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This Issue in the Journal

Estimated community costs of an outbreak of campylobacteriosis resulting from contamination of a public water supply in Darfield, New Zealand
Ian Sheerin, Nadia Bartholomew, Cheryl Brunton

In August 2012, there was an outbreak of campylobacter in Darfield, NZ which was most likely caused by failure of the community water supply. This was entirely preventable. The outbreak caused an estimated 828 people to become sick, but there may have been as many as 1987 people affected. The estimated cost to the community was $714,500 with the largest portion of that being approximately $500,000 from lost production from time off work.

Determining the health benefits of poultry industry compliance measures: the case of campylobacteriosis regulation in New Zealand
Gail E Duncan

A cost–benefit analysis is undertaken of the food safety regulation of poultry production for the New Zealand domestic market and the reduction in foodborne campylobacteriosis following this. A societal perspective is taken to demonstrate that regulation brings both benefits and costs. A Cost Benefit Analysis (CBA) is applied to a Cost of Illness (COI) estimate of foodborne campylobacteriosis derived from three previous studies, combined with the cost data supplied by industry and the regulator. The benefit:cost ratio was remarkable, showing a good return from the combined efforts of industry and the regulator; in dollar terms a gain of at least $57.4 million annually. In summary the study demonstrates the high value to the New Zealand economy of investment in food safety compliance at the primary industry level.

Infective endocarditis in New Zealand: data from the International Collaboration on Endocarditis Prospective Cohort Study
Genevieve Walls, Stephen McBride, Nigel Raymond, Kerry Read, Christin Coomarasamy, Arthur J Morris, Stephen Chambers, David Holland, David R Murdoch

This paper aims to describe the presentation, microbiology and management of infective endocarditis (infection of the heart valves) in New Zealand. It analyses data from New Zealand patients collected as part of a larger international study known as the International Collaboration on Endocarditis Prospective Cohort Study. Endocarditis in New Zealand and international patients in the modern era presents differently from older, classical descriptions of the disease, and is usually an illness of shorter duration than previously described. Many findings of the international study apply to endocarditis in New Zealand, although the most common cause of endocarditis in New Zealand was still organisms called ‘viridans streptococci’, which
is different from the international cohort. The paper will provide a reference for local New Zealand practitioners managing patients with endocarditis.

Is refractory angina pectoris a form of chronic pain? A comparison of two patient groups receiving spinal cord stimulation therapy
Nick Pak, Daniel A Devcich, Malcolm H Johnson, Alan F Merry

Spinal cord stimulation therapy is often used as an option for treating chronic pain conditions and refractory angina pectoris. Psychological-based pain management interventions may also be utilised, although typically less often for chronic refractory angina pectoris. Some studies, however, have shown that such programmes can be effective for treating chronic refractory angina pectoris, which suggests that it might be useful to view this condition as a form of chronic pain. In this study, we measured a range of important psychological and pain-related characteristics in patients receiving spinal cord stimulation therapy with either chronic pain or chronic refractory angina pectoris. Results showed that most self-rated psychological and pain-related characteristics were comparable across the two patient groups, which gives some support to the view that refractory angina can be considered as a form of chronic pain and may therefore be amenable to psychological-based treatment programmes.

A clinical psychologist in GP-Land: an evaluation of brief psychological interventions in primary care
Sunil Dath, Christine Yang Dong, Malcolm W Stewart, Eileen Sables

Our East Auckland-based study showed that psychological interventions in just four sessions make a large difference for general practice patients with mental health difficulties such as depression and anxiety disorders. The patients’ mental health and quality of life improved markedly after therapy. Weekly intervention sessions were held at the Pakuranga Medical Centre. The interventions were based on Cognitive Behavioural Therapy and were tailored to address the particular needs and situation of each patient. General practice staff reported that, as well as the value of therapy for the patients, having the psychologist present in their practice also improved communication with the mental health services and assisted them to help other patients with mental health difficulties. Previously, GPs only had been able to refer the most severely affected patients to mental health services due to constraints on hospital care budgets. This briefer, more targeted psychological intervention meant that patients with significant but less severe mental health problems had access to a service that helped them cope with life, and in many cases stay in work. There is strong interest from government in increasing the availability of psychological therapies (sometimes called talking therapies) and to improve how primary care and mental health services work together. This study shows a practical way these goals can be achieved.
A retrospective case series of 44 patients with community-acquired Staphylococcus aureus pneumonia
Darren Bowles, Kyle Perrin

This case series showed that pneumonia due to Staphylococcus aureus is often associated with the need for admission to the Intensive Care Unit (ICU), complications such as lung cavitation, and death. Staphylococcal pneumonia was more commonly seen in patients from socially deprived residential areas and with pre-existing health problems such as emphysema and kidney disease.
Campylobacteriosis in New Zealand: room for further improvement

Rebekah Lane, Simon Briggs

Campylobacteriosis is the most common notified disease in New Zealand with the 7031 cases in 2012 comprising 35% of all notifiable diseases reported to Public Health Services nationwide.\(^1\)

Its incidence in New Zealand peaked at 396 reported cases per 100,000 population in 2003;\(^2\) the highest rate reported by any developed country.\(^3\) The incidence remained at this level until 2006 when it dropped rapidly over a 2-year period to 157 reported cases per 100,000 population in 2008; it has remained stable since this time with 159 reported cases per 100,000 population in 2012.\(^1\)

The current incidence in New Zealand is still 1.5 to 3 times higher than reported incidence rates in Australia, England and Wales, and several Scandinavian countries in the early years of this century.\(^3\)

Campylobacteriosis is the most common cause of bacterial gastroenteritis worldwide. Taking into account the described ratio of reported to unreported cases of 9.3,\(^4\) it is very likely that more than 1% of New Zealanders currently acquire this disease every year.

Two articles in this issue of the NZMJ highlight the impact of campylobacteriosis and interventions that have recently reduced its incidence in New Zealand.

The first article, by Ian Sheerin, Nadia Bartholomew and Cheryl Brunton,\(^5\) describes a significant outbreak of campylobacteriosis in Darfield, Canterbury and estimates the economic costs of this outbreak to the community. The authors state that the likely source of the outbreak was faecal contamination of the town’s water supply compounded by the failure of a chlorination system. They estimate an economic cost to the community of between NZ$700,000 and $1.25 million.

The second article, by Gail Duncan,\(^6\) describes a cost benefit analysis of the introduction of the food safety regulation of poultry production in New Zealand and the reduction in campylobacteriosis that followed.

The Campylobacter Strategy was introduced in 2006 by the then New Zealand Food Safety Authority (NZFSA), now part of the Ministry for Primary Industries. This risk management strategy included the development and implementation of microbiological surveillance activities, the development and implementation of operational guidelines and control measures, communication between all involved parties and international collaboration.

The introduction of these measures is considered to be responsible for the more than 50% reduction in the incidence of campylobacteriosis that followed.\(^7\) The author states that the combined efforts of the NZFSA and the poultry industry resulted in an annual gain of at least $57 million to the New Zealand economy.
Campylobacter species, most commonly C. coli and C. jejuni, are commensal organisms found in the gastrointestinal tracts of birds, swine and cattle. These reservoirs are the usual source of infection in humans.

Consumption and handling of fresh poultry is thought to be the main source of human infection. Other sources include other contaminated foods (such as beef, pork and unpasteurised dairy products), direct contact with animals (either domesticated or farm stock) and environmental transmission from drinking and recreational waters.

The role that poultry plays in this illness in New Zealand was emphasised by a large national case-controlled study that found that campylobacteriosis was strongly associated with recent consumption of raw or undercooked chicken (OR 4.52, 95%CI 2.88–7.10) or chicken eaten in a restaurant (OR 3.8 5; 95%CI 2.52–5.88).

Campylobacter species infection is acquired by faecal–oral transmission. It results in illness characterised by fever, abdominal pain and diarrhoea. The illness is almost always of mild to moderate severity and is self-limiting, typically resolving without antibiotic treatment, within 5–6 days.

The mainstay of the management of campylobacteriosis, as with most enteric infections, involves adequate rehydration and electrolyte replacement. Antibiotics have very little impact on the duration and severity of symptoms and so are only rarely indicated. In a meta-analysis of the effect of antibiotic treatment on the duration of symptoms in patients with campylobacteriosis, 11 randomised, placebo-controlled trials were analysed. These trials assessed the impact of treatment with erythromycin (n=6), ciprofloxacin (n=3) or norfloxacin (n=2) which was started a mean of 3.5 days after the illness began. Pooled data from these trials showed a mean reduction of only 1.3 days of diarrhoea in the treatment group when compared to placebo. Antibiotic treatment of “real world” patients with campylobacteriosis, who may not receive treatment as early as those in the above trials, may well have even less benefit than this. If treatment is thought to be required, then the antibiotic of choice is erythromycin.

Of major concern, is the alarming increase in the rate of fluoroquinolone resistance found in human isolates of Campylobacter species in a number of countries. This has corresponded with the increased use of fluoroquinolones for growth promotion and the prophylaxis and treatment of infection in animals, particularly poultry, by the veterinary and food production industry. While fluoroquinolones are used by the veterinary industry in New Zealand, they are not licensed for use as growth promoters.

The prevalence of antimicrobial resistance in Campylobacter species isolated from humans in New Zealand remains low; 1.4% for erythromycin and 5.1% for fluoroquinolones during 2012. In comparison, the prevalence of fluoroquinolone resistance in Campylobacter species isolated from humans was 75% in Spain and 84% in Thailand during the 1990s.

As we close in on the 35th anniversary of campylobacteriosis notifications in New Zealand we should acknowledge the recent significant reduction in incidence of this disease that has resulted from the introduction of the NZFSA’s risk management strategy.
The two articles in this issue of the NZMJ have emphasised the direct and indirect costs associated with this infection and serve as a timely reminder that further significant reductions in the incidence of this disease in New Zealand are still required if we are to reach incidence rates seen in other comparable developed countries.

Given the very limited benefit of antibiotic treatment and the increasing rates of antibiotic resistance, prevention of campylobacteriosis must be the goal.

**Competing interests:** Nil.

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Infective endocarditis: trends in the disease and how we study them

Andrew J Kerr, Michael J A Williams

Infective endocarditis (IE) has long fascinated medical students, physicians and surgeons. Unfortunately, unlike many areas in cardiology where large scale randomised trials have helped to shape our management, such studies have not been performed in IE for a number of reasons. These include the infrequency of the disease, difficulties in making a definitive diagnosis, and variability in presentation.

In the most recent European Society of Cardiology Infective Endocarditis Guidelines there are no recommendations based on Level of Evidence A, i.e. multiple randomised clinical trials. Evidence to guide changes in treatment and outcomes has therefore come predominantly from observational studies of increasing sophistication. As in other areas of medicine these have evolved from single centre experiences to larger, multi-centre, national and international cohort studies.

In New Zealand there are a series of cohort studies spanning the last 50 years. In 1981 John Ormiston, John Neutze, Trevor Agnew, Jim Lowe and Alan Kerr reported the Green Lane Cardiology experience (n=177 with mean age 36y) from 1959 to 1976. Subsequently further single centre experiences were reported from South Auckland (n=78), Dunedin (n=62) and Tauranga (n=47).

The first multi-centre New Zealand (NZ) cohort is reported in this edition of the NZMJ by Genevieve Walls, David Murdoch, and colleagues. They participated in the International Collaboration on Endocarditis Prospective Cohort Study (ICE-PCS) which prospectively enrolled 2781 patients at 58 sites in 25 countries between 2000 and 2005 and reported in 2009.

The current study in the Journal reports on the 336 patients (median age 60y) in this study who were enrolled in Auckland, Counties Manukau, Waitemata, Capital and Coast and Canterbury District Health Board hospitals. Notably this is only slightly fewer patients than the sum of all prior NZ cohorts, and when combined with the ICE-PCS procedures to ensure consistent and comprehensive capture, it represents the best snapshot of this disease performed to date in NZ. There is a wealth of clinically relevant data presented.

Although most patients were hospitalised within a month of symptom onset only a minority were hospitalised within a week. Presentation was varied and non-specific with 76% having fever >38°C and only 36% with a new or worsening murmur. Splinter haemorrhages were seen in 19% but other classic manifestations (Osler’s nodes, Janeway lesions) were rare, a finding relevant for teaching medical students. Two thirds had a predisposing abnormal (37%) or prosthetic heart (31%) valves, which is relevant for both prevention and early diagnosis strategies.

Prior rheumatic heart disease was surprisingly uncommon. Blood cultures and confirmatory echocardiography were the mainstay of diagnosis. Blood cultures were
positive in 94% of those cultured. Viridans streptococci were the most common organism closely followed by *Staphylococcus aureus*. No data on antibiotic treatment is available, but a third had surgery. In-hospital mortality was lower than in prior series or in the remaining ICE-PCS cohort at 6%, but 14% of patient had strokes and 17% other systemic emboli.

What has changed? When compared with the historical NZ cohorts several broad trends emerge although these comparisons are limited by several considerations including variable cohort inclusion criteria and changing definitions. Patients with IE are older with more pre-existing degenerative valve disease or prosthetic valve disease. They are more likely to have recent health care exposure (a quarter in ICE-PCS), be diagnosed earlier, and have *Staphylococcus aureus* as the causative organism.

Where we need to know more:

- Are outcomes in IE improving? The in-hospital mortality rate of 6% is lower than in the older NZ series where it was around 20%. Whilst this may represent an improvement in outcomes, the relative roles of earlier diagnosis, improved diagnostic tools (e.g. echocardiography)/treatment or selection bias is unclear. Notably the in-hospital mortality in the international ICE-PCS cohort of 18% was similar to the rate of 16% reported in a review of 26 reports published between 1993 and 2003, suggesting that internationally in-hospital mortality is unchanged. At this time there is not sufficient evidence to conclude that in-hospital mortality has reduced in NZ.

- How is the incidence of IE changing? The current report is the best available multicenter NZ data for the period 2000 to 2005, but it is not comprehensive and there are no other data to allow us to assess temporal trends reliably. If we are to improve outcomes we need more up to date and regular data regarding presentation, treatment variables and outcome.

- Would more aggressive prevention help? Are patients with high risk of IE, in particular those with prosthetic valves getting recommended antibiotic prophylaxis? Are at risk patients getting recommended regular dental reviews?

- What treatment? Recommended treatment is based mostly on prior observational data and clinical “common sense” and embedded in international guidelines. These include considerations such as duration of antibiotic therapy, when to operate in patients with mobile vegetations to prevent stroke, when to operate after embolic stroke has occurred, what valve types to use, how long to treat with antibiotics prior to surgery. Are these recommendations correct and are they being adhered to?

We believe that there is an opportunity to move beyond periodic audits of practice to take a more systematic registry approach to IE. In NZ, under the auspices of the Ministry of Health, electronic national cardiac registries have been implemented in 2013 at all NZ public hospitals which now collect data on all patients having cardiac procedures, including coronary angiography and percutaneous coronary intervention, and will soon include most patients having heart attacks, cardiac surgery and receiving devices to treat cardiac arrhythmias.
These registries are linked to comprehensive NZ national and regional data collections, which include diagnosis, outcomes, laboratory and pharmaceutical data. This combination of registry and national collection data will be a powerful tool to better understand and improve patient management.

Although it is conceivable that IE cases may be able to be identified from national collections through ICD10 codes, it is likely that a registry approach to prospectively collect a core data set on these patients, with linkage to national data sets would be most useful. Such a registry could then serve to monitor incidence, management and outcome across NZ and serve as a source for more in-depth and representative audits.

Competing interests: None declared.

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References:


Estimated community costs of an outbreak of campylobacteriosis resulting from contamination of a public water supply in Darfield, New Zealand

Ian Sheerin, Nadia Bartholomew, Cheryl Brunton

Abstract

Aim To estimate the economic costs to the community of an outbreak of campylobacteriosis in August 2012 resulting from contamination of a public water supply in Darfield, New Zealand.

Method Probable incidence of waterborne disease was estimated. Reported cases were scrutinised to identify symptoms, duration, hospital admissions and those in the paid workforce. Extra public health and local authority costs were calculated. Estimated time off work was multiplied by the average wage to obtain a conservative estimate of lost production. Sensitivity analysis was used to estimate unreported cases and their associated costs.

Results There were 138 cases of confirmed or probable campylobacter, of whom 46 sought a medical consultation. Taking into account the usual pyramid of non-notified cases, estimates of the population infected range between approximately 828 and 1987. The dominant societal cost is lost production from time off paid work. Forty-six per cent were in the paid workforce, indicating a total estimated economic cost of at least $714,527 but it could have been as high as $1.26 million, depending on estimates of unreported cases.

Conclusion The likely cause of the Darfield outbreak was faecal contamination of the water supply, which with a multi-barrier approach would have been entirely preventable. The results provide economic evidence to support upgrading of water supplies to provide safe water and prevent waterborne disease.

In August 2012, there was an outbreak of gastrointestinal disease in Darfield, New Zealand (NZ) which evolved into one of the largest outbreaks of waterborne disease ever recorded in NZ. The majority of infections were confirmed or probable campylobacteriosis.

The outbreak occurred in a context of concern that there are unsafe water supplies in a number of NZ communities, compounded by a lack of action by local authorities, as the drinking water suppliers, who are responsible by law to provide safe water.

In the Darfield outbreak, elevated levels of faecal indicator bacteria *Escherichia coli* (*E. coli*) were confirmed in the water supply by laboratory testing. Before the outbreak, the Selwyn District Council had reverted to a river water source because of problems with its deep bore water source, then failure of a chlorination system allowed the survival of *Campylobacter* in the Darfield water supply, causing infection.
In the 4-year period before August 2012, the background population incidence of notified campylobacter in the Darfield area averaged between 1–2 cases per month. In August the number of confirmed and probable cases dramatically increased, as shown in Figure 1.

**Figure 1. Onset dates of probable and laboratory confirmed campylobacteriosis cases associated with the Darfield water contamination**

The Selwyn District Council contracted independent consultants to review their water system. They have been subsequently working through a number of recommendations including providing an additional deep bore source of water, continuous chlorination of any surface water used as back-up, and updating of hygiene systems.

Darfield is a small town, which in 2012 had an estimated resident population of 1790. It is located on a major highway approximately 40 km west of the Christchurch urban area.

Campylobacteriosis is a well-recognised issue in NZ, which has been most commonly associated with consumption of undercooked chicken.\(^1,2\) Considerable economic costs of foodborne campylobacteriosis and other gastrointestinal illness have been documented in NZ.\(^2,3\) Public health action has achieved much improved recognition of the risks of under-cooked chicken and of foodborne disease.

Although there have been ongoing attempts to increase awareness of the risks of unsafe water supplies, there remain a number of communities where significant concern exists about the safety of drinking water. Failure to take remedial action to address this is often accompanied by debates about its costs, but there is a relative lack of evidence on the potential economic costs of unsafe water supplies.

This paper aims to estimate the economic costs to the community of an outbreak of disease resulting from the contamination of a public water supply in Darfield in
August 2012. This data should help to inform debates about investment in safer water supplies.

**Methods**

Costs to the community were estimated from cases of confirmed as well as probable campylobacteriosis reported to the Canterbury District Health Board in August and September 2012 (n=138). The case definitions were as follows:

A person who had been in Darfield between 14 July and 30 August 2012, who was not overseas during the 10 days before the onset of symptoms and for whom there was no medical or other likely explanation for their symptoms and who either had:

- Diarrhoea and/or abdominal pain with fever—for at least 1 day (probable case of campylobacteriosis); or
- Laboratory confirmed campylobacteriosis (confirmed case).

Previous international research has shown that reported cases of infectious gastrointestinal illness represent the tip of the iceberg as most cases remain unreported to health authorities. Recent relevant research in the UK found that for every case of campylobacteriosis reported to national surveillance there were approximately 9.3 cases in the community (95% confidence intervals 6.0 to 14.4) that were unreported. Lake et al estimated that for every reported case of acute gastrointestinal illness in NZ, there were 222 cases that were not reported (95%CIs 199–247). However, the majority of this unreported disease pyramid is viral in origin and the UK studies indicate that ratios for unreported campylobacteriosis are likely to be considerably lower.

Therefore, the UK estimates by Tam et al were used to estimate the number of unreported cases of campylobacteriosis associated with the Darfield 2012 outbreak. The numbers of laboratory confirmed and probable cases were multiplied by the 95% confidence interval estimates of the number of unreported cases (based on Tam et al).

Uncertainty exists around this estimate of unreported cases, therefore the results include sensitivity analysis to indicate possible costs if the ratio in Canterbury was greater than the unreported to reported case ratio of 9.3:1. Possible reasons for this uncertainty are explored in the discussion section below.

The Community and Public Health Division of the Canterbury District Health Board provided a database of the 138 confirmed and probable cases, which included data on age, gender, occupational status, use of health services, laboratory testing, and type and duration of symptoms.

Costs of laboratory testing ($36.62 per faecal test) were as supplied by the Canterbury District Health Board in 2012. Costs of the primary care visit included the patient fee (as advertised for Darfield Medical Centre) plus the government capitation subsidy (total cost per visit $56.34), apportioned assuming an average of four GP consultations per person per year, which has been found in NZ Health Surveys.

The average age of reported cases was 30 years (range 0 to 89). Therefore the capitation subsidy for 25–44 year olds (averaged for males and females) was used for the purposes of these estimates. In the Darfield outbreak, the proportion of men and women was approximately equal among reported cases. Our estimates included an allowance for transport costs to and from a medical centre. For this we assumed a conservative distance of six kilometres per person at the average vehicle mileage paid by the Canterbury District Health Board in 2012 (70 cents per km).

We followed the assumption of Scott et al (2000) who received advice that approximately 10% of infected individuals would either receive a prescription for or buy an electrolyte replacement over the counter (OTC). For the purposes of this project, the OTC cost for Gastrolyte™ was used ($19.99).

To estimate losses from time off work (lost production), people who reported being in the active paid workforce were included, while preschoolers, school children, students and retired people were excluded.

Sixty-three confirmed and probable cases were currently in the workforce (46%). For the purposes of this analysis, we followed the assumption by Scott et al that each non-hospitalised case would have 5 days off normal work. This is conservative in view of the duration of symptoms reported in Table 1.
The average weekly wage ($922) for salaries and wages before tax in NZ published by Statistics NZ\(^8\) was used to calculate lost production.

Intangible costs were not included in this analysis. In their analysis of the costs of foodborne acute gastrointestinal illness in NZ, Scott et al\(^3\) included an estimate for intangible costs, by calculating time off usual activities associated with the illness, and then multiplying by the average wage. While there are undoubtedly intangible costs of such illness, we have not included any in our estimates because of the problems in quantifying them. Hence, our estimates could be viewed as conservative. Similarly, we have not included any potential damage to tourism and the “clean, green image” that is often promoted for NZ.

Inpatient costs of admissions to Christchurch Hospital were estimated from the average cost per bed day in 2012, supplied by the Canterbury District Health Board ($920 per day for an acute medical bed), multiplied by the length of stay (in days).

Extra work hours spent by District Health Board public health staff in containing the outbreak were multiplied by the relevant wage rates to obtain a cost estimate of the staff time directly attributable to investigating the outbreak and the Darfield water contamination. This method provides a conservative estimate of the opportunity costs of work time spent by staff who would otherwise have been working on other important public health issues.

Our estimates do not include any work time, operating or capital costs for the Selwyn District Council to repair and/or upgrade any of the local water reticulation and storage system. This is based on the assumption that providing clean water is part of a District Council’s legal responsibility, as the drinking water supplier, so that in theory all water reticulation and treatment systems should be maintained routinely, despite this outbreak. However, we have included the costs of a consultant’s report (Opus), which was commissioned by the District Council.

We have also included an allowance for costs of additional staff time (personal communication, 2013) by District Council staff involved in responding to enquiries, issuing boil water notices and responding to the outbreak.

**Results**

There were a total of 138 cases of confirmed and probable campylobacteriosis reported during the August 2012 outbreak (Table 1). Forty-six people reported seeking a formal medical consultation (either doctor or nurse), and laboratory testing was undertaken for 35 people.

