THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

Hospital admissions for people with diagnosed diabetes: challenges for diabetes prevention and management programmes
Gary Jackson, Brandon Orr Walker, James Smith, Dean Papa, Adrian Field

New Zealand is facing a growing tide of Type 2 diabetes, fuelled by rising obesity levels. Counties Manukau District Health Board (DHB) is at the forefront of this growth, as shown by this study. Thirteen percent of all people hospitalised in Counties Manukau had diabetes (1 in 5 aged 45+), with Pacific, Indian, and Māori people having the highest rates. Around 1 in 5 people with diagnosed diabetes will be admitted to hospital in any one year. Counties Manukau DHB is attempting to reduce the onset of new cases through preventive programmes such as *Lets Beat Diabetes*, while at the same time coping with the rising costs of care through its Chronic Care Management programme and hospital services.

Hospital expenditure on treating complications of diabetes and the potential for deferring complications in Canterbury, New Zealand
Ian Sheerin

There is considerable expenditure on potentially avoidable hospital admissions for treating the complications of diabetes. However, diabetes is often under-reported, particularly if it is a secondary diagnosis contributing to or causing a health complication that necessitates a hospital admission. There has been a lack of information on these costs of potentially avoidable hospital admissions related to diabetes. This study found that in the Canterbury District Health Board, in 2005/06, the total costs of avoidable hospital admissions where diabetes was a primary or secondary diagnosis amounted to $10.1 million and 9511 days stay in hospital. The major categories were cardiovascular disease, particularly angina, myocardial infarction, and heart failure.

Prevalence of undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose among Māori in Te Wai o Rona: Diabetes Prevention Strategy
David Simmons, Elaine Rush, Nic Crook

Te Wai o Rona: Diabetes Prevention Strategy recruited 4269 Maori without known diabetes to enter into a trial of lifestyle change to prevent diabetes. At the beginning of the study, the proportion with undiagnosed diabetes after adjusting for age was 6.5% among men and 4.2% among women, with a further 13.9% and 12.7% with pre-diabetes respectively. Among the 29.4% of women and 28.4% of men who were very obese, over one-third had undiagnosed diabetes or pre-diabetes. There were no differences between rural and urban Maori. Even after adjusting for age, gender and obesity, those with a community services card were 33% more likely to have diabetes or pre-diabetes. A high proportion of Maori with diabetes and pre-diabetes remain

undiagnosed, and therefore forego treatment to prevent subsequent premature death, cardiovascular disease and diabetes related damage.

The outcomes of transrectal ultrasound guided biopsy of the prostate in a New Zealand population
Andrew R Lienert, Peter J Davidson, J Elisabeth Wells

4316 biopsies were available for analysis. Biopsy was positive in 54.4%. Nomograms are formulated to help inform New Zealand men how likely they are to have a positive TRUS prostate biopsy and also how likely they are to have a higher grade cancer detected by TRUS prostate biopsy. Age-adjusted reference ranges did not improve prediction of cancer in this population, but the use of PSA density may enhance prostate cancer diagnosis.

Accuracy of prenatal diagnosis in a tertiary fetal medicine unit
Pippa M Kyle, Peter Coghlan, Jeannie Matthews, Rex de Ryke, Rosemary Reid

The aim of the study was to establish the accuracy of fetal diagnosis prior to birth in a tertiary referral fetal medicine unit by comparing those diagnoses made before birth with diagnoses around birth until discharge, or following fetal or neonatal loss. All cases seen in the Fetal Medicine Unit seen over 18 months were collected and sorted according to diagnosis. 681 cases seen which accounted for 1219 visits. 198 were classified before birth as a major abnormality, 46 cases minor, 56 with raised nuchal translucency, and 381 no abnormality. Follow-up rates were high; 99.7% for all liveborns. Of the liveborns, 93.6% of the before birth diagnoses were confirmed, 5.1% resolved (predominantly soft markers), and 1.3% resulted in an additional major abnormality that had a significant clinical effect. *Conclusion:* Accuracy of prenatal diagnosis in a tertiary fetal medicine unit is high. However, parents and staff need to be aware that not all abnormalities will be detected before birth, but inaccurate diagnosis is uncommon.

Obstructing the goal? Hospitalisation for netball injury in New Zealand 2000–2005
Pam Smartt, David Chalmers

Netball is the most popular game for New Zealand girls and women. Records of patients discharged from hospital in the period 2000–2005 were searched for netball injury cases. Age, ethnicity, body region, nature/severity of injury, and medical procedure were studied. 1126 cases were identified from the records; 81% were female. The average age was 29 years (5–82 years); 26% of cases were Māori.. Forearm fractures predominated in the 0–14 year age-group and Achilles tendon injury in the 15+ age-groups. In conclusion (a) the average age of hospitalised netball injury cases may be increasing, (b) forearm fractures in young netball players are a cause for concern, (c) surgical repair of Achilles tendon injuries appears to have increased, and (d) the indoor version of the game and male players may be important targets for injury prevention.

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Insights into future directions for diabetes management and prevention

Paul Chin, Helen Lunt

We live at a time when our health system is focussed on sustainability, yet recent data show and predict that the incidence of diabetes in New Zealand continues to rise.[1,2] Over the next decade, the diabetes epidemic will be driven in part by demographic changes—thus even if there is a beneficial change in our lifestyle, the number of people with diabetes will continue to increase.[1]

The rising impact of diabetes is recognised by most specialities and disciplines, usually as a comorbidity that has to be considered within the broader context of the patient's overall management plan. Diabetes and its complications are potentially preventable yet diabetes is taking an increasing slice of the health dollar, with a recent estimate of around NZ1.77 billion dollars by 2021 for Type 2 diabetes alone,[3] reducing the resources available to other areas. Research that offers insights into how best to manage some aspects of diabetes at a systems level, is therefore of relevance to all health professionals.

This issue of the *New Zealand Medical Journal* includes two papers that give a partial estimation of the cost associated with diabetes management.[4,5] Both papers include some discussion about approaches that might result in a reduction in this cost. Although the methodologies used differ between the two papers, their final conclusions are similar.

An estimation of the partial costs of admission for a patient with diabetes to a Canterbury DHB hospital (Sheerin) was just over $1000 per bed day.[4] The cost of a diabetes related stay for a Counties Manukau patient (Jackson et al) was conservatively estimated at $1858 per admission.[5] Sheerin's paper focuses on medical reasons for admission and concludes that a high proportion of admissions are theoretically preventable, for example those associated with microvascular and cardiovascular disease and acute metabolic decompensation.

Jackson et al focussed on differences in admission rates between ethic groups. Middle-aged Māori and Pacific patients with diabetes are most likely to be admitted. Jackson et al also compared overall annual healthcare costs of patients who were admitted to hospital during 2007, and found that those with diabetes were over four times more costly than those without. Diabetes admissions are costly, are increasing, and a proportion are theoretically preventable.

Another paper in this issue of the New Zealand Medical Journal focuses on the problem of marketing of unhealthy foods to children (Shaw),[6] and a fourth paper (by Simmons et al) discusses the prevalence of undiagnosed dysglycaemia in adult Māori residents in the mid-North Island.[7]

Shaw presents evidence for the association between obesity and the marketing of unhealthy food, especially in relation to marketing to children. Taking a population-

based approach to intervention, she argues that restricting television advertising of unhealthy food may result in a BMI change of 0.17 kg/m^2. Whilst this change is small, it represents one of the most cost-effective interventions available to government in reducing the burden of obesity. She then proposes government-driven regulatory change to minimise the adverse effects of advertising of unhealthy foods.

Simmons et al, using data from Te Wai o Rona: Diabetes Prevention Strategy, found that over 17% of those residents who were recruited and screened had either undiagnosed diabetes, impaired glucose tolerance, or impaired fasting glucose. The cohort is thought to represented approximately 13% of local Māori adults. Despite this caveat, a significant opportunity for improving Māori health has clearly been identified. Better case-finding will lead to an increased number of Māori with dysglycaemia requiring intervention, with associated resource implications.

Over the last few years, several changes have been implemented in diabetes prevention and management. The 'free' diabetes annual review programme (*Get Checked*) introduced into primary care in June 2000, is now embedded within the New Zealand health system.

Nationally, patient uptake is improving but remains suboptimal at 70% in 2007,[8] but there is ongoing debate and research around improving these participation rates. Introduction of the free annual check was associated with a shift in diabetes service delivery from secondary care to primary care and this has relieved some of the capacity related issues within ambulatory secondary care.

Some recent initiatives have aimed at lifestyle change at the population level and include HEHA (Healthy Eating Healthy Action), but the immediate impact of population based initiatives may be more difficult to measure than are changes in delivery in personal health. Advances in information systems promised an array of changes, some of which are now being realised. For example, the large scale data collection and data linkage incorporated into the analyses by Sheerin and Jackson would have been difficult to undertake a decade ago.

The topics raised by these four papers extend the debate about how best to manage diabetes in the future and, by extrapolation, how best to manage other chronic diseases. Where should we focus our future health investments? How much investment should go towards primary prevention—i.e. preventing diabetes through lifestyle change in conjunction with public health messages? Should public health messages be generic or should they have a diabetes specific focus and should they be incorporated into a systems wide strategy such as Counties Manukau's '*Let's Beat Diabetes*' programme?

Should there be greater emphasis on secondary prevention, for example through schemes aimed at increasing the uptake of the free diabetes annual check? Should there be more investment in strategies aimed at reducing costly diabetes related inpatient bed days? This might include a focus on facilitating early discharge, rather than emphasising a reduction in admissions that might in theory be preventable, but which do not always seem preventable in practice.

New strategies and goals will require better interaction and communication between primary and secondary care but it is unclear how this is best achieved. For example, should diabetes specialists play a more active role in the community, developing

teams across the health sectors?[9] Better health information systems will become integral to many of these changes. The establishment of a national diabetes register has been recommended as a priority.[10] This would allow for better interdisciplinary communication and clinical monitoring. Some promise has been shown around data linkage between primary and secondary care[11] and it remains to be seen if current information systems are sufficiently mature to pursue the option of a high-quality, low-maintenance national diabetes register.

