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This Issue in the Journal

A cluster of three cases of leptospirosis in dairy farm workers in New Zealand
Margot McLean, Quentin Ruscoe, Terence Kline, Caleb King, Annette Nesdale

Leptospirosis is a serious infectious disease that is spread from animals to humans. In New Zealand, farm workers and meat workers have the highest risk of becoming infected. This paper describes a cluster of three cases of leptospirosis in dairy farm workers. We describe the illness experienced by the three cases and discuss how leptospirosis rates in New Zealand can be reduced.

Chronic pain in New Zealand: a community sample
Nicola Swain, Matthew Johnson

It is well known that around 20% of New Zealanders live with some kind of long-term pain. This paper asked people who have this type of pain what their experience of it was like. A lot of people have pain in more than one body region. The pain has lots of different causes, including nearly a quarter of people who aren’t sure what caused it. An interesting finding in this paper is that accepting pain, rather than fighting it, is correlated with lower pain, less disability, depression anxiety and higher employment.

An assessment of an outcome of injury questionnaire using a Pacific model of health and wellbeing
Radilaite Delaibatiki Cammock, Sarah Derrett, Faafetai Sopoaga

Pacific peoples in New Zealand have poorer health outcomes and tend to be highly represented in high risk occupations. Pacific models of health can help in addressing Pacific peoples’ needs during injury and through recovery. Our study assessed the questionnaire of a study of injured New Zealanders (Prospective outcomes of injury study) using a Pacific model of health (Fonofale model). We found that the study questionnaire included questions covering all the elements of the Pacific model however was limited in questions regarding family, culture and spirituality. We suggest future researchers consider using Pacific models of health when undertaking research among Pacific peoples.
Incidence of maternal sensitisation to Rh(D) in Christchurch, New Zealand and reasons for prophylaxis failures
Krishna G Badami, Johanna Parker, Aoife Kenny, Sue Warrington

In terms of their blood, people can be D-negative or D-positive. D-negative mothers carrying a D-positive baby may make anti-D that can destroy the baby’s D-positive red blood cells. In D-negative women this can be prevented by giving anti-D injections. The anti-D prophylaxis regimen used in New Zealand covers many but not all situations. Therefore, though the risk in New Zealand of D-negative mothers making anti-D and their babies being potentially affected is low, it may be lower still with the addition of the routine prophylaxis regimen used in many other countries.

Postgraduation retention of medical students from Otago and Auckland medical programmes
William Shelker, Phillippa Poole, Warwick Bagg, Ian Wood, Paul Glue

By 10 years after graduating, about a third of NZ medical graduates leave NZ to work overseas. Different methods are used for selecting medical students into the Auckland and Otago medical programmes. We followed up medical students for up to 13 years after graduation to see if these different selection methods influenced the proportion of doctors remaining in NZ. We found similar retention of Auckland (74.9%) and Otago (73.6%) medical school graduates. Medical graduate retention is not influenced by different student selection methods.

Molecular epidemiology of group A streptococcus from pharyngeal isolates in Auckland, New Zealand, 2013
Deborah A Williamson, Nicole J Moreland, Philip Carter, Arlo Upton, Julie Morgan, Thomas Proft, Diana Lennon, Michael G Baker, Rod Dunbar, John D Fraser

Our study provides information on the circulating group A streptococcal strains in Auckland. Future clinical and molecular surveillance of these strains is essential in the context of ongoing vaccine development.
Leptospirosis is an important multi-species zoonotic disease in New Zealand

Chris Mansell, Jackie Benschop

Mclean and colleagues’ paper in this issue of the New Zealand Medical Journal\(^1\) reports an outbreak of leptospirosis on a dairy farm and illustrates issues in identifying and controlling animal sources. It highlights the practical difficulties in definitively diagnosing each individual case and also shows how increased awareness leads to recognition of additional, milder cases.

Leptospirosis is a globally important zoonotic disease, caused by the pathogenic spirochetes of the genus \textit{Leptospira}. The traditional serological classification, based on agglutinating antigens, classifies \textit{Leptospira} into 20 serogroups and over 300 serovars.\(^2\) Six serovars are endemic in New Zealand: Hardjobovis, Pomona, Ballum, Tarassovi, Copenhageni and Balcanica\(^3\) with the first three of these responsible for the majority of human cases. Apparent correlations between serovars or species and syndromes or severity of human disease are confounded by geographical distribution and other factors.\(^4\)

\textit{Leptospira} serovars have lower pathogenicity for maintenance than for accidental or spill-over hosts, while being similarly infectious. The consequence is that maintenance hosts remain infected and a host-pathogen equilibrium is established by a continued re-/infection cycle balanced by a marginal immune response.\(^5\)

The epidemiological pattern that predominates in New Zealand is occupational, due to domestic species being important maintenance hosts. A 2010 survey of 237 New Zealand farms found serological evidence of exposure to Hardjobovis and/or Pomona in over 50\% of adult sheep, 58\% of adult beef cattle and in 34\% of adult deer.\(^6\)

Therefore, workers other than dairy farmers are also at risk of infection. In a review of 97 notified cases from the Waikato region, dry stock farmers were the occupational group with the highest rates of leptospirosis and dairy farmers formed the second largest group.\(^7\)

In 2010, serovar Ballum emerged as the most frequently notified serovar in human cases\(^8\) and this emergence was coincident with a rise in the proportion of low risk occupations and the decline in affected meat workers.

Traditional maintenance hosts for Ballum are the mouse, black rat and hedgehog.\(^3\) Thus, as pointed out by McLean and her co-authors, an integrated programme that includes vermin control, avoidance of urine splash, use of personal protective equipment and vaccination of domestic species is required.

Infection commences with a leptospiraemic phase of a few days duration. Conjunctival suffusion is common. The second, “immune phase” then supervenes, when antibodies are present; leptospires are cleared from the blood and excreted in urine. These phases are not always distinct.
Classical clinical features include jaundice and renal failure (Weil’s disease) and, in a few cases, pulmonary haemorrhage. Leptospires can be found in the CSF during the leptospiraeemic phase. Meningitis is more common in young adults and children and manifests during the immune phase. Other complications can include myocarditis and uveitis.

Pathology is characterised by vasculitis rather than disseminated intravascular coagulation and, unlike in other spirochetal diseases, chronic stages of infection are not recognised.4

Reinfection can occur with different serovars. In animals, infection with non-maintenance serovars can cause clinical disease and subclinical losses. For example, Pomona infection causing sudden death in lamb flocks associated with high rainfall and surface flooding are reported.9,10

Human leptospirosis is uncommon and sporadic, but awareness among rural clinicians and patients is generally good. Diagnosis is erratic because culture and serology are slow and susceptible to early antibiotic treatment, so of limited assistance to the clinician. PCR shows promise11 and we need to better understand which specimens are most informative at each time and the effect of antibiotics.

Serum, CSF or urine may be tested and positive results may be found at unexpected phases of the illness (A. Werno, Canterbury Health Laboratories, Personal Communication, 22 Jan 2014). Serology (microscopic agglutination test, using live leptospires) is the reference standard, but requires paired samples.

Pre-existing titres are not uncommon in rural people, so a rise in titre is sought. Often, it takes 4 to 6 weeks for the acute infecting serovar to become well defined and even then immune reactivity may be blunted by effective antibiotic treatment. In 2013, the Waikato Hospital laboratory tested 612 patients by serology but only 16% had a convalescent serum submitted. Presumably, a large proportion of true cases are not being followed up and the diagnosis is never confirmed, nor included in surveillance notifications.

Preferred oral treatment is either doxycycline or amoxicillin, while ceftriaxone and penicillin may be used intravenously. It is straightforward to cover the possibility of leptospirosis in both general practice and in the hospital setting but, perhaps, at the cost of increased overall antibiotic use if this is not done selectively.

Accurate diagnosis is needed to focus antibiotic use, to elucidate the epidemiology and for ACC purposes. Leptospirosis is an occupational disease in Schedule 2 of the Accident Compensation Act 2001. ACC’s current standard for a diagnosis of leptospirosis is that of a clinically compatible illness and at least one of the following laboratory results:

- Isolation of leptospires from a clinical specimen.
- Detection of leptospiral nucleic acid from a clinical specimen.
- A four-fold or greater rise in leptospiral microscopic agglutination titre (MAT) between acute and convalescent sera.
- Single high antibody titre of ≥400 in the MAT.
Protection of dairy and meat workers against leptospirosis has focussed on the use of personal protective equipment, such as aprons, goggles, face masks and gloves. However there are issues with compliance, as these make routine work more difficult.\textsuperscript{12}

In the cross-sectional study of 567 meat workers in New Zealand, in which seroprevalence was 10.9\%,\textsuperscript{6} the strongest risk factor among sheep and deer meat workers was working prior to hide removal on the slaughter chain.

Vaccination, advocated by McLean and her co-authors, is feasible in New Zealand because the predominant serovars found in human cases are few and these are maintained in domestic species. However there are many practical and logistical considerations; in New Zealand it is only dairy cattle and pigs that are routinely vaccinated.

Approximately 10\% of beef herds and 9\% of deer herds practice vaccination for leptospirosis and vaccination of sheep is a rarity. Vaccination of deer in sub-clinically infected herds has been shown to improve reproductive and growth outcomes and to be cost-effective for farmers\textsuperscript{13} and this is currently under investigation in sheep flocks and beef breeding herds.

Antibiotic prophylaxis often used elsewhere in the world, during predictable periods of increased disease risk, for example in active outbreaks or in endemic areas post-flooding. A recent Cochrane review reported that the use of weekly oral doxycycline (200 mg) increases the odds for nausea and vomiting with unclear benefit in reducing \textit{Leptospira} seroconversion or clinical consequences of infection.\textsuperscript{14}

Research is key in informing control of human leptospirosis. Leptospirosis has complex interdependence on the interaction between the environment, domestic animals, humans and wildlife and requires a collaborative “one-health” approach.

This editorial and the paper by McLean et al exemplify the synergy of human and animal health clinicians and academics working in clinical practice, district health boards, universities, and laboratories. The current favourable economic situation for dairy, beef and sheep industries and rising expectations in occupational health and safety should support investment in research, diagnostic and control measures.

\textbf{Competing interests:} Nil

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Preventing death and injury to child motor vehicle passengers: achieving best practice requires simplifying restraint choice for parents and setting best practice as the societal norm

Lynne E Bilston

As noted in the viewpoint article by Kool et al in this issue of the NZMJ, injury remains the leading cause of death and disability for children beyond the perinatal period, with motor vehicle trauma accounting for a large proportion of the burden of disease.

While there is broad consensus internationally that the risk of serious injury to children in car crashes can be substantially reduced by the correct use of a size-appropriate child restraint, many children continue to travel in the wrong restraint for their size, and many restraints are used incorrectly, greatly reducing their effectiveness. The key to helping parents and carers to correctly use the right restraint for their child on every trip is making it as simple as possible to follow best practice and changing the societal norms to be ‘best practice’.

While raising the age for mandatory child restraint use is a step in the right direction, and will likely improve child restraint practices as it has done in Australia, choosing the 7th birthday as the upper age limit sends the message to parents that as soon as the child reaches their 7th birthday, their child can stop using their booster seat even though they are not big enough to get good belt fit in an adult seatbelt and are thus at higher risk of injury in a crash when using a seatbelt.

As noted by Kool et al, since 2009 European law has required use of boosters up to the age of 12 years—demonstrating that more stringent legislation is practical. It also redefines best practice as the expected ‘norm’. Situations in which this is difficult to achieve in practice can be managed as exceptions to the general rule, in much the same way as children who are taller than 135–145 cm (depending on the country) are allowed to use seatbelts in Europe before 12 years of age. Similar approaches are used in Australia to allow for exemptions to the rear seating law for families with more young children than rear seating positions.

Having the law require booster use up to age 12 also removes the general public perception that seatbelt use for children aged 7–12 is as safe as using a booster seat “because the law allows it” – a sentiment that is widely held by parents. Both New Zealand and Australia could learn from Europe’s example.

Another particular challenge in New Zealand is that it is difficult for parents to know when to transition their child from one restraint to the next, because these transitions are not defined clearly in the new law, and can be inconsistent between different restraints. This is difficult in any country due to the variation in the size of children at a given age, but this problem is greatly exacerbated in New Zealand because restraints
Certified to both US and European standards are accepted in addition to the joint Australian/New Zealand Standard, AS/NZS 1754. In contrast, only the joint Australian/New Zealand standard is accepted in Australia, and the child restraint law specifies a minimum age for use of each restraint type (e.g. 6 months for a forward-facing restraint, 4 years for a booster seat).

While acceptance of restraints from all over the world certified to multiple standards appears to offer more choice to New Zealand families, these potential benefits are more than outweighed by the complexity of choosing the ‘best’ restraint for a child, because different standards use different selection criteria (weight, age, height, shoulder height).

Particularly confusing is that a child who fits one type of restraint under one system may not fit a similar type of restraint designed to another standard. For example, a three year old child may be in the weight range for a US certified booster seat but not be tall enough to fit in an AZ/NZS 1754 certified one. This encourages premature graduation to a booster seat via choice of the US restraint, while evidence shows the child is much safer staying in their forward-facing restraint than in a booster seat at this age.

A good example of how to make restraint selection easy for parents is the new shoulder height marker system used on restraints certified under the Australian/New Zealand standard since 2010. These labels are placed on the restraint indicating the minimum and maximum shoulder heights for a child to fit properly in that restraint.

These labels make it clear at a glance when a child fits into a particular restraint, because their shoulders must be between the two labels on the restraint. It discourages premature graduation because if a child is too small for the next restraint category, their shoulders will be below the minimum height label. This is a much simpler and more reliable way of choosing a restraint than relying on the parent knowing the child’s weight or height (many parents either do not know or their estimates are wildly inaccurate), and gives better fit of the child in a restraint, and therefore better protection in a crash.

Another major safety concern is the continuing approval of child safety harnesses (or “H-harnesses”) for use by children under 7 in New Zealand (www.nzta.govt.nz/traffic/students-parents/child-restraints.html), as research has shown that they can be particularly dangerous in crashes, and that they offer no benefit over lap-sash seatbelts.

Indeed the latest Australian/New Zealand Standard restricts their use to lap only seatbelts together with a booster seat that can stop the harness pulling up in a crash, allowing a child to slide under it. Children under 7 should not use harnesses without a booster seat. The recently published Australian National Guidelines for Child Restraint Use, which were approved by Australia’s National Health and Medical Council in 2013, recommend against their use.

The other critical aspect of child passenger safety is using restraints correctly. Incorrect use of restraints also substantially increases injury risk, by up to 7 times. Child restraint checking and fitting schemes have been shown to significantly reduce misuse, and the recommendation that parents make use of these schemes by the New Zealand Paediatric Society is valuable.
Newer restraint installation systems, such as LATCH and ISOFIX can also reduce incorrect use. However, this is another area where the acceptance of multiple restraint standards in New Zealand makes parents lives much more difficult and potentially increases the risk of injury for New Zealand children, because different standards have different ways of installing restraints and different requirements for securing the child in the restraint. For example, top tether straps that connect the top of a child restraint to the car are highly effective in reducing injury, but their use on US and European restraints varies with the type of restraint and the type of installation system, while the joint Australian/New Zealand standard requires them for all infant and forward facing restraints, and many booster seats.

Parents therefore know that all restraints require top tethers, rather than having to remember which restraint needs one. This is reflected in the substantially lower rates of top tether misuse seen in Australia than in the USA (~20% vs ~60%). The bottom line is that we need to have a simple, consistent system for child restraint selection and use so parents can easily know what is ‘best practice’ and how to achieve it. This requires design of legislation that sets the societal norms for restraint use to be equal to best practice, while allowing for exceptions as required to make it practical. These laws need to be supported by targeted education and child restraint fitting advice services.

Competing interests: Nil.

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References:


A cluster of three cases of leptospirosis in dairy farm workers in New Zealand

Margot McLean, Quentin Ruscoe, Terence Kline, Caleb King, Annette Nesdale

Abstract

Aims We report a cluster of three cases of leptospirosis on a New Zealand dairy farm, with regard to clinical, laboratory, and environmental findings. The cluster is discussed against the annual incidence of leptospirosis in humans and cattle, and the vaccination of cattle as one means of preventing human cases on farms.

Methods The three cases were investigated by case interview and review of clinical and laboratory information. A site visit was made to the farm to assess environmental risk. Relevant veterinary information relating to the cattle herds was reviewed.

Results Most of the symptoms exhibited by the three patients were consistent with primary phase leptospirosis. Different methods of laboratory diagnosis were used with each case. However, two cases were confirmed as leptospirosis and in both the causative agent was Leptospira borgpetersenii serovar (sv) Hardjo. The third case had a milder illness, received doxycycline early, and was regarded as a ‘probable’ case as there were no confirmatory diagnostic results. All three cases had worked on the same dairy farm during their incubation period, where the highest risk environment was the milking shed and potential exposure to urine splashes from infected cattle. Also there were inadequacies in the herd vaccination programme.

Conclusions There are options for minimising risk to dairy farm workers in New Zealand. No human vaccine exists in this country. Leptospira borgpetersenii serovar (sv) Hardjo (serovar Hardjo) is endemic in New Zealand dairy cattle without causing apparent disease. L. Pomona is a sporadic infection but can cause abortions. A cattle vaccine against these serovars was introduced in New Zealand in 1979, after which there was a general fall in notifications of human cases of leptospirosis. This was attributed to the overall decrease in these two serovars among the livestock population.

Vaccination of farm livestock for leptospirosis is an integral factor in preventing human cases. We note the New Zealand initiative to combine vaccination with a risk management programme operated by veterinarians, called Leptosure®, to reduce the risk of human leptospirosis on dairy farms. The efficacy of using doxycycline as a prophylaxis for preventing human infection in trials is reviewed. Other preventative strategies include the use of personal protective equipment to cover the mouth and nose, eyes and all skin breaks, farm workers and rural clinicians being aware of the signs and symptoms of leptospirosis, and prompt treatment of cases with antibiotics.

Leptospirosis is a zoonotic bacterial disease, caused by spirochetes, that affects humans and many other animal species including livestock. It is spread to humans through urine from infected animals.1,2
Occupational exposure has been identified as a risk factor for *Leptospira* infection.\(^3,4\) This is true of human leptospiral infection in New Zealand, where a 2002 review found the incidence to be highest among meat processing plant workers and second highest among livestock farm workers.\(^5\)

Dairy workers were found to be the livestock workers most frequently represented. In 2012, “farmers or farm workers” was the occupational group with the highest number of cases.\(^6\)

Dairy cattle infected with leptospirosis may experience abortions and a decrease in milk production, both from acute and persistent infections, resulting in a significant loss of income to the farmer.\(^7,8\)

*Leptospira Hardjo* is commonly found in cattle and causes disease in humans. In humans, leptospirosis has four possible presentations. They are:

- A mild influenza-like illness (leptospiral or febrile stage);
- Weil’s syndrome with jaundice, renal failure, haemorrhage and myocarditis with arrhythmias (icteric stage);
- Meningitis/meningoencephalitis; and
- Pulmonary haemorrhage and respiratory failure.

Human leptospirosis is typically a biphasic disease with the symptoms of the first stage being nonspecific. They are similar to influenza symptoms with headaches, high fevers, myalgia (calves and lumbar region), coughing, vomiting, abdominal pain, diarrhoea and photophobia.