Thirty-five cases submitted stool samples of which 29 were positive for campylobacter. Twenty-three of these were *C. coli*, three of which also had *C. jejuni* isolated and one *Giardia*. Three cases were positive for *C. jejuni* only, and for three cases no further subtyping was done. All specimens (where tested) were negative for *Salmonella*, *Shigella*, *Yersinia*, rotavirus, norovirus and *E. coli* 0157.

Water samples taken from the well, residential water tank and Waimakariri River were all negative for *Campylobacter*. Sixteen sheep stool samples were taken, of which 25% were positive for *C. jejuni* and one was positive for *C. coli*. The *C. coli* strain was very closely related to the strain isolated in human cases.

The mean age of cases was 30 years, ranging from infants to 89 years of age. Men and women were affected equally. Cases commonly reported diarrhoea, stomach pain, vomiting and nausea (Table 1).

On average, symptoms lasted for approximately 5 days before they were cleared (range 1–28 days). One hospital admission directly related to campylobacteriosis was reported, requiring admission to an acute medical bed in Christchurch Hospital for 5 days, followed by discharge home with no further medical treatment required.
Table 1. Estimated costs to the economy of the reported* cases of campylobacteriosis August 2012 outbreak in Darfield

<table>
<thead>
<tr>
<th>Reported cases of confirmed &amp; probable campylobacter*</th>
<th>N</th>
<th>138</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases who sought medical consultations</td>
<td>N</td>
<td>46</td>
</tr>
<tr>
<td>Mean age (range)</td>
<td>years</td>
<td>30 (0.9–89)</td>
</tr>
<tr>
<td>Female</td>
<td>%</td>
<td>49</td>
</tr>
<tr>
<td>Notified cases in the active workforce</td>
<td>N (%)</td>
<td>63 (46%)</td>
</tr>
<tr>
<td>reporting diarrhoea</td>
<td>% (range in days)</td>
<td>97% (1–14)</td>
</tr>
<tr>
<td>Reporting stomach pain</td>
<td>% (range in days)</td>
<td>96% (1–28)</td>
</tr>
<tr>
<td>reporting vomiting</td>
<td>% (range in days)</td>
<td>26% (1–14)</td>
</tr>
<tr>
<td>reporting nausea</td>
<td>% (range in days)</td>
<td>60% (1–28)</td>
</tr>
<tr>
<td>Costs of primary care consultations</td>
<td>$</td>
<td>2592</td>
</tr>
<tr>
<td>Costs of laboratory tests and electrolytes</td>
<td>$</td>
<td>1558</td>
</tr>
<tr>
<td>Costs of hospital admission</td>
<td>$</td>
<td>6440</td>
</tr>
<tr>
<td>Extra public health staff time costs</td>
<td>$</td>
<td>7712</td>
</tr>
<tr>
<td>Costs of lost work</td>
<td>$</td>
<td>58,086</td>
</tr>
<tr>
<td>Total economic costs for reported cases</td>
<td>$</td>
<td>75,212</td>
</tr>
<tr>
<td>Estimated extra costs incurred by local authority</td>
<td>$</td>
<td>95,000</td>
</tr>
</tbody>
</table>

Notes: $ are in 2012 NZ$ values; *reported cases includes both confirmed and probable cases.

The majority of people with campylobacteriosis endure the condition in the community, with limited input from formal health services. The estimated costs of primary care consultations, laboratory tests and of the hospital admission are shown in Table 1.

Together with lost production from lost work, the direct economic costs of notified cases are estimated at $75,212. Approximately 77% of these are costs of lost production from time off work. Forty-six percent of confirmed and probable cases were people in the paid workforce (Table 1), so just over half were preschoolers, students, retired people or who those not in paid work.

The district council incurred additional direct costs of approximately $95,000, comprising an expert report from an engineering consultancy, as well as additional staff time for dealing with enquiries and issuing boil-water notices.

Research has established that in gastrointestinal disease outbreaks, reported cases represent only the tip of the iceberg and there is usually an underlying disease pyramid of unreported cases, of people who endure the symptoms without seeking consultations from formal primary health care services.4,5,7

If we use the best published evidence that there are an estimated 9.3 cases of unreported campylobacteriosis for every one reported case,5 the “best estimate” economic costs total approximately $714,528, which includes $170,212 of costs directly attributed to reported cases, plus $544,316 estimated costs of lost production from time off work (Table 2). However, the 95% confidence intervals reported by Tam et al5 indicate a possible range from $521,383 to $938,000, including estimated lost production in Table 2.
Table 2 shows that applying the Tam et al.\textsuperscript{5} ratio of unreported to reported cases (i.e. 9.3:1), provides an estimate of the population affected by the Darfield outbreak of 1283.

Tam et al.'s estimate was obtained from a UK study, which could potentially underestimate unreported campylobacteriosis in NZ, when it is considered that there are considerable general practice co-payments which provide a deterrent to primary care consultations in this country. Such co-payments do not exist in the UK. Also, cases are likely to include non-residents travelling through and/or working in the area—i.e. not restricted to residents of Darfield.

Hence a sensitivity analysis investigated costs if the ratio of unreported to reported cases was twice the level reported by Tam et al.\textsuperscript{5} i.e. 18.6:1, indicating that the costs of lost production could be as high as $1.09 million (Table 2). Adding the costs of directly reported cases from Table 1, would give an upper plausible total cost estimate of $1.184 million.

Table 2. Total estimated cases and economic costs, including unreported campylobacteriosis associated with the August 2012 outbreak in Darfield

<table>
<thead>
<tr>
<th>Variables</th>
<th>Using estimate by Tam et al\textsuperscript{*} (9.3 for every 1 reported case)</th>
<th>Lower 95% CI\textsuperscript{*} (6.0:1)</th>
<th>Upper 95% CI\textsuperscript{*} (14.4:1)</th>
<th>Sensitivity analysis – if the ratio was twice that estimated by Tam et al (18.6:1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases</td>
<td>N</td>
<td>1283</td>
<td>828</td>
<td>1987</td>
</tr>
<tr>
<td>In active workforce</td>
<td>N</td>
<td>590</td>
<td>381</td>
<td>914</td>
</tr>
<tr>
<td>Costs of lost production</td>
<td>S</td>
<td>544,316</td>
<td>351,171</td>
<td>842,811</td>
</tr>
</tbody>
</table>

Notes: *From Tam et al\textsuperscript{5}  

Discussion

These estimates indicate that the majority of economic costs to the community of this outbreak were from lost work time from people in the paid workforce taking time off work. This is similar to the conclusions of Scott et al.\textsuperscript{3} reporting on societal costs of foodborne disease.

These estimates should be viewed as conservative when it is considered that they do not include any allowance for non-paid work such as voluntary work, caring for others, lost school days, lost leisure time or social events. Also, our estimates do not include costs that employers may have incurred if they had to replace sick employees—e.g. those who may be teachers, in the health workforce or in industries needing replacement staff.

This study did not attempt to include any value for intangible costs such as suffering, pain, or lost social or leisure opportunities. These are valid costs, but methods for valuing them are subjective, controversial and may be open to criticism. In excluding intangible costs, we are not avoiding them—rather it indicates that our cost estimates are conservative.
This study has used the best published international evidence on the ratio of unreported to reported cases of campylobacteriosis (the Tam et al\(^5\) estimate of 9.3:1).

The most relevant NZ study is by Lake et al\(^7\) who estimated that there were 222 unreported cases in the community for every reported case of acute gastrointestinal illness. This estimate was made from the number of reported cases throughout NZ in 2006, from a nationwide telephone survey, and from a survey of community and hospital laboratories.\(^7,9\) They found that 22% of all people with acute gastrointestinal illness (AGI) had consulted their general practitioner (GP). However, only approximately 0.4% of community cases ultimately result in reporting of a case of notifiable disease to public health authorities.

Other research in the UK found that the majority of community cases of AGI are due to viruses and that the probable incidence of unreported cases of bacterial illness is smaller: for campylobacteriosis, it was estimated at 9.3 unreported cases for every reported case.\(^5\) An earlier UK study drew similar conclusions.\(^6\)

The Lake et al\(^7\) estimates are of a similar order of magnitude to other international studies, for example Majowicz et al\(^4\) for Ontario, Canada, and Tam et al\(^5\) for the UK. However, Tam et al\(^5\) found that the unreported disease pyramid for viral illness was much greater than for bacterial illness, of which Campylobacter is the most common.

Accordingly, in this study we have used the ratio reported by Tam et al\(^5\) (9.3 unreported cases for every reported one) as the most relevant to the Darfield water contamination.

Another possible reason for uncertainty as to the validity of applying UK estimates to a NZ situation is the possibility of the primary care co-payment as a potential barrier to seeking medical advice, especially when there might be public advice that a watchful waiting approach might be preferable to active medical intervention.

In the UK, there are no GP co-payments to provide such potential financial deterrents. This ratio of unreported to reported cases is the greatest potential source of uncertainty to our estimates, therefore we have included a sensitivity analysis that showed that if the ratio of unreported to reported cases was twice that found by Tam et al,\(^5\) i.e. if it was 18.6 cases for every reported case, the total estimated economic costs of the Darfield water contamination would have been $1.26 million.

There is yet another reason why our costs are probably conservative. We took into account only cases of campylobacteriosis and we made no allowance for infection with multiple pathogens. Waterborne outbreaks commonly involve more than one pathogen, as the three cases in this outbreak who were infected with more than one subtype of *Campylobacter* and the one who also had *Giardia* illustrate. The costs of treatment and the risks of complications, including need for hospitalisation, will vary for different diseases.

This estimate does not include any allowance for the Selwyn District Council to upgrade its water supply. This was not included because it is a statutory requirement for local authorities, as drinking water suppliers, to provide a safe water supply—i.e. there should have been a safe water supply to prevent any such outbreak.

A further point to note is that Darfield is a small town, but it is located on a significant highway, close to the Christchurch urban area and there is a large travelling public
who may not necessarily reside in Darfield, but who travel through there for work, study and leisure. Therefore, it is likely that those affected by the outbreak in Darfield will have included non-residents. There is some evidence to support this contention.

There was a rise in reported cases of campylobacteriosis in other parts of Canterbury (mostly in Christchurch city) that was coincident with the rise in reported cases from the Selwyn District (Community and Public Health Surveillance Unit data). This increased regional incidence lasted until mid-September and differed from the usual seasonal pattern of a “spring peak” in incidence of campylobacteriosis, which usually begins later.

The combination of events strongly suggests that contamination of the water supply was the source of the outbreak. Immediately before the outbreak, there were periods of heavy rainfall, run-off and flooding in the river (Figure 1). Without any filtration or chlorination, pathogenic organisms would have been able to enter and survive in the drinking water supply.

A common risk exposure amongst reported cases was having drunk unboiled water from the local water supply. Although it does not appear to have occurred in this case, the possibility that contaminated water could be used in commercial food preparation, thereby contaminating foodstuffs and subsequently resulting in foodborne infection of an even larger group of people, could have significant and widespread economic and commercial impacts.

There is continuing concern, publicity and debate about potentially unsafe water supplies in a number of NZ communities. This situation is compounded by relative inertia and reluctance to address these problems, at least in part because of the costs of upgrading and protecting water supplies.

Lessons should be learned from the 2012 Darfield outbreak, which was costly to the community as a whole and which was entirely preventable. We also note that the water contamination in Darfield was contained within a tight timeframe, with reported campylobacteriosis cases peaking in mid-August and a safer water source restored before the end of August.

Other communities with unsafe water supplies elsewhere sometimes experience much longer periods before adequate preventive measures are instituted and such longer periods of water contamination are likely to incur greater costs to these communities.

The cost estimates from our study provide evidence to inform decisions about upgrading water supplies and decreasing risks to public health.

**Competing interests:** Dr Sheerin was involved in peer reviewing a consultant’s report on the Darfield outbreak by Sapere Research Group who were contracted independently by the Ministry of Health to undertake a similar costing analysis.

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Acknowledgements: The authors thank the staff of the Community and Public Health Division of the Canterbury District Health Board as well as Selwyn District Council staff for discussion and information; Sapere Research Group for their involvement in research, discussion and feedback; and the Canterbury District Health Board for their permission to publish this article.

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References:

Determining the health benefits of poultry industry compliance measures: the case of campylobacteriosis regulation in New Zealand

Gail E Duncan

A paper derived from her 2012 thesis *The Economic Benefits of Food Safety Regulation*, Otago University [http://hdl.handle.net/10523/2463](http://hdl.handle.net/10523/2463)

And following on from her paper presented at the NZAE Conference, June 2011.

**Abstract**

I undertake a cost benefit analysis of the food safety regulation of production of poultry for the New Zealand domestic market and the reduction in foodborne illness following this. I take a societal perspective to demonstrate that regulation brings both benefits and costs. I derive a cost of illness (COI) estimate of foodborne campylobacteriosis from three previous studies. I apply a cost benefit analysis (CBA) to this estimate, combined with the cost data supplied by industry and the regulator. The benefit:cost ratio was remarkable, showing a good return from the combined efforts of industry and the regulator in reduction of campylobacteriosis; in dollar terms a gain of at least $57.4 million annually. In summary the study demonstrates the high value to the New Zealand economy of investment in food safety compliance at the primary industry level.

Expert reports to Food and Agriculture Organization/World Health Organization (FAO/WHO) conclude there are limited data quantifying and assessing the commercial application of interventions at primary production level in the poultry industry.

The effects of interventions applied in a commercial setting on reducing the prevalence of contamination by *Campylobacter* on broiler carcasses have not been quantified.\(^1\) There has been no validation of interventions, or any costing of such interventions, or quantifying of population health gains expected from them.

This study attempts to add to literature in this area by addressing the value of preventative measures at the primary industry level to reduce disease.

The disease problem was a rising epidemic of campylobacteriosis in New Zealand commencing in the 1980s with numbers around 300 and reaching a peak of 15,873 reported cases in 2006. It seemed there was no end in sight to the increase in cases.

In August 2006, the New Zealand Food Safety Authority (NZFSA) (the regulator; now within the Ministry for Primary Industries [MPI]) was developing the *Campylobacter* Strategy to determine risk factors and actions to be taken by the poultry industry to effect reductions in the foodborne illness *Campylobacter*. Soon after this, in early 2007, initial research findings identified that the consumption of poultry meat was related to 50% of *Campylobacter* cases.\(^2\)
Poultry supplied for New Zealand consumers is principally sourced from the New Zealand poultry industry. Consumer demand for poultry had been unaffected as it is a principal source of affordable protein. Sales had in fact increased and remained high from 2004. There had been no product withdrawals.

In April 2007 testing of poultry carcass rinse samples for *Campylobacter* enumeration at the end of primary processing commenced in addition to the existing (National Microbiological Database) NMD compliance programme which included routine *E. coli* (generic) enumeration and *Salmonella* presence/absence testing.

By April 2008 a *Campylobacter* performance target had been developed by the regulator. However over this period the poultry industry had been developing interventions based on a series of activities that reduced the pathogen loading on the bird on farm and through primary processing.

The immediate health benefit from actions undertaken by industry to meet the new compliance standards applied in 2007 and 2008 was a 58% reduction in campylobacteriosis notifications in New Zealand.

To evaluate the benefit in dollar terms, an estimation of the cost of illness (COI) of campylobacteriosis at the beginning of 2007 was derived from three recent studies by Scott et al., Cressey and Lake, and Gadiel. The COI was estimated at $99m in 2007 dollars. It was comprised of $6.2m in total health costs and $93m indirect costs.

Industry costs of capital investment over 2007/2008 was $2.014m, and increased operating costs, including chemicals were determined to be $0.88m. The new compliance programme also imposed additional annual costs on government which were estimated at $0.95m in 2009 dollars or $0.89m in 2007 dollars.

An economic cost benefit analysis (CBA) was undertaken with the following assumptions:

- Linear relationship between the number of cases and the estimated COI;
- The benefit accrues each year; and
- The reduction in cases is maintained by the compliance programme for at least 10 years (a 10-year horizon).

The CBA, on these assumptions, resulted in a strikingly high benefit:cost (B:C) ratio of 25.74, for a discount rate of 10% per annum and an internal rate of return (IRR) of 1925%.

**Brief history of *Campylobacter* in New Zealand**

Campylobacteriosis notifications in New Zealand increased steadily from 1980–2003 and were examined to determine if it was real as opposed to a surveillance artefact. The likely causes of the increasing rate were unclear.

Campylobacteriosis is a gastrointestinal illness caused by infection with *Campylobacter* bacteria. The onset of disease generally occurs 2 to 5 days after ingesting the bacteria and the most common clinical symptoms are diarrhoea, nausea and vomiting lasting 3 to 6 days. The main route of transmission is foodborne.
By the early 2000s, poultry product was being identified as a major source of campylobacteriosis in New Zealand. A high proportion of poultry product at retail was known to be contaminated by Campylobacter but proper cooking practices and care in handling were thought to be adequate measures to deal with this.\(^7\)

By the late 1990s and early 2000 independent research determined that a significant foodborne source of Campylobacter could be poultry. Further research supported this.\(^9\)

In 2009 the New Zealand Medical Journal referred to ‘a substantially foodborne epidemic of Campylobacter infection’ and updated on New Zealand’s largest ‘common source outbreak’\(^10\).

The Campylobacter epidemic in New Zealand received less attention than it warranted as there was public, scientific and government policy perception that the disease was ‘endemic’, \(^11\) a natural part of poultry final product.\(^12,13\) Case control studies, risk modelling studies and literature reviews were not sufficient to sway earlier action to support interventions by industry and government to reduce levels in Campylobacter in that poultry final product.

**Methods**

**Design**—To examine the economic justification of this regulatory intervention costs to the industry and the regulator, the cost of illness and any resulting health benefits were estimated. It appears to be the first time in New Zealand that industry compliance costs have been assessed in relation to reduction of the disease burden.

**Economic analysis**—To investigate the above I employ a CBA. The advantage of using a CBA is that the economic effectiveness of interventions applied by industry to ensure compliance with the microbiological programme can be determined.

In this case the proposals for more stringent monitoring went ahead without prior certainty, or pre-knowledge of the extent of the benefit to be expected. The CBA is a retrospective analysis.

Ten years is applied as the net present value (NPV) period as this would be the expected time a compliance programme would be undertaken before there were any major changes or applications.

A real discount rate of 10\% per annum as recommended by Treasury is applied.\(^14\) The discount rate is applied to all current and future costs and benefits to give a discounted NPV after deducting the discounted cost from the discounted benefit.

**Costs included in this study**—Three tiers of costs are investigated. Firstly the cost to industry of implementing the compliance programmes and attempting to change practices and processes to reduce the prevalence of Campylobacter in the final product for the market. Secondly the costs to the regulator to undertake and continue oversight of the compliance programme. Thirdly the cost of illness in New Zealand due to campylobacteriosis is estimated from previous studies.

For the purpose of the CBA some costs are excluded; these are: GST (goods and services tax) as GST is a transfer payment, and the capital, financing and depreciation costs as they do not correspond to actual resource use and are implicitly implied in the discount rate. However the regular maintenance of equipment undertaken by industry to improve process and microbiological outcomes are a real use of an economic resource, not the same as depreciation, and are included.

Sunk costs have occurred before the appraisal period and have no value for an alternative use. Sunk costs would include the initial Poultry Industry Association of New Zealand (PIANZ)/New Zealand Food Safety Authority (NZFSA) consultations to determine a way forward for the Campylobacter Strategy.

**Benefit**—The benefit is principally measured as reduction in health care costs, which will include reduction in loss of life or disability.

In 2006 there were 385.6 Campylobacter cases per 100,000. By the end of 2008 this had fallen to 166.3 per 100,000.\(^15\) This was a 58\% reduction in notifiable cases based on total case figures derived from the
final 2006 and 2008 figures. The principal factor that contributed to this decline was the implementation of the *Campylobacter* Strategy which involved substantial investment by the poultry industry.16

**Sensitivity analysis**—To examine if costs have been underestimated or benefits exaggerated a sensitivity analysis is conducted across a range of discount rates from 0% to 10%.

**Results**

**Non-capital compliance costs**—When this research project commenced in January 2010 there were nine poultry broiler processors participating in the National Microbiological Database (NMD) compliance programme (noting that these nine were the total number of poultry broiler processors operating in New Zealand at the time). Eight of whom were members of the Poultry Industry Association of New Zealand Inc. (PIANZ). Companies A (with multiple processing sites) and B (one processing site), both members of PIANZ, contributed costing information.

The non-capital costs for companies A and B of the NMD after the *Campylobacter* programme was added in April 2007 were estimated as $260,630 in 2008/2009. Total costs for these two companies represent approximately 70% of the total industry costs (personal communication Michael Brooks, chief executive, PIANZ).

Table 1 below represents the non-capital costs of implementation of the poultry *Campylobacter* programme for all poultry broiler processors.

**Table 1. Component costs of the Poultry *Campylobacter* NMD programme, April 2008 – end of March 2009**

<table>
<thead>
<tr>
<th>COMPANY A/NMD after <em>Campylobacter</em></th>
<th>Labour</th>
<th>Service/cash cost</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Programme activities</td>
<td>$1800</td>
<td></td>
<td>$1800</td>
</tr>
<tr>
<td>Staff training</td>
<td>$3,000</td>
<td></td>
<td>$3,000</td>
</tr>
<tr>
<td>IT facilities</td>
<td>$15,600</td>
<td></td>
<td>$15,600</td>
</tr>
<tr>
<td>Review of results</td>
<td>$18,200</td>
<td></td>
<td>$18,200</td>
</tr>
<tr>
<td>Sampling</td>
<td>nil</td>
<td></td>
<td>nil</td>
</tr>
<tr>
<td>Laboratory analysis</td>
<td>$16,500</td>
<td></td>
<td>$108,030</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>$124,530</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td></td>
<td><strong>163,130</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>COMPANY B/NMD after <em>Campylobacter</em></th>
<th>Labour/Service/cash cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Programme activities</td>
<td>$12,000</td>
<td>$12,000</td>
</tr>
<tr>
<td>Staff training</td>
<td>$4,000</td>
<td>$4,000</td>
</tr>
<tr>
<td>IT facilities</td>
<td>nil</td>
<td>nil</td>
</tr>
<tr>
<td>Review of results</td>
<td>$20,000</td>
<td>$20,000</td>
</tr>
<tr>
<td>Sampling</td>
<td>$11,500</td>
<td>$11,500</td>
</tr>
<tr>
<td>Laboratory analysis</td>
<td>$45,000</td>
<td>$45,000</td>
</tr>
<tr>
<td>Responses</td>
<td>$2,000</td>
<td>$2,000</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>$97,500</strong></td>
<td><strong>$260,630</strong></td>
</tr>
</tbody>
</table>

A and B make up 70% of industry

The costs included the cost of PIANZ providing technical advice to industry, meetings with industry, the regulator, and researching interventions and public health consequences.

Table 2 below represents estimates of total industry compliance costs.
Table 2. Total industry compliance costs

<table>
<thead>
<tr>
<th>Total industry</th>
<th>2008/2009 after Campylobacter testing included</th>
</tr>
</thead>
<tbody>
<tr>
<td>All processors</td>
<td>$372,329</td>
</tr>
<tr>
<td>PIANZ</td>
<td>$5308</td>
</tr>
<tr>
<td>TOTAL</td>
<td>$377,637</td>
</tr>
</tbody>
</table>

Note: The ‘All processors’ figure is based on dividing the sum of processor A and B by a factor of 0.7.

Capital costs of interventions—In addition to the annual compliance costs, there were initial investments in interventions. These investments occurred between late 2007 and April 2008 after some trialling and consideration of the applicability to the New Zealand Campylobacter epidemic and poultry industry standard practices. Capital costs occurred concurrently with the implementation of the programme. Capital upgrades made to improve washing/chilling and/or post chill dip over 2007/08 prior to the imposition of regulatory responses cost company A $950,000, and company B $460,000 or a total of $1,410,000 for both companies. The total actual capital investment in the Campylobacter strategy by industry is unknown at this stage. Applying the 70% factor, the total capital investment is estimated at $2,014,000.

The on-going cost following the capital investment in intervention systems is the cost of chemicals; citric acid and sodium chlorite, estimated at $500,000 per annum overall (personal communication Roy Biggs17). Ongoing maintenance costs have been included with the annual costs of the compliance programme in Table 3 below along with capital costs.