Clearly, all of the above issues are inter-related and changes to the current model of diabetes care must be played out within the complexities of the overall health system. Change is unavoidable and is driven by ongoing capacity and cost constraints. We should thank the authors of the papers discussed above, for highlighting many of the issues that inform this debate.

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Return of the great pretender: ectopic pregnancy

John Short

Many years ago, when I was a junior registrar, a senior colleague frequently referred to ectopic pregnancy as "the great pretender". He would always go on to elaborate- it is a condition that is sometimes difficult to diagnose, often unpredictable, and (most importantly of all) one should always be vigilant. Recent years may have seen great changes in the diagnosis and management of ectopic pregnancy but, as Drs Lee and Lamaro illustrate in their case report in this issue of the *Journal*,[1] my old mentor's message is as pertinent as ever.

In their report, Lee and Lamaro refer to the latest confidential enquiries into maternal death in the United Kingdom (CEMD), which covers the years 2003–2005 and should be essential reading for all health professionals. This highlights the potential dangers of ectopic pregnancy and reports 10 deaths over the 3-year period. The report's authors conclude that 7 of these deaths were associated with substandard care, which suggests they were potentially avoidable. Careful attention to the detail of this report also reveals that mortality from ectopic pregnancy remained virtually unchanged from 1991–2005.[2] This is something I find quite fascinating because over these 15 years the way ectopic pregnancy is diagnosed changed immensely.

For several years the combination of transvaginal ultrasound findings and serum quantitative human chorionic gonadotrophin (hCG) measurement, combined in diagnostic pathways and algorithms, has been the cornerstone of ectopic pregnancy diagnosis. This combination has led to a vast improvement in the accuracy of ectopic pregnancy diagnosis and has certainly led to a significant reduction in unnecessary intervention, compared with clinical diagnosis previously relied upon.[3] Together with increased awareness of early pregnancy problems, the widespread availability of these tests has also led to earlier diagnosis in most cases.

It seems plausible that improvements in the speed, accuracy, and reliability of diagnosis should be associated with a significant reduction in mortality from this condition. Why then the minimal impact on mortality from ectopic pregnancy and why do potentially avoidable deaths still occur?

The answer to this question may be that ectopic pregnancy is still "the great pretender". Avoidable mortality from ectopic pregnancy occurs because it is a condition that is sometimes difficult to diagnose, often unpredictable, and most importantly doctors forget to be vigilant. Earlier (and easier) diagnosis and the benign presentation of many ectopic pregnancies may promote complacency, and the successful use of diagnostic algorithms based on simple ultrasound and biochemical findings means that clinicians rarely need to use basic clinical skills and judgement anymore.

Of course clinical diagnosis of ectopic pregnancy is extremely unreliable,[4] and was associated with high rates of unnecessary intervention in the past. At the same time, many women would only present with symptoms at the time of tubal rupture.

However, there are also major limitations with the present day approach to diagnosing ectopic pregnancies.

hCG was introduced into practice to help clinicians further evaluate what is now termed "pregnancy of unknown location" (PUL), or where an ultrasound scan fails to confirm an intrauterine pregnancy, in women with minimal or no symptoms.[5] The value of hCG in this situation is based upon the correlation between absolute levels of hCG and the visibility of an intrauterine gestation at ultrasound, and the fact that it rises or falls predictably in the presence of ongoing intrauterine pregnancies or miscarriages respectively.

In theory, the variation of hCG over time in the presence of ectopic pregnancies does not to follow these patterns, thus serial measurements are helpful if initial diagnostic testing is equivocal.[6,7] Unfortunately, the behaviour of ectopic pregnancy is not always so easy to predict. The hCG profile in women with ectopic pregnancy can mimic that of an intrauterine pregnancy or a complete miscarriage in approximately 29% of cases.[7]

In many cases, multiple serial measurements of hCG or repeated scans are required prior to definitive diagnosis, leading to delays. A low or falling HCG is certainly no guarantee that an ectopic pregnancy will not become clinically significant.[1] Other problems in diagnosing ectopic pregnancy include atypical presentations, which are not uncommon and can easily be misdiagnosed. Also, a significant proportion of women with ectopic pregnancy with have clinical features identical to complete miscarriage, including the passage of tissue per vagina, which can be falsely reassuring and which underlies the need for histological confirmation of products of conception or strict HCG follow up.[8]

I suspect the major problem is that modern diagnostic methods, particularly since the introduction of hCG, have been so successful that the value of clinical judgement has almost been forgotten altogether. Anecdotally, there now appears to be great reluctance to act on clinical grounds alone and to consider diagnostic laparoscopy, with a preference towards repeated ultrasounds and hCG testing, even in the presence of significant symptoms. Some even go so far as to say laparoscopy is now obsolete.[3] Whilst this clearly prevents unnecessary intervention in some women, it may expose others to the greater risks of ruptured ectopic pregnancy.

Any diagnostic process is reliant upon someone interpreting the available information properly and responding appropriately. In the case of ectopic pregnancy, this sometimes requires an understanding of its unpredictable nature, clinical judgement, and a degree of vigilance. The authors of the last CEMD report describe "a recurring theme in this report of medical staff disregarding important, basic clinical signs" and failures to recognise atypical presentations. They also state "laparoscopy or laparotomy should be undertaken without delay if there are clinical signs suggestive of tubal rupture".[2]

A review of the literature would support the view that perhaps too much emphasis is placed on ultrasound, hCG, and diagnostic algorithms, with not enough emphasis on clinical judgement. A Medline search using combinations of "ectopic pregnancy" with "ultrasound" and/or "hcg" reveals over 1000 publications between 1991 and

2005. A search using the keywords “ectopic pregnancy” and “clinical diagnosis” reveals just 20 publications over the same time period.

Obviously, mortality from ectopic pregnancy is still rare. Based on the current UK figures we should expect about 1 death from ectopic pregnancy every 5 years in New Zealand. Yet for every death there may be many “near misses”. Cases such as that described by Lee and Lamaro, where women survive despite aspects of their care being substandard, and the lessons from CEMD remain just as important.

Unfortunately, it is likely that ectopic pregnancy will always be associated with maternal mortality. However, prompt accurate diagnosis should prevent those avoidable deaths. In future, the diagnosis of ectopic pregnancy of ectopic pregnancy should become even easier and quicker for most women. It now possible to diagnose most ectopic pregnancies with a high degree of accuracy from ultrasound alone at the time of first presentation, even in the presence of low hCG levels.[9–11] Thus hCG measurement may start to become increasingly unnecessary for diagnosis, as laparoscopy has done. However, to prevent avoidable deaths and “near misses” the diagnostic process will still need to include old-fashioned clinical judgement.

Clinicians must still be aware of the potential difficulties in making the diagnosis and the unpredictability of the condition. But most of all, clinicians must still be vigilant.

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Hospital admissions for people with diagnosed diabetes: challenges for diabetes prevention and management programmes

Gary Jackson, Brandon Orr Walker, James Smith, Dean Papa, Adrian Field

Abstract

Aim To identify the extent to which people with diabetes are admitted as inpatients to Counties Manukau District Health Board (CMDHB) hospitals compared to all New Zealand, and the costs of these admissions.

Method Search of National Minimum Dataset for inpatient public hospital discharges with any mention of Type 1 or Type 2 diabetes from 1996–2007.

Results Overall 13% of all CMDHB inpatient discharges mentioned diabetes in 2007 compared with 12% nationally. There were differences by ethnicity among those discharges aged 45 to 64 years, with Pacific (38%), Indian (31%) and Māori (31%) being more than three times as likely as European/Other people (10%) to experience a diabetes-recorded admission. There was a substantial increase in the rate of admission per 1000 where diabetes was recorded over 1996-2007, with Māori (31%) and Pacific (52%) people aged 45–64 years showing the largest increases. Findings from Counties Manukau tended to be comparable with total New Zealand data in terms of the percentage of people with diabetes who were inpatients in 2007, but relatively more inpatients in Counties Manukau had diabetes recorded (consistent with its high population of people with diabetes). Costs of admissions, laboratory investigations, and pharmaceuticals for people with diabetes were estimated to be $66 million per annum higher than for people without diabetes, in Counties Manukau alone.

Conclusion The findings give support to balanced system-wide strategies (such as the *Let's Beat Diabetes* programme in Counties Manukau) that focus on both upstream (prevention) and downstream (management and coordinated care) responses to diabetes. The study points to a long-term and significant capacity challenge for secondary care services to respond to the forecast growth in the incidence of Type 2 diabetes, particularly in Counties Manukau. The findings also pose challenges for transfer of care to primary care providers after a hospital episode and the flow of information between care settings.

The rate of obesity and Type 2 diabetes in the New Zealand population has been rising sharply, in common with most Western countries, and is expected to rise further.[1,2] Type 2 diabetes is a major driver of health-sector costs within both primary and secondary care, and is associated with increased cardiovascular disease, kidney disease, stroke, lower limb ulcers, and retinal damage.

Historically, diabetes services were the responsibility of specialist diabetes units that supported all people detected as having the disease. With the rapid growth of Type 2 diabetes, this model became unsustainable and diabetes management has become an

important dimension of primary health care, together with other chronic diseases. Specialist units have become more focused on treating diabetes and its complications at more advanced stages, and in supporting primary health care in diabetes management.

Counties Manukau District Health Board (CMDHB) is one of the key frontlines in efforts to prevent and manage diabetes in New Zealand. Within CMDHB it is estimated that around 27,000 people in the district have diagnosed diabetes, representing 8.3% of the population aged 15 years and over. The age- and sex-standardised prevalence of diabetes in Counties Manukau is estimated to be 15% in Pacific peoples, 10% in Māori, 8% in Asian groups (largely made up of Indian and Chinese ethnicities), and 5% in those of European/Other ethnicities.[3]

The 2006/07 New Zealand Health Survey has similar estimates.[4] Extrapolating from the Auckland Heart and Health Study[5] up to a further 9000 people are estimated to have diabetes in the district, but are as yet undiagnosed. Recent estimates from CMDHB indicate that 14% of the national diagnosed diabetic population live in Counties Manukau, although the area has only 11% of the total national population.[3]

If New Zealand is to effectively confront the diabetes epidemic, then Counties Manukau will be a pivotal area for action. Significant populations in the district are at high risk of diabetes: 38% of the national Pacific population, 12% of the national Māori population, and 34% of New Zealanders living in the most deprived areas reside in Counties Manukau.[6]

The scale of the Type 2 diabetes epidemic in Counties Manukau and elsewhere in New Zealand poses considerable challenges for health planners and frontline service delivery. With multiple contributory factors and disease development that occurs over decades, confronting diabetes requires responses that extend beyond traditional health service areas of activity.

