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This Issue in the Journal

Rates of *Chlamydia trachomatis* testing and chlamydial infection in pregnant women
B Lawton, S Rose, C Bromhead, S Brown, J MacDonald, J Shepherd

Chlamydia trachomatis is the most common bacterial sexually transmitted infection in New Zealand, and (if left untreated) has significant health consequences for pregnant women and the neonate. This study audited a community laboratory database to determine the rate of chlamydia testing and infection in pregnancy. Only 38% of the women had been tested, and high rates of chlamydia infection were detected, particularly among women under 25 years of age. This study highlights the need for testing for chlamydia to be carried out routinely in pregnancy.

Management of unstable angina and non-ST-elevation myocardial infarction: do cardiologists do it better? A comparison of secondary and tertiary centre management in New Zealand
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Coronary heart disease remains the leading single cause of death in New Zealand. Prompt and appropriate investigation and treatment is associated with better outcome. In New Zealand, patients are more likely to be promptly and appropriately investigated and treated after a myocardial infarction (heart attack) in a tertiary care centre (under the care of a cardiologist) than in a secondary care centre (under the care of a generalist physician). Therefore, such patients should be investigated and treated promptly regardless of the hospital site at presentation.

The New Zealand child work-related fatal injury study: 1985–1998
R Lilley, A-M Feyer, J Langley, J Wren

This study investigated the contribution of children to the burden of work-related fatal injury in New Zealand. Children, although not part of our formal workforce, participate in work arrangements such as casual holiday work, or part-time after-school work. In addition, they may be exposed to workplace hazards when they visit, or live on, worksites (eg, farms). This is the first such study in New Zealand investigating the role of work in fatal injuries in children <15 years old.

Oral health status and oral treatment needs of dependent elderly people in Christchurch
G Carter, M Lee, V McKelvey, A Sourial, R Halliwell, M Livingston

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to the gum. More effort is needed to ensure older people do not suffer from avoidable dental problems.

Lack of association between long-term illness and infectious intestinal disease in New Zealand

R Lake, M Baker, C Nicol, N Garrett

A small proportion of patients with infectious intestinal disease develop a variety of secondary long-term illnesses. Between 1988 and 2002, New Zealand experienced a marked increase in the number of notified cases of infectious intestinal disease. This paper reviews the information on hospitalisation for two long-term illnesses (in order to identify any correlation with these notified cases). Little or no correlation was found, which indicates that infectious intestinal diseases are not making a significant contribution to the burden of these long-term illnesses.

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The chlamydia problem in New Zealand

Nicky Perkins

When one reviews the epidemiological data on *Chlamydia trachomatis* infection in New Zealand, it is clear that we are significantly lagging behind other OECD countries in the control of this important sexually transmitted infection. The Institute of Environmental Science and Research (ESR) produces regular surveillance figures on Sexually Transmitted Infections (STI) in New Zealand. The latest annual STI surveillance report from 2003[1] is a good illustration of how far we have to go in New Zealand to gain a measure of control over the rising rate of STIs.

Chlamydia is now officially the most commonly reported STI and, over the past 5 years, the rate has risen 65.5%. The rate of chlamydia in Auckland, Waikato, and the Bay of Plenty (the regions with the most complete laboratory data) is six times higher than that reported in Australia and four times higher than that reported in the UK (excluding Scotland).

As with other STIs, young people under the age of 25, Maori, and Pacific Island people have the highest rate of infection. These figures are a sad indictment, especially as they are an under-representation of the extent of the chlamydia problem in New Zealand. The true prevalence of chlamydia is likely to be higher than that reported by ESR, because laboratory surveillance in this country is incomplete, and the figures reflect only the prevalence in those people who have presented for testing, since we have no screening programme in place nationally.

Chlamydia is a bacterial infection that is well placed to cause significant health problems in the population for several reasons:

1. It has a high rate of asymptomatic infection (approximately 70% in women and up to 50% in men), which means that infection is often not detected unless screening is undertaken.
2. It is relatively infectious (there is approximately a 20% chance of becoming infected per sexual contact with an infected person), and has a period of potential infectivity of months to years, which means that it can be sustained in a population with a reasonably low rate of partner change.
3. Lastly, it causes complications that can result in chronic health issues requiring significant health spending. Complications are more common in women and include preterm labour and perinatal sepsis, PID, tubal scarring and subsequent pelvic pain, infertility and ectopic pregnancy. Other complications include epididymo-orchitis, neonatal conjunctivitis, and early infant pneumonitis and otitis media[2].

Testing for chlamydia has previously been challenging, as it required the use of culture tests, which are invasive, insensitive and expensive. The advent of nucleic acid amplification tests (NAATs) has vastly improved detection of *Chlamydia trachomatis*. These are available in many centres in New Zealand, although a variety of different tests (including non-NAATs) are being used nationwide and there is no

national standard at present for chlamydia diagnostic testing. NAATs are generally very sensitive and specific, and can be used on a wide range of specimens (eg, vaginal swabs and urine) which facilitates non-invasive testing and screening.[3] Treatment of chlamydia is simple, as there have been very few reports of resistant organisms over the years, and standard regimens of azithromycin or doxycycline are highly effective[4].

In this edition of the NZMJ, Beverley Lawton and colleagues present *Rates of Chlamydia trachomatis testing and chlamydial infection in pregnant women*, an article on the prevalence of chlamydia in pregnant women in Wellington. The results indicate that only 37.5% of pregnant women are being tested for chlamydia as part of antenatal screening, and that a significant proportion of those tested are infected. Prevalence overall in the study population was 4.8% and it was particularly high in young women aged under 25 (12.2%), and in Maori (15.2%) and Pacific Island (12.5%) women. These data correlate well with ESR data on the prevalence of chlamydia and other STIs in New Zealand as a whole. An additional concern, raised by the authors, is the amount of neonatal chlamydial conjunctivitis occurring in New Zealand, which reflects untreated maternal infection. In the study population, there were four cases over the last 3 years; and data from the ESR surveillance reports show that in the Auckland, Waikato, and Bay of Plenty regions over the last 3 years there were 193 cases, an average of 64 per annum.

Although the data presented give some indication of chlamydia prevalence in pregnant women, caution must be exercised in extrapolating these figures to the general pregnant population. Only a third of pregnant women in the Wellington population were tested, and it is possible that maternity caregivers may have tested only those they felt to be at risk of infection, thus giving a falsely high impression of overall prevalence. In addition, data is needed for other regions in New Zealand to allow the emergence of a full national picture, as at present national antenatal prevalence data is lacking. However, given the ESR laboratory data for Auckland, Waikato, and the Bay of Plenty, it is possible that the prevalence of chlamydia in pregnancy in these regions is even higher than that found in the Wellington study.

This article highlights the issue of antenatal screening, and indeed screening in general, for chlamydia in New Zealand. Because *Chlamydia trachomatis* infection is largely asymptomatic, to reduce its prevalence, an effective screening programme is necessary. At present, there is no organised screening for chlamydia in any population group. The most efficient and cost-effective method would be to target sections of the population who are at high risk of acquiring infection—eg, young people between 15 and 25, particularly those of Maori or Pacific Island ethnicity, and persons with a recent change in sexual partner.

Screening pregnant women would be a useful place to start since they are generally all attending for healthcare during their pregnancies, they are young, and (by definition) sexually active. Efficiency and cost-effectiveness from such a programme could be maximised by screening pregnant women in the aforementioned target groups, rather than screening the entire pregnant population.

Currently routine antenatal screening for chlamydia is recommended in the USA,[2] but not in Australia, New Zealand,[5] or the UK[6]. However, in the UK, it is intended to begin targeted antenatal screening of women under the age of 25 as part of the national opportunistic screening programme, which is due to begin in 2004. It is

understandable that Australia does not have an antenatal screening programme in place, since it has a much lower prevalence of chlamydia, but it is unacceptable that routine antenatal screening for chlamydia is not being undertaken in New Zealand, where we have high rates of infection.

In modelling studies, screening for chlamydia in women has been shown to be cost effective at prevalences of between 3.1 and 10%.[7] Targeted screening in women has been shown to be effective in reducing the incidence of PID and chlamydia in studies in the USA[2,8]and Sweden[9]. In principle, screening of sexually active women under the age of 25 has been advocated by experts in England, Scotland, and the USA.[10] Therefore, it is clear (internationally) that screening in women is advocated and has been shown to be effective.

In the establishment of a screening programme, however, it would be important to screen men as well as women, since asymptomatic men are a reservoir for infection and tend to present for testing less frequently than women. To prevent New Zealand from falling further behind other OECD countries in its control of chlamydia, a well-defined strategy and fiscal commitment from the government are required. In particular, laboratory surveillance needs to be extended to include full national participation, a screening and treatment programme needs to be initiated, and health promotion programmes must be put in place to enhance education and encourage behaviour change to reduce sexual risk.

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The challenge of improving cardiac care in secondary centres

Paul Bridgman

Outside of the major New Zealand centres, acute inpatient medical care is delivered in secondary hospitals. Usually medical patients are admitted under general physicians with specialty back-up provided from a remote tertiary centre.

The pattern of care delivery varies considerably depending on the geographic and staffing situation for each individual hospital, and its relationship with the tertiary centre. Some aspects of care received by the patient in the secondary centre may be better than that received if they are admitted directly to the tertiary centre although certain aspects may be worse in the secondary centre. Each centre does its best depending on its situation, and one might hope (in the interests of regional equity) that overall outcomes would not be too dissimilar between centres.

However, not surprisingly, this is not always the case. Cardiac care outcomes provide an easy target for audit. Patients may be readily categorised on the basis of presentation, ST segment changes, and subsequent troponin T measurements. Treatment guidelines for these conditions are widely promulgated and well accepted within the cardiology community.

In the study by Conaglen and colleagues, patients admitted to Waikato Hospital with an acute coronary syndrome were 4.6 times more likely to receive diagnostic coronary angiography, and 4.2 times more likely to receive revascularisation, than those admitted to Taranaki Base Hospital.[1] This represents a large magnitude difference in care delivery so it merits close attention as these are interventions proven to improve outcomes in this patient group.[2] It also raises the question as to whether similar differences exist in care in other medical and surgical specialties, that are perhaps less amenable to audit.

Measuring and improving clinical care is a science that we must move forward in. The study by Conaglen and colleagues is a reasonable response to the challenge of measuring quality of cardiovascular care. Attempting to determine the source of a quality deficit is a much more difficult challenge. There are probably multiple reasons for the observed gap in cardiac care delivery. International data suggests that geographical access factors influence care. Admission to a hospital with on-site angiography is more strongly associated with the use of coronary angiography than any patient characteristic.[3] The specialty interest of the attending physician is also likely to be an important influence. Uptake of cardiology care guidelines by general physicians has been shown (in some instances) to be only moderate.[4] Additional factors are likely to also play a role. For example, it is possible that physicians in smaller hospitals may have inherited patterns of care delivery that are less amenable to change.

The science of intervening to improve care quality is still in its infancy. Conaglen and colleagues refer to recently promulgated guidelines, but passive dissemination of

knowledge (such as these guidelines) has been shown to have little impact on practice patterns.[4,5] It appears that quality improvement exercises that actively embed guideline knowledge into the care process itself are more successful.[6]

Hospitals with low angiography rates should be encouraged to develop protocols for the management of acute coronary syndromes, in association with clinical pathways that include a point of decision for angiography. This process needs to be driven by the hospital staff, but could include representative referral centre specialists. The literature shows that multifaceted interventions are more effective than single strategies to improve performance.[7] Many tertiary centre specialists now perform outreach clinics in secondary hospitals. This provides an opportunity to personalise the interhospital referral process.

From my experience with outreach cardiology clinics, I believe that regularly having morning tea with the local general physicians has played an important role in advancing secondary cardiac care across the board. In addition, it has provided a pleasant informal collegial environment in which to discuss the management of patients of interest. Indeed, any strategy aimed at improving referral rates for intervention should look to facilitate such direct contact between the secondary and tertiary physicians.

Ultimately, improvement in the access to care for secondary centre patients requires an open partnership between the secondary centre physicians and their tertiary centre colleagues. Care will never be identical across institutions, but every effort should be made to ensure that care for each individual is optimised.

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Rates of *Chlamydia trachomatis* testing and chlamydial infection in pregnant women

Beverley Lawton, Sally Rose, Collette Bromhead, Selina Brown, Jane MacDonald, and Jill Shepherd

Abstract

Aims To determine the rate of *Chlamydia trachomatis* testing and chlamydial infection in pregnancy (by auditing a community medical laboratory database).

Methods Data for women registered with a maternity care provider between 1999 and 2002 were matched with a community medical laboratory database for patients who met one of three criteria: tested for *C. trachomatis*, or had a first or second antenatal blood screen at that laboratory. The rate of *C. trachomatis* testing and of chlamydial infection was then calculated in this sample.

Results The overall rate of *C. trachomatis* testing for 6614 matched deliveries was 37.5%, with 4.8% of those tests positive for chlamydial infection. The rate of testing differed significantly between age-bands ($p<0.0001$), and by ethnicity ($p<0.0001$). The rate of infection showed a significant effect of age ($p<0.0001$) and ethnicity ($p<0.0001$). Maori and Pacific women, and those under the age of 25 years, had the highest rates—both of testing and of *C. trachomatis* infection.

Conclusions There is a high rate of maternal *C. trachomatis* in under 25-year-olds, and in Maori and Pacific women, together with incomplete testing for the infection in pregnancy. This highlights the need to instigate routine testing for *C. trachomatis* in pregnancy—to reduce the significant, yet preventable, morbidity associated with chlamydia in both the mother and the neonate.

Chlamydia trachomatis (*C. trachomatis*) is the most common bacterial sexually transmitted infection (STI) in New Zealand.[1] *C. trachomatis* has a high rate of asymptomatic infection—approximately 80% of cases in females, and 45% in males, are estimated to be asymptomatic.[2]

The exact prevalence of *C. trachomatis* in New Zealand is unknown, although rates are typically higher in females, in under 25-year-olds, and in Maori and Pacific people.[2,3] In 2000, a survey of laboratory and STI clinic data in the Waikato and Bay of Plenty regions revealed an incidence rate of 4162 per 100,000 for females aged 15–19.[4]

More recently, year 2002 laboratory data from Waikato and Bay of Plenty reported increasing rates of *C. trachomatis*—with up to 6998 cases per 100,000 for 15–19 year old females.[2] The overall rate of *C. trachomatis* was reportedly five times higher than that found in Australia (similarly calculated from laboratory surveillance data).[2] Dunedin laboratory figures have also shown that the incidence of *C. trachomatis* increased by 37% in females and 10% in males between March 2002 and March 2003.[5]

The prevalence of *C. trachomatis* in females who were sexually active and provided a urine sample for a New Zealand school-based study in Christchurch was 2.3%.[6] Despite the apparent increasing prevalence of *C. trachomatis* in New Zealand, it is not a notifiable STI, there is no national data collection, and there are no screening or treatment guidelines.[1]

There are currently no New Zealand prevalence data for *C. trachomatis* in pregnancy. Infection rates reported in pregnant women in the USA and Canada have varied from 5% to 20%.[7] The sequelae of untreated (which includes undetected or asymptomatic) chlamydial infections can be severe—both for the pregnant woman and for the neonate. Prenatal implications of chlamydial infection for the mother and newborn include associations with ectopic pregnancy, spontaneous abortions, preterm labour, amnionitis, premature rupture of membranes, low birth weight, prematurity, still birth, and neonatal deaths.[7–9] Women with chlamydia during pregnancy are also more likely to develop intrapartum fever and or late onset postpartum endometritis after vaginal delivery.

It has been estimated that 20%–40% of infected untreated women will progress to pelvic inflammatory disease (PID)[9]—and these women will subsequently be exposed to complications of infertility, chronic pelvic pain, ectopic pregnancies, and death from ectopic pregnancy. For the newborn of untreated mothers, inclusion conjunctivitis occurs in 11%–44% of cases, and pneumonia occurs in 11–20% of cases.[7] Furthermore, *C. trachomatis* in infancy has also been associated with otitis media, bronchiolitis, pharyngitis, rhinitis and gastroenteritis.[8]

Vertical transmission of *C. trachomatis* to the neonate occurs in approximately 50% of cases.[8,10,11] During 2002, up to 96 babies under the age of 12 months had *C. trachomatis* diagnosed at Auckland and Waikato/Bay of Plenty laboratories, which was an increase of almost 70% when compared to 2001.[2] By eliminating transmission of chlamydial infection from mother to child, it has been shown there are more favourable outcomes for both the mother and the newborn—including significant reductions in premature labour, low birth weight, and increased survival.[9]

Although screening for *C. trachomatis* in pregnancy is considered best practice internationally,[12,13] there are currently no guidelines in New Zealand advocating routine testing in pregnant women. Testing for *C. trachomatis* is desirable—for detecting and subsequently treating the chlamydial infection in pregnant women, and for reducing the associated morbidity, which is significant.

This audit process was carried out in a community medical laboratory to determine the prevalence of *C. trachomatis* testing and chlamydial infection in pregnant women who delivered between 1999 and 2002.

Methods

Ethics approval to carry out this audit was granted by the Wellington Ethics Committee and consultation took place with the Maori Health Directorate (Ministry of Health).

Crude rates. Rates of chlamydial infection were ascertained in all specimens (male and female) tested at Wellington Medical Laboratory in 1999, 2000, 2001, 2002 (September to September), and 2003 (September to June). Rates of chlamydial infection in pregnancy (that may include both completed and terminated pregnancies) were determined by matching all first antenatal bloods with *C. trachomatis* tests in the same laboratory in 1999–2003. The rate of *C. trachomatis* in infants (1-year-old or younger) was determined by retrieving results from all paediatric eye swabs tested during 2001, 2002, and 2003 (before 2001, data were not available for this type of test). This laboratory uses the polymerase chain

reaction (PCR) test (Amplicor CT/NG, Roche Diagnostics) to routinely detect *C. trachomatis* in both urine and swab samples.

Maternity care provider data matched to laboratory tests. To determine the prevalence of *C. trachomatis* testing in ongoing pregnancies, details of completed pregnancies that were registered with a maternity care provider were matched to laboratory data. For the years 1999–2002, name, date of birth, ethnicity and year of delivery were identified for all women with completed pregnancies who were registered with a maternity care provider.

Ethnicity was determined by self-identification using the 2001 census form completed by patients at an antenatal hospital booking. These data were forwarded to Wellington Medical Laboratory and matched with patients who met one of three criteria: had a *C. trachomatis* test; had a first antenatal blood screen, or had a second antenatal blood screen at that laboratory between 1998 and 2002. Data matching at the laboratory went back as far as 1998 to capture pregnancies that began in 1998 and delivered in early 1999.