Aseptic meningitis occurs in 25% of acute cases. Conjunctival suffusion is observed in about 30% of cases. Mild cases do not progress past the first phase. Moderate and severe infections progress to a secondary phase. The secondary or icteric phase of the disease is known as Weil’s disease. This is a very serious condition with symptoms including jaundice, renal failure, haemorrhage, cardiac arrhythmias, pneumonitis, and haemodynamic collapse and a death rate of 5–15%.\(^9\)

Leptospirosis is a notifiable disease in New Zealand. In 2012, 113 cases of leptospirosis were notified, a rate of 2.5 per 100,000 population, a significant increase from 2011 (1.5 per 100,000, 68 cases).

The highest rates in 2012 were in the Waikato, Hawke’s Bay, and MidCentral District Health Boards.\(^6\) The non-specific presentation of leptospirosis means that diagnosis, and determining a true incidence rate, is difficult.\(^10\)

**Methods**

Three cases of human leptospirosis, from the same dairy farm, were reported by clinicians to the local public health service in August and September 2010. The cases were investigated by case interview, review of clinical and laboratory information, site inspection to assess environmental risks, and review of relevant veterinary information about the cattle herds.

**Results**

**Clinical findings**—In August 2010, two male employees from the dairy farm presented with signs and symptoms of illness with onset 1 day apart. The signs and
symptoms included fever, headache, nausea, vomiting, conjunctival suffusion, photophobia, and dark urine.

The two employees worked in the milking shed at the farm. Both required hospitalisation. Case A for three days and Case B for one day. Case A was treated with intravenous fluids for mild dehydration and discharged on 100 mg doxycycline P.O., BD. Case B received flucloxacillin and acyclovir on admission and was treated with IV ceftriaxone/acyclovir while in the hospital. He was discharged without further treatment.

A third male farm worker, Case C, developed symptoms about three weeks after the other two. His symptoms were milder than those of the other two, but knowing about the other employees’ illness he saw a general practitioner and was prescribed doxycycline and recovered without any further problems.

The majority of the symptoms exhibited by the three patients were consistent with primary phase (leptospiremic) leptospirosis.

**Laboratory findings**—Leptospires have a slow growth rate and low metabolic activity making microbiological diagnosis difficult. A faster laboratory diagnosis can be achieved with serological titres using the Microscopic Agglutination Test (MAT) or identification of leptospiral DNA by Polymerase Chain Reaction (PCR).

Different methods of diagnosis were used for each patient (Table 1). Case A had a PCR on serum which was positive for leptospirosis. An acute serum was not done for this patient but a convalescent serum returned a leptospiral MAT value of 800, indicating a recent infection. The causative agent was determined to be serovar *Hardjo*.

**Table 1. Laboratory tests performed**

<table>
<thead>
<tr>
<th>Case (date onset)</th>
<th>Leptospiral DNA</th>
<th>Isolation of leptospires</th>
<th>Leptospiral screen IgM</th>
<th>Acute serum MAT</th>
<th>Convalescent serum MAT</th>
<th>Diagnosis</th>
<th>Causative agent</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (25/8)</td>
<td>Detected in plasma (30/8)</td>
<td>Not done</td>
<td>Equivocal (30/8)</td>
<td>Not done</td>
<td>800 MAT (28/9)</td>
<td>Recent infection</td>
<td>Serovar <em>Hardjo</em></td>
<td>Confirmed</td>
</tr>
<tr>
<td>B (27/8)</td>
<td>Not detected (1/9)</td>
<td>Not done</td>
<td>Presumptive positive (1/9)</td>
<td>200 MAT (1/9)</td>
<td>1600 MAT (22/9)</td>
<td>Recent infection</td>
<td>Serovar <em>Hardjo</em></td>
<td>Confirmed</td>
</tr>
<tr>
<td>C (19/9)</td>
<td>Not done</td>
<td>Not done</td>
<td>Equivocal IgM (21/9)</td>
<td>Negative</td>
<td>Requested but not done</td>
<td>Not confirmed</td>
<td>Not determined</td>
<td>Probable</td>
</tr>
</tbody>
</table>


Screening test: *Leptospira* IgM EIA by Panbio.

The Microscopic Agglutination test (MAT) testing and Leptospiral cultures were done at the Leptospira Reference Laboratory at The Institute of Environmental Science and Research (ESR), Wallaceville as described in: Guidelines for the control of Leptospirosis. WHO publication no.67 1982 S. Faine ED.

A PCR was performed for Case B but leptospiral DNA was not detected. Both acute and convalescent titres were run. The MAT for the acute serum was 200 and for the convalescent serum was 1600. A fourfold or greater increase in titre was indicative of
a current or very recent infection. The causative agent was again determined to be serovar *Hardjo*.

Case C became ill 3 weeks after his co-workers. His symptoms were not as severe. He did not have photophobia or conjunctival suffusion. He was aware of his co-workers’ illnesses and sought a general practitioner’s care as soon as symptoms developed. He received doxycycline early and his illness was mild. An acute phase leptospiral IgM enzyme-linked immunosorbent assay test (leptospiral screen) gave equivocal results. A convalescent serum MAT was not obtained.

Despite the lack of confirmatory laboratory results Case C was regarded as a “probable” case because of his symptoms and similar environmental exposure to the two co-workers with laboratory-confirmed disease.

**Environmental findings**—All cases worked on the same dairy farm. A health protection officer visited the farm to assess risks. The highest risk environment was assessed to be the milking shed. The three workers reported exposure to urine splashes from cattle.

Boots, gloves and aprons were worn but not face shields. In addition, there were inadequacies in the herd vaccination programme and 16 cattle of unknown vaccination status had been added to the herd the previous month.

This cluster of human illness was referred to the then Department of Labour (Occupational, Safety and Health Service), for further investigation and action to minimise future risks.

**Discussion**

In New Zealand, dairy farm workers are at occupational risk of leptospirosis though exposure to the urine of infected cattle. Options for minimising risk include vaccination of animals, animal chemoprophylaxis to reduce the number of animals shedding leptospires in urine, human chemoprophylaxis in outbreak situations, use of personal protective equipment, and greater awareness of symptoms and the need for early medical attention.

Human vaccines do not provide long-term protection, are very reactive and are not commonly used, although they have been effective in some epidemic situations.\(^11,12\)

No human vaccine is available in New Zealand.

An assessment of New Zealand dairy herds was conducted in 1975, 1976 and 1977. Sixteen herds that had experienced problems with abortions and five without any history of abortions were studied. Seventy-three percent of the animals that had aborted were found to be positive for *L. pomona*. Nineteen percent of the other cows in the same herds were also positive for *L. pomona*. Cattle from both groups were found to be positive for serovar *Hardjo* but it was not found in any of the cattle which had aborted.\(^13\)

A survey of dairy cattle in the Taranaki region of New Zealand in 1979-1980 found that 62% of the cattle were positive for serovar *Hardjo* by MAT. Serovar *Pomona* was only found in 4% of the cattle.\(^14\) It appears that serovar *Hardjo* is endemic in New Zealand dairy cattle without causing apparent disease, while serovar *Pomona* is a sporadic infection that causes pyrexia and abortion in cattle.
Vaccine is commercially available for cattle, although the level of protection provided may depend on the type of vaccine. Vaccines used in the United States contain serovars *Hardjo, Canicola, Pomona, Grippotyphosa*, and *Icterohaemorrhagiae*. Monovalent vaccines against serovar *Hardjo* were found to be more protective in cattle than a pentavalent vaccine.\(^{15}\)

Introduction of a cattle vaccine against serovars *Hardjo* and *Pomona* occurred in New Zealand in 1979. Human cases of leptospirosis in New Zealand dropped from 677 in 1979 to 179 in 1982.\(^{16}\) The decrease in human leptospirosis continued between 1990-1992 and 1996-1998. This was attributed to the overall decrease in serovars *Hardjo* and *Pomona* among the livestock population, although other serovars in wild animals showed an increase in prevalence.\(^5\)

Since 1997 there has been no decline in cases, with the number of notifications fluctuating around 100 cases per year (Figure 1). In 2012, 113 cases of leptospirosis were notified, a rate of 2.5 per 100,000 population, a significant increase from 2011 (1.5 per 100,000, 68 cases).

Of the 80 cases in 2012 with a high-risk occupation recorded, 58 (72.5%) were in farmers or farm-workers.\(^6\) Vaccination of farm livestock for leptospirosis is an essential factor in preventing human cases.

**Figure 1. Leptospirosis notifications and laboratory-reported cases by year, 1997–2012**

![Graph](image-url)

*Source:* Institute of Environmental Science and Research Limited.

In New Zealand the NZ Veterinary Association and the Society of Dairy Cattle Veterinarians have developed Leptosure®, a unique national risk management programme to reduce the risk of human leptospirosis on dairy farms. The farmer and veterinarian work together to design a specific vaccination programme for cattle, at the same time as including best-practice farm management.

Leptospirosis hazards are identified and a risk management programme established that eliminates, isolates, or minimises significant hazards. Monitoring and risk
management continue on an ongoing basis. There is an annual reassessment to ensure compliance with the programme and to maintain the farm’s Protected Leptosure® status.\(^\text{17}\) The programme, operated by veterinarians, also includes control of leptospirosis in other species such as sheep, pigs, deer, goats, and farm dogs.

Doxycycline has been used prophylactically for humans to prevent clinical leptospirosis in outbreaks, with good results.\(^\text{18}\) A randomised control study looking at the use of doxycycline as a leptospirosis prophylaxis found that while it didn’t decrease the infection rate between drug and placebo groups it decreased the number with clinical illness.\(^\text{19}\)

A study in US military troops also supported use of ongoing prophylaxis (200 mg doxycycline PO per week) in a specific high-risk environment.\(^\text{20}\) Whether doxycycline should be used as prophylaxis following exposure to an infected animal is not known.

The use of personal protective equipment, with special attention being given to covering the mouth, nose, eyes and all breaks in the skin, is recommended for all at risk workers.\(^\text{21}\) Meat-processing factories should have written protocols and equipment to minimise risk, as part of an industrial health and safety program.

Individual farmers and farm workers may be at risk because of their inadequate awareness of the risk, variation and compliance with vaccination protocols, and the tendency to vaccinate animals only rather than implement a comprehensive risk management programme.

Vaccination of animals already infected with *Leptospira* does not reduce their shedding of leptospires and consequently does not reduce the risk of exposure to farm personnel.\(^\text{21}\) It is important that dairy farmers and farm workers are well aware of the signs and symptoms of leptospirosis as prompt treatment with antibiotics will reduce the likelihood of severe or fatal illness. It is also important for clinicians working in rural areas to be vigilant for signs and symptoms of leptospirosis, as the disease is probably under-diagnosed.

Laboratory confirmation is also complex. Leptospires are only present in the first few days of the illness and are affected by antibiotic use. A study in Colombia compared microscopic diagnosis with PCR. The MAT and PCR both compared favourably with microbiological culture as means of diagnosis.\(^\text{22}\)

This cluster of illness was followed up with a community meeting, with participation from farmers and farm workers (including the cases), public health and veterinary staff, to raise awareness and discuss issues relating to the events. Subsequent to this event all cows older than two years of age on the farm were treated with parenteral amoxicillin to eliminate persistent leptospiral infection.

In summary this cluster of leptospirosis is likely to have occurred because dairy farm workers, who were not fully protected by personal protective equipment, were exposed to urine splashes from cattle of unknown vaccination status that were added to the herd in the previous month.
Competing interests: Nil.

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Chronic pain in New Zealand: a community sample
Nicola Swain, Matthew Johnson

Abstract

Aim The 2010 New Zealand Chronic Pain Survey aimed to gather information from people who experience chronic pain about their pain, mental health, disabilities, and acceptance of pain.

Methods In December 2010, surveys were distributed in paper or online to GPs, hospitals, pain clinics asking for people with chronic pain to respond. The survey was open for 6 weeks.

Results There were 142 responses to the survey. Most people reported more than one cause of their pain, and pain in more than one site. Although respondents reported a wide range of causes of their pain, most people were unsure of the cause, or endorsed injury or arthritis as the cause. The most common site of pain was the lower back, followed by the pelvis and joints. Levels of disability were high. Pain was significantly correlated with depression and anxiety, and negatively correlated with acceptance.

Conclusion New Zealanders experience chronic pain stemming from multiple origins, with multiple causes. Consistent with international data, the experience of pain causes high levels of mental health issues and disability, but can be modulated by acceptance. Future studies should examine treatment availability and effectiveness.

Chronic or persistent pain can be defined as pain that lasts longer than the usual time of healing. This type of on-going pain is a major health issue, both internationally and in New Zealand.1

In the New Zealand Health Survey 2011/12, 16% of adults reported chronic pain (defined as pain that occurs every day, for at least 6 months).2 Data from the New Zealand Burden of Diseases, Injuries and Risk Factors Study in 2006 note that chronic pain accounts for at least 5% of health loss recorded in the study.3 Health loss measures how much healthy life is lost due to premature death, illness or impairment. This makes chronic pain similar in size to that of anxiety and depression.

In New Zealand, rates of chronic pain are higher for older people, Māori, and people living in deprived areas.1 Internationally; data suggests chronic pain is more prevalent among females and older people.4

A person’s culture can impact not only how they perceive and experience pain but also how they interact with healthcare professionals and adhere to advice provided.5 It is evident that culture plays an important role in determining various aspects of pain experience and response.6 In Māori, cultural factors such as the role of whanau (family) and the importance of the development of relationships with healthcare providers are ways to enhance Māori health.5
Pain which has taken a chronic course is complex in presentation, it often has all aspects of a biopsychosocial condition present and each needs to be examined. It will have usually started with tissue damage but progressed to a pain problem influenced by a wide range of psychosocial factors.

Previous research suggests that those presenting to primary care services also often have more than one site of pain. For example, Raftery et al (2011) reported the mean number of pain sites to be 4.2, with almost 80% of people experiencing pain in more than one site.

Chronic pain is associated with a wide range of disabilities, including mental health issues. Many studies have reported an association between pain and depression. Baune et al (2008) reported that rates of comorbid major depression and chronic pain ranged from 30–54%.

Anxiety is also correlated with chronic pain. In a community sample, in which sociodemographic and medical conditions were controlled for, the odds ratio (OR) of anxiety disorder in chronic pain patients was reported as 2.13; similarly, the OR for depression in chronic pain patients was 2.00. Of those with multiple chronic pains, depression (without anxiety) had an OR of 2.5, and anxiety an OR of 2.3.

A study of over 13,000 community-based participants in New Zealand reports the prevalence of depression with multiple pain sites was 12.6%, compared to 5.4% for those with no pain. Specific anxiety disorders ranged from 5.8–8.3% for those with multiple pain sites compared to 1.6-4.5% for those with no pain.

As well as increasing mental health issues, pain also produces a wide range of other disabilities. For example, Raftery et al (2011) reported that 37% of people who have chronic pain have high levels of disability, defined as: impairment, activity limitations, and participation restrictions.

A study in Portugal found 92% of chronic pain sufferers have some disability related to their pain, most often family/home responsibilities, recreational activities, occupation/work, and sleep/rest. It is also well documented that sleep problems co-occur in up to half of those who experience chronic pain. The interaction is not as simple as pain preventing sleep however, with sleep problems also being likely to contribute to pain.

Although medical models can usually explain the cause of pain (e.g., as an experience that is directly related to a site of injury), given the complex nature of chronic pain, psychological models can be helpful in explaining the experience of chronic pain.

One model, which explains differing experiences of pain, is acceptance. Acceptance of pain can be seen as an alternative to experiential avoidance, that is, not participating in usual activities due to expected pain. Pain patients vary in the ability accept pain, or to let go of the struggle with pain, and be aware of it without attempting to change it.

Higher acceptance levels have been shown to be a reliable and valid predictor of lower levels of pain, disability, depression, anxiety, and also of a better work status. Acceptance has also been found to be positively associated with a shorter duration of pain reported in a New Zealand Tertiary Pain Management Centre.
Chronic pain is a major health issue, both in New Zealand and worldwide. It is prevalent, disabling and has strong associations with depression and anxiety.

The present study aimed to examine the experience of pain in a New Zealand community sample. To date, research has focused on either clinical samples or epidemiological data.

This research aims to gather participants from the community who consider themselves to suffer from chronic pain, and examine how their experiences compare to those reported in epidemiological studies and pain treatment studies.

This type of survey provides a snapshot in the lives of those suffering from chronic pain. Fitting with data regarding chronic pain, it was expected that people would report a variety of causes of pain, and a variety of locations of pain. In addition, it was hypothesized that pain would be related to depression and anxiety, disability and sleep problems, and that pain would be moderated by acceptance.

Method

Recruitment—Participants filled in a survey that was available in both paper and online versions. Paper copies of the survey were sent out to the waiting rooms of Pain Clinics, General Practitioners, Physiotherapists and outpatient clinics in several major cities (Dunedin, Wellington, Auckland and Wanganui).

A link to the online survey was posted on the Endometriosis NZ and Arthritis NZ websites as well as in the Multiple Sclerosis web newsletter, which directed participants to a version of the survey that was available on Google documents.

In both paper and online versions the cover page had “Do you suffer from chronic pain?” as the invitation to complete the survey. The first question was “do you suffer from persistent pain?” and if the respondent said no their survey was discarded.

Survey instrument—The survey consisted of a demographics section, and questions pertaining to pain, mental health, disability, acceptance, treatment and opinions (not all results reported here). The level of pain experienced was measured using the Brief Pain Inventory (BPI). Respondents identified the site of their pain from a list of 14 sites, and were able to nominate additional sites. The listed sites were: neck, lower back, upper back, wrist, head (headache or migraine), joints (e.g. knee, hip or elbow), muscles, feet, chest, stomach, dental, pelvic, skin (burns), amputation (stump or phantom).

There were 12 options that respondents could tick to identify the cause of their pain. The categories were: neuropathic, injury, cancer, amputation, osteoporosis, carpel tunnel syndrome, arthritis, due to surgery, burns, multiple sclerosis, fibromyalgia, and cardiovascular disease. Participants could also choose “I’m not sure” and “other, please list”.

For testing anxiety and depression we used the Hospital Anxiety and Depression scale (HAD). It consists of 14 questions, with responses rated from 0–3, giving a possible score of 0–42, with higher scores representing greater impairments. The short form of the Chronic Pain Acceptance Questionnaire (CPAQ) was used to measure acceptance.

Ethical approval was obtained from the Psychological Medicine Department at the University of Otago.

Results

Responses were collected and entered into a spreadsheet. Analysis was conducted using PASW (version 20) software (formerly SPSS statistics).

Participants—The 142 respondents were 16% men and 84% women. Seventy percent responded online. The average age was 40.6 years (SD=16.5; range 15–78). Ethnicity was: 88% NZ European and 8% Māori.
The responses came from all over the country: Otago (23%), Wellington (23%), Canterbury (11%), Manawatu-Wanganui (11%), Auckland (10%), other (13%). Most participants were married (57%), with 33% being single and 10% widowed or separated.

In terms of occupation, 30% worked fulltime and 16% part time, 14% were homemakers, 8% were students and 14% were retired. More than 1 in 5 (22%) participants were unemployed or on a sickness benefit.

Level of education was as follows; 40% had finished high school or less, 31% had completed some university or a diploma, and 30% had achieved a bachelors, masters or PhD.

**Cause of pain**—Participants with chronic pain were asked what had caused their pain. They were able to endorse more than one response. Table 1 summarises the most common responses, with the top responses being that they were not sure (23%), followed by injury (21%), and arthritis (20%).

Less common causes endorsed by respondents were; osteoporosis, complex regional pain syndrome, cancer, neuralgia, drug side effects, carpal tunnel, burns, pancreatitis, cardiovascular cause, reflux, ulcer, polio, polycystic ovaries, and amputation.