Let's Beat Diabetes (LBD) is a district-wide programme for Counties Manukau, aimed at long-term, sustainable change to prevent or delay the onset of Type 2 diabetes, slow disease progression, and increase the quality of life for people with the disease.[6,7] The programme has a broad set of community and health-sector partnerships, working within a 20-year horizon.

LBD has a system-wide focus, working across 10 areas of activity, including community-based programmes, social marketing, working with the food industry, supporting primary care, and improving service integration for advanced disease.[6,7] It is linked with the nationwide *Healthy Eating Healthy Action* (HEHA) programme.

Of particular note in the context of this study is LBD's support to the Chronic Care Management (CCM) programme in primary health care, and its focus on improving coordination across primary and secondary care. CCM supports community-based structured management of people with advanced and complicated diabetes, as well as cardiovascular disease, congestive heart failure, chronic obstructive pulmonary disease, and depression.

By November 2008, 8872 patients were enrolled in diabetes CCM from 88 participating practices, covering all 9 PHOs in Counties Manukau. An evaluation of the programme found statistically and clinically significant improvements over time.

In the diabetes module, the mean HbA1c levels of those patients followed for 5 years improved from 9.0% on entry to 8.4% at their 5-year review. This result is comparable to international studies of such programmes. Declines were also recorded for blood pressure, total cholesterol, and LDL cholesterol.[8]

Although an increasingly clearer picture is emerging of the prevalence of diabetes and its management (at least for Counties Manukau), the scale of the burden and potential opportunities for improving management of diagnosed diabetes across care settings remain important areas for exploration. This study sought to understand the extent to which diabetes is being revealed through inpatient hospital admissions, as a means of highlighting the scale of the challenge and providing pointers to potential strategic and service responses.

Method

The National Minimum Data Set (NMDS) was searched for all public hospital discharges with any mention of diabetes (either Type 1 or Type 2 diabetes) in either the principal or secondary diagnosis fields. Note that the admission is not necessarily for diabetes itself; simply that diabetes is mentioned on the patient record. Being coded on the record means that diabetes played some part in the care of that patient; either being treated in hospital or affecting the conditions being treated. For diabetes, it is less often the case that the disease itself necessitates hospital care, rather the many conditions that are complications of diabetes, such as renal disease and heart disease.

Only medical and surgical discharges were included; maternity, mental health, and health of older people services were excluded. Although gestational diabetes is an increasing problem, its coding in the datasets was not directly comparable. For mental health patients (who tend to be relatively young), approximately 6% had diabetes codes on their records; this proportion has been unchanged over the past 5 years. ACC purchased services and private hospital discharges were also not included.

Admission criteria for health of older people services differ markedly across New Zealand, and were excluded from analysis, although acute hospital admissions which led to health of older person admission are included in the analysis.

The years 1996–2007 were chosen. Earlier than 1996 the ethnicity recording was less robust and comorbidity recording was less emphasised. Indeed prior to 1995, only three secondary diagnoses could be recorded; since then there can effectively be an unlimited number. Changing coding standards from 1996 onwards, as New Zealand adopted the Australian coding system, led to an increasing number of secondary codes being included in the record. By the time of the change to ICD10 in 2000 this migration of coding standards was essentially complete.

The data were examined by ethnicity and age group, and the Counties Manukau population was compared with all New Zealand. Discharge rates were also compared with available figures from the United States.[9] Discharges were divided into daycases and inpatients (i.e. at least an overnight stay). Day case recording is variable around New Zealand in relation to patients treated and discharged from emergency departments. Although coding was stable for Counties Manukau over the time period under analysis, owing to national variability in recording, the analysis was restricted to inpatient data. Ethnicity is presented as prioritised, each person being assigned one group in the order shown in the tables.

Community laboratory and pharmaceutical claim data was used in conjunction with the hospital data to derive estimates of the total number of people with diagnosed diabetes in the population.[3] This enabled an estimate of the proportion of all people with diagnosed diabetes having an acute hospitalisation. It also allowed reimbursement costs to be aggregated for diabetes and non-diabetes cases; an average was derived for each population group using 2007 data.

Apportioned costs were standardised by age and sex. Costweights for NMDS hospitalisation data were aggregated for diabetes and non-diabetes cases, applying an estimate of $3740 per WIES (Weighted Inlier Equivalent Separation) for 2007/08 to arrive at total estimated costs; these were used as approximations of true cost and averaged by population group.[3]

Results

Overall, 13% of 45,970 medical-surgical inpatient discharges for Counties Manukau DHB residents had diabetes mentioned in 2007, compared to 12% of 388,093 nationally (Tables 1 and 2). The percentage of inpatient discharges with any mention of diabetes in both Counties Manukau and New Zealand increased with age, in line with the known prevalence of diabetes. The proportion of discharges with diabetes recorded climbed from 5% of 15–44 year olds, to peak at 26% among 65–74 year olds. Over 1 in 5 discharges in CMDHB in those aged 45+ had diabetes recorded—21% compared with 17% for all New Zealand.

In the 45–64 years age group (where premature mortality due to complications of diabetes is a particular concern), there were significant differences by ethnicity, with discharges by Pacific (38%), Indian (31%), and Māori (31%) being more than twice as likely as European/Other people (10%) to have diabetes recorded. People of Chinese or Other Asian ethnicities also had relatively low risks (13% and 11% respectively). Figures for New Zealand are comparable, with Pacific and Indian people similarly high.

Table 1. Percentage of inpatient hospitalisations with diabetes recorded (Counties Manukau DHB, 2007)

Ethnicity	Age 0–14	15–44	45–64	65–74	75+	Total
Māori	0.3%	6%	31%	41%	29%	14%
Pacific*	0.4%	6%	38%	46%	31%	17%
Chinese	0.0%	1%	13%	15%	26%	10%
Indian	0.3%	6%	31%	45%	34%	18%
Other Asian	0.7%	2%	11%	37%	17%	9%
European/Other	2.0%	3%	10%	16%	14%	10%
Total	**0.7%**	**5%**	**22%**	**26%**	**17%**	**13%**

Public hospitals, at least overnight stay, medical-surgical only; *Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Table 2. Percentage of inpatient hospitalisations with diabetes recorded (New Zealand, 2007)

Ethnicity	Age 0–14	15–44	45–64	65–74	75+	Total
Māori	0.8%	6%	28%	37%	30%	14%
Pacific	0.6%	7%	37%	46%	35%	18%
Chinese	0.1%	1%	14%	21%	29%	12%
Indian	0.4%	7%	33%	48%	36%	20%
Other Asian	0.3%	2%	16%	35%	30%	10%
European/Other	1.8%	3%	10%	17%	15%	11%
Total	**1.3%**	**4%**	**16%**	**21%**	**16%**	**12%**

Public hospitals, at least overnight stay, medical-surgical only.

Comparing the discharge numbers with the diagnosed diabetes numbers, the proportion of people with diabetes who were admitted to hospital in 2007 can be estimated (Table 3). Of note is that 18% of all people with diagnosed diabetes in

CMDHB had a hospital admission (staying at least overnight) in 2007—nearly 1 in 5. Rates climbed with age to 28% for the 75 years and over age group, and were higher for Māori (24%). Of the 3100 Māori with diagnosed diabetes aged 45 years and over, one quarter had an inpatient admission in 2007.

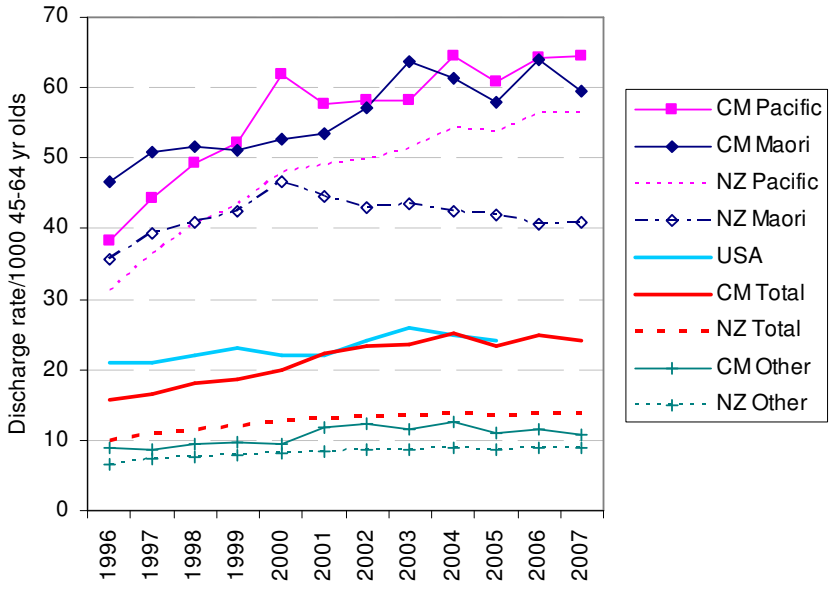
Table 3. Percentage of people with diagnosed diabetes admitted to hospital (Counties Manukau DHB, 2007)

Ethnicity	Age 0–14	15–44	45–64	65–74	75+	Total
Māori	30%	18%	23%	31%	32%	24%
Pacific	30%	15%	16%	23%	27%	18%
Chinese	–	8%	6%	12%	18%	9%
Indian	–	13%	12%	17%	23%	13%
Other Asian	–	6%	9%	19%	29%	12%
European/Other	36%	16%	12%	19%	29%	19%
Total	**33%**	**15%**	**15%**	**21%**	**28%**	**18%**

Public hospitals, at least overnight stay, medical-surgical only. Asian 0–14 numbers too small to calculate.