The laboratory assigned a study number to each patient, and the anonymised data were returned to the research team for analysis. Data included—study number; year of delivery; age at testing (under 25 years, 25 years and older); mean, median, and age-range of the sample; ethnicity; whether tested for *C. trachomatis*; and the outcome of that test (whether positive or negative for *C. trachomatis*).

Results

Crude rates. The (crude) rates of chlamydial infection in all male and female specimens (ie, from antenatal specimens and in paediatric eye swabs tested at the laboratory) are presented in Table 1.

Table 1. Prevalence rates of chlamydial infection in all laboratory samples (male and female) tested between 1999 and 2003

	Year of testing and percent positive for *C. trachomatis*				
	1999 (%)	**2000 (%)**	**2001 (%)**	**2002 (%)**	**2003 (%)**
All specimens	4.9	6	5.5	5.4	6.6
Antenatal specimens[*]	6.2	7.6	5.3	5.6	7.6
Paediatric eye swabs	Not available	Not available	20.0[†]	20.0[‡]	4.25[§]

[*] Includes both completed and terminated pregnancies; [†] n=5; [‡] n=10; [§] n=47.

Maternity care provider matched to laboratory tests. The maternity care provider supplied the laboratory with data for 7913 deliveries. Of those, 6614 matches were obtained for women who had had an antenatal blood test through the laboratory—so we could include them in the study sample. The 1299 women for whom no data was available at the laboratory were excluded due to the possibility that they may have been tested at another laboratory. The age range of the study sample was 14 to 52 years with a mean age of 30.6 years, and a median age of 31 years.

Tests for *C. trachomatis* had been performed for 37.5% of the 6614 deliveries between 1999 and 2002. Of those tested, 4.8% were positive for *C. trachomatis*, while 95.2% were negative. Invalid results were obtained for two tests (Table 2).

When analysed by age when tested (under 25 years, 25 years and older), the rate of testing for women 25 years and older (33.3%) was significantly lower than for women under 25 years (61.7%) ($p<0.0001$). Of those women tested, a significantly higher proportion of women under 25 years tested positive for *C. trachomatis* (12.2%) than those 25 years and older (2.3%), ($p<0.0001$).

Table 2. Rates of testing for *C. trachomatis* in pregnant women (who delivered between 1999 and 2002) and rates of chlamydial infection in those tested

	Total	Tested for chlamydia			Chlamydia positive*		
	n	n	%	p value	n	%	p value
Age-band							
Younger than 25 years	985	608	61.7	chi^2=287.89	74	12.2	chi^2=212.93
25 years and older	5629	1874	33.3	p<0.0001	44	2.3	p<0.0001
Ethnicity (all ages)							
NZ European	4015	1306	32.5	chi^2=227.86	26	2.0	chi^2=273.63
Maori†	505	277	54.9	p<0.0001	42	15.2	p<0.0001
Pacific	581	343	59.0		43	12.5	
Asian	495	202	40.8		1	0.5	
Not stated	479	170	35.5		5	2.9	
Other	539	184	34.1		1	0.5	
Total	**6614**	**2482‡**	**37.5**		**118**	**4.8**	

*Includes six 'equivocal' results, percentages calculated using denominator of all those tested;
†Includes 'sole' and 'mixed' Maori; ‡Includes all tests, including two 'invalid' results.

The rate of testing for *C. trachomatis* during pregnancy differed significantly across ethnic groups (p<0.0001), with testing in 59% of Pacific women, 54.9% of Maori women, and 32.5% of New Zealand European women. Of those tests that were carried out, the percentage of those who tested positive for *C. trachomatis* also differed significantly by ethnic group (p<0.0001); 15.2% of Maori women, 12.5% of Pacific women, 2% of New Zealand European women, and 0.5% of Asian women tested positive for *C. trachomatis*.

Table 3 presents a model of estimated national rates of *C. trachomatis* that have been scaled up using the rates of testing and infection presented in Table 1. Figures were calculated using the ethnicity-specific rates of testing and rates of chlamydial infection for Maori, Pacific, and New Zealand European women, as well as 2001 national birth statistics for these ethnic groups.[14]

Table 3. Model estimating national rates of *C. trachomatis* in pregnancy, as well as rates of undetected chlamydia in mothers and consequent infection in neonates (based on the rates of testing and infection found in the present audit)

Ethnicity*	Number of births	Estimated cases of chlamydia in mother†	Estimated cases of undetected chlamydia in mother‡	Estimated cases of chlamydia in neonates§
NZ European	38,307	766.1	517.1	258.6
Maori	12,689	1928.7	869.9	435.0
Pacific	6,321	790.1	323.9	162.0
Total	**57,307**	**3484.9**	**1710.9**	**855.6**

*Ethnicity of the mother giving birth; †Maximum number of cases modelled using the number of births in 2001 and the ethnicity (all ages) specific rates of chlamydia in Table 2; ‡Calculated by subtracting the number of cases of detected chlamydia (calculated using ethnicity specific rates of testing in Table 2) from the total estimated number of cases; §Calculation based on 50% transmission rate from estimated cases of undetected chlamydia.

The estimated infection in the neonate was modelled from the number of cases that would go undetected in pregnancy in the absence of testing, half of which would be transmitted to the neonate.[8,10,11]

Discussion

Less than half of all pregnant women in this audit population were tested for *C. trachomatis* between 1999 and 2002. These results along with increased detection of chlamydial infection in New Zealand neonates[2] suggest that *C. trachomatis* testing is not routinely carried out in pregnancy in New Zealand.

The rate of *C. trachomatis* in untested women is unknown, but the overseas evidence suggests that the overall rate is likely to be similar to the rate in those tested.[7] The rate of chlamydial infection in pregnant women (included in this audit) was high for Maori and Pacific women, and for women under the age of 25 years. This finding was consistent with previous New Zealand studies that have found a higher rate of infection in these groups.[2,3] However, there are limitations to the interpretation of this data as other risk factors such as socioeconomic status, previous STI, and educational level are not able to be taken into account. Women in the present audit could all be considered 'at-risk' due to the fact that they had unprotected intercourse.

The increasing incidence of chlamydial infection in the community has been well documented, along with an increase in cases of neonatal chlamydia.[2,5] Table 3 attempts to quantify the rate of possible undetected infection in New Zealand, as well as the subsequent rates of infection in neonates using the rates obtained in this audit. Furthermore, Table 3 shows an estimated 3485 women who gave birth in New Zealand who might have been positive for *C. trachomatis*. Indeed, based on ethnicity-specific testing rates, as many as 1711 cases might have gone undetected, and with a 50% rate of vertical transmission, up to 856 babies would have been infected with *C. trachomatis*.

We recognise that estimating the burden of chlamydial infection in this way has a number of weaknesses. The model does not take into account variation in rates of testing, age at delivery, or the ethnicity-specific variation in the age-structure of the population. Furthermore, this model presumes the same rates of testing nationally, and assumes that there will be an equal incidence of *C. trachomatis* in those women who were not tested. The median age of the mother at delivery in this sample (31 years) was higher than the national average.[14] Therefore, given the higher rate of *C. trachomatis* in younger women, the rates reported here may underestimate national rates of infection.

Diagnosis of maternal chlamydial infection requires either a cervical or urethral swab, or first pass urine specimen. Recent recommendations for the optimal diagnosis of *C. trachomatis* infection include adding a urine sample to conventional swab(s) for assessment, rather than replacing a swab with a urine sample.[15]

While inhibition is a recognised problem with non-invasive urine samples; when used in combination with swab(s), an increase in the chlamydial detection rate of 9% has been reported.[15] It is recommended that high-risk women are re-tested in the third trimester of their pregnancy to check for re-infection.[12] The recommended treatment for pregnant women is erythromycin 500 mg by mouth (4 times per day, for 7 days).[7]

However, a major drawback of this treatment is the high rate of gastrointestinal side effects and the length of treatment. Amoxycillin 500 mg by mouth for 7 days is shown to be equal in efficacy to erythromycin, with fewer side effects.[16] Azithromycin 1 g is an effective single dose treatment of *C. trachomatis*, is listed as an alternative regimen when compliance is an issue, and is being increasingly prescribed in pregnancy in New Zealand and overseas.[13,17] Contact tracing and treatment of partners are also essential to prevent re-infection.

Conclusions

There is a high rate of maternal *C. trachomatis* and incomplete testing for the infection in pregnant women. These findings highlight the need to instigate routine testing for *C. trachomatis* in pregnancy—to reduce the significant, yet preventable morbidity associated with chlamydial infection in both the mother and the neonate.

Routine screening for *C. trachomatis* in pregnancy is currently recommended in evidence-based international guidelines,[7,13] and has been advocated by other researchers in New Zealand.[1,2,5] Pregnant women are an easily reached population for testing and the beneficial health and economic consequences of detecting and treating *C. trachomatis* are significant.[9] The unacceptable rate of chlamydial infection in pregnancy, and the avoidable burden of disease in pregnant women and neonates must be highlighted to maternity caregivers.

Lastly, we recommend that testing for *C. trachomatis* should be added to best-practice screening that is already carried out in pregnancy.

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Management of unstable angina and non-ST-elevation myocardial infarction: do cardiologists do it better? A comparison of secondary and tertiary centre management in New Zealand

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Background Internationally, differences have been noted in how specialist cardiologists and general physicians manage acute coronary syndromes (ACS). Whether a similar practice difference exists in New Zealand is unclear.

Aim To test the hypothesis that management differences exist between cardiologists and general physicians in patients presenting with a non-ST-segment elevation acute coronary syndrome in a New Zealand setting—and whether these differences (if present) impact on patient outcome.

Methods A retrospective chart review of 324 consecutive patients presenting with a non-ST-segment elevation acute coronary syndrome to Taranaki Base and Waikato Hospitals from 1 January 1999 was undertaken. Patients in Taranaki were managed by general physicians and in Waikato they were managed by cardiologists.

Results Patients presenting to Taranaki Base Hospital were more likely to have high–risk ECG changes with ST-segment depression noted in 34.4% of patients there compared to 16.8% of patients in Waikato ($p<0.001$). Medical management during patient stabilisation was similar in Taranaki and Waikato with high use of anti-thrombotic (89%) and anti-platelet therapy (94%), respectively. However angiography (5.1% versus 23.4%; $p=0.0045$) and revascularisation procedures (4% versus 16.7%; $p=0.0002$) were performed less frequently in Taranaki. No significant difference was noted in mortality at 6 months (9.6% in Waikato versus 13.4% in Taranaki; $p=0.4$) Readmission rates were also similar; occurring overall in approximately one-quarter of the study population.

Conclusion In New Zealand, differences exist in how cardiologists and general physicians manage non-ST-elevation acute coronary syndrome. In particular, the low referral rates for angiography by general physicians is of concern and requires correction as current best-practice guidelines suggest high-risk patients are disadvantaged by a conservative approach to management.

Introduction

Acute coronary syndromes (ACS) are responsible for over 2 million hospital admissions per year worldwide.[1] It has been shown that cardiologists adopt an evidence-based approach more frequently than general physicians in the treatment of patients with ACS. Furthermore, cardiologists are more likely to undertake invasive investigations such as coronary angiography earlier.[2–4] Whether this practice pattern is similar in New Zealand is uncertain.

The aim of our study was to compare the management of patients presenting with non-ST-segment elevation ACS in two New Zealand centres: Waikato Hospital and Taranaki Base Hospital. During the study period, patients in Taranaki Base Hospital were managed by general physicians—with one cardiologist participating in the after-hours on-call roster. All patients with ACS presenting to Waikato Hospital are admitted directly to specialist cardiologist services. Services available at Waikato Hospital include coronary angiography, coronary angioplasty, and coronary artery bypass grafting. Waikato Hospital is the regional provider of these services for Taranaki Base Hospital.

Our hypothesis was to test whether patients presenting with a non-ST-segment elevation ACS (in a New Zealand setting) are managed differently by cardiologists compared to generalist physicians—and whether this difference in practice may result in different patient outcomes.

Methods

A retrospective audit of 200 consecutive patients (presenting with ACS to both Taranaki Base Hospital and Waikato Hospital from 1 January 1999) was performed. Patients eligible for inclusion in the study were identified by a discharge diagnosis of either unstable angina or myocardial infarction (MI) documented on clinical coding. The investigators subsequently reviewed all charts. Patients transferred to Waikato Hospital from other centres (including Taranaki Base Hospital) for coronary angiography or revascularisation procedures were excluded from the Waikato study population—as our intention was to compare local practice patterns only. The Waikato and Taranaki ethics committees granted ethical approval. Significance of variation between hospitals was tested by two-tailed student t-test, using Statistica version 5.1. A p value of <0.05 was considered significant.

Results

Data on 200 consecutive patients with a discharge diagnosis of unstable angina or myocardial infarction were reviewed at each site. Patients presenting with ST-elevation MI or new left bundle branch block were excluded from the study population due to the presence of a primary angioplasty programme in Waikato Hospital. A total of 167 patients at Waikato, and 157 patients at Taranaki, fulfilled inclusion criteria over the study period. Patient characteristics on admission are shown in Table 1.

The Taranaki population were slightly older—with a median age of 70 years compared to 68 years in Waikato. Similar risk factor profiles were noted between the two hospitals—with the exception of hypertension, which was more common in Taranaki (48.4% versus 34.7%; $p=0.013$), and a family history of ischaemic heart disease, which was more common in Waikato (41% versus 18%; $p<0.0001$).

Over 50% of patients in each hospital had documented ischaemic heart disease (defined as known coronary artery disease or previous myocardial infarct). Patients presenting to Taranaki Base Hospital were more likely to have high-risk electrocardiogram (ECG) changes—with ST-segment depression noted in 34.4% compared to 16.8% in Waikato Hospital ($p<0.001$).

No significant difference was noted in troponin status—with 36% of Waikato Hospital's patients and 27% of Taranaki Base Hospital's patients being troponin-positive. (p=not significant [ns]). The use of anti-anginal therapy, aspirin, and ACE-inhibitors was similar prior to admission. However, more Waikato Hospital patients were on statins prior to presentation (17.4% versus 7.6%; $p=0.008$).

Table 1. Patient characteristics on admission

	Taranaki (n=157)	Waikato (n=167)
GENERAL		
Median age	70	68
Mean age	68.5	68
(Age range)	(34–86)	(36–95)
Male	87 (55.4%)	89 (53.3%)
New Zealand European (Pakeha)	140 (89.2%)	139 (83.2%)
Maori	14 (8.9%)	14 (8.4%)
Smoking	66 (42.0%)	77 (46.1%)
Hypercholesterolaemia	91 (58.0%)	81 (48.5%)
Family history of IHD[†*] (p<0.0001)	28 (17.8%)	69 (41.3%)
Hypertension[*] (p=0.013)	76 (48.4%)	58 (34.7%)
Diabetes mellitus	28 (17.8%)	35 (21.0%)
Past history of IHD	85 (54.1%)	96 (57.5%)
ECG		
LBBB[‡]	6 (3.8%)	15 (9.0%)
ST depression[*] (p<0.001)	54 (34.4%)	28 (16.8%)
T-wave abnormalities[*] (p=0.02)	42 (26.8%)	65 (38.9%)
Normal	55 (35.0%)	59 (35.3%)
Troponin elevation	43 (27.4%)	60 (35.9%)
MEDICATIONS		
Aspirin	95 (60.5%)	108 (64.7%)
Beta-blockers	62 (39.5%)	66 (39.5%)
Nitrates	76 (48.4%)	85 (51%)
Calcium-antagonists	47 (30%)	67 (40%)
ACE-inhibitors	41 (26%)	46 (27.5%)
Statins[*] (p=0.008)	12 (7.6%)	29 (17.4%)

[*]Statistically significant; [†]Ischaemic heart disease; [‡]Left bundle branch block.

In both hospitals, medical management during patient stabilisation was similar—ie, high use of anti-thrombotic and anti-platelet therapy (Table 2). However, Waikato patients were more likely to undergo further inpatient risk stratification with treadmill testing (12.6% versus 3.8%), or to proceed directly to coronary angiography prior to discharge (23.4% versus 5.1%).

Not surprisingly, this more aggressive approach by Waikato Hospital was associated with a higher use of revascularisation strategies (16.7% versus 4%). Conversely, more Taranaki patients were discharged on nitrates as an anti-anginal therapy (84.7% versus 68.9%; p<0.001)—with similar use of beta-blockers and calcium channel blockers. The median length of stay during the index admission at Waikato Hospital was shorter than that at Taranaki Base Hospital (4 days versus 5 days, respectively).

At 6 months, 30% of Waikato patients had angiography performed on them, compared to 12.7% in Taranaki (p=0.002) (Table 3). Revascularisation rates remained higher in Waikato but were predominately performed during the index admission. Only 7.0% of patients admitted to Taranaki Base Hospital (with unstable angina or a non ST-segment elevation MI) had undergone a revascularisation procedure at 6 months. This contrasts with 27% of the Waikato study population.

Table 2. Patient management in hospital

	Taranaki (n=157)	Waikato (n=167)	P=
Inpatient medication			
Heparin	139 (88.5%)	150 (89.8%)	NS*
GP IIb/IIIa inhibitors	1 (0.6%)	4 (2.4%)	NS
Stress testing	6 (3.8%)	21 (12.6%)	0.0045
Coronary angiography	8 (5.1%)	41 (23.4%)	<0.001
Revascularisation	6 (4.0%)	28 (16.7%)	<0.001
PTCA†	2 (1.3%)	15 (9%)	<0.001
CABG‡	4 (2.5%)	13 (7.8%)	0.033
Medications on discharge			
Aspirin	148 (94.3%)	158 (94.6%)	NS
Beta-blockers	100 (63.7%)	114 (68.3%)	NS
Nitrates	133 (84.7%)	115 (68.9%)	<0.001
Calcium channel blockers	77 (49.0%)	78 (46.7%)	NS
ACE inhibitors	61 (38.9%)	56 (33.5%)	NS
Statins	28 (17.8%)	37 (22.2%)	NS

*Not significant; †Percutaneous transluminal coronary angioplasty; ‡Coronary artery bypass graft.

Table 3. Patient outcome at 6 months

	Taranaki (n=157)	Waikato (n=167)	P=
Exercise tolerance test	57 (36%)	49 (29%)	NS*
Coronary angiography	20 (12.7%)	50 (30%)	0.0002
Coronary angioplasty	5 (3.2%)	25 (15 %)	0.006
CABG	(3.8%)	20 (12%)	0.007
Revascularisation	11 (7.0 %)	45 (27%)	0.0001
Mortality	21 (13.4%)	16 (9.6%)	0.4
Readmission	35 (22.3%)	46 (27.5%)	NS
PTCA† on readmission	1 (3.4%)	8 (17.4%)	0.0001
CABG‡ on readmission	-	7 (15.2%)	0.00001
Revascularisation on readmission	1 (3.4%)	15 (32.6%)	0.0017

*Not significant; †Percutaneous transluminal coronary angioplasty; ‡Coronary artery bypass graft.