While 45% of respondents endorsed only one cause of their pain, 34% listed two causes and 21% listed three or more causes. The average number of causes per person was 1.8.

**Table 1. Responses to “what is the cause of your pain?” (n=142)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not sure</td>
<td>23(32)</td>
</tr>
<tr>
<td>Injury</td>
<td>21(29)</td>
</tr>
<tr>
<td>Arthritis</td>
<td>20(28)</td>
</tr>
<tr>
<td>Endometriosis</td>
<td>19(27)</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>18(25)</td>
</tr>
<tr>
<td>Surgery</td>
<td>16(23)</td>
</tr>
<tr>
<td>Neuropathic</td>
<td>14(20)</td>
</tr>
<tr>
<td>Irritable bowel</td>
<td>11(14)</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>9(12)</td>
</tr>
<tr>
<td>Other</td>
<td>31(44)</td>
</tr>
</tbody>
</table>

**Site of pain**—Respondents were asked where they felt pain. The most common complaint was pain in the lower back (59%). As shown in Table 2, 49% of participants reported pain in the pelvis or stomach, and 39% reported pain in joints such as knees, elbows or fingers. Similar to the causes of pain, people reported many sites of pain, with 56% reporting three or more pain sites and only 21% reporting a single pain site. The average number of pain sites per person was 3.6.
Table 2. Most common pain locations

<table>
<thead>
<tr>
<th>Pain site</th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower back</td>
<td>59(84)</td>
</tr>
<tr>
<td>Pelvis/abdomen</td>
<td>49(69)</td>
</tr>
<tr>
<td>Joints</td>
<td>39(56)</td>
</tr>
<tr>
<td>Neck</td>
<td>34(48)</td>
</tr>
<tr>
<td>Muscle</td>
<td>31(44)</td>
</tr>
<tr>
<td>Headache</td>
<td>31(44)</td>
</tr>
<tr>
<td>Foot</td>
<td>28(39)</td>
</tr>
<tr>
<td>Upper back</td>
<td>23(32)</td>
</tr>
<tr>
<td>Wrist</td>
<td>12(17)</td>
</tr>
</tbody>
</table>

Intensity of pain—On the VAS scale from 0 (no pain) to 10 (worst pain imaginable), the average pain rating was 5.8 (in general; SE=0.2). On average, participants rated their pain as 7.7 at its worst (SE=0.2), and 3.6 at its least (SE=0.2). When asked what their pain was at the time of completing the survey, the average score was 5.1 (SE=0.2). Pain constancy was rated 7.2, on average (SE=0.2).

Depression and anxiety—Levels of depressive symptoms were high (mean =7.1, SE=0.3). It was found that 32% of people had a HADS-D score of >7, indicating current depression. If a more conservative cut-off of 10 was used (Crawford et al, 2001), 19% of the participants in the current study met the criteria for potentially clinically relevant depression.

Scores for anxiety were high, with the average score of the entire sample (mean=8.5, SE=0.3) being classed as borderline anxiety on the HADS-A. One in three (33%) people were rated as having an anxiety disorder, using a cutoff score of >7, and 29% reported that anxiety was a problem for them, using a direct question.

Pain-related disability—Respondents were asked to endorse any disabilities in addition to mental health problems that they may have experienced in relation to their pain. Disability was high for this group of people, with the majority having trouble walking (76%), sleeping (75%), concentrating (64%) and maintaining relationships (56%) due to their pain (Table 3). Again, most people endorsed multiple disabilities, with the average number (from 10 choices) being 5.

Table 3. Reported disabilities as a result of chronic pain

<table>
<thead>
<tr>
<th>Disability</th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difficulty walking or moving</td>
<td>76(110)</td>
</tr>
<tr>
<td>Inability to sleep</td>
<td>75(108)</td>
</tr>
<tr>
<td>Inability to concentrate</td>
<td>64(91)</td>
</tr>
<tr>
<td>Strained relationships with family/friends</td>
<td>56(79)</td>
</tr>
<tr>
<td>Inability to meet family commitments</td>
<td>42(59)</td>
</tr>
<tr>
<td>Inability to drive</td>
<td>28(39)</td>
</tr>
<tr>
<td>Inability to care for self</td>
<td>25(35)</td>
</tr>
<tr>
<td>Loss of a job or chance of promotion</td>
<td>24(34)</td>
</tr>
<tr>
<td>None of these</td>
<td>0(1)</td>
</tr>
</tbody>
</table>
**Pain acceptance**—To investigate the effect of acceptance on respondents experiences of pain, we divided respondents into quartiles based on CPAQ scores, and related acceptance to mental health issues, pain experiences, and unemployment (see Table 4).

The Table suggests that, in all the variables measured, greater acceptance predicted better outcomes. That is - lower pain, disability, mental health issues and higher employment.

**Table 4. Scores of pain, mental health, and unemployment by acceptance ratings quartiles**

<table>
<thead>
<tr>
<th>Acceptance</th>
<th>Pain in general</th>
<th>Pain right now</th>
<th>Pain constancy</th>
<th>Disability</th>
<th>Depression</th>
<th>Anxiety</th>
<th>Unemployment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (least acceptance)</td>
<td>6.8</td>
<td>6.0</td>
<td>8.0</td>
<td>6.2</td>
<td>9.9</td>
<td>10.9</td>
<td>27%</td>
</tr>
<tr>
<td>2</td>
<td>5.9</td>
<td>5.2</td>
<td>7.2</td>
<td>4.8</td>
<td>8.4</td>
<td>9.1</td>
<td>24%</td>
</tr>
<tr>
<td>3</td>
<td>6.4</td>
<td>5.4</td>
<td>7.3</td>
<td>4.7</td>
<td>5.9</td>
<td>7.5</td>
<td>16%</td>
</tr>
<tr>
<td>4 (most acceptance)</td>
<td>4.2</td>
<td>3.9</td>
<td>6.3</td>
<td>3.6</td>
<td>4.2</td>
<td>6.6</td>
<td>12%</td>
</tr>
</tbody>
</table>

**Pain experienced**—A multiple linear regression was performed using “pain in general” as an outcome. Variables of interest were: being in paid employment, acceptance, and anxiety and depression. Research suggests fixed effects of gender and age so these were also added to the model as covariates. Variables were all added to the model and then removed in a stepwise fashion until all remaining predictors were statistically significant.

The relationship between “pain in general”, the CPAQ score, and the HAD score was statistically significant (F=14.6, p<.01). Pain in general could be predicted by the following equation:

\[
\text{pain in general} = 5.45 - 0.04\text{(CPAQ score)} + 0.07\text{(HAD score)} + 0.04\text{(age)}
\]

This indicates that the experience of pain increased in association with higher depression and anxiety scores, age, and decreased in association with higher acceptance scores.

**Discussion**

A survey of a group of community-based New Zealanders with chronic pain revealed high levels of disability and mental health issues. An examination of these issues are important because the population prevalence of chronic pain is estimated by one study to be 16.9%, which is likely to be an underestimate as many people who have conditions commonly associated with pain (e.g. migraine) do not consider themselves to have chronic pain."
The present results are consistent with previous epidemiological NZ research that showed injury or accident, and health condition to be the two most often attributed reasons people gave for their chronic pain.

The participants’ pain was experienced in multiple areas, consistent with a recent study in Ireland that was conducted with a community sample. Also consistent with the present research, recent research suggests that lower back pain is the leading cause of disability in Australasia. Interestingly, of those reporting back pain in the present research, only one person reported only lower back pain. This supports the argument that chronic pain is a problem with the pain system rather than an issue in the tissues.

As shown in the present study, chronic pain limits New Zealanders in a number of ways. Most participants in the present study reported that their pain made some usual daily activities difficult. This high level of disability is consistent with previous international studies.

As well as the physical disability we might expect with painful conditions, participants who experienced chronic pain also reported high levels of mental health issues. In New Zealand it has been reported that anxiety and depression interact synergistically with arthritis and neck/back pain disorders to increase the odds of reporting chronic pain beyond an additive model.

In the present study 19% met criteria for depression, which is consistent with international research, which showed that 15% of chronic pain patients met criteria for a significant depressive disorder. Rates of anxiety were also high in the present research. Clearly, although causation cannot be inferred, people who experience chronic pain also have significant psychological morbidity.

Given the slice-of-life nature of our survey, we were unable to determine whether mental health problems occurred as a result of chronic pain, or whether these problems were pre-existing and contributed to a person’s experience of pain. Other research suggests in most cases anxiety disorders exist before the onset of pain, and depressive disorders come on after the chronic pain experience.

The present anxiety rates are higher than those that have been reported in a representative community sample, indicating the respondents in this survey of people who consider themselves to have chronic pain may be different from people in the wider community who report having experienced pain in the previous year. This difference may be due to a response bias. That is, people who are considered resilient (have pain but little disability), may not have responded to a survey asking for respondents who “suffer from chronic pain”.

Although the participants may be self-selected, and thus not representative of all chronic pain sufferers, the current participants represent a significant number of pain sufferers, from a range of backgrounds, who are clearly impacted by their pain. It is useful to understand this group’s experiences of pain, as they are likely to represent the group of people who will be seeking support for their pain experiences. Although there was also an uneven gender split of respondents, this is consistent with epidemiological studies showing women experience more chronic pain.
In addition to finding that a number of disabilities were associated with participants’ experiences of pain, we found that acceptance moderated the experience of pain. Unlike depression and anxiety that are positively associated with pain (increase pain), acceptance was negatively associated with pain (reduces pain). This is consistent with previous research examining acceptance which suggests that higher levels of acceptance predict lower levels of pain. This finding supports a recent call to further examine the psychosocial factors which contribute to chronic pain in greater specificity. Blyth et al (2007) suggest we need to examine with more clarity what we mean when examining psychosocial aspects.

The finding that acceptance is associated with pain experienced adds to this dialogue. Acceptance and Commitment Therapy (ACT), a form of cognitive behavioural therapy, may help meet a need for accessible and cost-effective treatments for chronic pain. ACT has a growing evidence base. In New Zealand, level of acceptance of pain have been positively associated with reported duration of pain and negatively associated with total disability.

Recent research shows that chronic pain is a major health issue in New Zealand. The current research has provided a more in-depth picture of a group of chronic pain patients in New Zealand. These people provide a snapshot of their experience, which suggests pain stems from multiple origins, with multiple causes.

Consistent with international data, the experience of pain causes high levels of mental health issues and disability. Interestingly levels of pain can be modulated by acceptance. This community data is consistent with findings from epidemiological and clinical samples.

Further research into the treatment of these chronic pain sufferers would help to form a complete picture of what type of services are available and utilised in New Zealand.

Competing interests: Nil.

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An assessment of an outcome of injury questionnaire using a Pacific model of health and wellbeing

Radilaite Delaibatiki Cammock, Sarah Derrett, Faafetai Sopoaga

Abstract

Aim To use a Pacific model of health to describe relationships between questions within a structured questionnaire developed for a prospective study of injured New Zealand residents’ outcomes and important elements of Pacific people’s health; and identify health issues of particular importance for Pacific peoples that future studies may consider including.

Method The Fonofale model of Pacific health identifies culture, family, physical, spiritual, and ‘other’ elements (e.g. socioeconomic status and service use) as important. In consultation with Pacific researchers, each question from a Prospective Outcomes of Injury Study (POIS) questionnaire was assessed. Relationship between the type and number of POIS questions were considered in relation to each of the Fonofale elements.

Results Two-thirds of the POIS questions were able to be placed within a single element of the Fonofale model; remaining questions were placed into multiple elements. The POIS questionnaire strongly addressed the physical, mental and ‘other’ Fonofale elements. Culture, spirituality and family elements were not strongly addressed.

Conclusions The Fonofale model identified areas of strength in the POIS questionnaire, and areas of limitation. Researchers undertaking population studies or surveys could consider using a Pacific model to help inform structured questionnaire development.

Pacific peoples comprise 6.9% of New Zealand’s total population. Pacific peoples have lower income, higher levels of unemployment, poorer housing and lower life expectancy than other New Zealanders.1,2

Within New Zealand, injury-related mortality and morbidity is also not proportionately distributed. For example, Pacific males suffer on average higher rates of injury resulting in death,3 and Pacific peoples have higher rates of hospitalisation due to injury (2744 per 100,000) compared with the national average (2393 per 100,000).4

Pacific peoples are also highly represented in occupations associated with greater risk of injury.5,6 Additionally, many migrants from Pacific nations to New Zealand support extended families in the islands. Balancing the need to meet responsibilities, cultural traditions and the demands of living in a contemporary New Zealand society is difficult, particularly in times of economic recession.

Given the socioeconomic circumstances and risk of injury, it is important to assess injury and resulting disability among Pacific peoples.
The rate of overall disability reported among Pacific peoples in New Zealand is approximately 11%, with most living in the community. A greater proportion of Pacific peoples (57%) with a disability are aged less than 44 years, compared with European New Zealanders (27%).

Despite these findings, there remains a paucity of research addressing the social and cultural aspects of injury and rehabilitation outcomes for Pacific peoples in New Zealand.

Furthermore, if Pacific people are experiencing disabilities at younger ages and cannot work, the potential ramifications for their families and health are great. Research is required to understand outcomes following injury among Pacific peoples; ideally such research would address health and disability issues of importance and relevance to Pacific peoples.

Pacific models of health encapsulate Pacific values, beliefs and traditions. There is a general consensus in the literature that Pacific peoples’ views of health and disability are different to mainstream European ideologies.

Pacific models, tend to consider social and cultural dimensions of health and approach health and wellbeing holistically. Somewhat akin to the Māori ‘Te Whare Tapa Wha’ model of health and well-being, Pacific models of health illustrate the importance of balancing the well-being of the body, mind and spirit, and also the importance of the family.

Various ‘Pacific’ models have been developed. A distinguishing feature of Pacific models is the symbolic representation of concepts. These symbols tend to be rooted in the values, customs and traditions of particular Pacific ethnicities. For example the Tongan Fonua (nation) model represents the relationship between the environment and humanity, paying particular attention to Tongan hierarchy. Other examples include: the Tongan Kakala Model (process of making a fragrant garland), Samoan Fu’afaletui Model (Samoan knowledge system), the Cook Island—Tivaevae Model (traditional quilt patchwork) and Tokelauan Te Vaka Atafaga Model (canoe).

Many models can be adapted to function as useful frameworks for research and analysis. Models, such as the Fonua and Tivaevae models have been used in education and social science investigations. In New Zealand the Fonofale, Faafaletui and Te Vaka models have been used in health contexts.

This paper discusses the importance of applying Pacific frameworks of health to general population health studies and assesses a questionnaire used in a study of injured New Zealand residents to determine whether aspects of importance to Pacific peoples’ health were captured.

Specifically, this paper seeks to:

- Describe relationships between the Prospective Outcomes of Injury Study (POIS) questionnaire and important elements of Pacific people’s health and disability using a Pacific model; and
- Identify questions or dimensions that future studies could consider including to address health and disability issues of particular importance for Pacific peoples.
Methods

The Prospective Outcomes of Injury Study (POIS) aims to identify predictors of outcome among injured New Zealanders. POIS reports outcomes following injury for a large cohort from five regions of New Zealand—including Auckland and Manukau cities where the majority of Pacific New Zealanders reside.\textsuperscript{17}

POIS has recruited New Zealand citizens or residents who experienced an injury severe enough to be placed on an entitlement claims register with New Zealand’s no-fault compensation injury insurer—the Accident Compensation Corporation (ACC).\textsuperscript{18} POIS was developed with a particular emphasis on addressing bi-cultural research questions under the framework of the Treaty of Waitangi.\textsuperscript{19}

Analyses of POIS data from participants who identified as having at least one Pacific ethnicity, as per the New Zealand 2006 Census, was planned.\textsuperscript{20,21} As a precursor to these analyses, we assessed questions from the first POIS highly-structured questionnaire (usually administered by interview)\textsuperscript{22} to see whether it addressed aspects of health and disability important to Pacific participants. Responses to open-ended questions were also assessed to see if reference to Pacific values were made.

The POIS questionnaire used predominantly set-response-option questions, and included questions about injury characteristics, physical and mental wellbeing, health service experiences, disability, personal-wellbeing and occupational outcomes.\textsuperscript{17} Key Pacific models of health were reviewed to identify a framework to structure the analysis. The model selected was Fuimaono Karl Pulotu-Endemann’s Fonofale Model.\textsuperscript{14}

The Fonofale model is the only model, to our knowledge, developed in consultation with several Pacific groups in New Zealand. It draws on aspects identified by Pacific peoples as fundamental for health and well-being. For these reasons we believed it was an appropriate framework to use when considering Pacific peoples and injury in New Zealand.

The model integrates the metaphor of a house (\textit{fale}), complete with a roof and a foundation.\textsuperscript{14} The roof of the house represents culture, the beliefs and values Pacific peoples possess. These can include beliefs in both Western and traditional methods of healing.

Cultural aspects can include Pacific peoples being New Zealand or Pacific nation born and raised. It can also include the different ethnicities of individual Pacific people e.g. one parent being European (\textit{Kai valagi} or \textit{Palagi}) and the other parent being of a Pacific ethnicity.

The foundations of the house represent the family. Pacific families provide the support network and structures to support life. The Pacific family can comprise both nuclear and extended family members. This idea of family can also be retrospective, taking account of genealogy (\textit{gafa}).\textsuperscript{14}

The posts (\textit{pou}) supporting the roof represent continuous and interactive dimensions of Pacific peoples’ health. They are: spiritual health (well-being as a result of Christianity or traditional spirituality); physical health (biological and physical well-being, as well as the relationship of the body to physical or organic substances e.g. food, water, air and medications); mental health (health of the mind, involving thinking and emotion); and ‘other’ aspects which can influence health and well-being (e.g. gender, sexual orientation, age, social class, employment and educational status). The house itself is encircled by factors such as the environment, the sociopolitical context and time and place in which Pacific peoples live.\textsuperscript{14}

For our study, the research team analysed each question (n=212) from the first (and most comprehensive) POIS questionnaire, to see whether, and where, it may fit within the elements of the Fonofale model; with an emphasis on the central house.

The strength of each Fonofale element within the POIS questionnaire was determined according to the number and type of POIS questions. All categorizations were verified by the two Pacific authors (RDC and FS).

Results

Of the 212 questions in the POIS questionnaire, two-thirds (66\%) were able to be placed within a single element of the Fonofale Model (Table 1). Most of the remainder were placed in multiple Fonofale elements. These included ‘multidimensional’ questions (10\%) relating to two or three elements and,
‘overarching’ questions (22%) reflected in four or more elements of the model (e.g. participants ability to carry out usual activities, communicating and socialising).

Two percent of POIS questions were not directly applicable to Pacific peoples (e.g. questions asked only of Māori participants such as questions about Māori tribal affiliations).