Over the 1996–2007 time period there was a large increase in the number of hospitalisations. Coupled with the increasing proportion with diabetes coded, this led to a sizeable rise in diabetes hospitalisation rates over time as a rate per 1000 population. Again taking the 45–64 age group as a focus, Counties Manukau had a 58% increase over the 12-year period, with Māori and Pacific 45–64 year olds, rising by 31% and 52% respectively (Figure 1).

Figure 1. Counties Manukau (CM) 45–64 year olds, compared to New Zealand (NZ) and USA, discharges with diabetes mentioned per 1000 45–64 population, inpatients, 1996-2007[9]



Māori and Pacific people had similar discharge rates in 2007 for 45-64 year olds of 60 and 65 per 1000 people aged 45-64 respectively. The Counties Manukau rates for Māori and Pacific are higher than their New Zealand counterparts, with the difference particularly striking for Māori, where New Zealand rates have been declining since 2000. Looking at individuals, 5% of all Māori and 6% of all Pacific 45-64 year olds in CMDHB were admitted to hospital at least once in 2007 with diabetes.

By comparison, the United States rate rose from 21 to 24 per 1000 45-64 population from 1995-2005.[9] This is nearly twice the New Zealand rate (14 per 1000 in 2005), but is similar to the Counties Manukau rate (23 per 1000 in 2005). Whilst the US is often characterised as being ten years ahead of New Zealand in the obesity epidemic, it appears that the Counties Manukau population gained rapidly on the US over that period.

Overall costs of care were examined by combining mean costs for community pharmaceutical and laboratory claims with the mean cost of hospitalisation for diabetes cases and those without diabetes in CMDHB in 2007. When standardised by age and sex, the analysis indicates that these costs incurred an additional $66 million to CMDHB in 2007 (Table 5).

The estimated additional cost of diabetes in this analysis is likely to be an underestimate of the true cost of health care for diabetes in CMDHB. Several aspects of diabetes health care have not been included in the estimate—for example primary care visits, outpatients, and retinal screening.

Table 4: Age- and sex-standardised annual cost to CMDHB of medical/surgical discharges, community laboratory, and pharmaceutical claims for those with and without diabetes in 2007

Variables	Cost per person with diabetes ($)	Cost per person without diabetes ($)	Total additional cost to CMDHB ($ '000)
Laboratory claims	160	51	2910
Pharmaceutical claims	1229	181	28,270
Inpatient discharges	1858	561	34,980
Total	**3247**	**793**	**66,160**

Discussion

This study highlights that a significant proportion of inpatient care in middle-aged and older adults in Counties Manukau relates to diabetes: approximately 1 in 5 medical-surgical patients aged 45–54 years and 1 in 4 aged 65–74 years (who stayed at least overnight in a CMDHB facility) had diabetes (Table 1).

There were significant differences by ethnicity, with Māori, Pacific, and Indian people notably higher: up to 46% of all discharges in the 65–74 age groups for Pacific and Indian people had diabetes mentioned. There was also a significant increase in the rate of admission for diabetes (any mention) over the past 10 years. For the 45-64 age

group Counties Manukau has a similar rate of admission to that in the USA, with no evidence of a plateau.

There are a range of possible explanations for the increase in the rate of admission. Improved coding is likely to be an important factor. Coding improved in the 1990s as casemix funding provided financial rewards to hospitals to code accurately and comprehensively. However, at CMDHB there is little additional impact of these improvements from 2000 onwards.

It is likely that detection of diabetes has improved; with more people diagnosed there are likely to be more people who will be identified and coded at admission. There is evidence mounting that the proportion of known to 'unknown' diabetes has been steadily falling. Ratios as high as 1:1 were mooted in the early to mid 1990s for the 40–59 year age group.[10]

The ratio of unknown to known has since decreased over time, and recent evidence is pointing to 1 in 3 for European, and 1 in 4 or 1 in 5 for Māori and Pacific respectively over the age of 35 years (that is, for every 5 known to have diabetes there is 1 unknown).[5] The intensive priority given to diabetes screening in CMDHB may therefore be resulting in a lower undiagnosed rate in CMDHB than nationally.

These developments in diabetes coding and detection, coupled with a likely genuine increase in diabetes in parallel with the rising obesity rates will be driving the hospital admission increases. Supporting this, admissions with a primary diagnosis for diabetes, usually representing acute severe metabolic derangement have increased substantially, reflecting more people with diabetes.[11]

Despite 'upstream investment' in diagnosis and primary care management, the number of diabetic patients seeking secondary care input is rising above the rates of population growth increasing demands on services, as people with diabetes have longer hospital stays and greater morbidity and mortality.

It is clear that these data show a considerable burden of disease, both in the human impact and the health service costs, and pose significant challenges for models of hospital and primary care management of diabetes, and the future planning of health facilities and the health workforce.

Within CMDHB, these data have been incorporated into long-term planning and business cases have been made for significant expansion of facilities and workforce to meet demand. The expectation is that decisions such as these at the 'macro' (DHB) level will flow through to staffing at the 'micro' (hospital departmental) level.

The research raises questions that require further investigation: for example, what percentage of these patients are enrolled in chronic disease management programmes such as CCM; what percentage require secondary care or could have been treated in primary settings; and what information passed between secondary and primary care?

Central to these is a further question: what is the appropriate model of care for specialist diabetes services in meeting the growing demand for hospital-based treatments? Are new approaches needed so that secondary services can better support diabetes detection, engagement with primary health care and ongoing effective management of the condition?

With 1 in 4 Māori with diabetes in Counties Manukau being admitted to hospital overnight at least once each year (Table 3), and 1 in 5 Pacific people with diabetes, the importance of the flow of information between primary and specialist care associated with a hospital episode is highlighted. There may be potential in establishing a diabetes register to better understand the treatment pathways of people with diabetes, and to assist with designing more effective intervention processes.

A further implication to consider is if hospital services are making the most of the time the person is spending in hospital to improve their diabetes care by providing appropriate education. There may be opportunities at these times to reinforce advice that should also be being given in primary care, and which could be systematically followed up in primary care (targeted to those patients with inpatient events).

The findings give credence to cross-sectoral strategies such as *Let's Beat Diabetes*, which aim to optimally balance and coordinate efforts to reduce the negative upstream influences (such as diet, physical activity and their environmental influences) and enhance the effectiveness of downstream treatments in primary and secondary care settings. Clearly, diabetes and its complications are entrenched features of the populations studied here, and reducing the burden of diabetes will only be achieved through sustained action across different sectors.

Conclusion

For health planners and funders dealing with the incidence and impact of diabetes, the challenge is considerable and is still growing. Planning to meet this challenge requires a view of diabetes across the full spectrum of disease progression, and identifying where people flow through different parts of the care system, where the critical points of intervention are, and how flows of information can support optimal patient care.

Responding to the Type 2 diabetes epidemic requires consideration of the mix of interventions and a rebalancing of the investment mix, so that (over the long term) the disease burden can be lessened. Concurrent with upstream investment, however, the needs of those people with diagnosed disease must be met by appropriate resourcing of specialist services. The demands of diabetes for hospital resources will grow considerably before we see the returns from prevention and more effective primary care.

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Hospital expenditure on treating complications of diabetes and the potential for deferring complications in Canterbury, New Zealand

Ian Sheerin

Abstract

Aim To investigate annual expenditure by a major district health board in New Zealand on hospital admissions for treating the various complications of diabetes.

Methods Actual costs were analysed for 2005/06, for all inpatient hospital admissions in Canterbury, New Zealand, where diabetes was recorded as a primary or secondary diagnosis. Costs and lengths of stay for all such admissions were included for ICD-10 codes using the criteria from Australian studies of potentially avoidable hospitalisations. ICD-10 codes were used to identify the major types of complications of diabetes resulting in hospital admissions.

Results Total costs of all hospital admissions where diabetes was recorded as a primary or secondary diagnosis amounted to $10.1 million in 2005/06, and 9511 days stay in hospital; 69% of these costs were for admissions where diabetes was a secondary diagnosis, with the majority of such costs being for treatment of admissions for cardiovascular disease.

Conclusions The results generally indicated that diabetes associated with hospital admissions is a major factor, which is still under-reported, and which involves considerable costs of treating the longer term consequences of diabetes. The largest proportion of these costs is for cardiovascular disease, where diabetes is the secondary diagnosis.

Diabetes is a major public health problem which is associated with premature death and disease, with high costs of treating complications.[1] Recently, in Australia diabetes was identified as a major underlying cause of avoidable hospital admissions.[2]

It is often claimed that earlier detection of diabetes and intervention may save the costs of treating later complications, such as retinopathy, cardiovascular disease, foot amputations, and renal failure. There are current evidence-based guidelines that recommend diagnostic protocols, medical treatments, and lifestyle changes.[3]

These guidelines cite considerable evidence that such interventions can defer the onset and complications of Type 2 diabetes. However, there is a general lack of information on current health system costs of diabetes complications and on what proportion of these costs might realistically be deferred or prevented. Lack of information is further complicated by under-diagnosis and under-recognition of diabetes in hospital admissions and in contributing to the cause of death.[4]

This paper estimates costs of avoidable inpatient hospital admissions for diabetes and its various complications in the Canterbury District Health Board (CDHB), New Zealand, which covers a population of approximately 500,000 people. The

information may help to provide information for making decisions about investing in earlier more intensive management of diabetes and the potential for reducing overall costs of diabetes complications in the longer term.

Methods

Actual costs of inpatient hospital admissions for the 2005/06 financial year were provided by the Decision Support Unit of the CDHB (in New Zealand dollar values). They extracted costs and length of stay of all admissions, for ICD-10 codes, where diabetes was a primary or secondary diagnosis, using the criteria used in Australian studies of potentially avoidable hospitalisations.[2,5] These costs include all diagnostic and treatment costs during hospital admissions, including pharmaceuticals used during these admissions, and costs of administration, facilities, and hotel services.

Admissions were sorted by ICD-codes and their costs were analysed using a Microsoft Excel spreadsheet.