Table 3. Ongoing and capital costs for industry

<table>
<thead>
<tr>
<th>Cost factor</th>
<th>Industry cost per annum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total industry 2008/2009</td>
<td>$377,637</td>
</tr>
<tr>
<td>Intervention system chemicals per annum</td>
<td>$500,000</td>
</tr>
<tr>
<td>Total NMD compliance 2008/2009</td>
<td>$877,637</td>
</tr>
<tr>
<td>Capital investment 2007/2008</td>
<td>$2,014,000</td>
</tr>
</tbody>
</table>

Estimation of cost to the regulator (NZFSA)—The government cost for the poultry Campylobacter Strategy was $950,000 in 20096 which converts to $885,000 in 2007 dollars.

Estimation of the cost of illness—Scott et al, estimated the total cost of foodborne illness in 1999 was $55m of which $40m (73%) could be attributed to Campylobacter.4 Cressey and Lake estimated total foodborne disease at $85.3m in 2005 of which an estimated $74m (87%) was associated with foodborne campylobacteriosis.5 Gadiel estimated the total cost of foodborne disease in 2009 in
New Zealand, excluding government and industry costs, was $131m with $36m due to foodborne campylobacteriosis (27%).

Gadiel stated that the relatively low percentage attributable to poultry in 2009 was likely to be due to interventions undertaken by the poultry industry in response to the *Campylobacter* Strategy following 2007.

All totals exclude government and industry preventative measures. Table 4 below represents the above figures of cost of illness in constant 2007 dollars for comparison.

**Table 4. Cost of foodborne campylobacteriosis converted to constant 2007 dollars $m**

<table>
<thead>
<tr>
<th></th>
<th>1999</th>
<th>2005</th>
<th>2009</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Scott et al</td>
<td>Cressey and Lake</td>
<td>Gadiel</td>
</tr>
<tr>
<td><strong>Total health</strong></td>
<td>1.6</td>
<td>4.8</td>
<td>2.2</td>
</tr>
<tr>
<td><strong>Total indirect</strong></td>
<td>47.4</td>
<td>72</td>
<td>31.5</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>49</strong></td>
<td><strong>77</strong></td>
<td><strong>34</strong></td>
</tr>
</tbody>
</table>

The estimated cost of campylobacteriosis from Cressey and Lake is the nearest chronologically to 2007, but will underestimate the cost of disease at the beginning of 2007 as the epidemic had just peaked at the end of 2006 at 15,900 notified cases per annum; or approximately 159,000 community cases. The 10:1 ratio has been generally applied on the economic analyses I have reviewed, including the recent UK Infectious Intestinal Disease study.

There is an expanding pyramid from the number of notified/reported cases in government surveillance publications, those persons who have laboratory testing undertaken, those persons presenting for medical attention for gastrointestinal illness and the remainder who have the illness but endure it without presenting for medical attention. The total number of campylobacteriosis cases in the community is estimated to be at least ten times that of notified/reported cases at the top of the pyramid.

The number of community cases estimated by Cressey and Lake was 123,000. Thus the likely cost due to foodborne campylobacteriosis at the start of 2007 would be $77m × (159,000 cases/123,000 cases) = $99m per annum. For the total health care costs only this would be $4.8m × (159,000 cases/123,000 cases) = $6.2m. The cost of illness estimates by Scott et al, Cressey and Lake, and Gadiel, and my $99m COI estimate for the beginning of 2007 are represented in Table 5 and Figure 1 below.
Table 5. Campylobacteriosis notifications and estimated cost in millions

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases per annum</td>
<td>8924</td>
<td>11,572</td>
<td>8,161</td>
<td>8,432</td>
<td>10,145</td>
<td>12,494</td>
<td>14,788</td>
<td>12,214</td>
<td>13,836</td>
<td>15,837</td>
<td>12,778</td>
<td>6,694</td>
<td>7,177</td>
</tr>
<tr>
<td>Estimated cost in million (constant 2007 dollars)</td>
<td>$49</td>
<td>$77</td>
<td>$99</td>
<td>$34</td>
<td>$49</td>
<td>$77</td>
<td>$99</td>
<td>$12</td>
<td>$15,837</td>
<td>$12,778</td>
<td>$6,694</td>
<td>$7,177</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1. Relationship between the estimated cost of campylobacteriosis and the number of notifications

To summarise; the estimated cost of disease attributable to *Campylobacter* at the start of 2007, prior to the poultry *Campylobacter* NMD programme is $99m of which $6.2m is total health care costs.

**Reduction in the disease burden, the ‘benefit’**—There was a 58% reduction in *Campylobacter* (15,873 cases in 2006 compared to 6,694 cases in 2008). Assuming a linear relationship between the number of cases and estimated cost of illness this implies:

- 58% savings of $99m for TOTAL = $57.4m, or
- 58% savings of $6.2m, total health (excluding indirect costs) = $3.6m

These benefits accrue each year.
Assumptions—My assumptions are:

- That the compliance programme will maintain this reduction in *Campylobacter* levels;
- The total community cases are 10 times the notifications and
- That there is a linear relationship between notifications and health effects.

The application of this common compliance standard would be expected to have an initial impact, level out and then possibly rise again as the focus shifts away from the original epidemic. A loss of industry experience of factors related to contamination of broiler carcasses, and development of maintenance problems associated with interventions or other standard processes can occur. This type of pattern is described as the Bathtub curve.21

I have not assumed a possible future rise in notifications. Notifications have remained at around the 2009 level of 7177 and are presently recorded at 7033 campylobacteriosis notifications for the year from 1 January to 31 December 2012. ([http://www.surv.esr.cri.nz/PDF_surveillance/MthSurvRpt/2012/201212DecNat.pdf](http://www.surv.esr.cri.nz/PDF_surveillance/MthSurvRpt/2012/201212DecNat.pdf)).

Cost–benefit analysis—I estimate that from $99m cost of *Campylobacter* illness at the beginning of 2007, the reduction in notifications led to only $41.6m being incurred over 2007/2008. That is a benefit of $57.4m. To estimate the benefits from the investment in the compliance programme a net present value (NPV) calculation was undertaken, using a 10% discount rate.

An internal rate of return (IRR) was also calculated as IRR is a widely recognised technique used by business, industry and public policy agents to estimate the financial return of an investment.

In estimating the benefits to health from investment in compliance represented in Table 6 below the IRR calculated to 1925% and the benefit:cost ratio was 25.74 which is a very high benefit:cost ratio.

To examine if the costs have been underestimated or benefits exaggerated I conducted a sensitivity analysis with discount rates from 0% to 10%.

The results of this sensitivity analysis show high B:C ratios regardless of discount rate; a further confirmation that the investment has a high payoff.

Removing indirect costs—To check if the benefits have been exaggerated calculations are repeated in the following tables, excluding indirect costs (termed as indirect non-health care costs by Cressey and Lake). This is on the basis of work by Koopmanschap et al22 suggesting that such costs may be seriously over-estimated when costed by Human Capital methods.
Table 6. Estimating benefits to health from investment in compliance

<table>
<thead>
<tr>
<th>Year</th>
<th>Confirmed notified cases per annum</th>
<th>Costs undiscounted</th>
<th>Costs undiscounted</th>
<th>Benefits undiscounted: the 58% reduction in cost of illness</th>
<th>Net Benefit</th>
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<tr>
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Total costs

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PV 13.70  352.70  338.99  NPV

Discounted 10%

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<tr>
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<th>Benefits undiscounted</th>
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<td>0.68</td>
<td>22.13</td>
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PV 13.70  352.70  338.99  NPV

Benefit cost ratio

B/C ratio = 25.74
Table 7. Sensitivity analysis of net present value (NPV)

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<th>Discount rate</th>
<th>10.0%</th>
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Using campylobacteriosis records from 2005 (adapted from Tables 8 and 9 of Cressey and Lake⁵) 97.3% of illnesses are short-term gastroenteritis lasting for 3 to 7 days. The more complex sequelae (reactive arthritis (ReA), Guillain-Barré syndrome (GBS) and inflammatory bowel disease (IBD)) make up only 2.7% of the estimated total cases.

With ReA work is usually uninterrupted. IBD usually puts persons out of work intermittently. Only for GBS could sick leave be considered significant at 90 to 123 days.⁵ This translates to only 0.02% of cases resulting in long-term sick leave. It is these long-term cases for which Koopmanschap et al’s criticisms have the most validity.

The effect of this on the benefit cost ratio can be demonstrated by conducting an NPV removing all the indirect costs where the 58% savings of $6.2m Total health (excluding indirect costs) = $3.6m as demonstrated in Table 8 below.

Comparing the benefits if only direct and non-direct health benefits are included as represented in Table 8 above; the IRR reduces to 63% and the cost benefit ratio to 1.61 (Table 8 above). The cost benefit ratio is still greater than 1.0, and the IRR remains high. These two economic tests still show a strong argument for the investment in compliance.

I then checked the sensitivity to discount rate again as demonstrated in Table 9.

Once again the sensitivity analysis shows little variation, implying a good investment proposition.
Table 8. Comparing the benefits if only direct and non-direct health benefits are included

<table>
<thead>
<tr>
<th>Year</th>
<th>Confirmed notified cases per annum</th>
<th>Costs undiscounted</th>
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Total costs
Sum 10.81 9.68 20.49 36.00 15.51

Discounted 10%

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PV 13.70 22.12 8.42 NPV

Benefit cost ratio
B/C ratio 1.61

Source data undiscounted for a 10 year horizon.
Table 9. Sensitivity analyses

<table>
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<tr>
<th>Discount rate</th>
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Discussion

Cost to the New Zealand economy—If the NMD Specifications as per 2001 (without Campylobacter measurement and regulatory controls) were continued it can be assumed that the rates of campylobacteriosis in New Zealand would have remained high.

The cost of this to the New Zealand economy can be conservatively demonstrated by calculating the area between the curves of actual reduction and anticipated continuation of campylobacteriosis at the average of yearly rate from 2000 to 2006 if the poultry Campylobacter NMD was delayed until 3 years later. The comparison in cost savings if Campylobacter NMD implementation was delayed is represented in the following graph.

Figure 2. Illustration of the potential cost to the economy if implementation of effective compliance measures is delayed

Note: ‘if delayed’ means if the implementation of NMD had been delayed a further 3 years.
The area between curves, represented by the shaded parallelogram, can be estimated by height of $57.4\text{m}$, the reduction of disease cost, times the base which is 3 years totalling $172.2\text{m}$ saved. This demonstrates the cost to the economy of delays in implementing effective responses to foodborne disease epidemics.

**Future targets**—A further 50% reduction in *Campylobacter* would result in a further $20.8\text{m}$ saved per annum ($99\text{m}–$57.4\text{m} = $41.6\text{m}$ divided by 2). The level of investment required by the poultry industry to effect or contribute to a further 50% reduction is however unknown.

Reviewing the attribution studies for 2006 and 2008 published by Lake, Hall and Ball in 2011 the proportion accounted for by an identifiable source was poultry ~60% in 2006 compared to ~40% in 2008. The estimated notifications attributed to other pathways remained relatively constant for both years (p51, Figure 11). This supports my assumption that the reduction in notifications was due principally to changes in the poultry processing. Other attribution pathways in New Zealand include pets, red meat, recreational water, drinking water, other animal contact, other foods and recreational exposure.

As about 40% of campylobacteriosis is still estimated to be sourced from poultry, further investment in poultry by both the regulator and industry is the most likely next step to deliver further significant health benefits. The final reduction in disease burden from *Campylobacter* will be related to improvements in farming practices in ruminants, particularly sheep and cows, environmental management of water catchments and reduction in unregistered water supplies.

**Conclusions**

The *Campylobacter* epidemic in New Zealand has been brought into check by poultry industry actions undertaken to achieve the targets of a compliance programme. There are costs associated with this programme that have been borne by industry. In a normal commercial environment the capital expenditure associated with reducing the level of *Campylobacter* on poultry carcasses at the end of slaughter and dressing would not have been invested. Neither could the effectiveness of the health outcomes have been predicted at the time. This paper shows the benefit:cost ratio of the capital investment in retrospect.

The preceding NMD programme with only *E. coli* and *Salmonella* testing requirements did not have any significant effect on industry practices related to *Campylobacter*. “On farm” did it for *Salmonella* biosecurity, but these feed/hatchery and on farm biosecurity changes seemed to do nothing for *Campylobacter*. It was changes during primary processing from crates used to carry the birds, equipment maintenance, evisceration procedures, chlorination levels in spin chill, use of interventions, smarter procedures and attention to detail of good management practice that worked for *Campylobacter*.

New Zealand’s campylobacteriosis epidemic had been identified as largely a foodborne disease, with poultry as a significant part of the problem. To fix the problem the level of *Campylobacter* contamination of poultry carcasses at end of slaughter and dressing needed to be measured. The NMD was/is useful in monitoring production every processing day with the data being submitted to the regulator.
Industry then re-evaluated investment in interventions or upgrading of processes with the intention of improving hygienic standards. At the time their investments were made neither they nor the regulator could have anticipated that it would make such a considerable difference to the number of Campylobacter notifications.

I found in the case of campylobacteriosis there are a very small number of fatalities and/or long-term complications. The economic burden to New Zealand is in the actual lost days (sick leave) and lost production of a large number of people off work for short periods. Thus even making allowances for the difficulties in getting accurate cost data, and recognising that savings from reduction on non-health indirect costs (also termed as ‘indirect non-health-care costs’ by other authors) are probably over-estimated substantially when using the Human Capital method approach, the economic CBA clearly demonstrates that the intervention was beneficial to New Zealand.

Estimates are conservative in that they do not capture the costs associated with suffering when a person is ill with Campylobacter or where their loss and the wider community’s loss will be larger than the loss of his or her earnings. To the extent this is true the effects of the NMD testing regime have been under-estimated independently of other sources of under- and over-estimation.

A clear positive linkage between the industry and regulatory cost of compliance and the internal social benefit to the New Zealand economy has been demonstrated. By addressing the primary source of this foodborne disease a more economically efficient outcome in the reduction of disease has been achieved. Further significant reductions in the disease burden can be expected if further focus and investment can be undertaken by the poultry industry in collaboration with the regulator.

To sum up an economic benefit has been clearly demonstrated by the application of a suitable compliance programme requiring investment by industry. This is supported by recent observations that Guillain–Barré Syndrome (GBS) hospitalisations are significantly correlated with campylobacteriosis notifications, and have declined since 2007.24

Note that campylobacteriosis is still not eliminated in New Zealand. Current levels of campylobacteriosis notifications are equivalent to approximately 7000 notifications per annum or 70,000 incident cases. New interventions need to be identified and developed to further reduce cases. These will probably be applied at the farm level, and involve further improvements in poultry primary processing.

Competing interests: Nil.

Author information: Gail E Duncan, Specialist Adviser, Ministry for Primary Industries, Wellington

Acknowledgements: I thank NZFSA (now incorporated in MPI) for granting me a Chief Executive’s Award in December 2007 to undertake this study. I am also grateful to Des O’Dea (Otago University Wellington, School of Public Health), Grant Scobie (Treasury), Poultry Industry Association of New Zealand Inc. (PIANZ), and poultry industry contributors.

Correspondence: Ms Gail Duncan, Ministry for Primary Industries, 25 The Terrace, Wellington 6140, New Zealand. Email: flutetunes@hotmail.com
References:


Infective endocarditis in New Zealand: data from the International Collaboration on Endocarditis Prospective Cohort Study

Genevieve Walls, Stephen McBride, Nigel Raymond, Kerry Read, Christin Coomarasamy, Arthur J Morris, Stephen Chambers, David Holland, David R Murdoch

Abstract

Aims The International Collaboration on Endocarditis Prospective Cohort Study (ICE-PCS) collected worldwide data on the presentation, management and outcome of infective endocarditis (IE). We present data from patients with endocarditis enrolled from New Zealand.

Methods Patients who fulfilled the Duke criteria for definite or probable endocarditis were enrolled from five district health boards: Auckland, Counties Manukau, Waitemata, Capital and Coast, and Canterbury, between June 2000 and September 2005.

Results There were 336 New Zealand patients enrolled in the ICE-PCS. Prosthetic valve endocarditis occurred in 31%. Underlying medical conditions were present in 28% of patients, but only 4% of patients had rheumatic heart disease. Forty patients (12%) had healthcare-associated endocarditis. Viridans streptococci were the most common cause of IE (32%), followed by *Staphylococcus aureus* (24%). Patients with *S. aureus* IE were more likely to present within a week of symptom onset than those with viridans streptococcus IE (OR 4.18, 95% CI 2.36–7.42). Surgery was performed in 33% of patients. In total, 20 patients (6%) died in hospital. Those with endocarditis caused by coagulase-negative staphylococci had an increased risk of death compared with those viridans streptococcus endocarditis (RR 4.7, 95% CI 1.2–17). The risk of stroke was higher in those with endocarditis caused by *S. aureus* and coagulase-negative staphylococci (RR 2.7, 95% CI 1.2–6.05, and 4.9, 95% CI 1.9–13, respectively).

Conclusion While viridans streptococci remain the predominant causative organisms of IE in New Zealand, many ‘traditional’ clinical and management aspects of this disease no longer apply. This paper provides a reference for local practitioners assessing and managing IE.

Despite medical advances, infective endocarditis (IE) continues to cause substantial morbidity and mortality. The International Collaboration on Endocarditis Prospective Cohort Study (ICE-PCS), a worldwide study of IE in the 21st century, included patients recruited from New Zealand. Regional variations in presentation and management of IE exist, and knowledge of these may assist local physicians.

In this paper we present the New Zealand data from the ICE-PCS study. This is the largest series of patients with IE in New Zealand.
Methods

Design—New Zealand study sites involved in the ICE-PCS were Auckland, Counties Manukau, Waitemata, Capital and Coast, and Canterbury District Health Boards (DHBs). The methods of the ICE-PCS are described in detail elsewhere,¹ and are briefly reiterated below. Study sites enrolled patients aged 18 years or over with a diagnosis of definite or probable IE, according to the modified Duke criteria.²

Study sites were required to fulfil the following criteria: minimum enrolment of 12 cases of IE per year, access to cardiac surgery, patient identification procedures ensuring consecutive enrolment and minimising ascertainment bias, high quality data, including query resolution, and Ethics Committee approval or waiver based on local standards.¹ Patients were enrolled between 1 June 2000 and 1 September 2005. One-year follow-up data were collected.

A standardised case report form of 275 variables was used. These variables and definitions are described elsewhere.²,³ Data were collected by investigators at the participating site during the index hospitalisation and then collated by the coordinating centre.

Definitions—Community-acquired IE was diagnosed within 48 hours of admission in a patient who did not fulfil the criteria for healthcare-associated infection. Healthcare-associated IE was classified as nosocomial or non-nosocomial.

Nosocomial IE was diagnosed in a patient who was hospitalised for more than 48 hours before onset of symptoms or signs of IE.

Non-nosocomial healthcare-associated IE was diagnosed within 48 hours of admission in an outpatient with extensive healthcare contact, either: receipt of intravenous (IV) therapy, wound care, or specialised nursing care at home within the 30 days before IE onset; attendance at a hospital or haemodialysis clinic or receipt of IV chemotherapy within the 30 days prior to onset of IE; hospitalisation in an acute care hospital for two or more days in the 90 days prior to onset of IE; or residence in a nursing home or long-term care facility.¹

Statistical analysis—Statistical analysis was performed using SAS version 9.3 software. Fisher’s exact test and Chi-squared tests were used to compare proportions. Logistic regression models were used to produce odds ratios.

Results

Internationally, 3284 patients were enrolled into ICE-PCS, with definite IE by the modified Duke criteria present in 2781. New Zealand recruited 336 patients (266 with definite IE and 70 with probable IE). Table 1 shows the baseline characteristics and medical conditions of patients in the New Zealand cohort.

The median age at enrolment of New Zealand patients was 59.5 years (range 15 to 98 years, interquartile range (IQR) 41-73), and the majority of patients were male (229 males, 68%). Prosthetic valve endocarditis occurred in 104/336 (31%); the remainder had native valve endocarditis or another type of endocarditis, e.g. related to other prosthetic intracardiac material (11/336, 3%). Around a quarter of patients (93/336, 28%) had underlying medical conditions.

Conditions predisposing to IE were common, notably underlying structural heart disease. Data on the prevalence of underlying rheumatic heart disease, prior invasive procedures and the presence of intravascular devices at the time of IE diagnosis, were available for 274 patients (274/336, 82%).

Thirty-four patients (34/336, 10%) had already had an episode of IE before the enrolment episode; eleven were previously included in the ICE-PCS. Of patients with a previous episode, 6/34 (18%) had a history of injecting drug use (IDU).
Forty patients (40/336, 12%) had healthcare-associated endocarditis: nosocomial 78% (31/40) and non-nosocomial 22% (9/40).

### Table 1. Baseline characteristics and predisposing factors

<table>
<thead>
<tr>
<th>1. Baseline characteristics</th>
<th>New Zealand cohort, n=336 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age, years (range)</td>
<td>59.5 (15 – 98)</td>
</tr>
<tr>
<td>Male gender</td>
<td>229 (68)</td>
</tr>
<tr>
<td>Haemodialysis</td>
<td>10 (3)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>40 (12)</td>
</tr>
<tr>
<td>HIV</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Cancer</td>
<td>34 (10)</td>
</tr>
<tr>
<td>Native valve IE</td>
<td>221 (66)</td>
</tr>
<tr>
<td>Prosthetic valve IE</td>
<td>104 (31)</td>
</tr>
<tr>
<td>Infected pacemaker/ICD</td>
<td>0</td>
</tr>
<tr>
<td>2. Predisposing conditions</td>
<td></td>
</tr>
<tr>
<td>Current IDU</td>
<td>18 (5)</td>
</tr>
<tr>
<td>Previous IE</td>
<td>34 (10)</td>
</tr>
<tr>
<td>Invasive procedure in preceding 60 days</td>
<td>40/274 (15)</td>
</tr>
<tr>
<td>Long-term IV access at onset of IE</td>
<td>24/274 (9)</td>
</tr>
<tr>
<td>Pacemaker in situ</td>
<td>9 (3)</td>
</tr>
<tr>
<td>ICD</td>
<td>0</td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td>45 (13)</td>
</tr>
<tr>
<td>Native valve predisposition</td>
<td>124 (37)</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>12/274 (4)</td>
</tr>
</tbody>
</table>

HIV: human immunodeficiency virus; ICD: implantable cardiac defibrillator; IDU: injecting drug user

The clinical and echocardiographic findings in this cohort are shown in Table 2. Most patients (257/336, 76%) had fever and 120/336 (36%) had a new or changed heart murmur. Classical signs of IE were rare, with few patients exhibiting Osler’s nodes (6/336, 2%), Roth spots (5/336, 1%) or Janeway lesions (7/336, 2%).

Echocardiography was performed in 334/336 patients (99%): 191/334 (57%) had both a trans-thoracic echocardiogram (TTE) and a transoesophageal echocardiogram (TOE). Forty-one (41/334, 12%) had TOE alone and 102/334 (31%) had TTE alone.

The time of onset of infective endocarditis was known in 187/336 patients (56%) and estimated in the remainder. Eleven patients with a known date of IE onset developed
IE after hospitalisation; the remaining 176 patients developed symptoms in the community. The median time between development of symptoms and hospital admission was four days (range 1-126 days, IQR 1-12 days). Most patients with a known duration of symptoms were admitted within a week of symptom onset (111/176, 63%).

The time from onset of symptoms to hospitalisation varied with the causative organism (Figure 1). Patients with *S. aureus* IE were more likely to present within a week of symptom onset than those with viridans streptococcus IE (OR 4.18, 95% CI 2.36–7.42). Only 5/36 patients (14%) with *Enterococcus faecalis* IE presented within a week of symptom onset.