Table 1. Overview of POIS questions (%) in relation to the Fonofale model

<table>
<thead>
<tr>
<th>Fonofale model</th>
<th>POIS questions n (%)</th>
<th>Examples of POIS questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Culture (Roof)</td>
<td>8 (4)</td>
<td>Ethnicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Country of birth and years lived in New Zealand</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cultural sensitivity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sense of community</td>
</tr>
<tr>
<td>Family (Foundation)</td>
<td>8 (4)</td>
<td>Family involvement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Household responsibilities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Global/social relationships</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Marital status</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Household/family arrangements</td>
</tr>
<tr>
<td>Physical (Post)</td>
<td>27 (17)</td>
<td>Injury characteristics (pre and post)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hearing and sight</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Physical activities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hand dominance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Work capacity</td>
</tr>
<tr>
<td>Spiritual (Post)</td>
<td>2 (1)</td>
<td>Religion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comfort in spiritual beliefs</td>
</tr>
<tr>
<td>Mental (Post)</td>
<td>30 (14)</td>
<td>Depression, sadness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Loss of interest</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nervousness</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alcohol and drugs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Life satisfaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Expectation</td>
</tr>
<tr>
<td>Other (Post)</td>
<td>47 (22)</td>
<td>Socioeconomic status: costs, work situation, pay, demand, work skill and expertise, financial support, household dwelling, income, material standard of living, education</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Services: health service use, experiences with service, health system contact and trust</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gender</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>Environment</td>
<td>8 (4)</td>
<td>Distance to services</td>
</tr>
<tr>
<td>Time/Context</td>
<td></td>
<td>Relationship at work</td>
</tr>
<tr>
<td>Overarching/</td>
<td>67 (32)</td>
<td>Self-efficacy, optimism</td>
</tr>
<tr>
<td>Multidimensional</td>
<td></td>
<td>Overall health and wellbeing</td>
</tr>
<tr>
<td>Unplaced</td>
<td>4 (2)</td>
<td>Māori languages and Māori tribal affiliation</td>
</tr>
</tbody>
</table>

POIS questions relating to the physical post included general health status, BMI, prior chronic illness and mobility. POIS questions relating to the mental post included those asking participants about depression, anxiety, and alcohol and drug use. There were many POIS questions addressing socioeconomic status and service use.

Although the POIS questionnaire assesses spirituality, this was not comprehensive. POIS included only two questions directly eliciting information about spirituality—
one about religious affiliation and the other about the level of comfort participants found in their faith or spiritual beliefs.

Culture was another element represented by few POIS questions. The cultural questions identified included ethnicity, migration, number of years lived in New Zealand and relationships with community. POIS questions related to the family post asked about level of family involvement in life, household responsibilities, marital status, household and family living arrangements and satisfaction with social relationships.

These are important questions to ask; however they do not sufficiently cover family and its importance in the lives of Pacific peoples. Additionally, POIS lacked questions relating to sexuality or gender roles (including fa’afafine), and questions examining conflict between traditional Pacific roles and New Zealand status roles.

Fonofale elements least addressed within the POIS questionnaire were those of spirituality, culture and family. Due to the paucity of questions addressing spirituality, culture and family, we also reviewed responses to open-ended free-text questions where participants were able to raise any factors they perceived as helping or hindering their recovery from injury.

Analysis of these open-ended questions found no additional information in relation to spirituality, family or culture; instead the emphasis was on access to health services and the nature of the injury itself.

**Discussion**

We found that the POIS questionnaire contained questions addressing all of the core elements within the Fonofale Model however it was restricted in three aspects: spirituality, culture and family.

For most Pacific peoples, Christian spirituality plays a fundamental role in attitudes, expectations and relationships. One question within the POIS questionnaire was from the Functional Assessment of Chronic Illness Therapy—Spiritual well-being scale (FACIT-Sp-12; permission to use the item was granted by www.facit.org).

Bearing in mind the ever-present issue of responder burden in structured interviews, including the other eleven FACIT-Sp-12 questions may be advisable in future studies. Other measures could also be considered such as the 20-item Spiritual Well-being Inventory, and the 79-item Spiritual Assessment Inventory (SAI).

We have been unable to identify spirituality questionnaires that have been validated for use in Pacific populations. Further research could specifically address the role of Pacific spirituality (which may include the presence of spirits and connection with the dead) in the recovery process.

We acknowledge that culture is a complex aspect to assess in questionnaires. It is likely that all the dimensions in the Fonofale model have interactions with, and possess aspects that reflect, culture.

There is no one measure that is able to fully measure culture or encompass all the factors associated with Pacific culture. Given this, a useful area to explore in future surveys would be the level of cultural alignment to New Zealand Pacific society.
Such assessments would give insights into cultural and social factors that aid in the lives and decisions of Pacific peoples. For example, while a question in POIS enquired about the length of time living at participants’ current address, additional questions could be asked about cultural reasons that may influence residency—e.g. proximity to family and Pacific communities.

Other cultural areas that could be explored include language, or languages, spoken by individuals at home; work, in communities, or at church. In the POIS questionnaire, there were questions that referred to the ability of participants to speak ‘te reo Māori’, but not for other languages.

Future surveys could include similar questions about Pacific languages spoken. However, the relationship of language with culture is a contested subject, as fluency in Pacific languages may not necessarily reflect strong cultural alignment or tendencies.

Nevertheless, it seems useful to consider alongside other questions such as the number of years lived in New Zealand and community relationships, as included in POIS.

Questions could also explore cultural traditions, customs and obligations as well as engagement in cultural activities or processes. The hypothesis is that individuals with a stronger cultural sense and belonging, more often understand and continue to practice cultural traditions and customs. These connections may cause individuals to feel a sense of belonging and provide support networks in times of difficulties, such as injury or illness. Alternatively, cultural obligations and traditions may be perceived as placing stress upon Pacific people living in New Zealand.

The other Fonofale element addressed in a somewhat limited way within the POIS questionnaire was ‘family’. Family has been identified as the fundamental unit of Pacific society, and is the foundation for the Fonofale model of health and wellbeing.

While considering future questions, researchers may want to keep in mind the structure of most Pacific families which is not restricted to the nuclear family but includes the extended family. Unfortunately measures that have been developed with Pacific populations in mind are scarce. Perhaps other questions such as the five item Family Adaptability, Partnership, Growth, Affection and Resolve (APGAR) Test could be considered.

The APGAR assesses family members’ satisfaction with five components of family function. Future studies could also assess the impact of the injury or disability on the family. Here, the 27-item Impact on Family Scale may be useful; addressing the economic, social, familial and personal strain health and illness bears on the family.

Although useful, these questions do not comprehensively address family issues of importance to Pacific peoples such as kinship, reciprocity, and communality, therefore, further work seems warranted to develop improved measures.

The health status of Pacific peoples in New Zealand needs to be improved. Injury and disability is an important area to address given its effect on the socioeconomic status and well-being of Pacific peoples.
Our assessment found the POIS survey included a wide range of questions that addressed each aspect of the Fonofale model. We found that it was particularly strong in relation to the physical, mental and ‘other’ aspects of health (particularly service use and socioeconomic status). However, more attention to spirituality, culture and family appears warranted.

We acknowledge that there are usually tensions between breadth and depth in population surveys; between asking sufficient questions and not overburdening individual participants. Minimising responder burden was always a consideration in the design of POIS. POIS has investigated a range of outcomes—including outcomes specific to Pacific people.

Currently, a POIS qualitative study is underway with Pacific women who have not recovered from injury, to investigate in an open-ended manner the issues of greatest importance to them and their wellbeing. What our present analysis of the POIS quantitative questionnaire suggests, is that more work exploring how best to strengthen some of the Fonofale elements within future quantitative studies would be useful.

With today’s global societal, economic and environmental changes the health of Pacific peoples is challenged. Researchers undertaking population studies or surveys have a role to play in strengthening the ‘fale’ within their surveys. Our findings suggest that using a Pacific model to help inform questionnaire development could help with this task.

**Competing interests:** Nil.

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Incidence of maternal sensitisation to Rh(D) in Christchurch, New Zealand and reasons for prophylaxis failures

Krishna G Badami, Johanna Parker, Aoife Kenny, Sue Warrington

Abstract

Aim To estimate the current incidence of maternal sensitisation to Rh(D) and examine reasons for prophylaxis failures.

Method Retrospective chart review of new sensitisations to Rh(D) detected in antenatal records, between 2005 and 2012 in Christchurch, New Zealand and systematic examination of circumstances likely to have caused prophylaxis failures.

Results Fifty-four new sensitisations in an at-risk population of about 4624 in 8 years means an incidence of roughly 1.1%. In 86.6% of 45 sensitisations where information was available, there was a recognised sensitising event including previous deliveries while in 13.3% there were none. Of those with recognised sensitising events, 46.1% had anti-D prophylaxis per local guidelines, in 12.8%, prophylaxis was given though it did not conform, entirely, to guideline. No prophylaxis at all was given to 41% despite a sensitising event being recognised.

Conclusion The incidence of maternal sensitisation to Rh(D) in Christchurch, New Zealand, is as expected given our prophylaxis regimen. Half the sensitisations were associated with complete or partial failure to follow local guidelines. Better adherence to this may reduce incidence of sensitisation. It is also thrice as high as might be expected with a routine antenatal anti-D prophylaxis (RAADP) program. An economic analysis of RAADP in New Zealand will be useful.
Method
Retrospective chart review of all those with alloantibody to Rh(D), detected for the first time between 2005 and 2012, in the New Zealand Blood Service (NZBS) Christchurch Hospital Blood Bank antenatal records.

Using a specially-designed data collection form we systematically examined the circumstances that might have led to failure of protection against sensitisation to Rh(D)—specifically:

- Was there a recognised antenatal sensitising event?
- If so, at what stage of pregnancy did it occur?
- What dose of prophylactic anti-D, if any, was given to cover this event?
- If indicated, was a Kleihauer (or equivalent) test done to determine if a further prophylactic anti-D dose was indicated?
- Was there any history of transfusion of Rh(D) incompatible blood components e.g. platelets and, if so, was this covered with prophylactic anti-D?

As this study meets the criteria for studies that do not require New Zealand Health and Disability Ethics Committees review, ethics approval was not sought.

Results
In the antenatal records we found 54 new sensitisations to Rh(D) in the 8 years from 2005–2012. All appeared to be related to the pregnancy with none, apparently, related to an Rh(D) incompatible transfusion. The parity of the women involved varied from 0–5. Seven women (13%) were primigravidae. Table 1 summarises our data on sensitisations to Rh(D) and prophylaxis during this period.

Table 1. Summary of data on sensitisations to Rh(D) and anti-D prophylaxis in Christchurch, 2005–2012

<table>
<thead>
<tr>
<th>Recognised sensitising event (including previous delivery)</th>
<th>Prophylaxis</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>Yes</td>
<td>Given per guideline</td>
<td>Given but not per guideline</td>
<td>Not given (not given despite recognised sensitising event)</td>
</tr>
<tr>
<td>Pregnancy stage when sensitisation first detected</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1(^{st}) trimester</td>
<td>13</td>
<td>1</td>
<td>12</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>2(^{nd}) trimester</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3(^{rd}) trimester</td>
<td>23</td>
<td>4</td>
<td>19</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Peri-partum / post-natal</td>
<td>8</td>
<td>1</td>
<td>7</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>No information</td>
<td>9</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>totals</td>
<td>54</td>
<td>6</td>
<td>39</td>
<td>18</td>
<td>5</td>
</tr>
</tbody>
</table>

To calculate incidence of maternal sensitisation to Rh(D), we estimated the population-at-risk. For this we considered the number of confinements (defined as ‘a pregnancy resulting in either live or stillborn children, irrespective of whether a single or multiple birth results’\(^8\)) in Christchurch. There are about 6300 of these annually and 85% of them (about 5355) may be assumed to be in women of European Caucasian origin\(^8\) of whom 18% (about 964) may be expected to be Rh(D) negative.\(^9\)
It should be noted that the other ethnic groups of any size in Christchurch (Māori, Pacific Islander and East Asian) are predominantly Rh(D) positive. It can be calculated that about 60% of all pregnancies in the Rh(D) negative, European Caucasian population (578 pregnancies) will result in an Rh(D) positive fetus the consequence of which may be maternal sensitisation to Rh(D). This assumes that the father is also Caucasian and takes in to account the proportions of Rh(D) positive Caucasian Europeans who are likely to be either heterozygous or homozygous for Rh(D) and the likelihood that only 50% of pregnancies where the father is heterozygous for Rh(D) will result in an Rh(D) positive fetus whereas this will be 100% if the father is homozygous.

Thus, the population-at-risk—the population of Rh(D) negative pregnant women likely to be carrying an Rh(D) positive fetus—in Christchurch can be calculated to be about 578 per year or 4624 for the 8 years from 2005–2012. We can then calculate a rough incidence proportion of 54/4624 or 1.1% for this 8 year period and an incidence rate of about 1.4 per 1000 person years in this population.

For 9/54 (16.6%) sensitisations in our study, no clear information on timing of sensitisation, events predisposing to this, or prophylaxis, was available. These will, essentially, not be considered further. Thirteen (24%) new sensitisations to RhD were detected during the 1st routine antenatal blood tests (around 10–12 weeks gestation), one (1.8%) in the 2nd trimester, 23 (42.5%) in the 3rd trimester, and 8 (14.8%) in the peri-partum or post-natal periods.

Among the 45 sensitisations where information was available, 39 (86.6%) were preceded by a documented sensitising event, including previous deliveries, while for six (13.3%) there were no documented events. Table 2 summarises the nature of sensitising event depending on stage of pregnancy when sensitisation was first detected.

Of the 39 with a recognised sensitising event, 18 (46.1%) had anti-D prophylaxis per NZBS guidelines. In a further 5, (12.8%), prophylaxis was given though it did not conform entirely to the guideline (all of which were failures to perform a follow-up Kleihauer test to determine the quantum of fetomaternal haemorrhage when indicated). No prophylaxis at all was given to 16/39 (41.0%) women despite a sensitising event being recognised nor is there a record of a Kleihauer test being done in these cases.

Overall, of the 54 new sensitisations to Rh(D), at least 18 (33.3%) appear to have occurred despite the standard, local anti-D prophylaxis guideline being adhered to, at least five (9.2%) with partial non-adherence to the NZBS guideline and 22 (40.7%) with anti-D never having been administered. As stated, for nine (16.6%), information was inadequate.
Table 2. Likely sensitising event in the 45 women in Christchurch sensitised to Rh(D), 2005–2012 for whom this information was available

<table>
<thead>
<tr>
<th>Pregnancy stage when sensitisation first detected</th>
<th>Sensitising event</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt; trimester:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous delivery only</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Miscarriage&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Still-birth&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Massive fetal haemorrhage&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>No sensitising event</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>2&lt;sup&gt;nd&lt;/sup&gt; trimester:</td>
<td>Previous delivery only</td>
<td>1</td>
</tr>
<tr>
<td>3&lt;sup&gt;rd&lt;/sup&gt; trimester:</td>
<td>Previous delivery only</td>
<td>14</td>
</tr>
<tr>
<td>Miscarriage&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>PV spotting&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Uterine rupture&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Intrauterine death (29/40)&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Termination of pregnancy&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>No sensitising event</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>Per-partum / post-natal:</td>
<td>Previous delivery only</td>
<td>3</td>
</tr>
<tr>
<td>Miscarriage&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Fall on to abdomen&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Amniocentesis&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Termination of pregnancy&lt;sup&gt;*&lt;/sup&gt;</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>No sensitising event</td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>

<sup>*</sup> previous delivery was also a potential sensitising event in these cases

Discussion

The incidence proportion of maternal sensitisation to Rh(D)—the major cause of HDFN—used to be about 13–14% before any prophylaxis was available, this fell to 1–2% after routine post-natal prophylaxis was started in the 1960s, to about 1% after antenatal prophylaxis for sensitising events and further to about 0.2–0.3% after the introduction of RAADP in the 1970s.<sup>1–3</sup>

In contrast to some countries, RAADP is not official policy in New Zealand.<sup>1,4</sup> Though some practitioners use it, its application here is patchy at best.<sup>10</sup>
From our data it would appear that the calculated incidence proportion in Christchurch is at least 1.1% of the population at risk per year or 6.3 women per year. These figures may in fact be slightly higher because in some instances sensitisation may only be detectable following re-stimulation by the antigen—for instance in a subsequent pregnancy and this may not have happened. Nevertheless, the calculated incidence is roughly what one might expect with prophylaxis for antenatal sensitising events and routine post-natal prophylaxis (conventional prophylaxis) but without RAADP and is at least thrice as high as might be expected if an RAADP programme was also in place.

Because less than half the sensitisations with a documented sensitising event (18/39) had anti-D prophylaxis per guideline, improved adherence to protocol, supported by continuing education and, perhaps, checklists may reduce incidence of sensitisation. An NZBS audit of anti-D prophylaxis showed that greater than 95% of all post-natal indications for anti-D prophylaxis audited were covered appropriately in terms of initial dose and its timing. However this audit also highlighted the overall poor, and very variable, adherence to the guidelines for the Kleihauer test as also the overall low (5% of eligible candidates), and variable, use of RAADP. It should be kept in mind that this audit was concerned with the way anti-D was used when it was used. It does not address, as does the present study, the incidence of sensitisations and the causes of prophylaxis failures.

In 41% (16/39) of sensitisations with a documented sensitising event, anti-D prophylaxis was simply not administered. If an RAADP programme had been in place, it is likely that some of these sensitisations might have been prevented. Why anti-D prophylaxis was omitted in these 16 women is unclear excepting in the case of two Jehovah’s Witnesses who refused prophylaxis.

In nearly half the sensitisations with a documented sensitising event (18/39), prophylaxis was administered per guideline. This suggests that other, unrecognised, sensitising events—perhaps not covered with prophylaxis—also occurred. It is reasonable to think that some of these sensitisations too may have been prevented through an RAADP programme.

In 6/45 sensitisations where the clinical record appeared to be complete, no sensitising event had been recorded—not even a previous delivery. We are not sure what the mechanism of sensitisation in these cases was. Some women may not truly have been primigravidae; some primigravidae may have failed to report a sensitising event and, in some sensitisation could have occurred without obvious exposure to Rh(D) positive RBC—through pregnancy, transfusion, or other means. Mechanisms by which this might occur include the sensitisation of Rh(D) negative mothers during fetal life by exposure to Rh(D) positive RBC from their mothers or what’s been called the ‘grandmother hypothesis’. A similar audit was performed in the UK covering a period when RAADP was not yet standard practice (1988–1991). In this study, of 129 women with 312 pregnancies and 98 potentially sensitising events between them, information was inadequate for 40% of events, 52% of women with Rh(D) sensitisation had not had a recognised sensitising event (other than a prior delivery), sensitisation occurred after 20% of
events despite anti-D prophylaxis per local protocol and 48% of events were associated with complete or partial failure to follow local guidelines. ¹⁴

Experience shows that even with an RAADP programme sensitisation to Rh(D) still occurs. ⁴,¹⁵ These may be due to failure to adhere to the RAADP protocol, refusal to accept prophylaxis, and, possibly, biological reasons for failure to respond as expected to prophylaxis.

Nevertheless, a recent bias-adjusted meta-analysis has confirmed that RAADP, in addition to conventional prophylaxis, is more effective than conventional prophylaxis alone. ¹⁶ There are several studies of cost-benefit analysis of RAADP, additional to conventional prophylaxis, compared to the latter alone. These take in to account the additional costs of the anti-D for the RAADP, and of administering it, compared to the costs, if RAADP was not used, of managing sensitised pregnancies and neurodevelopmental problems in affected children. Even without reckoning societal and other costs, RAADP is believed to be economically attractive—especially for primigravidae but also if used in all eligible women. ¹⁷–¹⁹

To summarise, the incidence of maternal sensitisation to Rh(D) in Christchurch, New Zealand, is about 1.1%. Between 2005–2012, 44 neonates in the Christchurch region were affected by Rh isoimmunisation. Consequently, 13 fetuses received intrauterine transfusions and 7 neonates, exchange transfusions. This is as expected given our prophylaxis regimen. Half of all sensitisations appeared to be associated with complete or partial failure to follow the current guideline. Better adherence to this may reduce incidence of sensitisation. The incidence is three times higher than it might be if an RAADP program was also in place.

