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This Issue in the Journal

Fatal injury epidemiology among the New Zealand military forces in the First World War
Nick Wilson, Jennifer A Summers, Michael G Baker, George Thomson, Glyn Harper

We studied injury-related mortality amongst NZ military forces in the First World War. Out of a total of 16,703 deaths occurring during the War (28 July 1914 to 11 November 1918), injury deaths predominated: 65.1% were “killed in action” (KIA), 23.4% “died of wounds” (DOW). During the course of the war, the annual mortality rate from injury (for KIA + DOW) per 10,000 NZEF personnel in the North Hemisphere peaked at 1335 in 1915 (Gallipoli campaign) and then peaked again in 1917 at 937 (largely the Battle of Passchendaele). Participation in the First World War was by far the worst fatal injury event in New Zealand’s history. Many of these injury deaths could be considered to have been preventable through: better diplomacy (to prevent the war), improved military planning to reduce failed campaigns (e.g., Gallipoli, Passchendaele), earlier use of protective equipment such as helmets, and improved healthcare services.

Evaluating the impact of implementing an early warning score system on incidence of in-hospital cardiac arrest
David Drower, Renee McKeany, Pranesh Jogia, Andrew Jull

Patients in hospital can suddenly or progressively deteriorate physiologically. Part of patient care is routine observation and monitoring of the patients physiological condition by assessing vital signs. The introduction of an adult early warning scoring system at Waikato Hospital occurred in 2010. The system includes a visual representation of a patient’s progress captured by a new adult ‘vital signs’ chart. Staff can see at a glance if there has been any deterioration in a patient’s condition, and if there has been, what steps they need to take. The system, called ADDS (adult deterioration detection system), was formally evaluated in both a trial and subsequent hospital wide introduction. A key measure of the impact of the introduced change was to consider what effect ADDS had on the incidence of adult cardiac arrest in the hospital. This paper reports results of a before-after study which showed a reduction in incidence of cardiac arrest following the introduction of ADDS. It is important to realise that the study has limitations. These mainly relate to the quality and accuracy of data captured routinely as part of the hospital cardiac arrest response team process. Every attempt was made to only report in the study that data which was reliable and accurate.
Stereotypes do not always apply: findings from a survey of the health behaviours of mental health consumers compared with the general population in New Zealand
Amanda J Wheeler, Brian McKenna, Dominic Madell

Research suggests that people with mental illness frequently have worse physical health than the general population. Our study investigated the health behaviours of mental health consumers in New Zealand, as we hoped that by clearly describing this group more targeted healthcare could be provided. We surveyed 404 adult mental health consumers about their height and weight, fruit and vegetable intake, exercise activity, smoking, alcohol and drug use. We found that mental health consumers abstained from alcohol more often than those from the general population sample. However, we also found increased obesity, poorer eating habits, less physical activity, and a higher proportion of smokers among mental health consumers. Health professionals should therefore strongly encourage engagement from this group, and should also take care not to stereotype individual mental health consumers, as our study showed variations in health behaviours, as well as alcohol abstinence across a large proportion of the group.

Introduction of sacral neuromodulation for the treatment of faecal incontinence
Sarah Benson-Cooper, Emily Davenport, Ian P Bissett

Faecal incontinence is a debilitating condition that impairs quality of life and causes social isolation. Over the last 15 years a promising new therapy (sacral neuromodulation, SNM) has been introduced which has been associated with marked improvement in many incontinence symptoms. Our early results at Auckland City Hospital have shown a significant improvement in faecal incontinence following sacral neuromodulation, in terms of symptom severity and quality of life. These results are dramatic and comparable to other international centres of excellence. SNM offers a good alternative treatment for patients with severe faecal incontinence who have failed other measures.

Numerical identity: the creation of tri-parental embryos to correct inherited mitochondrial disease
Michael Legge, Ruth Fitzgerald

Mitochondria are responsible for most of the energy production in human cells. There is a range of maternally inherited defects in the function of mitochondria, which can cause serious body organ metabolic dysfunction including heart and liver failure, blindness and premature death in the children of an affected carrier mother. Research in the UK has indicated that these disorders may be corrected by transferring the chromosomes from an affected oocyte to an unrelated donor oocyte that is unaffected thereby ‘correcting’ the disease. This has given rise to the concept of a three-parent child, as it would inherit the parents DNA plus a small amount of DNA contained in the donor oocyte mitochondria. This paper discusses issues related to this new (but as yet unapproved) treatment in the context of the metabolic implications and related Acts in the UK and New Zealand governing reproductive technologies.
The Green Lane and Auckland City Hospital cardiac resynchronisation therapy experience
Andrew Martin, Susan Sinclair, Nigel Lever, Jim Stewart

The Green Lane Cardiovascular Service performs well with respect to CRT complications, though less well when failure of implantation and lead dislodgement are considered. Where complete echocardiographic data was present, 52% of our patients derived significant improvement in cardiac function.
Lest We Forget: the First World War’s terrible toll on New Zealanders

On 4 August 2014, New Zealand along with Britain, our other war-time allies, and our foes of a century ago will commemorate the 100th anniversary of the outbreak of the First World War, so named because by its end some 4 years later it was to have touched many countries around the globe and claimed millions of lives.

The War was to see some 100,444 New Zealanders serve overseas, of whom 16,703 were to lose their lives by the time of the Armistice, with a further 1604 deaths directly attributed to the war once the fighting was concluded.

This edition of the Journal contains an important examination of injury-related mortality among the New Zealand Military forces in the Great War.1 The authors have extensively reviewed complex statistical data—data not necessarily easy to extract nor entirely accurate from one source to the next. Indeed the authors detected and corrected some errors in the War Graves databases, thus ensuring subsequent researchers have a more level playing field.

Whilst the causes of the Great War and its subsequent military and political conduct have been fiercely debated ever since the war began, New Zealand’s willingness to join the fight has never been doubted.

From a nation of just over 1 million in 1914, with a population of approximately 250,000 men of military age, the Government was able to equip and despatch a force to capture German Samoa on 29 August 1914—just 25 days after the declaration of war.2 Furthermore by October the Main Body of the New Zealand Expeditionary Force, some 8000 men and 3000 horses, was ready to sail for the battlefields3. Those officers and men who rushed to serve were all volunteers, and whilst conscription was introduced in 1916, the vast majority of all New Zealanders who fought were in fact volunteers.

Injury and death were to be frequent if not constant companions. Early deaths from illness and injury occurred as the Main Body sailed—for example Lieutenant EJH Webb, a medical student, died of a broken neck sustained in a “crossing the line” ceremony on board a troopship just prior to reaching Ceylon (Sri Lanka).

The first combat death was Private WA Ham of the Canterbury Regiment who died from wounds sustained in defending the Suez Canal from Turkish attack in early February 1915. From “small beginnings”, the Roll of Honour and casualty lists published in the daily newspapers of the Dominion were to grow and touch life in every town and city in the country.

Gallipoli was to see the first great loss of life. Disease and a tough enemy would extract a terrible toll with only a very few avoiding any physical injury. Following Gallipoli, the New Zealand force would divide—mounted troops remaining in Egypt to eventually defeat the Turks in Palestine, and the remainder of the army, reinforced...
by new arrivals from Home forming the New Zealand Division that was to fight with distinction on the Western Front in France and Belgium.

The Western Front saw the greatest casualties for Allied troops in the Great War, New Zealand included. Some 12,000 New Zealanders were lost in the mud and barbed wire of battles that to this day are metaphors for death and the inhumanity of war—the Somme, Ypres and Passchendaele perhaps the most infamous.

As the War developed so too did the methods of inflicting death on the enemy. Machine guns, creeping barrages, poison gas, and tanks all found their place in the battle for victory. In turn the ability of the Medical Corps to treat and rehabilitate the wounded and sick improved dramatically. This is emphasised by the work of Wilson and colleagues. Their research highlights the decrease in the risk of death from combat as the war continued, although this decline was not uniform and did spike again with the slaughter that was Passchendaele.

Interestingly, whilst the absolute risk of dying decreased from 1915 to the end of the war, the risk of dying of wounds did not decrease. As the authors conclude, this is potentially due to casualties being evacuated more rapidly as the war progressed—thus those who would otherwise have died on the field of battle were at least retrieved and offered medical care, but the rise in death by wounds may also have increased due to alterations in weaponry and tactics over the course of the war.

To explore possible actions that could have reduced the deaths of New Zealanders, the authors question the tactical decision-making of Field Marshall Haig, British Commander in Chief, in his conduct of the battle of Passchendaele. This has been a popular if not controversial topic of many writers over the last century, but it would be unfair to demonise Haig as he was ultimately the man who guided the allied forces to victory.4

Terrible though the losses were, the Western Front was always going to be a war of attrition as the Germans were in fortified positions (usually on the high ground) that had to be destroyed if victory was to be obtained.5 That destruction generally had to be completed by the infantry. Less controversially, the authors point to developments in protective equipment such as helmets that could have saved lives as well as the role that improved nutrition could have played, particularly on Gallipoli.

Ultimately the fate of those wounded lay very much in the hands of the stretcher-bearers and the medical staff and facilities to which the wounded were delivered. The authors describe the deficiencies of the medical services during the Gallipoli campaign and rightly conclude that improvements may have reduced the death rate amongst the New Zealanders.

Many lessons were learned from Gallipoli, and the medical history of the Great War is rich in advances that ultimately did save many lives.6 Associate Professor Wilson and colleagues are to be commended for an important piece of research and it is hoped we will see more as we approach the centenary of the Great War for Humanity.

Competing interests: None known.

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References:


“Easy to cure but difficult to detect”—the case for early warning systems in the acute hospital

Ross Freebairn

“…in hectic fever, that in the beginning of the malady it is easy to cure but difficult to detect, but in the course of time, not having been either detected or treated in the beginning, it becomes easy to detect but difficult to cure”

(Nicolo Machiavelli, The Prince Chapter III, Concerning Mixed Principalities)

Warning systems that reveal “easy to cure but difficult to detect” malady hold the promise of improved patient outcomes. Early Warning systems (EWS) were designed specifically to secure the timely presence of skilled clinical expertise at the bedside of patients exhibiting physiological signs compatible with established or impending critical illness. However unless supported by an organised in-hospital responses, with appropriately resourced and trained personnel, who in turn have access to support for those patients requiring an escalation in therapy (such as access to acute operating theatre, intensive care or high dependency services), an early warning system on its own is destined to fail.

Despite enthusiasm for early warning system activation and medical emergency teams, initial attempts to demonstrate their benefits in a scientific way were frustrating. Complex study design using cluster randomised sites, variation in practice between hospitals, case mix and treatments creating the potential of a large noise to signal ratio. Early evidence suggested decreased cardiac arrest rates, without clear evidence of other patient-centred benefit.

Drower et al, in this edition of the Journal, demonstrates a similar result, decreased cardiac arrest rate, this time in a New Zealand setting after the introduction of a EWS.

The criteria used to trigger escalation is of obvious concern, as too low a threshold for medical emergency team response results in increased “cry wolf” responses, which come at increased resource utilisation, while too high threshold defeats the purpose of the “early” warning system. The Waikato groups have utilised well-researched trigger criteria. Previously Jones reported an inverse relationship between call intensity and the reduction in cardiac arrests, suggesting that there may be a dose response to the EWS and subsequent medical emergency team (MET) intervention. Identifying, calibrating and testing the criteria for intervention is essential.

Interestingly the Waikato criteria did not include the subjective “worried carer” criteria that has been popular on many other EWS system. In previous studies the “worried” criterion was the single most common reason (29%) for a MET activation, but if it was the sole criterion for MET activation resulted in a cardiac arrest rate one seventh of that of those that met the objective physiological criteria, although overall outcome were no different.

If reduction in cardiac arrest is the primary aim of the intervention it would appear that objective rather than subjective criteria would be a desirable. The Waikato group experienced a slight, but not sustained rise in the medical emergency team response.
Objective criteria, although not foolproof may allay anxieties of Intensive care staff about inducing “acopia” in the wards, and the fear in ward-based clinicians of loss of clinical autonomy in patient management.

Reduction in cardiac arrest rates following MET and early warning score implementation have been previously demonstrated, although these reductions have not always resulted in a positive effect on mortality or other patient focused outcomes.\(^4,6\) Should we be discouraged if all that is shown initially is a reduction in cardiac arrest rate? Probably not, for at least two good reasons.

Firstly, in a longer term analysis of over 5.9 million hospital admissions and over 73 thousand deaths in Victoria of the effect following a EWS and MET team intervention, Tobin and Santamaria showed that while mortality in the first 2 years after the introduction was not different from the pre-MET period. However, in the longer term, MET team exposure resulted in a reduction in hospital mortality.

It is encouraging therefore that the New Zealand study in this early cohort demonstrated a reduction in cardiac arrest rate so early on, there is the promise that the EWS and MEC response, carefully applied may provide support to acute hospital care. Although not a primary focus, available data suggest a trend towards overall reduction in Waikato’s in-hospital mortality. Whether the enhanced early warning results in overall better patient outcomes after a longer bedding down remains to be seen, but certainly warrants further study.

Secondly, Drower’s study focus was the cardiac arrest rate. In the absence of prospective directive to the contrary, for patients arresting in hospital the default position in New Zealand would be would be the delivery of CPR.\(^7\) One possible reason for the reduction in cardiac arrest rate, as Drower suggests, could be increased diversion of patients from full resuscitation pathway to a palliative-only approach their care, as the Liverpool care pathway was introduced in the same time frame.

Other studies have found high proportion of deaths associated a do not resuscitate order after a MET team suggesting that early identification of the need for palliation is a potentially important role of the MET team. It is possible the Waikato finding was the result of more timely, appropriately conducted end of life discussions, precipitated by a EWS score, with or without a MET response. An earlier detection of deterioration that highlights that a patient options need to be considered and their wishes respected, this would also be a positive outcome even in the absence of a reduced mortality or length of hospital stay.

Many of the innovations aimed at improving patient care and safety that are being advocated by district health boards require multifaceted interventions. It is often difficult to identify which facet of the complex intervention is critical to improvement, and what effect any modification may have. Recent projects have been based upon plausible theorising about the cause and effect of observational findings. For example the shorter stays in emergency departments (ED) health target is based upon the assumption that mortality will be reduced if ED are less crowded, and that this can most cheaply be achieved by reduced length of ED stay.\(^8,9\) However, shorter length of stays themselves are not associated with lower mortality, and simply achieving this surrogate marker of improvement does not guarantee success.\(^10\) A similar situation exists with the introduction of the EWS and the associated medical
response. While “cardiac arrests” events may be reduced, unless this is associated with better patient outcomes, the intervention may not be cost effective, or even beneficial to the average patient.

Rapid response team implementation in other health systems did not improve the severity-of-illness-adjusted outcome of patients transferred from the ward, but did increase ICU admission rates, and early transfer of other patients to the ward, bringing with it the real risk of unforeseen costs without obvious benefit.11 This is a situation we need to avoid.

The Waikato experience is set against the background of a well-established high dependency and intensive care service, in a tertiary hospital.12 Their well-constructed audit, provides comfort that the EWS and the associated response they have developed, seems to be beneficial. For other hospitals the process needs to be carefully thought through, taking into account current practice, case mix and resources.

Most importantly, when similar plans are implemented, careful analysis of the effect on patient care and outcomes over extended periods of time will be needed so we can monitor and tweak the system.

Competing interests: None known.

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Fatal injury epidemiology among the New Zealand military forces in the First World War

Nick Wilson, Jennifer A Summers, Michael G Baker, George Thomson, Glyn Harper

Abstract

Background Despite the large mortality burden of First World War (WW1) on New Zealand (NZ) military forces, no analysis using modern epidemiological methods has ever been conducted. We therefore aimed to study injury-related mortality amongst NZ military forces in WW1.

Methods An electronic version of the Roll-of-Honour for NZ Expeditionary Force (NZEF) personnel was supplemented with further coding and analysed statistically. We also performed literature searches to provide context.

Results Out of a total of 16,703 deaths occurring during the war (28 July 1914 to 11 November 1918), injury deaths predominated: 65.1% were “killed in action” (KIA), 23.4% “died of wounds” (DOW), 1.0% were other injuries (e.g. “accidents”, drownings, suicides and executions), and 10.5% were other causes (mainly disease). During the course of the war, the annual mortality rate from injury (for KIA + DOW) per 10,000 NZEF personnel in the North Hemisphere peaked at 1335 in 1915 (Gallipoli campaign) and then peaked again in 1917 at 937 (largely the Battle of Passchendaele). Some of the offensive campaigns involved very high mortality peaks (e.g. 2 days with over 450 deaths per day in October 1917).

Conclusions Participation in First World War was by far the worst fatal injury event in New Zealand’s history. Many of these injury deaths could be considered to have been preventable through: better diplomacy (to prevent the war), improved military planning to reduce failed campaigns (e.g. Gallipoli, Passchendaele), earlier use of protective equipment such as helmets, and improved healthcare services.

The literature on injury epidemiology associated with warfare is largely focused on particular injury types, and on wars since Vietnam (e.g.1,2). Indeed, there is relatively little modern literature on injury epidemiology for the First World War, and even that is largely about specific injuries (e.g.3,4). In particular, we could not identify any analytical epidemiological studies of the fatal injury burden for a nation from WW1. This gap also applies to New Zealand military forces, even though this nation had a proportionately high mortality burden from this war.

Interest in this war is likely to grow in New Zealand and in other countries involved with the forthcoming centenary, and so it is of relevance to critically consider the injury epidemiology of this event. Therefore, we aimed to briefly study injury-related mortality amongst New Zealand military forces in WW1 and consider some issues around potential preventability. Mortality was chosen as the most important and measurable indicator of the wider injury burden associated with WW1.
Methods

An electronic version of the Roll-of-Honour for New Zealand Expeditionary Force (NZEF) personnel in WW1 was obtained courtesy of the compiler, Professor Peter Dennis (University of New South Wales at the Australian Defence Force Academy).

Further work on this dataset included duplicate removal and coding for ethnicity. For the latter, personnel were classified as having Māori ethnicity if any of the following applied: having a Māori language name (first, second or surname); likewise for having a parent with a Māori language name; being buried in a Māori cemetery or having a memorial in such a cemetery; or having a iwi (tribal) affiliation listed in a biographical database (“Cenotaph database”) covering NZEF personnel and held by the Auckland War Memorial Museum® (from whom we purchased a dataset).

Personnel were classified as having Pacific peoples ethnicity if they came from a South Pacific island providing military personnel for the NZEF (i.e. Fiji, Gilbert and Ellis Islands, Niue, Samoa, Tonga and the Cook Islands) and had any of the following: a Pacific name; a parent with a Pacific name; or they came from a named village. The approach of using the language of a name for considering ethnicity has been used elsewhere in historical work in New Zealand. It is also used for identifying from the electoral roll potential Pacific people respondents to public health surveys in New Zealand (the LEXICON method used by Massey University) and we used it in a previous study on pandemic influenza.

For the denominator data we extracted a random sample of 1000 individuals (about 1%) of personnel who served in the NZEF as detailed in the Cenotaph database. This denominator sample was then adjusted further to replace (with additional random selection).

Ethnicity coding was then performed as for the numerator data. A validation study was performed for the method of ethnicity coding for Māori. It involved one of us (GT) with local history expertise who independently classified the ethnicity of WW1 participants in a rural area in which he had performed historical research.

The results indicated that the coding system we have used was under-ascertaining Māori ethnicity (a sensitivity of 73% (i.e. n=11/15). Of note however, is that the rural locality used in this validation study had a relatively high Māori population in the pre-WW1 era and intermarriage between Māori and New Zealand European was relatively common. As such the under-ascertainment found would be a worse case assessment if applied to New Zealand in general. In contrast all of those classified as Māori via our coding system were also classified as Māori in this validity study (specificity of 100%, n=17/17).

We also updated the Roll-of-Honour dataset with cause of death information including deaths relating to executions and suicides. The latter was via a search in March 2013 of the online Cenotaph database given new information added to this from on-going archival research by others on this topic.

From the final dataset we extracted information on the date, cause and place of death, and the unit, rank and ethnicity of the personnel. To add to the spatial dimension of the mortality, we also extracted the site of the cemetery or memorial, since for some NZEF personnel the site of actual death was different (e.g. they died from wounds when being transported from the battlefield to hospital facilities in another country e.g. the United Kingdom or Egypt).