**Table 2. Clinical, laboratory and echocardiography findings in NZ-ICE patients**

<table>
<thead>
<tr>
<th>Clinical sign/labatory finding</th>
<th>New Zealand patients with sign, n=336 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever (temperature &gt;38°)</td>
<td>257 (76)</td>
</tr>
<tr>
<td>New murmur</td>
<td>90 (27)</td>
</tr>
<tr>
<td>Worsening murmur</td>
<td>30 (9)</td>
</tr>
<tr>
<td>Splinter haemorrhages</td>
<td>65 (19)</td>
</tr>
<tr>
<td>Janeway lesions</td>
<td>7 (2)</td>
</tr>
<tr>
<td>Osler’s nodes</td>
<td>6 (2)</td>
</tr>
<tr>
<td>Roth spots</td>
<td>5 (1)</td>
</tr>
<tr>
<td>Conjunctival haemorrhage</td>
<td>6 (2)</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>12 (4)</td>
</tr>
<tr>
<td>Other emboli</td>
<td>13 (4)</td>
</tr>
<tr>
<td>Elevated CRP</td>
<td>266 (79)</td>
</tr>
<tr>
<td>Elevated ESR</td>
<td>234 (70)</td>
</tr>
<tr>
<td>Microscopic haematuria</td>
<td>15 (4)</td>
</tr>
<tr>
<td>Positive rheumatoid factor</td>
<td>5 (1)</td>
</tr>
</tbody>
</table>

**Echocardiographic findings, n=334 patients**

| Aortic regurgitation                     | 73 (22)                                  |
| Mitral regurgitation                     | 79 (24)                                  |
| Tricuspid regurgitation                  | 20 (6)                                   |
| Any vegetation                          | 229 (69)                                 |
| Aortic valve vegetation                  | 109 (33)                                 |
| Mitral valve vegetation                  | 115 (34)                                 |
| Tricuspid valve vegetation               | 17 (5)                                   |
| Chordae tendinae vegetation              | 6 (2)                                    |
| Paravalvular abscess                     | 43 (13)                                  |
| Paravalvular perforation                 | 10 (3)                                   |
| Paravalvular fistula                     | 4 (1)                                    |
| Prosthetic valve dehiscence              | 8/104 (8)                                |
| Prosthetic valve regurgitation           | 10/104 (10)                              |
Blood cultures were obtained from 333 patients (333/336, 99%) and were positive in 94% (314/333 patients). Data on number of bottles of blood cultures were available for 273 patients (273/333, 82%). A median of six blood culture bottles was collected per patient (range 0–22 bottles, IQR 4–8). Amongst patients with positive blood cultures, a median of four bottles per patient was positive (range 1–16, IQR 2–6) and 46% of patients (153/333) had growth from blood samples drawn at least 12 hours apart.

The causative organism was cultured from three or more sets (where one set consists of two bottles) of blood cultures in 61% of patients (204/333). All blood cultures were negative in 22/333 patients (7%); 12/22 (55%) had been on antibiotics within seven days prior to obtaining blood cultures.

Culture of excised valvular tissue or vegetations from 13 blood-culture-negative patients was positive in four: *Haemophilus parainfluenzae*, *Haemophilus*/*Aggregatibacter aphrophilus* and *Propionibacterium acnes* were the organisms cultured; one valve had polymicrobial growth. Seven patients had additional serological testing, including two blood-culture-negative patients, but these tests were non-contributory. Polymerase chain reaction (PCR) was performed on excised valvular tissue from two blood-culture-negative patients, which led to identification of *Staphylococcus lugdunensis*, the presumed causative organism. In total, 80 valves and three vegetations were removed at surgery and sent for culture; organisms were cultured from 42/83 specimens (51%). The 41 patients whose valves and vegetations were culture-negative had had pre-operative antibiotics for longer than the 42 whose valve cultures were positive (median 11 days versus 4 days).

Table 3 outlines the organisms causing endocarditis in this cohort. Viridans streptococci were the most common (109/336 patients, 32%), followed by *S. aureus* (80/336 patients, 24%). Viridans streptococci were more likely to cause native than prosthetic valve endocarditis (36% versus 24%; difference in proportions 0.12, 95%CI 0.014–0.22), as was *S. aureus* (28% versus 15%; difference in proportions 0.13, 95%CI 0.042–0.22). *Enterococcus faecalis* (OR 2.44, 95% CI 0.19–0.89) and coagulase-negative staphylococci (OR 4.17, 95% CI 0.075–0.75) were both more likely to cause prosthetic valve endocarditis than viridans streptococci.
Table 3. Organisms causing infective endocarditis in the NZ-ICE cohort

<table>
<thead>
<tr>
<th>Organism</th>
<th>NZ cohort total n=336 (%)</th>
<th>Native valve IE n=221 (%)</th>
<th>Prosthetic valve IE n=104 (%)</th>
<th>Differences in proportions with 95% CIs</th>
<th>Managed surgically n=110 (%)</th>
<th>Managed medically n=226 (%)</th>
<th>Differences in proportions with 95% CIs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viridans streptococci</td>
<td>109 (32)</td>
<td>79 (36)</td>
<td>25 (24)</td>
<td><strong>0.12 (0.014, 0.22)</strong></td>
<td>29 (26)</td>
<td>80 (35)</td>
<td>-0.090 (-0.19, 0.013)</td>
</tr>
<tr>
<td><em>Streptococcus bovis/gallolyticus</em></td>
<td>5 (1)</td>
<td>3 (1)</td>
<td>2 (2)</td>
<td>-0.0057 (-0.036, 0.025)</td>
<td>0</td>
<td>5 (2)</td>
<td><strong>-0.022 (-0.0413, -0.0029)</strong></td>
</tr>
<tr>
<td>Other streptococci</td>
<td>21 (6)</td>
<td>13 (6)</td>
<td>9 (8)</td>
<td>0.028 (-0.09, 0.035)</td>
<td>6 (5)</td>
<td>15 (7)</td>
<td>-0.012 (-0.065, 0.042)</td>
</tr>
<tr>
<td><em>S. aureus</em></td>
<td>80 (24)</td>
<td>61 (28)</td>
<td>15 (15)</td>
<td><strong>0.13 (0.042, 0.22)</strong></td>
<td>30 (27)</td>
<td>50 (22)</td>
<td>0.061 (-0.039, 0.161)</td>
</tr>
<tr>
<td><em>Enterococcus faecalis</em></td>
<td>40 (12)</td>
<td>22 (10)</td>
<td>17 (16)</td>
<td>-0.064 (-0.15, 0.017)</td>
<td>8 (7)</td>
<td>32 (14)</td>
<td><strong>-0.069 (-0.14, -0.0024)</strong></td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td>14 (4)</td>
<td>6 (3)</td>
<td>8 (8)</td>
<td>-0.05 (-0.11, 0.0057)</td>
<td>6 (5)</td>
<td>8 (4)</td>
<td>0.019 (-0.030, 0.068)</td>
</tr>
<tr>
<td>HACEK</td>
<td>12 (4)</td>
<td>8 (4)</td>
<td>4 (4)</td>
<td><strong>-0.0023 (-0.047, 0.042)</strong></td>
<td>5 (5)</td>
<td>7 (3)</td>
<td>0.015 (-0.031, 0.060)</td>
</tr>
<tr>
<td>Other</td>
<td>24 (7)</td>
<td>12 (5)</td>
<td>11 (10)</td>
<td>-0.052 (-0.12, 0.015)</td>
<td>10 (9)</td>
<td>14 (6)</td>
<td>0.029 (-0.033, 0.091)</td>
</tr>
<tr>
<td>Polymicrobial</td>
<td>3 (1)</td>
<td>2 (1)</td>
<td>1 (1)</td>
<td>-0.0006 (-0.023, 0.022)</td>
<td>2 (2)</td>
<td>1 (0.4)</td>
<td>0.014 (-0.013, 0.040)</td>
</tr>
<tr>
<td>Culture-negative</td>
<td>16 (5)</td>
<td>10 (5)</td>
<td>6 (6)</td>
<td><strong>-0.012 (-0.065, 0.040)</strong></td>
<td>7 (6)</td>
<td>9 (4)</td>
<td>0.024 (-0.028, 0.076)</td>
</tr>
</tbody>
</table>

HACEK: Haemophilus/Aggregatibacter spp., Aggregatibacter actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella species.

**A** Native valve + prosthetic valve do not always equal cohort total as there were some cases of non-valvular endocarditis. There were 12 cases of IE where the organism was not reported or the codes used uninterpretable.

**B** Compares the proportion of prosthetic and native valve IE caused by each organism in the New Zealand cohort; statistically significant differences are in bold italicized script.

**C** Compares the proportion of patients managed surgically and medically according to causative organism; statistically significant differences are in bold italicized script.

**D** ‘Other’ organisms: Abiotrophia adjacens (3 patients); Candida albicans (2); Corynebacterium jeikeium (1); Enterococcus casseliflavus (1); Enterococcus sp. (1); Erysipelothrix rhusiopathiae (1); Escherichia coli (1); Gemella hydrolysans (3); Gemella morbillorum (1); Lactobacillus sp. (1); Leptotrichia sp. (1); Micrococcus sp. (1); Micromonas micros (2); Propionebacterium acnes (2); Serratia marcescens (2); Stomatococcus mucilaginosus (1).
Most *S. aureus* were methicillin-susceptible (MSSA, 75/80 patients, 94%). There were five cases of methicillin-resistant *S. aureus* (MRSA) endocarditis. Nine (9/40, 23%) *E. faecalis* isolates demonstrated resistance to high-level gentamicin. There were no vancomycin-resistant enterococci (VRE).

Seventy-two percent of patients with viridans streptococcus IE (79/109) had isolates susceptible to penicillin (minimum inhibitory concentration (MIC) \( \leq 0.25 \text{ mg/L} \)); and 2% (2/109) had IE caused by viridans streptococci resistant to penicillin (MIC >2 mg/L). Antibiotic treatment was administered to 335 patients; in most cases, antibiotic treatment involved a beta-lactam. Complete specific data on antibiotic treatment are not available for this cohort.

Surgery was performed in 110/336 patients (33%). Table 4 outlines the surgical management and findings for patients in the New Zealand ICE-PCS cohort. Five patients in whom surgery was indicated were too unwell for surgery.

**Table 4. Surgical management of patients in the NZ-ICE cohort**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number of patients n=110 (%)^4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic valve replacement (AVR) or repair</td>
<td>77/110 (70)</td>
</tr>
<tr>
<td>AVR – mechanical valve</td>
<td>32/77 (42)</td>
</tr>
<tr>
<td>AVR – bioprosthetic valve</td>
<td>43/77 (56)</td>
</tr>
<tr>
<td>Repair – no prosthesis</td>
<td>1/77 (1)</td>
</tr>
<tr>
<td>Unspecified aortic valve replacement/repair</td>
<td>1/77 (1)</td>
</tr>
<tr>
<td>Mitral valve replacement (MVR) or repair</td>
<td>42/110 (38)</td>
</tr>
<tr>
<td>MVR – mechanical valve</td>
<td>28/42 (67)</td>
</tr>
<tr>
<td>MVR – bioprosthetic valve</td>
<td>6/42 (14)</td>
</tr>
<tr>
<td>Repair – no prosthesis</td>
<td>3/42 (7)</td>
</tr>
<tr>
<td>Unspecified mitral valve replacement/repair</td>
<td>5/42 (12)</td>
</tr>
<tr>
<td>AVR + MVR</td>
<td>12/110 (11)</td>
</tr>
<tr>
<td>Tricuspid valve replacement (TVR) or repair</td>
<td>7/110 (6)</td>
</tr>
<tr>
<td>TVR – mechanical valve</td>
<td>1/7 (14)</td>
</tr>
<tr>
<td>TVR – bioprosthetic valve</td>
<td>2/7 (29)</td>
</tr>
<tr>
<td>Unspecified tricuspid valve replacement/repair</td>
<td>4/7 (57)</td>
</tr>
<tr>
<td>Pulmonary valve replacement</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Remove infected pacemaker</td>
<td>0</td>
</tr>
<tr>
<td>Other procedure</td>
<td>16 (15)</td>
</tr>
</tbody>
</table>

^4 Several patients had more than one procedure. Other procedures: replace VSD patch (1 patient); remove vegetation (2); aortic root graft repair (1); aortic root replacement (2); coronary artery bypass graft (3); closure ventricular septal defect (VSD) (4); closure patent ductus arteriosus (1); drainage infected pericardial effusion (1); replace incompetent conduit (1); pulmonary artery homograft (1); remove infected VSD patch (1).

Enterococcal IE and *Streptococcus bovis/gallolyticus* IE were more likely to be managed medically rather than surgically; there was no significant difference between surgical versus medical management for other organisms.

Those who were managed medically were older than those managed surgically (median ages 67 and 46 years respectively), with 39/83 (47%) of those aged 15–40...
years receiving surgery compared with 5/88 patients (6%) aged 73-98 years (p<0.001).

The median time from initial contact with healthcare services to surgery was four
days (range 0 to 259 days, IQR 1-12). Most patients (109/110, 98%) received
antibiotics before surgery. There was evidence of endocarditis at surgery or on
surgical specimens in 107/110 patients (97%).

Measured outcomes in the ICE-PCS were in-hospital mortality, length of hospital stay
and complications of disease or treatment. These outcomes are shown in Table 5.

In total, 20/336 (6%) patients died in hospital. The median time to death after
admission was 15.5 days (range 1 to 80 days, IQR 9.5-22.5), and the median age at
death was 76 years (range 39 to 90 years, IQR 61-80.5). Those with IE caused by
cogulase-negative staphylococci had an increased risk of death compared with those
with IE caused by viridans streptococci (RR 4.7, 95% CI 1.2, 17).

The most common complications of IE were congestive heart failure (71/336, 21%)
and systemic embolisation (59/336, 17%). IE caused by coagulase negative
staphylococci conferred a higher relative risk of several complications (Table 5).

There were 104 patients with prosthetic valve endocarditis. The microbiology of
prosthetic valve IE is shown in Table 3. TOE was performed in 85 patients with
prosthetic valve IE (85/104, 82%). Paravavular complications were identified in 17%
(18/104 patients): prosthetic valve dehiscence in 8/104 patients (8%) and paravalvular
regurgitation in 10/104 patients (10%). Vegetations were identified in 60 patients
(60/104, 58%). Thirty-six patients required surgery (36/104, 35%).

Adverse drug effects, including rash, hepatotoxicity, neutropenia and Clostridium
difficile colitis, complicated the treatment of 7/336 patients (2%).

One-year follow-up data were available for 316 patients. Out-of-hospital mortality
data were collected. A further 18 patients (18/316, 6%) had died at one-year follow
up, two from strokes, four from heart failure, and the remaining patients for other or
unknown reasons. Fourteen patients (14/316, 4%) required further surgery on their
aortic valve (eight patients) or mitral valve (two patients).
Table 5. Complications of IE by valve type and causative organism

<table>
<thead>
<tr>
<th>Complication</th>
<th>Complication by native versus prosthetic valve</th>
<th>Complications by causative organism with absolute and relative risk compared with viridans streptococci IE (reference group)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NZ cohort n=336 (%)</td>
<td>Native valve n=221 (%)</td>
</tr>
<tr>
<td>Death</td>
<td>20 (6)</td>
<td>12 (5)</td>
</tr>
<tr>
<td>Absolute risk (AR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative risk (RR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>48 (14)</td>
<td>32 (14)</td>
</tr>
<tr>
<td>Absolute risk (AR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative risk (RR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systemic embolism</td>
<td>59 (17)</td>
<td>42 (19)</td>
</tr>
<tr>
<td>Absolute risk (AR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relative risk (RR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure (CHF)</td>
<td>71 (21)</td>
<td>53 (24)</td>
</tr>
<tr>
<td>Absolute risk (AR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complication by native versus prosthetic valve&lt;sup&gt;A&lt;/sup&gt;</td>
<td>Complications by causative organism with absolute and relative risk compared with viridans streptococcus IE (reference group)&lt;sup&gt;AB&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>RR</strong></td>
<td>1.5</td>
<td>1.7</td>
</tr>
<tr>
<td><strong>Persistent bacteraemia</strong></td>
<td>(0.87, 2.6)</td>
<td>(0.92, 3.2)</td>
</tr>
<tr>
<td>10 (3)</td>
<td>6 (3)</td>
<td>4 (4)</td>
</tr>
<tr>
<td><strong>AR</strong></td>
<td>0.063</td>
<td>0.05</td>
</tr>
<tr>
<td>(0.021, 0.14)</td>
<td>(0.01, 0.17)</td>
<td>(0.0018, 0.3)</td>
</tr>
<tr>
<td><strong>RR</strong></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Intracardiac abscess</strong></td>
<td>30 (9)</td>
<td>19 (9)</td>
</tr>
<tr>
<td><strong>AR</strong></td>
<td>0.046</td>
<td>0.025</td>
</tr>
<tr>
<td>(0.015, 0.10)</td>
<td>(0.001, 0.13)</td>
<td>(0.084, 0.59)</td>
</tr>
<tr>
<td><strong>RR</strong></td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Median length of stay: days (quartiles)</strong></td>
<td>17.5 (11-29)</td>
<td>23.0 (15-35)</td>
</tr>
</tbody>
</table>

<sup>A</sup> Significant differences in bold italic script.

<sup>B</sup> The first row for each complication represents the Absolute Risk (AR) and the second row shows the Relative Risk (RR) along with their respective 95% confidence intervals.

<sup>C</sup> The microbiology for 16/20 patients who died is given in this table. The four other patients who died had IE caused by unknown organism (1); culture-negative (2); *Enterococcus casseliflavus* (1).

<sup>D</sup> Stroke types were: embolic without haemorrhage (35 patients); embolic with haemorrhage or intracranial haemorrhage (11); unknown (3). Data for transient ischaemic attack (TIA) are not included.

<sup>E</sup> One patient with an ‘other’ type of IE had CHF.

Discussion

The ICE-PCS provided valuable global information on endocarditis in the 21st century, although the bulk of data was obtained from Europe (1213 patients, 44%) and North America (597 patients, 22%),\(^1\) which may have influenced descriptions of the characteristics of IE. Others have published retrospective single-centre or referral centre data on IE in New Zealand,\(^5\text{-}10\) however we believe this is the only prospective multicentre study and the largest modern series of New Zealand patients with IE.

As noted in the international cohort, most New Zealand patients with IE were men (68%) with a median age of 60 years. A significant proportion of New Zealand patients with IE had underlying structural cardiac abnormalities, including congenital heart disease (13%), prosthetic heart valves (31%) and a native valve predisposition (37%). The prevalence of underlying rheumatic heart disease, however, was low (4%), a significant change from historical descriptions of IE, where up to 50% of patients had rheumatic heart disease,\(^11\) and less than may have been anticipated in New Zealand given the high incidence of acute rheumatic fever among Maori and Pacific Island people.\(^12\) It is possible that some IE patients with rheumatic heart disease were not included in the study because they did not live in a participating DHB’s catchment area. We were not able to match these data with specific DHBs and so are not able to comment on this. However, Counties Manukau DHB, which has one of the highest incidences of acute rheumatic fever in New Zealand,\(^13\) was included in the ICE-PCS, suggesting that the rheumatic heart disease data presented here should be representative.

Other studies of IE in New Zealand describe a variable prevalence of underlying rheumatic heart disease in the 21st century: 1 of 62 patients (2%) in a study from Dunedin Hospital\(^5\) (a tertiary referral centre with cardiothoracic surgical services) and 11 of 57 patients (19%) with IE in Tauranga\(^6\) (a peripheral hospital with no cardiothoracic surgical services). A study of IE in South Auckland in the 1980s reported an underlying prevalence of rheumatic heart disease of 45%.\(^9\)

The clinical presentation of IE in the modern age is also different from historical descriptions. The majority of patients had had symptoms for less than one month before presentation, and those with \textit{S. aureus} IE tended to have a shorter illness than those with IE caused by other organisms. Clinical signs of IE were less frequent in this cohort than traditional descriptions of the disease, with very few exhibiting the classical Roth spots, Osler’s nodes or Janeway lesions.

This change in the clinical presentation of IE has been noted elsewhere:\(^1,11\) IE is less frequently a sub-acute illness presenting with classical signs engendered by prolonged inflammation, but rather an illness of relatively short duration, which may have few objective clinical signs. This change may be due to a number of factors including changing patient characteristics, for example, more patients with prior healthcare contact, prosthetic valves or degenerative heart disease rather than underlying rheumatic heart disease; the increasing role of \textit{S. aureus} as a causative agent; and earlier diagnosis through improved microbiological methods and readier access to echocardiography.
Viridans streptococci remained the most common causative organisms in the New Zealand cohort (32%), followed by *S. aureus* (24%). Internationally, *S. aureus* has emerged as the commonest cause of IE, accounting for 31% of IE cases worldwide and 43% of cases in the USA, compared with 17% of cases worldwide caused by viridans streptococci (9% in the USA).\(^1\)

The preponderance of *S. aureus* IE internationally differs from historical studies, in which viridans streptococci were more prevalent.\(^1\)\(^1\) This is thought to reflect the changing epidemiology of *S. aureus* infection in the Western world, with increased contact with healthcare services,\(^1\)\(^4\) high rates of *S. aureus* bacteraemia reported among hospitalized patients,\(^1\)\(^4\)\(^,\)\(^1\)\(^5\) increased numbers of patients receiving prosthetic endovascular and intracardiac devices with subsequent infection,\(^1\)\(^6\) and increased incidence of invasive procedures, all of which are associated with *S. aureus* bacteraemia or endocarditis.\(^3\)\(^,\)\(^1\)\(^4\) Other risk factors for *S. aureus* IE (e.g. IDU), also exist globally, and contribute to making *S. aureus* the leading cause of IE in many parts of the world.

While there appeared to be less *S. aureus* IE in the New Zealand cohort than internationally, it is difficult to make direct comparisons between the groups The international study analysed data only from patients with definite IE, whereas New Zealand patients with both definite and probable IE were included in this paper. There appeared to be a lower prevalence of healthcare-associated IE, previous healthcare contact, and IDU in the New Zealand cohort, which may potentially contribute to less *S. aureus* IE than internationally.

Only 12% of the New Zealand cohort had healthcare-associated IE; in the North American sub-group 32% had healthcare-associated IE.\(^1\)\(^1\) In the New Zealand cohort there was a low prevalence of prior invasive procedures (15%), endocavitary cardiac device infection (0), and patients receiving haemodialysis (3%); in the North American cohort the prevalence was 27%, 8% and 21% respectively. The prevalence of IDU in the New Zealand cohort is also low (5%), reflecting a low prevalence of IDU in the general New Zealand population.\(^1\)\(^7\)

The in-hospital mortality rate in the New Zealand cohort was relatively low (6%) compared with historical New Zealand data.\(^8\)\(^-\)\(^10\) Internationally, in-hospital mortality remains high, between 12 – 22%.\(^1\)\(^,\)\(^3\) The low in-hospital mortality in this cohort may possibly be explained by a lower proportion of *S. aureus* IE in New Zealand than internationally, but also possibly by the contribution of bias in the type of New Zealand patient who was included in the study, leading to an under-representation of severe IE in this cohort. For example, a number of patients from non-study-centre hospitals may have been too unwell to undergo surgery or transfer to a study centre. The inclusion of ‘probable’ endocarditis in this analysis may have also contributed to an apparently lower mortality. It is difficult to comment on whether this reflects an actual decline in IE mortality in New Zealand.

Only 2 of 22 (10%) blood-culture negative patients had additional serological or molecular testing to identify a causative organism (e.g. *Bartonella* species or *Tropheryma whippelii; Coxiella burnetii*, a well recognised cause of culture-negative endocarditis, is not endemic in New Zealand). Overseas studies show that these fastidious organisms cause culture-negative endocarditis relatively frequently, and they should be borne in mind as part of the differential diagnosis.\(^1\)\(^8\) Molecular
methods are increasingly used for elucidating the microbiological aetiology of IE and would almost certainly be found to be more widely used if this study were repeated.

This study was not specifically designed to look at IE in New Zealand; however, it is a large, prospective study involving several local centres. All sites were major centres with ready access to cardiothoracic surgical services. This may have introduced observational bias towards patients with more severe disease or a requirement for surgery, or, conversely, towards those who were well enough to undergo surgery. A truly representative picture of IE in New Zealand would involve both tertiary centres and peripheral hospitals.