Readmission rates with an acute coronary syndrome were similar—with approximately one-quarter of the study population re-hospitalised at 6 months. In this small selected group, revascularisation rates remained low in Taranaki (3.4% versus 33% in Waikato; p=0.0017). No mortality difference was noted at 6 months, although a trend was seen in favour of Waikato patients (9.6% versus 13.4%; p=0.4).

Discussion

Coronary heart disease remains the leading single cause of death in New Zealand, accounting for 23% of all deaths in 1998.[5] In addition, patients frequently present with an acute coronary syndrome resulting in hospital admission. Differences in the management of this common condition by general physicians and specialist

cardiologist have been noted.[2–4] Specifically, cardiologists appear to have a more evidence-based approach to ACS treatment, are more likely to prescribe cardiac medications (including aspirin and beta-blockers) and are less reluctant to undertake invasive investigations.[2–4]. Our study shows little difference in the medical stabilisation of patients. Aspirin use was high in both hospitals. The overall use of beta-blockers is slightly disappointing, however—with less than 70% of patients receiving these agents on discharge. Current beta-blocker use in ACS has improved at Waikato Hospital and is in excess of 80% in the ongoing GRACE Registry , which compares favourably with global patterns in this registry. Low statin use on discharge likely reflected New Zealand regulatory issues during the study period.

Recently, management of patients with ACS has become more complex—with growing evidence supporting a more aggressive interventional-based approach in high-risk individuals.[6–8] It is therefore conceivable that patients may be disadvantaged in the future by non-cardiologist care, particularly if further stratification of high–risk cases is not guided by angiography.

Our results certainly show angiography to be undertaken more frequently when cardiologists (in a New Zealand setting) manage patients with acute coronary syndromes. This discrepancy (between Taranaki and Waikato) does not appear to be related to patient population—as similar demographics were noted in each region.

The reasons why fewer patients in Taranaki were referred for angiography are purely speculative. Guidelines for the management of acute coronary syndromes have been updated in recent times and promulgated in the literature and by key opinion leaders at national meetings. For example, The Cardiac Society of Australia and New Zealand recommend an early invasive strategy in patients presenting with high-risk features[9]—these include individuals with elevated cardiac–markers and dynamic ECG changes. Of concern, significantly more patients in the Taranaki region were high risk, with ST changes noted in 34% of admissions.

Access and familiarity with coronary angiography have previously been shown to influence the frequency of performing studies.[10] This may partly explain the differences noted in our study. At the time of our study, diagnostic angiography was not routinely performed in Taranaki. Indeed, patients in whom angiography was considered necessary were discussed with the cardiology team in Waikato Hospital, and transferred (often several days later) when beds were available. The delay in access to timely intervention is undoubtedly a significant contributory factor to low angiography rates in Taranaki Base Hospital.

What are the advantages of early intervention for patients presenting with an acute coronary syndrome in New Zealand? A mortality benefit has been documented (at 1 year) in the FRISC II trial—with 1.7 lives saved per 100 patients treated.[6] In contrast, TACTICS did not show any mortality benefit at 6 months.[7] In our study, a favourable trend towards mortality reduction was only noted at 6 months (9.6% versus 13.4% p=ns) in patients managed in an interventional centre. Readmission rates at 6 months were uninfluenced by a higher use of revascularisation procedures at Waikato hospital. (These findings are similar to the TACTICS trial.)[7]

A reluctance to refer for angiography was again noted when patients were readmitted to the Taranaki region. Unfortunately, the impact of intervention on quality of life was not assessed following discharge. However, a difference was noted in anti-anginal

use—with significantly more patients in Taranaki on nitrate preparations. This is perhaps not surprising, given lower intervention rates.

Our study has significant limitations due to its small and retrospective nature. Clinical coding on discharge identified the study population and it is therefore possible that not all patients presenting with an acute coronary syndrome were included. The size reduced the power to detect small changes in treatment between the two hospitals. This may be particularly relevant given the trend to reduced mortality.

Similarly, the use of only two hospitals (Waikato Hospital and Taranaki Base Hospital) was also a limitation. However, one possible advantage of the study's retrospective nature was that the clinicians were unaware of the study and the results are therefore more likely to reflect 'real world' practice.

In conclusion, patients presenting with acute coronary syndromes in a New Zealand setting appear to be managed differently by cardiologists and general physicians. Whilst initial medical stabilisation is similar, patients are referred more frequently for coronary angiography by cardiologists. In addition, a trend was noted towards mortality reduction when cardiologists managed patients. However, no difference was noted in readmission rate at 6 months.

The low referral rates for angiography are concerning and need addressing—as current best-practice guidelines suggest that high-risk patients are disadvantaged by a conservative approach to management. Moreover, patients presenting with a high-risk acute coronary syndrome should undergo further risk stratification with angiography (regardless of the hospital site at presentation).

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The New Zealand child work-related fatal injury study: 1985–1998

Rebbecca Lilley, Anne-Marie Feyer, John Langley, and John Wren

Abstract

Aims To estimate the numbers and rates of work-related fatal injury for children under the age of 15 years.

Methods Potential cases of work-related injury deaths of persons aged <15 years of age were identified from the national electronic mortality data-files for the period 1985-1998 inclusive. The circumstances of the death in each fatality incident were reviewed directly from coronial files to determine work-relatedness.

Results A total of 87 workplace work-related fatalities were identified. The vast majority of children identified were fatally injured while a bystander to another person's work. Workplace bystander involvement was found to vary by age, with the majority of workers identified aged 10–14 years old. With a third of all fatalities, the agricultural industry was the most common industry for workplace work-related fatalities in children. In the period 1985–94, children <15 years of age were found to account for 46% of New Zealand's total workplace bystander deaths.

Conclusions Children contribute significantly to the overall burden of work-related fatal injury in New Zealand, especially as bystanders to other people's work. The high contribution to bystander deaths by children aged <15 years suggests that hazard control in certain work settings is lacking.

Childhood injury is a major public health problem in New Zealand. Injury among New Zealand children and teenagers (<20 years of age) accounts for 34% of fatalities in this age group, and is the leading cause of death for this age group, with the exception of those less than 1 year old.[1] Comparisons with other OECD countries show that New Zealand's injury mortality rate of 13.7 per 100,000 for children aged <14 years ranked New Zealand poorly at 22nd out of 26 OECD countries.[2]

Although not traditionally viewed as part of the formal workforce, children under 15 years of age do participate in work under less formal work arrangements, such as casual holiday work, or part-time after-school work, and may be exposed to workplace hazards when they visit, or live on, worksites (eg, farms). There is evidence to suggest that children below official working age are at considerable risk of work- related fatal injury (WRFI).

From recent Australian data, WRFIs to children were identified as a substantial proportion of work-related deaths.[3] Furthermore, the Australian data revealed that 47% of workplace bystander WRFIs were to those aged <15 years. These data also revealed that those children living on (or visiting) farms were more likely to sustain WRFIs, with 54% of all WRFI in children occurring on farms.[3] Given the similarities between New Zealand and Australia, the New Zealand experience of WRFI to children is also likely to be similar. For instance, a New Zealand study of fatal injuries

on farms revealed that 17% of fatal injuries on farms occurred in children aged <16 years.[4]

In New Zealand, the Health and Safety in Employment Act (1992) clearly requires that employers and the Occupational Safety and Health Service protect all people who come into 'contact' with workplaces. Indeed, there is a clear legislative mandate to investigate and prevent child work-related deaths, yet comprehensive and reliable data on child WRFIs in New Zealand is non-existent. This study aimed to address this gap by identifying and describing work-related fatal injuries in children aged <15 years of age.

Methods

Identification of work-related deaths—This study followed the protocol used by Feyer et al[5] to create an adult WRFI data set. Potential cases of work-related injury were identified from the New Zealand Health Information Service (NZHIS) electronic national mortality file—using the external cause of injury coding, commonly known as E-codes. The same E-codes, as used previously,[7] were used to identify all potential work-related cases. Additional E-codes (E810-829, 846-848, and 919) were considered for the child work-related fatal injury study (CWRFIS)—to capture the potential work-related fatal injuries that occurred due to traffic crashes on public roads, thus allowing for a more complete capture of WRFI. This differs from the adult WRFIS, which excluded all deaths that occurred due to traffic crashes on public roads.[5] For the purposes of this study, a child was defined as any person less than 15 years of age.

Coronial files were reviewed in random sequence to determine work-relatedness. A detailed description of the selection process followed is described by Feyer et al (2001).[5] Deaths classified as definitely work-related were included in the study.

Data coding—Information in each coronial file was coded to obtain an electronic description of each work-related case. Data for the CWRFIS was simultaneously coded with that for another study investigating work-related road traffic crash fatal injuries using the same coders. Although rater agreement was not undertaken specifically for the CWRFIS, it was undertaken for the traffic crash study. Inter-rater agreement was found to be better than 91% for case determination by the same coders. Coding of variables has been described previously by Feyer et al (2001).[5] Additional coding of the child's activity at the time of fatal injury was undertaken by categorising coronial descriptions of the child's activity at the time of the incident.

Analysis—Rates (including 95% confidence intervals) were calculated for work-related injuries per 100,000 children per year; counting each individual in the denominator equally as described previously.[5] Census data from 1986, 1991, 1996, and 2001 were used to estimate denominator data for the calculation of rates. The contribution of work to all fatal injury to children was calculated as a percentage of all child injury fatalities recorded by NZHIS. All child injury fatalities were identified from the NZHIS electronic national mortality file using cases coded within the range E800–E999.

The inclusion criteria and categorisation for fatally injured persons were as follows:[8]

Workers—Children employed for pay, profit, payment-in-kind, in a job or business or on a farm; and children who worked without pay in a family business, or on a farm.

Bystanders—Children not engaged in work but who were fatally injured as a direct consequence of the work of another person. Bystanders were further divided into workplace and road bystanders. Workplace bystanders were children who were not working but were fatally injured as a result of workplace activities, generally not associated with public roads or public transport. As an exception, some incidents involving public roads and public transport were included in the workplace category where the child was exposed to similar hazards to those faced by the working person controlling or working the vehicles—for example, child passengers in the cabin of a working truck. Road bystanders were children who were not working but who were fatally injured in a motor vehicle incident on a public road (or on public transport) as a result of other people's work.

Students—Students were those from primary school age or older where the incident occurred during school time, on school premises, or while they were performing a task directly connected with their course.

The intention of this study was to describe (for children <15 years of age) the total burden of WRFI by considering additional E-Codes that captured potential cases of WRFI that occurred due to traffic crashes on public roads. While sufficient information was available from coronial files to identify the WRFI events that occurred due to motor vehicle traffic crashes, any further information regarding the road-bystander deaths, in particular, was limited.

In contrast to non-traffic crashes (eg, vehicle crash on farm), there was often insufficient information for traffic crashes (eg. vehicle crash on public road) on the bystander. Typically, the coronial report focused on the driver of the motor vehicle. Motor vehicle death on public roads are contributors to the total burden of work-related deaths differing from other motor vehicle deaths by virtue of a working vehicle being involved in the fatal incident. Motor vehicle deaths to New Zealand children have been described in-depth elsewhere.[6] This paper, while quantifying the total burden of WRFI for children aged <15 years of age, only describes the worker and workplace bystander deaths in more detail. The data set described in detail in this paper is comparable with published Australian data.[3]

Data were analysed using the SPSS Version 9.0 statistical package.

Results

During the period 1985–98, a total of 238 work-related fatalities were identified for children aged <15 years of age. This was an average of just over 17 work-related fatalities per year. The majority of fatalities were deaths to children who were bystanders to someone else's work (Table 1).

The bystander fatalities were mostly road bystanders (66%) on a public road, with the remainder of bystander deaths having occurred at a place of work. The most common scenarios for road bystander fatalities were vehicle versus vehicle incidents (eg, a child travelling in a car that crashed into a working vehicle), or pedestrian versus vehicle incidents (eg, a child hit by working vehicle while crossing the road), which contributed to 34% and 42% of all road bystander fatalities, respectively.

Table 1. Work status of child work-related fatal injury (WRFI): 1985–98

Work status	Number (%)	Rate* (95% CI)
Working	12 (5%)	0.11 (0.06–0.19)
Bystander total	222 (93%)	1.97 (1.72–2.25)
- *road*	147	
- *workplace*	75	
Student	4 (2%)	
Total	**238**	**2.11 (1.85–2.4)**

*Per 100,000 children per year.

For the purposes of this paper, as indicated earlier, only deaths that occurred to workers (n=12) or workplace bystanders (n=75) (hereafter collectively referred to as workplace fatalities) are described in further detail.

Gender, age, and ethnicity—Of the 87 workplace fatalities, 41% were in children aged <5 years old (Table 2). The majority (82%) of child fatalities were male. Ethnicity could not be determined from the coronial files in over half of all child fatalities. For the 41 cases where ethnicity could be determined, 27 were New Zealand European/Pakeha and 9 were Maori. Males had a higher incidence rate of WRFI than females. In particular, males aged 0–4 years had the highest gender/age-group-specific rate of WRFI (Table 3). During the study period, WRFI accounted for 6% of all fatal injuries in male children and 2.5% of all fatal injuries in female children.

Table 2: Age-related characteristics of the child workplace WRFI: 1985–98

	Age group (years)			
	0–4	**5–9**	**10–14**	**Total**
Number of cases	**36**	**26**	**25**	**87**
% female	17	27	20	**18**
% on farm	53	31	28	**39**
% on public road	14	23	36	**23**
% bystanders	100	85	68	**86**
% workers	0	15	32	**14**
% weekend deaths	28	31	16	**25**

Table 3. Child workplace WRFI by age and sex: 1985–98

Age group	Rate* (95% CI)		Percentage†	
	Males	**Females**	**Males**	**Females**
0–4 years	1.55 (1.05–2.22)	0.33 (0.12–0.71)	5.28	1.59
5–9 years	1.0 (0.60–1.56)	0.39 (0.16–0.80)	8.11	4.17
10–14 years	1.03 (0.63–1.59)	0.27 (0.09–0.63)	5.84	3.07
Total	**1.20 (0.93-1.51)**	**0.33 (0.19-0.52)**	**6.03**	**2.54**

*Per 100,000 population per year; †Work-related fatal injuries as a percent of all fatal injury to children (1985–98).

Work involvement in fatality—Workplace bystander involvement varied with age. All workplace fatalities in children aged <5 years old were bystander deaths. Eighty-five percent of workplace fatalities in the group aged 5–9 years were bystander deaths—while 68% of workplace fatalities in 10–14 year olds were bystanders. Only 12 children were identified as working at the time of the fatal incident, with two thirds of the working children aged 10–14 years. The most common working scenario at the time of the fatal incident was working riding a motorbike to shift stock on a farm.

Location of fatal incident—Fatalities on farms dominated the 0–4 years age group with just over half of all fatalities occurring on a farm; while for older children, close to a third of all deaths occurred on a farm. Even with road bystander fatalities excluded, workplace fatalities on public roads were notable with 36% of fatal incidents in 10–14 year olds occurring on a public road.

Time of injury—Overall, a quarter of all child work-related deaths occurred on the weekends with close to a third of all deaths for children aged 5–9 years occurring on weekends. Exclusion of incidents during official school hours on a weekday reveals that 62% of 10–14 year olds (and 55% of 5–9 year olds) were fatally injured before, or after, official school hours on a weekday.

Agency and mechanism of fatal incident—For children aged <5 years, the most common mechanism of injury (Table 4) was being hit by a moving object (56%). Drowning (14%) and vehicle crashes (17%) were also common mechanisms of fatal injury for these young children.

Table 4. Mechanism and agent of child WRFI: 1985–98

	Age categories (n)			
	0–4 yrs	**5–9 yrs**	**10–14 yrs**	**Total n (%)**
Mechanism of injury				
Falls	-	1	3	4 (5)
Hitting object with part of body	3	1	1	5 (6)
Hit by moving object	20	5	2	27 (31)
Heat, radiation, and electricity	1	-	1	2 (2)
Drowning	5	6	1	12 (14)
Chemicals and other substances	1	-	2	3 (3)
Vehicle accident	6	13	14	33 (38)
Unknown			1	1 (1)
Agency of fatal incident				
Vehicles				
- *Truck*	9	2	4	15 (17)
- *Bus*	-	2	2	4 (5)
- *Car/Ute*	6	1	2	9 (10)
- *Motorbike/ATV*[*]	-	5	2	7 (8)
- *Other*	2	1	-	3 (3)
Aircraft	1	4	4	9 (10)
Farm machinery				
- *Tractor*	4	1	2	7 (8)
- *PTO*[†]	2	-	-	2 (2)
- *Other*	2	-	-	2 (2)
Portable plant	1	1	1	3 (3)
Poison and explosive	1	-	1	2 (2)
Body of water				
- *Pool*	1	3	-	4 (5)
- *Dam*	2	-	-	2 (2)
- *River*	1	1	1	3 (3)
- *Other*	1	1	-	2 (2)
Materials	1	1		2 (2)
Animal and insects				
- *Horse*	1	-	1	2 (2)
- *Other*	-	1	1	2 (2)
NEC[‡]	1	2	4	7 (8)
Activity at the time of fatal incident				
Ambulatory				
- *At play*	19	8	4	31 (36)
- *Helping out*	4	3	2	9 (10)
Other	4	-	2	6 (7)
Passenger in/on vehicle	9	11	12	32 (37)
Driving vehicle	-	4	3	7 (8)
Riding (horse, bicycle, etc)	-	-	2	2 (2)

*All terrain vehicle; [†]Power take off; [‡]Not elsewhere classified.

Vehicle crashes (off the public road) were the most common mechanism of fatal injury for children aged 5–9 and 10–14 years of age. For children aged 5–9 years, drowning (23%), and being hit by moving objects (19%), were common.

The agents most commonly involved in fatal injury were vehicular agents such as trucks, and cars/utilities, as well as aircraft and tractors (Table 4). Apart from vehicles, farm machinery (such as tractors) and bodies of water (such as dams and rivers) were common agents of the fatal incident among 0–4 year olds.

For older children, vehicular agents were the most common agents of the fatal incident. Notably, bodies of water (such as a public pool or irrigation dam) contributed to 19% of fatalities in 5–9 year olds.

Activity at time of fatal incident—The most common activities being performed at the time of the fatal injury were being a passenger in, or on, a vehicle (37%) and 'playing' in or near the workplace (34%). Ambulatory activities, especially playing (53%), at the time of fatal incident were much more common for very young children than vehicular activities (Table 5). As children increased in age, being a passenger in (or on) a vehicle was a more common activity at the time of the fatal incident with 42% of 5-9 year olds, and 48% of 10–14 year olds, killed as passengers.