We also performed literature searches to provide context, especially around aspects of the injury burden that could have been prevented at the time. In particular, we searched the literature for the contemporary diplomatic, military planning, equipment, shelter and health service means that could have reduced the fatal injury burden at the time.

Results

Out of a total of 16,703 deaths among NZEF personnel during the war (28 July 1914 to 11 November 1918), injury deaths predominated (Table 1). Being killed in action (65.1% of all deaths) exceeded dying of wounds (23.4% of all deaths) (Table 1). Injury deaths occurred in 14.9% of the military personnel who had embarked for overseas service (14,946/100,444).
Table 1. Causes of death among NZEF personnel during the First World War (both Northern and Southern Hemispheres 1914–1918)

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Year of First World War</th>
<th>1914*</th>
<th>1915</th>
<th>1916</th>
<th>1917</th>
<th>1918*</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Injuries</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Killed in action (KIA)</td>
<td>0</td>
<td>1920</td>
<td>2203</td>
<td>3831</td>
<td>2916</td>
<td>10,870</td>
<td>65.1</td>
</tr>
<tr>
<td>Died of wounds (DOW)</td>
<td>0</td>
<td>594</td>
<td>814</td>
<td>1316</td>
<td>1179</td>
<td>3903</td>
<td>23.4</td>
</tr>
<tr>
<td>“Accident”</td>
<td>1</td>
<td>4</td>
<td>28</td>
<td>41</td>
<td>35</td>
<td>109</td>
<td>0.7</td>
</tr>
<tr>
<td>Drowned</td>
<td>0</td>
<td>36</td>
<td>6</td>
<td>5</td>
<td>10</td>
<td>57</td>
<td>0.3</td>
</tr>
<tr>
<td>Suicide**</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>4</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Executed</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>5</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Subtotal (injuries)</td>
<td>1</td>
<td>2555</td>
<td>3055</td>
<td>5196</td>
<td>4141</td>
<td>14,948</td>
<td>89.5</td>
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<tr>
<td>Other causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Died of disease (DOD)</td>
<td>9</td>
<td>283</td>
<td>171</td>
<td>250</td>
<td>584</td>
<td>1297</td>
<td>7.8</td>
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<tr>
<td>Other**</td>
<td>2</td>
<td>48</td>
<td>133</td>
<td>101</td>
<td>174</td>
<td>458</td>
<td>2.7</td>
</tr>
<tr>
<td>Total deaths</td>
<td>12</td>
<td>2886</td>
<td>3359</td>
<td>5547</td>
<td>4899</td>
<td>16,703</td>
<td>100</td>
</tr>
<tr>
<td>%</td>
<td>0.1%</td>
<td>17.3%</td>
<td>20.1%</td>
<td>33.2%</td>
<td>29.3%</td>
<td>100%</td>
<td></td>
</tr>
</tbody>
</table>

* For 1914, starting from 28 July; for 1918 ending on Armistice day (11 November). Only 4 of the injury deaths occurred in NZ; 2 amongst those who had never embarked for service overseas.

** A genealogical website suggests an additional 3 suicides among the NZEF for this 1914–1918 period (“Miscellaneous New Zealand War Deaths 1914–1923”). But such details were not available on the primary databases we used for this study (2 of these were listed as “Section 2” in the Roll-of-Honour and one name was not included).

*** “Other” mainly includes “Section 3” (n=266); “Section 2” (n=151) and unknown causes. “Section 2” was those “who died after discharge from the NZEF from wounds inflicted or disease contracted while on active service.” “Section 3” were “those who died from accident occurring or disease contracted, while training or attached to the NZEF in NZ.” While many of the Section 2 and 3 deaths appear to have been from disease, it is possible that some of these were also injury deaths (e.g. from the long-term effects of wounds).

Demographic characteristics—There were 179 injury deaths among those classified as being Māori (1.2% of the total) and seven such deaths among Pacific soldiers. The cumulative injury mortality risk was highest for European/Other (1514 per 10,000 population); then Māori (1103 per 10,000); and then Pacific soldiers (101 per 10,000). Ten of the injury deaths (0.1% of the total) were women. These were all nurses who drowned when the ship they were travelling in was torpedoed (HM Transport SS Marquette, on 23 October 1915; and see also details in this NZMJ piece on the memorial chapel in Christchurch: [http://journal.nzma.org.nz/journal/119-1244/2292/]).

The date of birth, or age at death, have not been routinely recorded in the Roll-of-Honour or Cenotaph database we examined. Nevertheless, as part of other work (a study of an outbreak of pandemic influenza on a troopship in 1918) we found that the mean age of the NZEF personnel on-board was 26.7 years.

In terms of military rank and role, injury deaths were distributed as follows: officers (n=686, 4.6% of the deaths), non-commissioned officers (n=2686, 18.0%), health care workers in the New Zealand Medical Corps and the New Zealand Army Nursing
Service (n=146, 1.0%), and all other ranks (n=11,428, 76.5%). Denominator data are not available to calculate mortality risk by these ranks.

Temporal patterns—During the course of the war the annual risk of injury death (for KIA + DOW in the Northern Hemisphere) declined after peaking at 1335 per 10,000 in 1915 (Gallipoli campaign) and then peaked again in 1917 at 937 (largely the Battle of Passchendaele) [see Figure 1]. However, the proportion of DOW deaths out of all injury deaths did not decline over time and peaked in 1918 at 28.8%.

Figure 1. Annual risk of injury mortality for New Zealand military personnel in the First World War in the Northern Hemisphere (with only 1 death in 1914)

The injury deaths varied by season with a relatively small proportion of deaths in the Northern Hemisphere winter (range: 0.0% to 5.4% of all the deaths). The peaks in numbers of deaths by season were autumn 1916 (n=2313, 15.5%, Somme offensive) and autumn 1917 (n=2317, 15.5%, Battle of Passchendaele).

The seasonal variation is also apparent in Figure 2 which shows the injury deaths by month and year. Also apparent are that the peaks in numbers of deaths generally related to Allied offensives (an exception being the March 1918 offensive by Germany).
Figure 2. Monthly pattern of injury mortality for New Zealand military personnel in the First World War in the Northern Hemisphere (1915 to 1918)*

* Peaks in 1915 reflect the Gallipoli campaign (Allied offensive). The large peak in 1916 reflects the Somme offensive in France. The first peak in 1917 reflects the Battle of Messines, Belgium (an Allied offensive); the second, the Battle of Passchendaele (Third Battle of Ypres), Belgium (another Allied offensive, see Figure 3). In March/April 1918 there was a response to a major German offensive in Western France and from July onwards there were multiple Allied advances on the Western Front.

Some of the offensive campaigns involved very high mortality peaks over just a matter of days. Indeed, for the worst month of the war for the NZEF (October 1917), two extreme mortality peaks are shown in Figure 3. In this month the New Zealand forces took part in two major actions at Passchendaele, in Belgium.

Figure 3. Daily pattern of injury mortality for New Zealand military personnel in the month of the First World War with the highest number of NZEF deaths (October 1917, Battle of Passchendaele, Belgium)*

* For injury deaths in France, Belgium, and the United Kingdom (and excluding 5 deaths from accidents and 9 deaths in Palestine that month).
The first peak in Figure 3 (for 4 October) reflects a relatively successful military action—but still with major loss of life. The second peak (12 October) reflected a failed offensive due to heavy rain and the inability of the artillery to destroy barbed wire and German defensive bunkers (pillboxes) that housed machine guns.\(^{12}\) (p51,90) Figure 3 also shows that in subsequent days the proportion of deaths that were from wounds (relative to KIA), were relatively high compared to on the two days of the offensives.

### Table 2. Spatial distribution of injury mortality for NZEF personnel in the First World War as reflected by the locations of cemeteries and memorials*  

<table>
<thead>
<tr>
<th>Region/Country**</th>
<th>Cemeteries/ memorials (N)</th>
<th>Individuals named (N)</th>
<th>Proportion of all names (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Europe</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>France</td>
<td>242</td>
<td>7207</td>
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</tr>
<tr>
<td>Belgium</td>
<td>111</td>
<td>4588</td>
<td>30.7</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>56</td>
<td>193</td>
<td>1.3</td>
</tr>
<tr>
<td>Greece</td>
<td>8</td>
<td>52</td>
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</tr>
<tr>
<td>Malta</td>
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<td>28</td>
<td>0.2</td>
</tr>
<tr>
<td>Gibraltar</td>
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<td>6</td>
<td>0.0</td>
</tr>
<tr>
<td>Ireland</td>
<td>4</td>
<td>5</td>
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</tr>
<tr>
<td>Germany</td>
<td>2</td>
<td>3</td>
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</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>432</strong></td>
<td><strong>12,082</strong></td>
<td><strong>80.8</strong></td>
</tr>
<tr>
<td><strong>Middle East</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turkey</td>
<td>84</td>
<td>2303</td>
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<td>278</td>
<td>1.9</td>
</tr>
<tr>
<td>Palestine/Israel</td>
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<td>192</td>
<td>1.3</td>
</tr>
<tr>
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<tr>
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<tr>
<td>Iraq</td>
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<td>1</td>
<td>0.0</td>
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<tr>
<td><strong>Subtotal</strong></td>
<td><strong>109</strong></td>
<td><strong>2821</strong></td>
<td><strong>18.9</strong></td>
</tr>
<tr>
<td><strong>South Pacific/Other</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>NZ – memorials***</td>
<td>5</td>
<td>20</td>
<td>0.1</td>
</tr>
<tr>
<td>NZ – cemeteries***</td>
<td>12</td>
<td>20</td>
<td>0.1</td>
</tr>
<tr>
<td>Sri Lanka</td>
<td>2</td>
<td>2</td>
<td>0.0</td>
</tr>
<tr>
<td>Samoa</td>
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<td>1</td>
<td>0.0</td>
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<tr>
<td>Tahiti</td>
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<td>1</td>
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</tr>
<tr>
<td>Not known</td>
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<td>1</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>22</strong></td>
<td><strong>45</strong></td>
<td><strong>0.3</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>563</strong></td>
<td><strong>14,948</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>

* Some of these cemeteries and memorials include names of those whose bodies were never found, or who died at sea or in transit (e.g. on a hospital ship taking the wounded away from a conflict zone); ** As per country boundaries in 2013 and not distinguishing between contested boundaries in Palestine/Israel; *** Only 4 of these injury deaths recorded at New Zealand (NZ) memorials and cemeteries actually occurred in New Zealand (3 suicides and 1 drowning). Most (n=18) died of wounds while at sea (mainly on route to New Zealand).
Spatial patterns—The location of injury deaths is shown in Table 2, based on the location of cemeteries and memorials.

At the regional level, most of these deaths were in Europe (80.8%) and particularly France (48.2% of the total) and Belgium (30.7%). The next major region was the Middle East (18.9%), particularly Turkey (15.4%). Some of these cemeteries and memorials include names of those whose bodies were never found, or who died at sea or in transit (e.g. on a hospital ship taking the wounded away from a conflict zone).

Discussion

Main findings and interpretation—The fatal injury burden from WW1 for New Zealand was extremely high and clearly exceeded that for New Zealand from World War II (which was 11,625 deaths from all causes\textsuperscript{13}). These WW1-related deaths need to be put in the context of the population of 1,149,225 of New Zealand in the 1916 census,\textsuperscript{14} (i.e. 1.5% of the total population). The burden was also much greater than that from the largest natural disaster in New Zealand’s history, the 1918 influenza pandemic (estimated at 8573 deaths\textsuperscript{15}). Similarly, for the earthquake causing the highest mortality burden, the 1931 Hawke’s Bay earthquake with 258 deaths.\textsuperscript{16}

Another striking aspect of the injury burden was how far it exceeded deaths from disease in this “industrial war” (Table 1). In preceding wars such as the American Civil War and the South African War (Second Boer War), most deaths among soldiers were from disease (e.g. two-thirds for British troops in the South African War, particularly from typhoid).\textsuperscript{17}

As shown in Figure 1, the proportion of “died of wounds” (DOW) deaths out of all injury deaths did not decline as the war progressed. This finding suggests that the improvements in medical services for the wounded were probably outweighed by other factors. These could have included the ongoing changes in weaponry such as the greater number and more effective use of artillery as the war progressed, but also changes in military tactics, both defensive and offensive.

The effective use of artillery is particularly relevant as it has been calculated that artillery caused 58% of the war’s military fatalities.\textsuperscript{18} (p184) It is also plausible that improvements in evacuation also contributed to this pattern. That is rather than the wounded dying in the field and being classified as “KIA”, better evacuation processes later in the war\textsuperscript{19} may have resulted in more wounded getting carried to a field station, where they subsequently still died and were classified as “DOW” instead of “KIA”.

The other temporal pattern of note is how the largest peaks in injury mortality corresponded to Allied offensive actions (versus the defensive response to the March 1918 German offensive—see Figure 2). Offensive action in settings where artillery had not effectively removed German machine gun posts was particularly devastating (e.g. 12 October 1917, Battle of Passchendaele, Figure 3).

The New Zealand Division’s role on the Western Front, as with the British Expeditionary Force in general, was primarily an offensive one. The New Zealand Division experienced just one defensive battle on the Western Front: the German offensive of 21 March to 5 April 1918.
The cumulative injury mortality proportions for WW1 were highest in the European/Other ethnic group. This probably reflects the differing roles of Māori and Pacific soldiers in the NZEF, with these personnel generally having more involvement working in non-combat units such as a Pioneer Battalion. Most casualties occurred in infantry units, followed by artillery. But the injury mortality contrasts with the pattern for disease deaths where Māori and Pacific soldiers had higher cumulative mortality burdens, and indeed a higher all-cause mortality burden for Māori than for the European/Other ethnic group.

**Study limitations**—We found (and corrected) minor errors with the Roll-of-Honour dataset and indeed it is likely that various other minor errors still exist. For example, an online dataset which is regularly updated by a New Zealand genealogist has an additional four deaths included (i.e. n=18,311 vs n=18,307 in the dataset we used, for the 1914–1923 period). Furthermore, some injury deaths might still be misclassified as “killed in action” or “accident” when these were actually suicides.

Indeed, by restricting our analysis to the date of the end of the war (11 November 1918), we have excluded some unknown proportion of the 1604 deaths in the subsequent period (to the end of 1923 in the Roll-of-Honour), that could have been due to injuries.

The deaths in this period are generally not well classified as to the cause, given that many occurred in personnel discharged from the military. Many are likely to have died from one or more waves of the 1918/19 influenza pandemic, and other diseases and “accidents”. Long-term mental health burdens associated with the war, may also have increased intentional injury risk via suicide in subsequent decades, given the evidence for mental health sequelae amongst veterans, including New Zealand veterans.

The lack of collated denominator data for the entire NZEF has limited our ability (in this unfunded study), to determine the risk of injury death by age-group and by other variables of interest such as occupational class and military rank. But such data could be extracted from the archival records in future analyses. Consideration could also be given to estimating the injury impact on New Zealand citizens who enlisted in other armies (especially that of the United Kingdom and Australia).

**Aspects of preventability**—While we acknowledge it is somewhat speculative to retrospectively consider issues around preventability for historical events, the available literature does suggest these to varying extents. Primarily, it appears very plausible that better diplomacy and communication could have prevented the war entirely. Also, once the war was underway, it also appears that diplomatic action failed to prevent or constrain the further use of chemical weapons, once the existing international law on chemical weapons use (the Hague Convention of 1899) had been initially violated by Germany.

Improved military planning might have also helped to reduce the fatal injury burden, by for example avoiding the failed Gallipoli Campaign, in which the NZEF suffered severely (most of the deaths in 1915 and specifically estimated at 2779 deaths, which is slightly more than an official figure of 2721).

Similarly, more thoughtful military decisions by the high command on the Western Front could have avoided the Battle of Passchendaele or at least halted military
operations when weather and terrain conditions meant there was little prospect of success. If Field Marshal Sir Douglas Haig had called off this battle on the afternoon of 4 October 1917 when the spell of reasonable weather ended, many lives, including New Zealanders, would have been spared.

Haig’s decision to continue the offensive after this point, against the advice of his army commanders, was the most controversial of his career. The influential military theorist and historian Major General JFC Fuller believed that Haig’s decision to continue with a “tactically impossible battle … was an inexcusable piece of pig-headedness on the part of Haig”. Unfortunately too many soldiers paid the price for this “pig-headedness”.

Poor planning was also associated with more specific events. For example, the sinking of the Marquette was potentially avoidable since “a marked hospital ship, by definition safe from attack, had left the same port on the same day as the Marquette, completely empty. By putting the medical staff in an unmarked transport in a convoy carrying troops and ammunition, the authorities unnecessarily risked their lives.”

The deaths of 10 NZEF personnel in a railway accident in the UK also had preventable elements.

Improved protective equipment such as the use of steel helmets by New Zealand troops earlier in the war would probably have helped reduce head wounds and associated fatalities. Photographs of soldiers in Gallipoli in 1915 show a variety of soft hats in use and steel helmets did not appear in use until 1916 (frontline NZ troops in Western Europe—using the British-designed “Brodie helmet”, see Figure 4). In contrast, the steel “Adrian helmet” started to be used by the French army at an earlier stage, in the summer of 1915. Helmet design could also have been better since the German helmet (the “stahlhelm”) was stronger than the Brodie helmet and gave better protection to the back of the head and neck (though it was also more expensive and heavier). The American version of the Brodie helmet was also stronger than the British version.

Tighter restrictions on the provision of alcohol in military rations may also have reduced a range of fatal injuries. Views on the benefits and hazards of alcohol rations for allied soldiers varied and the amounts provided were often small. Nevertheless, for the NZEF at Gallipoli the daily alcohol ration was “½ gill rum at discretion of G.O.C. on recommendation of S.M.O”. This is equivalent to 71 ml of rum or around 2.2 standard drinks (assuming rum at 40% alcohol by volume and using the official NZ formula).

Improved nutrition of NZEF personnel may also have reduced injury deaths. For example, nutrient deficiency diseases such as scurvy (e.g. at Gallipoli) may have increased the risk of death once wounded. It is also plausible that some personnel at Gallipoli may have had night blindness from low vitamin A intake (which may have increased the risk of injury when fighting at night).
Better disease prevention (to prevent dysentery, typhoid, and malaria among NZEF personnel\textsuperscript{29}) may also have helped avoid the situation of weaken troops who might have succumbed more readily to death from wounds.

Finally, improved design and resourcing of military medical services is likely to have helped reduce deaths from injury.\textsuperscript{38–40} Examples of deficits included the lack of enough medical supplies, health workers and hospital ships in the Gallipoli Campaign.\textsuperscript{39,40} The lack of adequate medical facilities was one of a number of deficiencies outlined in the final report of the Dardenelles Commission although its censures were “polite and vague, rather like the orders for the Suvla landing”.\textsuperscript{41}

New Zealand conducted its own investigation into “various matters” concerning the NZEF in September-October 1915. The investigating officer, Lieutenant Colonel the Hon R Heaton Rhodes, reported that during the April landing and the August offensive “congestion and consequent delays in getting the sick and wounded away” had been a serious problem. He sought assurances that such delays would not occur again.\textsuperscript{42} The use of hospital facilities in Egypt for the wounded in this campaign were also criticised as being a poor choice (e.g. due to fly problems) relative to using hospitals in Malta or England.\textsuperscript{43}
There were sometimes shortages of stretcher-bearers and initially less fit men were selected for this work—until this was subsequently changed to the fitter and stronger men.\textsuperscript{19} The initial use of horse-drawn ambulances (rather than motorised ones) on the Western Front\textsuperscript{38} was also problematic. Indeed, use of motorised ambulances (Figure 4), well-trained stretcher bearers, and improvements in fracture/wound management by medical services have been described as factors reducing the case fatality level among the wounded in later stages of the war.\textsuperscript{19}

Paradoxically though, improvements in medical treatment may have helped prolonged the war. As David Stevenson has noted:

\begin{quote}
Still more remarkable was medicine’s success in rehabilitating the wounded: and this more than anything else accounted for the armies’ ability to keep fighting despite seemingly prohibitive casualty rolls. Most lists compiled during the war lumped together dead and wounded without indicating that only a minority of the latter were unable to ever serve again.\textsuperscript{18} (p207-8)
\end{quote}

**Conclusions**

The First World War was by far the worst fatal injury event in New Zealand’s history, exceeding all other wars and natural disasters by a large margin. Many of these injury deaths could be considered to have been preventable at the time through better diplomacy, improved military planning, use of protective equipment and better medical services.