Another limitation is the lack of data on medical management of IE in New Zealand. Other interesting data, which were initially collected by local investigators in this cohort but are no longer available for analysis, include information about dentition and dental procedures, antimicrobial therapy used, previous valve surgery for IE, duration between IE episodes and indications for and findings at surgery.

**Conclusion**

The ICE-PCS provides valuable information on IE in the modern era, and most of the findings in the international cohort apply to IE in New Zealand. IE in the modern age is less likely to be a prolonged illness with classical clinical signs and is more likely to present acutely.

In the New Zealand cohort, there was a ‘traditional’ predominance of viridans streptococci over *S. aureus* as causative organisms, though the increasing role of *S. aureus* in IE observed internationally may soon be relevant to New Zealand as well. This large contemporary series of IE in New Zealand provides a reference for local practitioners assessing and managing infective endocarditis.

**Competing interests:** Nil.

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References:


Is refractory angina pectoris a form of chronic pain? A comparison of two patient groups receiving spinal cord stimulation therapy

Nick Pak, Daniel A Devcich, Malcolm H Johnson, Alan F Merry

Abstract

Aim To compare psychological and pain-related characteristics of patients with chronic pain and patients with refractory angina pectoris who had been treated with spinal cord stimulation (SCS) therapy.

Method Twenty-four patients receiving SCS therapy were interviewed. Four psychological variables were assessed using standardised questionnaires for pain catastrophising, health locus of control, anxiety sensitivity, and self-efficacy. Patients also completed the revised version of the Short-Form McGill Pain Questionnaire, the Short-Form Health Survey, and self-reported measures of global perceived effect, pain, functionality, and satisfaction with SCS therapy.

Results Most patients reported improvements in pain, functionality, and improvement overall. Some health locus of control dimensions were significantly higher for the angina group than the chronic pain group, and chronic angina patients reported significantly lower levels of intermittent pain. Virtually all patients reported being satisfied with SCS therapy.

Conclusion Most self-rated psychological and pain-related characteristics were no different between the two groups, which gives some support to the view that refractory angina is a form of chronic pain. The results also add to evidence supporting the use of SCS therapy for refractory angina pectoris; however, differences observed on a few variables may indicate points of focus for the assessment and treatment of such patients.

Spinal cord stimulation (SCS) therapy has been used for over four decades in a variety of chronic pain conditions and for over two decades in refractory angina pectoris. The UK-based National Institute for Clinical Excellence (NICE) has recommended SCS therapy for chronic neuropathic pain conditions but not for refractory angina, because, for the latter, randomised controlled trial evidence shows that SCS therapy is merely equivalent to coronary artery bypass grafting (CABG) and percutaneous myocardial laser revascularisation. This is an interesting perspective, given that the risks of CABG surgery are considerably greater than those of SCS.

In New Zealand, patients with chronic pain and patients with refractory angina pectoris have been treated with SCS therapy in pain clinics for at least the past 15 years. In these clinics, prior to commencing SCS therapy, patients with chronic pain are typically assessed for intensive (e.g., three-week, full-time) pain management programmes consisting of education, relaxation training, and activation components.
In some cases, by contrast, patients with refractory angina pectoris may progress to SCS therapy without undergoing any psychological assessment or undertaking a pain management programme. However, given that several psychological characteristics that correlate with chronic pain may influence the outcomes of SCS therapy despite technical success, it is worth asking whether this approach allows adequate opportunity to identify patients with refractory angina pectoris who may not respond well to SCS therapy.

As with other chronic pain conditions, it has been shown that cognitive-behavioural interventions for refractory angina pectoris can improve symptoms and quality of life as well as reduce hospitalisations and myocardial infarctions. While these studies add value as examples of applied research that addresses alternative treatment options for these patients, they also raise the theoretical question of whether refractory angina pectoris may be considered within the spectrum of chronic pain conditions. If so, this may place more emphasis on the importance of the psychological assessment of patients with refractory angina pectoris and may have implications for clinical pathways in treating the condition.

The aim of this study was to provide a descriptive account of psychological and pain-related characteristics of two patient groups receiving SCS therapy: patients with refractory angina pectoris and patients with chronic pain. We were interested in assessing variables that have been shown to predict aspects of functioning and outcomes in patients with chronic pain, and we also took the opportunity to assess levels of satisfaction with SCS therapy.

**Method**

**Participants**—Patients who had received SCS implantation through The Auckland Regional Pain Service (TARPS) in Auckland, New Zealand before the end of September 2010 and who were still using it were invited to participate in the study. The inclusion period ran from July 1991 through to September 2010, and there were no exclusion criteria for patients other than their decision to not participate.

Thirty-four out of a total of 57 patients were eligible to participate in the study. Exclusions included 13 patients who had stimulators inserted over the trial period but had discontinued using them (5 patients with chronic pain and 8 with refractory angina pectoris) and 10 angina patients who had since deceased. Of the 34 patients who met the inclusion criteria, 24 agreed to take part—12 patients with chronic pain, 12 with refractory angina pectoris. Patients who were unwilling to take part in the study cited poor health (n=4) and personal reasons (n=2), and of the four remaining non-participating patients, two were unable to take part because of pending surgery and overseas travel, respectively, and the remaining two were unable to be contacted by the researchers.

**Procedure**—The study was approved by the New Zealand Ministry of Health Northern Y Regional Ethics Committee (NTY/09/114/EXP). Eligible patients were contacted by mail and telephone and were invited to participate in the study. Participation involved an interview of approximately two hours in length at Mercy Hospital in Auckland. Patients’ travel expenses were reimbursed through research donations provided by Medtronic and Advanced Neuromodulation Systems (ANS). The interviews were conducted by a medical student (NP) trained in interview methods by a clinical psychologist (MHJ), who also monitored the first day of interviews. Neither researcher was involved in the clinical management of the participants.

During the interviews, participants completed a questionnaire comprising various standardised measures to assess pain relief, quality of life, functionality, and a selection of psychological characteristics. After their interview, patients were offered the opportunity to be seen by the SCS nurse specialist for an adjustment of their stimulator and to discuss any unresolved issues if necessary. The research nurse was also present and available to provide assistance if required at all times during the interviews. Once all data were collected, analysis was conducted using SPSS statistical software. All
statistical tests conducted in the analysis were two-tailed, and statistical significance was considered to be established at the .05 level.

Measures

Pain Catastrophising Scale (PCS)\(^7\)—The PCS is a 13-item questionnaire that assesses pain catastrophising—the disposition to negatively evaluate pain and one’s ability to deal with it—which has been linked to higher levels of psychological distress and pain-related disability.\(^7\) Patients are asked to indicate the degree to which particular thoughts and feelings are associated with their experience of pain on a 5-point Likert-type scale (0=not at all; 4=all the time). Item scores are summed to give an assessment of pain catastrophising (range=0–52), and scores for subscales measuring ruminating, magnification, and helplessness are yielded similarly by summing the relevant items.

Pain catastrophising scores of 30 or more are considered clinically relevant, as 70% of chronic pain patients scoring above 30 describe themselves as totally occupationally disabled, and 66% report moderate depression.\(^8\) Good to excellent levels of reliability and validity have been reported for the measure (including subscales) among chronic pain populations.\(^9\)

Pain Self-Efficacy Questionnaire (PSEQ)\(^10\)—Self-efficacy is a measure of confidence that someone has in his or her ability to engage in a course of action sufficient to achieve a desired outcome.\(^11\) High self-efficacy has been correlated with lower pain, less psychological distress, and better outcomes to medical treatments.\(^10\)

Comprising 10 items, the PSEQ assesses patients’ confidence in accomplishing daily activities while in pain. Responses are made on a seven-point rating scale (0=not confident at all; 6=completely confident), and a final score is produced by summing all responses (range=0–60), with higher scores signifying stronger pain self-efficacy beliefs. Normative data from a large sample of patients with chronic pain show mean PSEQ scores ranging from 24.9 to 28.5, depending on the site of the pain, and an overall mean of 25.5 across all patients.\(^5\) \(^7\) Studies have reported evidence of construct validity and excellent internal consistency in research involving patients with chronic pain.\(^13\)\(^14\)

Anxiety Sensitivity Index (ASI)\(^15\)—The 16-item ASI assesses beliefs about the harmful consequences of anxiety-related symptoms (i.e., anxiety sensitivity: the fear of anxiety and related sensations that arise from the belief that these sensations can have harmful physical, psychological, or social consequences).\(^16\) Each item—a statement relating to a possible negative consequence (e.g., fear, embarrassment, etc.) of the experience of anxiety—is rated by patients on a five-point scale (0=very little; 4=very much).

Summing the scores of the items gives a final score of sensitivity to anxiety (range=0–64), with normative data indicating an overall ASI mean of around 19.0 among European and American samples.\(^17\) Higher scores on the ASI in patients with persistent pain have been shown to be associated with greater disability, pain, and distress;\(^18\) higher scores are also related to higher fear of pain, which in turn is linked to reduced activity and movement.\(^19\) Research into the psychometric properties of the ASI has revealed high internal consistency (\(\alpha=0.88\)), with factor and correlational analyses demonstrating meaningful construct independence from standard measures of anxiety.\(^16\)

Multidimensional Health Locus of Control (MHLC)\(^20\)—The 18-item MHLC (Form C) measures individuals’ beliefs about the extent to which they are able to control the status of their health—that is, whether control beliefs are oriented towards factors that are internal or external (i.e., chance, doctors, and other people).\(^21\) The MHLC requires respondents to score each item, a statement about their health, on a six-point Likert-type scale (1=strongly disagree; 6=strongly agree). Scores are summed for each of the four Form C subscales (ranges=6–36 for the internality and chance subscales and 3–18 for the doctors and other people subscales).

High scores on a particular subscale indicate stronger representations of the related locus of control—higher scores on the internality subscale, for example, represent stronger beliefs that one’s health status is largely influenced by personal factors, indicating that these individuals are more likely to take responsibility for their own health, while high doctors and other people scores suggest the individuals are more likely to believe that interventions by others will be necessary to manage their health. Since publication, the MHLC has been used widely, with a majority of studies showing good support for the validity, reliability, and the multidimensional structure of the measure.\(^21\)\(^23\)

Demographic and other pain-related variables—The study questionnaire also comprised some standard, psychometrically robust measures commonly used in pain research. Patient disability was assessed using the physical functioning scale of the Short-Form Health Survey (SF-36),\(^24\) a widely used
health-related quality-of-life measurement tool that incorporates assessments of physical and mental health status. Sensory and affective dimensions of pain experience were measured with the 22-item revised version of the Short-Form McGill Pain Questionnaire (SF-MPQ-2), which has been validated for both neuropathic and non-neuropathic pain conditions. In addition, we collected data on a range of demographic variables (age, gender, and ethnicity), and, using three seven-point scales (1=completely recovered; 7=worse than ever), self-reported perceptions of global effectiveness, pain, and functionality in relation to SCS therapy were also measured (e.g., “How would you rate your level of function now compared with before you had your spinal cord stimulator inserted?”).

Finally, as an assessment of patients’ satisfaction with SCS therapy, two dichotomous items on patients’ willingness to (1) go through the procedure again and (2) have their spinal cord stimulator repaired (should the need arise) were also included in the study questionnaire.

Results

Patient information across the two groups is displayed in Table 1.

Table 1. Patient information (demographic and spinal cord stimulation [SCS]-related data) across the two study groups

<table>
<thead>
<tr>
<th>Demographic data</th>
<th>Patient group</th>
<th>Pain (n=12)</th>
<th>Angina (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic data</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Age, M (range)</td>
<td></td>
<td>56.3 (37–79)</td>
<td>66.2 (48–81)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand European</td>
<td></td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Māori</td>
<td></td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td>Other European</td>
<td></td>
<td>–</td>
<td>3</td>
</tr>
<tr>
<td>Indian</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Not specified</td>
<td></td>
<td>1</td>
<td>–</td>
</tr>
<tr>
<td><strong>SCS therapy data</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulation months, M (range)</td>
<td></td>
<td>51 (6–222)</td>
<td>37 (6–150)</td>
</tr>
<tr>
<td>Complication-free patients</td>
<td></td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Total complications, n</td>
<td></td>
<td>14</td>
<td>16</td>
</tr>
<tr>
<td>Total procedures, n</td>
<td></td>
<td>35</td>
<td>40</td>
</tr>
<tr>
<td><strong>Procedures following SCS insertion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stimulator inserted</td>
<td></td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Battery replacement</td>
<td></td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Stimulator replacement</td>
<td></td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Trial lead inserted</td>
<td></td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Other procedures</td>
<td></td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Lead replacement</td>
<td></td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Lead revision</td>
<td></td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Stimulator removed</td>
<td></td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td><strong>SCS complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection of stimulator site</td>
<td></td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Technical problems with stimulator</td>
<td></td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Unsatisfactory coverage, reprogrammed</td>
<td></td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Undesired stimulation effects</td>
<td></td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Lead migration</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Stimulator unit caused discomfort</td>
<td></td>
<td>2</td>
<td>–</td>
</tr>
</tbody>
</table>

Note: Except where indicated, values represent number of participants.
In general, the two groups were fairly evenly matched in demographic and SCS-related variables, including procedures on SCS and experience of SCS-related complications. They were also well balanced in respect of their perception of the effects of SCS and in their satisfaction with this therapy (see Table 2). However, patients with refractory angina pectoris were, on average, older than patients with chronic pain (mean difference of 11.4 years, p=0.03), and while all but one of the patients with chronic pain had a formal psychological assessment prior to stimulator insertion, no patients with angina underwent such assessment.

Table 2. Pain characteristics (pain experience, self-reported disability) and patient-perceived spinal cord stimulation [SCS]-related outcome variables across the two study groups

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patient group</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pain</td>
<td>Angina</td>
</tr>
<tr>
<td>SF-MPQ-2 subscales</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continuous, M (SEM)</td>
<td>18.25 (4.20)</td>
<td>10.67 (3.42)</td>
</tr>
<tr>
<td>Intermittent, M (SEM)</td>
<td>15.08 (5.08)</td>
<td>3.33 (1.98)</td>
</tr>
<tr>
<td>Neuropathic, M (SEM)</td>
<td>13.92 (3.37)</td>
<td>9.00 (2.26)</td>
</tr>
<tr>
<td>Affective, M (SEM)</td>
<td>12.75 (3.45)</td>
<td>7.17 (1.37)</td>
</tr>
<tr>
<td>Total score, M (SEM)</td>
<td>60.00 (13.81)</td>
<td>30.17 (8.43)</td>
</tr>
<tr>
<td>SF-36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical functioning, M (SEM)</td>
<td>18.09 (1.96)</td>
<td>17.33 (1.26)</td>
</tr>
<tr>
<td>Perceived effects of SCS therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Improved overall</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>Improved pain</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Improved functionality</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Patient satisfaction with SCS therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Would repeat the procedure</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Would have repairs done</td>
<td>12</td>
<td>12</td>
</tr>
</tbody>
</table>

Note: Except where indicated, values represent number of participants. SF-MPQ-2=revised version of the Short-Form McGill Pain Questionnaire; SF-36=Short-Form Health Survey. *p=0.05.

Five patients in the chronic pain group participated in a clinic-administered pain management programme, which focussed on education, relaxation, and activation across an intensive 3-week period. No patients with refractory angina pectoris underwent such a programme.

Pain characteristics (see Table 2) were mostly comparable across the two patient groups: between-group differences in subscale scores of the SF-MPQ-2 were not observed for most of the measure’s pain dimensions (i.e. continuous pain, neuropathic pain, and affective descriptors). However, patients with chronic pain reported experiencing significantly more intermittent pain than patients with angina (15.08 vs. 3.33, p<0.05). Levels of disability (physical functioning scale of the SF-36) were similar across the two groups—18.09 for patients with chronic pain and 17.33 for patients with refractory angina pectoris.

In the questionnaire, patients were asked to rate their perceptions of the effect of SCS. To assist analysis, we split participants’ responses (which were made on seven-point scales) into two categories: (1) improved, which incorporated responses of
‘completely recovered’, ‘much improved’, and ‘slightly improved’ (scores 1–3 on the scale) and (2) not improved, which combined the ‘no change’, ‘slightly worsened’, ‘much worsened’, and ‘worse than ever’ responses (scores 4–7).

Results showed that perceptions of the efficacy of SCS therapy were mostly favourable: with few exceptions, the majority of patients in both groups reported improvement overall (global effect) since beginning SCS therapy; likewise, most patients in both groups reported improvements in pain and functionality. Patient satisfaction with SCS therapy was high across both groups, with nearly all patients indicating they would repeat the procedure and would have their spinal cord stimulator repaired (e.g., new lead inserted) if the need arose. Two patients in the chronic pain group indicated they would not undergo the procedure again.

Analysis of the psychological characteristics data (see Table 3) revealed many parallels between the two patient groups. Specifically, no significant between-group differences were found with regard to pain catastrophising, self-efficacy, and anxiety sensitivity. It is worth noting, however, that PCS scores for patients with refractory angina pectoris appeared to be approaching clinical significance cut-offs for pain catastrophising.

Between-group differences were observed for some of the MHLC subscales: the chance dimension of the MHLC was significantly higher in the angina group than in the chronic pain group (14.08 vs. 20.09, p=0.01), indicating that patients with refractory angina pectoris were more likely to hold beliefs about fate and luck as determinant factors of their pain condition and outcomes. The same trend was found for the doctors subscale, where significantly higher scores (which indicate stronger beliefs that health status is predominantly influenced or determined by doctors and medically trained professionals) were observed among patients with refractory angina pectoris (11.46 vs. 15.08, p=0.01).

Table 3. Psychological characteristics (pain catastrophising, self-efficacy, anxiety sensitivity, and health locus of control) across the two study groups

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patient group</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pain</td>
<td>Angina</td>
</tr>
<tr>
<td>Pain catastrophising (PCS)</td>
<td>16.08 (4.01)</td>
<td>23.33 (3.29)</td>
</tr>
<tr>
<td>Self-efficacy (PSEQ)</td>
<td>34.92 (4.06)</td>
<td>33.42 (3.44)</td>
</tr>
<tr>
<td>Anxiety sensitivity (ASI)</td>
<td>17.42 (3.63)</td>
<td>18.83 (3.03)</td>
</tr>
<tr>
<td>Health locus of control (MHLC subscales)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internality</td>
<td>26.42 (1.25)</td>
<td>26.08 (1.07)</td>
</tr>
<tr>
<td>Chance</td>
<td>14.08 (1.00)</td>
<td>20.09 (1.82)</td>
</tr>
<tr>
<td>Powerful others</td>
<td>20.83 (1.74)</td>
<td>24.50 (2.10)</td>
</tr>
<tr>
<td>Doctors</td>
<td>11.46 (1.18)</td>
<td>15.08 (0.65)</td>
</tr>
<tr>
<td>Other people</td>
<td>11.27 (1.02)</td>
<td>11.75 (1.04)</td>
</tr>
</tbody>
</table>

Note: Values represent means, with standard error of the mean in parentheses. PCS=Pain Catastrophising Scale; PSEQ=Pain Self-Efficacy Questionnaire; ASI=Anxiety Sensitivity Index; MHLC=Multidimensional Health Locus of Control.
Discussion

In this study we used a range of psychometrically robust measures to assess important pain and psychological characteristics of two patient groups receiving SCS therapy. For the most part, between-group differences in self-reported disability and pain experience across the dimensions of the SF-MPQ-2 could not be detected in the data—the only exception being the results from the intermittent subscale, where patients with refractory angina pectoris reported significantly less intensity with regard to intermittent-type symptoms (e.g., ‘shooting pain’, ‘sharp pain’). Virtually all patients reported improvements following insertion of their spinal cord stimulators and indicated that they would repeat the procedure or repair their spinal cord stimulator if the need arose.

Our results show many parallels between these two patient groups in terms of psychological characteristics. Specifically, we found no differences in pain catastrophising, self-efficacy, or anxiety sensitivity, with both groups demonstrating, on average, at least moderate ratings on the respective measures (although it is noteworthy that pain catastrophising scores were approaching clinical significance among the angina group). We did, however, find some key differences in some dimensions of health locus of control: analysis revealed significantly higher scores on the chance and doctors subscales among patients with refractory angina pectoris. These findings indicate stronger beliefs that health status is predominantly influenced or determined by particular externality-related factors.

Taken together, these data indicate that the two patient groups shared many similarities where psychological characteristics are concerned, and the findings perhaps give some support to the view that refractory angina pectoris lies somewhere in the spectrum of the chronic pain syndrome and may therefore be amenable (at least in terms of its psychological sequelae) to integrated psychological-based pain management interventions.5,6

Research involving patients with angina has shown that maladaptive beliefs about angina appear to have a negative effect on functional and psychological outcomes.26 Such findings, along with the controversial view that refractory angina pectoris could be conceptualised as a chronic pain condition, not only challenge assumptions about conventional clinical pathways for patients with refractory angina pectoris, they also support the call for considering, as is the case with other chronic pain conditions, the integration of psychological and educational treatment approaches.27,28

The differences between the groups, where observed, perhaps have some implications for specific health outcomes, particularly for patients with refractory angina pectoris. The MHLC provides an assessment of expectancies associated with health status and health care, and the measure has been demonstrated to be a good predictor of health behaviours29 and recovery.30 Patients with refractory angina pectoris reported significantly higher scores on two subscales related to externality, and this raises an interesting research question of whether these relatively greater externality beliefs among these patients might be related to poorer health outcomes, as has been shown in other pain-related conditions.31,32

The pain catastrophising results, although not entirely conclusive, at least raise the possibility that among patients with refractory angina pectoris there could be a
tendency towards maladaptive cognitive and emotional coping responses to pain, particularly in terms of rumination and feelings of helplessness (e.g., ‘ticking time bomb’ beliefs). While we remain cautious about overstating any unfounded implications of these data, we are of the view that they do at least suggest that such patients would most likely benefit from psychological assessment prior to SCS implantation, as is typically done in the case of patients with chronic pain conditions. And while interventions tailored specifically for this group of patients may have benefits for their symptoms and quality of life, further research into pain catastrophising among patients with refractory angina pectoris is needed to warrant its inclusion as a theme of intervention in any such programme. The same caveat could also extend to the health locus of control dimensions mentioned above or any other putatively important psychological characteristics for that matter.

There are some limitations to consider regarding the generalizability of the study findings, chiefly because, first, the chronic pain group comprised patients across a number of different conditions and, second, because of the small number of study participants. Indeed, the small sample size is a major limitation that restricts the extent to which strong conclusions can be drawn from the data. Nevertheless, it is worth highlighting that even though the sample of patients was relatively small for both groups, we were yet able to detect significant differences in a few psychological and pain-related characteristics. And even though it may well be that there are other important differences that exist and that the present study was not sufficiently powered to detect, the differences that were observed may suggest topics of research involving patients with refractory angina pectoris.

In conclusion, the similar patterns in psychological characteristics give some credence to the view that refractory angina is a form of chronic pain, and this supports (but also raises some interesting questions about) psychological-based treatment for these patients. Therefore, while it is encouraging that studies involving patients with refractory angina pectoris have reported positively on the effects of psychological-based interventions that are modelled on interventions for chronic pain, the results from the present study suggest that perhaps such programmes could be enhanced by addressing some of the unique psychological and pain-related characteristics of these patients.

**Competing interests:** Nil.

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References:


A clinical psychologist in GP-Land: an evaluation of brief psychological interventions in primary care

Sunil Dath, Christine Yang Dong, Malcolm W Stewart, Eileen Sables

Abstract

Aim To evaluate the clinical outcomes and other impacts of brief therapy provided in a primary care setting by a clinical psychologist who was mainly employed in secondary mental health.

Method The outcomes of 23 primary care patients referred to a clinical psychologist were evaluated using the General Health Questionnaire (GHQ), the World Health Organisation Quality of Life (WHOQoL) scale, and the Beck Depression Inventory (BDI). A mixture of quantitative and qualitative data from patients and staff were analysed to identify other impacts of the intervention.

Results Large improvements in BDI, GHQ, and WHOQoL scores were found, with strong changes consistent with the targets of the intervention. Patients reported primary-based clinical psychology input was more convenient and many engaged who had resisted referral to secondary mental health services. Other benefits to the service, including improved primary-secondary service integration, improved primary management of mental health difficulties, and improved liaison with mental health specialists, were reported by primary health staff.