With such a programme, the number of new sensitisations in Christchurch can be expected to drop from about 6.3 to about 2 per year. An economic analysis of RAADP in New Zealand, comparing various models of RAADP with the status quo, will be useful.

Competing interests: Nil.

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References and websites:


Postgraduation retention of medical students from Otago and Auckland medical programmes

William Shelker, Phillippa Poole, Warwick Bagg, Ian Wood, Paul Glue

Abstract

Aim Auckland and Otago medical programmes have different methods for selecting students. This study compared postgraduate retention in New Zealand (NZ) of medical graduates from the two medical programmes, to assess whether different selection methods influenced retention. Other variables assessed included entrance category and age at graduation.

Methods Anonymised databases were created of all graduates from the Otago Faculty of Medicine (1999–2011) and the Auckland medical programme (2000–2012). Demographic and entry category data were recorded. Retention was defined as presence on the NZ Medical Register in December 2012. Risk differences (RD) were calculated to compare retention between the two medical programmes using the Mantel-Haenszel method. The influence of medical programme entrance category on retention was also tested. The influence of covariates on retaining graduates on the register was evaluated using a multiple logistic regression model.

Results The postgraduate retention of graduates of the two medical programmes over 13 years was identical (Auckland 74.9%, Otago 73.6%, P=0.48). Retention of graduate and non-graduate entry students from both medical programmes was similar by 6 years after graduation. Age during medical school did not affect retention.

Discussion University of attendance had no effect on postgraduation retention of students on the NZ Medical Register, suggesting that retention is not influenced by the different student selection methods at each programme. The data presented shows that New Zealand graduates regardless of programme completed show a similar profile in terms of retention.

The retention of doctors in New Zealand (NZ) is a workforce issue with many practical, economic and political components.1

NZ has become the most heavily dependent country in the world on overseas-trained doctors to fill the medical workforce.2 In part this is due to years of enrolment of relatively low numbers of medical students, but NZ also loses many graduates overseas.3

It has been estimated that over one-third of medical graduates are not registered with the NZ Medical Council by 10 years after graduating, with many presumably working overseas temporarily or permanently.4

While the NZ government has recently increased medical student numbers significantly,5 very little has been done to understand or indeed enhance retention in NZ. Our earlier research using graduates from the University of Otago explored aspects of medical student selection that might lead to increased retention in the NZ
medical workforce. One of these studies identified that completion of a prior degree or allied health professional qualification was associated with higher retention at 13 years than for school leaver entry (relative increase of 7% and 20%, respectively), and that this was not due to an effect of age.

There are two medical programmes in NZ, run by the Universities of Otago and Auckland respectively. Entrance pathways to the two medical programmes are broadly similar. Students may enter the programmes at year 2 of a 6-year medical degree after completing either a health sciences first-year course (labelled HSFY) or a degree (competitive graduate entry or CGE).

Each programme has Māori and Pacific and rural origin affirmative schemes, but students selected must still enter via one of the two aforementioned pathways, after achieving an academic threshold grade. Where the programmes differ is mainly in the use of selection tools (Table 1; note there is also one additional entry pathway at Otago).

Otago does not interview HSFY or CGE applicants, whereas Auckland has a 25-minute interview which is structured and contains questions about commitment to a medical career. While this interview does not predict performance at medical school or dropping out, we considered that it might plausibly help in selecting those more predisposed to stay in NZ after graduating.

The objective of this research was to examine the influence of a number of variables, including medical programme, age, and category of medical programme entry, on the postgraduate retention of NZ medical students.

We hypothesised differences in selection processes and criteria might lead to different rates of postgraduate retention in NZ, and might inform decision-making as to whom to offer medical school places.

### Table 1. Tools used for selection of medical students at the Universities of Otago and Auckland

<table>
<thead>
<tr>
<th></th>
<th>OTAGO</th>
<th>AUCKLAND</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Entry via Health Sciences First Year (HSFY) course</strong></td>
<td>Student ranking</td>
<td>Academic performance (66%)</td>
</tr>
<tr>
<td></td>
<td>Academic performance (66%)</td>
<td>UMAT (34%)</td>
</tr>
<tr>
<td></td>
<td>No interview</td>
<td>No interview</td>
</tr>
<tr>
<td><strong>Entry as a Graduate (CGE)</strong></td>
<td>Student ranking</td>
<td>Weighted academic score for applicants who meet UMAT threshold</td>
</tr>
<tr>
<td></td>
<td>Academic performance (60%)</td>
<td>UMAT (15%)</td>
</tr>
<tr>
<td></td>
<td>No interview</td>
<td>No interview</td>
</tr>
<tr>
<td><strong>Entry via Other Category</strong></td>
<td>Student ranking</td>
<td>At least B average, or to complete HSFY with B average</td>
</tr>
<tr>
<td></td>
<td>Students may apply once per category</td>
<td>Interview</td>
</tr>
<tr>
<td><strong>Additional considerations</strong></td>
<td>Compilation of entry list</td>
<td>Others&gt;Graduate&gt;HSFY, to obtain ~5:20:75 proportional entry</td>
</tr>
<tr>
<td></td>
<td>Number of applications to medical school</td>
<td>Students may apply once per category</td>
</tr>
</tbody>
</table>

UMAT: Undergraduate Medicine and Health Sciences Admission Test.
Methods

Approval for this project was given by both the Otago University Ethics Committee and the University of Auckland Human Participants Ethics Committee. The Otago database was created with the names of students graduating from the University of Otago, along with their entrance categories, provided by the Health Sciences Admissions Office.

Data regarding the year of birth, gender and year of graduation were obtained from the University of Otago student database, under the supervision of an authorised staff member. An identical database for Auckland medical students was created by a University of Auckland staff member.

The NZ Medical Register was accessed in December 2012 to identify which graduates were registered in NZ, and to collect postgraduate information (such as medical specialisation or training program), and geographical location within NZ. Names were matched by using string functions, or other demographic information. Sponsored foreign students were not included in either database, and Other Category students from Otago were also excluded, as there was no corresponding category at Auckland.

Anonymised data from both databases were analysed using summary statistics. The influence of age at graduation, entry category and the year of graduation on remaining on the NZ Medical Register were evaluated using logistic regression (Stata v11). Risk Differences (RD) were calculated to compare retention between entry categories by year of graduation and by medical programme, using the random effects Mantel-Haenszel method (Review Manager 5.0).

Results

The database of medical graduates between 1999 and 2011 included all 1611 eligible students from the Auckland medical programme and 2254 students from the Otago medical programme (Table 2). Reliable data comparing the entrance categories between the two programmes were only available for Auckland graduates from 2006-2011, hence the totals for these subcategories are smaller than the Total (Table 2, see rows 2,3,5,6)

Age at graduation and gender ratio were similar in both medical programmes. CGE students were statistically significantly older at graduation compared with HSFY students. There were similar proportions of graduates entering at each programme via HSFY and CGE (Auckland: 86% from HSFY and 14% from CGE, Otago: 83% from HSFY and 17% from CGE).

Table 2. Demographics of the Auckland and Otago Medical School graduates

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>% of category</th>
<th>mean (SD) age at graduation</th>
<th>% male</th>
</tr>
</thead>
<tbody>
<tr>
<td>AUCKLAND Total*</td>
<td>1611</td>
<td></td>
<td>25.17 ± 3.14</td>
<td>46.12%</td>
</tr>
<tr>
<td>HSFY**</td>
<td>616</td>
<td>85.91%</td>
<td>24.81 ± 3.16</td>
<td>42.86%</td>
</tr>
<tr>
<td>CGE**</td>
<td>101</td>
<td>14.09%</td>
<td>28.41 ± 3.72</td>
<td>43.56%</td>
</tr>
<tr>
<td>OTAGO Total*</td>
<td>2254</td>
<td></td>
<td>25.17 ± 3.27</td>
<td>46.67%</td>
</tr>
<tr>
<td>HSFY**</td>
<td>845</td>
<td>83.17%</td>
<td>24.26 ± 2.11</td>
<td>44.62%</td>
</tr>
<tr>
<td>CGE**</td>
<td>171</td>
<td>16.83%</td>
<td>27.06 ± 2.86</td>
<td>37.43%</td>
</tr>
</tbody>
</table>

*1999–2011 graduates. **2006–2011 graduates. t=10.33, p<0.0001. q=14.82, p<0.0001.

Figure 1 shows the number of graduates from each medical programme by year of graduation, as well as the number that were still on the NZ Medical Register in December 2012. A total of 1206 of 1611 Auckland graduates (74.9%) and 1659 of
2254 Otago graduates (73.6%) were on the NZ Medical Register in December 2012. This difference was non-significant (Mantel-Haenszel Risk Difference (RD) 1% (95% CI=-2, 4)).

Figure 1. Forest plot comparing retention of medical graduates from Auckland and Otago Universities between 1999–2011

The proportion of medical graduates remaining on the NZ Medical Register by time elapsed since graduation was shown in Figure 2. The loss at 5 years was greater for HSFY students than for CGE students, with similar trends for both medical programmes, but this was not statistically significant.

Retention of HSFY students from Auckland and Otago was similar, (RD =0.03, 95%CI -0.01–0.08, p=0.11). Retention of CGE students from Auckland and Otago was also similar (RD=0.01, 95% CI=-0.11–0.13, p=0.84).

Comparison of 6-year retention rates for CGE vs. HSFY students from the two medical programmes identified that 238 of 272 CGE students (87.5%) and 1255 of 1461 HSFY students (86.0%) were on the NZ Medical Register during December 2012. Again this was not significantly different (RD=0.01, 95% CI=-0.03, 0.06). Retention of Otago CGE students was 7% greater than HSFY students, based on 13 year data.6
The effect of age at graduation on retention over 5 years for the two medical programmes was shown in Figure 3. Age categories were selected to give approximately similar group sizes. Visual inspection of these plots does not suggest differences in retention by age category.

Because mean age at graduation differed by entrance category (Table 2), multiple logistic regression was used to evaluate the relative influences of entrance category and age at graduation on retention in each centre. There was an overall significant effect for 1999–2011 Otago graduates (n=2137; p=0.012) with the relationships described by the equation Logit P=0.05 + (0.02*agegrad) + (0.31*entrancecat).

Assessment of individual variables identified Entrance Category as statistically significant (OR 1.36, 95%CI 1.01–1.83; p=0.04); however age at graduation was not (OR 1.02, 95%CI 0.98–1.07; p=0.31). While there was no significant effect for either the 2006–2011 Auckland graduates (n=717; p=0.64) or the 2006–2011 Otago graduates (n=1017; p=0.43) the equations describing these relationships were similar to the 13-year Otago graduates analysis: Auckland: Logit P=1.25 + (0.01*agegrad) + (0.24*entrancecat); Otago: Logit P=1.06 + (0.01*agegrad) + (0.29*entrancecat).
Figure 3. Graduate retention by age at graduation category in (A) Auckland and (B) Otago

Note: Values shown are 2-year rolling averages.

Discussion

Using the natural experiment afforded by having two 6-year medical programmes in one country, we have found similar patterns in both the Auckland and the Otago medical programmes in terms of medical student demographics and NZ medical workforce retention up to 13 years after graduation.

About a quarter of 1999–2011 graduates were no longer practising in NZ by December 2012. Although the reasons for the losses were not identifiable from the present study, we have previously shown that most are registered overseas. Apart from the natural tendency of New Zealanders to travel overseas for a period, other drivers could include postgraduate training requirements and places available, as well as the fact that a high proportion of NZ medical students were born overseas. The retention of ~three-quarters of NZ medical graduates is similar to that reported previously.

Our study suggests that medical student retention postgraduation is not influenced by the different student selection strategies employed by the two medical programmes. Moreover, these selection strategies have changed over time, especially at Auckland, yet there has been little difference in cohort retention rates.

Specifically, we have found that an interview does not add value to NZ workforce retention, despite the potential for it to identify those with a commitment to remaining and working in NZ.

A second finding was that despite independent and varied selection strategies, the total percentage of students admitted from HSFY in both centres was similar (Auckland: 85.91%, Otago: 83.17%). One explanation for this may be that NZ has a fairly limited pool of graduates with the aptitude and drive to enter medicine, whereas there are many more eligible HSFY students than there are places.

Another finding was that entrance category to medical school influences retention rates but only to a very small extent, with lower rates of retention for HSFY students,
and higher rates for CGE students. Although this was apparent from visual inspection of Figure 2, it was only statistically significant when examining data over 13 years from the Otago medical programme, and not from the comparison of 6-year combined data from Auckland and Otago medical programmes.

Possible explanations for this are that differences in retention occur gradually, and 6 years is not long enough to demonstrate this. For example, New Zealanders often head overseas after university and return to NZ later.\(^3\) There may also be statistical power considerations due to the smaller size of the Auckland cohort (n=716), compared with the 13-year Otago cohort (n=2137). This finding is consistent with our previous publication\(^6\) and would support continuing to admit a significant proportion of graduate students in medical student cohorts as a means to enhancing retention of doctors in NZ. However, this effect is modest, and given the limited pool of suitable CGE applicants, may not provide strong reasons to increase the pool of graduate entrants further. Furthermore, a strategy to enrol more CGE applicants could increase costs to students and society by prolonging the time to earn a medical degree, as well as having a possible detrimental effect on the affirmative schemes.\(^8\)

Our final finding, that age at graduation does not affect rates of retention, is consistent with our earlier findings.\(^6\)

Possible shortcomings of this study should be acknowledged. Entrance category data were only available for 6 years for Auckland graduates, and this may have limited our ability to identify trends in retention that may only become evident over a longer timeframe.

As discussed in an earlier publication, there may be other life experiences associated with completion of a degree, rather than the academic achievement, that could account for increased medical graduate retention in CGE students. Among these are marriage status, number and age of children, student loan status, and/or employment satisfaction. These data were not collected in this project and cannot be further explored.

In conclusion, both the Auckland and Otago medical programmes have similar rates of retention of their medical graduates in NZ, with possibly higher retention rates for students enrolling after completing a degree. This finding of similar retention rates between medical programmes was not anticipated, as we predicted that differences in student selection strategies might lead to differences in rates of retention post-graduation. Taken together, our findings do not indicate a way to change the selection policies of either programme to aid NZ workforce retention, but do suggest the interview adds no value to this particular outcome.

We suggest schools continue to enrol the current proportion of graduate students until longer term data shows convincingly that graduates are more likely to stay in the NZ medical workforce.

Competing interests: Nil.

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References:
Molecular epidemiology of group A streptococcus from pharyngeal isolates in Auckland, New Zealand, 2013

Deborah A Williamson, Nicole J Moreland, Philip Carter, Arlo Upton, Julie Morgan, Thomas Proft, Diana Lennon, Michael G Baker, Rod Dunbar, John D Fraser

Abstract

Aims To describe the molecular epidemiology of emm types associated with circulating pharyngeal group A streptococcus (GAS) isolates in Auckland, New Zealand.

Methods GAS isolates were collected over a 10-day period from a community pathology provider in Auckland. PCR analysis and sequencing of the emm gene was performed at the Institute of Environmental Science and Research.

Results A total of 52 emm types were identified from 278 GAS isolates. The three most common emm types were emm1, emm89 and emm12. Overall, the experimental 30-valent GAS M protein vaccine covered 19 / 52 (37%) of emm types in our study.

Discussion Our study provides baseline data on the circulating pharyngeal GAS emm types in Auckland. Future clinical and molecular surveillance of GAS pharyngitis is essential in the context of ongoing GAS vaccine development.

Group A streptococcus (GAS) is a major human pathogen and is responsible for considerable morbidity and mortality. GAS infections cause a range of acute clinical manifestations, including pharyngitis, skin and soft tissue infection (SSTI), and serious invasive disease such as bacteraemia, necrotising fasciitis and streptococcal toxic shock syndrome. Moreover, the non-suppurative sequelae of GAS infection (rheumatic fever and post-streptococcal glomerulonephritis) result in a substantial clinical and economic burden.

New Zealand has one of the highest reported incidence rates of rheumatic fever, with significant sociodemographic disparity. Consequently, a number of initiatives designed to reduce the incidence of rheumatic fever have recently been implemented in New Zealand. These include measures to: (i) improve housing conditions; (ii) systematically identify and treat childhood sore throats in the school and primary care settings; and (iii) improve patient health literacy. In addition to these public health approaches, there has been renewed interest in developing an effective vaccine to prevent GAS infections and their consequences.

Recently, an international workshop was held in Auckland to assess potential GAS vaccine candidates [Moreland NJ et al, manuscript in draft]. Although several GAS vaccines are currently in development, only two have reached clinical trial stage—the most advanced being a multivalent vaccine based on the GAS M protein, encoded by the emm gene. As such, knowledge of the locally circulating GAS emm types is a prerequisite when considering the potential effectiveness of this vaccine in a specific population.
To date, there are limited contemporary data on the circulating GAS emm types in New Zealand. In this context, and to inform discussion for the above workshop, we performed a 'snapshot' survey of the molecular epidemiology of circulating GAS emm types in Auckland.

Methods

LabTests Auckland (LTA) provides the majority of community diagnostic microbiology services to the 1.4 million population of the greater Auckland region. This includes all referrals from primary care, such as general practitioners, midwives, and the school-based throat-swabbing programme. Over a 10-day period in January 2013, all non-duplicate group A streptococcus isolates growing from throat swabs were collected. Throat swabs were plated onto tryptic soy sheep blood agar and incubated in 5% CO2 overnight at 37°C. GAS isolates were identified using a MALDI-TOF MS Biotyper (Bruker, Germany) and purity plated onto nutrient agar slopes. All GAS isolates were forwarded to the Invasive Pathogens Laboratory at the Institute of Environmental Science and Research (ESR) for further analysis. Polymerase chain reaction (PCR) analysis and DNA sequencing of the emm gene was performed using previously described methods.

Simpson’s index of diversity was used to assess variation in emm types. This index indicates the probability that two emm types randomly selected are of different types – i.e. the higher the index, the greater the diversity of emm types in a particular population. 95% confidence intervals (CI) for the Simpson’s index were calculated as previously described.

Results

Between the 7th and 16th of January 2013, 1418 throat swabs were received at LTA. Of these, 282/1418 (19.8%) specimens grew GAS. The median age of the patients with GAS isolated was 12 years (range 3–69 years), and 120/282 (43%) of patients were male. Of the 282 GAS isolates, 278 were emm typed. Overall, a total of 52 different emm types were identified (Figure 1).

A relatively small number of emm types predominated, such that six emm types (emm1; emm89; emm12; emm28; emm75; and emm22) together accounted for 59% of all isolates. The Simpson’s index of diversity was 0.904 (95% CI, 0.883–0.924). Overall, 19/52 (37%) emm types were represented in the experimental 30-valent M protein vaccine (Figure 1). These emm types included 17/30 (57%) of the most common circulating emm types (Figure 1).

Recent data suggest the 30-valent M-protein vaccine evokes cross-opsonic antibodies against non-vaccine emm types. When the putative effect of cross-opsonic antibodies against other emm types was considered, the potential vaccine coverage increased to 29/52 (56%) of all emm types, and 21/30 (70%) of the 30 most common emm types (Figure 1).
Conclusions

Our study provides a contemporary ‘snapshot’ of the circulating pharyngeal GAS emm types in Auckland, New Zealand. Although we found substantial diversity in emm types, only a few types predominated.