**Competing interests:** None known.

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Evaluating the impact of implementing an early warning score system on incidence of in-hospital cardiac arrest

David Drower, Renee McKeany, Pranesh Jogia, Andrew Jull

Abstract

Aim To evaluate the introduction of an early warning score (EWS) system on incidence of in-hospital adult cardiac arrest.

Methods A before-after evaluation of an EWS system (in the form of a patient observation chart with escalation protocol) in a 600 bed tertiary teaching hospital in New Zealand during the two 12-month periods between March 2009 and March 2011. Difference in incidence rates was compared using Student’s t test.

Results There were 168 cardiac arrests during the 24 month period. The incidence rate of cardiac arrests per 1000 admissions was 4.67 during 2009–2010 and 2.91 during 2010–2011 (mean difference of 1.77, 95%CI 0.59–2.94). The number of cardiac arrests dropped from an average of 8.5 arrests per month during 2009–2010 to 5.5 arrests per month during 2010–2011 following the introduction of ADDS (mean difference 3.0, 95%CI 0.78–5.22). There was no significant increase in the number of medical emergency calls (7.5 calls versus 9.1 calls per month).

Conclusion Introduction of an EWS system in addition to an existing cardiac arrest team response decreased the incidence of in-hospital cardiac arrests in a tertiary hospital in New Zealand.

Unintended harm can occur to hospitalised patients from unrecognised physiological deterioration. Cardiac arrest may result if the deterioration is not detected early and is unable to be reversed.1,2 Previous studies have confirmed that hospitalised ward patients exhibit signs of physiological instability prior to experiencing a cardiac arrest.3-10

Use of early warning score (EWS) systems have been advocated in order to support early detection of the physiological deterioration often seen prior to cardiac arrest in hospital settings.11 Based on key vital sign parameters, the original EWS system was designed to aid early detection of developing critical illness by using weighted scoring of physiological variables.12

This score is utilised to guide staff action based on an associated graded response algorithm. EWS systems depend heavily on staff ability to undertake timely, and appropriate, assessment and monitoring of patient vital signs (and other key pertinent physiological values), understand the significance of any changes, and follow agreed escalation protocols. However, evidence to support use of an EWS system to reduce the incidence of cardiac arrest is limited. Despite this limitation, a landmark investigation in a large New Zealand (NZ) urban hospital in 2004 prompted a Health and Disability Commissioner (HDC) inquiry,13 resulting in a recommendation that district health boards (DHBs) implement EWS systems.14
The implementation of a EWS system at Waikato DHB presented an opportunity to evaluate the impact of introducing an EWS system to the incidence of in-hospital cardiac arrest.

**Method**

Waikato Hospital is a 600-bed hospital that has a cardiac arrest team that responds to all arrest calls, activated through telecommunication system operators. The team usually comprises an intensive care unit (ICU) registrar, a senior ICU nurse, a senior cardiac care unit nurse, and various others depending on the time of day or night. After-hours, a senior clinical resource nurse also attends all arrest calls. The evaluation study covered a 24 month period between the 1st April 2009 and the 31st March 2011, comprising a before and after retrospective assessment. The first 12 months constituted data gathering for in-hospital cardiac arrest at Waikato Hospital and also design and development of the EWS tool appropriate to the Hospital environment. The subsequent 12 month study period included ongoing data collection with introduction of the EWS, firstly to a number of target clinical areas and then during subsequent wider rollout across the hospital. Staff education on use of the new tool occurred throughout.

The EWS system and associated escalation protocol was incorporated into a standardised observation chart, known as the Adult Deterioration Detection System (ADDS). The ADDS chart included criteria specifically designed to activate the cardiac arrest team. An ADDS chart template was made available by the developers from the University of Queensland.\(^\text{15}\) The original ADDS chart was developed utilising human factor design principles and tested in experimental laboratory studies to elicit an evidenced based observation chart and EWS tool.\(^\text{16-18}\)

Local modification of the ADDS chart and process involved key senior medical and nursing staff from various specialities including critical care, internal medicine, anaesthesia, and surgery. Modifications to the original template primarily consisted of changing chart orientation, modification of the parameters that attracted a score (Figure 1), and localisation of the escalation protocol that outlined various actions to be taken by staff, depending on the total ADDS score.

**Figure 1. ADDS scoring tool**

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<tr>
<th>Level of consciousness</th>
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<th>2</th>
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<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>99777*</th>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>99777*</td>
</tr>
<tr>
<td>Responds to Pain</td>
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</table>

<table>
<thead>
<tr>
<th>Respiratory Rate (breaths/min)</th>
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<th>6 - 8</th>
<th>9 - 11</th>
<th>12 - 20</th>
<th>21 - 24</th>
<th>25 -35</th>
<th>30 - 39</th>
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<tr>
<td>O₂ Flow Rate (L/min)</td>
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<td></td>
<td></td>
<td></td>
<td>≥ 2 or on room ar.</td>
<td>3 - 6</td>
<td>&gt; 6</td>
<td></td>
</tr>
<tr>
<td>O₂ Saturation (%)</td>
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<td>90 - 92</td>
<td>93</td>
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<tr>
<td>Heart Rate (beats/min)</td>
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<td>40 - 49</td>
<td>50 - 109</td>
<td>110 - 119</td>
<td>120 - 129</td>
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<td>Systolic BP (mmHg)</td>
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<td>90 - 99</td>
<td>100 - 179</td>
<td>160 - 199</td>
<td>≥ 200</td>
<td></td>
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<tr>
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<td>36 - 37,9</td>
<td>38 - 38,9</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>4 Hour Urine output (ml)</td>
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<td>110 - 159</td>
<td>160 - 300</td>
<td>&gt; 800</td>
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</tbody>
</table>
The primary outcome of interest was the incidence of in-patient adult cardiac arrest, as captured by the hospital Resuscitation Audit form. This form is expected to be completed by attending staff at an arrest to which the cardiac arrest team has been called. Cardiac arrest was defined as an arrest that comprised either a cardiac or a respiratory arrest in an adult patient $\geq 16$ years old, and occurred first in the hospital.

Cardiac arrest was seen to occur if the patient received chest compressions (CPR), had a documented primary arrest rhythm of ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), pulseless electrical activity (PEA), or asystole (cessation of electrical activity of the heart). Respiratory arrest was defined as apnoea (absence of spontaneous respiration) with a palpable pulse.1 By default, an arrest that did not fit this definition was considered to be a medical emergency.

Hospital cardiac arrest data was accessed from the New Zealand Resuscitation Council National CPR registry. The registry provided details of patient national health identifier numbers, dates and general location of cardiac arrests on the hospital campus. This information was then correlated with the arrest call data sheets and pager logs available via the hospital telecommunication system operators. The number of monthly adult patient hospital admissions, via the emergency department (ED), during the study period was accessed from hospital administration staff. All cardiac arrests that occurred in the ED, ICU or operating theatres were excluded.

Given the evaluation study primarily involved audit, ethics approval was waived by the Northern Y Ethics Committee. However, locality approval was obtained for access to clinical and hospital data. Data analysis was conducted using Microsoft® Excel. Rates and counts for the two periods were tested for normality. Unpaired two sample Student’s $t$ tests were used to compare incidence rates and count data. Results were considered significant if the $p$ value was less than 0.05. Control charts were produced using Minitab version 16 software.

**Results**

There were 621 arrest calls made via the hospital telecommunication system over the 24 month study period, 324 during 2009-2010 and 297 during 2010-2011. From the CPR registry 168 (27%) were cardiac arrest responses, as defined in this study. The remainder of calls fell into two categories. The first (199, 32%) were deemed medical emergency calls, and the second (254, 41%) were calls for which there was no audit form completed or the form contained insufficient information (Table 1).

About 93%(156) of the 168 cardiac arrest responses were due to potentially lethal dysrhythmias or pulseless electrical activity (PEA). The frequency observed were as follows: PEA 37.8% (59), asystole 26.2% (41), pulseless ventricular tachycardia 20.5% (32), and ventricular fibrillation 15.3% (24). The remaining cardiac arrests were respiratory arrests or no known arrest rhythm yet undergoing CPR.

There were a total of 44,184 adult hospital admissions, 21,806 during 2009–2010 and 22,378 during 20102011.
Table 1. Arrest calls by category and rate of cardiac arrests

<table>
<thead>
<tr>
<th>Month</th>
<th>Arrest calls</th>
<th>CPR registry</th>
<th>Cardiac arrest</th>
<th>ME</th>
<th>No information</th>
<th>Admissions</th>
<th>Rate of arrests per 1000 admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apr-09</td>
<td>24</td>
<td>14</td>
<td>4</td>
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<td>4</td>
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<td>8</td>
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<td>6</td>
<td>11</td>
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<td>7</td>
<td>7</td>
<td>1918</td>
<td>4.17</td>
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<td>6</td>
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<td>2</td>
<td>1957</td>
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<td>6</td>
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<td>1901</td>
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<td>8</td>
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<td>5</td>
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<td>4</td>
<td>13</td>
<td>7</td>
<td>1904</td>
<td>2.10</td>
</tr>
</tbody>
</table>

ME = medical emergency.

The rate of cardiac arrest responses captured by the CPR registry over the entire period was 3.79 per 1000 admissions. The rate of cardiac arrests per 1000 admissions was 4.67 during 2009–2010 and 2.91 during 2010-2011 (Figure 2). The mean difference during the two periods was 1.77 (95%CI 0.59–2.95, p=0.005), equating to a 38% reduction in cardiac arrest responses captured by the CPR registry between the two periods.

Following introduction of ADDS in March/April 2010 the number of cardiac arrest responses dropped from an average of 8.5 arrests per month during 2009–2010, to 5.5 arrests per month during 2010–2011 (mean difference 3.0, 95%CI 0.78–5.22).

The number of medical emergency calls increased from 90 during 2009–2010 to 109 during 2010–2011, an increase from an average 7.5 medical emergencies per month during 2009–2010 to 9.1 per month during 2010–2011. The increase was not statistically significant, and these results did not alter when calls with no information were included as medical emergency calls.
Discussion

Our study is the only study to have attempted to identify the effect of introducing an EWS system over an existing cardiac arrest team response, and to examine the effect on the rate of actual cardiac arrests as a component of total cardiac arrest calls.

Only about one-third of all cardiac arrest calls constituted an actual cardiac arrest, with the remaining calls deemed to be of a medical emergency nature. The large amount of missing information demands caution in regards to any further interpretation.

Introducing an EWS system in the form of the ADDS chart appears to have reduced the number of cardiac arrest responses captured by the CPR registry during the implementation period, without significantly increasing the number of medical emergency calls.

District health boards have adopted variants of EWS systems, driven largely by recommendations from the HDC mentioned above, but the evidence supporting their use has been limited. While the effect of improvements in hospital response to patient clinical deterioration has been investigated, the effect of system improvement in identification and activation of hospital response to patient clinical deterioration, in order to reduce incidence of cardiac arrest, has not been clearly established. Thus a Cochrane review of critical care outreach and EWS systems called for use of EWS systems in the practice setting to be evaluated.

Two other studies have reported a reduction in the incidence of in-hospital cardiac arrest as a result of introducing an EWS system. The first study considered data on in-hospital arrest during a 5-month period when the EWS was being piloted on a general surgical ward, by comparing the numbers of arrests on the pilot ward with two other surgical wards. They found a threefold reduction in incidence of cardiac arrest on the pilot ward (four compared with 12 and 13 arrests in the comparison wards), a result attributed to the introduction of the EWS. However, the study was conducted over a short period of time and there was no information on rates of cardiac arrest,
which would allow for differences in volume. Furthermore, it is not clear whether the difference represents an improvement in the pilot ward, or continuity of a baseline low incidence.

The second study presented the results of a retrospective audit of prospectively gathered data over two 4-year periods from 2002 to 2009. The second period followed the introduction of a Modified EWS with a new vital sign chart, and a critical care outreach team covering 24 hours per day for seven days of the week. These innovations significantly reduced the number of cardiac arrest calls relative to adult hospital admissions, the cardiac arrest calls per hospital death and the number of patients who received CPR before admission to intensive care. However, isolating the effect of the changes on incidence of cardiac arrest is uncertain, as it was not clear how many of the cardiac arrest calls were ‘actual’ arrest events and not just medical emergencies in nature.

Our evaluation found that the introduction of an EWS did not significantly increase the number of medical emergency calls responded to by the cardiac arrest team as an arrest call, described as a consequence of introducing a more comprehensive rapid response system in another large NZ hospital. It may be that such an increase observed in the other hospital was driven by the formal and informal communications built around a more wider initiative than reported in this study, as well as possible case finding activity associated with a critical care outreach type system.

Our findings suggest that an EWS alone does not increase medical emergency calls, when implemented in addition to an existing cardiac arrest team response. Another explanation may lie with the length of the evaluation period in our study, making it difficult to establish the full effect of implementation. A rise in medical emergency calls, captured by the CPR registry, did occur during the three months immediately following the introduction of ADDS; in some way a hoped for outcome of the EWS introduction, and may have resulted from increased vigilance and earlier recognition of patient deterioration or evolving medical emergency, thereby having the effect of preventing cardiac arrest. However, this rise in calls was not sustained.

This study was limited by two main issues. Firstly, attributing the observed reduction of cardiac arrest responses to the introduction of ADDS should be cautiously interpreted in the absence of a randomised design. There was a strong organisational imperative to get going and introduce the change; the paucity of resource and time constraints meant a before-after design was necessary. Consequently, isolating the effect of an EWS over other changes is difficult.

The introduction of the Liverpool Care Pathway (LCP) to Waikato hospital at the same time as ADDS, and the occurrence of the H1N1 pandemic in 2009, the baseline year, may have confounded the results. However, there was no significant reduction in total cardiac arrest calls made via the hospital telecommunications system in 2010-2011 from 2009-2010; suggesting any impact from the introduction of LCP was minimal.

Furthermore, a review of the hospital mortality rate (sum of adult deaths/adult discharges per month) during February 2009 to January 2010 and February 2010 to January 2011 identified no significant difference between the two periods (1.22 deaths/100 Discharges vs 1.13 deaths/100 discharges, mean difference 0.09, 95%CI -
0.11 to 0.29). The quantum of deaths was similar for the two periods (684 vs 666), suggesting that impact of the H1N1 pandemic on the results of this study was also minimal.

Secondly, the significant amount of missing data is unhelpful. There was missing cardiac arrest call information due to the Resuscitation Audit forms not being completed or containing insufficient information for approximately 40% of the cardiac arrest calls. In some instances an arrest call was cancelled. In addition, it is possible that a number of calls were medical emergencies for which an audit form could have been considered unnecessary by some staff.

It is equally possible that a number of calls could have been to patients who were designated 'not-for-resuscitation', but this status may have been unclear at the time the arrest call was made. However, the average rates of cardiac arrest calls with no information were similar for both periods (6.1/1000 admissions versus 5.5/1000 admissions) and the 0.6 mean difference in these rates would not explain the observed difference in actual cardiac arrests.

**Conclusion**

Following the introduction of an EWS system that involved colour coding to indicate abnormal vital signs, scoring of abnormality and an escalation protocol driven by the EWS score, we observed a reduction in the incidence of cardiac arrest responses in a tertiary hospital in NZ.

**Competing interests:** None known.

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**Correspondence:** David Drower. Email: drowerd@gmail.com

**References:**


Stereotypes do not always apply: findings from a survey of the health behaviours of mental health consumers compared with the general population in New Zealand

Amanda J Wheeler, Brian McKenna, Dominic Madell

Abstract

Aim Research suggests that people with mental illness frequently have worse physical health than the general population. Our study sought to investigate the health behaviours of mental health consumers in New Zealand, as we hoped that by clearly describing this group more targeted healthcare could be provided.

Method We surveyed 404 adult mental health consumers about their: height and weight, fruit and vegetable intake, exercise activity, smoking, alcohol and drug use.

Results We found that mental health consumers abstained from alcohol more often than those from the general population sample. However, we also found increased obesity, poorer eating habits, less physical activity, and a higher proportion of smokers among mental health consumers. We also found that those who did drink alcohol did so more heavily than drinkers from the general population sample. Finally, there were a number of interesting demographic variations among mental health consumers in terms of their health behaviours. For example, those in the European/Other ethnic group more often consumed an alcoholic drink at least 3 or 4 times a week than those in other ethnic groups, and those in the Pacific group did this less often.

Conclusions Mental health consumers cannot be stereotyped as a single homogeneous group in relation to their health behaviours.

Implications Health professionals should strongly encourage engagement from mental health populations to identify their physical health behaviours. Individual differences in health behaviours should not be ignored just because a mental illness is present.

It has been argued that those with mental health problems frequently have worse physical health than the general population, and experience barriers to receiving adequate care from fragmented health systems that are disconnected from their needs. For example, Scott et al found associations between obesity and depressive disorders and anxiety disorders in a survey that drew respondents from a number of countries across the world. Similarly, McIntyre et al found that those with a mood disorder were more likely to be obese than those without in a Canadian survey.

Research also shows that mental health consumers are more often smokers, and smoke more cigarettes than those in the general population. In a South Australian survey, Bowden, Miller and Hiller found that 26.4% of those with a general mental illness and 51.2% of those with a severe mental illness smoked, compared to 18.7% of those without a mental illness.
Research has also identified associations between alcohol and other substance use and mental illness. Barnett et al considered a sample of referrals to a specialist early intervention service for first-episode psychosis in England and found alcohol abuse in 43%, cannabis abuse in 51% and Class A drug use in 55%. A New Zealand study of community patients attending four public psychiatric services in New Zealand found substance use disorder in 15.4% of those with schizophrenia, 12.8% of those with bipolar disorder and 11.3% of those with depression.

Literature has also suggested that levels of physical activity among people with mental health conditions may be limited and that their diets may be worse. In an English study of middle aged people with a diagnosis of schizophrenia, Brown et al found that 36% of males and 32% of females had taken no exercise in the past week.

Similarly, McCreadie found that only 59% of their sample experiencing schizophrenia considered themselves physically active. McCreadie also noted that fewer males in this sample than males in the general population reached acceptable levels for the consumption of fresh fruit, vegetables, skimmed or semi-skimmed milk, potatoes, pasta or rice, and pulses. In addition, fewer females with schizophrenia than females in the general population reached acceptable levels for the consumption of skimmed or semi-skimmed milk, potatoes, pasta or rice.

There is ample international evidence to suggest that the health behaviours of people with serious mental illness are often worse than those in the general population. With this in mind, our study sought to investigate the health behaviours of mental health consumers in NZ, as this is a group that has received little attention and there are cultural differences which may impact on physical health outcomes. We also hoped that by providing a clearer picture of the health behaviours of this group, more targeted healthcare could start to be provided.

Methods

Study setting and participants—This study aimed to survey 500 consumers aged 18 to 65 receiving care from mental health services of one District Health Board (DHB) in Auckland. Adults living in the community attending general mental health, early psychosis intervention, forensic and culturally specific Māori and Pacific Island mental health services were invited to take part between July and November 2009. Collectively, these teams have around 2500 open cases at any point in time. However, fewer people are likely to be actively and regularly using these services. For example, just over 1,400 people had a face-to-face contact with their mental health team in a four week period between September and October 2009 (Southen C, Personal communication, 1 October 2009). Guidance was sought from the DHB’s mental health consumer advisors, locality managers and individual mental health teams around appropriate recruitment and participant support strategies to encourage participation. Information packs (including the survey) were distributed through community mental health team (CMHT) reception areas, directly to consumers by their mental health clinician and via non-government organisations (NGOs) who specifically provided support for mental health consumers engaged with this DHB. In addition, the researchers presented information about the study at consumer group meetings.

Questionnaires could be self-completed by consumers or with the support of a researcher, a consumer researcher, or their mental health support worker. Completed questionnaires could be returned using pre-paid envelopes, or via drop-boxes at mental health service and NGO locations. All participants were eligible to win one of five $100 gift vouchers in a prize draw. Entry forms containing participant names and contact details were separated from questionnaires to ensure questionnaire data remained anonymous. Mental health support workers and clinicians were eligible for...
one prize draw entry for each consumer they supported to take part to win one of five $100 gift vouchers.