Conclusion Brief psychological interventions by a visiting clinical psychologist in a general practice setting had substantial benefits for the patients and for the practice. This project indicates the value of integrated psychological input consistent with recent moves to better primary-secondary integration in mental health care.

Mental health difficulties are common in New Zealand with a lifetime prevalence of 47%, and 21% prevalence in the previous 12 months. Mental health difficulties are common in primary healthcare, with a third of general practice patients in New Zealand having experienced a diagnosable mental health disorder within the previous 12 months. Addressing these issues effectively is important for patients’ overall mental and physical wellbeing and quality of life. Providing timely, effective, and cost-effective assistance with mental health needs places considerable demands on primary and secondary health services.

Substantial work has been undertaken to improve the availability of mental health care in primary health settings, including increased funding and initiatives to broaden the range of mental health interventions available. Blueprint II identifies better integrated treatment across primary and secondary services as important for improving mental health services.

Many patients presenting to primary care with mild to moderate mental health difficulties do not meet referral criteria for secondary mental health services, or refuse referral to specialist services. This creates a challenge for primary health to provide
optimal treatment within existing time and resource constraints. Provision of brief psychological therapy services in the primary health setting can be important in meeting this challenge.\textsuperscript{6}

International literature has indicated a variety of benefits for provision of clinical psychology services in primary care.\textsuperscript{7–11} For example, Seekles, and colleagues\textsuperscript{12} reported a moderate effect size in a meta-analytic study of psychological treatment of anxiety disorders in primary care. New Zealand evaluations of brief psychological interventions in primary care have also shown positive clinical outcomes.\textsuperscript{6,13,14}

Different models of providing psychological therapy in primary care have been used, including: consultation-liaison, where mental health staff provide consultation and support for a primary care provider who is offering mental health care; shifted outpatient model, where mental health staff run clinics within primary health settings; and formal shared care, where responsibility is shared between primary healthcare staff and specialist mental health staff.\textsuperscript{15}

Another model involves mental health providers being employed by primary health services.\textsuperscript{13,14} Stepped Care approaches are widely advocated and increasing widely used.\textsuperscript{6} Stepped Care approaches define interventions into discrete levels of intensity and use well-defined processes to 1) match the patient to the optimal level of care to most effectively and least restrictively meet their needs and 2) transition patients between levels if their needs are not met or if they change.

A stepped care approach may be used in conjunction with any of the models above. Hallmarks of the approaches above are often brief treatment duration and use of evidence-informed therapeutic approaches such as cognitive behaviour therapy (CBT).\textsuperscript{13,14}

This study evaluates the outcomes of the provision of evidence-informed psychological therapy in a primary care setting by a clinical psychologist who mostly worked in secondary mental health and provided outreach services to primary care on a weekly sessional basis.

Services were provided as shared care between the psychologist and primary care without the patient otherwise engaging with secondary mental health services. Hypothesised outcomes included positive changes on clinical measures; positive changes in psychological wellbeing and quality of life; patient report of beneficial outcome and satisfaction; and primary healthcare staff report of benefits for patients and for mental health care provision in primary health.

Methods

Participants

Patients—Inclusion criteria for this study were:

- 18 years or older,
- Showing mild-moderate mental health disorder of at least 6 months duration (in most cases, longer),
- Not assessed as having a severe acute mental health disorder or high risk of harm to self or others,
- Their mental health disorder had not been substantially alleviated by medication and/or other support interventions already provided by primary care staff, and
Current patients of a primary care service with approximately 12,000 patients. General practitioners used clinical assessment and patient self-report to identify potential participants rather than a standardized measure, in part due to the diversity of presenting problems. Thirty-seven patients were referred, of which 29 (76%) met criteria and gave consent. These 29 patients entered therapy and 23 (80%) completed the intervention.

**Primary care staff**—Eight GPs and a practice nurse who worked at the Primary Care Practice were interviewed.

**Procedure**

**Intervention**—Psychological interventions were undertaken by male doctoral-level clinical psychologist with 17 years post-qualification experience, nine years of this in adult community mental health.

After initial assessment at the GP clinic, patients undertook a time-limited (4 session) individually-tailored psychological intervention targeting mood improvement and based on CBT and associated skills-based techniques such as problem-solving, lifestyle enhancement, behavioural activation, emotional regulation techniques, interpersonal/relationship effectiveness, or other interventions negotiated with the patient following discussion of the psychological formulation, needs, and therapeutic goals. The patient’s GP was informed about the diagnosis, treatment plan and recommendations for additional support by primary care staff.

The standard four session intervention typically involved a 90 minute initial assessment, two one-hour intervention sessions at two-weekly intervals, and a final one-hour session including completion of outstanding aspects of therapy, relapse prevention, and therapy termination.

Additional self-help resources, such as free on-line mindfulness training and stress, anxiety or depression management packages were introduced as appropriate. Depending on patient need and preference, up to two telephone follow-ups were undertaken by practice nurses. These provided support, encouraged use of therapeutic strategies, and enhanced motivation.

**Research**—Evaluation of the intervention involved three components: clinical outcome measures, a patient satisfaction survey, and primary care staff interviews.

**Clinical outcomes**

Three patient-completed rating scales were given as pre- and post- measures at the first and last sessions. The 12-item General Health Questionnaire (GHQ)\(^{16}\) assessed mental health concerns including depression, anxiety, stress, and the patient’s perceptions of their general health. The 8-item World Health Organization Quality of Life measure (WHOQoL)\(^{17}\) assessed quality of life. The 21-item Beck Depression Inventory – II (BDI)\(^{18}\) is a widely-used measure of depression.

**Patient satisfaction**

A 10-item satisfaction survey designed by the principal investigator was distributed to all patients who completed treatment. The survey was returned anonymously. A copy of this survey is available in this article’s appendix.

**Primary care staff**

After the intervention period ten general practitioners (GPs) and one practice nurse were invited to participate in a 30-minute structured interview about the project.

The interview covered five main areas:

- Reasons/criteria for referral to the project,
- Impact of the project on the relationship between primary and secondary mental health services,
- Impact of the project on access for primary health patients to mental health services,
- Outcomes of the project, and
- Advantages and disadvantages of the project.

The interview was undertaken by a researcher/health professional not otherwise involved in the study. Eight GPs and one practice nurse (82%) participated.
Analysis

Quantitative data—Statistical analyses were performed using Statistical Analysis Systems (SAS) software for Windows (version 9.3, 2010). All statistical tests were two-sided at a 5% significance level. The pre-post difference in mean total scores for the GHQ, WHOQoL and BDI were tested with paired t-tests. The proportion of patients with a total score in the clinical range on the GHQ and BDI were compared for pre- and post-treatment assessments. Multivariate statistical tests were undertaken to assess the relationship between demographic variables and change in total scores but none were significant so the results are not presented. Wilcoxon Signed-Rank tests with subsequent Stepdown Bonferroni adjustment for multiple comparisons were used to test the pre-post differences in individual item scores for the three clinical measures. The clinical significance of treatment impact can be assessed using the caseness (defined as “the proportion of patients with a particular condition”). The pre- and post-treatment proportions of caseness for individual items were compared.

Qualitative data—Participants’ responses were analysed using the Inductive Categorisation method of content analysis. This method involved the systematic categorisation of data into themes by reading through the interview responses, and the themes that emerge are grouped into categories. Such analyses focus particularly on the range of ideas reported, but the frequency of similar ideas was also recorded. The results are presented either in the text (summarizing the range of ideas) or as a table showing the themes and sub-themes with asterisks after each sub-theme to indicate the frequency of similar ideas from different respondents.

Results

Participants

The patient’s demographic and clinical characteristics are shown in Table 1. The small number of non-completers makes comparisons tentative, but non-completers were more likely to have anxiety conditions and had slightly higher pre-treatment GHQ scores than completers.

Table 1. Demographics of participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>Completing participants (N=23)</th>
<th>All participants (N=29)</th>
<th>Non-completing participants (N=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>12</td>
<td>52%</td>
<td>17</td>
</tr>
<tr>
<td>Male</td>
<td>11</td>
<td>48%</td>
<td>12</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-30</td>
<td>5</td>
<td>22%</td>
<td>6</td>
</tr>
<tr>
<td>31-40</td>
<td>9</td>
<td>39%</td>
<td>11</td>
</tr>
<tr>
<td>41-50</td>
<td>3</td>
<td>13%</td>
<td>5</td>
</tr>
<tr>
<td>51-65</td>
<td>5</td>
<td>22%</td>
<td>6</td>
</tr>
<tr>
<td>66+</td>
<td>1</td>
<td>4%</td>
<td>1</td>
</tr>
<tr>
<td>Primary diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>1</td>
<td>4%</td>
<td>4</td>
</tr>
<tr>
<td>Depression</td>
<td>18</td>
<td>78%</td>
<td>21</td>
</tr>
<tr>
<td>Other*</td>
<td>4</td>
<td>17%</td>
<td>4</td>
</tr>
<tr>
<td>Pre-intervention total scores</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GHQ</td>
<td>M (sd)</td>
<td></td>
<td>M (sd)</td>
</tr>
<tr>
<td>WHO-QOL</td>
<td>26.3 (5.9)</td>
<td></td>
<td>25.8 (5.7)</td>
</tr>
</tbody>
</table>

*Notes: N = number in sample. M = mean. (sd) = standard deviation. Other includes: adjustment disorder, psychosis in remission and impulse control difficulties.
Clinical outcomes

The changes in clinical measures total scores are shown in Table 2. The change in total scores between pre- and post-treatment for all outcome measures were statistically significant (p<0.001 for all). The power of the pre-post paired difference for the GHQ, BDI and WHOQOL total scores (1-β) was greater than 0.9 for all, indicating that the sample size is very likely to be large enough to detect the true differences.

The proportion of patients in the clinical range on the GHQ (total score of 12 or more) reduced from 78% (18 patients) to 9% (2 patients) from pre- to post-treatment. On the BDI the proportion of patients in the severe or moderate range (score of 20-63) decreased from 63% (12 patients) to 11% (2 patients) from pre- to post-treatment.

Table 2. Comparison of total scores for the clinical outcome measures for pre- and post-treatment assessments

<table>
<thead>
<tr>
<th>Clinical outcome measures</th>
<th>N</th>
<th>Pre-treatment M (SD)</th>
<th>Post-treatment M (SD)</th>
<th>M of the difference (SD of the difference)</th>
<th>95% CI of the difference</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GHQ</td>
<td>23</td>
<td>16.3 (5.77)</td>
<td>7.5 (3.95)</td>
<td>-8.9 (7.37)</td>
<td>(-12.1, -5.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>WHOQoL</td>
<td>22</td>
<td>26.3 (5.90)</td>
<td>31.1 (4.48)</td>
<td>4.8 (5.72)</td>
<td>(2.3, 7.4)</td>
<td>0.0007</td>
</tr>
<tr>
<td>BDI</td>
<td>19</td>
<td>21.5 (7.57)</td>
<td>7.6 (6.99)</td>
<td>-13.8 (6.55)</td>
<td>(-17.0, -10.7)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

BDI = Beck Depression Inventory: <9 = normal, 9-20 = mild, 21-63 = moderate-severe. GHQ = General Health Questionnaire: >11 = clinical range. WHOQoL = World Health Organisation Quality of Life measure. N=number of pairs; M=mean; SD = standard deviation; CI = confidence interval

Table 3 shows the individual item scores for the three outcome measures at pre- and post-treatment. All six GHQ negative (symptom and distress) items showed significant improvement (p<0.02 for each).

The proportion of patients in the caseness score range for these items also reduced substantially. Half of the six GHQ positive items showed significant improvement (p<0.05). For the WHOQoL, the item “overall quality of life” and two other items showed significant change from pre- to post-treatment (p<0.05), with a substantial increase in the number of patients scoring in the two most positive categories on several items. Eight of out the 21 BDI items showed significant improvement (p<0.05) with items “sadness”, “worthlessness” and “self-dislike” improving the most.

The proportion of patients in the caseness range for the BDI individual items also reduced markedly for many of the items. Suicide risk, as assessed by a BDI item, showed a reduction that was not statistically significant due to a “floor effect” as suicide risk was relatively low at the outset.
Table 3. Comparison of pre- and post-treatment individual item scores for the clinical outcome measures

<table>
<thead>
<tr>
<th>Outcome measure</th>
<th>Caseness</th>
<th>% pre</th>
<th>% post</th>
<th>Effect size (r)</th>
<th>Adj. P</th>
</tr>
</thead>
<tbody>
<tr>
<td>GHQ 12 (N=23)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GHQ negative items</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressed</td>
<td>% in categories</td>
<td>65%</td>
<td>9%</td>
<td>0.53</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Under strain</td>
<td>reflecting distress,</td>
<td>61%</td>
<td>9%</td>
<td>0.47</td>
<td>0.01</td>
</tr>
<tr>
<td>Overwhelmed</td>
<td></td>
<td>48%</td>
<td>4%</td>
<td>0.47</td>
<td>0.01</td>
</tr>
<tr>
<td>Lost confidence</td>
<td>pathology, or poor</td>
<td>48%</td>
<td>4%</td>
<td>0.46</td>
<td>0.01</td>
</tr>
<tr>
<td>Lost sleep</td>
<td></td>
<td>39%</td>
<td>4%</td>
<td>0.47</td>
<td>0.01</td>
</tr>
<tr>
<td>Worthlessness</td>
<td>function</td>
<td>35%</td>
<td>4%</td>
<td>0.49</td>
<td>0.01</td>
</tr>
<tr>
<td>GHQ positive items</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generally happy</td>
<td>% in categories</td>
<td>56%</td>
<td>100%</td>
<td>0.45</td>
<td>0.01</td>
</tr>
<tr>
<td>Able to concentrate</td>
<td></td>
<td>52%</td>
<td>96%</td>
<td>0.40</td>
<td>0.04</td>
</tr>
<tr>
<td>Able to face problems</td>
<td>reflecting wellbeing</td>
<td>70%</td>
<td>91%</td>
<td>0.38</td>
<td>0.04</td>
</tr>
<tr>
<td>Able to be decisive</td>
<td>or good function</td>
<td>65%</td>
<td>91%</td>
<td>0.31</td>
<td>0.12</td>
</tr>
<tr>
<td>Enjoy activities</td>
<td></td>
<td>52%</td>
<td>83%</td>
<td>0.30</td>
<td>0.12</td>
</tr>
<tr>
<td>Feel useful</td>
<td></td>
<td>78%</td>
<td>96%</td>
<td>0.29</td>
<td>0.12</td>
</tr>
<tr>
<td>WHOQoL total (N=22)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall quality of life</td>
<td></td>
<td>55%</td>
<td>86%</td>
<td>0.42</td>
<td>0.04</td>
</tr>
<tr>
<td>Energy level</td>
<td></td>
<td>36%</td>
<td>82%</td>
<td>0.43</td>
<td>0.04</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>% in categories</td>
<td>32%</td>
<td>82%</td>
<td>0.43</td>
<td>0.03</td>
</tr>
<tr>
<td>Activity of daily living</td>
<td>“good/satisfied”</td>
<td>50%</td>
<td>77%</td>
<td>0.31</td>
<td>0.20</td>
</tr>
<tr>
<td>Satisfaction with health</td>
<td>or “very good /</td>
<td>36%</td>
<td>72%</td>
<td>0.26</td>
<td>0.24</td>
</tr>
<tr>
<td>Personal relationships</td>
<td>very satisfied”</td>
<td>50%</td>
<td>77%</td>
<td>0.27</td>
<td>0.24</td>
</tr>
<tr>
<td>Financial security</td>
<td></td>
<td>36%</td>
<td>64%</td>
<td>0.29</td>
<td>0.24</td>
</tr>
<tr>
<td>Living conditions</td>
<td></td>
<td>64%</td>
<td>73%</td>
<td>0.17</td>
<td>0.25</td>
</tr>
<tr>
<td>BDI (N=19)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sadness</td>
<td></td>
<td>16%</td>
<td>84%</td>
<td>0.58</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pessimism</td>
<td></td>
<td>16%</td>
<td>74%</td>
<td>0.52</td>
<td>0.02</td>
</tr>
<tr>
<td>Self-dislike</td>
<td></td>
<td>21%</td>
<td>74%</td>
<td>0.52</td>
<td>0.02</td>
</tr>
<tr>
<td>Punishment feelings</td>
<td>% in most</td>
<td>37%</td>
<td>95%</td>
<td>0.51</td>
<td>0.04</td>
</tr>
<tr>
<td>Feel like crying</td>
<td>positive</td>
<td>37%</td>
<td>84%</td>
<td>0.51</td>
<td>0.04</td>
</tr>
<tr>
<td>Agitation</td>
<td>(least)</td>
<td>37%</td>
<td>79%</td>
<td>0.48</td>
<td>0.04</td>
</tr>
<tr>
<td>Worthlessness</td>
<td>pathological</td>
<td>16%</td>
<td>79%</td>
<td>0.49</td>
<td>0.04</td>
</tr>
<tr>
<td>Indecisiveness</td>
<td>response</td>
<td>21%</td>
<td>79%</td>
<td>0.44</td>
<td>0.04</td>
</tr>
<tr>
<td>Self-criticalness</td>
<td>category</td>
<td>26%</td>
<td>74%</td>
<td>0.46</td>
<td>0.05</td>
</tr>
<tr>
<td>Feeling a failure</td>
<td></td>
<td>21%</td>
<td>58%</td>
<td>0.47</td>
<td>0.05</td>
</tr>
<tr>
<td>Loss of interest in sex</td>
<td></td>
<td>26%</td>
<td>58%</td>
<td>0.48</td>
<td>0.05</td>
</tr>
<tr>
<td>Loss of interest</td>
<td></td>
<td>32%</td>
<td>79%</td>
<td>0.45</td>
<td>0.05</td>
</tr>
<tr>
<td>Tiredness/fatigue</td>
<td></td>
<td>21%</td>
<td>63%</td>
<td>0.45</td>
<td>0.05</td>
</tr>
<tr>
<td>Loss of pleasure</td>
<td></td>
<td>11%</td>
<td>37%</td>
<td>0.41</td>
<td>0.08</td>
</tr>
<tr>
<td>Sleep changes</td>
<td></td>
<td>11%</td>
<td>58%</td>
<td>0.41</td>
<td>0.08</td>
</tr>
<tr>
<td>Guilty feelings</td>
<td></td>
<td>26%</td>
<td>58%</td>
<td>0.36</td>
<td>0.18</td>
</tr>
<tr>
<td>Loss of energy</td>
<td></td>
<td>26%</td>
<td>58%</td>
<td>0.34</td>
<td>0.18</td>
</tr>
<tr>
<td>Irritability</td>
<td></td>
<td>32%</td>
<td>79%</td>
<td>0.34</td>
<td>0.18</td>
</tr>
<tr>
<td>Suicidality</td>
<td></td>
<td>74%</td>
<td>90%</td>
<td>0.26</td>
<td>0.30</td>
</tr>
<tr>
<td>Concentration</td>
<td></td>
<td>48%</td>
<td>63%</td>
<td>0.24</td>
<td>0.30</td>
</tr>
<tr>
<td>Changes in appetite</td>
<td></td>
<td>53%</td>
<td>63%</td>
<td>0.10</td>
<td>0.55</td>
</tr>
</tbody>
</table>

Notes: BDI = Beck Depression Inventory; Caseness = item score > 0. GHQ = General Health Questionnaire; Caseness = item score > 1. WHO QOL = World Health Organisation Quality of Life measure: Caseness = item score >3. Adj P= p values adjusted for multiple comparisons by the Stepdown Bonferroni Method.
Patient perceptions of service

On the client satisfaction survey, 93% (13 respondents) indicated being very or mostly satisfied with their psychology treatment and 100% (14 respondents) reported the treatment helped them manage their mental health problems better. Eighty-five percent of patients reported being very or mostly satisfied with the number of sessions they received. Barriers to seeking help earlier were: concern about cost (43%), not aware of how to access services (36%), and stigma (21%). One hundred percent reported that they would prefer to receive therapy at their GP clinic rather than go to a mental health service.

Staff perceptions of the service

Table 4 summarises primary healthcare staff perceptions of the psychologist’s impact on four aspects of primary mental health care. These qualitative results indicate the program improved timely access to mental health care for primary care patients, assisted GPs to manage the mental health needs of their patients better, and increased how well patients’ mental health needs were addressed.

Eight of nine (89%) primary healthcare staff reported this project improved access to therapeutic services and added additional resource for managing patients’ difficulties. One GP reported not referring any patients due to not identifying any suitable candidates.

Primary healthcare staff indicated frequent frustration with secondary mental health services prior to this project. Particular difficulties were: gaining access to services (typically because patients did not meet entry criteria); requests for assistance not being adequately responded to; or the secondary services not offering the kinds of support that primary staff believed the patient needed.

Staff perceived the project as supporting improved mental healthcare through: easy and relatively inexpensive access to psychological services that patients otherwise could not have accessed; improved uptake because it was offered in the familiar and non-stigmatising environment of the GP practice; improved support for GPs and practice nurses to meet the healthcare needs of their mental health patients; the synergistic relationship of the psychologist being able to extend the work primary healthcare staff could do and the primary healthcare staff being able to reinforce the psychologist’s work with the patient; and the ability to catch mental health problems earlier when adequate treatment takes less time. Another perceived advantage was that providing help in a primary healthcare setting may decrease the likelihood of the patient becoming reliant on the primary or secondary services.

The project was perceived as improving communication between primary care and secondary mental health services. Primary health staff perceived this benefit as deriving from (in decreasing order of frequency): the regular presence of a psychologist at the service, the psychologist’s role in up-skilling and supporting staff, and the ease of communication that proximity allowed. Improved communication with, and input from, other secondary mental health staff, was also noted as resulting from this project.
Table 4. Primary care staffs’ perspectives on the impact of the primary care psychologists’ pilot on aspects of service provision

<table>
<thead>
<tr>
<th>Access to mental health services for primary care patients</th>
<th>Timelines for mental health service provision</th>
<th>Assisting GPs’ management of mental health issues</th>
<th>Rate of mental health service use in the community</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Has helped</strong></td>
<td><strong>Has helped</strong></td>
<td><strong>Has helped</strong></td>
<td><strong>Has helped</strong></td>
</tr>
<tr>
<td>• Has improved access******</td>
<td>• Speed of access improved******</td>
<td>• Has helped GPs to improve their management of cases******</td>
<td>• Has improved rate of service use******</td>
</tr>
<tr>
<td>• Most referred patients would not otherwise had access*</td>
<td>• Able to get onto contact with patients quickly*</td>
<td>• Useful as part of better, sooner, more convenient*</td>
<td>• Definitely within this practice*</td>
</tr>
<tr>
<td>• Other options too expensive (self-pay, limited sessions through insurance)*</td>
<td>• Waiting times have improved*</td>
<td>•</td>
<td>• Probably, but little direct experience*</td>
</tr>
<tr>
<td>• It is a good start*</td>
<td>• Patients generally seen within a month*</td>
<td>•</td>
<td></td>
</tr>
<tr>
<td>• Good response around suicidal patient recently*</td>
<td>•</td>
<td>•</td>
<td></td>
</tr>
<tr>
<td><strong>Reasons it has helped</strong></td>
<td><strong>Reasons it has helped</strong></td>
<td><strong>Reasons it has helped</strong></td>
<td><strong>Reasons it has helped</strong></td>
</tr>
<tr>
<td>• Has fostered more connection between primary and secondary services.*</td>
<td>• Having psychologists at clinic on designated day helps*</td>
<td>• Another option for patients***</td>
<td>• Patients not as scared by referral for mental health help*</td>
</tr>
<tr>
<td>• Improved dialogue with nurses and psychiatrists has also helped.*</td>
<td>• Able to do brief interventions early*</td>
<td>• Good for patients who don’t want medication as the only option**</td>
<td>• Has helped recognise problems sooner*</td>
</tr>
<tr>
<td><strong>Further developments</strong></td>
<td><strong>Overall perceptions of service</strong></td>
<td><strong>Disadvantages of the project</strong></td>
<td><strong>Notes:</strong> Asterisks indicate number of respondents making this comment or similar</td>
</tr>
<tr>
<td>Looking forward to more contact with older persons services*</td>
<td>• Service has improved over last year*</td>
<td>• The disadvantages or limitations identified by respondents were: The availability of sessions only during working hours, the restricted number of sessions was sometimes insufficient, and that patients on other specific Chronic Care Management programmes could not access the service. One GP also speculated that a few people may find it stigmatising to receive this kind of care through their GP practice.</td>
<td></td>
</tr>
</tbody>
</table>

Notes: Asterisks indicate number of respondents making this comment or similar

**Clinical outcomes**—A large majority of the primary healthcare staff reported psychologist’s input to be useful and reported positive outcomes. Several GPs reported positive or very positive feedback from patients. The aspects of the service they regarded as most helpful included: The service comes to the patient, the listening and interpersonal skills of the psychologist, helpful advice and suggestions, and the treatment. Several indicated that additional or follow-up sessions would be helpful.