Excluded are primary care costs, outpatient costs, pharmaceuticals prescribed in community settings, and admissions to private hospitals. Also excluded were any costs of treating comorbid psychiatric conditions in the community or in psychiatric hospitals.

Costs of diabetes management and prevention in community settings are also excluded from the costs in this paper. For example, costs of the *Get Checked* annual diabetes checks and of retinal screening are excluded.

The costs of renal dialysis are also not included in this paper because in Canterbury, dialysis is mostly performed in the community.

Costs for previous years were not extracted, partly because of changes in ICD classifications and associated problems in comparing costs over time.

Results

Total costs of all inpatient hospital admissions where diabetes was recorded as a primary or secondary diagnosis were estimated to be $10.1 million in 2005/06, involving 9511 days stay in hospital; 69% of these costs were attributed to complications of diabetes mellitus (DM), where DM was a *secondary* diagnosis.

Table 1 summarises the costs of admissions where DM was the *primary* diagnosis. It shows that the majority of the costs were associated with Type 2 diabetes mellitus (T2DM). Of admissions where T2DM was a primary diagnosis, 214 bed days costing $498,368 were for ophthalmic complications. Renal complications accounted for another 251 bed days and costs of $223,942. Table 1 identifies 598 bed days and over $560,000 in costs were for ICD code E11.7 and the majority of these were for foot ulcers.

Table 1 also shows 532 bed days for ICD diagnosis E11.6, and the majority of these costs were for admissions for hypoglycaemia and/or for poor control.

Table 2 shows that more than twice as much cost ($6.98 million) was associated with admissions where diabetes was the *secondary* diagnosis, compared with those for diabetes as a primary diagnosis. Ninety percent of these costs were for admissions for diseases of the circulatory system. Numbers of admissions and costs of both renal failure and preparation for dialysis were low in comparison to those for cardiovascular disease.

Table 1. Expenditure and bed days in the Canterbury District Health Board (in 2005/2006) on complications of diabetes where diabetes is the primary diagnosis

ICD-10 code	Type of disorder	Expenditure (NZ$)	Bed days (N)
E10.1	IDDM with ketoacidosis	352,211	415
E10.2	IDDM with renal complications	52,167	46
E10.3	IDDM with ophthalmic complications	81,094	29
E10.4	IDDM with neurological complications	32,735	48
E10.5	IDDM with peripheral circulatory complications	3060	1
E10.6	IDDM with other specified complications	143,386	134
E10.7	IDDM with multiple complications	122,893	132
E10.8	IDDM with unspecified complications	7480	5
E10.9	IDDM without complications	112,004	90
E11.0	T2DM with coma	16,983	30
E11.1	T2DM with ketoacidosis	64,228	85
E11.2	T2DM with renal complications	223,942	251
E11.3	T2DM with ophthalmic complications	498,368	214
E11.4	T2DM with neurological complications	93,314	152
E11.5	T2DM with peripheral circulatory complications	238,315	219
E11.6	T2DM with other specified complications	432,601	532
E11.7	T2DM with multiple complications	564,426	598
E11.9	T2DM without complications	31,807	48
E13	Other specified DM	80,563	90
E14	Unspecified DM	2941	7
Total admissions for diabetes as primary diagnosis		**3,154,516**	**3126**

IDDM=insulin-dependent (Type 1) diabetes mellitus; T2DM=Type 2 diabetes mellitus.

Table 2. Expenditure in the Canterbury District Health Board (in 2005/2006) on diabetes where diabetes is the secondary diagnosis

ICD-10 code	Type of disorder	Expenditure (NZ$)	Bed days (N)
E87.2	Acidosis	5527	4
G45 to G63	Diseases of the nervous system	175,176	184
H25 to H41	Diseases of the eye and adnexa	61,954	23
I20 to I75	Diseases of the circulatory system	6,361,324	5909
N10 to N23	Diseases of the genitourinary system	271,437	206
Z49.0	Preparatory care for dialysis	102,649	59
Total admissions for diabetes as secondary diagnosis		**6,978,067**	**6385**

Table 3 provides a more detailed summary of the major complications of admissions where diabetes was recorded as a secondary diagnosis. It shows clearly the importance of angina, myocardial infarction (MI), and heart failure as the major cost categories for cardiovascular disease (CVD). Thirty-six percent of costs where diabetes was a secondary diagnosis were for admissions for MI with another 22% for angina. For angina pectoris unspecified, the most common type of angina reported, the average length of stay was 3.67 days (range 1–56). For the most common type of MI (acute subendocardial MI), the average length of stay was 8.35 days (range 1–36).

Table 3 also shows disorders of the genitourinary system accounting for much lower numbers of discharges and costs in comparison with CVD.

Table 3. Expenditure and bed days in the Canterbury District Health Board (2005/2006) for selected complications of diabetes where diabetes is the secondary diagnosis

ICD-10 code	Type of disorder	Expenditure (NZ$)	Bed days (N)
G45.9	Transient cerebral ischaemic attack	86,544	113
I20.0	Unstable angina	734,287	602
I20.9	Angina pectoris, unspecified	830,304	518
I21.0	Acute transmural MI of anterior wall	257,825	187
I21.1	Acute transmural MI of inferior wall	186,071	145
I21.4	Acute subendocardial MI	2,095,802	1786
I25.11	Chronic ischaemic heart disease	122,942	73
I50.0	Congestive heart failure	697,560	1024
I50.1	Left ventricular failure	202,206	244
I63 to I63.9	Cerebral infarction	501,909	666
I64	Stroke	183,566	274
I71.3	Abdominal aortic aneurysm, ruptured	124,754	36
I71.4	Abdominal aortic aneurysm, without rupture	117,098	52
I74 to I74.9	Arterial embolism and thrombosis	57,482	23
N17 to N19	Renal failure	77,676	64
N20 to N23	Urolithiasis	146,651	97
Z49.0	Preparatory care for dialysis	102,649	59
Total admissions for DM as secondary diagnosis		**6,978,067**	**6385**

Discussion

Information on hospital admissions, bed days, and costs may help to indicate where investment could be redirected to emphasise earlier intervention and prevention of complications of diabetes.

There is much published evidence on the potential to defer or prevent the development of these complications. In Australia, diabetes was shown recently to be a major cause of avoidable hospitalisations.[2] In Canterbury New Zealand, Endre, Beaven, and Buttimore[6] indicated that 14% of all hospital bed days are due to undiagnosed or inadequately diagnosed diabetes.

However, there is published evidence that diabetes is under-reported in contributing to deaths and to hospital admissions. Chen et al[3] found that in New Zealand, diabetes was under-reported on more than 50% of *death certificates*, which was consistent with findings from other studies in Otago,[7] in Australia,[8] and Europe.[9]

In New Zealand, the extent of under-reporting of diabetes in *hospital discharges* is uncertain, but is likely to be significant.[3] A Dunedin study indicated that hospital admissions involving DM as a secondary diagnosis were not recorded in 45% of cases.[7]

In 2002/2003, Sheerin et al[11] found that avoidable admissions to Christchurch Hospital for CVD accounted for 34,390 bed days and $50.6 million in hospital costs. The estimates from this current 2005/06 study of $6.4 million of costs and 5,909 bed days where diabetes was explicitly listed as a secondary diagnosis in admissions for

CVD (Table 3), seem relatively low in comparison to the costs of all avoidable admissions for CVD found in 2002/03. This seems likely to reflect under-reporting of diabetes as a contributing factor in hospital admissions for CVD.

Decision-making about improvements in diabetes management would be assisted if more accurate information was available, including the extent of diabetes as a contributing factor in hospital admissions. This would be assisted by continuing efforts to promote recording of diabetes where it may be involved as a secondary diagnosis.

This type of information could potentially indicate the importance of hospital costs of admissions for T2DM and the potential costs of deferring complications and their associated costs.

The complications involving the dominant costs and the areas of greatest potential indicated from this study included:

- Ophthalmic complications.
- Foot ulcers.
- Cardiovascular disease.
- Renal failure.

Studies have shown that retinal screening programmes are cost saving[12,13] and that the incidence of blindness could be reduced by 35% by screening and early treatment.[14] Foot care has also been identified as cost saving.[12] The majority of amputations are preceded by foot ulcers, but foot care programmes have been shown to reduce frequency of lower extremity amputations by approx 50 to 85%.[15,16]

The information in Table 3 indicates the importance of CVD. The New Zealand guidelines for management of Type 2 diabetes also emphasise this and estimate that approximately two-thirds of people with T2DM die of CVD.[3] These guidelines also state that 50% of microvascular complications and of CVD events could be reduced by pharmacologic treatment and lifestyle changes—chiefly nutrition and physical activity. This study did not incorporate comparisons of costs of hospital admissions for similar conditions with and without diabetes as a secondary diagnosis, which would be a useful focus of further investigation.

The Costs of Diabetes in Europe-Type 2 study (CODE-2) found that in eight European countries, the presence of microvascular and macrovascular complications increased patient management costs more than 3.5 fold.[17] They concluded that to reduce the overall costs of diabetes, policies should aim to reduce hospital stays and to delay the onset of complications.

Both the NZ guidelines and trials in the UK indicate the importance and effectiveness of intensive metabolic control.[3,18]

Endre, Beaven, and Buttimore[6] estimated that probably half of the total expenditure on diabetes end stage chronic kidney disease (CKD) could be saved or deferred by more strategic management. A US study[14] found that screening and early treatment was estimated to reduce lifetime occurrence of kidney failure by 26%. In this paper, the costs of renal failure appear low compared with CVD, however costs of renal

dialysis are not included in these tables because, in Canterbury, dialysis is mostly provided in community settings.

The costs of renal dialysis in Christchurch have previously been reported to total $5.9 million for 2003.[6] Therefore, prevention of renal failure should be given priority, even though it may appear relatively low compared with costs of hospital admissions.

A limitation of this study is the lack of information on ethnicity and the age of onset of diabetes. There is concern about the increasing numbers of younger people with obesity and T2DM, particularly with evidence that younger onset may have the potential to increase the numbers who develop nephropathy and end stage renal disease.[19,20] This appears to be an important area to monitor in order to be in a position to promote policies aimed to prevent increasing costs of renal failure.