Response rates from mental health consumers receiving the services of non-culturally specific (mainstream) adult CMHTs were lower than anticipated. In order to improve participation of this group postal recruitment was also conducted. Mental health consumers aged 18–65 years with a minimum of one recorded face-to-face contact with these teams in the previous 4 weeks and three face-to-face contacts in the previous 6 months met the inclusion criteria (n=880). These criteria were set to ensure that those receiving postal surveys were current and active service users.

To reduce the likelihood of multiple entries, the completed prize draw entry forms were checked against the mail out database and these names were removed from the mail out.

It was not possible to know exactly how many consumers were asked to take part by mental health service teams. However, based on numbers of unused questionnaires returned, we know that a maximum of 543 questionnaires were handed out, meaning a minimum response rate of 51.6% (280/543). The response rate to the mail out survey was 14.1% (124/880) meaning a minimum response rate of at least 28.4% (404/1423) for the whole study.

It is possible that the response rate was higher as non-respondents could have received a personal invitation to participate (been included in the 543 questionnaires handed out) and received a postal invitation (and been included in the 880 questionnaires mailed out); hence the minimum response rate of 28.4%.

Ethics approval was obtained from the Ministry of Health Northern Region Ethics Committee (NTX/09/44/EXP).

Instrument—The health survey used consisted of five parts. This article is concerned with Parts Two and Five. Part Two asked about health behaviours including fruit and vegetable intake, exercise activity, height and weight, and smoking, drinking and drug use. Part Five asked about socio-demographic information of the mental health consumers, including age, gender, and ethnicity. Questions in Part Two were aligned with those asked in the NZ Health Survey, with an additional question about illicit drug use for this study. Findings from other parts of the survey are reported elsewhere.

Data analysis—Survey data were analysed using SPSS (Version 13). Apart from descriptive statistics, Spearman’s correlations, Chi-square, Mann-Whitney and ANOVA tests were used to analyse the data along with relevant post-hoc analysis. Demographic differences within the mental health consumer sample were investigated, and major findings were compared to those of the NZ Health Survey.

Results

Table 1 shows the sociodemographic characteristics of the surveyed sample (n=404) along with the prevalence of key health indicators. These are compared with the demographic characteristics of respondents to the NZ Health Survey (n=12,488).

Just over half of the consumer sample was female (n=224; 55.4%). The participants ranged in age from 18 to 70 years, with the majority being 25 years old and over. The mean age of females (n=223) was 41.5 years and the mean age of males (n=163) was 40.7 years. Questions relating to height and weight had a relatively low response rate, this may reflect respondents not knowing this information and not wanting to go to extra effort to find it out. However, from the data collected, the mean height for females (n=174) was 165 centimetres and for males (n=119) was 175 centimetres. The mean weight for females (n=180) was 78.8 kilograms and for males (n=138) was 93.9 kilograms.

The demographic characteristics of the consumer survey respondents are comparable with those who responded to the NZ Health Survey (Table 1) in terms of gender and age. Māori and Asian service users were under-represented in the current sample.
compared to respondents for the NZ Health Survey, and Pacific service users were over-represented (see Table 1).

Table 1. Mental Health Consumer Survey and corresponding New Zealand Health Survey results

<table>
<thead>
<tr>
<th>Demographic</th>
<th>Mental health consumer survey population (n=404)</th>
<th>New Zealand Health Survey population (n=12,488)</th>
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</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males n (%)</td>
<td>164 (40.6)</td>
<td>5,273 (42.2)</td>
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<tr>
<td>Females n (%)</td>
<td>224 (55.4)</td>
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<tr>
<td>Unknown n (%)</td>
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<td>–</td>
</tr>
<tr>
<td>Age</td>
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<td></td>
</tr>
<tr>
<td>Mean</td>
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<td>Under 25 years old n (%)</td>
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<td>Unknown n (%)</td>
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<td>8,593 (68.8)</td>
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<td>24 (5.4)</td>
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<td>BMI mean</td>
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<tr>
<td>Obese (BMI = 30+) %*</td>
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<td>Adequate fruit intake %*</td>
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<tr>
<td>Active %*</td>
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<td>Smoking</td>
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<td>Current smoker %*</td>
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<td>Never tried smoking %*</td>
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<td>11–15</td>
<td>20.5</td>
<td>21.4</td>
</tr>
<tr>
<td>16–20</td>
<td>25.3</td>
<td>15.9</td>
</tr>
<tr>
<td>21–25</td>
<td>11.4</td>
<td>6.4</td>
</tr>
<tr>
<td>26–30</td>
<td>4.2</td>
<td>3.0</td>
</tr>
<tr>
<td>31+</td>
<td>6.0</td>
<td>1.5</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency of drinking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never %*</td>
<td>42.5</td>
<td>16.3</td>
</tr>
<tr>
<td>Monthly or less %</td>
<td>32.4</td>
<td>26.1</td>
</tr>
<tr>
<td>Up to 4 times a month %</td>
<td>10.1</td>
<td>19.7</td>
</tr>
<tr>
<td>Up to 3 times a week %</td>
<td>9.1</td>
<td>17.7</td>
</tr>
<tr>
<td>4 or more times a week %</td>
<td>6.0</td>
<td>20.2</td>
</tr>
</tbody>
</table>
Table 2 shows answers to a question in the survey which asked whether respondents have or have had any of a list of conditions. Multiple responses could be selected for this question. This shows that over half of the sample reported depression, a third reported anxiety disorder, a third reported schizophrenia, and nearly a fifth reported bipolar disorder. The most common physical health conditions were indicated to be high cholesterol (roughly a quarter of the sample) and high blood pressure (roughly a fifth of the sample).

**Table 2. Self-report data on physical and mental health conditions***

<table>
<thead>
<tr>
<th>Condition</th>
<th>Yes n</th>
<th>Total n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>204</td>
<td>372</td>
<td>54.8</td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>120</td>
<td>371</td>
<td>32.3</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>116</td>
<td>370</td>
<td>31.4</td>
</tr>
<tr>
<td>High cholesterol (hyperlipidemia)</td>
<td>98</td>
<td>370</td>
<td>26.5</td>
</tr>
<tr>
<td>High blood pressure (hypertension)</td>
<td>75</td>
<td>371</td>
<td>20.2</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>69</td>
<td>371</td>
<td>18.6</td>
</tr>
<tr>
<td>Asthma</td>
<td>58</td>
<td>372</td>
<td>15.6</td>
</tr>
<tr>
<td>Diabetes: Type 2</td>
<td>34</td>
<td>359</td>
<td>9.5</td>
</tr>
<tr>
<td>Stroke</td>
<td>11</td>
<td>370</td>
<td>3.0</td>
</tr>
<tr>
<td>Kidney disorders</td>
<td>9</td>
<td>372</td>
<td>2.4</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>9</td>
<td>372</td>
<td>2.4</td>
</tr>
<tr>
<td>Angina (ischemic heart disease)</td>
<td>8</td>
<td>372</td>
<td>2.2</td>
</tr>
<tr>
<td>Heart attack</td>
<td>7</td>
<td>372</td>
<td>1.9</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>6</td>
<td>370</td>
<td>1.6</td>
</tr>
<tr>
<td>Diabetes: Type 1</td>
<td>5</td>
<td>358</td>
<td>1.4</td>
</tr>
<tr>
<td>Heart failure</td>
<td>5</td>
<td>372</td>
<td>1.3</td>
</tr>
<tr>
<td>None of those listed</td>
<td>16</td>
<td>375</td>
<td>4.3</td>
</tr>
</tbody>
</table>

*Respondents could tick as many as apply.

**BMI**—The mean BMI for the mental health consumer sample was 29.2 (n=274). Overall 23.0% of the sample (n=63/274) were classified as overweight and 40.9% (n=112/274) as obese. The proportion of the sample that was overweight was lower
than for the NZ Health Survey (36.2% of n=12,488), but a higher proportion was obese (NZ Health Survey: 26.5% of n=12,488) (overweight: Z=-4.51, p<0.0002; obese: Z=5.32, p<0.0002).

There was a small positive correlation between age and BMI ($r_{271}=0.137$, $p=0.024$). In addition, those in the European/Other (M=28.1, SD=6.8) and Asian (M=27.8, SD=8.2) groups had a lower BMI than those in the Māori (M=34.6, SD=9.8) and Pacific groups (M=35.8, SD=10.6) ($F_{(4,299)}=10.416$, $p<0.0005$, post hoc test: Tukey’s HSD, $p<0.05$).

There was no significant difference between the genders in their BMI scores.

**Fruit intake**—In the NZ Health Survey, adequate fruit intake was defined as eating two or more servings of fruit each day (a serving can be defined as the amount which fits into the palm of one’s hand). By this definition, 44.9% (n=174/388) of the mental health consumer sample ate an adequate amount of fruit. This proportion was lower than for the NZ Health Survey (60% of n=12,488) ($Z=-0.15$, $p<0.0002$).

There were no significant associations between how many servings of fruit were eaten per day and gender, age or ethnicity.

**Vegetable intake**—Adequate vegetable intake was defined in the NZ Health Survey as three or more servings of vegetables each day (a serving can be defined as the amount which fits into the palm of one’s hand). By this definition, 30.2% (n=117/387) of mental health respondents ate an adequate amount of vegetables. This proportion was lower than in the NZ Health Survey (64.1% of n=12,488) ($Z=-13.60$, $p<0.0002$).

Males ate fewer servings of vegetables per day than females ($U=14772.0$, $n_{(m)}=159$, $n_{(f)}=217$, $p=0.014$): only 23.9% (n=38/159) of males ate adequate amounts of vegetables as compared with 35.9% (n=78/217) of females. Māori were less likely to eat three or more servings of vegetables per day than other ethnic groups ($X^2_{(4)}=18.065$, $p=0.001$). Age was not correlated with number of servings of vegetables eaten.

**Exercise activity**—In the NZ Health Survey, ‘regularly physically active’ was defined as undertaking at least 30 minutes of physical activity per day on five or more days of the last week. ‘Sedentary’ was defined as less than 30 minutes of physical activity in the last week.

For the mental health consumer survey, 25.1% (n=88/351) of respondents were regularly physically active and 16.2% (n=57/351) were sedentary by these definitions. A greater proportion of respondents from the mental health consumer survey was sedentary than the NZ Health Survey (9.9% of 12,488) and a lower proportion was active (NZ Health Survey: 52.1% of 12,488) (sedentary: $Z=3.89$, $p<0.0002$; active: $Z=-9.991$, $p<0.0002$).

Males did 30 minutes of moderate activity on more days (M=3.5, SD=2.3) in the last week than females (M=2.6, SD=2.1) ($t_{(343)}=3.665$, $p<0.0005$). Age and ethnicity were not associated with number of days in which 30 minutes of moderate activity were undertaken.
Smoking—Smokers made up 41.6% (n=165/397) of the mental health consumer sample, 17.4% (n=69/397) were ex-smokers and 41.1% (n=163/397) had never smoked. There was a higher proportion of current smokers in the mental health sample than in the NZ Health Survey population (19.9% of 12,488) but also a higher proportion who had never tried smoking (NZ Health Survey: 35.4% of 12,488) (current smokers: Z=10.513, p<0.0002; never smoked: Z=2.317, p=0.02).

More males (51.8%; n=85/164) than females (33.8%; n=75/222) were smokers (X^2(2)=12.685, p=0.002) in the mental health sample. More of the Māori group were smokers (59.7%; n=37/62) and fewer had never smoked (19.4%; n=12/62) than those in other ethnic groups, and more of those in the Asian group had never smoked (73.9%; n=17/23) (X^2(8)=25.301, p<0.001). There was no association between age and smoking.

Of consumer respondents who smoked, 55.3% (n=89/161) had a relative, friend, doctor or other health worker concerned about their smoking or suggest they cut down during the last year. 11.8% (n=19/161) of respondents had received such comments prior to the last year, and 32.3% (n=52/161) had never received such comments.

There were no associations between whether a relative, friend, doctor or other health worker had suggested a reduction in smoking either in the last year or prior to this and gender or age. It was not possible to test for an association between this variable and ethnicity because of limited numbers in individual ethnic groups.

Alcohol—Of the mental health consumer sample, 42.5% (n=164/386) never had a drink containing alcohol, 32.4% (n=125/386) had a drink monthly or less, 10.1% (n=39/386) had a drink up to four times a month, 9.1% (n=35/386) had a drink up to three times a week, and 6.0% (n=23/386) had a drink four or more times a week. Mental health consumer respondents were more likely to completely abstain from alcohol than those from the NZ Health Survey population (Z=13.46, p<0.0002).

There were small negative correlations between age and frequency of alcohol drinking (r(s)=-0.166, n=376, p=0.001). In addition, those in the European/Other group more often consumed an alcoholic drink at least 3 or 4 times a week than other ethnic groups, and those in the Pacific group did this less often (X^2(8)=18.615, p=0.017). For example, 19.4% (n=54/278) respondents in the European/Other group had an alcoholic drink three times a week or more often, as compared with 3.8% (n=2/52) of Pacific respondents. There was no association between how often alcoholic drinks were consumed and gender.

Males consumed more alcoholic drinks on a day when drinking than females (U=4281.0, n(0)=87, n(1)=126, p=0.003). For example, 23.8% (n=30/126) females consumed five or more drinks on a day when drinking, as compared with 39.1% (n=34/87) of males. There was also a small negative correlation between age and how many drinks containing alcohol were consumed (r(s)=-0.262, n=217, p<0.0005).

Finally, those in the Pacific group were more likely than those in other groups to have five or more drinks on a day when drinking than those in other groups (X^2(3)=16.981, p=0.001); 54.2% (n=13/24) of this group drank at this level.

Of respondents who drank alcohol, 25.5% (n=56/220) had a relative, friend, doctor or other health worker that had been concerned about their drinking or suggested they
cut down during the last year; 8.2% (n=18/220) had received such comments prior to the last year, and 66.4% (n=146/220) had never received such comments.

There was no significant difference in whether males and females had received a suggestion to cut down drinking either in the last year or prior to this ($\chi^2 (2) = 4.116$, $p = 0.128$). Those who had received suggestions in the last year tended to be younger than those who had received suggestions prior to this, and also younger than those who had never received a suggestion ($F_{(2,211)} = 7.514$, $p = 0.001$, post hoc test: Tukey’s HSD, $p < 0.05$). It was not possible to test for an association between this variable and ethnicity because numbers in individual ethnic groups were too small.

Illicit drug use—A total of 294 participants answered whether they had used an illicit substance in the past year; 24.5% (n=72/294) had used cannabis, 4.4% (n=13/294) had used amphetamines, 3.0% (n=9/294) had used hallucinogens and 4.4% (n=13/293) of respondents had used opiates in the past year (multiple responses could be selected for drug use on the questionnaire). This question was not asked in the NZ Health Survey.

Males were more likely to have used cannabis in the past year (31.3%; n=36/115) than females (19.8 %; n=34/172) ($\chi^2 (1) = 4.974$, $p = 0.026$). In addition, the age of those who had used cannabis ($t_{(285)} = -6.95$, $p < 0.0005$), amphetamines ($t_{(285)} = -4.745$, $p < 0.0005$) and hallucinogens ($t_{(285)} = -4.645$, $p < 0.0005$) in the past year was significantly lower than those who had not.

There were no associations between use of amphetamines, hallucinogens or opiates and gender. Nor were there significant differences in the ages of those who had used opiates and those who had not. Finally, there was no association between ethnicity and use of cannabis in the past year. Sample sizes within individual ethnic groups were not large enough to test for associations between ethnicity and use of amphetamines, hallucinogens or opiates.

Discussion

The first major finding of our study was that there was increased obesity in the mental health consumer group as compared with the NZ Health Survey population (Table 1). A similarly lower proportion of the general population survey in Australia has been shown to be overweight or obese, and high obesity levels have also been found among other mental health samples. There may be a number of reasons why obesity is more prevalent among populations with mental health issues.

Mental illness can have major impacts on appetite, energy and motivation. For example, neurobiological factors associated with mental illness and the side effects of medications used for treatment can also encourage weight gain. In addition, socio-demographic factors such as increased poverty and lower educational attainment among people with mental illness may mean that healthy foods (such as fresh fruit and vegetables) and health care services, that could encourage healthy eating behaviours, are less accessible.

Findings from our study suggest that the eating habits of the mental health consumer group were not as healthy as those from the general population in New Zealand, with lower proportions eating adequate fruit and vegetables (Table 1).
Our study also found a lower proportion of mental health consumers being classified as ‘active’ and a higher proportion being classified as ‘sedentary’ than in the NZ Health Survey population (Table 1). This factor could also contribute to obesity among people with mental illness.

Other literature has also suggested that people with mental health conditions are less physically active than the general population, and this may relate to the effects of medications or the symptoms of mental illness inhibiting motivation.7

There were major differences between the mental health consumer sample and the NZ Health Survey population in smoking behaviour. Around twice the proportion of mental health consumers were smokers as compared to the NZ Health Survey population. The same is also true of the mental health consumer sample in comparison with the general population sample in Australia.14 The consumer sample smokers also tended to smoke more than smokers from the NZ Health Survey population. These findings are echoed in other research into populations with mental health issues.4,19

The increased prevalence of smoking among mental health consumers is a complex issue which may relate to a range of factors. For example, cigarettes may be used as a tool to cope with the symptoms of mental illness, and also as a means of fitting in and feeling comfortable with other people in a similar situation.

In addition, coping with the side-effects of medications may make it more difficult for people with mental illness to meet the extra challenge of stopping smoking.20 Cigarettes have also been used as a reward to manage the behaviour of people in mental health institutions in the past, which would have encouraged addiction.21

Another major finding of this study was that the mental health consumer sample drank alcohol less frequently than the NZ Health Survey population. The reasons for this may be that alcohol can worsen the symptoms of mental illness or interfere with responses to medications.22 However, despite the reduced likelihood of the consumer sample drinking alcohol, those that did drink did so more heavily than drinkers from the NZ Health Survey.

Past research has identified associations between alcohol use and mental illness, and this may be a maladaptive strategy for coping with the increased stress and unpleasant symptoms caused by mental health conditions.

Our research suggests then that mental health consumers are polarised in terms of their alcohol intake. The large majority of mental health consumers drank infrequently or never, but those who did drink sometimes did so more heavily than those in the general population. Our research also suggests cultural variations in the patterns of drinking behaviour. The European/Other ethnic group were more likely to consume alcohol on an on-going weekly basis, but the Pacific Island group were more likely to be involved in binge drinking behaviour.

Our study also looked at drug use among the mental health consumer sample, although there was no comparative data from the NZ Health Survey. Given that nearly a quarter of the group had used cannabis in the past year, with lower percentages having used amphetamines, hallucinogens or opiates, these findings will be of interest to health professionals. Illicit drug use can have adverse impacts on the course of
mental illness including symptom exacerbation, increased hospitalisation, medication adherence, problem behaviours, and decreased social functioning.24

**Limitations**—This study has a number of limitations. First, how well the sample represented the overall population of mental health consumers with serious mental illness who regularly access community mental health services in the region is unknown due to the lack of official statistics available for this group. Recruitment was driven by consumer responsiveness, rather than a representative sample being selected because we wanted to encourage participation in this often hard to reach population. The present study also suffers from a potential response bias, as individuals who were more unwell may not have responded to the survey.

Reliance on self-report data may also reduce the validity of the findings. It has been argued that the meaning of results from self-report measurement involving seriously mentally ill populations should be interpreted with caution, as they may contain biases due to cognition, periodic affective swings, and recent life events that may better reflect psychopathology and symptoms than actual life conditions or functions.30

The present study, however, surveyed a community sample of mental health consumers, a group of people with serious mental illness who were not acutely unwell. Finally, comparisons were made with the NZ Health survey which was conducted in 2006/7 and there may have been changes in the two year intervening period.

Some of the NZ Health Survey population reported experiencing a mental illness sometime in their life (10.5% depression, 4.3% anxiety, 1.0% alcohol use disorders, 0.7% bipolar disorder, 0.3% schizophrenia) and it is possible that a number of these people were consumers with a serious mental illness engaged with a community mental health service.