The three predominant emm types in our region (emm1; emm89 and emm12) are similar to those described from GAS pharyngeal isolates in other developed countries. For example, Shulman et al analysed over 7000 GAS pharyngeal isolates in North America over a 7-year period from 2000–2007. In both the United States and Canada, the two predominant emm types were emm1 and emm12. Similar to our setting, they found that a relatively small number of emm types predominated, such that, in their study, 10 emm types accounted for approximately 90% of all isolates. Moreover, Steer et al performed a systematic review of global emm types, and found that in high-income countries, the two most common emm types were emm1 and emm12.

Interestingly, a recent study described the emergence of emm89 (the second most common emm type in our study) as a major emm type in a Canadian population, increasing from 2.7% of GAS isolates in 2002 to 14.7% in 2010. Of note, the third most common emm type in our study (emm12) was not represented in the 25 most common emm types in a previous study in the Auckland region.
This study assessed the emm types associated with invasive GAS disease in Auckland from January 2005 to December 2006. However, temporal variation in circulating GAS emm types is well described, as are differences in emm types according to clinical syndrome.

Despite the short sampling frame in our study, we observed considerable diversity in the circulating GAS pharyngeal emm types in Auckland. We found that the experimental 30-valent M protein vaccine covered only 37% of emm types in our sample, although this coverage increased to 57% when only the 30 most common emm types were considered.

Our estimated vaccine coverage is higher than that calculated in the previous Auckland study of emm types associated with invasive GAS disease, where only 17/58 (29%) emm types were covered by the 30-valent vaccine. However, the recent demonstration that immune sera evoked by the 30-valent vaccine contains significant levels of bactericidal antibodies to 24 of 40 non-vaccine serotype indicates the coverage of the multivalent M-protein vaccine may be greater than originally predicted.

When this additional potential coverage was extrapolated to our study sample, vaccine coverage increased to 56% of all strains and 70% when only the 30 most common emm types were considered. Despite this, ongoing questions remain around the widespread usage of M-protein based vaccines, including coverage of emm types in less developed parts of the world, and the theoretical potential for serotype replacement, resulting in the emergence of non-vaccine serotypes.

There were several limitations in our study. We did not have clinical information relating to each patient and as such, were unable to distinguish colonizing and infecting GAS pharyngeal isolates. However, given that each patient had a throat swab taken as part of a primary care consultation it is probable that these patients had pre-test clinical symptoms suggestive of pharyngitis.

A further limitation was our short sampling frame, which meant we were unable to assess longitudinal changes in the molecular epidemiology of circulating emm types. In addition, our sampling frame was during the 2012–2013 school summer holidays, and as such would not have included children presenting as part of the school-based throat swabbing programme.

In summary, our study provides baseline information on the molecular epidemiology of GAS pharyngeal isolates in Auckland, New Zealand. Despite high national rates of rheumatic fever, and ongoing work around sore throat prevention in schools and primary care, there is no formal system of surveillance of GAS pharyngitis in New Zealand.

Future work should aim to systematically assess the national clinical and molecular epidemiology of this significant disease burden.
Competing interests: Nil.

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References:


‘A child restraint for every child on every trip’

Bridget Kool, Rebekah Ryan, Keira Radice, Elizabeth Segedin, Gabrielle Nuthall, Michael Shepherd, Julie Chambers

Abstract

Child passenger injury from road traffic crashes is a leading contributor to New Zealand’s paediatric trauma-related mortality and morbidity. New Zealand has significantly higher rates of child passenger injury than internationally comparable countries. Correctly used child restraints can prevent death and severe injury of child passengers. Despite huge efforts by individuals and Non-Government Organisations to promote up-to-date height-based legislation and to distribute child restraints, the New Zealand Government has a tepid commitment to promoting child passenger safety. Further change is needed, in both our child restraint legislation and practice. This paper highlights the recommendations from a Paediatric Society of New Zealand Position Statement for the correct use of child restraints. This information should be used by all health professionals to advocate for and implement this important injury prevention initiative.

Correctly used child car restraints can prevent death and severe injury of child passengers. The most effective way of ensuring all children are provided with a child restraint when they are travelling in a vehicle is to have legislation that reflects...
current evidence, is appropriately enforced, and is coupled with widespread education and car seat distribution programmes.5–9

'A child restraint on every trip for every child' is the key message New Zealand's Paediatric Society members emphasise in their Child Passenger Safety Position Statement adopted at the Society’s 2012 Annual General Meeting.10

The position statement was developed in consultation with Plunket and is a synopsis of evidence for the safe transport of children in vehicles. It is targeted towards child health professionals, and includes a list of recommendations (Table 2).

Topics include information on the safest seating position for children; the dangers of lap belts; when children can safely use adult seat belts; the age at which infants may be seated in forward-facing restraints and child restraint related hypoxia.

The statement also urges health practitioners to engage New Zealand Qualifications Authority (NZQA) certificated child restraint technicians to provide child restraint advice.

Table 2. The Paediatric Society of New Zealand recommendations for the correct use of child car restraints

<table>
<thead>
<tr>
<th>The Paediatric Society of New Zealand recommends:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A child restraint is used on every trip for every child. Child passengers travelling in motorised vehicles should at all times be seated in a restraint that is correctly fitted into the vehicle, meets accepted Standards, and is suitable for the child's age, height, weight and development.</td>
</tr>
<tr>
<td>2. Child Health Services and Well Child Service providers work with Certificated Child Restraint Technicians to ensure health professionals; whānau and caregivers receive expert technical advice and have access to information and products that promote and ensure the safe transport of children.</td>
</tr>
<tr>
<td>3. The transfer and transportation of children by child health services should at all times be carried out in a manner consistent with best practice child restraint advice and families, whānau and caregivers are provided with every opportunity to access and use child restraints when travelling to and from hospital and/or child health services.</td>
</tr>
<tr>
<td>4. Families, whānau and caregivers of children with special health needs receive expert advice on the safe transportation of their child from Certificated Child Restraint Technicians who are working in collaboration with their Child Health Service Provider. This includes situations where the use of a usual child restraint is not achievable or may compromise the child's health, for example, children with hip spicas, cardiopulmonary conditions and/or behavioural issues.</td>
</tr>
<tr>
<td>5. Child restraints that are semi-reclining are used only for travel in the first months of life and travel time spent in a child restraint should be minimised.</td>
</tr>
<tr>
<td>6. Child Health Services and Well Child Service providers routinely advise New Zealand families to:</td>
</tr>
<tr>
<td>6.1 Seek advice from a Certificated Child Car Restraint Technician when purchasing and installing child car restraints.</td>
</tr>
<tr>
<td>6.2 Seat children rearward facing up until the age of two years, and then continue to seat them rearward facing for as long as practicable.</td>
</tr>
<tr>
<td>6.3 Ensure young infants are not left unattended to sleep in semi-reclining child restraints.</td>
</tr>
<tr>
<td>6.4 Use head positioning inserts to ensure infants are correctly positioned and able to maintain a clear airway at all times they are in the child restraint.</td>
</tr>
<tr>
<td>6.5 Ensure children younger than the age of fourteen always travel seated in the back seat, for their safety.</td>
</tr>
<tr>
<td>6.6 Ensure children are never placed in a restraint in the front seat of a vehicle where an airbag might be activated. This is critical with respect to rear-facing child restraints.</td>
</tr>
<tr>
<td>6.7 Only use lap belts when there is no safer alternative.</td>
</tr>
<tr>
<td>6.8 Continue to use a child restraint or booster seat until the child reaches 148 cm in height.</td>
</tr>
</tbody>
</table>
Despite the overwhelming scientific evidence of the effectiveness of child restraints and long-standing recommendations that they be used, the New Zealand Government has a tepid commitment to promoting child passenger safety. Change is needed, in both our child restraint legislation and practice.

New Zealand’s child restraint legislation was introduced in 1994 as a first step towards achieving better passenger safety for children. It languished unattended for over 15 years. A crucial issue for paediatricians has been the law’s failure to mandate that older children remain in child restraints (i.e. booster seats) until it is safe for them to use adult seat belts. In 2010 a Road Safety strategy Safer Journeys 2020 recommended updating the law.

In 2011, a New Zealand Cabinet paper confirmed to the Executive that children are being injured and killed after prematurely graduating from child restraints into adult seat belts. The Cabinet paper acknowledged that New Zealand child restraint law has lagged behind other international jurisdictions, and noted an extensive public education campaign promoting the voluntary use of booster seats had not resulted in any improvement and recommended a law change.

The law change, which was enacted in November 2013, includes a Rule that children must be seated in a child restraint until their seventh birthday, two years longer than previously required (Table 3). This Rule brings New Zealand into line with recently enacted Australian Federal law, but falls short of recommendations in many other parts of the world including the UK, Europe and Canada.

### Table 3. November 2013 New Zealand child restraint law change

<table>
<thead>
<tr>
<th>The law says you must:</th>
<th>Until 31 October 2013</th>
<th>From 1 November 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correctly secure your child in an approved child restraint</td>
<td>Until their 5th birthday</td>
<td>Until their 7th birthday</td>
</tr>
<tr>
<td>Correctly secure your child in an approved child restraint if one is available in the vehicle (and if not, in any child restraint or safety belt that is available)</td>
<td>From their 5th birthday until their 8th birthday</td>
<td>From their 7th birthday until their 8th birthday</td>
</tr>
</tbody>
</table>

Source: [http://www.nzta.govt.nz/about/media/releases/2669/news.html](http://www.nzta.govt.nz/about/media/releases/2669/news.html)

These changes have been greeted coolly by paediatricians as 'a step in the right direction'. The paediatrician’s lack of enthusiasm for this recent update is explained within the Paediatric Society’s Statement, which says best practice is to keep children in child restraints until they reach a minimum height of 148 cm. That height is usually not reached until approximately a child’s 11th birthday. For this reason many overseas child restraint laws encode minimum height requirements along with age; for example the United Kingdom, European Union and Canada.

The most recent published car restraint use surveys in New Zealand found that between 45% and 65% of child passengers were in incorrectly fitted restraints. Lack of information and resources have been cited as factors contributing to the incorrect
use of child restraints.\textsuperscript{19,20} In addition to the law change, greater effort is needed on methods shown to be effective within at risk communities, which is the distribution of child restraints through multifaceted campaigns within community settings.\textsuperscript{21}

New Zealand health services do not need to look far to find gaps and limitations in the distribution of child restraint information and resources. The New Zealand Road Rules do not require an infant or child of any age to be seated in a child restraint when they are travelling in a registered passenger vehicle including minivans, taxis, or buses.\textsuperscript{22} Infants and children travelling between health services or home from hospital in taxis can legally be completely unrestrained, held in someone’s arms, or restrained only with an adult seat belt.\textsuperscript{20}

This exemption, disappointingly, has resulted in occasions where rather than being viewed as an opportunity to provide child restraints for at-risk families using their services, health professionals have turned a blind eye to families engaging in obviously unsafe behaviour.

The Paediatric Society’s endorsement of Plunket’s message ‘A child restraint for every child on every trip’, and their call for health services to work more directly with community-based child restraint rental and loan agencies, are prompts aimed directly at challenging and changing this practice.\textsuperscript{10}

The adoption of this position Statement by Paediatric Society members represents a refreshed focus by child health professionals on the promotion of child passenger safety. This information should be used by all health professionals to advocate for and implement this important injury prevention initiative.

The full version of the Paediatric Society’s Position Statement is available online at: http://www.paediatrics.org.nz/index.asp?pageID=2145878337

Competing interests: Nil.

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Acknowledgements: We thank the Paediatric Society of New Zealand’s Injury Special Interest Group for their feedback on the Position Statement as well as Plunket New Zealand for the use of their slogan ‘A child restraint for every child on every trip’.

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References:

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Relief from cancer chemotherapy side effects with pharmacologic vitamin C
Anitra C Carr, Margreet C M Vissers, John Cook

Abstract
Fatigue is a common, often debilitating, side effect of cancer chemotherapy. Pharmacologic vitamin C has been used as an alternative treatment for the disease itself but its effects on fatigue have not often been documented. Here we report on the case of a woman with recurrent breast cancer, undergoing weekly chemotherapy, with lethargy as a major symptom. Vitamin C (50 g/session) was administered twice weekly and quality of life and multidimensional fatigue symptomology questionnaires were undertaken. Dramatic decreases in fatigue and insomnia were observed, as well as increased cognitive functioning. There were no adverse side effects of i.v. vitamin C.

Fatigue is the most common symptom reported by cancer patients and can affect quality of life more than pain.\textsuperscript{1,2} Fatigue can be expressed at physical, emotional and mental levels, and questionnaires that cover these multidimensional aspects have been developed for use with cancer patients.\textsuperscript{3}

The use of i.v. vitamin C in cancer is relatively common, but there is controversy as to any proven benefits. Vitamin C has numerous functions, including a co-factor role in collagen, carnitine, neurotransmitter and neuropeptide hormone synthesis and in the regulation of epigenetics and gene transcription.\textsuperscript{4} Many of these functions could potentially influence quality of life and fatigue.\textsuperscript{5}

Case report
Here we report the case of a 45-year-old female diagnosed in May 2009 with invasive ductal carcinoma of the left breast (grade 2, ER+, PR+, HER2-). She immediately underwent wide local excision (no lymphovascular invasion evident) and radiation therapy to the breast. Tamoxifen was terminated after 4 days due to intolerance. Axillary lymph node recurrence occurred in Feb 2013 followed by axillary lymph node clearance (4/22 lymph nodes with extra nodal disease).

In March 2013 a CT scan of chest, abdomen and pelvis showed no evidence of distal metastatic disease. Two cycles of fortnightly chemotherapy with doxorubicin and cyclophosphamide were initiated in April and May, and in June once weekly paclitaxel was initiated for 12 weeks. Lethargy was a major symptom of chemotherapy.

Prior medical history included gastric bypass in 2004 for weight reduction and a hysterectomy in 2005 for adenomyosis. At that time the patient was tired and lethargic and blood tests indicated low iron and B12 levels. In 2007 a toxic nodule on the thyroid was diagnosed and in 2012 a total thyroidectomy was carried out for a multinodular goitre.
A buccal swab was analysed for selected single nucleotide polymorphisms by the Department of Genetics, La Trobe University (Melbourne, Australia). Profiling of specific inflammatory, immune, antioxidant and detoxification genes indicated a number of adverse gene polymorphisms in this patient (Table 1).

Of particular interest for this case are the CYP1B1 (cytochrome P450 1B1) and NQO1 (nicotinamide quinone oxidoreductase 1) polymorphisms. CYP1B1 is located mainly in breast, endometrium and ovaries and is involved in oestrogen metabolism. Increased activity due to the indicated homozygous polymorphism, leads to enhanced activation of pro-carcinogens. NQO1 has strong antioxidant capacity and has been described as an anti-cancer enzyme. The heterozygous polymorphism indicates extremely reduced enzyme activity. It is possible that these deleterious gene polymorphisms contributed to the development of breast cancer in this patient.

Table 1. Selected genes involved in inflammation, immune systems, cell antioxidant defence and detoxification

<table>
<thead>
<tr>
<th>Gene product</th>
<th>Dysfunction</th>
<th>Polymorphism</th>
<th>Heterozygous</th>
<th>Homozygous</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP-1</td>
<td>Increased risk of inflammation</td>
<td>0169171C&gt;T</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>CRP-2</td>
<td>Increased risk of inflammation</td>
<td>0143294G&gt;A</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>CAT</td>
<td>Increased risk of oxidative stress</td>
<td>262C&gt;T</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>CYP1B1</td>
<td>Increased levels of reactive toxins</td>
<td>Val432Leu</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>GSTP1</td>
<td>Decreased capacity to clear toxins and reactive oxygen species</td>
<td>313A&gt;G</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>NQO1</td>
<td>Highly decreased antioxidant capacity</td>
<td>690C&gt;T</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>HO-1</td>
<td>Increased risk of oxidative stress and inflammation</td>
<td>-413A&gt;T</td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>

To investigate the effects of pharmacologic vitamin C on quality of life and fatigue due to chemotherapy, intravenous vitamin C (50 g/session, AscorL500, McCuff Pharmaceuticals, Santa Ana, USA) was initiated twice weekly, 2 days either side of each chemotherapy session (doxorubicin/cyclophosphamide in May and paclitaxel in June of 2013). Quality of life (EORTC QLQ-C30) and fatigue (MFSI-SF) questionnaires were undertaken before and after 4 weeks of vitamin C intervention.

The quality of life questionnaire showed dramatic decreases in fatigue, pain, appetite loss, nausea/vomiting and insomnia following vitamin C administration (Figure 1A). Increases in physical, emotional, cognitive and social functioning were also observed, as well as a doubling of the patient’s “global health status” (Figure 1B).

The multidimensional fatigue symptomology questionnaire showed decreases in general, physical, emotional and mental fatigue, as well as increased vigour, following vitamin C administration (Figure 2).

Final assessments were made after 4 weeks of vitamin C administration, but positive effects were noticed following the first administration. No adverse side effects of the vitamin C administration were observed by the patient or her GP.
Figure 1. Patient’s health-related quality of life scores before (black bars) and after (grey bars) i.v. vitamin C administration

Note: All of the scales range in score from 0 (no bar)–100, with a high score representing a higher response level, i.e. a high score for a symptom scale (A) represents a high level of symptomology/problems, whereas a high score for the global health status scale (B) represents a high quality of life and a high score for a functional scale (B) represents a high/healthy level of functioning.
Figure 2. Patient’s multidimensional fatigue scores before (black) and after (grey) i.v. vitamin C administration

Note: All of the single-item measures range in score from 0 (not at all = no bar) to 24 (extremely). Total fatigue represents the sum of general, physical, emotional and mental fatigue scores minus the vigour score.

Discussion

Research has shown that fatigue has a constant presence following chemotherapy and also increases incrementally with consecutive cycles of chemotherapy. A retrospective, multicentre, epidemiological cohort study has indicated that intravenous vitamin C administration improves quality of life, including fatigue, in breast cancer patients during chemo-/radiotherapy and aftercare.

Our case report supports the findings of Vollbracht et al and extends these by further investigating the effects of intravenous vitamin C on the multidimensional aspects of fatigue. Following pharmacologic vitamin C administration there were dramatic decreases in chemotherapy-related fatigue and other symptoms, as well as increased functioning and overall health.

It is not possible to rule out a placebo effect, particularly as this effect tends to be more prevalent with measures of subjective symptoms. However, based on the
varied functions of vitamin C in the body, it is plausible that vitamin C contributed to some of the observed quality of life effects and similar findings have recently been reported.

Overall, our report has shown that pharmacologic vitamin C may be considered for patients experiencing side effects from chemotherapy. Furthermore, based on our prospective findings and others and the retrospective findings of Vollbracht et al., a double-blind placebo-control study of the efficacy of intravenous vitamin C on chemotherapy-related quality of life and fatigue appears warranted.

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**References:**

Congestion bleeding of the head and neck following myocardial infarction

Caroline Mahon, Paul Maurice, Dougal McClean

Abstract

We present an unusual case of congestion bleeding of the head and neck following myocardial infarction. A 51-year-old man presented with widespread facial petechiae and subconjunctival haemorrhages following a collapse associated with evolving electrocardiographic changes. Emergency coronary artery stent placement was undertaken. No cardiopulmonary resuscitation (CPR) was performed.

We hypothesise that the presence of facial petechiae in our case following transient loss of consciousness due to a presumed ventricular arrhythmia in the setting of acute myocardial ischaemia, may have been precipitated by a Valsalva manoeuvre on regaining consciousness with sudden acute increase in venous pressure and consequent venous congestion of the head and neck, and that congestion bleeding of the face may occur in acute cardiac events without a history of CPR.