Table 5. Main activity of child at time of child workplace WRFI: 1985-98

Activity	Number of children	Percent (%)
Ambulatory		
- *At play*	30	34
- *Helping out*	9	10
- *Other*	6	7
Passenger in/on vehicle	31	36
Driving vehicle	4	5
Riding (ie, horse, motorbike)	7	8

Occupation and industry—Of the 12 children identified as working at the time of fatal injury; 2 were working as street milk vendors, 1 was delivering newspapers, and the remaining 9 were working as farm labourers.

A third of all workplace WRFIs in children occurred in the agricultural industry. Other industries with notably high representation of workplace WRFIs in children include the road transport (14%), air transport (10%), and the sport and recreation industries (10%). It should be noted that while the air transport industry represented 10% of work-related fatal injuries in children, there were only 3 incidents—each involving multiple deaths.

Discussion

This study found that it was unusual for children in New Zealand to be fatally injured while involved in work. We identified 87 workplace fatalities in children (<15 years of age) from 1985 to 1998. We identified 10 children who were performing work tasks at the time of the fatal incident; and we identified 2 children commuting to (or from) work who were fatally injured. The vast majority (86%) of workplace fatalities were in workplace bystanders. During the period 1985–98, WRFI accounted for at

least 6% of total child fatal injuries in males, and 2.5% of total child fatal injuries in females. In males aged 5–9 years, at least 8% of all child injury fatalities were work-related. Male WRFI, in particular, contributes to a significant proportion of all child fatal injuries.

That it was more likely that a child sustained fatal injuries as a bystander to someone else's work, rather than as a worker, is significant. Bystander deaths reflect inadequate control over hazards in the workplace and, as such, can provide insight into aspects of the breakdown in the control of hazards in the work setting.[8]

Among adult WRFIs,[9] 67 workplace bystander deaths were identified for persons aged 15–84 in the period 1985-94 while (over the same period) the present study identified 58 workplace bystander deaths in children <15 years of age. Therefore, children <15 years of age accounted for 46% of New Zealand's total workplace bystander deaths (excluding motor vehicle traffic crashes) in the period 1985–94.

Similarly, Australian children aged <15 years contributed to 47% of Australia's total workplace bystander deaths.[8] This highlights that children are an important and particularly vulnerable group who are exposed to hazards in the workplace which, for the most part, are under the control of others.

In this study, the level of bystander involvement changed with age. With children aged 0–4 years old, 100% of WRFIs were as workplace bystanders. By age 10–14 years, 68% of fatalities were classified as workplace bystander deaths, with the remaining fatalities in this age group classified as worker deaths. The observed increase in the proportion of worker fatalities with increasing age reflects the movement of older children towards informal employment (eg, newspaper deliveries) as they get closer to the official working age. With increasing age, the movement into the informal workforce changes the exposure of the child to workplace hazards, and the pattern of fatalities becomes more like that of adults [3].

Males were identified as being at greater risk of work-related fatal injury, with only 18% of deaths overall occurring in females. A similar proportion of male and female work-related fatal injuries were identified in Australia.[3] Within male workplace fatalities, there was a preponderance of males aged 0–4, thus suggesting that this group of very young and vulnerable males may be at greater risk of WRFI.

The most common location of the fatal incident was on a farm. On the farm, home and work activities overlap, and informal participation in farm work is commonplace amongst New Zealand children. A survey of 160 New Zealand farms found that 77% of 4–7 years olds, 90% of 8–11 year olds, and 85% of 12–16 year olds living on farms participate in work activities on the farm.[10]

On farms, children are more likely to face (and have contact with) the well-documented and prevalent hazards of motor vehicles and farm machinery. Early involvement in informal farm work further exposes rural children to workplace hazards at an early age. With nearly one fifth of all injury on New Zealand farms occurring in children aged <16 years,[4] children represent a significant proportion of all farm injuries.

A high proportion of the fatal incidents occurring on a farm were in children aged 0–4 years. This may reflect a number of factors specific to rural work environments. Firstly, there is a lack of access to childcare services in rural areas,[11] with rural parents

perhaps being more likely to take a child out to work with them while the other partner is away, for example. Children aged <5 years were likely to be playing at the time of the fatal incident; often they were playing around a parent in the workplace while the parent was working. The difficulty in providing constant supervision while parents are performing farming duties could explain why young children in rural farm environments are particularly vulnerable to work-related hazards.

The type of fatal incident varied by age. Fatal incident involving vehicles were common across all ages with children aged <5 years most likely to be hit by a moving vehicle and those aged 5–14 most likely to be a passenger in (or on) a vehicle. Drowning was another notable mechanism of fatal incident in children aged 0–9 years old. The agency of fatal incident reflects the mechanism of injury with vehicular agents being the most common cause for all age groups, and bodies of water being a relatively common cause in younger children. These common mechanisms and agents of fatal incident reflect those identified in other countries as being of high risk to children.[12,13]

Agriculture is a significant contributor to the overall WRFI burden for children—with well over a third of all child fatalities (identified in this study) occurring on farms. This is not surprising. The agricultural sector is consistently identified as being one of the highest risk groups for fatal injury in adult workers,[7] as well as in children.[12,14,15]

The patterns of child WRFI identified in this study are not unique to New Zealand. In New Zealand, as elsewhere, these common patterns of fatal injury have been found to be associated with: (1) inadequate supervision of small children in work settings; (2) permitting children to be in the area of moving or unguarded machinery; (3) allowing children to accompany workers using machinery; and (4) having children performing work-related tasks inappropriate for their age and physical size[13,16]. The preponderance of farm and vehicle injuries observed in this study could indicate the existence of similar risk factors in New Zealand. Compared with adults, children lack work experience, physical size, physical strength, and attention to task,[17] thus placing children at greater risk of fatal injury in the workplace. Children are also more vulnerable to severe injury when injury events occur due to the child's physical fragility.

The findings of this study have important implications for the way workplace hazards are controlled in New Zealand. The results indicate that children contribute significantly to the overall burden of WRFI in New Zealand, especially as bystanders to other peoples work—and the results also indicate that work-related incidents contribute to the total burden of child injury deaths. The high contribution to workplace bystander deaths by children aged <15 years indicates that hazard control in certain work settings is lacking. Greater attention needs to be paid to managing hazards and children's activities in (or near) workplaces; a priority in this respect is farms and vehicles.

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Oral health status and oral treatment needs of dependent elderly people in Christchurch

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Abstract

Aim To describe the general health, oral health status, and treatment needs of dependent older people living in Christchurch rest homes.

Methods 210 Grade 3 residents from seven randomly selected care facilities were examined for: dental/denture status, caries, periodontal disease, and oral cleanliness.

Results Age ranged from 65 to 103 with an average age of 84.6 years. The female-to-male ratio was 3:1. Thirty-two percent of residents were dentate (average 14 teeth). This figure was up from 16–19% one decade ago ($p<0.01$; chi-squared test). Sixty-five percent of dentate residents had caries. Each dentate person had an average of 2.38 teeth with carious lesions in the root or crown. 51.5% of residents required restorative treatment and 38.2% required an extraction. Twenty residents had at least one 'carious stump'. Eighty-two percent of residents required scaling of the teeth. Two thirds had no natural teeth, and one third of those with dentures required treatment. Eighty-nine residents (42%) were unable to communicate about past dental behaviours.

Conclusions These data indicate that (compared to one decade ago) more dependent elderly people are retaining their natural teeth—but they are keeping no more teeth, and the health of the teeth has not improved. Most elderly people do not regularly receive dental assessments or care.

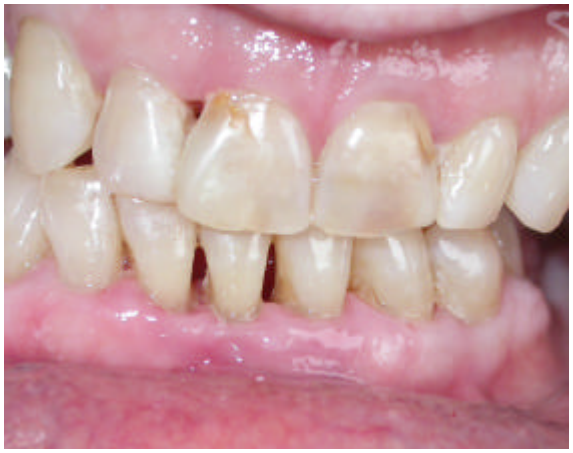
Canterbury, like the remainder of New Zealand and many other countries, has an increasingly ageing population. This will be compounded by numerous 'baby boomers' reaching the age of 65 during the next 10 years. Although most people aged 65 or over are fit and healthy, a minority are frail and vulnerable and require a high level of care and disability support. The number of people aged 85 years and over in Canterbury will more than double over the next 20 years (from 6,318 to 14,750), and many of these older people will retain some of their own teeth.[1,2]

Overseas studies have shown that older people in care have poorer oral health than their community-dwelling counterparts,[3,4] have poor oral hygiene,[5,6] and many do not receive regular dental care.[7–12] A poor nutritional status is associated with poor oral health.[13,14]

A study of institutionalised older people in the Manawatu-Horowhenua region showed that tooth-decay was active in the population.[15] Thomson suggested 'that dentate elderly are indeed a decay-active population and that (when both types of caries [coronal and root surface] are taken into account) they may be more at risk than the children and adolescents upon whom most organised preventive efforts have been concentrated to date'.[1]

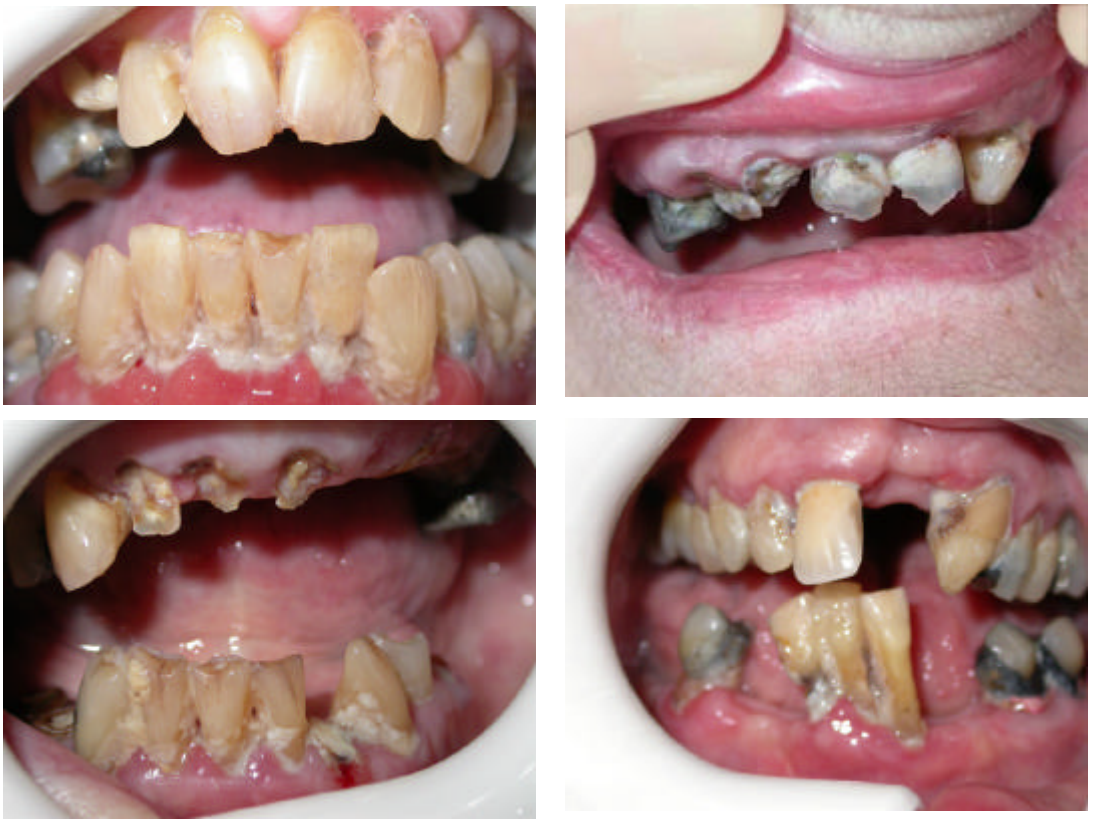
Poor oral health is not an inevitable consequence of aging—some elderly are able to maintain a functional and attractive dentition (Figure 1).

Figure 1. An elderly person with good, healthy dentition



However many dependent elderly are unable to maintain their dentition—leading to marked deterioration in comfort, function, hygiene, and appearance (Figure 2).

Figure 2. Examples of elderly persons with poor, unhealthy dentitions



There are no recent data to show that the predicted trend of more people retaining their teeth into old age is actually occurring in New Zealand. Furthermore, there is no information on the oral health of older people of Canterbury and how their treatment needs are being met. The aim of this study is to describe the general health, oral health status, and treatment needs of dependent older people living in Christchurch rest homes.

Methods

For this study, a dependent elderly person was defined as a resident who requires extensive assistance in order to carry out everyday self-care activities. Those included in the study were long-term residents in private hospital beds, as well as dementia residents (classified as D4 or D6) and Grade 3 rest home residents (those requiring assistance from more than one staff member).

Facilities were randomly selected from a list of rest homes and geriatric hospitals in Christchurch obtained from the Ministry of Health. Participation in this study was obtained by asking all residents in the selected homes (who met the above criteria for a dependent elderly person) to consent to an oral examination. In the situation when a resident was unable to give informed consent, consent was obtained from family, or from a welfare guardian. If consent was not obtained, or a resident was considered by the manager to be too unwell to participate, or the resident was aged under 65 years then that resident was excluded from the study and no data were collected about that person.

One dentist examiner and a recorder carried out all examinations using a dental mirror, headlight, and a WHO periodontal probe. The number and distribution of the teeth and oral prostheses were recorded. Carious lesions were recorded by surface, with crown and root lesions recorded separately.[16]. The need for periodontal treatment was measured using the Community Periodontal Index of Treatment Needs (CPITN) ,[17] oral hygiene was recorded with the Oral Hygiene Index (OHI) [18], and soft tissue lesions were recorded. Dental treatment was recorded on the basis of requiring restorative care or extraction. Required restorative care was recorded as a one surface, two surface, or complex restoration. Details of currently prescribed medications and existing medical conditions were obtained from rest home or hospital records.

The data were recorded on paper, transcribed into an Epi Info™ 2000 data-entry module (CDC, Atlanta, GA), and subsequently analysed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, Ill).

Results

Sample—210 residents (from a total of 318 eligible residents) were examined. Residents who were not examined were either under 65years (8) or could not give consent (100). Their ages were 65–103 years and the average age was 84.6 years. The ratio of females to males was 3:1, and the ethnicity was predominantly NZ European (208).

Health status—188 residents (89.5%) were under hospital care—with just 7 (3%) in dementia care and 15 (7%) in rest home care. The most common medical condition was cardiovascular disease; the most commonly prescribed medications were laxatives and analgesics. On average, each person was on 9 different prescription drugs.

When asked, about their last dental visit, 89 residents (42%) could not remember when it was, or they were unable to communicate.

Dental status—Sixty-eight (32%) of the 210 residents were dentate, and had a total of 949 teeth. Each dentate person had on average 14 teeth and the number of teeth present ranged from 1 to 27 (Table 1). Twenty percent of all residents (40) had more than 10 teeth.

Table 1. Numbers of teeth for male and female residents

Number of teeth	Males	Females	Males and females combined
0	33	109	142
1–4	1	3	4
5–8	5	14	19
9–12	6	5	11
13–16	1	6	7
17–20	2	8	10
21–24	4	5	9
25–28	2	6	8

Dental caries—Thirty-five percent (24) of residents (with teeth) were caries free. Sixty-five percent (44) of those with teeth had at least one carious lesion in the root or crown, and twenty of these had one or more carious stumps. Five dentate residents had five or more carious stumps.

The 'average' dentate resident had 14 teeth. Each dentate person had an average of 2.38 teeth with carious lesions in the root or crown.

168 (17%) of all teeth (949) had active carious lesions. 77 carious teeth (8%) could be restored. 96 teeth (10%) required extraction for dental caries or other reasons.

Treatment needs—The 68 dentate residents required an average of 1.1 tooth restorations; 22 (31.4 percent) needed one or two restorations and a further 13 (19.1 percent) three-or-more restorations. Most (93 percent) of the restorations required were single-surface restorations.

On average, each dentate person required 1.4 teeth to be extracted, with 28 (41 percent) needing at least one extraction. Thirteen percent (9) required the extraction of three-or-more teeth, with one resident requiring the extraction of 18 teeth.

Two-thirds (45) of the dentate residents had a CPITN score of 2 or less—thus requiring scaling and improved oral hygiene to resolve their periodontal problems. Only two people had the highest score of 4, which indicates the need for complex periodontal treatment. The mean Oral Hygiene Index score was 1.3

Dentures—Most residents (142) were completely edentulous. Many wore removable dentures. One hundred (70%) residents used both upper and lower dentures, 25 (17.6%) did not wear one or other of their dentures, and 17 (8.1%) did not have either an upper or a lower denture. The examining dentist recommended treatment for about a third of the denture-wearing residents whereas only one fifth thought they needed treatment. A further fifth could not communicate. Some dentures were not clean. The mean percentage of denture area covered in biofilm was 28% for upper dentures and 26% for lower dentures.

Soft tissue lesions—The most common soft tissue lesions were *Candida*-associated conditions (44) and traumatic ulcers (7). No new neoplastic lesions were identified.

Discussion

Compared with previous New Zealand studies on the oral health of the elderly (all conducted over 10 years ago); in this study, the proportion of elderly people retaining some of their teeth has increased.[15,19,20] The participants in Thomson's 1989 rest home

study were similar to this study in terms of age, however the proportion of dentate residents (19.5 percent) was less than two-thirds of that found in this present study (Table 2**)**.[15,19–22] Thirty-six percent of Thompson's sample included people who met the criteria for dependency used in this study. The remainder 64% were classified as moderately dependent. Other New Zealand studies have included independent elderly people in their sample (Table 2).[19,20]

It does not appear that dependent dentate elderly people are retaining more teeth—because the number of teeth retained by the dentate individuals in this study (average 14) is similar to that found in earlier New Zealand studies.[15,19,20]

The dependent elderly in New Zealand have poor oral health. Oral health and oral disability in older people is not seen as part of overall health by central government funders,[23] and this (in part) negatively influences the delivery of care. We believe that oral health should be considered part of overall health when the Ministry of Health writes service specifications and minimum requirements for rest homes and residential care facilities. In the current contract, services of dentists are specifically excluded from the specifications. Because of this, oral health care and the recognition of overt oral disease are not easily integrated into an overall health care plan for dependent older people.