Whilst the size of this group who had the potential to bias the findings is unknown, the effect would be to reduce the size of any difference we have found between the consumer and general population groups. However the fact that the differences between the two groups were statistically significant suggests that this bias was not substantial.

**Implications**—Despite its limitations, our study suggests that the physical health behaviours of mental health consumers in NZ are frequently poorer than those from the general population. Health professionals should therefore strongly encourage engagement from this group, especially as they may be less likely to initiate contact with healthcare providers themselves.25

Resources to address the physical health of mental health populations also need to be made available. However, health professionals should also take care not to stereotype individual mental health consumers, as our study showed a variety of within-group variations in health behaviours, as well as alcohol abstinence across a large proportion of the group. Just as in the general population, individual differences in health behaviours should not be ignored just because a mental illness is present.
Competing interests: None known.

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References:
Introduction of sacral neuromodulation for the treatment of faecal incontinence

Sarah Benson-Cooper, Emily Davenport, Ian P Bissett

Abstract

Introduction Faecal incontinence (FI) is a condition that impairs quality of life and ability to function socially. Over the last 15 years a promising new therapy (sacral neuromodulation, SNM) has been introduced which has been associated with marked improvement in many incontinence symptoms.

Aims To assess the early results of SNM in Auckland in terms of improved continence in those undergoing implantation of a permanent stimulator, and determine whether these results are comparable to overseas data.

Methods Patients who met the criteria for SNM; severe faecal incontinence, failure to respond to other measures including biofeedback, dietary modification, and appropriate surgical intervention were offered this treatment.

After an initial bowel diary, patients underwent lead placement connected to an external stimulator and only those who responded had an implanted stimulator placed. Results were assessed by repeated bowel diary, QoL scores and continence scores.

Results Of 29 patients who had initial percutaneous stimulation, 27 showed adequate improvement and went on to permanent implanted stimulator. Of these, results were available on 26. The median number of FI episodes per week preoperatively was 7.25. The median post implantation was one. FI episodes improved following SNM by a mean of 12.21 episodes per week (95% confidence interval 4.91 to 19.51, p value=0.002). For those with longer follow up the initial improvement was sustained. There was a mean follow up time of 10.7 months (range 1 to 30 months).

Conclusion Early results are encouraging, with a significant improvement in faecal incontinence following SNM. The results in Auckland in terms of improvement in symptom severity and quality of life are significant and comparable to other centres. SNM offers a good alternative for patients with end-stage FI.

Faecal incontinence (FI) is a socially stigmatised condition that has a major effect on a patient’s quality of life. Many patients are reluctant to seek help until their symptoms are severe or because they fear that there is no available treatment. It affects approximately 13% of the New Zealand (NZ) adult population.1

The vast majority are treated with conservative management—dietary manipulation, appropriate medication to improve stool consistency, and pelvic floor physiotherapy. Only a few patients require surgical repair of sphincter injuries or prolapse. There remain a small number of patients, refractory to these treatments and incapacitated by faecal incontinence, who are suitable for treatment by sacral neuromodulation (SNM), also known as sacral nerve stimulation. In NZ these patients have previously been
managed by offering a stoma to control the incontinence or by the use of incontinence pads.

SNM is a promising new minimally invasive therapy, which involves implanting a Tined lead (Model 3889 or 3093, Medtronic) into the S3 or S4 foramen and initially connecting this to an external nerve stimulator via a percutaneous lead. It is cost effective and significantly cheaper when compared with stoma formation.

The present study seeks to determine whether encouraging results achieved overseas with SNM are replicable in a new program set up in New Zealand.

Methods

Patients—Auckland City Hospital offers a tertiary service for the management of patients with severe FI refractory to other measures. SNM is reserved for patients who have failed all other treatments and have at least two episodes of FI per week for a minimum of 12 months.

The decision to perform SNM on each patient was agreed at the Auckland City Hospital monthly pelvic floor multidisciplinary meeting after extensive assessment at the Colorectal Pelvic Floor Clinic. Only four patients had not undergone previous surgery for incontinence. Most patients included had undergone multiple previous procedures—15 patients had sphincter repair, 13 had undergone an anterior delormes, 7 had anterior mesh rectopexy and 4 patients underwent PTQ injection.

Obstetric trauma was the commonest aetiology, followed by non-obstetric trauma such as perianal injury and sphincterotomy, and then post-radiation and post-anterior resection incontinence. To date, more than 800 patients with faecal incontinence or obstructed defecation have been assessed at this clinic and their results entered on a database.

Data collection—Prior to undergoing SNM patients underwent pelvic floor training from a dedicated pelvic floor physiotherapist, and complete a comprehensive faecal incontinence questionnaire (C-FIQ) which includes a faecal incontinence severity index score (FISI), and a NZ validated incontinence quality of life score. A two week incontinence diary, trans-anal ultrasonography and anal manometry were also recorded.

The tined four-electrode lead (Model 3889, Medtronic, Minneapolis, MN, USA) and external stimulator (Model 3625, Medtronic) were used to identify patients that were likely to be responders. Lead placement was determined by the best stimulation response in theatre. The technique of lead placement has been previously described in detail by Wöllner. Outcome was assessed with a screening 2-week incontinence diary, and responders defined as having at least a 50% reduction in the number of FI episodes per week compared with baseline. A permanent implanted stimulator (InterStim Model 3023, Medtronic) was then placed, and incontinence diaries and QoL measures repeated at intervals post operatively to assess results. All patients who had permanent stimulator implanted for treatment for FI at Auckland City Hospital were included in our study.

Statistics—The primary outcome in this study was the difference in the mean number of FI episodes per week after permanent SNM as compared with baseline score. A
secondary outcome was the difference in total quality of life scores between baseline and post SNM implantation. The paired t test was used to analyse continuous parametric data. P<0.05 was considered statistically significant.

Results

Between 2009 and 2012, 29 patients underwent initial temporary lead placement for faecal incontinence at Auckland City Hospital. Five of these were men and 24 were females, with a median age of 60 years (range 39–84). Eleven of the leads were placed at S3 on the left, 11 at S3 on the right, and 7 at S4 on the left.

Faecal incontinence episodes: baseline vs screening—27 of the 29 showed an adequate improvement and met criteria, determined as 50% reduction in diary-recorded faecal incontinence episodes (with a mean difference of +10.17 (95%CI: 3.848–16.50; p=0.0027) (Figure 1). Two patients did not meet criteria; one patient had no decrease in FI weekly score, and one patient did not improve by 50% or more.

Figure 1. Baseline versus screening FI diaries
Faecal incontinence episodes: baseline versus last follow up—27 patients went on to have a permanent stimulator implanted. One patient had the permanent stimulator removed following infection after one month and therefore had no post implantation diary completed.

Of the 26 patients with complete data (Figure 2) the median number of incontinence episodes per week at baseline was 7.25 (range 1–90), and post-implantation was 1 (range 0–7). There was a mean difference of -12.2 (95%CI: -4.9–19.5; p=0.002). Mean follow up for the 26 patients was 10.7 months (range 1–30 months). For those with longer follow up the initial improvement was sustained.

Quality of life—15 patients had both baseline and post-implantation QOL data completed. Scores were a total quality of life score out of 104. Fourteen patients reported improvement in total quality of life score (Figure 3), with a mean difference of +23.0 (95%CI 12.8, 33.2 - P=0.0003). One patient reported a decrease in QoL (43/104 to 40/104). This did not correlate with the patients FI diary.

Figure 2. Baseline FI episodes versus last follow up FI episodes

Paired t-test P=0.0020, mean difference -12.21 (95%CI: -4.910, -19.51).
Figure 3. Total quality of life score, pre versus post implantation

Paired t-test P=0.0003, mean difference +23.0 (95%CI 12.76–33.24).

**Complications**—This procedure was not without complications. There were five patients who developed infections: two of these following temporary lead placement, who then had the lead removed and treatment with intravenous antibiotics. They then went forward for the second stage. Three patients developed infection after permanent stimulator placement, two being successfully treated with intravenous antibiotics alone, and one patient required removal of the permanent stimulator and then declined replacement. Two patients had lead breakage requiring lead replacement. One patient had the stimulator battery fail requiring stimulator replacement. Most patients required a change in programme settings during their follow up.

**Discussion**

There was a statistically significant improvement in the number of faecal incontinence episodes in patients who underwent permanent stimulator placement for SNM at Auckland City Hospital. In addition to a sustained functional improvement, quality of life was significantly enhanced as measured by total quality of life scores in the 15 patients for whom we had complete data.
A screening trial is an important predictive test for response to SNM; patients who had a good response to the screening trial had an improved and sustained response to the permanent implant.

Unfortunately, as the data was collated retrospectively and post procedure we were unable to obtain complete quality of life data for each patient pre- and post-intervention. However, despite these shortcomings, the data we do have has shown a significant improvement in quality of life scores following SNM. Ideally in future quality of life data would be further analysed in domains (physical, social, emotional, general wellbeing) to provide further information.

So far experience with SNM is short in NZ, with a mean follow-up 10.6 months for this study. Further follow up would be required to assess long-term outcomes and longevity of treatment. We would expect lead fractures or lead failure to become more of a problem in coming years. However, reassuringly, those with longer follow up between one to 3 years have shown no apparent deterioration in FI episodes.

These results are consistent with earlier studies of SNM in FI. In the study by Hetzer et al, 44 patients with severe incontinence were initially assessed with 84% having an improvement in continence on initial, temporary testing and these were then fitted with an implantable stimulator. In these 37 patients there was a significant reduction in faecal incontinence scores, and 34 of them (92%) were deemed to have successfully treated their incontinence.

In a further randomised controlled trial (involving 60 patients in each arm) patients were randomised to either SNM or best supportive therapy. The SNM patients had significantly fewer incontinence episodes, a better quality of life and almost 50% had perfect continence. Long-term results of SNM in treatment of FI has been reported from Mellgren, and reassuringly results appear durable. We would expect similar durability from our study.

**Conclusion**

Early results are encouraging, with a significant improvement in FI and improvement in quality of life following SNM. A screening trial is an important predictive test for response to SNM. The results in Auckland, in terms of improvement in symptom severity and quality of life are significant and comparable to other centres. SNM offers a good alternative for patients with end-stage FI.

**Competing interests:** None known.

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**Acknowledgement:** We thank Dr Tom Wang at Auckland City Hospital for his statistical analysis.

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References:


The Green Lane and Auckland City Hospital cardiac resynchronisation therapy experience

Andrew Martin, Susan Sinclair, Nigel Lever, Jim Stewart

Abstract

Aim To evaluate the outcome of cardiac resynchronisation therapy (CRT) in Auckland since its inception in 2000. Outcome measures were periprocedural events including procedure duration, fluoroscopic time, procedure success, complications, and echocardiographic markers of response.

Methods Retrospective review of those undergoing CRT implantation at Green Lane and Auckland City Hospitals.

Results 159 CRT procedures were performed on 139 patients between 2000 and April 2011. Of all procedures: 63% had new device implants, and 23% were upgraded from a pacemaker or defibrillator to CRT. Mean age 62 years, 77% male, mean left ventricular ejection fraction (LVEF) 25%, mean QRS duration 175 ms, 91% LBBB, 36% ischaemic cardiomyopathy. Mean procedure time 147 min and fluoroscopy time 30 min. Eight patients had significant periprocedural complications (6%), 17 experienced lead displacement (12%), and 15 (11%) had unsuccessful procedures. 46% of patients had complete echocardiographic data, of these LVEF improved by >5% in 52%, and decreased by >5% in 6% over a 16-month follow-up.

Conclusions Our service performs well with respect to complication rate, though less well when failure of implantation and lead dislodgement are considered. Where complete echocardiographic data was present 52% of our patients derived significant improvement in LVEF.

Cardiac resynchronisation therapy (CRT) with optimal medical therapy, has been demonstrated to improve heart failure symptoms, quality of life, exercise capacity, left ventricular (LV) systolic function, and reduce mortality in patients with severe impairment of LV systolic function, severe heart failure symptoms (New York Heart Association (NYHA) functional class III or ambulatory class IV), and a wide QRS complex. Recent evidence supports using this in addition to implantable cardioverter defibrillator (ICD) in patients with mild to moderate heart failure symptoms, wide QRS complex, and severe LV systolic dysfunction.

New Zealand guidelines published in 2010 recommend CRT for patients with reduced left ventricular ejection fraction (LVEF) ≤35% after ≥6 weeks of optimal medical heart failure treatment, whose QRS duration is >149 ms or is 120–149 ms with two additional criteria for dys-synchrony (aortic pre-ejection delay >140 ms, interventricular mechanical delay >40 ms, or delayed activation of the posterolateral LV wall). Ideally these patients should be NYHA Class III, have had no major cardiovascular event in the prior 6 weeks, and be in sinus rhythm. In addition there should be no major comorbidity that would reduce survival to less than 18 months, or seriously impair quality of life.
The most commonly encountered problem with the CRT procedure is the inability to successfully implant the LV lead. This is typically the result of unfavorable coronary venous anatomy and/or proximity to the left phrenic nerve (resulting in diaphragmatic stimulation during pacing).

A systematic review by McAlister and colleagues involving 6123 patients across 54 studies reported an implant success rate of 93% (95% confidence interval (CI), 92–94%). This same review identified a peri-implantation mechanical complication rate of 4.3% (95% CI, 3.6–5.1%), which included coronary sinus dissection, cardiac tamponade, pneumothorax, and pocket erosion. During a median 11 month follow-up, lead problems (primarily as a consequence of displacement) occurred with an incidence of 6.6% (95% CI, 5.6–7.4%), with lead dislodgement described in up to 10% by others. During a median 6 month follow-up, hospitalisation for infection involving the implant site occurred in 1.8% (95% CI, 1.3–2.5%). Peri-implant death occurred in 0.3% of patients (95% CI, 0.1–0.6%). There was no statistically significant difference between implant success rates or frequency of adverse events (including lead dislodgement) across studies involving CRT alone or CRT combined with ICD devices.

The first CRT implant occurred at Green Lane Hospital (GLH) in the year 2000, and until 2003 a single physician performed device implantation. The service was relocated from GLH to Auckland City Hospital (ACH) in 2004, and the service has expanded such that devices are now primarily implanted by three physicians. In addition, ACH has provided training in CRT implantation for fellows in electrophysiology since 2007.

The referral catchment for device implantation at ACH includes Auckland District Health Board (DHB), Counties Manakau DHB, Northland DHB, and until mid-2012 Waitemata DHB and Hawke’s Bay DHB, with a combined population of approximately 1.79 million.

We have sought to describe our CRT patient population, and audit periprocedural factors (including procedure duration, procedure success, and periprocedural complications), and echocardiographic markers of response.

Materials and Methods

Patients—All patients who underwent a CRT procedure from the inception of the program at Green Lane Hospital in 2000 until April 2011 at Auckland City Hospital were included in this study. This includes de novo implantation of CRT, upgrade from pacemaker/ICD to CRT, or other CRT-related procedure (including lead remanipulation/replacement, and revision of the device site). Procedures involving solely CRT pulse generator replacement were excluded.

Data collection—Patients were identified using established data management systems employed by our device clinic. Data pertaining to the procedure and the post-procedure period were obtained via review of records held at our device clinic and present on Clinical Record Information System (CRIS). Data collected included patient demographic data, procedure-related data, clinical events occurring in the three months following the procedure (defining the periprocedural period), and any echocardiogram performed prior to and following the CRT procedure. Statistics—Results were tabulated using Microsoft Excel software, and reported using the mean±standard deviation (SD). P values were calculated using the Student’s t-test method.
Results

139 patients underwent procedures between 2000 and April 2011. 100 (72%) had new systems implanted, 37 (27%) were upgraded from either pacemaker or ICD to CRT, and 2 (1%) had lead replacement/remanipulation where the initial implantation had been performed at another centre.

In total, 159 procedures were performed on these 139 patients over this time period. In addition to the 139 procedures described above there were 19 procedures where a pacing lead was either repositioned or changed, and three procedures for evacuation of pocket haematoma that did not result in lead manipulation. Forty-eight patients underwent procedures during the first 6 years of our experience and 91 patients over the last 6 years (Figure 1).

Figure 1

The mean age of our patient population was 62±10 years (range 25–79 years), and 77% were male. The mean LVEF was 25±10%, and the aetiology for impairment of LV systolic function was ischaemic in 36%. 91% had LBBB, and the mean QRS duration was 175±33 ms (range 100–350 ms). At the time of the procedure 11% of patients were in atrial fibrillation (AF) (Table 1). 49% received CRT combined with an ICD device.
Table 1. Characteristics of the patients at baseline

<table>
<thead>
<tr>
<th>Variables</th>
<th>First six years N=48</th>
<th>Second six years N=91</th>
<th>Overall N=139</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age – years</td>
<td>65±9</td>
<td>60±10</td>
<td>62±10</td>
</tr>
<tr>
<td>Male gender – N (%)</td>
<td>39 (81)</td>
<td>68 (75)</td>
<td>107 (77)</td>
</tr>
<tr>
<td>LVEF – %</td>
<td>22±7</td>
<td>26±10</td>
<td>25±10</td>
</tr>
<tr>
<td>Ischaemia as cause for LV impairment – N (%)</td>
<td>26 (54)</td>
<td>24 (26)</td>
<td>50 (36)</td>
</tr>
<tr>
<td>LBBB present – N (%)</td>
<td>42 (88)</td>
<td>85 (93)</td>
<td>127 (91)</td>
</tr>
<tr>
<td>QRS duration – ms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrinsic QRS duration (N=102)</td>
<td>173±31</td>
<td>170±23</td>
<td>171±26</td>
</tr>
<tr>
<td>Paced QRS duration (N=37)</td>
<td>218±37</td>
<td>183±45</td>
<td>187±45</td>
</tr>
<tr>
<td>AF at time of procedure – N (%)</td>
<td>5 (10)</td>
<td>10 (11)</td>
<td>15 (11)</td>
</tr>
</tbody>
</table>

Abbreviations: LVEF—left ventricular ejection fraction. LV—left v entricle. LBBB—left bundle branch block. AF—atrial fibrillation.

Data are reported as mean±standard deviation.

The mean procedure duration was 147±60 min. The procedure duration was not different over the first 6 years of our experience at 141±53 min, when compared with our more recent 6 years of 152±64 min (p=0.76). The mean fluoroscopy time was 30±19 min which was also not different over the first 6 years of our experience at 33±21 min, when compared with our more recent 6 years of 27±17 min (P=0.93).

Failure to implant the CRT device (either as a new implantation or as an intended upgrade from pacemaker or ICD) occurred in 15 patients (11%). For all cases this was due to factors pertaining to placement of the coronary sinus lead. There was no change in the incidence of procedure success (defined as successful coronary sinus lead placement with biventricular pacing) between the first 6 years of our experience when compared to the last 6 years (88% vs 90%, p=0.54).

Seventeen patients (12%) experienced lead dislodgement, with three of these patients having two leads dislodge concurrently, and one patient where all three leads dislodged. The coronary sinus lead was the most common lead to dislodge, occurring in 10 patients (7%). Six patients had right ventricular lead dislodgement, and five had right atrial lead dislodgement. There was a trend towards a reduction in the incidence of coronary sinus lead displacement over the latter half of our experience, which was not statistically significant (8% vs 5%, p=0.64).

Eight patients experienced a significant procedure-related complication (6%), including: one death, two device and/or lead infections requiring extraction, three haematomas requiring evacuation, one pneumothorax requiring intercostal drain placement, and one cardiac tamponade. No patient experienced two or more significant complications.

The patient that died developed respiratory failure when the CRT procedure was nearing completion, necessitating endotracheal intubation and positive pressure ventilation. As a consequence of low cardiac output multi-organ failure occurred, ultimately leading to death several days later. There was not an excess of these complications during the initial half of our experience with the complication rate during our first 6 years being 4%, compared to 5% (p=0.53) over the subsequent 6 years (Figure 1).
Sixty-four of the 139 patients (46%) had echocardiograms before and after CRT implantation where LVEF and LV end diastolic dimension was reported on both studies. Echocardiographic measures of dys-synchrony are not routinely measured at our institution prior to CRT. For these 64 patients the mean LVEF at baseline was 25±11%.