In 2005/2006 the CDHB spent approximately $3.9 million on diabetes management and prevention in community settings.[21] Despite considerable progress in implementing free annual checks for people with diabetes, there remains a range of opportunities for improved targeting of preventive services as illustrated by some examples:

- Only an estimated 32% of Māori people in Canterbury with diabetes, actually received an annual check in 2006;
- Only 22% of Māori people with diabetes had glycaemic levels within recommended guidelines (and 45% of the general population with diabetes);
- Approximately 32% of people with diabetes in Canterbury received retinal screening, and only 14% of Māori with diabetes;
- The Local Diabetes Team reported there are long standing and continuing problems with access to foot clinics.

The CDHB has recently completed a review of diabetes services in the region, in which these problems were identified. It is currently reviewing options for future priorities.

The main limitation to this study was the lack of data on costs incurred privately by individuals with diabetes. For example, the CDHB has good information on inpatient costs and on its investment in community preventive programmes such as annual check-ups, diabetes management, and retinal screening. However, there is a lack of information on privately incurred costs of co-payments for attending general practitioners and private specialists, lost work time, and other costs to families. Nevertheless, this study does provide important information on how resources are used by the Canterbury District Health Board in treating diabetes and its complications. This is important because resources are limited and decisions must be made about how they should best be spent, particularly the balance between early intervention and management, compared with the costs of hospital admissions for longer term complications of diabetes.

It may be questioned if the results of this study can be generalised to other district health boards. However, these findings are generally consistent with research in other Western countries.[1,22]

For example, a European study found that the costs of treating the complications of diabetes (mainly during hospital admissions) were approximately 3.3 times the amount spent on attempting to manage and prevent diabetes in the community.[17] Therefore, the evidence indicates that a similar situation is likely to exist in other areas of New Zealand.

Conclusions

This study shows that in 2005/2006, $10.1 million of costs and 9511 bed days were spent by the CDHB on hospital admissions for complications of diabetes, where diabetes was specifically identified as a primary or secondary diagnosis; 69% of costs were associated with admissions where diabetes was recorded as a secondary diagnosis. Diabetes appears to be under-reported as a factor in many hospital admissions.

This paper shows that expenditure by one of the largest district health boards on hospital admissions for complications of diabetes was considerably greater than the amount spent on attempting to manage diabetes in the community. However, there is ample published evidence indicating that many of these complications could be deferred and/or prevented.

The information indicates that the options which may offer the most potential are: retinal screening; foot care; control of blood pressure in primary care settings; and lifestyle changes, focussing on increasing physical activity and improving nutrition.

Investment on these services should defer or prevent a significant proportion of expenditure on treating complications of more advanced diabetes.

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Prevalence of undiagnosed diabetes, impaired glucose tolerance, and impaired fasting glucose among Māori in Te Wai o Rona: Diabetes Prevention Strategy

David Simmons, Elaine Rush, Nic Crook

Abstract

Aims To describe the prevalence of undiagnosed diabetes, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) ("dysglycaemia") among Māori.

Methods Te Wai o Rona: Diabetes Prevention Strategy was a trial of lifestyle change among Māori families in the Waikato/Lakes areas of New Zealand. All Māori family household members aged ≥28 years, without known diabetes, were invited to participate through primary care, community, and media approaches. Participants were invited to have an oral glucose tolerance test (OGTT).

Results Of the 3817 eligible Māori, mean BMI was 32.9±7.8 kg/m^2 (women) and 33.1±6.7 kg/m^2 (men). The age standardised prevalence of undiagnosed diabetes was higher among men than women (6.5[5.8–7.4]% vs 4.2[3.6–4.8]%), as was that for IFG (5.4[4.7–6.1]% vs 3.0[2.3–3.5]%), but not IGT (8.5[7.6–9.4]% vs 9.7[8.7–10.6]%) with no rural-urban differences. The prevalence of dysglycaemia increased with increasing BMI with no clear inflection point and was 1.33(1.11–1.60) greater among those with a community services card after adjusting for age, sex and BMI.

Conclusions Undiagnosed diabetes, IGT, and IFG remain common among Māori, particularly men, the very obese, and those with greater socioeconomic disadvantage. There remains significant opportunity to reduce Māori morbidity and premature mortality through diabetes case-finding and intervention.

The management of known Type 2 diabetes and its complications accounts for a significant proportion of health expenditure in New Zealand.[1] However, much of diabetes remains undiagnosed[2] and levels of hyperglycaemia below the diagnostic threshold for diabetes (impaired glucose tolerance [IGT] and impaired fasting glucose [IFG]) are also associated with an excess risk of cardiovascular disease.[3] Such levels of hyperglycaemia are also at high risk of progressing to Type 2 diabetes, progression now known to be preventable by intensive lifestyle change and some medications.[4–6]

The potential benefits of detecting any abnormal glucose tolerance vary between populations depending on risk of developing diabetes and risk of complications once diabetes has been diagnosed. Calculating the costs and benefits of intervening at an early stage require detailed information including the proportion with undiagnosed diabetes, IGT and IFG.

Māori have higher rates of diabetes than European New Zealanders and disproportionately higher rates of many of the complications caused by diabetes.[2] As such, a successful diabetes prevention strategy among Māori is crucial for New Zealand and Māori alike.

Te Wai o Rona: Diabetes Prevention Strategy was a 4-year randomised controlled trial among Māori communities in the Waikato and Lakes District Health Board areas,[7] registered with the Australasian Controlled Trials Registry (ACTRN012605000622606). The trial did not proceed after the first 3 years for funding reasons. However, the baseline data, being population based may be able to be used to provide the needed detailed information regarding the proportion with undiagnosed diabetes, IGT, and IFG and we now present these data and discuss the caveats behind the data.

Methods

Participants—All Māori resident within the boundaries of the Waikato DHB, and the tribal area of Ngati TuWharetoa in the neighbouring Lakes DHB were invited to participate with their families. The age cutoff for entry was taken as ≥28 years on 30 September 2005. Recruitment was by personal invitation from local general practitioners in association with media releases (television, radio, posters, newspapers) announcing times/venues of screening, workplace screening, and personal contact.

Personal contact through different health organisations and their staff, and announcements at a number of Māori community activities became increasingly important through the recruitment phase. Those who attended were asked to inform other family members and friends.

Participants were asked to attend after a 10-hour overnight fast and were advised that breakfast would be provided. Advice was also given that attending non-fasting was also acceptable although full testing (including OGTT) would not then be performed. Those unfit to sign a consent form, with terminal disease, or not permanently residing in the study area at the time of the baseline data collection were excluded.

Ethical approval was provided by both the Waikato and Bay of Plenty Ethics Committees. All participants gave signed informed consent.

Screening sessions were held 0700–1400 hours in a variety of community venues across the study area. Where possible, transport was provided for participants. After registration/consent and ascertainment of fasting status, fasting participants had a finger-prick glucose (venous plasma equivalent) using a glucose meter (Advantage, Roche, Switzerland) and venesection. Those who were non-fasting had a single venesection for glucose and HbA1c (Bio-rad Diamat Variant, [upper limit of reference range 6.4%], Bio-Rad Laboratories, USA).

Samples for HbA1c were sent to the same laboratory for analysis. Glucose samples were centrifuged, separated, and refrigerated within 30 minutes on site in a mobile laboratory and subsequently measured using the Hitachi 911 (Hitachi Limited, Tokyo, Japan). All assays were within target limits specified by the RCPA Quality Assurance Program. These assays were carried out by the Waikato District Health Board Laboratory which has IANZ ISO9002 Accreditation.

Fasting participants with a fingerprick glucose ≥4.4 mmol/L were advised to undertake a 75g 2 hour oral glucose tolerance test (OGTT), although those with values below this were also invited to have an OGTT.

Trained staff facilitated standard questionnaire and measurement completion. Questionnaires including demographic data were completed. Ethnicity was determined by self identity. During the OGTT, other measurements included height±0.5 cm (portable height scale PE087; Mentone Education Centre, Victoria, Australia) and weight±0.1 kg (Wedderburn TI-TH316 Personal scales or Wedderburn TI-BWB800 Personal scales [up to 200kg] for oversize participants).

Based upon previous research including Māori,[8] if no OGTT had been completed and the fasting glucose was ≥5.3 mmol/L, or a random glucose ≥5.3 mmol/L or the HbA1c ≥ 5.3%, participants were asked to subsequently attend the local community laboratory for an OGTT (screen positive subjects).

Screen-negative subjects were defined as those with HbA1c, fasting, and random glucose results below these criteria. Diabetes, IFG, and IGT were diagnosed using 1998 World Health Organization criteria.[9] If no OGTT was undertaken and the fasting glucose was ≥7.0 mmol/L and/or the random glucose was ≥11.1 mmol/L, diabetes was considered to be present.