People who cannot look after themselves rely on institutions with policies that provide—healthcare (including oral healthcare), managers who ensure the policies are implemented, and carers who have skills that will deliver straightforward and effective oral hygiene and be able to recognise overt oral disease. Unfortunately even apparently simple preventive measures (eg, cleaning teeth and dentures) are not carried out effectively by older people or their carers. Indeed, currently there is no formal training program in oral hygiene and the recognition of oral disease for carers.

Most residents in this study had lost contact with their dentist but were seen regularly by their medical practitioner. Few received regular dental care, but all received medical care. In this situation, it is likely that the resident's medical practitioner would be able to recognise and refer patients who would benefit from dental care. The medical practitioner has a key role in ensuring timely dental care for this population. In addition, general dental practitioners should encourage recall of elderly people and be willing to assess dependent residents in residential homes.

Table 2. Comparison with estimates based on reported findings of other New Zealand and Australian studies of older people

	Type of population					
	Institutionalised		Community-dwelling		Australian	
Centre	Christchurch	Manawatu[15]	Dunedin[20]	Mosgiel[19]	Melbourne[21]	Adelaide[22]
Year	2003	1989	1987	1992	2001	2002
Number of participants	210	359	272	815	175	224
Age in years (average)	84.6	82.5	72.4	76.7	83.7	83.2
Dentate proportion (%)	**32.4**	**19.5**[a]	24.3	16.1[d]	35.4	36
Mean number of teeth	14	13.3	13	14.4	13.8	13[f]
Mean number of decayed teeth	2.4	4.6	0.5[b]	2.2	0.92[b]	1.9[g]
% of teeth requiring treatment	18.1	28.6	4.4[c]	18[e]	13.6[c]	20
% requiring no periodontal treatment	2.9	0	16.7	7	7.2	
% requiring oral hygiene instructions only	8.9	0	3	11.5	22.8	
% requiring simple scaling and root planing	85.3	92	80.3	81.5	70	
% requiring complex periodontal treatment	2.9	8	0	10.5	0	

[a]P<0.01, chi-squared test, comparison between estimates from the Christchurch study and the Manawatu study; [b]Coronal surfaces only; [c]A small number of teeth may have been counted twice where they required coronal and root restorations; [d]98 dentally examined; [e]Only includes 'simple' restorations; [f]11.9 teeth + 1.1 roots; [g]1.1 teeth + 0.8 roots.

It is often difficult to obtain consent from a dependent elderly person for elective procedures. Obtaining consent for proposed elective treatment from a third party takes time. For example, in this study, although consent was never refused, we were unable to gain consent within 12 weeks for examination of 100 residents (out of a total of 318 eligible residents). In addition, standard interview methods for obtaining a history may be impossible. When asked about their last dental visit, 89 residents (42%) could not remember when it was, or they were unable to communicate. For these reasons, it may be difficult and, in some instances, impossible for a dependent elder, who may have impaired cognition, to initiate dental treatment.

Traditionally, the major measurement used in studies of dental caries has been the Decayed, Missing, Filled Teeth index (DMFT). This index was developed for use in children and has been adapted for use with the elderly.[24,25] The DMFT index has an important place in longitudinal studies; however, in prevalence studies of older populations, the DMFT index is not capable of representing the true impact of a lifetime's experience of dental caries, and the consequent cycles of repair and re-repair on both an individual's quality of life, and on providers and funders of health services. In particular, retained tooth roots are frequently excluded from assessments of dental status as they are not accounted for by the DMFT index—these 'stumps' may have a considerable bearing on an individual's quality of life, and require relatively complex procedures for removal.

A similar situation used to exist in the recording of the severity of periodontal disease until the WHO developed the Community Periodontal Index of Treatment Needs (CPITN), which measures the extent of common treatable periodontal conditions, thus allowing for the planning and provision of appropriate healthcare services for individuals and populations.[26]

The use of the CPITN index is now widespread, and was used in this study. We have taken a similar approach in not recording DMFT index scores—but have recorded the presence of carious lesions (a measure of dental neglect) and the need for treatment (a measure of the burden of unmet treatment need).

The many different outcome measures used in Australasian studies of the oral health of the elderly[15,19–22] suggests that there is a need for standardisation of recording oral conditions in this increasingly important area of healthcare of the elderly person.

Only clinical examinations were carried out for this study. It is likely that the amount of disease present has been underestimated, and if radiographs were available, additional carious lesions and periapical pathology would have been detected.

The oral hygiene of the dentate participants in this study was poor. However, there was no statistical association between oral hygiene and the presence of dental caries or periodontal disease. On the other hand, clean teeth in this population may be essential for general health. Dependency for oral care and the number of decayed teeth are predictors for aspiration pneumonia.[27] In addition, elderly people who clean their teeth or dentures only occasionally (or never) are more likely to develop aspiration pneumonia.[27]

In our study, only one-quarter of residents cleaned their own dentures or teeth; the remainder had their teeth cleaned by carers. We believe that personal oral hygiene instruction may not be appropriate for someone with restricted dexterity or with

cognitive disturbance, and it may be more appropriate to provide the carers with strategies and motivation for cleaning dentures and natural teeth.

Soft tissue lesions were not a major problem in this group of elderly people—with most soft tissue lesions being either directly caused by (or associated with) denture use. Many of the soft tissue lesions appeared to involve *Candida* infection, and it is possible that salivary hypofunction (secondary to aging and/or drug use) could be a factor.

In summary, more elderly are retaining teeth than a decade ago—over 50% of those persons retaining teeth have dental caries and require conservative treatment; over 40% require one or more extractions because of dental caries, and about 30% of denture wearers may benefit from treatment.

A report commissioned by the National Health Committee (detailing preventive dental strategies for older people) was published in 1997.[2] The results of our Christchurch study show little evidence of implementation of the recommendations of that report.

There is a need for commitment (at a national level) to care for this vulnerable population. Clear public policy backed by sufficient resources is required. As the children and adolescents of New Zealand already have, the dependent elderly person should have access to affordable, appropriate dental services.

In particular, a national public policy on oral health care for elderly people is necessary to meet the oral health needs of this vulnerable group.

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Lack of association between long-term illness and infectious intestinal disease in New Zealand

Rob Lake, Michael Baker, Carolyn Nicol, and Nick Garrett

Abstract

Aims To investigate whether the increase in notified cases of infectious intestinal disease in New Zealand from 1988 to 2001 has resulted in a concurrent increase in associated secondary illness cases.

Methods National surveillance system data were compared to hospital discharge data.

Results No statistically significant correlation between the number of cases of campylobacteriosis and Guillain-Barré syndrome (GBS) was found. There was no statistically significant correlation between the number of cases of campylobacteriosis, salmonellosis, shigellosis, and any of the categories of reactive arthritis; apart from two correlations with campylobacteriosis: with arthropathy associated with Reiter's disease and nonspecific urethritis (Pearson correlation R^2=0.69; p<0.02) and unspecified infective arthritis (Pearson correlation R^2=0.75; p<0.008). The later category is likely to include cases of both infective and non-infective aetiology.

Conclusion In New Zealand, infectious intestinal diseases are not making a significant contribution to the burden of hospitalisation for reactive arthritis or GBS.

From 1988 to 2002, New Zealand experienced a marked increase in the number of notified cases of infectious intestinal disease, especially campylobacteriosis.[1] It has been estimated that 2–3% of infectious intestinal disease cases develop a variety of secondary long-term illnesses.[2] The most recognised of these are haemolytic uraemic syndrome (HUS) after infection with Shiga-like toxin producing *Escherichia coli,* reactive arthritis, and Guillain-Barré syndrome (GBS).[3]

Reactive arthritis is associated with preceding infection by a number of organisms causing intestinal disease—including *Campylobacter*, *Salmonella*, *Shigella,* and *Yersinia*; as well as genital infections. An analysis of reactive arthritis in Otago from 1986 to 1993 suggested that enteric infections were the predominant cause, with *Yersinia enterocolitica* being the commonly isolated organism.[4]

Campylobacter is the most common antecedent pathogen for GBS[3], and the attributable risk of GBS for laboratory confirmed cases of *Campylobacter jejuni* infection has been estimated as 30.1 per 100,000 cases (95% confidence interval (CI): 13.9–57.8) in a Swedish study.[5] A review of this association estimated that 30–40% of GBS cases had suffered *Campylobacter jejuni* infection prior to the onset of GBS.[6]

As part of a study of the number of cases of food-borne intestinal disease in New Zealand[7], we analysed communicable disease notification (ESR, EpiSurv) and hospital discharge data (New Zealand Health Information Service) for infectious intestinal disease and associated secondary long-term illness cases. This study did not consider trends in disease incidence. The number of cases of HUS has increased from

an average of 9 a year from 1990 to 1995, to 23 a year from 1996 to 2002—and much of this increase may be attributable to the rising incidence of Shiga-like toxin producing *Escherichia coli* infection in New Zealand.[8]

In this paper, we consider whether the increase in notified cases of infectious intestinal disease was associated with increases in the number of admissions for GBS and reactive arthritis. The period between prodromal infection and onset of GBS symptoms has been estimated as up to 3 weeks,[9] while reactive arthritis symptoms begin approximately 7 to 30 days after intestinal illness[3]. Therefore the incidence of secondary illness should follow closely any changes in infectious intestinal disease.

Methods

Notification data were obtained from surveillance reports.[10] Hospital admissions were obtained from the New Zealand Health Information Service for all cases, with one of the codes of interest recorded in the first nine diagnosis fields. The conditions of interest were GBS (357.0), arthropathy associated with Reiter's disease and nonspecific urethritis (711.1), postdysenteric arthropathy (711.3), unspecified infective arthritis (711.9), and other inflammatory spondylopathies (720.8). All readmissions were excluded over the entire time period. Hospitalisations were categorised based on the first diagnosis code that contained one of the above codes.

Results

Table 1 shows the number of notified cases of enteric disease, together with the number of hospital admissions coded to relevant arthritis types and GBS, for the 15-year period from 1988 to 2002.

These data suggest only a weak association between the rise in campylobacteriosis and the incidence of GBS (Pearson correlation R^2=0.40; p=0.14; for 1988 to 2002).

Penner serotyping based on the heat stable (HS) antigen(s)[11] has been conducted for 1130 *Campylobacter* isolates obtained from human cases in New Zealand between 1996 and 2001. The serotypes identified include almost all of those that have been associated with GBS[12]: HS:1,44 (16% of serotyped isolates); HS:2 (23%); HS:4 complex (15%); HS:5 (0.6%); HS:10 (0.6%); HS:19 (0.8%); HS:23 (8%); HS:35 (1.3%); HS:37 (4%); HS:41 (0.5%).

Serotypes HS:19 and HS:41 have been suggested as being associated with GBS in some parts of the world, and the low proportion of these serotypes amongst isolates from New Zealand could partially explain the lack of correlation between campylobacteriosis notifications and GBS in this country. However, this association is not consistent. Internationally, a wider range of serotypes have been identified amongst isolates from GBS cases.[12]

Table 1. Number of cases of selected infectious intestinal diseases reported in New Zealand 1988 – 2001, and number of discharged patients coded to reactive arthritis and Guillain-Barré syndrome (GBS) for the same period

Disease	**1988**	**1989**	**1990**	**1991**	**1992**	**1993**	**1994**	**1995**	**1996**	**1997**	**1998**	**1999**	**2000**	**2001**	**2002**
Salmonellosis	1128	1860	1619	1244	1239	1340	1522	1334	1141	1177	2069	2077	1796	2417	1870
Shigellosis	145	137	197	152	124	128	185	191	167	117	122	147	115	157	112
Campylobacteriosis	2796	4187	3850	4148	5144	8101	7714	7442	7635	8924	11575	8161	8418	10145	12489
Total	**4069**	**6184**	**5666**	**5544**	**6507**	**9569**	**9421**	**8967**	**8943**	**10218**	**13766**	**10385**	**10329**	**12719**	**14471**
Arthropathy associated with Reiter's disease and nonspecific urethritis (711.1)	1	0	0	3	0	2	2	2	3	4	7	4	0	0	0
Postdysenteric arthropathy (711.3)	0	3	1	1	1	7	2	2	6	1	2	0	0	0	0
Unspecified infective arthritis (711.9)	16	20	22	21	23	21	29	33	30	39	46	299	399	393	384
Other inflammatory spondylopathies (720.8)	3	0	1	1	2	0	1	1	1	3	9	17	7	11	16
GBS (357.0)	99	60	82	75	103	84	87	87	91	109	95	80	71	103	106

There was generally no association between the various enteric diseases (campylobacteriosis, salmonellosis, and shigellosis) and the number of cases coded to post-dysenteric arthropathy, or other potential sequealae codes. An association between campylobacteriosis and unspecified infective arthritis admissions was more marked over the period 1988 to 1998 (Pearson correlation R^2=0.75; p<0.008).

However, cases assigned to this code will be a mixture of both infective arthritis and non-infective arthritis (Dr John O'Donnell, Canterbury District Health Board, personal communication, 2003) and (without examining case details more closely) this association must be considered as suggestive only. A statistically significant association between campylobacteriosis and arthropathy associated with Reiter's disease and nonspecific urethritis (NSU) (Pearson correlation R^2=0.69; p<0.02) was observed for the same period, but this will be complicated by the potential for arthritis in response to NSU as well as enteric infections.

Coding changes (International Classification of Diseases ICD-9-CMA-II to ICD-10-AM) are thought to have caused the significant changes to reported numbers of reactive arthritis cases from 1999 to 2002—so these years were not included in the analysis.

Discussion

This ecological analysis therefore suggests that, in New Zealand in recent years, infectious intestinal diseases are not making a significant contribution to the burden of hospitalisation for reactive arthritis and GBS. If it is correct that 30–40% of GBS cases have an antecedent infection with *Campylobacter jejuni*,[6] then a strong association between the number of cases could have been expected. Using the predicted rate from the Swedish study,[5] and assuming that the unreported to reported campylobacteriosis case ratio is 7.6:1 for New Zealand,[7] then the number of associated GBS cases could have been expected to rise from 7.3 in 1988 (95% CI; 3.3–13.9) to 32.7 in 2002 (95% CI; 14.9–62.0). This increase was not seen.

The coding of reactive arthritis cases makes examination of any association difficult, but the lack of correlation between the number of cases of any of the enteric diseases and those of post dysenteric arthropathy suggests that any link is tenuous.

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Familial Mediterranean Fever: 36 years to diagnosis

Paul Casey, Mark Lane, and Rohan Ameratunga

Familial Mediterranean fever (FMF) is a rare cause of recurrent abdominal pain in New Zealand, but it is more common in the Middle East and Mediterranean regions. With increasing immigration to New Zealand from these areas, it is a diagnosis that must be considered in patients presenting with unexplained recurrent abdominal pain.

Case report

A 44-year-old woman was referred to the Gastroenterology Service at Auckland Hospital with a history of chronic recurrent abdominal pain. She was born in New Zealand but lived in South Africa for 20 years. She subsequently returned to New Zealand.

Her symptoms began in childhood at 8 years of age when she presented to hospital with fever and abdominal pain. On further questioning in New Zealand, she described severe lower abdominal pain that typically occurred 3 to 4 days into menstruation—with fevers and abdominal distension. During several of these painful episodes, she developed a well-demarcated erythematous rash around her upper limbs, chest wall, and legs. Each episode would last approximately 5 days and then resolve spontaneously.

She initially underwent appendicectomy. Subsequently, she was diagnosed with peritonitis, which settled spontaneously. She continued to have recurrent episodes of pain over the years increasing in frequency over the last 6 years. Courses of prednisone (for a presumed autoimmune process) had minimal effect. Investigations were unremarkable except for an elevated white cell count of 13.5 and an elevated ESR of 32 on one occasion. Abdominal X-rays, abdominal and pelvic ultrasound, and laparoscopy were normal with no evidence for endometriosis.

Her sister had similar symptoms but with less frequent and severe attacks. On further questioning, she indicated she was an Ashkenazi Jew.

Once the possibility of a periodic fever syndrome was considered, mutation analysis was performed for the Familial Mediterranean fever gene (MEFV). The test was undertaken at Gene Dx and she was shown to be homozygous for the V726A mutation of MEFV. Her sister also tested positive for the same mutation.

She was commenced on colchicine prophylaxis and there has been a marked improvement in the frequency and severity of her symptoms.

Familial Mediterranean Fever (FMF)

FMF is an autosomal recessive disease. It affects people predominantly from the Mediterranean basin—including Sephardic Jews, Arabs, Turks, Armenians, and (less commonly) Greeks and Italians. Ashkenazi Jews are also affected, although less frequently.

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Clinically, FMF manifests as short attacks of serositis (peritonitis, pleuritis, or arthritis) and fever. The peritonitis often resembles an acute surgical abdomen, and a history of previous exploratory laparotomy is often given. Symptoms last from hours up to 4 days. Ninety percent of patients have their first attack before age 20 years.[1] An erysipelas-like skin rash can be seen in up to 40% of cases.[2]

The frequency of attacks is variable. Defined triggers for attacks are rare, but attacks related to the menstrual cycle are found in about 7% of cases.[3]

During attacks, white cell count and acute phase proteins become elevated, but return to normal between attacks. Diagnosis is based on clinical findings of episodic fever, serositis, and absence of an alternative cause in a person from an at risk population. Although clinical diagnostic criteria have been proposed,[4] mutation analysis of the MEFV is required for definitive diagnosis.[5]

The most important long-term complication in FMF is the development of secondary amyloidosis. Renal involvement with proteinuria and eventually renal failure is the predominant presentation of FMF related amyloidosis. In spite of 36 years of recurrent serositis, our patient did not have clinical evidence of amyloidosis.

The pathophysiology of FMF appears to involve abnormal recruitment and activation of neutrophils at serosal surfaces. This may be a consequence of the action of the gene product of the MEFV gene, pyrin. Pyrin may have a role in dampening neutrophil activation.

The treatment of choice is colchicine. Colchicine is known to impair neutrophil function in gout, and its beneficial effect in FMF is likely to be via the same mechanism. Furthermore, colchicine reduces the frequency and severity of attacks. Colchicine also prevents amyloidosis, and can reverse proteinuria even after amyloidosis is established.[6]

This case illustrates the difficulty in making a diagnosis of FMF in a low prevalence area. Chronic or recurrent abdominal pain is a common problem presenting to medical practitioners. With increased immigration to New Zealand from the Middle East in recent years, this is a disease which should be considered, particularly in patients of Mediterranean or Middle Eastern extraction.

