diseases affecting that system, including pathological changes. I did not think these discussions were of an equivalent high standard to the rest of the book. But they are a minor component and do not significantly detract from the overall excellence of the book.

In summary, I found this book very useful and a pleasure to use. The author is an effective communicator who has utilised an outstanding collection of illustrations to accompany his lucid text, thereby providing an excellent overview of normal structure and function of human cells and organ systems.

Andrew Miller

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Enhancing Patient Care: a practical guide to improving quality and safety in hospitals

Alan Wolff and Sally Taylor. Published by [MJA books](#), 2009. ISBN 9780977578665. Contains 234 pages. Price AU\$49.95

When research found that death, disability, and prolonged hospital stay was often the outcome of medical treatment rather than the disease there was optimism that recognition of the problem would lead to effective control of medical errors and adverse events.



Some of the reasons why this did not occur are familiar to the Wimmera Health Care Group that faithfully pursued the patient safety improvement goals for 19 years.

Their efforts established a model that has now been adopted by many hospitals ranging from tertiary centres in capital cities to rural hospitals.

This book which is sub-titled as practical guide to improving quality and safety in hospitals is written by hands-on physicians, but it has a large reference base that provides explanations of organisational theory, and definitions for every aspect of risk management.

There are very detailed explanations of the components and processes required to build quality and safety into established systems that are resistant to change, but too little attention is given to the need for executive support and how to change a dysfunctional system.

An individual or institution seeking to better understand the complexity of the patient safety challenge will be well served by “Enhancing Patient Care” which should be available in every hospital library, because, as the authors conclude, inertia is probably the biggest obstacle to change, and taking effective action is up to individuals within organisations.

John Morton
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