In clinical medicine petechiae of the head and neck and subconjunctival haemorrhage are common examination findings in cases in which there is a history of recurrent vomiting or forceful coughing.

Subconjunctival haemorrhage and petechiae of the face and neck are also the hallmarks of asphyxial death, particularly in cases of strangulation. The proposed pathophysiologic mechanisms for head and neck congestion bleeding are the combined effects of increased cephalic venous pressure generated by raised intrathoracic pressure or external compression of the neck or chest wall and subsequent hypoxic injury to the vascular endothelium of the skin and conjunctival microvasculature.

Congestion bleeding of the head and neck observed in patients experiencing non-traumatic near death events has long been thought to be a consequence of CPR, though this presumed association has been challenged in the forensic literature. We present the case of a patient with acute myocardial infarction presenting with head and neck congestion bleeding, without an antecedent history of CPR.

Case report

A 51-year-old man with a background of hyperlipidaemia treated with bezafibrate presented to the emergency department having had a collapse with loss of consciousness at his home. There was a preceding 3-day history of chest pain. The patient reported on the day of the collapse that he felt hot and complained of chest pain.

He was witnessed by his son to collapse to ground and was unresponsive, pale and sweaty. His son noted the appearance of a rash over the face and neck. The patient
took an estimated one minute to regain consciousness and then continued to complain of chest pain.

Ambulance staff noted on arrival at his home that the patient was alert and that his face was deeply flushed in appearance. His vital signs were within normal limits and he was in sinus rhythm. On arrival in the emergency department triage staff also documented the presence of a rash over the face and neck.

An ECG showed an evolving acute anterior ST elevation myocardial infarction and the patient went on to have urgent percutaneous coronary intervention, with placement of a single stent into the left anterior descending coronary artery and three stents into the right coronary artery.

He was given aspirin and clopidogrel immediately prior to the procedure, which was uncomplicated. A single intracoronary bolus of IIb/IIIa platelet inhibitor, abciximab was also given. An echocardiogram performed at the same time showed mildly impaired systolic function with an ejection fraction of 50%. There was no evidence of venous obstruction. Further comment was made of the presence of facial rash in the clinical record by the attending nursing staff at the time of the procedure.

The following morning the patient was noted to have widespread petechiae of the face and neck with bilateral subconjunctival haemorrhages. The platelet count, and INR/APTT were within the normal range. The dermatology service was asked to see him 60 hours after his admission.

**Figure 1. The patient captured his image on his own cellphone the day after his presentation to hospital**

![Image of the patient's cell phone capture](image-url)
On examination, extensive petechiae were seen over the face and neck, most prominent over the forehead and cheeks. There was bilateral subconjunctival haemorrhage, more pronounced on the left, with a left-sided upper eyelid ecchymosis in the region of the inner canthus (Figure 1).

Specific questioning of the patient and his relatives confirmed that CPR had not been performed in the community by family members or by ambulance staff.

Discussion

The clinical findings of facial petechiae, ecchymosis and subconjunctival haemorrhage in our patient are consistent with those typically seen in clinical medicine in cases of raised cephalic venous pressure induced by preceding prolonged or repeated Valsalva manoeuvre. There have been case reports of facial petechiae in cases of recurrent forceful vomiting in healthy newborn infants, infants with pyloric stenosis, as well as in weightlifters and bungee jumpers and as a rare complication of upper gastrointestinal endoscopy. Postictal facial petechiae have also been reported.

Facial petechiae have also been reported as post mortem findings in cases of death due to cardiac causes. The petechiae have long been thought to be related to venous congestion of the head and neck due to CPR attempts or iatrogenic post mortem handling of the corpse. However, periorbital and conjunctival petechiae have also been documented in post mortem findings in 21% of cases of cardiac death in which no resuscitative attempts were made.

In a recent prospective study of 196 patients presenting to an emergency department in cardiac arrest due to non-traumatic causes (including myocardial infarction, arrhythmia, pulmonary oedema, pulmonary embolism and cerebral infarction), 4% were found to have periorbital and/or conjunctival petechiae before CPR was initiated.

Two survivors diagnosed with decompensated cardiac insufficiency, without petechiae at initial presentation or immediately after CPR, were found to have facial petechial haemorrhages hours after admission to an intensive care unit for management of ongoing cardiac insufficiency. Therefore, establishing a direct causative relationship between congestion bleeding of the head and neck and CPR in acute cardiac events is somewhat controversial.

It has been postulated that the appearance of congestion bleeding of the head and neck in survivors of cardiac events in which no CPR has been performed may be due to a period of acute right heart failure with sudden precipitous impairment of venous return to the heart but ongoing left ventricular output. In sudden death due to cardiac arrest a plausible physiologic mechanism for the appearance of facial and conjunctival petechiae is more difficult to hypothesise.

In the forensic literature, conjunctival and facial petechiae are well established findings in deaths due to ligature or manual strangulation, partial hanging, plastic bag-ligature suffocation, carotid sleeper holds and crush injuries to the chest. In all of
these mechanisms, the prerequisite event appears to be impairment of venous drainage of the head and neck whilst partial or intermittent arterial supply is maintained.

The subsequent mechanical trauma caused by venous congestion to the microvasculature of the ocular and facial tissues is thought to result in rupture and cutaneous bleeding.

The role of hypoxia and tissue acidosis in provoking petechial bleeding is controversial and has been challenged in the literature, as facial congestion bleeding is not a characteristic of autoerotic deaths due to plastic bag suffocation without ligature or deaths due to gag obstruction of the airway.

In addition, studies in which healthy volunteers were subjected to inverse suspension have shown that conjunctival petechiae can be induced after one minute of suspension and without associated loss of consciousness, and therefore mechanical trauma rather than tissue hypoxia is likely to be the primary insult to tissue microvasculature in the head and neck.\textsuperscript{15}

**Conclusion**

It is likely that our patient collapsed due to cerebral hypoperfusion resulting from transient low cardiac output from a presumed ventricular arrhythmia in the setting of acute myocardial infarction. He may have been choking as he recovered, with the associated Valsalva manoeuvre causing an acute increase in venous pressure leading to congestion bleeding of the head and neck.

Prolonged hypoxia followed by reperfusion may have contributed. To our knowledge, although the presence of facial congestion bleeding in cardiac deaths and their presumed relationship with CPR has been studied and challenged in the forensic literature, there have been no case reports in the clinical literature of head and neck petechiae or subconjunctival haemorrhage in survivors of myocardial infarction.

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**References:**


Pituitary metastasis: an unusual cause of hypopituitarism

Yared N Demssie, Kavita Kulavarasalingam, Mohit Kumar, Akheel Syed

Clinical—A 51-year-old man was referred to our endocrine clinic with symptoms of erectile dysfunction and loss of libido of 4 months duration. He was otherwise asymptomatic and general physical examination was unremarkable. Initial investigation revealed secondary hypogonadism and further assessment of full pituitary hormonal profile showed panhypopituitarism including central diabetes insipidus. He was started on hydrocortisone, thyroxin and testosterone replacement therapy along with oral desmopressin. Subsequent investigation with magnetic resonance (MR) imaging of the brain revealed an enhancing pituitary mass and three subcortical ring enhancing masses involving the left frontal lobe (Figure 1, Panels A, B and C). A whole body computed tomography scan showed a large right middle lobe primary tumour and extensive bilateral pulmonary metastasis. CT scan guided bronchial biopsy of the primary tumour confirmed adenocarcinoma of the lung.

Figure 1. Panels A and B show an enhancing pituitary mass abutting on the optic chiasm and panel C shows a ring enhancing mass in the left frontal lobe (white arrows)

The patient has been treated with a course of chemotherapy and cranial radiotherapy. He remains stable 24 months after diagnosis except for episodes of generalised seizures which have been adequately controlled with sodium valproate.

Discussion—Symptomatic pituitary metastasis is a very rare clinical entity but latent metastasis is much more commonly encountered in autopsy series. Breast and lung cancer are the two most common cancers that metastasise to the pituitary gland.
The posterior pituitary is reported to be the most common site of metastasis and hence central diabetes insipidus is one of the most common clinical manifestations. Other presenting symptoms include visual field defect, ophthalmoplegia, headache and symptoms of anterior pituitary hormonal deficiency such as excessive tiredness, reduced libido, erectile dysfunction and amenorrhea.

Clinical and radiological distinction from other non-functioning pituitary tumours is difficult but the diagnosis should be suspected in older patients with rapidly progressing neuro-ophtalmologic symptoms and central diabetes insipidus.

Treatment is palliative but subtotal resection could be undertaken for patients with neuro-ophtalmologic symptoms. Conventional radiotherapy and gamma knife stereotactic radiosurgery have also been utilised for symptom relief and local tumour control.

The overall prognosis is poor with a reported mean survival between 6–7 months and only 10% of patients surviving beyond 1 year.

**Learning points**

- The finding of secondary hypogonadism should prompt a complete assessment of pituitary hormonal function along with pituitary imaging to rule out hypopituitarism and pituitary mass lesion respectively.

- Pituitary metastasis should be considered as a differential diagnosis of a non-functioning pituitary tumour in patients with central diabetes insipidus and rapidly progressing neuro-ophtalmologic symptoms.

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**References:**


Two earholes

Kenta Watanabe

A previously healthy 27-year-old female visited our clinic for evaluation of a second hole in her right ear. The patient reported that the hole had been present since infancy, and it was likely congenital. She did not have any severe symptoms with the exception of slight ear discomfort several times per year.

A hole 4 mm in diameter was found at the anteroinferior part of cavum conchae of the right auricle, which was separate from the true external auditory canal (Figure 1: arrow), and which contained a small amount of scurf.

Figure 1. An extra hole was found at the cavum conchae of the patient’s right auricle

Computed tomography revealed an extracranial blind-ended sinus beneath the true external auditory canal (Figure 2: arrow). Audiometry was normal on both sides, and no other malformations were found around the acoustic organs.
Figure 2. Computed tomography revealed an aerated blind-ended sinus beneath the true external auditory canal

Although the definite pathogenesis of the second hole was unknown, it seemed to be a type of first branchial cleft anomaly.¹ Usually, the treatment of the anomaly is complete surgical excision of the sinus, fistula, or cyst.² In this case, however, surgery was not selected because the patient had no history of active infection in the sinus, which opened widely and was well aerated. The patient was followed up, and the hole was periodically cleaned.

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References:
Marketing and supplying alcohol to young people

The posting of a video of an extremely intoxicated 9-year-old boy resulted in a media furore in recent weeks reflecting the extreme youth of the individual and a heightened focus on Internet postings; it also illustrated increased public concern over alcohol related harm and highlighted some key public health issues which require urgent action.

This case is a ‘tip of the iceberg’ illustration of what our survey data suggests is happening in New Zealand among young people (although data does not cover children as young as 9 years). As controls on sale from licensed premises have become better enforced and compliance has increased—only 9% of 16-year-old drinkers and 14% of drinkers aged 17 purchasing their own alcohol in 2012 (unpublished data, HRC funded Alcohol Policy in New Zealand [APINZ] survey)—social supply has become a major source of alcohol for those under the minimum purchase age. Much supply is from older friends or relatives and, on average, about 11 cans of ready-to-drinks (RTDs) or equivalent are supplied.1

The Sale and Supply of Alcohol Act 2012 introduced greater clarity to the law regarding supplying alcohol to minors. The Act now makes it an offence to supply without ‘express consent’ of the parent or guardian. Charges have been laid against three people in connection with this case providing an opportunity to capitalise on a public health message about the law and the likelihood of prosecution.

The supply of 8 cans of Cody RTDs and 2 shots (about 150 ml of absolute alcohol in total) illustrates the importance of RTDs. In a recent study RTDs were found to be the beverage young suppliers reported supplying most often to those under the minimum purchase age.1

Attempts overseas to introduce specific taxes on RTDs have not been successful because of substitution to alternative beverages2 or amelioration of costs by provision of multipack options3 but evidence shows affordability of alcohol is very important4 and the cost entailed in this supply (about $20.60) was obviously not sufficient to deter the generous spirit of the suppliers. Increasing excise tax by 50% as recommended by the Law Commission in 2010, but rejected by government, would have increased the cost by only about $3 but would have been a move in the right direction. The results from the APINZ survey show heavier drinkers purchase cheaper alcohol.5 Tax increases are effective to reduce alcohol-related harm and significant tax increases, as have been applied to cigarettes in New Zealand, are urgently needed.

Equally urgent is action to reduce the exposure of young people to the marketing of all alcohol beverages including RTDs. Cody, with the third largest share of the high potency RTD market in NZ, carried out a significant television campaign in 20126 and is prominent in the bottle store displays to which children are exposed.

Then there is the question of distribution: high potency RTDs were to be subject, in the new Act, to greater controls. The original recommendation from the Minister,
following the Law Commission review, was to prohibit RTDs above 5% alcohol potency. This was reduced to prohibiting sale from off licenses of 6% RTDs while still allowing sale from on-license premises. However, after government consulted the producers of RTDs these clauses were withdrawn with a promise the industry would adopt a voluntary code and government would regulate quickly if needed. It is not clear what would trigger regulation or if an organisation independent of the industry will monitor.

Which brings me to a final relevant issue in the media coverage of this story: while there were no comments from the vested interest groups, producers or retailers, one NZ Herald story did quote Dr Eric Crampton, a University of Canterbury economist, who said the video was shocking because ‘rare and sad events are shocking’. He also said ‘while several prominent anti-alcohol commentators have used this tragic case to argue for higher alcohol prices and broader restrictions on where alcohol can be sold, the overall statistics on youth drinking suggest that things are improving’.

Dr Crampton was referring a decrease in the proportion of young drinkers classified as hazardous drinkers or binge drinkers in recent surveys. The Ministry of Health (MoH) NZ Health Survey for example, reported 1 in 5 of those aged 15–17 years were hazardous drinkers (down from about 1 in 4 in 2006/7). There is no doubt there is some improvement but whether enough to argue against improved alcohol control policies is another question.

The University press release and NZ Herald story did not contextualise this ‘expert’ opinion, but in a recent news item it was announced that Dr Crampton and the University of Canterbury had accepted 3 years of funding from the Brewers Association of New Zealand.

In the words of the Director of External Relations of the Brewers Association, Jenny Cameron: “This funding … will add to the voices in the public debate over alcohol and alcohol policy.”

Figure 1. Russell Rd Liquor (70–72 Russell Rd, Manurewa, Auckland)
References:


Dizziness caused by medications

The terms ‘dizziness’ and ‘vertigo’ are often used interchangeably but have different meanings. ‘Dizziness’ is now precisely defined as “a sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion (vertigo)”. Well recognised causes of dizziness are extremes of blood pressure, cardiac arrhythmias and hypoglycaemia. However, an increasingly common cause is the side-effects of prescribed medications.

In a private practice (J.H.) specialising in the management of vertigo and dizziness, over a 10-year period, 70 patients were identified as having dizziness as a medication side-effect. The typical presentation is “dizziness” and often “staggeryness”, which comes on after breakfast builds during the morning and often fades by the evening.

This was established by either changing the time of use from morning to evening or (usually) cessation of the drug. The drugs were, in order of frequency: simvastatin 15 (patients), quinapril 10, terazosin 8, diltiazem 6, cilazapril 5, doxazosin 4, metoprolol 4, candesartan 2, felodipine 2, atorvastatin 2, and 1 each for 12 other drugs.

The 2013 MIMS New Ethicals lists dizziness as a side effect of all commonly used antihypertensives. However, in the clinical literature there is an absence of likely mechanisms. One explanation is that an noradrenergic receptor blocker (e.g. doxazosin) or a calcium channel blocker (e.g. diltiazem) could affect the vestibular nucleus, which has noradrenergic receptors and calcium channels.

Noradrenaline is known to modulate neuronal responses to GABA in the vestibular nuclei and may participate in the regulation of the vestibulospinal and vestibulo-ocular reflexes. Another explanation is that changes in blood pressure affect the vestibular nucleus. It is well known that vestibular stimulation affects blood pressure. Conversely there is animal experimental evidence that acute hypotension induces electrical activity mediated by the excitatory transmitter glutamate.

Although MIMS does not include disabling dizziness as a side-effect of statins it is not infrequently encountered in publications, but more difficult to explain. The statins are HMG-CoA reductase inhibitors whose primary role is reduction of cholesterol. They also have poorly
understood cardioprotective effects which include vasodilatation, and therefore may modulate function in the vestibular nuclei.

In summary, in a patient complaining of non-specific fluctuating dizziness, a medication side-effect should be considered. It seems that general practitioners and urologists have long been aware of this for terazosin and doxasosin, but few are aware for most antihypertensives and the statins.

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Emergency departments’ cost and primary care

Jones and Thornton’s paper’s conclusion—diverting funds from secondary care to offset the cost of after-hours primary care is unlikely to reduce emergency department presentations—is methodologically flawed and may be asking the wrong question.¹

The extent to which cost might be a barrier surely will vary depending on how high the cost of a GP visit might be, how big the cost of travel to the emergency department (ED) might be, and how poor the population being studied are. All of these vary considerably from place to place in New Zealand (Dunedin compared to Middlemore) and over time. Only two of the cited studies were more recent than 26 years ago. Prior to 1987 we had less child poverty and less income disparity. It is hard to draw any meaningful conclusions from such a temporally and geographically varied sample.

The conclusion seems to be at odds with a recent report from Wellington that “Capital and Coast District Health Board has saved more than $1 million and avoided 400 emergency department admissions since free healthcare for under-6s was introduced.”² However the problem is the question asked. The cost barrier to accessing primary care for children led to more ambulatory sensitive admissions that cost the board a lot of money. The money saved was in the in-patient service not in the ED. If the question is whether a cost barrier affects visits to ED then with better data you might be able to conclude that it does not. If the question is whether a cost barrier leads to greater hospital costs in this instance that it does.

EDs are not good places to provide primary care. Because there are so few of them they are a long way away from where many people live. They are designed to provide emergency care and in my experience tend to over-investigate and spend more time on primary care problems than a GP would. Whether the reasons for attendance are cost, ease of access or misunderstanding of the role of GP we should continue to discourage people with primary care problems from attending ED.

The study addressed people who attended ED, not all those who had a need to attend ED. They noted that of those surveyed in the 2011–12 New Zealand Health Survey 14% (n=12,370) had an ‘unmet primary care need’ due to cost and 7% had an ‘unmet after hours need’ due to cost.

The latest survey 2012–13³ showed that 27% of adults and 21% of children had an unmet need for primary care for all reasons. For people without access to primary care many hospital services are not available. Our first priority should be removing cost and access barriers to ensure that everyone has access to primary care.

Diverting money to primary care may not save ED costs but it has been shown to save overall hospital costs and is the best way to provide for unmet need in primary care.

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References:


Mortality from coronary heart disease: the (unnoticed) elephant in the room

Ellis et al\(^1\) in their third audit of hospital management of acute coronary syndromes (ACS) in New Zealand (NZ) hospitals report a continuing gap in optimal management between hospitals equipped and not equipped with invasive cardiac facilities.

The figures quoted by Ellis et al, however, show important long-term trends, which are not commented upon by the authors. First, to those of us who remember when in-hospital case fatality from ACS was 25%, the fact that there were only 20 in-hospital deaths (2%) among 1007 suspected cases of ACS and 17 (3.2%) among those in whom the diagnosis was confirmed, is truly remarkable and testifies to continual improvements in treatment as well as to declining severity of attacks.