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Complete atrioventricular block in Becker muscular dystrophy

Ramazan Akdemir, Hakan Ozhan, Huseyin Gunduz, Mehmet Yazici, Enver Erbilen, Cihangir Uyan, and Necat Imirzalioglu

Cardiac involvement in Becker muscular dystrophy (BMD) is one of the leading problems during the progression of the disease. Cardiac involvement includes dilated cardiomyopathy, mild-to-moderate mitral regurgitation, cardiac conduction system abnormalities, and various arrhythmias. But complete atrioventricular (AV) block associated with Becker muscular dystrophy (necessitating permanent pacemaker implantation) is rare.

The presented case report is of a patient with Becker muscular dystrophy, whose condition is complicated with complete atrioventricular block and dilated cardiomyopathy.

Case report

A 49-year-old male admitted to our cardiology department complaining of shortness of breath and fainting episodes during the previous 2 weeks. He had muscle weakness during the previous 30 years. When he was 25 years old, he became unable to walk without help, and became wheelchair-bound at 34 years old. Family history revealed a brother with BMD complicated with cardiomyopathy.

On admission, his physical examination revealed a heart rate of 30 beats/minute with irregular rhythm, S3 gallop, and apical 2/6 systolic murmur. Neurological examination showed atrophic proximal muscles in the upper and lower limbs, and pseudo-hypertrophy in the calf muscles. Nerve conduction velocity studies showed normal findings, and an electromyogram revealed myopathic degeneration.

The chest X-ray examination revealed an enlarged cardiac silhouette. The electrocardiogram showed complete AV block with varying heart rate between 30–36 beats/minute (Figure 1). The echocardiogram (ECG) showed findings about dilated cardiomyopathy and left ventricular ejection fraction was calculated as 30% (Teicholz method). Cardiac valvular structures were normal. On Doppler investigation, there was mild-to-moderate mitral regurgitation. Gene analysis (using polymerase chain reaction and Southern blotting analyses) demonstrated that there was deletion of exons 45, 46, and 47 of the dystrophin gene of the patient's DNA (Figure 2).

A permanent cardiac pacemaker (DDDR) was implanted, and medication for heart failure was started—including digoxin, ACE inhibitors, and furosemide. After 6 months, the patient was haemodynamically stable and had no cardiac complaints.

Figure 1. ECG showing complete atrioventricular block

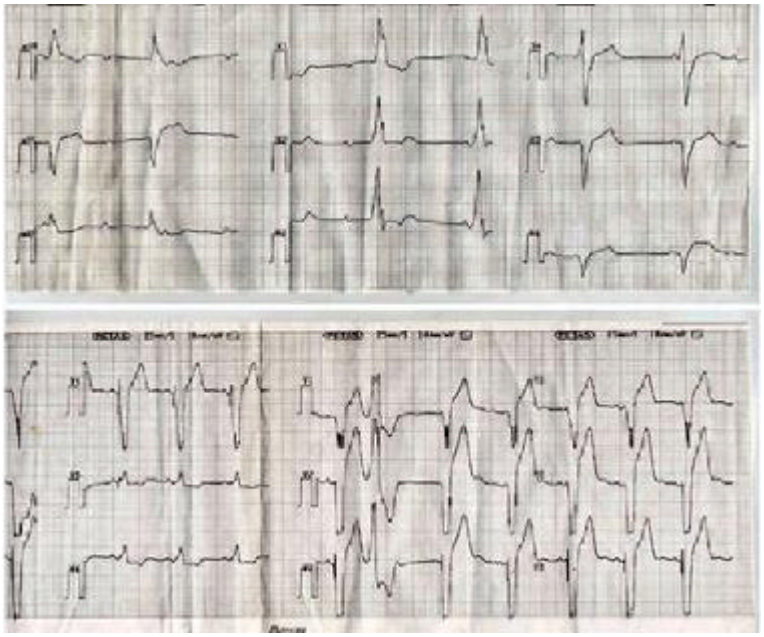
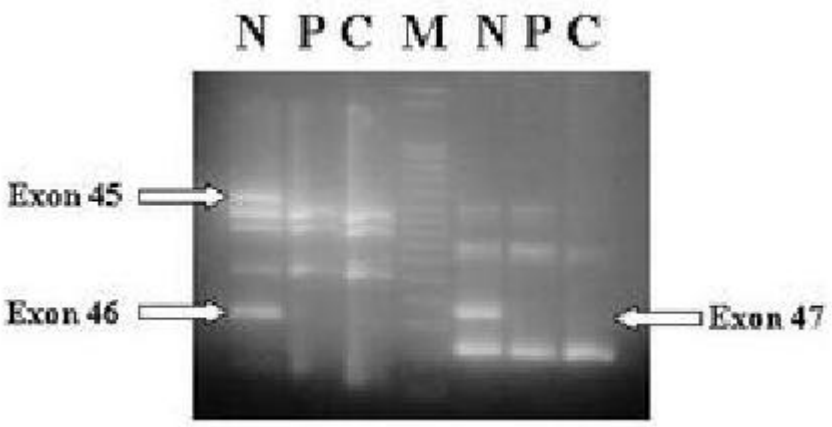


Figure 2. Genetic analysis shows deletions of exons 45, 46, and 47 of the dystrophin gene of the patient's DNA, using the polymerase chain reaction (N: Normal, P: Patient, C: Control patient with deletion)



Discussion

BMD is a congenital muscle disease caused by mutations in the dystrophin gene. Dystrophin gene-related diseases comprises two other entities, Duchenne muscular dystrophy (DMD) and X-linked dilative cardiomyopathy.[1] Although all the three so-called dystrophinopathies result from different types of mutations in the dystrophin gene, there are phenotypic differences between them and the heart is affected to some extent.[2]

In BMD, the degree of cardiac abnormality varies greatly—from completely normal heart tissue to severe rhythm abnormalities, dilated cardiomyopathy, and heart failure leading to sudden cardiac death (due to ongoing replacement of the cardiac tissues by connective tissue).[2]

BMD presents heterogeneously; however, the classical presentation is between 10 and 15 years of age with an abnormal waddling gait. First presentation with cardiovascular symptoms is rare.[2,3] Cardiac involvement in BMD is typically subclinical for long periods during adolescence and early adulthood due to progressive replacement of cardiac myocytes by connective tissue.

Most deaths in BMD result from heart failure or sudden cardiac death.[2–4] Symptoms are more frequent in ambulatory patients, possibly due to the reduced cardiac workload in wheelchair-bound patients.[4] Cardiomyopathy is characterised by early right ventricular involvement, followed by left ventricular reduction in systolic function and dilation.[5]

Left ventricular dilation may begin at any time during the disease course and may be complicated by life-threatening ventricular arrhythmias, which may lead to sudden cardiac death.[5] Cardiac involvement is usually unrelated to the skeletal muscle involvement.[6,7] Overt heart failure is estimated to be a terminal event in about 50% of the cases.

In BMD, frequent ECG abnormalities are sinus tachycardia, atrial fibrillation, intraventricular conduction delay with QRS broadening,[8] and hypertrophy pattern.[9] The typical ECG abnormality is a decreased R wave or prominent Q wave in D1, aVL, and V6.[10] Right or left bundle branch block, T wave abnormalities, and abnormal QS waves are relatively rare.[5] Complete AV block is very rare; indeed, the conduction abnormality as noted in the presented case, has only been reported in one patient.[11]

The most encountered echocardiographic features of BMD patients are myocardial thickening apical thrombi, dilation of right or both ventricles, wall motion abnormalities, secondary valve insufficiency, and reduced systolic function[8, 9]. Severe systolic dysfunction requiring heart transplantation may be even the presenting manifestation of BMD[12]. Another dystrophinopathy that affects the human heart is Duchenne muscular dystrophy (DMD), which is 10 times more frequent than BMD in the general population. It remains subclinical until the teens or late stages of the disease. The absence of cardiac symptoms in the early stages is possibly due to the physical inability. Subclinical cardiac involvement may start between 6–13 years of age, when the skeletal muscle is already evidently involved. Cardiac involvement becomes symptomatic in about 60% of patients.[13]

Classical ECG abnormalities (at the subclinical stage) are sinus tachycardia; tall R waves in V1; increased R/S ratio in V1; deep Q waves in D1, aVL, V5–V6, D2, D3, and aVF; and increased QT dispersion.[14]

Although cardiac involvement (as detected by ECG and echocardiography) occurs in 90% of DMD patients, overt heart failure is a terminal event in only 10–20% of cases.[15] Furthermore, cardiac involvement in DMD does not relate to the severity of the skeletal muscle involvement. Patients with strong muscles are even more likely to die from cardiomyopathy than the patients with weak muscles.[16]

Another dystrophin-related disease (X-linked dilated cardiomyopathy [XLDCM]) typically presents in patients with heart failure in their teens or early 20s. The disease course is rapidly progressive, resulting in heart transplantation or cardiac death from bi-ventricular heart failure within 1–2 years after presentation.[17] Female carriers of XLDCM typically present with atypical chest pain in their middle ages, without having skeletal muscle weakness.[2] Onset of symptoms in manifesting female carriers is usually the 5th decade. Heart failure in female carriers progresses over several years, is slower than in affected males, and is also frequently fatal.[2]

No specific therapy exists for cardiac involvement in BMD as well as other dystrophinopathies.[18] Families of patients with BMD must be screened for evaluation and early detection of cardiac damages. Patients should undergo a first detailed cardiovascular system examination as soon as their diagnosis is established. Patients examined by the cardiologist for the first time with myocardial dysfunction, ECG abnormalities, and CK elevation of unknown cause should be referred to the neurologist.

Systolic dysfunction should be treated with high-dose ACE inhibitors, diuretics, and (in case of tachycardia) beta-blockers in low dosages. Intractable chronic heart failure may necessitate heart transplantation. Patients should be anticoagulated if there is intracardiac thrombus formation and atrial fibrillation. If atrial fibrillation is tachycardious, then digitalis, amiodarone, or beta-blockers should be administered. In case of severe symptomatic bradycardia intractable with drugs and complete AV block, a pacemaker is indicated.[8] The ACC/AHA Guidelines For Cardiac Pacing and Anti-arrhythmic Devices suggests permanent pacemaker implantation as class I indication in patients with muscular dystrophies.[19] If there are symptomatic ventricular tachycardia or fibrillation an automated intracardiac defibrillator may be required to prevent sudden cardiac death. Supraventricular re-entry tachycardia can be treated by radiofrequency catheter ablation.[20]

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Helicobacter pylori: a historical perspective 1983–2003

Alan Fraser

1983—the beginning of a revolution

A short letter to the Lancet in 1983 by Dr Marshall reporting on the culture of a bacterium from the stomach started a revolution in our thinking about upper gastrointestinal disease.[1] This report was viewed with great interest around the world. One of the centres that began research activity very soon after this report was Auckland Hospital. Dr Arthur Morris, a microbiology registrar, teamed up with Dr Gordon Nicholson, gastroenterologist. Dr Morris was keen to fulfil some of Koch's postulates regarding *Helicobacter* and gastritis. He performed a self-ingestion study that has been a widely quoted paper in the area.[2]

The initial establishment of infection was found to require a larger than expected 'dose' of viable bacterium as well as needing H_2-antagonists to reduce gastric acid. Abdominal pain and nausea occurred within a few days but resolved completely after 12 days. The infection proved to be difficult to eradicate. Antibiotics that had been shown to be effective in the test-tube caused only temporary suppression of the infection. It wasn't until antibiotic combination treatments become established that the infection was finally eradicated several years later.[3]

Standard triple therapy—an imperfect but useful workhorse

Triple therapy with De-Nol, Metronidazole and tetracycline became the established treatment. This had modest success rates but was poorly tolerated because of side-effects and the large number of tablets that needed to be taken over 2 weeks.[4] Although compliance was an issue, ulcer patients were well motivated to look for a cure for their chronic symptoms.[5]

Moreover, despite the drawbacks of 'standard' triple therapy, a large number of ulcer patients were successfully treated over a 10-year period.[6] Through case-finding, both in primary care and hospital clinics, the cohort of ulcer patients was largely identified and treated.

The use of De-Nol as part of the eradication treatment was a 'rediscovery' of an old drug. A study at Auckland Hospital by Lane and Lee had shown that De-Nol was more effective than H_2-antagonists in preventing duodenal ulcer recurrence. The reason for this observation was not apparent until the discovery of *H.pylori*.[7]

Bismuth compounds are highly bacteriocidal to *H. pylori*. Scanning electron microscopy studies at the University of Auckland showed the acute destructive effect of bismuth on the cell wall.[9] Dr Morris was initially treated with 4 weeks of Pepto-Bismol (an old formulation of bismuth). He appeared to have been 'cured' based on normal histology and the absence of *H. pylori* on biopsies taken a week after completing treatment. Gastroscopy performed 1 year after initial ingestion showed a mild chronic inflammatory infiltrate and *H. pylori* was again isolated. Molecular

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comparison showed that this bacterium had identical molecular patterns to the original bacterium.[3] Subsequent studies have confirmed that re-infection in adults is very rare.[8] The re-appearance of *Helicobacter* after apparently successful treatment is nearly always due to recrudescence of the same strain of bacteria.

Epidemiology

The epidemiology of the infection was a puzzle. Dr Morris rapidly developed an ELISA antibody test and was able to demonstrate that IgG antibodies to *Helicobacter* were a valuable tool for epidemiology. It became apparent that *H. pylori* was a chronic infection with positive antibodies persisting throughout life. Early observations were that there was a higher prevalence of antibody to *H. pylori* with older age and a higher prevalence in Maori and Pacific Islanders.[10] The seropositivity for blood donors aged 21–30 years was 24%; for 41–50 years, 53%; and 71–80 years, 68%. The ethnic differences observed in serological studies were extended with endoscopy studies of patients presenting with dyspepsia in Fiji and Tonga (93% and 78%, respectively were positive for *Helicobacter)*.[11,12] There was a high rate of ulcer disease—mostly duodenal ulcer (Fiji 28/42 and Tonga 8/18).

H. pylori is an infection acquired in early childhood. There is a higher risk of infection with lower socioeconomic living conditions. Childhood living conditions have dramatically changed in New Zealand over the last 50–60 years; consequently, acquisition of *H. pylori* in childhood has become much less common. This change in living standards explains the observation that *H. pylori* infection is more common with older age.[13,14] This 'cohort effect' explains why *Helicobacter* infection is gradually becoming less common in our community independent of the effect of antibiotic treatment.

Epidemiological studies have identified some risk factors for early childhood infection. These include lack of hot running water, overcrowding and sharing a bed. Some other potential risk factors that need to be explored further are the pre-mastication of food prior to feeding infants, crowded daycare facilities, and the possible protective effect of breast-feeding. Limited data is available for New Zealand (NZ), but lower socioeconomic status has been confirmed as a risk factor.[15] Serological studies of children are able to inform us of the potential burden of gastroduodenal disease in the future. Recent studies from Christchurch, Dunedin, and Auckland show that about 5% of European children become infected with *H. pylori* by the age of 20 years.[15–17] However in contrast, almost half of Pacific Island children (living in NZ) are infected. These children will have a significant burden of gastroduodenal disease in adult life.[15]

Duodenal ulcer disease appears to be relatively recent consequence of *H. pylori* infection; perhaps only over the last 150 years. However there is good evidence available which shows that *H. pylori* has infected human stomachs for thousands of years. The best data on these trends in peptic ulcer disease comes from hospital admissions for ulcer bleeding or perforation.

The incidence of duodenal ulcers started to increase around 1900 and then peaked in the 1950s. Since then, there has been a gradual decline in the incidence of duodenal ulcers.[14] In New Zealand, endoscopists practicing in areas with a predominantly

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European middle class population now rarely observe duodenal ulcers. The reasons for this remarkable change in pattern of disease are speculative, but one explanation is the trend towards higher levels of acid secretion over the last 150 years, perhaps due to improved nutrition.

When *H. pylori* is acquired in a stomach with low levels of acid, there is a diffuse pattern of inflammation and the potential adverse outcomes are gastric ulcer and gastric cancer. In contrast, when *H. pylori* is acquired in a stomach with high acid secretion, the inflammation is confined to the antrum. This leads to a further increase in acid secretion (because antral inflammation stimulates gastrin that is able to act on the healthy, responsive acid-secreting mucosa in the body of the stomach) and this high level of acid output is crucial for the formation of duodenal ulcers.

Indeed, the last 100 years has been a 'window of opportunity' for *Helicobacter* to cause duodenal ulcers. The continuation of the trend towards higher living standards is causing its gradual demise because of the interruption of transmission during childhood.

One interesting avenue of study is the use of strain types of Helicobacter to determine population migrations patterns. O'Toole et al showed that strains of *Helicobacter* strains from Maori and Pacific Island patients were distinctly different from strains obtained from European patients.[18] The DNA profiles of the Maori and Pacific Island patients were remarkably homogeneous. Worldwide studies of DNA profiles (polymorphisms) have recently shown that the strains types from Maori and Pacific Island patients are very similar to isolates obtained from South East Asian (and to a lesser extent American Indian patients). In this worldwide study, the Polynesian isolates were the most uniform of any group studied, suggesting separate migration over at least 1000 years.[19]

The goal of effective and well-tolerated treatment

Metronidazole resistance became the critical factor associated with failure of standard triple therapy. The resistance rate has been observed to increase from 13% of isolates in 1986 (before eradication treatment was ever given) to around 50% in many centres.[6] Metronidazole resistance is now less of a concern with the establishment of omeprazole, amoxicillin, and clarithromycin (OAC) as first-line treatment. The use of clarithyromycin was the major breakthrough in treatment combinations. Treatment became shorter (7-day duration) and more effective (eradication rates above 90%).[6] The main determinant of treatment failure is now clarithromycin resistance.

Resistance rates have risen from low levels (<2%) to around 10% but may be staying stable at this level. Resistance to clarithromycin is nearly universal after failed treatment with OAC. In addition, repeating OAC has a very low chance of success (less than 10%). The preferred second-line treatment is quadruple therapy—De-Nol one tabs *qid* (four times daily), omeprazole 20mg *bd* (twice daily), tetracycline 500mg *qid*, and metronidazole (daily dose probably needs to be 1200 mg).[6]

If this treatment fails, then the merits of further attempts at eradication needs to be carefully considered. Often it is more appropriate to suggest that no further eradication treatment is attempted. Furthermore, patients with definite ulcer disease

may need to accept 'old-fashioned' maintenance treatment with proton pump inhibitors (PPI) or H_2-antagonist.