Statistics—The overall prevalence of diabetes (among those aged ≥30 years) was calculated by direct age standardization to the 2006 Census.[10] Comparisons are made by either Chi-squared test or by

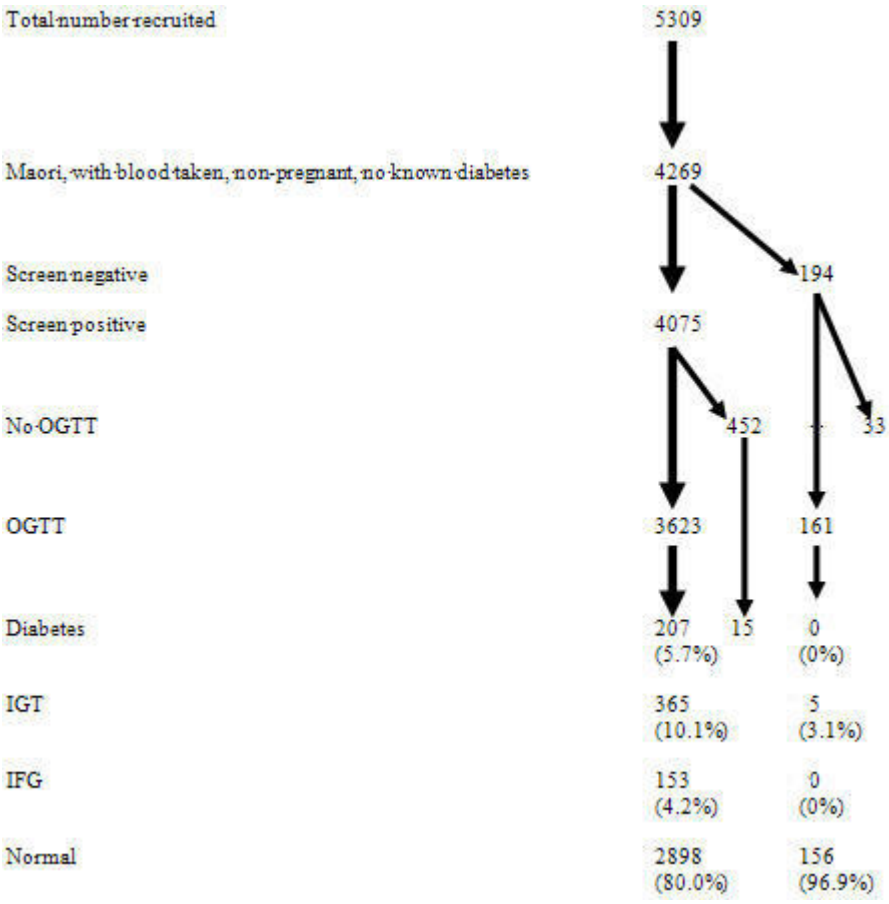
comparing 95% confidence intervals. Tests are 2-tailed with $p<0.05$ taken as significant. Mantell Haenszel test was used to compare the prevalence of dysglycaemia by community services card use, after adjustment for age and gender.

Results

Of the 5059 non-pregnant adults screened for diabetes, 4269 were Māori aged ≥28 years (approximately 13% of the comparably aged Māori population in the recruitment area).[10]

Figure 1 shows the attendance at OGTT (n=3784) including among those with a negative screen. Among screened Māori, 2726 (63.9%) were women, the mean age was 48±12 years, 67.4% were rural residents, and 65.9% were known to have a family member with diabetes. Very few (3.7%) of those with a negative screen had IGT and none had diabetes or IFG.

Figure 1. Response to screening and crude proportions with diabetes, impaired glucose tolerance (IGT), and impaired fasting glucose (IFG)



Among men, 86.7% and 79.2% of those above and below the criteria for OGTT respectively had an OGTT, the proportions being 85.2% and 89.9% respectively among women. Among those not attending OGTT and screen-positive (above the

threshold for OGTT), 15 were considered to have diabetes on the basis of their screening test.

Among the screen-positive participants, the fasting blood glucose (n=109), random blood glucose (n=338), and HbA1c were similar between those attending and not attending OGTT (5.3±0.9 vs 5.5±2.1 mmol/L; 5.5±1.0 vs 5.6±1.8 mmol/L; 6.0±0.6 vs 6.0±1.0% respectively). As the proportion with a "negative screen" was small (4.5%), and most of these had an OGTT (among whom few had IGT and none with diabetes or IFG), analyses assume that the negative screen group were all normal and that those not having an OGTT were comparable to the overall cohort. Age-specific prevalence is therefore calculated from those who had an OGTT or who were screen negative without an OGTT (n=33).

Table 1 shows that the prevalence of diabetes increased with age, although no Māori aged over 80 years was found to have undiagnosed diabetes. The age standardised prevalence of undiagnosed diabetes was higher among men than women (6.5[5.8–7.4]% vs 4.2[3.6–4.8]%), as was that for IFG (5.4[4.7–6.1]% vs 3.0[2.3–3.5]%), but not IGT (8.5[7.6–9.4]% vs 9.7[8.7–10.6]%).

Table 1. Response to screening and estimated prevalence of undiagnosed diabetes, IGT, and IFG among Māori

Females (age groups)	**28–29**	**30–39**	**40–49**	**50–59**	**60–69**	**70–79**	**80+**
N	93	630	737	537	328	128	15
Diabetes	2.2%	2.4%	3.8%	5.0%	8.5%	7.0%	0%
IGT	5.4%	6.5%	7.9%	10.6%	15.9%	14.8%	53.3%
IFG	1.1%	1.3%	2.4%	4.8%	6.7%	3.1%	0%
Males (age groups)	**28–29**	**30–39**	**40–49**	**50–59**	**60-69**	**70–79**	**80+**
N	44	301	413	286	206	89	10
Diabetes	2.3%	2.3%	7.7%	8.4%	11.2%	12.4%	0%
IGT	2.3%	4.3%	5.8%	12.6%	17.5%	14.6%	20.0%
IFG	0%	3.3%	7.7%	5.6%	4.9%	6.7%	0%
Female vs male	ns	ns	<0.001	ns	ns	ns	ns

Those who were screen negative were <15.0% in all age-sex groups.

There was no significant difference in prevalence of diabetes, IGT, or IFG between those living in rural and urban areas, nor between those living in different rural tribal areas. Among those classified as having diabetes, 12.8% had a fasting glucose <6.1 mmol/L and 8.2% had a fasting glucose <5.6 mmol/L. Among those with IGT, 76.2% had a fasting glucose <6.1 mmol/L and 46.7% had a fasting glucose <5.6 mmol/L.

Mean BMI among women was 32.9±7.8 kg/m^2 and among men was 33.1±6.7 kg/m^2. The prevalence of diabetes, IGT, and IFG are shown by body mass index (BMI) group in Figure 2 (women) and Figure 3 (men). The BMI groups were defined by 1 kg/m^2 across the overweight and obese ranges (25–36 kg/m^2).

There was no significant age difference between the BMI groups. Prevalence of dysglycaemia increased with increasing BMI with no clear inflection point. Among the 29.4% of women and 28.4% of men with a BMI ≥36 kg/m^2, over one-third had some degree of dysglycaemia, particularly IGT.

Figure 2. Prevalence of diabetes, IGT, and IFG by body mass index (*women*)

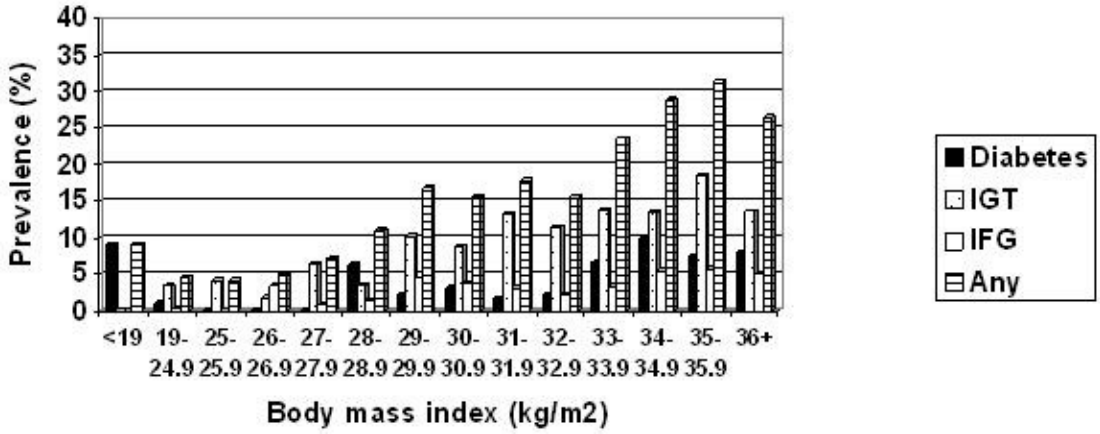
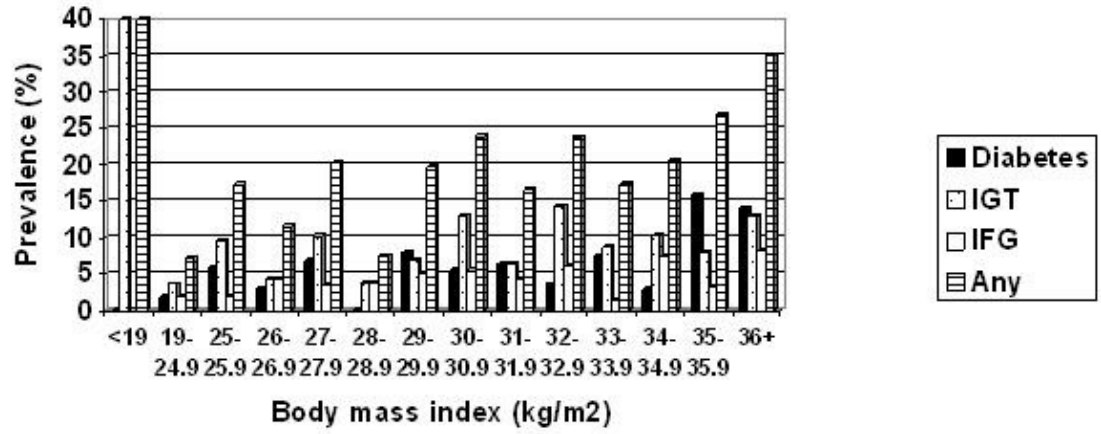


Figure 3. Prevalence of diabetes, IGT, and IFG by body mass index (*men*)



Note: The 1 subject with a BMI <19 kg/m2 had IGT.

The prevalence of dysglycaemia was 1.38(1.16–1.65) fold greater among those with a community services card after age and gender and 1.33(1.11–1.60) fold greater after additionally adjusting for BMI. Undiagnosed diabetes alone was 1.24(0.91–1.69) fold higher among those with a community services card (i.e. non significant).

Discussion

This is one of several studies reporting the prevalence of undiagnosed diabetes, IGT, and IFG among Māori in different parts of New Zealand.[2] The prevalence of undiagnosed diabetes was higher than in the Diabetes Heart and Health Study[11] and comparable to that in the Ngati and Healthy Project.[12]

Abnormal glucose tolerance, particularly undiagnosed diabetes, was particularly high among Māori men and the very obese. As diabetes, IGT, and IFG remain harbingers

of significant cardiovascular disease,[3] and lifestyle and pharmacological intervention can significantly reduce the risk of future morbidity and mortality, we need to find ways to identify such dysglycaemia as early as possible.