**Disadvantages of the project**—The disadvantages or limitations identified by respondents were: The availability of sessions only during working hours, the restricted number of sessions was sometimes insufficient, and that patients on other specific Chronic Care Management programmes could not access the service. One GP also speculated that a few people may find it stigmatising to receive this kind of care through their GP practice.
Discussion

This study has evaluated the introduction into a primary care setting of very brief, time-limited, individually tailored psychological interventions based on evidence-informed psychological therapies. This study extends previous literature by demonstrating that in primary care even very short interventions (4 sessions) have substantial positive clinical outcomes. Such positive outcomes are rarely as quickly achieved by patients who have become sufficiently unwell that they meet the criteria for admission to secondary mental health services prior to beginning effective treatment.

The study further extends previous literature by demonstrating that, in addition to the clinical outcomes, regular psychological input from the secondary services promoted a better relationship and integration between primary and secondary mental health services, leading to primary health staff feeling able to better manage patients with mental health difficulties.

The clinical outcomes, as reflected in change in standard measures, client satisfaction and self-assessment of outcome, and primary health staff perceptions, all indicate that this input added considerable value for the patients and the service. The outcomes showed significant improvements with decreased depression, anxiety, stress, and self-esteem.

Positive improvements were seen in both cognitive and physical symptoms related to depression. The overall quality of life also improved. The transition of a substantial proportion of patients from the clinical to the non-clinical range on the measures and many of the individual items is indicative of clinical significance as well as the statistical significance of the changes. These changes closely matched the targets of the intervention, indicating that even though this intervention was very brief, for this population it appears to have made a substantial difference.

Other variables, such as financial status and satisfaction with living conditions, did not change. This is to be expected as these were not generally intervention targets and were unlikely to be influenced by it. Feedback from patients and primary health staff was positive regarding satisfaction with the service and with perception of outcomes attributable to the intervention.

These findings are consistent with previous New Zealand studies in urban\(^6\) and rural\(^{14}\) settings, and overseas studies from the UK\(^{21}\) and South Africa\(^{22}\) that have shown benefit using different outcome measures.

This study extends these studies by evaluating the outcomes of a psychologist primarily working in secondary services and providing this input in primary health with a shared care approach; assessing the impact on aspects of quality of life in addition to symptom resolution; and exploring in greater detail the benefits to the primary healthcare system and its ability to provide mental health care.

Feedback from primary healthcare staff suggested satisfaction with the project. Impacts included: improved access to psychological services for patients and improved uptake because it was offered in the familiar and non-stigmatising environment; improved support for the GPs and practice nurses to meet the healthcare needs of their mental health patients; the synergistic relationship of the psychologist
being able to extend the work the GP could do, and the GP being able to reinforce the work the psychologist; and the providing a pathway for improved communication between primary health and the specialist mental health services.

These findings are consistent the Blueprint II prescription for the development of more integrated, shared care, and early intervention approaches. This study provides empirical evidence that this approach is potentially valuable.

This evaluation suggests that many primary care patients may benefit from brief intervention for mental health issues but current models of care across primary and secondary health services may put barriers in the way of people accessing psychological assistance unless they develop more severe mental illness. However, continued mild-moderate mental health difficulties still have considerable impacts on quality of life and other adverse outcomes such as reduced vocational status, relationship disturbance, diminished physical wellbeing, etc. 3

One of the limitations of this research is the small sample size. However, the generally medium to large effect sizes reported, and the evidence of clinically as well as statistically significant change despite the small sample size is indicative of robust outcomes. Another limitation is the absence of a control group. However, the patients had suffered with their mental health difficulties for at least six months prior to entering the project (often longer) despite the efforts of primary care clinicians. Thus, the patients can effectively be considered to having been their own “wait-list control” group during the period of months and years prior to the start of the intervention.

The stability of their symptoms with “treatment as usual” reduces the likelihood that the changes found with this intervention were due to non-treatment-related factors.

Competing interests: Nil

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References:


APPENDIX
Primary Care Psychology Pilot Survey (CMDHB)

Introduction: Your feedback
We would like to ensure that we are providing a quality and easily accessible service that is integrated into Primary Health Care services. Please take some time to complete this survey form and answer as frankly as possible. The survey is anonymous; you do not need to provide your name. To ensure that your comments are kept confidential the report produced will not identify any respondent. A copy of the final analysis will be available with your doctors sometime this year.

Please complete this questionnaire and return by mail (stamped addressed envelope enclosed) or drop it off at [location] on or before [date].

Thank you. We greatly appreciate your help.

Please indicate the type of services you received from this therapist:

- One-off assessment
- Brief Psychology service
- Did not complete treatment

Please circle your answer for each of the following questions:

1. How would you rate the quality of service you received from the psychologist?
   - Excellent
   - Good
   - Fair
   - Poor

2. Did you receive the type of therapy/service you wanted?
   - No, definitely not
   - No, not really
   - Yes, generally
   - Yes, definitely

3. How well did the service meet your needs?
   - Almost all of my needs met
   - Most of my needs have been met
   - Only a few of my needs have met
   - None of my needs have been met

4. Would you recommend this service to your friends or family if they were in need of similar help?
   - No, definitely not
   - No, not really
   - Yes, generally
   - Yes, definitely
5. Are you satisfied with number of times you were followed up?

- Quite dissatisfied
- Indifferent or mildly dissatisfied
- Mostly satisfied
- Very satisfied

6. Did this service help you to manage your problems better?

- They helped a great deal
- Yes they helped somewhat
- No, they didn’t really help
- No, they seemed to make things worse

7. In an overall, general sense, how satisfied are you with the service that you received?

- Very satisfied
- Mostly satisfied
- Indifferent or mildly dissatisfied
- Quite dissatisfied

8. If you were to seek help again, would you consider coming back to this program?

- No, definitely not
- No, I don’t think so
- Yes, I think so
- Yes, definitely

9. Do you think it was helpful to see the psychologist within the GP surgery instead of going to the mental health clinic?

- No, did not matter
- No, somewhat mattered
- Yes I do think it helped me
- Yes Definitely

10. Please circle any barriers that might have prevented you from seeking help earlier on?

- Stigma
- Distance to mental health service
- Finance/Cost
- Did not know whom to go to

Please provide any additional comments or suggestions you may have in the space below:

________________________________________________________________________

________________________________________________________________________

Thanks for completing this survey!
A retrospective case series of 44 patients with community-acquired Staphylococcus aureus pneumonia

Darren Bowles, Kyle Perrin

Abstract

Aim Staphylococcus aureus (S. aureus) community-acquired pneumonia (CAP) is a potentially devastating and life-threatening infection. Early detection and appropriate treatment is important to prevent morbidity and death. The aim of this case series was to investigate the patient demographics, clinical features, antibiotic treatment and complications of cases of community-acquired S. aureus pneumonia occurring in the Wellington region.

Method The case records of patients with radiographically confirmed community-acquired pneumonia and laboratory evidence to support S. aureus as the causative organism admitted to Wellington Regional Hospital over a 5-year period (2007-2012) were retrospectively reviewed.

Results A total of 48 presentations in 44 patients met the inclusion criteria. The majority of patients (63.6%) had underlying comorbidities. Although the mean CURB65 score was only one and fever was uncommon, 30% of patients were admitted to ICU and 16% died in hospital. Significant infective complications occurred in 48% with new lung cavitation in 20%.

Conclusion This series of patients with staphylococcal pneumonia confirms the significant morbidity and mortality of the infection. A low CURB65 score and lack of objective fever should not detract from the possibility of S. aureus. The presence of bacteraemia in patients with S. aureus pneumonia needs to be regarded as a potentially deleterious finding that may necessitate a change in treatment.

Staphylococcus aureus (S. aureus) remains a significant cause of community-acquired infection as indicated by steadily increasing hospital admission rates since the early 1990s. While skin and soft tissue infections account for the majority of staphylococcal infections in New Zealand (NZ), S. aureus is also a recognised cause of both community- and hospital-acquired pneumonia. S. aureus is a relatively uncommon cause of community-acquired pneumonia (CAP), accounting for 0.2%–4% of all cases in the UK, between 1 to 10% in the US, and 2%–3% in NZ. The proportion of community-acquired pneumonia infections attributable to S. aureus has remained fairly constant over the last half-century. S. aureus has been found to be responsible for 23% of all cases of severe community-acquired pneumonia admitted to an intensive care unit in NZ.

Early diagnosis of S. aureus CAP is important to effectively treat this potentially fulminant infection. This serious condition has in recent times been further brought to prominence following episodes of pandemic influenza and the emergence
of Panton-Valentine leukocidin toxin (PVL)-producing strains, including community-associated methicillin-resistant *S. aureus* (CA-MRSA) in the Oceania region.\(^2\,13\)

The aim of this case series was to investigate the patient demographics, clinical features, antibiotic treatment and complications of cases of *S. aureus* CAP in the Wellington region of NZ.

**Method**

All patients diagnosed with CAP with concomitant isolation of *S. aureus* from sputa, pleural fluid and/or blood admitted to Wellington Regional Hospital (WRH) over a 5-year period (2007-2012) were retrospectively reviewed. Approval to use patient record data was sought from the Decision Support Unit (DSU) at WRH. The authors decided that Ethics Committee approval was not required for the case series review. Patient data was de-identified and treated with the utmost confidentiality.

Case records were identified using the ICD-10 code for ‘pneumonia due to staphylococcus’ (J15.2). Additional cases were identified from the sputum culture results database of the WRH microbiology laboratory. Inclusion criteria included focal consolidation on chest radiograph (CXR) consistent with a new diagnosis of CAP and microbiological confirmation of *S. aureus*.

We excluded cases with no evidence to support *S. aureus* as the underlying infective cause of CAP, hospital-acquired *S. aureus* pneumonia and cases where evidence supported a primary vascular site of inoculation with *S. aureus*.

Sources of information included patient case files, digital radiology, microbiological data and patient electronic records. Data were collected on patient demographics, clinical history and observations, laboratory blood results, radiological investigations, antibiotic treatment and infective complications.

Information was collected using a data template and transferred to a Microsoft Excel software database prior to analysis.

**Results**

Forty-four subjects meeting the inclusion criteria were identified over a 5-year period (2007–2012). Four patients re-presented with *S. aureus* pneumonia between 5 days and 4 months following their initial presentation, hence there were 48 presentations in total available for analysis.

The median age was 57 years; 36% were aged 56–75 years; 20% were aged over 75 years and 16% (n=7) were aged 16–25 years. The majority (56.6%) were current or ex-smokers.

Ethnicity was stated as NZ European in 28 and Māori in 8 patients; 54% (n=37) of patients were resident in areas designated either deciles 9 or 10. (Deciles 9 and 10 signify socially deprived residential areas according to the New Zealand Deprivation Index.\(^{14}\))

A total of 63.6% (n=28) of the patients had underlying comorbidities as shown in Table 1.
Table 1. Comorbidities

<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>% (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic lung disease</td>
<td>34 (15)</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>(1)</td>
</tr>
<tr>
<td>Chronic renal impairment</td>
<td>18 (8)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11 (5)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>9 (4)</td>
</tr>
<tr>
<td>Intravenous drug use</td>
<td>7 (3)</td>
</tr>
<tr>
<td>Chronic alcoholism</td>
<td>7 (3)</td>
</tr>
<tr>
<td>Chronic hepatitis C viral infection</td>
<td>7 (3)</td>
</tr>
<tr>
<td>Long-term corticosteroid therapy</td>
<td>13.6 (6)</td>
</tr>
<tr>
<td>Long-term immunosuppressive drug therapy</td>
<td>11 (5)</td>
</tr>
</tbody>
</table>

Of the eight patients with chronic renal impairment, two were receiving regular haemodialysis at the time of admission and five patients had an estimated glomerular filtration rate (eGFR) between 19–33 ml/min/1.73m².

Overall 39.6% (n=19/48) of presentations occurred in the 3 months Sept–Nov. For all 48 presentations the median time from symptom onset to hospital admission was 4 days.

Sweats, rigors and chills were reported by three, seven and six patients respectively. One patient described a preceding flu-like illness. Five patients, whose ages ranged from 57 to 88 years, were acutely confused at presentation.

The mean and median CURB65 scores for all 48 presentations were 1; 14 patients had a score of 1 and 13 patients had a score between 2–5. The frequency of CURB65 scores of 2 and above is provided in Table 2.

Data was available to assess Systemic Inflammatory Response Syndrome (SIRS) criteria for 45 presentations of which 84% (n=38) satisfied the American College of Chest Physicians (ACCP) definition of SIRS.¹⁵

Table 2. Distribution of CURB65 scores

<table>
<thead>
<tr>
<th>Score</th>
<th>Number of presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

Baseline clinical features are shown in Table 3.
Table 3. Baseline clinical features

<table>
<thead>
<tr>
<th>Observations</th>
<th>% (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>64.5 (31/48)</td>
</tr>
<tr>
<td>Cough</td>
<td>68.8 (33/48)</td>
</tr>
<tr>
<td>Fever (≥ 37.5)</td>
<td>43 (19/44)</td>
</tr>
<tr>
<td>Chest pain</td>
<td>43 (21/48)</td>
</tr>
<tr>
<td>Chills</td>
<td>6.8 (3/44)</td>
</tr>
<tr>
<td>Rigors and chills</td>
<td>6.8 (3/44)</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>18 (8/44)</td>
</tr>
<tr>
<td>Oxygen saturation (median, %)</td>
<td>93</td>
</tr>
<tr>
<td>SBP/DBP (median, mmHg)</td>
<td>125/70</td>
</tr>
<tr>
<td>Respiratory rate (median, breaths per min)</td>
<td>24</td>
</tr>
<tr>
<td>Confusion</td>
<td>11 (5/44)</td>
</tr>
</tbody>
</table>

39.6% (n=19/48) of admission chest radiographs showed evidence of multifocal consolidation. Radiological evidence of new lung cavitation was seen in 20% (n=9), secondary pneumothorax in 6.8% (n=3) and pleural effusion in 27% (n=12).

Median results for the baseline blood samples showed: Hb 122 g/L; White cell count 10.64×10⁹/L; Neutrophil count 9.17×10⁹/L; Platelet count 199×10⁹/L; Alb=30 g/L and CRP=142 mg/L.

*Staphylococcus aureus* was cultured from 90.7% (n=39/43) of sputum samples collected. Of these 87% (n=34) were reported as moderate or heavy growth of *Staphylococcus aureus*. In total 33 sets of blood cultures were collected from 31 patients. Blood cultures were positive for *Staphylococcus aureus* in 36% (n=12). Of these, three patients did not produce sputum, four patients had moderate or heavy growth of *Staphylococcus aureus* in sputa, three patients had light growth of *Staphylococcus aureus* in sputa and two patients’ sputa were negative for *Staphylococcus aureus*.

Pleural aspirates were performed in six patients with radiographic evidence of a pleural effusion, of these 50% (3/6) were culture positive for *Staphylococcus aureus*.

*Staphylococcus aureus* was flucloxacillin-sensitive in 86% (n=24/28) of isolates. Erythromycin resistant *Staphylococcus aureus* was found in 12.5% (n=2/16) of sputum isolates. MRSA was identified on sputum culture in four patients and blood culture in one additional patient. Four of the five patients tested for influenza were positive for concurrent infection.

Intravenous cefuroxime or ceftriaxone were used as initial therapy in 79% (n=38) of presentations. Addition of a macrolide in combination with a cephalosporin occurred in 65% (n=31) of presentations.

Of those who were coadministered a macrolide, 67% received oral roxithromycin, 16% received intravenous erythromycin and 7% received intravenous clarithromycin. Intravenous flucloxacillin was used in 9% (n=4) and anti-MRSA agents (glycopeptides, clindamycin and cotrimoxazole) were used in 9% (n=4) of patients, including three treated with intravenous vancomycin.

Revision of intravenous antibiotic therapy occurred in 62.5% of presentations (n=30); in 60% (n=18/30) this included a change to intravenous flucloxacillin. The median time from admission to the first revision of antibiotic therapy was 3 days for 89.5%
(n=43) of presentations, which generally correlated with confirmation of culture results.

An anti-MRSA agent was introduced at subsequent revisions of therapy in a further 27% (n=12). The median time from admission to revision of antibiotic therapy to flucloxacillin and/or an anti-MRSA agent was 4 days for 36 presentations.

In total, intravenous flucloxacillin was used in 64% (n=28), intravenous clindamycin in 16% (n=7) and intravenous vancomycin in 18% (n=8).

Duration of inpatient antibiotic therapy ranged from 3 to 49 days with a median of 10 days for all 48 presentations. Length of hospital stay ranged from 3 to 50 days, with a median of 11 days for 45 presentations.

In total 48% (n=21) of patients developed significant infective complications as shown in Table 4.

### Table 4. *S. aureus* CAP complications

<table>
<thead>
<tr>
<th>Complications</th>
<th>% (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>New lung cavitation</td>
<td>20 (9/44)</td>
</tr>
<tr>
<td>Pleural effusion (including empyema)</td>
<td>27 (12/44)</td>
</tr>
<tr>
<td>Emphyema</td>
<td>7 (3/44)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>7 (3/44)</td>
</tr>
<tr>
<td>ARDS</td>
<td>16 (7/44)</td>
</tr>
<tr>
<td>Pleural aspirate positive</td>
<td>50 (3/6)</td>
</tr>
<tr>
<td>Blood culture positive</td>
<td>36 (12/33)</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>7 (3/44)</td>
</tr>
</tbody>
</table>

Seventy-five percent (n=9/12) of cases with positive blood cultures developed significant infective complications compared to 38% (n=8/21) with negative blood cultures.

A total of 13 (30%) patients were admitted to ICU, all required mechanical ventilation, and 11 additionally required inotrope therapy. Of the 13 ICU patients, nine had multifocal consolidation on their admission CXR, 8 had blood cultures positive for *S. aureus* and 4 died in hospital.

Overall in-hospital mortality was 16% (n=7/44). A further three patients with significant comorbidities died within 6 months of discharge from hospital.

**Discussion**

This is the first retrospective case series of consecutive patients with CAP due to *S. aureus* in NZ. It confirms that this infection is more prevalent in patients with comorbidities who have social deprivation and demonstrates that the infection is associated with significant morbidity and mortality.

Despite initially low CURB65 scores and a lack of fever at presentation, patients in this case series had a high rate of ICU admission and death.

There was high use of intravenous cephalosporins, particularly cefuroxime, and oral roxithromycin, in accordance with the WRH antibiotic guidelines for CAP. Antibiotic
treatment was changed in the majority of patients after an appropriate period correlating to confirmation of culture results.

Patients who required ICU admission tended to have multifocal consolidation on their admission CXR and bacteraemia on blood culture.

Limitations of this case series include the retrospective nature of the data and the small number of patients found to meet the inclusion criteria. The presence of *S. aureus* in sputum in isolation is not necessarily indicative of underlying pulmonary infection. However the official microbiological sputum analysis report, and patient’s past sputa data, were reviewed to distinguish between colonisation and infection.

The findings were similar to a UK case series by Woodhead et al. who found a comparable number of cases complicated by new cavitation and pneumothorax, and that the presence of bacteraemia was associated with adverse outcomes.

At baseline assessment, *S. aureus* pneumonia is clinically indistinguishable from that caused by other pathogens. However, useful indicators include radiological evidence of multifocal consolidation, cavitation, pneumatoceles or secondary pneumothorax.

The treatment of undifferentiated community-acquired pneumonia should follow the local antibiotic guidelines, and be guided by severity assessment at baseline. Upon microbiological confirmation of *S. aureus* a change of treatment to antibiotic therapy with high anti-staphylococcal activity should be considered. Intravenous flucloxacillin is appropriate for the initial treatment of the majority of methicillin-susceptible *S. aureus* (MSSA) strains. Longer intravenous antibiotic courses for up to two weeks duration have been recommended to treat patients with uncomplicated staphylococcal bacteraemia.

Virulent PVL-positive *S. aureus* strains, including both MSSA and CA-MRSA, resistant to standard anti-staphylococcal antibiotic treatment are a recognised cause of severe CAP associated with high mortality. In recent years there has been a reported increase in the prevalence of PVL-positive CA-MRSA strains in Australia. In NZ, 18% of Auckland MSSA clinical isolates causing pneumonia have been found to be PVL-positive.

Several cases of fatal necrotising pneumonia due to PVL-positive MSSA and CA-MRSA have been described in Australia. At least one case of necrotising pneumonia due to PVL-positive MSSA has been described in NZ.

11% of *S. aureus* isolates in this case series were methicillin-resistant. No *S. aureus* isolates in this series were tested for PVL toxin. It is possible that PVL-positive *S. aureus* went undetected among the most severely affected patients in this case series.

CA-MRSA strains are universally resistant to all currently available beta-lactams. Recommended antibiotic therapy for CAP caused by PVL-positive *S. aureus*, both MSSA and CA-MRSA strains, includes intravenous clindamycin and linezolid, and rifampicin. Clindamycin and linezolid achieve good lung penetration and have the ability to reduce toxin formation via bacterial protein synthesis inhibition, while rifampicin kills intracellular staphylococci. A caveat to the use of clindamycin for
long treatment courses is the exclusion, subsequent to starting treatment, of inducible resistance using a D-test.\textsuperscript{35}

Treatment failure has been associated with the use of vancomycin as first-line antibiotic therapy for necrotising staphylococcal pneumonia\textsuperscript{36}. Vancomycin poorly penetrates lung tissue, failing to attain levels at or above the MIC for MRSA\textsuperscript{37}. Other concerns with vancomycin include its lack of bacterial toxin inhibition and increasing reports of resistance.\textsuperscript{38,39} For these reasons vancomycin is generally not recommended for the treatment of MRSA pneumonia. Another agent that should be avoided for the treatment of staphylococcal pneumonia is the lipoglycopeptide daptomycin which is inactivated by pulmonary surfactant.\textsuperscript{40}

Testing for PVL toxin may be indicated in patients presenting with confirmed severe \textit{S. aureus} CAP, particularly with necrotising features and haemoptysis. A longer course of intravenous antibiotic therapy of up to 2 weeks duration should be considered for patients with uncomplicated \textit{S. aureus} bacteraemia or culture positive pleural fluid.

For severe MSSA and MRSA CAP it is recommended that the managing medical/intensive care team seek the advice of the local infectious disease/clinical microbiology team at the earliest opportunity.

In conclusion, \textit{S. aureus} as a causative organism should be suspected in patients presenting with severe community-acquired pneumonia, particularly with radiological evidence of multifocal consolidation and findings suggestive of underlying tissue necrosis such as cavitary changes.

This case series suggests that low CURB65 scores and absence of fever at presentation should not detract from the possibility of \textit{S. aureus} as the underlying cause. Adequate doses of intravenous anti-staphylococcal antibiotics must be started as soon as microbiological confirmation is available, and there should be a high index of suspicion for subsequent complications should patients fail to respond appropriately to treatment.

\textbf{Competing interests:} None.

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References:
Asystole with carotid sinus hypersensitivity

Jackson J Liang, Eric R Fenstad

A 72-year-old male experienced syncope after standing from a seated position. Bystanders noted he was pulseless, and cardiopulmonary resuscitation was briefly performed until emergency medical services arrived, when he was noted to have a pulse. On hospital admission, electrocardiogram showed normal sinus rhythm with no ST-segment or T-wave abnormalities. Heart rate was 64 beats per minute and blood pressure was 133/67 mmHg. He admitted to syncope 4 years prior while driving.