The 1990s—a time of widening indications

The availability of effective and well-tolerated treatments shifted attention to indications other than peptic ulcer disease (duodenal and gastric ulcers). Patients with duodenitis, and those with evidence of duodenal scarring but no active ulcer, were also found to have a good symptomatic response—similar to patients with duodenal ulcer disease. They should be considered as part of the duodenal ulcer spectrum or 'ulcer diathesis'. Patients presenting with bleeding peptic ulcer definitely need treatment (if *H. pylori* positive) but they often leave hospital without starting eradication treatment.[20]

The benefit of eradication treatment for patients with dyspepsia but a normal endoscopy (non-ulcer dyspepsia) is debatable. There have been at least two conflicting meta-analysis of available trials. It all depends on which trials you select! The magnitude of any effect is certainly small. Less than 15% of patients achieve symptomatic benefit (that is greater than the effect of placebo).[21,22] Treatment, if given, should be directed at those with upper abdominal discomfort improved by meals (ulcer-type dyspepsia). Patients with heartburn alone are unlikely to gain any symptomatic improvement.

Uninvestigated dyspepsia is a different issue from 'non-ulcer dyspepsia'. The 'test and treat' approach is a valid and successful way of managing dyspepsia in patients under 50 years in areas where the prevalence of *H. pylori* is over 25–30%.[23–25] Some patients will have underlying peptic ulcer disease and they will be effectively treated without the need for endoscopy. The proportion of patients with dyspepsia and positive *H. pylori* tests and who have an underlying peptic ulcer has been debated. Studies from South Auckland showed the proportion to be as high as 40% but the proportion could be much lower in other areas.[26]

There is some literature suggesting that *H. pylori* causes a variety of non-gastrointestinal problems; However, none of these claims have been validated in the long-term. One of the most difficult contentions to prove (or disprove) is the association of *H. pylori* infection with coronary heart disease. There is probably no biological association, but there have been some positive studies probably because of similar confounders (such as socioeconomic status).[27,28]

1995 onwards—are there some good *Helicobacter* species that should be preserved?

The possible negative association between *H. pylori* and gastro-oesophageal reflux (GORD), as well as reports of aggravation of reflux oesophagitis after eradication treatment, gave some caution to the widespread treatment of *H. pylori* in primary care. There is no doubt that GORD is becoming more common in our community and that this change has happened at the time when the prevalence of *H. pylori* is decreasing. This is probably not 'cause and effect'. Indeed, recent studies have argued against any contention that *H. pylori* eradication aggravates reflux.[29,30] Another controversy is the potential benefit of eradication of *H. pylori* in patients taking long-term proton pump

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inhibitors (PPIs). Atrophic gastritis appears to develop at a faster rate in *H. pylori*-infected patients taking long-term PPIs.[31]

For some time, it will not be known whether this is an important association, but it cannot be considered good for patients to develop atrophic gastritis (as this is known to be the first step in a progression towards gastric cancer). Therefore, eradication treatment should be given to all patients who are likely to require long-term treatment with proton pump inhibitors.

Helicobacter and gastric cancer

Epidemiological studies have consistently shown a 2–3 fold increased risk of gastric cancer with *H. pylori* infection.[32] Gastric cancer is generally believed to be a multi-step progression from chronic gastritis to atrophic gastritis, intestinal metaplasia to dysplasia, and subsequently to cancer. Intestinal metaplasia is more common, and occurs at an earlier age in *H. pylori*-infected individuals.

Furthermore, intestinal metaplasia is more common, and more extensive, in ethnic groups at higher risk of gastric cancer (eg, Maori and Pacific Islanders).[33] Several large studies of gastric cancer rates after *H. pylori* eradication are in progress but 5–10 years of follow-up will be required before any meaningful results are obtained.[34] *H. pylori* eradication could be a cost-effective public health strategy if large population studies show a reduction in rates of gastric cancer. Much attention has focused on an extended Maori family with familial gastric cancer related to an E-cadherin mutation.[35] This important and ground-breaking research finding but should not detract from the effect of *H. pylori* in causing the high rates of gastric cancer in Maori and Pacific Island men.

Indeed, most estimates would attribute 50% of the gastric cancer risk to *H. pylori* infection.[36] Therefore, eradication of this infection will always be the most easily altered risk factor for gastric cancer.

Helicobacter and the NSAID controversy

If *H. pylori* causes some gastric ulcers and non-steroidal anti-inflammatory drugs (NSAIDs) cause others, then surely both factors together would be worse? However, this simple notion has proved difficult to prove or disprove—with many conflicting results. An important meta-analysis has helped to resolve some issues. *H. pylori*-infected NSAID users are 60 times more likely to have a peptic ulcer than non-infected non-users. Furthermore, *H. pylori* increases the peptic ulcer risk in NSAID-users by three fold. The risk of gastric bleeding is also greater with *H. pylori* infection[37] To ‘test and treat’ for *H. pylori* infection (prior to initiation of NSAID) is a proven strategy that has yet to be become popular.[38]

The last 20 years has seen an explosion in our knowledge of *Helicobacter pylori.* Over the years there have been many areas of controversy but most of the areas of debate have been resolved. It is now possible to have evidence-based practice for the management of this infection.

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Note on the suprapubic operation for enlarged prostate, in two stages

This extract was taken from an article by Dr T. Hope Lewis, Auckland, published in the New Zealand Medical Journal Vol 3 (12), p426–8

Following on the lines laid down by that master of English surgery, the late Professor Greig Smith, where he writes of operations in stages, “For, after all, the safest surgery is the best” (Vol. ii., page 637), which refers to resection of the bowel, I determined that such a procedure would suit certain cases of enlarged prostate.

Every practitioner, if he casts his mind back a little, will remember one or more old men to whom he has been called to pass a catheter to relieve a generally very much distended bladder with weak walls, and where there has been some difficulty in getting through the prostatic portion of the urethra.

You try, perhaps, a variety of catheters, and then with a caudee or a large curved No. 12 you manage to get in, and find the eye jammed with mucus. Finally you empty the bladder and some bleeding ensues. These are cases, left rather long in sending, in which, after the preliminary treatment described, you get feverishness and a further inability to pass urine, rendering catheterization still necessary, and where washing out the bladder is a farce.

These old men are septic and debilitated from the start. They have not had an undisturbed night’s rest for months. Their days have been days of straining. They have suffered much pain and apprehension of impending inability to pass urine at all. They are often very old and feeble, and they are not good subjects for a suprapubic prostatectomy. My ideal for these cases is a deliberate operation in two stages. You may pass a catheter when summoned in the emergency, but next day, or as soon as possible, the bladder should be opened suprapubically.

A warm bed and general surgical cleanliness, with suitable feeding, comprises the treatment for the next week or ten days. The wearied and exhausted old man of eleven days back will, with good nursing, be bright and comfortable, and ready for an enucleation, which can now be done with greater ease than before. The suprapubic wound allows an easy entrance of the finger, and after the mucous membrane over the most prominent part of the prostate is torn through the operation is completed in the usual way.

I well remember my old master, Sir William Saviny, shaking a catheter at his surgical class and exclaiming that such instruments had caused more deaths than the actions in the Crimea. And I am sure that many lives of old prostatics go to swell that list from the trauma caused by difficult catheterization.

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Proceedings of the Waikato Clinical School Research Seminar, Thursday 25 March 2004

Is tuberculosis transmitted to staff in Waikato Hospital? S Burcher[1], Noel Karalus[2], S Holmes[2]. [1]University of Auckland, Auckland; [2]Waikato Hospital, Hamilton.

Aim To assess the risk of transmission of tuberculosis to staff working in Waikato Hospital, and the workload involved in monitoring this, based on the hypothesis that there is no transmission.

Methods The medical records of patients admitted to Waikato Hospital with pulmonary tuberculosis between 1998 and 2002 were reviewed. The level of staff contact tracing performed by the Public Health Unit was assessed, and the surveillance of staff working in the 'monitored unit' was examined.

Results Thirty-seven pulmonary tuberculosis patients were admitted to Waikato Hospital between 1998 and 2002. This lead to the investigation of 142 staff for mantoux conversion. There were 10 mantoux conversions. Two staff were prescribed chemoprophylaxis for latent infection while none was treated for active disease. Between 2002 and 2003, three staff in the monitored unit demonstrated mantoux conversions.

Conclusions The transmission of TB to staff at Waikato Hospital is not a major problem. No staff developed disease though some are infected. While the measures in place for preventing tuberculosis transmission within the hospital are adequate, a cautious approach is taken to staff contact tracing resulting in a high workload for those involved. The initial testing of all staff contacts within 21 days of exposure would provide the best chance of demonstrating a mantoux conversion. The 'monitored unit' model is a pragmatic approach to tuberculosis control in staff. This audit has allowed us to make recommendations regarding contact tracing of exposed staff.

Women with low sexual desire—responses to pictorial and semantic tasks and questionnaires. H M Conaglen[1], J V Conaglen[2], B Hedge[3]. [1]The Psychology Centre, Hamilton; [2]Waikato Hospital, Hamilton; [3]Psychology Department, University of Waikato, Hamilton.

Aim This study compared persons presenting with low sexual desire problems with volunteers from a non-clinical sample with respect to responding to pictorial and semantic sexual and non-sexual cues. In addition, a comparison of questionnaire responses sought to clarify whether assumptions underpinning current therapeutic approaches are realistic.

Method In this study, participants completed mood, sexual anxiety, sexual desire and body-esteem questionnaires and carried out information processing tasks, including picture rating and recognition tasks, and timed responding to semantic stimuli; this replicates aspects of earlier work but with a group of women reporting low sexual desire.

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Results The two groups of women did not differ significantly with respect to their rating of pictures, their recognition time for previously viewed pictures or for the extent of sexual content induced delay. The tasks with the semantic stimuli were completed more slowly by the low desire women, and they also rated sexual words as less familiar than the contrast women. The questionnaire responses were significantly different for sex anxiety, $t(40)=4.52$, $p<0.0001$; both sexual desire measures, $t(40)=$ -12.01, $p<0.0001$ and $t(40)=-6.35$, $p<0.0001$; and the sex attractiveness sub-scale of the body esteem scale $t(40)=-2.97$, $p=0.005$.

Discussion This study has shown that previous findings in a non-clinical sample cannot necessarily be generalised to clinical groups. The study samples were small, and a larger study may have yielded findings more in line with those found in the previous work. However the study also served to confirm the reliability of the sexual desire measures in discriminating between the two groups of women, and reinforced the understanding of the strong association between anxiety and desire problems. These findings together with the qualitative interview data further the theoretical understanding of factors that may influence some women's problematic levels of sexual desire.

Exploring satisfaction and worries of Maori people with diabetes in the Waikato region. J Haar (Ngati Maniapoto/Ngati Mahuta), D Simmons, S Lillis, J Swan. Waikato Clinical School, University of Auckland, Hamilton.

A study of Maori people who live with diabetes in the Waikato region was conducted to explore satisfaction with care, and worries about their diabetes. From 553 responses, satisfaction was higher if blood glucose was monitored ($t=2.753$, $p<0.01$), or treatment included lipid lowering medication ($t=2.640$, $p<0.01$). Respondents with complications were less satisfied with their care ($t=-4.344$, $p<0.001$) and more worried about their diabetes ($t=6.980$, $p<0.001$). No differences were found by treatment with antihypertensive medication, oral antihyperglycaemic medication, or insulin; or by smoking or gender.

Using a Maori Tikanga (customs/beliefs) structured thematic analysis, respondent worries were categorised into four major themes. 1.*Rongo* (experience/information) - history of whanau (family) and friends dying "I have seen the extremes of diabetes and I don't want to die like my sister and brother did", or having complications. Limb amputations were often mentioned. 2. *Whanau* (extended family) - respondents didn't want to die and leave their whanau (e.g. children, grandchildren) "I need to take better care of myself for me and my children", or have their whanau developing diabetes (through inherited bad habits). 3. *Kai* (food/eat) - kai holds a central role in Maori society. It is often the heart of family gatherings. Consequently, the ability to control type and volume of kai is a major concern "I find great difficulty always having to avoid…kai (food) which as a Maori I miss". This leads to worries about weight. 4. *Makatu* (fear/afraid) - this theme was dominated by the fear of losing limbs and loss of eyesight, as well as the fear of having to inject insulin. A common response was "I worry because I may lose a limb or worse". In conclusion, to provide the best care for

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Maori people who live with diabetes, it is important that consideration be given to these cultural dimensions, particularly with regard to whanau.

Child and adolescent obesity: A qualitative exploration of its assessment, management and treatment by practitioners in the Waikato region. J Howarth, B Hedge. Psychology Department, University of Waikato, Hamilton.

Child and adolescent obesity is a rapidly rising epidemic in New Zealand. This is a matter for concern as children and adolescents who are obese are likely to continue to be so through to adulthood, and obesity can be associated with a wide range of medical, social, and psychological difficulties. This study, complimentary to a questionnaire study, aimed to explore the current strategies used, as well as the obstacles faced, by health professionals in the Waikato region in the assessment and treatment of child and adolescent obesity. General practitioners, public health nurses, paediatricians, and dieticians who practise in the Waikato were invited to participate in semi-structured interviews exploring these issues. Nine interviews were conducted, audio taped and transcribed. Thematic analysis, based on a framework model, was used to explore and interpret interview content.

Identified themes related to: 1. the presentation of child and adolescent obesity; 2. the impact of culture, ethnicity, socioeconomics and education; 3. assessment; 4. barriers to effective treatment; 5. causes; 6. elements of treatment programmes; 7. successful aspects of treatment programmes; 8. nationwide strategies to reduce child and adolescent obesity. The implications of these findings will be discussed in relation to the treatment of obese and overweight children and adolescents in the Waikato.

Does percutaneous endoscopic gastrostomy influence the course of gastroesophageal reflux? E M I Kim, U Samarakkody, R Richmond, S Brown. Department of Paediatric Surgery, Waikato Hospital, Hamilton.

Purpose Percutaneous endoscopic gastrostomy (PEG) has been widely used for children with feeding difficulties and low caloric intake. There are controversial data in the literature reporting causation and development of gastroesophageal reflux (GER), subsequent to PEG. The aim of this prospective study, commenced in the year 2001, is to analyse the influence of PEG on GER.

Methods Ambulatory 24-hour esophageal pH monitoring was performed on the patients before PEG and subsequently at the time of Mickey button placement, which was approximately 3 months after PEG. Boix-Ochoa score was used for the analysis. The medical records of all patients were reviewed to record GER related symptoms and signs before and after PEG. The site of PEG was recorded and photographed endoscopically, in order to analyse the other possible contributing factors in the development of GER after PEG.

Results The complete results of twenty patients were available. Unpredictable changes were evident in the number of acid refluxes, the number of long acid refluxes, the duration of the longest acid reflux (min), and the fraction of time pH below 4.00 (%) before and after PEG. The Boix-Ochoa scores improved in most

children with few exceptions. One patient subsequently required a Nissen fundoplication.

Conclusion Our results suggest that PEG does not precipitate or exacerbate GER. Anti-reflux surgery is not necessary with PEG placement, even if there is evidence of GER. The pH monitoring prior to PEG can be used to screen the patients who may require anti-reflux surgery if they become symptomatic.

Child and adolescent obesity: assessment, management and treatment by practitioners in the Waikato region. J M McClintock, B Hedge. Psychology Department, University of Waikato, Hamilton.

The rapidly rising prevalence of obesity amongst children is of particular concern in New Zealand because children who are obese are more likely to be obese into adolescence and adulthood. This rising prevalence is likely to be associated with a corresponding increase in obesity related disorders; problems for health service practitioners to contend with in the future. Based on research conducted in the United States, the purpose of this study was to identify current assessment, management, and treatment strategies for child obesity used by health professionals in the Waikato region. 250 questionnaires were sent out to general practitioners and child health specialists in the Waikato region. 56 participants returned fully completed questionnaires.

Although the results indicate that child health practitioners are particularly concerned with childhood obesity, very few of the practitioners follow published guidelines for the medical, laboratory, and family evaluations of obesity. A greater number of participants carry out appropriate psychological and behavioural assessments. Lack of patient motivation and support services were the most heavily endorsed barriers to intervention. These findings suggest the need for increased training in the appropriate recognition, assessment and initiation of treatment for childhood obesity.

The use of ultrasound to detect position and patency of endotracheal tubes. B Manikkam[1], J Sleigh[2], H Round[3]. [1]University of Auckland; [2]Anaesthetics Department, Waikato Hospital, Hamilton; [3]University of Waikato, Hamilton.

Endotracheal tubes may be incorrectly placed in the oesophagus or beyond the carina, or may become kinked or blocked with mucus. This occurs particularly in the neonate and comorbidities include cerebral hypoxia and death. We attempted analysis of ultrasonic echoes, to quantify and locate blockage, and determine ETT position. The use of piezoelectric transducers was abandoned as a result of lengthy ringing and therefore poor temporal and spatial resolution. Manufacture of capacitive / electrostatic transducers was attempted to allow determination of transducer characteristics although with little success. Polaroid electrostatic transducers produced preliminary results.

After digital signal processing including filtering, we were able to quantify larger blockages to within 10% and determine its position to within 5mm of their actual values. Refinement of these methods will involve better means of directing sound

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from these large transducers into such small tubes. Alternately on may develop small transducers, which fit into the ETT, that are capable of delivering a single unipolar pulse. The development of a small ultrasonic device would offer a cheap, fast and reliable method of determining ETT position and patency. This could eliminate the need for, potentially hazardous, routine suctioning and facilitate prompt resolution of situations in which ETT position and patency is uncertain.

The availability and efficacy of written information addressing obesity in Hamilton. J Roach, B Hedge. Psychology Department, University of Waikato, Hamilton.

Child and adolescent obesity is reaching epidemic proportions in New Zealand. One way to affect change is by using written information. However, large discrepancies exist between the reading levels required to understand many information pamphlets and the reading levels of average people. In addition, people are only likely to attend to those health messages that are presented in attractive packages.

The aims of the study were to investigate the availability in Hamilton pamphlets targeting obesity. For those pamphlets targeting childhood obesity, their efficacy in providing good obesity related health education was assessed. Thirteen venues were investigated for the availability of pamphlets that targeted obesity.

Available pamphlets were evaluated with respect to their aims and target audience, content and general message. For those targeting childhood obesity prevention (or treatment), presentation, format, content and readability was evaluated.

Pamphlets were obtained from 13 sites. Of the eight pamphlets that targeted child and adolescent obesity, one targeted weight reduction, and seven targeted the maintenance of healthy weight. Using an 80% criterion for acceptability, only two pamphlets reached 80% for presentation, none of the pamphlets reached the 80% criterion for format, and two of the eight pamphlets were readable by 80% of the population. The best ‘all round’ pamphlet was rated 80% for presentation and was readable by 75% of the population.

High-quality written information concerning childhood and adolescent obesity is difficult to access in Hamilton. This suggests that there is an opportunity for enhancing child healthcare through the development and distribution of more effective pamphlets that target obesity.