At a median follow-up of 16±27 months there was a trend towards an improvement in LVEF (33±13%, p=0.50) and this was associated with a trend towards a reduction in LV end diastolic dimension (72±11 mm at baseline to 68±11 mm at follow-up, P=0.46) (Figure 2).

Thirty-three (52%) of these patients experienced an increase in LVEF of greater than 5 %. 20 (31%) patients experienced an increase in LVEF by >10 %, 13 (20%) an in LVEF increase of 5–10 %, 27 (42%) remained unchanged (LVEF within 5% of that at baseline), and 4 (6%) patients had a decrease in LVEF by greater that 5% (Figure 3).
Discussion

To the best of our knowledge this is the largest series of CRT procedures and procedure-related outcomes and complications published in New Zealand, and the second largest in Australasia. Ours is an expanding service, with a marked increase in procedures performed over the past 6 years when compared with the first 6 years of our experience.

Our patient demographics including age, QRS duration, LVEF, proportion with LBBB, and proportion with AF are similar to that of previously published large randomized trials. 36% of our patients have ischaemia as the cause for their LV dysfunction, which is lower than the 40–65% typically seen in these trials. This could potentially be due to several observations that those with a non-ischaemic cardiomyopathy are more likely to respond to CRT than those with an ischaemic aetiology, and this observation may have affected local referral.

Our rates of lead dislodgement (12%) and failure to achieve device implantation (11%) are higher than that published in large trials and meta-analysis (5–10% and 6–8% respectively), while our rate of other complications (6%) is similar. There was no statistical difference between the first and second half of our experience across procedure and fluoroscopy time, procedure success, and incidence of lead dislodgement or significant complication. As such, we demonstrate no clear evidence...
for an institutional learning curve. Such learning curves have been well documented by others,\textsuperscript{15,18} and our inability to demonstrate this is most likely attributable to several factors.

These factors include the training of fellows in CRT at our institution, which has been more common over the latter half of our experience. In addition to this, guidelines advise that a minimum of 10 CRT device implantations per implanting physician per year is necessary to maintain competency in this procedure.\textsuperscript{19} We achieved and sustained this implantation rate for the first time in 2008. Our previously low implantation rate may lead to a longer institutional learning curve than has been observed elsewhere thus evidence for this may not be observed for several more years.\textsuperscript{15,18}

Overall, our patients were full echocardiographic data was available gained improvement in left LV function as measured by LVEF and LV end diastolic dimension, though given the small number of patients involved in our series we are unable to demonstrate this with statistical significance. Over a 16-month follow-up period 52\% of our patients derived an improvement in LV ejection fraction of >5\%, while only 6\% of our patients had a reduction in LVEF by >5\%. The nature of our data is such that it is not possible to conclude whether the 42\% of those with no significant change in LVEF have not responded to CRT or whether they have derived benefit by preventing further deterioration.

This study is limited by the retrospective nature of data collection, and incomplete data relating to echocardiographic measures of LVEF and LV end diastolic dimension before and after CRT. We also acknowledge that the indications and technology for CRT have significantly evolved between 2000 and 2011 therefore limiting our ability to compare our population across this time period.

**Conclusion**

Our service compares well with large published trials in measures of procedure-related complications, though less well with respect to the incidence of procedure success and lead dislodgement. 52\% of our patients derived significant improvement in LVEF, and only 6\% of our patients had deterioration in LVEF over a 16-month follow-up.

**Competing interests:** None known.

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**Acknowledgments:** We thank Steve Withy (Team Leader, Cardiac Physiology, Auckland City Hospital) for assistance with database analysis.
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References:

Understanding medication errors in the elderly

Monica Gupta, Monika Agarwal

Abstract

Elderly are the most vulnerable population group owing to increased prevalence of chronic diseases and drug consumption. Several characteristics of aging and geriatrics medicine affect drug prescribing for elderly. Thus the selection of appropriate pharmacotherapy for elderly becomes a complex and challenging process. Association of these factors with metabolic changes predisposes the older people to suffer drug interactions and adverse drug events.

The objective of this article is to understand the concept, causes and measurement of medication errors in elderly. Various interventions to improve drug prescribing such as educational approaches, geriatric medicine services, computerised prescribing and multifaceted approaches have also been highlighted. In conclusion there is an overwhelming need for optimising the prescribing in elderly through an integrated approach involving physician, pharmacologist, pharmacist and the patient himself.

Background

A medication error can be defined as a failure in the treatment process that leads to or has the potential to lend to harm to the patient. Most of the medication errors are the result of faulty prescribing and failure in prescription writing that leads to wrong interventions. It has been found that medication errors are most likely to be made by healthcare professionals on account of several reasons. These include inexperience, lack of attention, hurry, distraction, fatigue or depression.

Medication errors deserve careful attention more so, because they are most common in very young and very old patients. Older people are rapidly increasing in number throughout the world, both in developed and developing countries. Among older age group multiple chronic and degenerative disorders are highly prevalent. Therefore knowledge of geriatric prescribing has become essential for clinicians.

The overall incidence of drug reactions in geriatric patients is estimated to be at least twice that in younger population. Despite that, there is deficiency of geriatric specialists among health care providers. In developed countries, elderly use at least three prescribed concurrently.

In developing countries, 85–90% of elderly use at least one medication daily. It has also been estimated that 30% of hospital admission in elderly patients are due to drug-related problems.

In this review, we aim to outline the causes of medication errors in elderly, focus on importance of measuring medication errors and compare various optimization tools for prescribing in elderly. Sources of data search were Google scholar, pub med and Cochrane library.
Causes of medication errors in elderly

Older people often experience multiple comorbidities for which they are prescribed multiple medications. Thus they fall prey to a prescribing cascade leading to increased risks of drug-drug, drug-disease interactions. Along with this, several characteristics of ageing and geriatric medicine affect drug prescribing for elderly. Therefore, selection of appropriate pharmacotherapy is complex as well as challenging. There are some pathophysiological peculiarities in this age group that leads to differences in pharmacokinetics and pharmacodynamics of the medications administered. In this way the prescription process becomes complex and inappropriate.

Studies have shown that almost all pharmacokinetic process (absorption, first pass metabolism, bioavailability, distribution, protein binding, renal and hepatic clearance) are altered in elderly patients. It has been found that bioavailability of enalapril, a prodrug can be affected by reduction in its first pass metabolism leading to reduction in its efficacy. Moreover, there is increased risk of adverse reactions for example drugs with high hepatic extraction ratios such as the nitrates, barbiturates and propranolol may have reduced hepatic metabolism in older adults.

Causes of medication errors in elderly are listed in Panel 1.

<table>
<thead>
<tr>
<th>Panel 1. Causes of medication errors in elderly</th>
</tr>
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<tbody>
<tr>
<td>Dispensing errors</td>
</tr>
<tr>
<td>Medication non compliance</td>
</tr>
<tr>
<td>Medication administration errors</td>
</tr>
<tr>
<td>Suboptimal prescribing</td>
</tr>
<tr>
<td>• Overuse</td>
</tr>
<tr>
<td>• Inappropriate use</td>
</tr>
<tr>
<td>• Underuse</td>
</tr>
</tbody>
</table>

In a case control study by Golden et al, it was observed that those exposed to skeletal muscle relaxants had an increased risk of fractures (OR=1.40, 95%CI 1.15–1.72). The combination of a skeletal muscle relaxant with either a long or short-acting benzodiazepine was associated with a greater risk OR 2.66, 95%CI 1.45–2.4. These findings are important because falls and related injuries are common problem for adults >65 years.

Under-prescribing has also been associated with increased morbidity, mortality and decreased quality of life especially in elderly patients. Under-treatment can be defined as omission of indicated drug therapies for the treatment or prevention of a disease. In a study by Tulner et al, it was found that one factor associated with under-treatment was polypharmacy 76.5% vs 68.8% without polypharmacy (p=0.066).

One study documented that 19% of drug-associated admissions in older people were due to therapeutic failure owing to inadequate drug therapy. Under-utilisation of
beta-blockers in older survivors of acute attack of myocardial infarction has also been associated with increase in mortality.\textsuperscript{12}

Polypharmacy is the concomitant use of multiple drugs or the administration of more medications than are clinically indicated.\textsuperscript{13} Polypharmacy is problematic for older patients because it may increase the risk of adverse drug reactions, geriatric syndromes, diminished functional status and healthcare costs.\textsuperscript{14}

Polypharmacy may also involve excessive or unnecessary medication use, rather than an arbitrary number. Studies have shown that the percentage of patients with adverse reactions increases from about 10\% when a single drug being taken to nearly 100\% when 10 drugs are taken.\textsuperscript{2} Schmader et al found that 53\% of 208 outpatients were taking drugs with no indication, 32.7\% were taking ineffective drugs and 16.8\% were taking drugs with therapeutic indication.\textsuperscript{14}

Inappropriate prescribing has been defined as prescribing of medications that has more potential risk than benefit or prescribing that does not agree with medical standards.\textsuperscript{15}

Particular drugs pose special risks top older people—e.g. increased risk of upper GI bleed with NSAIDs or prolonged sedation and increased risk of falls with long-acting benzodiazepines. Prescription of such drug is potentially inappropriate especially when safer alternatives exists.\textsuperscript{16} A list of commonly prescribed drugs causing potential adverse effects has been shown in Table 1.

**Table 1. Potentially inappropriate medications in elderly**

<table>
<thead>
<tr>
<th>Commonly prescribed drugs in elderly</th>
<th>Potential adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lorazepam</td>
<td>Sedation</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>Central nervous system (CNS) confusion</td>
</tr>
<tr>
<td>Bisacodyl</td>
<td>Bowel dysfunction</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>CNS stimulation, sleep disturbances</td>
</tr>
<tr>
<td>Naproxen</td>
<td>Gastrointestinal bleed</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Muscle relaxants</td>
<td>Anticholinergic effects</td>
</tr>
<tr>
<td>Doxazosine</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>Confusion, slurred speech</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td>Decrease renal function</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Osteoporosis</td>
</tr>
<tr>
<td>Cholinesterase inhibitors</td>
<td>Peripheral cholinomimetic effects</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Heart failure, anticholinergic effects</td>
</tr>
</tbody>
</table>

**How to measure medications errors in elderly**

The important approaches to measure inappropriate prescribing include identifying drugs to avoid using for older people, using retrospective information from databases to identify problems such as dosage range duration, drug interactive and applying explicit criteria for clinical review of medications (Table 2).
Table 2. Measurement of medication errors in elderly

<table>
<thead>
<tr>
<th>Variables</th>
<th>Explicit methods (criterion based)</th>
<th>Implicit methods (judgment based)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methods</td>
<td>Published reviews</td>
<td>Focus on individual patients</td>
</tr>
<tr>
<td></td>
<td>Expert opinions</td>
<td>Information's from patients</td>
</tr>
<tr>
<td></td>
<td>Consensus techniques</td>
<td>Published studies</td>
</tr>
<tr>
<td>Advantages</td>
<td>Consistency of approach to individual cases</td>
<td>Allow flexibility in individual patients</td>
</tr>
<tr>
<td></td>
<td>Can be adapted to computerised systems</td>
<td>Do not require problems to be prespecified</td>
</tr>
<tr>
<td></td>
<td>Can incorporate information from published literature and expert consensus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Can easily be used for educational purposes, drug utilization reviews, and epidemiological studies</td>
<td></td>
</tr>
<tr>
<td>Disadvantages</td>
<td>Do not allow flexibility in individual patients</td>
<td>Depend on knowledge, experience, and skills of healthcare professionals</td>
</tr>
<tr>
<td></td>
<td>Can produce false-positive results</td>
<td>More difficult to use consistently</td>
</tr>
<tr>
<td></td>
<td>Need problems to be prespecified</td>
<td>More difficult to measure outcomes in valid and reliable ways</td>
</tr>
<tr>
<td></td>
<td>Miss some problems that may be identified only during a full assessment of the patient</td>
<td>Time consuming</td>
</tr>
</tbody>
</table>

For a long time, Beers criteria were used in clinical practice and research to measure appropriateness of prescribing. Beers and colleagues developed a bit of explicit criteria for identifying in appropriate drugs.\(^{17}\) Take an example of benzodiazepines.

According to process measure explicit criteria, prescription of benzodiazepines is inappropriate if prescribed for insomnia in patients with history of fall and no attempt to withdraw the drug. Implicit criteria states, if patient is prescribed a long-acting benzodiazepine for insomnia for 5 years, the patient can demand discontinuation of treatment. If outcome measures are followed then explicit states, patient admitted to hospital for fall and taking long-acting benzodiazepine indicates inappropriate prescription.

According to implicit criteria, patient admitted for falls and with history of benzodiazepine or cold remedy use in previous 3 days, needs to be evaluated by clinicians for possible relation between fall and drug whether it could be prevented.

There are several advantages of explicit criteria. These indicators are developed from expert opinion, reviews and consensus techniques. Hence they take into account evidence based aspects of treatments. They can also be applied with little or no clinical judgment since they are drug or disease oriented. However they are costly to apply. They lack validity and reliability since causal link to outcome is absent. Also they do not include all factors that define health care for individuals. They do not underscore disease burden and patient preference.

In contrast, implicit criteria take into account patient rather than drug or disease. Although they are more sensitive at the same time more time consuming and less reliable. Some newer explicit measures like screening tool of older patients prescriptions have been introduced. But it is pertinent to note that both explicit and
implicit methods must be rationally combined to individualize drug treatment in elderly.

In 2011, the American Geriatric Society updated the Beers criterion for potentially inappropriate medication use in older adults. It catalogues medications that cause adverse drug events in older adults due to their pharmacologic properties and the physiologic changes of aging.\textsuperscript{18}

**Others tools to measure inappropriate medications**

- START and STOPP are newer criteria to identify potentially inappropriate medications in elderly, including drug–drug and drug–disease interactions, drugs which increase risk of falls and drugs which duplicate therapy.

- START (screening tool to alert doctors to the right treatment) is an evidence-based screening tool that can detect potential omissions in therapy of elderly patients. It includes 22 scenarios divided by physiological system (cardiovascular, respiratory, CNS, GI, locomotor and endocrine) where specific medications are recommended.\textsuperscript{19}

- STOPP (screening tools of older persons’ potentially inappropriate prescriptions) incorporates commonly encountered instances of potentially inappropriate prescribing in older people including drug-drug and drug-disease interaction.\textsuperscript{20} STOPP criteria are arranged according to relevant physiological systems for ease of use as in case of most drug formularies. Each criterion is accompanied by a concise explanation as to why the prescription is potentially inappropriate. A study which compared STOPP and Beers criteria concluded that STOPP identified significantly more PIMs than Beers criteria.\textsuperscript{20} STOPP also encourages clinicians to consider medication as a possible cause of ADEs in older people thereby avoiding potentially harmful prescribing cascades. According to the updated AGS 2012 Beers criterion implicit criterion such as STOPP/START criterion and medication appropriateness index should be used in a complimentary manner with the Beers criterion to guide clinicians in making decisions about safe medication use in older adults.\textsuperscript{18}

- An expert panel from Canada also developed a list of generally contraindicated drugs and clinically important drug-drug and drug-disease interactions.\textsuperscript{21}

- A set of investigators from the UK developed inappropriate prescribing indicators via the consensus of a multidisciplinary healthcare team of older in patients.\textsuperscript{22}

- Lipton et al developed a scale that examines six domains: no indications, improper schedule, inadequate dosage, potential drug interactions, therapeutic duplication or allergy—and allowed for the calculation of weighted score.\textsuperscript{23}

- Medication appropriateness index (MAI) developed by Samsa G et al for each of 10 criteria defined the index has operational definitions and instructions and allows three possible ratings. MAI has demonstrated good reliability, validity and feasibility in geriatric inpatients and outpatients.\textsuperscript{24}
• ACOVE (assessing care of vulnerable elders) project was the most comprehensive project involving systematic reviews of publications and expert opinions to develop a set of quality care indicators relevant to vulnerable elders.\textsuperscript{25}

**How to optimise prescribing in elderly**

Several studies have indicated that geriatric medicine service approaches, pharmacist involvement in patient care and computerised decision support can improve appropriate prescribing in elderly patients.\textsuperscript{26}

A number of interventions have been suggested to optimise prescribing such as mailed prescribing feedback and education materials, small group workshop and decision tree to manage osteoarthritis, and computerised decision support system (Panel 2). It is possible, however, that these approaches might fail because of no direct interaction with the main prescriber, low participation rate of health care professionals and environmental barriers.

<table>
<thead>
<tr>
<th>Panel 2. Approaches to improve inappropriate prescribing in elderly</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mailed prescribing feedback and education materials</td>
</tr>
<tr>
<td>Small-group workshops</td>
</tr>
<tr>
<td>Decision tree</td>
</tr>
<tr>
<td>Computerized decision support systems</td>
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<tr>
<td>Pharmacist involvement in patient care</td>
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<td>Geriatric clinic</td>
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<td>Multidisciplinary geriatric team care</td>
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<td>Multifaceted approaches that incorporate two or more distinct strategies</td>
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Importance of communications from the family has been highlighted by a case study wherein an 80-year-old patient suffered from respiratory arrest and hypoxic encephalopathy. In this case the patient was given pethidine despite a warning by his son that his father was allergic to it.\textsuperscript{27} Thus the involvement of patients or their care givers in decision-making is a seemingly promising approach. For this, the prescriber should be willing to share the prescribing decisions.

Apart from this, further multicentre studies are required to assess the effect of these approaches. What also needs to be done is development of resources for widespread diffusion of approaches. Researchers should disseminate interventions with the help of publications in academic journals. As far as clinical research is concerned the evidence from clinical trial is highly generalised since they exclude older and frail patients so the trial should be better designed to address the issue of inequity.\textsuperscript{28}

Role of information technology is very important in improving the use of drugs. Schiff and Rucker have concluded that prescribing in future will use three interacting
Reducing the rate of medication errors in geriatric patients is a challenging task, due to risk factors, lack of available data on efficacy and safety of medicine and many chronic diseases that contribute to the risks. Thus it is necessary to ensure effective, safe, cost-effective medication and good quality of life in the older citizen.

Physician should integrate clinical expertise with comprehensive knowledge about patients’ disabilities, prognosis, preferences, family support and cost of treatment. In doing so, knowledge of geriatric pharmacology plays an important role. Apart from this, there are certain ways of which we can reduce medication errors in elderly.

Firstly, patients should be counseled effectively to improve compliance with medications, regimens, side effects and adverse drug events. This can only be achieved by enhancing communication between doctor and patient. In a randomised study, a simple education intervention significantly reduced inappropriate prescribing for elderly patients.  

Secondly, physician should review his patients’ medications regularly to reduce polypharmacy and medication errors. A prospective observational study showed discrepancy between prescribed medicines and those that patients was actually taking. Ideally review and modification of a patients medication regimen should be done by a multidisciplinary team comprising a pharmacist, physician and nurse.

Thirdly, computerised alerts provide warnings to physicians using computerised order entry systems. An alert is generated by the system when there is potential drug allergy, intolerance or interaction. Moreover a medications grid should be used as a visual reminder and over the counter (OTC) agents like vitamins and minerals should be reviewed.

Fourthly, physician should describe to the patient, any foreseeable risk or benefit from the treatment. This should also include instructions regarding food, drinks, lifestyle or ‘when to call’ for example call if you develop rash, sore throat, dark urine or anything unexpected or unusual.

Finally, there is an overwhelming need for more education in geriatric prescribing through an integrated approach involving physician, pharmacologist, pharmacist and the patient himself.

Medication errors in elderly are a public health problem and must be evaluated constantly with changes in pharmacotherapeutics. It would be pertinent to develop country-specific list of medications inappropriate for elderly and include this list in national drug formularies so as to reduce their prescription and use in this age group.

Competing interests: None known.

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Numerical identity: the creation of tri-parental embryos to correct inherited mitochondrial disease

Michael Legge, Ruth Fitzgerald

Abstract

Inherited mitochondrial disorders affect between 1 in 5000 to 1 in 8000 people. These are a heterogeneous group of maternally-inherited disorders, with an array of outcomes such as heart and liver failure, defects in energy metabolism, blindness, deafness, loss of motor skills and premature death.

Recently the Human Fertilisation and Embryology Authority provided advice to the UK Government to permit the use of enucleated donated oocytes with normal (wild-type) mitochondria (a currently prohibited IVF technique) to be used as recipients of nuclear DNA from intending mothers to overcome transmission of mitochondrial disorders.