When carotid sinus massage was performed, his breathing ceased and he immediately lost consciousness. Simultaneous telemetry captured an 8-second period of sinus arrest and ventricular asystole. Dual-chamber pacemaker was implanted for symptomatic carotid sinus hypersensitivity and he remains asymptomatic at long-term follow-up.

Carotid sinus hypersensitivity, a cause of syncope, can be readily detected with physical examination in a monitored setting. Permanent pacing is indicated for recurrent syncope when carotid sinus massage induces 3 or more seconds of ventricular asystole.

Figure 1. Telemetry recording demonstrating 8-second period of sinus arrest with ventricular asystole (time 16:46:57–16:47:05) induced by carotid sinus massage
Learning points:

- Carotid sinus hypersensitivity is an uncommon cause of syncope which can be diagnosed with carotid sinus massage during cardiac monitoring.
- Implantation of permanent pacemaker in symptomatic patients is indicated when carotid sinus massage induces at least 3 seconds of ventricular asystole.

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A rare case of occult gastrointestinal bleeding: a submucosal colonic lipoma mimicking a malignant tumour

José A Guirola Ortíz, Guillermo Muñoz González, Victoria Mayoral Campos, José L de Benito-Arévalo, Beatriz Carro Alonso

A 52-year-old man presented a 2-month history of cramping lower abdominal pain, change in bowel habits with reduced consistency, altered stool frequency and excessive flatus.

His physical examination and laboratory test were normal except for a routine stool study that showed a positive result in fecal occult blood test. A colonoscopy revealed an ulcerated tumour with friable tissue and a pediculated polyp of 8 mm at the splenic flexure.

Biopsies obtained in the colonoscopy shown a focal fibrosis, granulated tissue and increased vascularity in which the pathologist concluded as a possible colonic hemangiosarcoma.

Abdominal CT revealed a large mass located in the colon at the splenic flexure with an approximated size of 45 mm in diameter and a low-density attenuation (-80 HU), which corresponds to fatty tissue (Figure 1).

Abdominal CT also revealed a large thickening of the colonic wall (8.0 × 5.0 cm) with a collapse of the colonic lumen (Figure 1); the radiologist concluded a submucosal lipoma with a doubtful colonic neoplasm.

Figure 1. Abdominal CT (axial and coronal views). Shows a large fatty mass (yellow arrows) located in the splenic flexure of the colon, with a low density same as the normal adjacent mesenteric fat. Also it shows a thickening of the colonic wall with a collapsed lumen (white arrows)
Laparotomy surgery was performed showing a large fatty mass and an extended right hemicolectomy procedure was done. The surgical specimen obtained showed a pediculated tumour located within the colonic submucosa (Figure 2) with a broad eroded and ulcerated surface, oval shape, a size of 6 cm in the maximum diameter located at 19 cm of the ileocaecal valve (Figure 2).

**Figure 2.** Macroscopic view of the surgical anatomical specimen, shows a fatty tumour located within the submucosa (white arrow), an ulcerated surface from chronic friction and the origin of the intestinal bleeding (black arrow), and the normal colonic mucosa (blue arrow)
Microscopy view showed a proliferation of mature adipocytes within the colonic submucosa, showing a wide and clear cytoplasm and small-elongated nucleus located in the periphery.

The fatty lesion presented a covering sheath that corresponds to colonic mucosa with fibronectic changes, inflammatory cells and newly formed vessels secondary of chronic friction and the presence of acute and chronic hemorrhagic findings (Figure 3). The final diagnosis was a submucosal lipoma, a benign fatty tumour seen in the colon with right side preference (51–70%).

These benign tumours are 0.035–4.4% of all intestinal neoplasms.¹

**Figure 3.** Microscopy view of the specimen. Shows a large infiltration of inflammatory cells and bleeding due to the chronic friction (white arrow). Multiple normal mature adipocytes with peripheric nucleus (black arrow)

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**Learning Points:**

- Not all gastrointestinal bleeding comes from malignant tumours
- Always remember Hounsfield units

- −100 to −60 HU, is always fat
- Colonic lipoma are 0.035-4.4% of all intestinal neoplasms,¹ with a right-sided preference (51–70%)¹
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References:
Is *Campylobacter concisus* an unrecognised cause of diarrhoea in New Zealand?

The introduction of a range of interventions to reduce *Campylobacter* spp. contamination of poultry is thought to coincide with a marked decline in campylobacteriosis in New Zealand in recent years.\(^1\)

However, in contrast to international data, infection rates in New Zealand remain high, particularly in young people.\(^2\) This gains greater significance with emerging data linking recent campylobacter infection to increased risk of chronic gastrointestinal (GI) disease.\(^3\) It is also likely that the true incidence of campylobacteriosis is currently underestimated.

Studies have identified emerging and potentially pathogenic species of these bacteria,\(^4\) and these species are unlikely to grow at the higher temperature routinely used to isolate the more common thermophilic strains of *C. jejuni* and *C. coli*. Thus, *C. concisus*, a nonthermophilic *Campylobacter* spp. that is reportedly associated with human gastroenteritis\(^5\) and Crohn’s disease,\(^6,7\) may also account for a proportion of cases of acute diarrhoea of otherwise unknown aetiology.

We cultured 200 stool samples to determine the prevalence of *C. concisus* in faecal samples from individuals presenting with acute enteritis in Christchurch, New Zealand. Samples were plated on CCDA, a blood-free campylobacter isolation media and incubated at 42°C in 10% CO\(_2\) for 48 hours.

In parallel, a faecal suspension was transferred to the surface of a 0.6 µm mixed cellulose ester filter placed on the surface of blood agar plates prepared using 10% sterile defibrinated horse blood.\(^6\) After 40 min incubation at 37°C, the filters were aseptically removed and the plates incubated at 37°C in a hydrogen enriched microaerobic environment for up to 4 days. Small circular colonies or spreading films showing evidence of highly motile spiral bacteria when viewed by light microscopy were then identified using MALDI-TOF mass spectrometry.

*C. jejuni/coli* were isolated from 14 samples using both methods. An additional four isolates were recovered following incubation at 42°C on selective media only, whereas another 8 culture positive isolates were detected by the filtration method only.

These findings suggest that either method will detect the presence of *Campylobacter* spp. in human stool samples, and that the prevalence of *C. jejuni/coli* in people presenting with acute enteric infection is approximately 13%. In contrast, only one *C. concisus* isolate was detected in the 200 samples, and this was by the filtration method only. This finding is in sharp contrast to that of a recent study in the same community where PCR-denaturing gradient gel electrophoresis was used to examine faecal samples from healthy volunteers and individuals with diarrhea.\(^8\) In that study, *C. concisus* was found notably more often than *C. jejuni/coli*, irrespective of whether the participant was healthy or suffering from diarrhoea.
This implies that carriage of these bacteria is more widespread than previously recognised in New Zealand but the prevalence of oral versus gut genomospecies of *C. concisus* was not determined. Our finding of only one *C. concisus* isolate from 200 samples (0.05%) suggests that these bacteria are unlikely to account for more than a small proportion of diarrheal cases of unknown aetiology in our community.

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**References:**

Medical inspection of school children

Excerpt from Editorial published in 1912 March issue of the NZMJ.

Finally, it is necessary to refer to the scheme of medical inspection of school children. We are not yet convinced that such a scheme is either necessary or advisable in New Zealand, and would be glad to hear arguments instead of assertions in its favour.

It has at least one very serious objection, for this scheme will take away from parents, to a great extent, the natural obligation of providing for their sick and defective offspring. We have had some experience acting as a member of an Orphanage Board, and we have heard parents apply to have their children taken into the Orphanage, on the ground that they (the parents) could manage much better if we "took the kids off their hands."

If our readers will pardon the vulgarity of the phrase, it appears to be established that when children are of school-age, as far as their ailments are concerned, Dr. Valintine and the teachers will oblige parents by taking "the kids" off their hands. The "boys of the bull-dog breed" in future will not only honour their parents, but also the Department of Public Health.

As for the scheme itself, it is ill-devised, and wholly discreditable to its originators. There is very little in it that is original, and the worst features of the English scheme have been borrowed. The system in England of making use of the out-patient departments of hospitals for treatment of school children has already been shown to be a failure from every point of view, and yet it is to be one of the strong points of Dr. Valintine's intentions.

If Dr. Valintine had consulted the profession, who after all can make or mar his scheme, he would have brought forward something more worthy of the occasion, but a gentleman whose own means of livelihood are not likely to be affected by innovations cannot properly appreciate what these changes may mean to the general practitioners.

It is clear that the only people who are to suffer for this plan of medical inspection are the private doctors. The doctors who have regular salaries are not to be given the right to earn a martyr's crown.
E-cigarettes in the USA

Electronic cigarettes, or e-cigarettes—battery-operated nicotine-delivery devices that mimic the look and feel of smoking by vaporizing a liquid solution such as propylene glycol—have been shown to be useful in helping smokers to cease using tobacco. However, there may be a downside to their use and three perspective papers in the NEJM review this topic.

A concern has been raised that non-smoking teenagers experimenting with e-cigarettes may be led to real cigarettes—a gateway effect. Another obvious concern is that the long-term effects of e-cigarette usage are not known. Obviously a lifelong addiction to nicotine is highly likely to be harmful. Bearing this in mind it has been suggested that there should be a ban on selling all such products to anyone under 21 years of age, given the risks for lifelong nicotine addiction associated with early use.

A proposal—“Tobacco 21”—from May 2014 it will be illegal to sell tobacco products and e-cigarettes to persons younger than 21 years in New York. A sensible idea?


Effect of beta blockers on mortality after myocardial infarction in adults with chronic obstructive pulmonary disease (COPD)

Beta blockers (β blockers) are known to reduce infarct size and improve survival after myocardial infarction. However, they may be underused in those with COPD as they may worsen the COPD. This UK population-based cohort study looks at this problem.

Over 1000 COPD patients who suffered a myocardial infarction over a 6-year period were included in the evaluation. β blockers initiated during the admission for myocardial infarction were associated with substantial survival benefits (fully adjusted hazard ration 0.50). Patients with COPD already taking a β blocker before their myocardial infarction also had a survival benefit (0.59).

The researchers conclude that these data suggest that β blockers should be used more widely in COPD patients who have had a myocardial infarction. Safety to date is good in these patients, but further evaluation of the safety of β blockers in this high risk group could be required to change current prescribing practice.


Adalimumab plus methotrexate or methotrexate alone in the treatment of rheumatoid arthritis

Biological agents offer good control of rheumatoid arthritis, but the long-term benefits of achieving low disease activity with a biological agent plus methotrexate or methotrexate alone are unclear.
This report is of a trial evaluating this problem. Over 1000 patients with early rheumatoid arthritis (<1 year) naïve to methotrexate were involved. The patients were recruited from 161 sites worldwide. They were randomised to receive adalimumab (40 mg every other week) plus methotrexate (initiated at 7.5 mg/week, increased by 2.5 mg every 1–2 weeks to a maximum weekly dose of 20 mg by week 8) or placebo plus methotrexate for 26 weeks.

At 26 weeks more patients in the combination group (44%) had achieved a low disease activity target compared with 24% of the methotrexate group. Patients achieving the stable low disease activity target on adalimumab plus methotrexate who withdrew adalimumab mostly maintained their good responses.

An editorial commentary points out that it would have been preferable to have had methotrexate plus corticosteroids rather than methotrexate monotherapy as the comparator group. We note that methotrexate is very cheap and adalimumab is extremely expensive.

John Edwin Horton

1931–2012

John was born in London, England in 1931. His father was a New Zealander and a member of the family associated with the NZ Herald. His mother was part French.

The family returned to New Zealand in 1939 at the outbreak of the War and John’s schooling continued as a boarder at St. Peters in Cambridge where he gave great service in subsequent years culminating in his appointment as Chairman of the Board of Governors, a position which he held for many years.

In 1945 John entered Kings College as a boarder in School House where he was well liked by his peers. He was academically smart and made steady progress through school before proceeding to University in 1950 and a career in Medicine. John Horton graduated from the University of Otago in 1955 followed by 2 years as a House Surgeon in Auckland.

Deciding on a surgical career, John travelled to the United Kingdom for postgraduate study and subsequently obtained Fellowships in Surgery from the Royal Colleges of Surgery of Edinburgh and England.

John returned to New Zealand in 1964 and was appointed Surgical Tutor Specialist at Green Lane Hospital (a full-time surgical position) plus teaching 6th year medical students attached to the Auckland sub-faculty of the University of Otago. John was an excellent colleague and as a surgeon very competent, showing good judgement and looking after his patients very well.

Completing this appointment in 1968, John joined the New Zealand Services Medical Team in Vietnam at the Bong Son 100-bed hospital in Binh Dinh province. The primary role of the team was the provision of medical and surgical care to South Vietnamese civilians but they also treated military casualties. 1968 was the height of the Vietnam War and the unit was extremely busy.

After completing his tour of duty John returned to Auckland and an appointment to the part-time visiting staff of Green Lane Hospital. After retirement from Green Lane he continued his private practice at St. Marks Clinic in Remuera where he invited the writer to join him in the mid-1980s. He retired completely in the early 1990s.

For many years he was a member of the Northern Club and the Auckland Golf Club, where he played regularly.

John married twice, firstly to Margaret (nee Ross); 3 children. In 1976 he married Judith (nee Turner) who survives him; 2 children.

John: Rest in Peace my friend.

R. G. Kay (a friend since Kings College and colleague of John) wrote this obituary.
Barbara Farnsworth Heslop

Immunologist, academic

There was probably very little possibility the University of Otago would ever forget one of its most brilliant graduates—and academics—but in establishing the Barbara Heslop Memorial Fund, it has wisely left nothing to chance. The fund has been set up to support a scholarship for research students at Otago, a thoroughly appropriate purpose given Emeritus Prof Heslop’s second-to-none record as a medical researcher.

Dr Heslop, CBE, MD, FRCPath, FRACS, FRSNZ, died in Dunedin in late December, 2013, just a month before her 89th birthday.

Among Dr Heslop’s many post-retirement activities was typing up her recollections of a life well lived and it is on these revealing writings this obituary leans rather heavily.

For example, here is how she reflected on her “retirement”: “I retired from working four times. The first was the compulsory retirement at age 65 (from the university, in 1990). It is no longer compulsory to retire at 65. But at the time I had a programme grant from the MRC [Medical Research Council] that had another year or two to run, so I stayed with that. When that came to an end I still had the FRACS [Fellow of the Royal Australasian College of Surgeons] course that I was convening and teaching.

“I was teaching immunology in Melbourne (coals to Newcastle!) and Singapore and elsewhere into the 1990s. I decided to stop teaching in Dunedin in the mid-1990s when one of the candidate assessments said ‘perfect’. I might perhaps not have stopped teaching if the assessments had not been as good. But it was clear that there was only one way to go from there down. So it was the right time to stop. That was my third retirement.

“Then, I was on the hospital board (then called a CHE for Crown Health Enterprise and board members were company directors) for three years from 1993. But that’s another story. It wasn’t exactly a job, although we got paid for it, but I learned a bit about hospital management mainly how not to do things in those years.” She also notes that in 1999 she returned to teach undergraduates to “help out with a staffing crisis.”

But it is necessary to turn the clock right back to get a full picture of the exceptional life of Dr Heslop, who was born in Auckland in January, 1925, to John Sampson Cupit (emigrated by Derbyshire, UK) and his wife Isabel. They gave their daughter
“Farnsworth” as her second name because her paternal grandmother was Caroline Farnsworth.

Dr Heslop's parents had a major influence on her life, especially her father who worked in the child welfare department in Auckland. She says both parents were "bookish—we had quite a scholarly home library for New Zealand suburbia, virtually the complete works of the classical English poets and writers. Nobody else that I knew had anything quite like this. I never had trouble looking up literary references at school—we had them in the house. My mother rated Darwin’s *Origin of Species* as a favourite book."

While attending Epsom Girls Grammar School, Dr Heslop says she always knew she would go on to university as well even though “for most girls” in the 1930s, that was not a realistic goal—“in my primary school days, compulsory schooling stopped at about 12, when one got the so-called proficiency certificate.”

But it never occurred to her that university study was an unusual expectation. “My father had always said ‘You can do anything you want to’, and I didn't question it, so I was really somewhat cocooned from the prevailing social mores. My mother also advised against getting married too early and without qualifications and getting stranded in suburbia. So the ‘glory box’ was a non-issue—in fact the whole idea was a bit of a joke,” she wrote.

But Dr Heslop's field of interest was medicine, not one that many women were attracted to in those days. She recalls many fathers obstructed most ambitious girls, or at least steered them into domesticity. Hers, however, did the opposite, so, after winning a University Entrance scholarship, she moved south to Dunedin to attend Otago University.

Getting into the Medical School during the latter stages of the Second World War was relatively “easy” and finding student life in the south greatly agreed with her, she graduated with a degree in medicine in 1948, then began working towards her MD. Incidentally, 14 members of her graduating class went on to become professors.

It was around this time she met her future husband John Heslop, who was one year behind her at medical school. They were to get to know each other a lot more as house surgeons at Dunedin Hospital around 1950.

Looking back at her life at the Medical School, Dr Heslop recalls her ambitions to become a “career woman” were at variance with the prevailing attitudes of the day which “didn't really expect very much of medical women after graduation”, an attitude that lasted until well into the 1970s.

“The opinion was widely expressed that the women dropped out (or dropped down) for domestic reasons. In many ways this viewpoint seemed justified—women simply didn’t go for the top jobs. It was only when the numbers of women in the medical classes started to increase that the necessity to arrange jobs a little more imaginatively than had been done previously occurred to the administration.” It was, however, very much a male-dominated world. Women were invited to attend functions after the men-only dinners. Yet, she wrote, “strangely enough, we didn't object; it was just the way things were.”
Undaunted, Dr Heslop's first career step was to become an assistant lecturer at the Medical School’s Department of Pathology in 1950, where she stayed until 1953. She recalled living as a group in the house surgeon’s quarters was “great fun…working long hours and letting off steam in the remainder.”

But a life-changing romance was also blooming in the background. She married John Heslop in Auckland in January, 1953 and by the end of that year found herself on a cargo ship, the Port Vindex, en route to the UK (her husband was the ship’s surgeon), fulfilling a directive from her mother years earlier that she “must travel.”

The young couple settled in London where John became a surgical officer at the King Edward Memorial Hospital in Ealing while his wife landed a research job at Great Ormond Street Hospital, for sick children, which had been organised for her by Charles Hercus in Dunedin.

At the end of 1956 the Heslops returned to New Zealand, this time with 10-day-old baby daughter Helen in hand, as John had been appointed a senior register and surgical tutor at Dunedin Hospital. Being a young mother could have spelled the end of Dr Heslop’s academic career, but although she made no immediate plans to return to work, it wasn’t long before her medical credentials were in demand again.

“While I was still in Auckland, ‘JH’ rang up one day and said that the pathology department (in Dunedin) was asking where I was. So he told them that I’d work part time and that ‘Grandma’ (John’s mother) would look after Helen. I don’t know what I would have done had she not been prepared to do this. It was unusual at the time—society was not geared for working mothers. Indeed, it looked upon them askance.”

Dr Heslop returned as a lecturer but, the following year, she became a senior research officer in the department of surgery, working with the transplantation research group alongside orthopaedics professor Norman Nisbet. “It was the early days of transplantation, and it all felt a bit more intellectually alive,” she wrote. “It was necessary to learn a lot of new stuff, and it was nice to think about Mendelian genetics again.”

During this time Mrs Heslop worked on what would be her major contributions to immunology, notably her research on how to prolong organ graft survival. Eventually she rose to become head of the research group, holding the position until 1990. In that time she published more than 130 research papers and because of her special interest in transplantation and immunology, was often invited to lecture and teach overseas.

In 1972 she was made an associate professor, at that time believed to be the first woman in New Zealand to achieve this status in a department of surgery. More honours were to follow, in fact, there was a veritable flood of them. She was made a Fellow of the Royal Australasian College of Surgeons in 1975, for her contribution to surgical science, and, in March 1984, became a professor of surgery at the Otago Medical School, the first medically-qualified woman to do so.

In 1978 Dr Heslop became the convenor of the Dunedin Basic Medical Sciences Course Trust, following the retirement of Assoc Prof John Borrie. “At its height we were running two courses a year for a total of about 130-odd students, most from Australia,” she wrote. “It made a significant profit—enough to donate $300,000 to the
neurosurgery appeal in 2012, without impairing its capacity to issue its smallish annual scholarships.”

In the summer of 1989–90 there was a “double banger” of awards bestowed upon Dr Heslop, learning, on the same day, she was to be made a Fellow of the Royal Society of New Zealand and also to be awarded a CBE in the New Year’s honours list. Five years later her husband was given the same honour, a “rare double” indeed for the same household.

Not that it was unusual for them to have shared interests. Both she and her husband were also heavily involved with the Cancer Society (both becoming life members in the 1990s). At the time of Dr Heslop’s honour (1997) the Society’s medical director, Dr Peter Dady, described her to the ODT as “a powerful advocate for research who gave the committee some profound insights into the strange Machiavellian workings of New Zealand universities.”

Officially Dr Heslop “retired” in 1989 but, as mentioned earlier, she found retirement a difficult status to achieve, such was the ongoing demand for her services and for access to her formidable intellect and vast reservoir of knowledge.

In August, 2004, she put those qualities into a 15-page paper, published in the New Zealand Medical Journal, entitled All About Research—looking back at the 1987 Cervical Cancer Inquiry in which she examined the controversial work of Dr Herbert Green and the subsequent Cartwright Inquiry.

Her family says she also enjoying writing non-fiction entries for the Manhire Prize and got shortlisted three or four times, her favourite being Eomaia’s Children, a 2008 work about rats, the laboratory animal she worked with. It is an incredibly detailed story, accompanied with a bibliography of 25 references.

On the rare occasions when she wasn't writing, Dr Heslop loved her music—she was an accomplished pianist—cooking, and spoiling her dogs Sam and Henry.

In a more recent development, Dr Heslop now has her own Wikipedia page, which was mentioned in last week’s Guardian newspaper in a story titled “Don’t just use women in science—listen to them too.” Dr Heslop’s family say such publicity, about the under-representation of women scientists in Wikipedia, would have amused her.

When Dr Heslop knew her death was imminent, her attention to detail continued, leaving clear instructions there was to be no funeral, rather a private cremation. But she also wanted her family, old friends and colleagues, to enjoy a get-together in Dunedin last month where the life of a truly remarkable woman was celebrated—and a fine time was had by all.

Dr Heslop is survived by her husband John and daughters Helen and Hilary.

Dave Cannan of the Otago Daily Times (http://www.odt.co.nz) wrote this obituary. We thank them for the reprint permission.

Wikipedia webpage, including link to 2004 NZMJ article: http://en.wikipedia.org/wiki/Barbara_Farnsworth_Heslop
Medical Benevolent Fund

NZMA Members, and families of deceased Members, may apply for aid when in situations of financial hardship or distress.

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington

Tel: 0800 656161
Resilient Health Care

Edited by Erik Hollnagel, Jeffrey Braithwaite and Robert L Wears. Published by Ashgate, August 2013. ISBN 978-1-4094-6978-0. Contains 296 pages. Price £54.00

If you can get through the acronyms and the jargon, this book has some very helpful suggestions for organising health care.

The many different authors from Europe, Denmark, Taiwan, Canada, Australia, the UK and the USA posit that health care is a Complex Adaptive System which is not explained well by the usual logical, linear models.

Adaptability rather than flexibility is the key to resilience with some centralised standardisation so that the part of the system on the front line can proactively deal with the ever-changing complexity.

The book comes from the perspective of making the health care system safer by focusing on what is going right as well as the things that go wrong. It also encourages looking at where things need changing at the management end in addition to the provider end, and looking at the whole system from the patients’ viewpoint in addition to that of the service provider.

The authors argue that adding more accountability, protocols and regulations make service provision less safe, but using technology and systems to empower clinicians to adapt to the unpredictability of clinical situations will produce more effective health care.

Sue Bagshaw
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