Epidemiology of Meckel’s diverticulum in the Central North Island of New Zealand. R P Sakalkale, U Samarakkody, N Noor-Mohd, P Newman, S Brown. Waikato Hospital, Hamilton.

Purpose The epidemiology of Meckel’s diverticulum (M.D.) in children in New Zealand has not been reported in the literature. We studied the children referred to us from the Central North Island of (C.N.I.) New Zealand between 1997-2003 and who had their Meckel’s diverticulum resected.

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Methodology Hospital medical records were reviewed (including pathological database) of children who had their M.D. resected. Pathology was correlated with clinical presentation. Census data for the region was used to determine the annualized incidence.

Results A total of 19 children (13 boys, 6 girls, M: F=2.1:1, 3 patients <2 years old) were identified. Their ages ranged from 4 months to 13 years (mean age: 8.2 years). The annualised incidence rate was 1.89% for the region. Seven (36.8%) presented with rectal bleeding and in these Tc99m-pertechnetate scan was positive. In the remaining 12 patients (63.2%), findings were, acute inflammation 2, volvulus alone 3, volvulus with gangrene or perforation 3, intussusception, 1 and entirely normal 3. Presentation-wise, there was no difference between the younger (<2 years) and older (>2 years) children. On histopathology, (heterotopic) epithelia were gastric 7, duodenal 1, pancreatic 3, colonic 1 and highly necrotic in 2. On a mean postoperative follow-up of 3.8 years, all have been asymptomatic.

Conclusions The Central North Island of New Zealand can be expected to have around 3 children per year with an M.D. Age at presentation is much higher than reported elsewhere. Uncommon types of epithelia are detected in many cases.

Differing perceptions of barriers to diabetes care among medical, nursing and other health staff in secondary health services in the Waikato. D Simmons, J Haar, S Lillis, J Swan. Waikato Clinical School, University of Auckland, Hamilton.

Diabetes continues to cause premature death and disability in spite of a range of effective interventions. We have undertaken a postal survey among medical, dietetic and senior nursing staff involved in the management of patients with diabetes asking perceptions of ways to improve care, issues preventing quality care and concerns about diabetes care. Initial surveys were followed up with repeated contact. Overall, 171 staff were identified, of whom 64/100 (64%) medical and 57/71 (80%) other staff responded. The number of comments (of variable length) ranged from 1-18 per respondent and 1053 comments were provided. Doctors gave 7.3 comments each and others 10.3 comments.

Comments were given one or more of 34 “barrier” codes based upon prior validated work (1) using triangulation. The major perceived barriers overall were patients knowledge of diabetes (67.8%), patient’s motivation/denial (66.1%), staffing levels/appointment systems (66.1%), unsatisfactory diabetes care/education in the past (65.3%), patients readiness to change (58.5%) and inadequate information management including professional education, coordination and audit (52.5%).

Doctors (vs others) were significantly more likely to report that obesity was an issue (21.9 vs 5.6%, p=0.012), while doctors were significantly less likely to report 10 barriers including personal finance (28.1 vs 51.9%, p=0.008), lack of community based services (26.6 vs 51.9%, p=0.005), lack of family support (4.7 vs 22.2%, p=.004) and the unsupportive macroenvironment (26.6 vs 59.3%, p<0.001). Twenty one respondents were employed by the diabetes services and were more likely to comment (vs others) on priority setting (28.6 vs 1.0%, p<0.001), time (38.1 vs 11.3%,

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p=0.007) and emotional responses such as fear (19.0 vs 4.1%, *p*=0.047) as barriers. There were no differences in perceptions of cross-cultural issues (33.1%) as barriers. Similar numbers (35.6%) indicated concern about the size of the diabetes epidemic.

We conclude that major differences in perceptions of barriers to care exist and these may influence service planning. The survey has also generated a large number of suggestions about how to improve services and these are under consideration. Surveys of patients and those in primary care are underway.

1. Simmons D, Weblemoe T, Voyle J, Prichard A, Leakehe L, Gatland B. Personal barriers to diabetes care: Lessons from a multiethnic community in New Zealand. Diab Med. 1998;15:958–64.

Is a routine serial ultrasound examination necessary after an initial negative complete lower limb ultrasound study to exclude DVT? R Subramaniam, K Cox, R Heath, R Allen, G Davis. Department of Radiology and Haematology, Waikato Hospital, Hamilton.

Objective To determine whether routine serial ultrasound imaging is necessary to safely exclude DVT or its complications in patients with an initial negative complete lower limb ultrasound study.

Methods 320 patients presented to the Emergency Department with suspected DVT were recruited prospectively from June 2002 to May 2003. A complete lower limb ultrasound examination was used as the gold standard to diagnose DVT. The main sonographic criterian for diagnosis of DVT was a noncompressible vein.13 variables were collected before the ultrasound examination. Simplify D dimmer and 'D-dimer plus' D dimer were done in all patients. All patients were followed up for 3 months for detecting any suspected evidence of recurrent DVT or Pulmonary embolism (PE).

Results A total of 68 (21.25%) patients were found to have DVT. 252 patients (78.75%) had an initial negative lower limb ultrasound study. Of those who had an initial negative study, 28 (11%) patients re-presented to Emergency Department with various presenting complaints within the 3-month follow-up period. Among these patients, 10 of them had lower limb ultrasound for the purpose of diagnosis or exclusion of DVT and one had a CTPA and Ultrasound for diagnosis or exclusion of PE / DVT. None of these 11 patients had evidence of thromboembolism. The other 252 patients with an initial negative study had no suspected episode of thromboembolism or re admission to Emergency Department. The specificity and negative predictive value of a complete lower limb ultrasound is 99.8% (95% CI 98.2%-100%) to exclude clinically significant DVT.

Conclusions A negative complete lower limb ultrasound study is a safe examination and routine serial ultrasound is unnecessary to exclude clinically significant DVT.

Screening for deep venous thrombosis using digital photoplethysmography following hip or knee arthroplasty: T Swift, P Jones. Surgical Unit, QE Health, Rotorua.

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Objective Deep vein thrombosis occurs more frequently in joint arthroplasty patients than inmost other surgical groups. The purpose of this study was to evaluate the effectiveness of Digital Photoplethysmography (D-PPG) as a screening tool for DVT in this population group.

Method A prospective study of 50 patients who had undergone elective hip or knee joint replacement. Each patient was assessed by duplex ultrasound (the gold standard) and D-PPG between the 3rd and 5th postoperative day. Patients received routine DVT prophylaxis. Analysis was by 2x2 table and Chi-squared statistic for goodness of fit.

Results 6% of patients (n=3) were found to have DVT as demonstrated by duplex ultra sound. Using a refill time of 21 seconds as the optimal cut off point D-PPG achieved 100% sensitivity and negative predictive value, specificity of 32% and positive predictive value of 8.6%. Using a cutoff point of 10 seconds sensitivity and NPV remained at 100% and specificity increased to 76%, PPV 21%.

Conclusion Using published protocols for refill time cut off point D-PPG is not a useful screening tool for DVT in postoperative joint replacement patients. Varying the refill time cut off point improves the test performance but the very low rate of DVT in this patient group precludes a definite conclusion.

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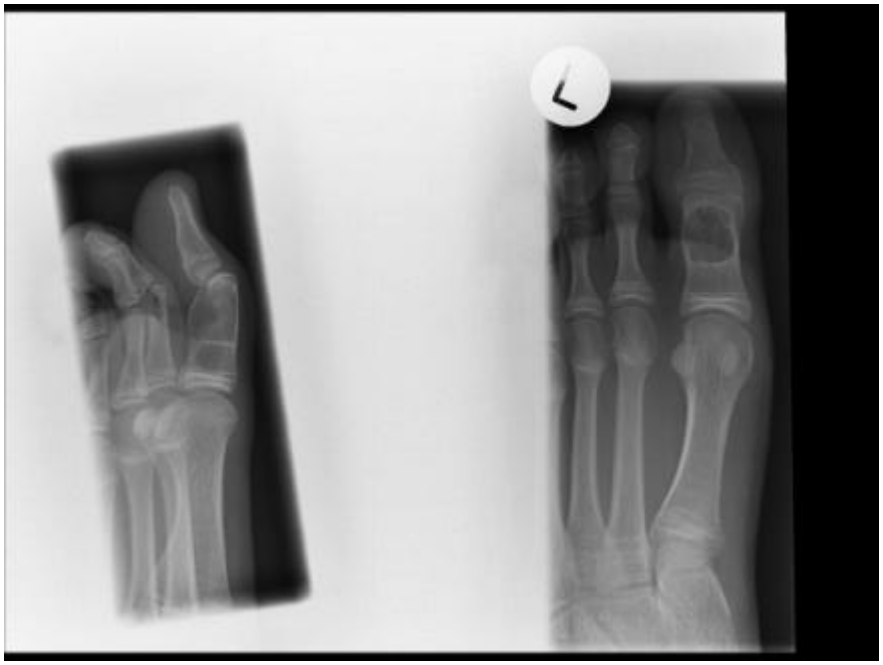
Dactylalgia

A 35-year-old woman presented with pain localised to the great toe. She was referred for anteroposterior (AP) and lateral radiographs. See Figure 1 below.

Figure 1



What is the diagnosis?

See below (next page) for the answer.

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Diagnosis:

Figure 2

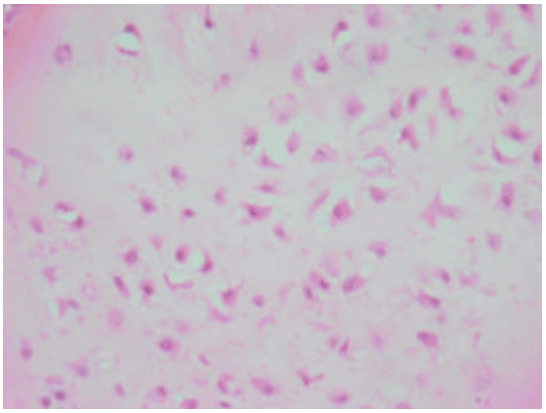
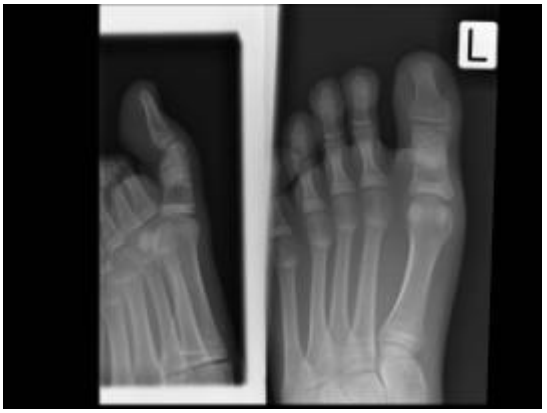


Figure 3



The radiographs show a simple cystic lesion in the diaphysis of the proximal phalanx. No calcification can be seen and there is no pathological fracture. The appearances are of an enchrondroma, but the differential diagnosis includes aneurysmal bone cyst. The lesion was curetted; and Figure 2 is a histological slide showing plump chondrocytes with bland nuclei and occasional binucleated forms, without mitoses or necrosis.

The enchondroma was packed with bone chips, as shown on Figure 3, to prevent pathological fracture.

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Better late than never

The EU is to withdraw its massive subsidies to tobacco growers following a bitter battle among agricultural ministers in Brussels. The withdrawal of payments for what is the most subsidised crop in Europe reflects unease about helping tobacco farmers while EU states campaign against smoking. The UK, which pays US$155m of the US$1.4bn annual subsidy, was among a group of northern European states that demanded an end to the payment. The EU has 1,000 tobacco growers and is the world's fifth largest tobacco producer, with 75% of its crop being grown in Greece and Italy.

Smoking kills an estimated 500,000 Europeans a year, yet EU farmers are paid US$9,274 a hectare to grow tobacco. Wheat farmers receive $US424 a hectare. In the UK alone the health service spends US$2.6bn a year treating people with smoking-related diseases. The government spends around US$53m on anti-smoking education campaigns and another US$70m helping people to stop smoking.

Guardian Weekly (UK) 29/4/04–5/5/04 p4

Asthma and aspirin

Aspirin induced asthma is characterised by the onset of asthma 30 minutes to three hours after the ingestion of aspirin. Affected patients are cross sensitive to all non-steroidal anti-inflammatory drugs (NSAIDs) that inhibit cyclo-oxygenase (COX) enzymes, but seldom cross sensitive to paracetamol. The prevalence of aspirin induced asthma is higher than previously suggested. A recent systematic review found that the prevalence of aspirin induced asthma was higher when determined by oral provocation testing than verbal history (21% *v* 3% in adults, 5% *v* 2% in children).

Most patients were sensitive to NSAIDs, but sensitivity to paracetamol (7% of patients) was more likely in patients highly sensitive to aspirin. Since aspirin and other anti-inflammatory drugs are often self prescribed, patients with asthma should be alerted to the possibility of cross reaction. Simple, standardised warnings on packs of aspirin and NSAIDs may be indicated.

BMJ 2004:328:434–7

Europe and herbal medicines

Europe is set to tighten its regulation of traditional herbal medicines. European manufacturers will have to register supplements in the same way as mainstream pharmaceuticals, according to a directive adopted by the European Union on 11 March. If manufacturers can prove their product has been in use for at least 30 years before they apply for registration, including 15 years in Europe, then they will not have to perform safety and efficacy trials. However, they will still have to submit documentary evidence from the authoritative source that their product is safe and has a history of traditional use.

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The information will be passed to a newly created European Committee for Herbal Medicinal Products, which can ask for more data if necessary. Once the authenticity of a generic product has been established, the committee will add it to a "positive list" of traditional supplements. The directive will also force manufacturers to report adverse reactions to their products. National regulatory bodies, such as the UK's Medicines and Healthcare Products Regulatory Agency also actively scan for reports of adverse reactions.

And the EU has gone further than the US in banning some products, such as the anti-anxiety supplement kava kava, which causes liver damage. The sale of ephedra has not been banned, but it is severely restricted.

New Scientist 10 April 2004

Clozapine improves dyskinesias Parkinson disease

Lovodopa-inducted dyskinesias are a disabling side effect of long-term levodopa therapy in Parkinson disease. Dyskinesias, which occur at the time of maximal clinical improvement, can be reduced by decreasing and spreading the daily doses of levodopa. However, a change in the levodopa regimen often leads to the emergence of motor fluctuations in the form of "on-off" phenomena.

Clozapine, an antipsychotic used in managing schizophrenia, could help treat dyskinesias resulting from long-term levodopa therapy in patients with severe Parkinson's disease, according to a study in *Neurology*. 50 patients participated in the 10-week clinical trial, using a diary to record duration and intensity of fluctuations in motor performance. The results suggested that low-dose clozapine substantially reduces dyskinesia, a serious side-effect of levodopa therapy that is normally difficult to treat, said the researchers.

Neurology 2004;62:381–8

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University of Otago Faculty of Medicine Freemasons Postgraduate Fellowships in Paediatrics and Child Health

The above Fellowships or Scholarships are open to University graduates who intend long term to pursue work in Paediatrics or Child Health within New Zealand. The Fellowships include full-time salary for one year with provision for a further year.

Applications close on **30 June 2004** with the Secretary to the Faculty of Medicine,
University of Otago Medical School,
P O Box 913,
Dunedin,
from whom further details may be obtained.

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The Royal Australasian College *of* Physicians

Adult Medicine Division

Call for Applications for

Foundation Fellowship of the Australasian Chapter of Sexual Health Medicine

The RACP has formed the Australasian Chapter of Sexual Health Medicine within the Adult Medicine Division. Foundation Fellowship will be available to experienced registered medical practitioners who practice in Sexual Health Medicine in Australia and New Zealand.

Those applying for admission will be considered on the basis of the following criteria:

1. Fellowship of the Australasian College of Sexual Health Physicians (FACSHP);
2. Broad experience in all aspects of clinical Sexual Health Medicine;
3. Ongoing contribution to Public Health policy development in the control of sexually transmitted infections on a population basis in Australasia or overseas;
4. Full-time academic position in Health Sciences relevant to Sexual Health Medicine at senior lecturer level or above;
5. Evidence of clinical training in Sexual Health Medicine;
6. Attainment of academic qualifications in Sexual Health Medicine;
7. Evidence of participation in Continuing Medical Education and Quality Improvement in the field of Sexual Health Medicine;
8. Evidence of contributions to the field of Sexual Health Medicine by:

- participation in research in the field with appropriate supervision and collaboration
- development of professional or academic activity
- regular contributions to undergraduate/postgraduate education; and/or
- publications in scientific journals and/or contributions to scientific meetings.

For specific details concerning eligibility, please refer to the detailed criteria in the *Guidelines for Determining the Eligibility of Candidates for Foundation Fellowship* in the Application Package.

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Applicants must demonstrate a satisfactory practice history (no professional misconduct, or disciplinary issues).

Foundation Fellows will participate in ongoing professional activity in the field of Sexual Health Medicine and are strongly encouraged to supervise trainees and participate in a Maintenance of Professional Standards (MOPS) Program. Payment of the annual subscription for Fellows is a requirement of the Chapter. Continued Fellowship is conditional upon a satisfactory practice history.

Application Process

Application Packages may be downloaded from the RACP Website at http://www.racp.edu.au/public/sexualhealth.htm

or obtained from:

Australasian Chapter of Sexual Health Medicine
Telephone: **+61 (0)2 9382 7457**
Email: sexualhealthmed@racp.edu.au

Closing date for applications: Wednesday 28 July 2004

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While You're Away: New Zealand Nurses at War 1899–1948

Anna Rogers. Published by Auckland University Press, 2003. ISBN 1-86940-301-0. Contains 360 pages. Price $39.99

This book concentrates on the vital work done by nurses in war zones from 1899–1948. The author has skilfully used personal material from letters, diaries, and oral histories; as well as from official papers, unpublished theses, and books. Her ready pen has woven a compelling story of how it is to serve the wounded and dying in a war zone. Of added interest are the many photographs, and verses penned by nurses, volunteer aides (VADs), and members of the Women's Army Auxiliary Corps (WAACs), when they were overseas.

I found it hard to put this book down, so well are the stories told. The author clearly shows why these nurses were revered by the troops. At times, the nurses faced overwhelming numbers of casualties, shortage of supplies, insufficient staff, and some illness—yet they coped. Their courage (especially when close to the firing line), resilience, skill, and devotion to duty is documented throughout the book. Also mentioned are factors that are still relevant today: recognition of status and unequal pay—the sisters (charge nurses) earned less than the untrained medical orderlies.

A recommended read for anyone who would understand what war means to those people closest to it.

Alice Silverson
Archivist and retired nurse
Medical History Trust
Christchurch