In this short communication we present the basis for this radical new IVF technology, and discuss the implications for its use both in the context of treating a group of inherited disorders and the current New Zealand IVF legislation.

The recent outcome of the national consultation by the Human Fertilisation and Embryology Authority on possible changes to the UK Human Fertilisation and Embryology Act (HFEA) to allow the use of novel technologies for mitochondrial replacement to correct inherited mitochondrial disease raises a number of issues.

The HFEA “Advice to the Government” is with the UK government to consider whether to permit the new IVF technology, which is currently banned by the HFEA (1990). In essence the advice requests that donor oocytes with normal mitochondria be used to host the nuclear DNA from women with a history of inherited mitochondrial disease.

Two techniques have been proposed. The first involves the removal of the oocyte meiotic spindle (nuclear DNA) from the donor oocyte and replacing it with the meiotic spindle from the intending mother’s oocyte (the person at risk for transmitting mitochondrial disease), thereby providing healthy mitochondria. The reconstituted oocyte is then fertilised with sperm from the intending father or donor.¹

The alternative procedure involves pro-nuclear transfer to a donor oocyte whereby the intending mother’s oocyte is fertilised using IVF and the two pro-nuclei (one male and one female) are removed and transferred into the enucleated donor zygote at the same stage of development.²

In both situations the embryos then proceed to develop unaffected by mitochondrial disease. A third procedure cytoplasmic transfer, is not considered as it uses an admixture of intending mother’s oocyte cytoplasm and donor oocyte cytoplasm, which retains a significant proportion of mutated mitochondria, and is banned in the UK.
The proposed technologies will give the resulting child DNA from both intending parents plus DNA from the donor oocyte mitochondria. In essence, creating a three-parent baby.

Currently, neither of the two proposed techniques have been approved for use in treating inherited mitochondrial disorders in humans therefore the outcomes of the new techniques are not known. However, previous research using primates indicates that the transfer of the meiotic spindle to a host enucleated oocytes produced normal offspring.

Although this indicates the relative success of the technique the primates did not have any mitochondrial disorders and therefore the influence of mutated mitochondrial DNA could not be established. However, in a subsequent publication using human oocytes from normal donors and recipients these authors demonstrated comparable normal fertilisation rates between meiotic spindle transfer oocytes and controls but 52% of the meiotic spindle transferred oocytes had abnormal pro-nuclei development. Of the remaining 48%, 62% developed to the blastocyst stage.

Later work using abnormally fertilised pro-nuclear stage human embryos whereby the abnormal pro-nuclei were replaced by normal pro-nuclei demonstrated that 22% of the manipulated embryos developed to the 8-cell stage and 8% of those with two pro-nuclei developed to the blastocyst stage and from these data it was reported that less than 2% of the ‘foreign’ mitochondria had been transferred. However, once again the oocytes used did not contain any abnormal mitochondrial DNA.

Mitochondria are cell organelles responsible for approximately 95% of the energy requirements of the cell via the biosynthesis of adenosine triphosphate (ATP), by oxidative phosphorylation. Although the majority of DNA is in the chromosomes located in the nucleus, up to 1% of DNA is located in the mitochondria.

Typically mammalian cells contain $10^3$ to $10^4$ copies of mitochondrial DNA (mtDNA), which encodes for 2rRNAs, 22 tRNAs and 13 polypeptides; the latter are all involved in the respiratory chain. All other mitochondrial proteins, including those involved with replication, transportation and translation of mtDNA are encoded by nuclear genes targeted to the mitochondria by specific transport systems thereby creating a functional interdependence between nuclear and mitochondrial genomes.

Unlike nuclear DNA, mtDNA contains no introns and no protective histones and is error prone during replication. The accumulation of mtDNA mutations is approximately 10-fold greater than those identified in nuclear DNA and are widely associated with a number of cancers.

Although 39 of the 46 sub-units of complex 1 are encoded by nuclear genes, the entry of the proteins in to mitochondria is still not fully understood for about 20 of the sub-units. Currently over 100 pathogenic point mutations and 200 deletions, insertions and rearrangements have been described with tRNA mutations accounting for approximately 60% and polypeptide subunits affected by approximately 35% mutations.

It is estimated that inherited mitochondrial disorders are the most common inherited metabolic disorders affecting between 1 in 5000 and 1 in 8000 of the general population, with some presenting as well defined clinical syndromes but others with...
unique phenotypes with variable onset and often multiple organ involvement particularly in those organs with high oxidative phosphorylation requirements.\textsuperscript{8,10}

Affected individuals are often heteroplasmic (a mixture of wild-type and mutant mtDNA), which creates a ‘threshold’ level for disease manifestation.\textsuperscript{8,11} Currently there are limited options for the diagnosis and treatment of this group of inherited diseases and reports of pharmacological agents and vitamin supplements have not made significant inroads in effective therapies.\textsuperscript{10}

Additional issues relate to the genetic counselling of this group of inherited diseases. Depending on the presentation and family history genetic counselling may need to resort to empiric risk especially when the mutation is unknown.

Where the mutation is unknown it has been proposed that the recurrence risk for children from an affected female would be in the order of 10 to 20\% and for an affected male the risk might be approximately 2\%.\textsuperscript{8}

For some of these disorders pre-implantation genetic diagnosis is possible using two of the pre-implantation embryo cells, however many of the different types of mtDNA mutations are inherited in a complex and poorly understood way causing issues relating to mutation load and difficulty in predicting the prognosis.

Recent research indicates that if the mutation load is 18\% or less then there would be a 95\% chance that the individual would not be affected.\textsuperscript{12} However, a heteroplasmic woman with this mutation load may produce embryos with mutation loads at 20 to 25\%, which would create a dilemma in decision-making and may have to rely on reproductive and family history.\textsuperscript{12}

As all human mtDNA is inherited from the mother via the oocyte (sperm mtDNA is not detectable following fertilisation and early pre-implantation development), the development of mitochondrial replacement therapies at the oocyte/zygote stages appears to offer the greatest hope for those families affected by these groups of inherited diseases.\textsuperscript{13} What then would be the issues?

First there is the issue of a three-parent child, both the HFEA and the Nuffield Bioethics Centre (UK) have indicated that the oocyte donor should remain anonymous—a decision contrary to the spirit of New Zealand’s HART legislation.

Second, although the intending mother’s nuclear DNA is transferred to a normal oocyte, mitochondria from her ‘affected’ oocyte may also be transferred with the cytoplasm surrounding either the spindle or the pro-nuclei.

What role will the intending mother’s mtDNA have in the host oocyte? Will the stress of the IVF procedures create an environment for mitochondrial fusion thereby creating hybrid mitochondria?\textsuperscript{14–16} possibly changing metabolic function and signalling cascades?

Little is understood about retrograde mitochondrial signalling to the nucleus, how important will this be in regulating mitophagy mediation in the oocyte/embryo?\textsuperscript{17,18}\ As little is known about how mitochondria segregate during cell division and tissue development, will heteroplasmic distribution of normal and mutant mitochondria still occur and present with differing disease patterns?
The ‘new’ (donor) mitochondria would be incorporated in to all cells and therefore would result in the modification of the germ-line as well as somatic cells raising an issue regarding any impact of the therapy on future generations19.

In New Zealand (as in the UK and Australia) legislation prohibits the modification of nuclear and mtDNA in oocytes, sperm and embryos as well as prohibiting somatic cell nuclear transfer (cloning). In addition, while pre-implantation human embryo research may be licensed to approved clinics in Australia under the “Research Involving Human Embryos Act (2002), current New Zealand policy is to allow research only on non-viable embryos.

Therefore, any developments and use in human reproduction would require a change in the Human Assisted Reproductive Technology Act (HART) (2004)10 in New Zealand, the HFEA (2008) in the UK and Australian legislation, which is where the HFEA is at present. In Australia, recent consideration relating to a review of both the Research Involving Human Embryos Act, (2002) and the Prohibition of Human Cloning for Reproduction Act, (2002) upheld section 13 of the latter Act in recommendation 7 in that “There should be no change to the current legislation in relation to the use of DNA from more than two persons”.20

Not withstanding the legislation, issues relating to consent should be considered, as the future child has not been involved in the treatment decision to modify its genome. Will there be issues connected to the three parent IVF and the perceived identity of the future child/children? In addition to the anonymity of the oocyte donor the HFEA and the Nuffield Bioethics Centre have proposed that the oocyte donor should have a similar status to tissue donors. How would this develop in a New Zealand context?

First, the HART Act requires donors to be identified and a register is maintained to facilitate donor identification for children born from gamete and embryo donation, enucleated oocyte donation would have to be considered in this context.

Second, the New Zealand Human Tissue Act (2008)21 does not cover reproductive tissue donations. Would this need to be amended as the UK proposal has recommended that oocyte donation be considered as a tissue donation?

Finally if the technique were used off-shore would donor oocytes from the host country be used rather than those from New Zealand adding an additional layer of complexity relating to donor identification, payment (commercial transactions for gametes and embryos is illegal in New Zealand) and future child identity.

Competing interests: Both authors declare that they have no competing interest related to this manuscript.

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Bleeding from gastrointestinal tract recurrence of non-seminomatous germ cell tumour testis, showing temporary response to gemcitabine and oxaliplatin chemotherapy

Stephanie K Prince, Garry Forgeson

Abstract

The first reported case of gemcitabine/oxaliplatin (GemOx) chemotherapy used for recurrent non-seminomatosus germ cell tumour of the testis metastatic to the gastrointestinal tract causing uncontrolled bleeding which induced a temporary response.

Testicular cancers are the most common malignancy in men aged 15–35 years and have an incidence of 0.8% of all cancers in men worldwide, with a mortality rate of 0.1%.1 A higher incidence in Australian and New Zealand men (1.2%) is described. Non-seminomatous germ cell tumours (NSGCT) account for just under half of all testicular cancers reported.

With advancements in chemotherapy disease-free survival (DFS) and overall survival (OS) rates have been improving, but the 14% of patients with poor prognosis NSGCT have a 5-year OS rate of 48%.2 Patients who relapse after second-line or high-dose chemotherapy have a poor prognosis, with uncertain benefit from further chemotherapy.

Common sites of metastatic disease include; retroperitoneal lymph nodes, lungs and brain. A rare site of metastatic deposits is the gastrointestinal (GI) tract. When this occurs it can generate symptomatic haemorrhage leading to difficult treatment decisions.

Case report

A 24-year-old Caucasian male presented with a 4-month history of left testis swelling and a short history of 12 kg weight loss, lethargy, and fever. He had no past medical history.

Computed tomography (CT) scan showed extensive metastases in both lungs, and, left sided para-aortic lymphadenopathy. Orchidectomy demonstrated mixed non-seminomatosus germ cell testicular cancer, 30% embryonal carcinoma, 70% immature teratoma. Serum tumour markers were all significantly elevated. Beta-HCG was 2800 IU/L (reference range [RR] 0–5 IU/L). Final staging was stage IIIC NSGCT of the testis.

He received bleomycin, etoposide and cisplatin chemotherapy. He relapsed at 7 months and received high dose chemotherapy. After both regimens his tumour markers normalised but had only modest reductions in the metastatic disease. Repeated cardiothoracic opinion stated that the thoracic disease was unresectable.
At further relapse after 15 months, gemcitabine and oxaliplatin (GemOx) palliative chemotherapy was accepted by the patient. Whilst being organised he presented acutely with collapse, melaena and a haemoglobin (Hb) of 37 g/L (RR for males 130–170 g/L).

CT showed increase in the size of his known disease with the development of extensive new liver and right kidney metastases. He required 2 units of blood per day to maintain his Hb.

Oesophagogastroduodenoscopy (OGD) and colonoscopy demonstrated only mucosal erosion. CT angiogram showed a small pooling of contrast in a loop of small bowel (Figure 1). A trial of subcutaneous infusion of octreotide did not reduce the bleeding.

**Figure 1. CT angiogram demonstrating pooling of contrast into the small bowel and other areas of metastases**

![CT angiogram demonstrating pooling of contrast into the small bowel and other areas of metastases](image)

A – Pooling of contrast into the small bowel; B – Para-aortic metastases; C – Metastases in the right kidney; D – Liver metastases.

Due to the likelihood of multiple lesions a capsule endoscopy was organised but whilst awaiting the results his clinical condition deteriorated with a rise in beta-HCG from 60688 to 79423 IU/L over 3 days. The surgeons felt that intervention was unlikely to be beneficial. The remaining options were chemotherapy or terminal care. His case was discussed with our multidisciplinary team and the patient who agreed to have GemOx with an initial 25% dose reduction with the risk of causing catastrophic bleeding due to chemotherapy-induced thrombocytopenia.
Thalidomide 200 mg orally was started on day 8 from the start of the GemOx chemotherapy as advised by the gastroenterologist in attempt to reduce the bleeding. The capsule endoscopy showed multiple tumour deposits in the stomach and small bowel therefore, two lesions were clipped in the stomach via OGD. On day 10 his Hb dropped by 40 g/L and his platelets fell to $49 \times 10^9/L$ (RR 150–400 $\times 10^9/L$), increase in the amount of melaena was reported.

On day 11 his platelets spontaneously rose to $80 \times 10^9/L$ then continued to rise and his Hb remained stable for 7 days without blood transfusion, his melaena ceased. His beta-HCG dropped in this time to 7136 IU/L indicating a response to the chemotherapy.

On day 20 small amounts of melaena reoccurred and his beta-HCG rose consistently to 20785 IU/L. He was offered further chemotherapy on a more intensive schedule with a greater risk of bleeding or palliative care; he chose palliative care and died 30 days after the start of chemotherapy.

**Discussion**

Cases of metastatic NSGCT to the GI tract have been reported either as an initial presenting feature or as relapsed disease. The most frequent histological type metastasising to the GI tract reported in the literature is choriocarcinoma. Other reported cases include teratoma and embryonal carcinoma.

Frequently patients with GI metastases have surgery to remove the GI lesion prior to chemotherapy. However, accurate mapping of lesions is difficult and lesions may be missed during surgery. The reported chemotherapy regimens administered whilst GI bleeding due to metastases was ongoing include; ifosfamide based, and actinomycin-D, etoposide and methotrexate. The first case presented with GI metastases and chemotherapy induced complete resolution of macroscopic tumour. The second case died due to metastases in the lung and brain. Another case treated with unspecified chemotherapy died 3 days later due to worsening haemorrhage.

On review of the literature this is the first case where GemOx and thalidomide have been used in a patient with bleeding NSGCT GI metastases.

GemOx was considered in this case, who had relapsed after two previous lines of chemotherapy, due to the results of 2 phase II trials using GemOx in relapsed NSGCT. These enrolled 28 and 35 patients respectively, with relapsed or cisplatin-refractory NSGCT. Response rates of 32 and 46% respectively were seen, 14 and 9% achieved a complete response. In the first trial 62% of patients experienced grade 3/4 neutropenia and 41% grade 3/4 thrombocytopenia. In the second trial 54% experienced grade 3 myelosuppression.

The usual indication of thalidomide in GI bleeding is for angiodysplasias or vascular malformations refractory to other treatments. There is only one reported case of thalidomide use with a non-haematological malignancy. This case was a gastric cancer causing severe bleeding resistant to tranexamic acid, etamsylate and sucralfate. Thalidomide was given at a dose of 300 mg orally a day and bleeding stopped within a week. The response appeared to be dose dependent with bleeding recurring at 100 mg per day.
This case demonstrates the difficult treatment decisions faced when managing palliative NSGCT patients with bleeding from GI metastases. The bleeding from the GI metastases reduced in concordance with the decrease in beta-HCG suggesting a response to the GemOx.

The GemOx did cause an increase in bleeding due to grade 3 thrombocytopenia for 1 day but induced a temporary tumour response which rendered the patient transfusion free for a week. It is unclear if there was any additional benefit from the use of thalidomide in our case.

Further research is required in the use of thalidomide in bleeding primary or metastatic GI cancers and optimum dosage. This is the first report of a patient receiving GemOx for this indication and gaining a temporary response.

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Bilateral plunging ranula: two case reports and a review of the literature

Steven McKenzie, Cameron Lewis

Abstract

Aim Presentation of two bilateral plunging ranula cases and then review of the plunging ranula literature to understand current concepts on aetiology, imaging for diagnosis and management.

Method A literature review using PubMed (92 papers) and Google Scholar (18 papers) has revealed only 18 cases of bilateral plunging ranulas ever published and with the addition of the two cases presented this makes a total of 20.

Results These cases are reviewed and information related to aetiology and management is presented. The cause appears to be a combination of mylohyoid dehiscence, racial predisposition and previous trauma to the mouth/face or previous oral surgery.

Conclusion Plunging ranula are a rare cause of bilateral and unilateral neck swellings but more common in Māori, Polynesian and Asian people. Their cause is multifactorial and ultrasound scan (USS) is the current investigation of choice. Management relies on excision of the sublingual gland with the cystic contents via a trans-oral approach.

The term ranula is derived from the Latin word “rana” meaning little frog and is descriptive of the blue translucent swelling in the floor of the mouth, which is said to resemble the under belly of a frog. A simple ranula is the result of extravasation (leakage) of saliva from any of the 20 ducts that arise from the sublingual gland and empty into the floor of the mouth or into the anterior portion of the submandibular duct.

Ranulas are characteristically large (3–6 cm) and form a blue, tense vesicle in the floor of the mouth. Some ranulas will attain sufficient fluid pressure to herniate through the mylohyoid muscle into the submandibular space within the neck and are termed plunging or diving ranulas. A plunging ranula usually presents as a painless fluctuant lateral neck swelling which does not change with swallowing or eating. It is most commonly centred within the submandibular space and averages 4–10 cm in size.

Bilateral plunging ranula are very rare with only 18 cases reported in the literature. A broad differential diagnosis exists for unilateral neck swellings and this also applies to bilateral swellings. Other causes of submandibular swelling that need to be considered in both the community and hospital settings include submandibular space abscess/collection from an odontogenic source, metastatic disease from cancers of the lip, skin of the face and oral cavity and tumours of the submandibular gland (50% of which are malignant). Other cystic lesions that present within this position in the neck
include dermoid cyst, cystic hygroma (lymphangioma) or haemangioma. Additionally adolescents may develop acute inflammatory lymphadenopathy from bacterial and viral infections such as glandular fever.

It has been widely suggested that the cause of a plunging ranulas is due to a congenital predisposition involving dehiscence of the mylohyoid muscle, allowing the sublingual gland to herniate into the cervical tissues, and racial predisposition.\textsuperscript{1,3,4} There is also a possible correlation with trauma to the floor of mouth and sublingual duct rupture\textsuperscript{5} or previous oral surgery.\textsuperscript{3}

A unilateral plunging ranula is a rare entity with the chances of it occurring bilaterally even less likely but should be considered by general practitioners, emergency medicine, ear nose throat and oral/maxillofacial specialists when presented with neck lumps. We describe two cases with one having had previous trauma to the mandible and both having radiological evidence of bilateral dehiscence of the mylohyoid muscle and herniation of the sublingual gland.

**Case 1**

A 20-year-old fit and well Māori man, presented with a left-sided submandibular swelling. He had sustained trauma to the left side of his face 4 days prior to presentation. Initially there was minimal swelling but was followed by a rapid left submandibular pain and swelling over the next day with extension down the lateral neck.

The swelling was firm and diffuse, extending from the left inferior border of mandible to thyroid cartilage inferiorly. Wharton’s duct was patent and he had no cervical lymphadenopathy. Bloods and a panoramic film were unremarkable.

Computed tomography (CT) neck demonstrated a multiloculated rim enhancing fluid collection around the left angle of the mandible within the submandibular space (Figure 1). The possibility of an anterior mylohyoid defect on the left side was raised. It was also noted that there was an additional plunging ranula on the right side with herniation of sublingual gland posterior to this defect on the right side. The mylohyoid defect was within the anterior two-thirds of the muscle.

**Figure 1. Axial CT showing fluid collections within bilateral submandibular spaces**
This mylohyoid defect was better demonstrated on ultrasound scan (USS) through which a small amount of sublingual gland was seen to herniate. See Figure 2.