But this good news from the hospitals ignores more unpleasant public health realities. According to the NZ Ministry of Health\(^2\) there were 5389 deaths from coronary heart disease in NZ in 2010; allowing for the continual fall in mortality there should have been about 5230 in 2012.

The 20 hospital deaths recorded by Ellis et al happened over 2 weeks, so assuming that the 2-week study was representative, there must have been about 20 × 26 = 520 hospital deaths over the whole of 2012. So about 90% of all deaths from ACS happened outside hospital—not altogether surprising in view of the fact that hospital mortality is declining even faster than community mortality, and suggestions from overseas studies that the ratio of out-of-hospital to in-hospital deaths is increasing.\(^3,4\)

What should be done about this “elephant in the room”? Short of prevention (primary and secondary), more lives are saved in ACS by defibrillation than by any other treatment.\(^5\) Defibrillation is more likely to be successful at the start of a heart attack than later, and early access to defibrillation depends on the patient calling for help from the ambulance with minimal delay, and the promptness of ambulance paramedics in answering the call.

Available evidence suggests that ambulance response is usually prompt, but there is concern that the behaviour of patients in calling for help, and the speed of general practitioners in responding (if they rather than the ambulance are called), leaves much to be desired.\(^6,7\)

From a public health rather than from a purely hospital perspective in the management of ACS, the “elephant in the room” surely deserves further study. The out-of-hospital toll from ACS could at least be mitigated by expediting access to a defibrillator even earlier than at present for patients with prolonged chest pain.

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References:

   http://journal.nzma.org.nz/journal/126-1387/5939


Is the benefit/risk ratio for cardiac defibrillators implanted in an older population (age 75 or more) still favourable?

An implantable cardioverter-defibrillator (ICD) is a treatment associated with significant cost and risk of morbidity. Elderly patients (arbitrarily defined as age 75 or more) were under-represented in large clinical trials (e.g. MADIT-II\(^1\) and SCD-HeFT\(^2\)) which showed a mortality benefit with ICD implantation in patients with reduced left ventricular function. Therefore, the net mortality benefit for this age group is unclear.

Van Rees et al\(^3\) found that the cumulative incidences of appropriate therapy and appropriate shocks in patients age 75 or more with primary ICD implanted were 28% and 13% respectively. Their all-cause mortality was 29% with a cumulative incidence for death of 35% at 1 year following appropriate shock. Currently, there is no such New Zealand data available.

The primary objective of our study was to ascertain survival in elderly patients (age 75 or more) who have had ICDs implanted together with device related morbidity. The secondary objective was to compare these outcomes with a younger subset of patients (age 70–74) receiving ICD.

This was a descriptive study with retrospective data collection on 41 patients aged 75 or more living in the Auckland region who received ICDs between 1 January 2000 and 31 December 2010. Demographic, clinical and survival data were collected retrospectively from Auckland District Health Board (DHB), Counties Manukau DHB, Waitemata DHB and regional ICD databases.

For comparison, data on 47 consecutive patients receiving ICDs at age 70-74 between 1 January 2001 and 31 December 2010 were retrieved from the National Wellington ICD registry. Mean follow-up was 50 months for the Auckland (age 75 or more) cohort (range 7 to 150 months) and 56 months for the Wellington (age 70–74) cohort (range 3 to 143 months).

Apart from age, the baseline characteristics were comparable (Table 1). All recorded variables were defined according to literature or common practice. Ischaemic heart disease was defined as the presence of a diameter stenosis of at least 50% in at least one coronary artery\(^4\).
Table 1. Clinical characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Auckland 75 or more cohort (n=41)</th>
<th>Wellington 70–74 cohort (n=47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>34 (83%)</td>
<td>40 (85%)</td>
</tr>
<tr>
<td>Mean age (range)</td>
<td>77 years (75-84)</td>
<td>72 years (70-74)</td>
</tr>
<tr>
<td>Primary implants</td>
<td>10 (24%)</td>
<td>13 (28%)</td>
</tr>
<tr>
<td>Secondary implants</td>
<td>31 (76%)</td>
<td>34 (72%)</td>
</tr>
<tr>
<td>&gt; for cardiac arrest</td>
<td>10</td>
<td>18</td>
</tr>
<tr>
<td>&gt; for ventricular tachycardia</td>
<td>21</td>
<td>16</td>
</tr>
<tr>
<td>History of atrial fibrillation</td>
<td>18 (44%)</td>
<td>9 (19%)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>29 (71%)</td>
<td>38 (81%)</td>
</tr>
<tr>
<td>Non ischaemic cardiomyopathy</td>
<td>14 (38%)</td>
<td>7 (22%)</td>
</tr>
<tr>
<td>Prior revascularisation</td>
<td>18 (44%)</td>
<td>25 (53%)</td>
</tr>
<tr>
<td>&gt; CABG alone</td>
<td>15 (83%)</td>
<td>12 (48%)</td>
</tr>
<tr>
<td>&gt; PCI alone</td>
<td>2 (11%)</td>
<td>8 (32%)</td>
</tr>
<tr>
<td>&gt; CABG &amp; PCI</td>
<td>1 (6%)</td>
<td>5 (20%)</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>14 (34%)</td>
<td>9</td>
</tr>
<tr>
<td>Class II</td>
<td>18 (44%)</td>
<td>14</td>
</tr>
<tr>
<td>Class III</td>
<td>7 (17%)</td>
<td>5</td>
</tr>
<tr>
<td>Class IV</td>
<td>2 (5%)</td>
<td>0</td>
</tr>
<tr>
<td>Unknown</td>
<td></td>
<td>19</td>
</tr>
<tr>
<td>QRS&gt;150ms</td>
<td>15 (37%)</td>
<td>17 (36%)</td>
</tr>
<tr>
<td>LBBB</td>
<td>28 (68%)</td>
<td></td>
</tr>
<tr>
<td>Mean LVEF (range)</td>
<td>35% (19–56)</td>
<td>35% (11–70)</td>
</tr>
<tr>
<td>EF 35% or less</td>
<td>28 (68%)</td>
<td>26 (55%)</td>
</tr>
</tbody>
</table>

ICD therapy (including shock and anti-tachycardia pacing (ATP)) was classified as appropriate when initiated by ventricular tachycardia (VT) or ventricular fibrillation (VF) that was still present when therapy was delivered. Twenty-two patients (54%) in the Auckland cohort and 26 patients (55%) in the Wellington cohort received appropriate ICD therapy. There was no significant difference between the two groups (Table 2).
Table 2. Appropriate ICD therapy

<table>
<thead>
<tr>
<th>Variables</th>
<th>Auckland 75 or more cohort (n=41)</th>
<th>Wellington 70–74 cohort (n=47)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appropriate ICD therapy</td>
<td>22 (54%)</td>
<td>26 (55%)</td>
<td>0.88</td>
</tr>
<tr>
<td>Shock</td>
<td>16 (39%)</td>
<td>18 (38%)</td>
<td>0.95</td>
</tr>
<tr>
<td>&gt; more than 3 episodes</td>
<td>8</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>&gt; shock alone</td>
<td>4</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>ATP</td>
<td>18 (44%)</td>
<td>23 (49%)</td>
<td>0.64</td>
</tr>
<tr>
<td>&gt; 1-3 episodes</td>
<td>8</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>&gt; more than 3 episodes</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

Device-related morbidity comprised inappropriate ICD therapy, ICD implant complications, subsequent ICD generator and lead complications including lead failure and lead dislodgement. ICD therapy was deemed inappropriate when triggered by supraventricular tachycardia, sinus rhythm, T-wave oversensing, lead dysfunction or occurred after spontaneous termination of a ventricular arrhythmia while lead dislodgement was defined as movement of a lead necessitating another procedure for repositioning.  

Overall, there were 16 deaths (39%) in the Auckland cohort and 15 deaths (32%) in the Wellington cohort. All cause mortality and device related morbidity were comparable between the two groups (Table 3). Mortality rates at 1 year (2.4% for Auckland cohort vs 4.2% for Wellington cohort, p=0.64) and 2 years (14.6% for Auckland cohort vs 8.5% for Wellington cohort, p=0.23) post ICD implantation were not significantly different between the two groups (Figure 1).

Table 3. All-cause mortality and device-related morbidity

<table>
<thead>
<tr>
<th>Variables</th>
<th>Auckland 75 or more cohort (n=41)</th>
<th>Wellington 70–74 cohort (n=47)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>16 (39%)</td>
<td>15 (32%)</td>
<td>0.46</td>
</tr>
<tr>
<td>&gt; cardiac</td>
<td>9</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Device related morbidity</td>
<td>16 (39%)</td>
<td>20 (43%)</td>
<td>0.79</td>
</tr>
<tr>
<td>&gt; Inappropriate therapy</td>
<td>5 (12%)</td>
<td>9 (19%)</td>
<td>0.38</td>
</tr>
<tr>
<td>– 1 to 2 times</td>
<td>3</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>– more than 2 times</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&gt; Implant complications</td>
<td>5 (12%)</td>
<td>3 (6%)</td>
<td>0.35</td>
</tr>
<tr>
<td>&gt; Generator complications</td>
<td>5 (12%)</td>
<td>2 (4%)</td>
<td>0.17</td>
</tr>
<tr>
<td>&gt; Lead complications</td>
<td>1 (2%)</td>
<td>6 (13%)</td>
<td>0.08</td>
</tr>
</tbody>
</table>
We compared the survival and device related morbidity of a group of elderly patients (age 75 or more) receiving ICDs in Auckland with a younger subset of patients (age 70–74) receiving ICDs in Wellington and found no significant difference in terms of outcomes. However, the small sample sizes could have prevented any significant differences from being detected.

Compared to the cohort of patients age 75 or more receiving primary ICDs by van Rees et al\textsuperscript{3}, the Auckland cohort had a longer median follow up, higher percentage of appropriate therapy and appropriate shocks but lower 1-year mortality post appropriate shock only. However, a majority of patients in the Auckland cohort had a secondary ICD indication which may have been a factor in the better outcome (Table 4).

**Table 4. Comparison between Auckland cohort with Van Rees et al**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Auckland 75 or more cohort</th>
<th>Van Rees et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median follow up</td>
<td>4.25 years</td>
<td>1.6 years</td>
</tr>
<tr>
<td>Appropriate therapy</td>
<td>54%</td>
<td>28%</td>
</tr>
<tr>
<td>Appropriate therapy</td>
<td>39%</td>
<td>13%</td>
</tr>
<tr>
<td>1 year mortality post appropriate shock only</td>
<td>25%</td>
<td>35%</td>
</tr>
<tr>
<td>Primary ICD implant</td>
<td>24%</td>
<td>100%</td>
</tr>
</tbody>
</table>

The incidence of inappropriate shocks for the Auckland cohort was 9.8% which is higher than van Rees et al’s 8%.\textsuperscript{3} However, it is comparable with those reported in MADIT-II (11.5%)\textsuperscript{6} and SCD-HeFT (9.9%).\textsuperscript{7}
In conclusion, while the total number of patients is small, our data support the value of ICD implantation in carefully selected elderly patients as outcomes for patients of age 75 or more were similar to a subset of patients of age 70–74.

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**References:**

Elderly locums

Roger Ridley-Smith is to be congratulated on pointing out the increasing cost of reaccreditation and the inevitable increase in fees to the patient.¹ I agree with his opinion that the Medical Council has not thought through the consequences of its insatiable quest to protect itself from criticism that a medical mishap could be attributable to its lack of regulatory foresight.

No one criticises its aim in maintaining standards, but to impose on GPs not in a formal vocational training scheme (and previously practising under collegial supervision) the same criteria to achieve accreditation as an inexperienced overseas trained graduate, with a poor command of the English language, defies logical explanation. I’m sure it did not foresee the exodus of recently retired GPs, described in Dr Ridley-Smith’s letter, from the locum pool.

Council may not be aware that the bpaq programme is not universally accepted as a suitable competency test for potential locums. Those in many group practices responsible for assessing new locums or associates prefer the previously widely accepted regular collegial supervision process as a more reliable guide of competency.

These are debatable issues. Council remains, however, obdurate in its refusal to discuss the content of the programme, nor the reason for and the consequences of its introduction. It produces an annual report, but on the grounds of cost is not sent to each member. It does not have open meetings, certainly not an AGM, and is immune from scrutiny of the Official Information Act. Letters from the rank and file are seemingly ignored: the only contact I have had followed a complaint about its silence to the Minister of Health, who requested a reply with a copy to me.

We at the receiving end of all this deserve a better response. By its silence Council is treating neither the profession nor the public, both of whom it serves, as it should.

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Reference:

Children of the outer Cook Islands have lower BMI compared to their urban peers

Childhood obesity rates have risen globally and especially among Pacific Island children.¹

In the recent NZMJ publication² regarding obesity management, Carroll et al point to the need for changes in lifestyle at a personal level and note that adherences to the intervention and hence behavioural change is a predominant factor in success. However, Pacific Island children are also thought to be genetically large and this genetic predisposition may well weaken resolve to address lifestyle issues through effective intervention³ if obesity is seen as part of the Pacific Island phenotype and thus out of personal control.

This contribution of the Pacific Island phenotype could be put in perspective by comparing Pacific Island children who are not subject to urban lifestyle influences to genetically similar populations who are.

One of us (DS) in 2012 had the opportunity to observe children in the remote Northern Group of the Cook Islands. The islands are between 270 and 737 nautical miles from the most populated island of Rarotonga in the South. Some of these remote islands have no airstrip and are supplied by occasional freighters. There are no shops and the diet is essentially taro, coconut and seafood. The main imported items are rice and sugar. There is no distraction from regular exercise and no screen time. Most children were seen as part of a general physical check up and height and weight were measured. The impression was that these children were not obese.

Cook Island Māori are the predominant ethnicity in the Northern Islands (97%),⁴ making this group ethnically comparable to children in Rarotonga, and children identified as Cook Island Māori living in Auckland, New Zealand. We thus took the opportunity to compare the body mass index (BMI) of the Northern Island children with BMI of children in Rarotonga and Cook Islanders in Auckland.

The Northern Island data excluded children with medical conditions that may affect growth and those older than primary school age. Based on the 2011 census count, our sample of children represents around 40% of the population under 15 years. The Rarotonga data was collected by us from randomly selected primary school children. The Auckland Cook Island children data were from the Pacific Islands Family Study (PIF), a longitudinal study of Pacific Island children living in Auckland.⁵

For our comparison a single time point BMI from each Cook Island primary school aged child in the PIF was randomly selected. BMI were converted to age and sex corrected standard deviation scores (SDS) based on the World Health Organization (WHO) standards.⁶

On average the BMI SDS of the children living in the Northern Cook Islands was almost one SDS lower than that of Cook Island children living in Auckland, with children in Rarotonga in-between (Table 1). That children of the Northern Islands
have a higher average BMI than the WHO standard (zero) supports the notion that even with non-obeseogenic lifestyles, Pacific children have greater BMI than European, and other ethnicities.

Table 1. Mean body mass index standard deviation score (BMI-SDS) of Cook Islands children from the Northern Cook Islands, Rarotonga and Auckland

<table>
<thead>
<tr>
<th>Location</th>
<th>N</th>
<th>Female %</th>
<th>Age mean (y)</th>
<th>Age range</th>
<th>Mean BMI-SDS</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northern Cook Islands</td>
<td>172</td>
<td>49</td>
<td>7.3</td>
<td>(1.5–11.9)</td>
<td>0.72</td>
<td>(0.57–0.87)</td>
</tr>
<tr>
<td>Rarotonga</td>
<td>95</td>
<td>49</td>
<td>8.9</td>
<td>(3.2–14.3)</td>
<td>1.29</td>
<td>(1.08–1.50)</td>
</tr>
<tr>
<td>Auckland</td>
<td>213</td>
<td>61</td>
<td>7.3</td>
<td>(2.1–11.9)</td>
<td>1.62</td>
<td>(1.46–1.77)</td>
</tr>
</tbody>
</table>

Assuming these Cook Island groups are genetically similar, then the difference in BMI may be understood in terms of different environmental influences over and above genetic susceptibility.

These results are observational and we provide no direct evidence of lifestyle differences between the groups, but we think the data indicates that the further Cook Island children are away from the Northern Islands lifestyle the heavier they become, suggesting that lifestyle change in urban communities is worthwhile and could meet with success in managing Pacific Island childhood obesity.

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References:

Dominion Notes. Drink Bill

Excerpt from Dominion Notes published in NZMJ 1911 May;10(38):35–37.

The following is a summary of the main features of the Rev. Edward Walker’s statement showing the “drink bill” for 1910. For some years past Mr. Walker has annually calculated and published the Dominion’s expenditure on alcoholic liquors.

The drink bill of the Dominion for 1910, calculated as usual at per gallon rates on the quantity, which passed through the Customs and excise, amounted to £3,803,438, being an increase on the previous year of £175,310, or £3 13s. 1¼d per head of population, being an increase per head on the previous year of 2s. 0¾d.

The bill is like a barometer for showing the current spending powers of the people. Anyone may form his own estimate of what the figures would be if the increase, after passing the Customs and excise, of the quantity of liquor, by methods known to the trade; and the actual cost to the consumers, not at per gallon, but as sold across the bar, could be calculated.

Probably five million pounds sterling, and five pounds per head of population, or £25 per household, is less than was really spent on liquor in New Zealand last year. The Customs and excise revenue from it was £799,634.
Selective serotonin reuptake inhibitors during pregnancy and risk of autism

Selective serotonin reuptake inhibitors (SSRI) are increasingly used in the treatment of depression and anxiety disorders. Depression is common in pregnancy and SSRIIs are often prescribed in these circumstances. As SSRIIs cross the placenta there are potential foetal and infantile hazards. In particular, the risk of autism in the offspring has been raised.

This has prompted this cohort study of all singleton live births in Denmark from 1996 through 2005 (676,875), with follow-up through 2009. Information on maternal use of SSRIs before and during pregnancy and autism spectrum disorders diagnosed in the offspring was obtained from Danish population registries.

The conclusions were that the researchers “did not detect a significant association between maternal use of SSRIs during pregnancy and autism spectrum disorder in the offspring. On the basis of the upper boundary of the confidence interval, our study could not rule out a relative risk up to 1.61, and therefore the association warrants further study.”


Does perinatal probiotic supplementation prevent asthma and childhood wheeze?

Recent increases in asthma prevalence could be related to disruption of the infant gut microbiota and associated immune system dysfunction; therefore, perinatal probiotics have been proposed as a novel prevention strategy.

This report from Canada concerns a meta-analysis which examines this hypothesis. It analyses evidence from 20 randomised controlled trials evaluating probiotic supplements administered to mothers during pregnancy or to healthy infants during the first year of life. The primary outcome sought was doctor diagnosed asthma.

The median age at final follow-up was 24 months. The conclusions reached were that probiotic supplementation in pregnancy or infancy did not protect against asthma or childhood wheeze.

Salty food and hypertension

Excess salt intake increases blood pressure (BP). Identifying individuals with excess salt intake is, therefore, important for the prevention of hypertension. Measurement of urinary sodium excretion is a reliable method of evaluating salt intake. These researchers have recently reported that the frequency of salty food intake measured subjectively was positively associated with urinary sodium excretion in individuals who underwent a general health examination.

970 non-hypertensive subjects (mean age 44 years) were asked about their subjective frequency of salty foods intake (seldom, sometimes or always), and they were divided into three groups according to their answers. Hypertension was defined as systolic/diastolic BP ≥ 140/90 mmHg or use of antihypertensive medications.

At 4 year follow-up there were no significant differences in the incidence of hypertension in the three groups. Further investigations, particularly a longer follow-up time, may be interesting.