Figure 2. Ultrasound scan of the neck showing the mylohyoid (MH) dehiscence

At this stage the decision was made to address the symptomatic left plunging ranula with aspiration, incision and drainage of the collection and then left sublingual gland excision via a trans-oral approach with removal of the cyst contents. The aspirate was positive for amylase. Histology showed focal mucous extravasation and patchy chronic inflammation of the gland.

Unfortunately 6 weeks later, he developed a sudden right-sided neck swelling. The right sublingual gland was then excised in a similar fashion to the left with access via a trans oral approach. A small part of the herniated sublingual gland was lifted from the neck below the mylohyoid muscle dehiscence. The histology report was similar to the left gland with mucous extravasation and chronic inflammation. He has remained asymptomatic in subsequent clinic reviews.

Case 2

A 21-year-old Māori man presented with a 3-day history of a painless, enlarging, fluctuant left neck swelling which he initially described starting after having a sore throat and it enlarging over the course of a day. He was otherwise well and denied any dental pain or trauma.
The left submandibular swelling was non-tender and extended down the lateral neck, measuring 10 cm in diameter. He had no intraoral signs of odontogenic infection. His floor of mouth was soft and he had neither trismus nor cervical lymphadenopathy. CT neck confirmed the presence of bilateral plunging ranulas, with the symptomatic left plunging ranula larger than the right.

He underwent aspiration, incision and drainage of the left submandibular space. The drained fluid was clear and mucoid in nature and was free of pus. Biochemical analysis revealed the "straw-coloured" fluid being mucous and saliva.

The patient made an uneventful recovery but unfortunately failed his outpatient review and has been lost to follow-up before the definitive excision of the gland was able to be offered.

Discussion and review of the literature

A literature review using PubMed (92 papers) and Google Scholar (18 papers) has revealed only 18 cases of bilateral plunging ranulas ever published and with the addition of the two cases presented this makes a total of 20.

A total of nine of the 20 cases have been reported from the Auckland region and has led to the suggestion that there is a congenital predisposition in people of Polynesian and Māori descent to have a defect in the mylohyoid muscle, which forms a muscular diaphragm in the floor of the mouth, allowing ectopic sublingual gland tissue to escape below the muscle.

All but one of the cases are from either New Zealand or the Asia-Pacific region. This aberration in the mylohyoid muscle has been described before, reported by Engel (1987) and other papers have documented this observation.

If people of Polynesian and Māori descent have a genetic predisposition to having a unilateral dehiscence in their mylohyoid muscle it is quite reasonable to assume that this sometimes occurs bilaterally, which may predispose some individuals to having bilateral plunging ranulas.

Four bilateral cases are reported in a recent large review of 77 patients by Morton et al. Their surgical findings support the theory of a dehiscence in the mylohyoid muscle in 67 of 69 operated cases. They also comment there is a strong predilection to Māori and Pacific Island people and that there may be a genetic component to this condition with Māori and Pacific Island people making up 82% of the cases.

Morton et al published a case series of 20 patients over a 9-year period in the Auckland region and 100% were Māori or Pacific Islanders. Along with other large studies most have a common racial origin as demonstrated by over 80% of all cases being of Asian descent.

Further weight to this hypothesis comes from Mahadevan and Vasan. They published a case series of 21 paediatric patients from the Auckland region over a 5-year period with 20 of them being Māori and Pacific Islanders. There were three bilateral plunging ranulas in a 6, 9 and 15 year old respectively.

Previous blunt trauma to the face/neck and previous head/neck surgery were identified as further risk factors for plunging ranula development with 9 of 21 patients having had surgical procedures such as submandibular gland excision prior to
developing the plunging ranula. Tail et al also reported six patients developing intraoral ranulas after submandibular duct transposition surgery.

There are a further 11 bilateral plunging ranula cases in the literature. All but one are from Asian countries, although a British case appears to have been the first, described by Barnard in 1991. Trauma and previous oral surgery are often mentioned in these case reports as part of the history.

The diagnosis of plunging ranula can be difficult and different techniques have been employed over the years, ranging from aspiration and examination of the cyst fluid for salivary amylase and protein content or fine needle aspiration and cytology to imaging with MRI, CT and now ultrasound scan (USS) becoming the modality of choice.

Jain et al investigated 33 cases of plunging ranulas over 4 years with high resolution USS. The potential benefits of this investigation include measuring the extent and dimensions of the plunging ranulas, confirming the cystic nature and the status of the mylohyoid muscle (100% of cases had a defect) and evaluating the sublingual gland for rupture and herniation. Patients in the study had a median age of 20 years old and USS would avoid radiation exposure to these younger patients.

Various surgical approaches to excising the plunging ranula and/or the sublingual gland have been described in the papers reviewed, through either a trans-oral or cervical approach. Some have recommended excision of the pseudo cyst also or inducing sufficient fibrosis to seal the mucous leak.

The optimum surgical treatment has been proposed by Morton and colleagues from their large South Auckland experience and relies on complete trans-oral excision of the sublingual gland and evacuation of cervical cystic contents. A trans-oral approach to the gland was the preferred method in the majority of the cases reviewed and is the technique of choice in our department.

Competing interest: None known.

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References:
A rare “mimicker” of lung malignancy

Sujith V Cherian, Elena Thampy, Subhraleena Das

Clinical presentation—A 50-year-old African-American woman was referred to our institution for workup of a right hilar mass. The patient reported that she had been evaluated 2 years prior and underwent a right upper lobar resection of a mass which turned out to be benign. At the time of presentation, she mainly complained of dyspnoea on exertion and occasional chest pain. On physical examination, she was seen to have increased fullness of her face, but oxygen saturations were 95% on room air. X-ray of the chest (Figure 1A) and CT scan of the chest (Figures 1B, 1C & 1D) are shown below. What is the diagnosis?
Answer & Discussion—Fibrosing mediastinitis. Chest CT scan showing a right hilar mass with calcification and circumferential involvement of the right main stem bronchus, pulmonary artery and superior venacava with consequent narrowing. Splenic calcifications (arrows) are present consistent with sequelae of histoplasma infection.

Proliferative fibrotic process in the mediastinum with associated airway or vascular compromise is called fibrosing mediastinitis, with morbidity and mortality related to the location and extent of fibrosis.

It is a rare disease, with a vast majority of cases (>90%) secondary to previous histoplasma infection, especially in the United States. Pathogenesis is believed to be secondary to leak of fungal antigens from lymph nodes into mediastinim causing a hypersensitivity reaction with excess fibrotic response. Diagnosis is based on clinical and radiological presentation with associated calcification.¹

There is no effective treatment. Antifungals have been found to have some potential benefit in case reports, while surgery to palliate symptoms by relieving airway and vascular compromise have been tried with variable success.²

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Bowel cancer screening in New Zealand

A recent editorial\(^1\) in the *NZMJ* contains several erroneous statements and presents a poor discussion of the options for bowel screening in New Zealand.

The statement “colonoscopy is the only test that provides prevention as well as early detection” is not true. Flexible sigmoidoscopy has also been shown to prevent bowel cancer in several clinical trials.\(^2\)\(^-\)\(^4\) For example, the United Kingdom (UK) trial achieved a 43% reduction in the incidence of bowel cancer from having one flexible sigmoidoscopy between 55 and 64 years of age.\(^4\)

Moreover, this reduced risk appears maintained 11 years after having the flexible sigmoidoscopy, suggesting the possibility of a permanent reduction in risk from the removal of adenomatous polyps. These trial results were similar to our previous estimates, based on the available research at the time, of the potential effect of flexible sigmoidoscopy screening in New Zealand.\(^5\)

A lack of qualified sigmoidoscopists and colonoscopists is recognised as a potential problem. Training in flexible sigmoidoscopy screening for non-medical staff is available overseas. If, as in the UK, general practitioners, nurses and trained technicians conducted this once-in-a-lifetime flexible sigmoidoscopy, screening could be provided with little increased demand for colonoscopy services. This is because of the one-off nature of flexible sigmoidoscopy, whereas screening by 2-yearly iFOBT between 50 and 74 years of age has a 25% chance of at least one false positive iFOBT test and consequent colonoscopy.

Are people 55 to 64 years of age willing to have a flexible sigmoidoscopy to significantly reduce their risk of dying of, or subsequently developing, bowel cancer? A study of rigid sigmoidoscopy in Dunedin, New Zealand, suggested that about 57% of Southern residents invited may participate.\(^6\) Given this earlier research, it is not surprising that surveillance colonoscopy as a screening test is well accepted by people with a family history of bowel cancer in New Zealand.\(^7\)

The editorial did not present all the screening options for bowel cancer. There are at least three main options, not two: FOBT, flexible sigmoidoscopy and colonoscopy. Failure to consider flexible sigmoidoscopy resulted in the following erroneous conclusions:

- “Population colonoscopic screening is not possible and so prevention will not occur”.
  (It is correct that population colonoscopic screening is not possible, but bowel cancer prevention can occur with flexible sigmoidoscopy.)
- “Early detection is the best that can be hoped for but even this is not currently available on a programmatic level”.
  (By using flexible sigmoidoscopy we could achieve both *prevention* and early detection.)
“New Zealanders must rely on family practitioner-based faecal occult blood screening, on accurate family histories to determine level of risk, and on prioritisation of what colonoscopy slots are available to screen those at high risk”.

(New Zealanders do not need to rely on family practitioner-based faecal occult blood screening. They can advocate strongly for one-off flexible sigmoidoscopy screening so that by 2016, when flexible sigmoidoscopy screening begins in the UK, we do not need to make excuses for why New Zealand, a country with very high incidence of bowel cancer, is not implementing the most effective proven screening method.)

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References:
Dr James Church’s response

I appreciate the letter from Drs Cox and Sneyd, making the point that flexible sigmoidoscopy is an effective method of early detection and prevention of rectal and sigmoid cancers.

The data are there to support this, and in my editorial it would have been appropriate to say that endoscopic screening in general is both diagnostic and preventive. However, as with any method of screening for colorectal cancer except colonoscopy, flexible sigmoidoscopy represents a compromise. It accepts the limitation that colon proximal to the reach of the sigmoidoscope remains uninspected, and has been likened to performing a “mammogram of one breast”.¹

In addition, the same concerns that limit colonoscopy—poor quality examinations, poor bowel preparation, discomfort, missed polyps—apply equally, if not more, to sigmoidoscopy. This may be a reason for the threefold increase in left sided interval cancers reported after flexible sigmoidoscopy compared to colonoscopy.²

Sigmoidoscopy is cheaper to perform (especially when performed by nurses), does not involve all the disadvantages of sedation, and is more suited to mass screening in high throughput clinics. Cost benefits analyses show that the relative advantage of colonoscopy over other methods of screening depends strongly on compliance with screening and with levels of cancer protection achieved in the right side of the colon.³

I am not privy to the discussions held by the New Zealand Colorectal Screening Advisory Group and so I am unaware of the reasons why they favoured immunochemical faecal blood testing as the population screening tool.⁴ However the pilot study of this programme is ongoing.

I would reiterate that identification and targeted colonoscopy screening of high-risk cases is an important aspect of population screening, whatever the underlying method for the low risk cases happens to be.

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References:

Prostate cancer screening and ethical use of limited resources

The contrasting letters by Ben Gray and Peter Zohrab highlight the complexity of allocating limited health resources. PSA screening for prostate cancer in asymptomatic men promises early and potentially life-saving detection of a serious illness. However, the evidence from trials on benefits versus harms is equivocal, the number needed to treat (screen) is large, and untargeted screening increases the likelihood of harm due to over-diagnosis and over-treatment.

We note with interest the warning provided by Erik Monasterio of the adverse effects a culture of over-diagnosis and over-treatment may have on patients and health systems generally. The effects of over-diagnosis of prostate cancer specifically, and the medicalisation of elevated risk regardless of treatment effects on length or quality of life, have also long been recognised.

Mr Zohrab calls for more clinical studies to resolve the issue, particularly those which investigate shorter screening intervals and use control arms ‘uncontaminated’ by men who have had PSA screening outside the trial. The lure of ‘perfect’ data is seductive, but difficult if not impossible to achieve in practice.

Convincing men to accept recruitment to a control arm that prohibits access to an otherwise available test will not be easy. Similarly, researchers proposing to investigate more frequent screening would have to argue that this would not unduly increase risks of harm. As alluded to above, harms from PSA screening may outweigh benefits in a wide range of scenarios.

The US Preventive Services Task Force does not consider cost or cost effectiveness in making its recommendations. The political and economic factors that make the US health system resistant to cost considerations would likely require substantially longer treatment than would be possible here.

Other countries, including New Zealand, have explicitly recognised the need for cost effectiveness, and the inevitable trade-offs this brings. We therefore support Dr Gray’s call for greater consideration of cost-effectiveness in prostate screening policy and, by implication, health interventions generally.

New Zealanders are arguably most familiar with cost-effectiveness analyses for pharmaceuticals, as exemplified by Pharmaceutical Management Agency (PHARMAC). Pharmacoeconomics applies the principles of evidence-based medicine to the population as a whole. It strives to maximise benefits from health spending, mindful that every dollar spent on one intervention imposes an opportunity cost of foregone benefits from other interventions. As Dr Gray points out, there are already large sections of the New Zealand population harmed by lack of access to needed and beneficial treatment.
Based on the foregoing, a number of steps can be followed when deciding to offer an intervention at individual, institutional or national levels:

- What is the evidence of effectiveness, both in absolute terms and relative to other interventions used or proposed for the indication?
- Does the evidence show that the benefits outweigh the harms?
- If the benefits outweigh the harms, what is the absolute gain, for example in Quality Adjusted Life Years (QALYs)?
- What are the likely costs for each QALY gained, and how does this compare to other possible uses of available resources?
- If the cost/benefit comparison is unfavourable, is there some other compelling reason -- such as lack of other treatment options, a national health priority, or reducing an undisputed health inequality -- for favouring this intervention over others?

If these criteria are met, a case for the intervention can be made, whether it is a screening test, surgical procedure, pharmaceutical or other treatment. If not, it risks unfairly favouring one group of patients at the expense of others.

To conclude by returning to the prostate, Mr Zohrab asserts that individual men and not governments should judge harms versus benefits of screening. This is illogical in the case of government funding and indeed Zohrab acknowledges that “the decision as to which costs are bearable is a political one”.

In this age of financial restraint and rapid technological change, it is irresponsible for governments to fund any intervention without considering available evidence of its linked benefits, harms, and costs.

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Editorial: dyspepsia or indigestion (part 1)


Wellington, August, 1911.

Editorial

THERE is no complaint more common than indigestion, or dyspepsia. There is hardly a human being who has not suffered from it, and there are few doctors who have very clear views in connection with its treatment. Any treatment to be scientific and efficacious must rest on a basis of etiology and classification. Who is bold enough to classify the protean forms of this disease, and to be content with his endeavours in this direction?

There are, of course, two great classes of dyspepsia, organic and functional. The former do not present any insuperable difficulties to our understanding, but when we come to deal with the functional varieties we are often hopelessly astray. The nomenclature of these functional disturbances of gastric activity varies enormously. The older division into sthenic and asthenic types had the advantage of simplicity, and a recent authority has reverted to this classification and added a third type, which he calls fermentative indigestion. We are inclined to think that in marked cases of fermentation and flatulence the nervous system, or the intestines, and not the stomach, are at fault.

A fairly good and serviceable division of functional cases of dyspepsia is—1. Acid Dyspepsia; 2. Atonic Dyspepsia; and 3. Nervous Dyspepsia, which may otherwise be called neurasthenic; but the most rational classification has been supplied by Robert Hutchison as follows:—

1. Secretary

   Excess — Hyperchlorhydria and hypersecretion.
   Defect — Hypochlorhydria and achylia.

2. Motor

   Excess — Pyloric spasm.
   Defect — Atony and motor insufficiency.

3. Sensory

   Excess — Hyperaesthesia.
   Defect — ? Anorexia.

If a specific case of functional indigestion can be placed in one of these compartments, there is a reasonable hope that treatment will be scientific in theory and successful in practice, but in the great majority of cases the symptoms overlap; for instance, hyper-secretion, hyper-aesthesia, and pyloric spasm are often present in the same case. Gastric irritation, gastric catarrh, nervous dyspepsia, and ulceration of the stomach are not easily distinguished.
In recent years we have learned something from the examination of the gastric contents; and the frequency of abdominal operations has shown that many hitherto regarded as functional are really organic, but otherwise our knowledge of the diagnosis and treatment of indigestion has not very greatly advanced since the time of Dr. Beaumont and his patient, Alexis St. Martin; the Canadian soldier.
Glucose levels and risk of dementia

Diabetes is a risk factor for dementia. It is unknown whether higher glucose levels increase the risk of dementia in people without diabetes.

This prospective study involved 2067 subjects who did not have dementia (mean age 76 years). 232 were diabetic and 1835 were not diabetic. 60% of the subjects were women.

During a median follow-up of 6.8 years, dementia developed in 524 participants (74 with diabetes and 450 without). Thus 32% of the diabetic subjects developed dementia versus 24% of the non-diabetic group. Among participants without diabetes, higher average glucose levels within the preceding 5 years were related to an increased risk of dementia (P=0.01).

In the diabetic cohort higher average glucose levels were also related to an increased risk of dementia (P=0.002). The researchers conclude that “our results suggest that higher glucose levels may be a risk factor for dementia, even among persons without diabetes.”


Severe hypoglycaemia and cardiovascular disease

This meta-analysis sets out to answer the question of whether severe hypoglycaemia is associated with risk of cardiovascular disease in people with type 2 diabetes. Severe hypoglycaemia is defined as at least one episode that impaired level of consciousness or required medical intervention.

Six studies involving more than 900,000 subjects were included. The primary outcomes were cardiovascular events and this meta-analysis showed a strong association between severe hypoglycaemia and a higher risk of cardiovascular disease—relative risk 2.05 and P<0.001.

The researchers acknowledge that comorbid severe disease is also relevant. They conclude that “a bias analysis indicates that the observed association between severe hypoglycaemia and cardiovascular disease may not be entirely due to confounding by comorbid severe illness.


Intermittent pneumatic compression in reduction of risk of deep vein thrombosis in patients who have had a stroke

Venous thromboembolism has been detected in 20–42% of patients in hospital who have had a stroke. Graduated compression stockings have been shown to be ineffective and may damage the skin. Anticoagulants are effective but potentially dangerous.
In surgical patients, intermittent pneumatic compression (IPC) reduces the risk of deep vein thrombosis (DVT), but no reliable evidence exists about its effectiveness in patients who have had a stroke.

This report concerns a multicentre randomised trial from 94 centres in the UK. 2876 immobilised stroke patients with a median age of 76 years were randomised to IPC or no IPC. The primary outcome was a DVT in the proximal leg veins within 30 days. There was an absolute reduction in the risk of 3.6% in the IPC group. The overall risk of DVT in the IPC group was 0.65 (P=0.001). Skin breaks occurred in 3% of the IPC group and 1% of those without IPC.

Stephen Paul Dinniss


Born in Palmerston North, New Zealand, Steve remained passionately Kiwi throughout his life.

He studied medicine in Otago University, Dunedin and completed house jobs in Wellington and Masterton, where he met his future wife, Kelly.

Steve began psychiatry training in Cheltenham and Gloucester on their return to the UK in 1998. He moved to Plymouth as a Specialist Registrar in Psychiatry in 2003. In between, he enjoyed several travel career breaks in Asia and Africa with Kelly.

As a Consultant he worked with both Home Treatment and latterly Adult Community teams in Plymouth.

He championed high-quality services for psychiatric patients and was a highly respected colleague. He led Undergraduate Psychiatry training in Plymouth for the Peninsular Medical School and was an enthusiastic MRCPsych Course lead.

His passion for training others in psychiatry was undoubted and he even allowed filming of his final operation to provide teaching material for future surgeons. He approached his health problems with a fortitude and resilience admired by all.

Steve enjoyed sports both as a participant and spectator. He regularly met with friends and colleagues to play cricket, rugby and badminton. He was able to attend the London 2012 Olympics with family, with which they were all thrilled.

He will be remembered as a vital, intelligent and passionate psychiatrist, a loyal colleague and above all a friend. He leaves his wife, Kelly and two children, Sam and Emma.

Sara Cunningham, Mike Cooper, Naresh Buttan, and David Bickerton (Consultant Psychiatrists, Plymouth, UK) compiled this obituary.