Those identified as having diabetes in this study were followed up for microvascular disease (retinopathy and nephropathy) and while retinopathy was prevalent in very few, microalbuminuria and albuminuria were present in 29.6% and 7.7% respectively.[7]

We have previously shown that microalbuminuria is often present before diagnosis of diabetes in Māori and that it is more related to the familial risk of renal disease than diabetes.[13] The low prevalence of retinopathy was heartening and is a measure of the duration that individuals remained undiagnosed.[14]

The prevalence of known diabetes in the area is unknown, and the low rate of retinopathy at diagnosis might suggest a greater uptake of screening in this district. A combined diabetes specialist clinic and retinal screening register has estimated the prevalence of known diabetes among Waikato Māori to be 1.4% (30–39 years), 4.0% (40–49 years), 10.9% (50–59 years), and 17–20% (60+ years).[15]

These data exclude some important patients (e.g. those attending eye clinic but not the diabetes specialist clinic) and are not gender-specific, but suggest that (perhaps) over 50% of diabetes is undiagnosed in those 30–49 years, but that 25–40% are undiagnosed over this age.

In view of the risk from undiagnosed Type 2 diabetes in pregnancy among Māori women,[16] and the risk of renal disease and other long-term complications from uncontrolled diabetes among Māori,[2] diabetes screening and diagnostic strategies among Māori under 50 years warrants greater attention.

With such a high prevalence of undiagnosed diabetes shown in these analyses, and the excess risk of cardiovascular disease associated with dysglycaemia, there is clearly scope to increase identification of Māori with any form of dysglycaemia, who would benefit from intervention.

Whether this would help address the higher mortality rates among Māori, particularly Māori men[17], is yet to be seen. This difference is known to be associated with greater deprivation, and in our study, use of a community services card, accessible only to those on reduced incomes, was associated with a greater prevalence of dysglycaemia. The lack of a significant association with new diabetes was possibly due to insufficient statistical power.

Of interest was the lack of difference in prevalence between rural and urban Māori (as well as between tribal areas), suggesting a balance between access to diabetes screening between rural and urban Māori and actual diabetes/dysglycaemia risk.

We have carefully looked at the prevalence of IGT, IFG, and undiagnosed diabetes in relation to obesity as measured by BMI. Naturally, prevalence increased with increasing BMI, but there was no natural inflection point to assist with defining obesity within Māori.

A range of studies have recommended that Māori have different criteria for obesity using BMI in view of their lower fat content at a given BMI.[18] Clearly what is of

importance in defining a cut off for risk is relating the cutoff to a hard end point such as diabetes or CVD. While such analyses should preferably be prospective using a baseline BMI, in reality, BMI changes over time and a greater weight gain is associated with greater diabetes risk.[19]

In our study, using cross-sectional data, there is no BMI between 25 and 36 which would help define overweight or obesity. These data do need to consider penetration of screening, which may have been greater among more obese Māori.

There are a range of caveats to interpreting these data. These are recruits into a trial of lifestyle change to prevent diabetes. We are aware that some participants attended to be screened for diabetes (i.e. the trial may have attracted those who were symptomatic). Conversely, those attending for a lifestyle trial are possibly more likely to lead a healthier life and to have started making healthier food and physical activity choices, which would impact on risk of dysglycaemia.

Notwithstanding this self selection, the vast majority were obese and hence this is unlikely to have had a major impact on the nature of the cohort. The cohort represents approximately 25% of Māori women and <15% of Māori men in the area, again suggesting that caution should be used in extrapolating these findings to the wider local or national Māori population.

One of the major strengths of the cohort is the high proportion who underwent OGTT without reliance on a fasting test alone. The criteria used to avoid OGTT were largely ignored by participants, resulting in a high proportion of screen negative individuals having an OGTT. This was important, with the high proportion of Māori with diabetes and IGT with a lower fasting glucose. These data are important to inform future screening campaigns and emphasising the importance of the OGTT in identifying those with undiagnosed dysglycaemia who could benefit from intervention.

In conclusion, we have shown that undiagnosed diabetes, IGT, and IFG remain common in this Māori cohort (particularly in men and the very obese) and that there remains significant opportunity to reduce Māori morbidity and premature mortality through case-finding and intervention.

Competing interests: None known.

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The outcomes of transrectal ultrasound guided biopsy of the prostate in a New Zealand population

Andrew R Lienert, Peter J Davidson, J Elisabeth Wells

Abstract

Introduction We aim to produce nomograms relating age, digital rectal examinations (DRE), and prostate specific antigen (PSA) to probability of a positive transrectal ultrasound guided (TRUS) prostate biopsy for a New Zealand population. Usefulness of age-adjusted PSA reference ranges and PSA density are also examined.

Methods Data was extracted retrospectively from electronic records of prostate biopsies performed between 1995–2007 in Christchurch, New Zealand. Nomograms were created using logistic regression models. The area under the curve (AUC) for age-adjusted PSA ranges, PSA density, and PSA in predicting a positive biopsy was calculated and used to compare these methods.

Results 4316 biopsies were available for analysis. Data was incomplete for 1177 (27%) of patients. Biopsy was positive in 54.4%. PSA level and DRE finding were strong predictors of a malignant biopsy in our multivariable model but age was not. PSA level and DRE were also predictors of a higher Gleason score (7 or greater). Nomograms are presented relating PSA and DRE to both a positive biopsy result and to biopsy with Gleason score 7 or greater. AUC for age adjusted reference ranges was no better than PSA using a single cutpoint of 4.0. (0.54 vs 0.53). AUC for PSA density using a cutpoint of 0.15 was 0.72. Receiver Operator Characteristic (ROC) curves showed a clear advantage for PSA density over PSA regardless of cutpoint. (AUC 0.80 vs 0.67).

Conclusions Nomograms are formulated to help inform New Zealand men how likely they are to have a positive TRUS prostate biopsy and also how likely they are to have a higher grade cancer detected by TRUS prostate biopsy. Age-adjusted reference ranges did not improve prediction of cancer in this population, but the use of PSA density may enhance prostate cancer diagnosis.

Prostate specific antigen (PSA) is a prostate cancer tumour marker that was first purified in 1979 by Wang et al.[1] In 1987, Stamey et al[2] were the first to report on the clinical use of PSA as a marker of prostate cancer. Over the last 20 years PSA testing, in combination with digital rectal examinations (DRE), has revolutionised the diagnosis of prostate cancer.

Screening for prostate cancer, however, remains controversial. There are conflicting recommendations with regards to the use of PSA and DRE in testing for prostate cancer.[3–6] Most policy statements regarding PSA/DRE testing and subsequent prostate biopsy do agree that it is most important to accurately inform men of the risks and benefits[3–6] of such testing.

Two important aspects to this information are the probability that a prostate cancer will be diagnosed by transrectal ultrasound guided (TRUS) biopsy and the potential morbidity of the biopsy procedure. Whilst overseas data is available on both of these aspects, there is no data published in a New Zealand population.

At the standard cutpoint of >4.0, PSA has a high sensitivity but low specificity for detecting prostate cancer.[7–10] This leads to high numbers of negative biopsies being performed. Age-adjusted reference ranges and PSA density (PSAD) have been suggested as ways to improve specificity.[12,13] Again, no data exists to examine the validity of these PSA refinements in a New Zealand population.

The aim of this study is to examine a large cohort of New Zealand men having TRUS biopsies, thus producing nomograms relating age, DRE finding, and PSA to the probability of a positive prostate biopsy. Additionally we will assess whether age adjusted reference ranges or PSA density can improve sensitivity and specificity in a New Zealand-based population. We also report post-biopsy sepsis rates for a subset of our cohort.

Patients and Methods

From January 1995 until March 2007, all patients undergoing transrectal ultrasound (TRUS) guided biopsies of the prostate in Christchurch, New Zealand, have had clinical data recorded on electronic databases. This includes patients in both the private and public sectors. Patient details, PSA levels, DRE findings, prostate volume measurements, and number of cores taken were downloaded from these databases. A retrospective electronic search of histology records was performed to assess the outcome of the biopsies.

Approximately 25% of patients had data missing and in these patients a manual search of the clinical records was performed. For calculation of the nomograms, men were excluded if a previous diagnosis of prostate cancer had been made, or there had been a previous prostatic biopsy, or if the histology from the biopsy was not attainable.

Several clinicians performed the biopsies including 7 consultant urologists and multiple rotating trainee registrars. 18 gauge core biopsies were taken under transrectal ultrasound guidance. There is no standard protocol in our department for taking a biopsy. Biopsies were performed on an outpatient basis with no sedation. Local anaesthetic use was at the discretion of the person performing the biopsy. The number of cores taken was also determined by the person performing the biopsy. Ciprofloxacin 500 mg orally twice daily for 4 days, with first dose 2 hours prior to biopsy, was the most common form of antibiotic prophylaxis.

Prostate volume measurement was not recorded routinely for patients in the public sector. Private sector patients who had a biopsy taken after 1 January 1997 had prostate volume recorded routinely. Therefore PSA density analyses were performed only on patients from the private sector who had a biopsy taken after 1 January 1997. All of these biopsies for which volume was available were used.

A subgroup of patients who underwent biopsy between 1 May 2004 and 30 April 2006 had their clinical records reviewed to identify any admissions with post biopsy sepsis. All patients with post biopsy sepsis are routinely admitted to Christchurch Hospital for intravenous antibiotics. They are also recorded at the weekly mortality and morbidity meeting. The records of these meetings were also explored to check that none of these patients were missed. Microbiology cultures from these patients were reviewed.

Analyses were carried out using SAS v9.1 software. For variables investigated singly, Chi-squared tests for contingency tables and t-tests for independent groups were used. Logistic regression with multiple predictors was used to investigate age, PSA, and DRE together as predictors of malignancy, and also as predictors of Gleason scores of 7 or greater.

Nomograms were derived from these models without interaction terms because the model fit was good according to the Hosmer-Lemeshow test. Use of the main effects models provided some smoothing and improved the precision of estimates for small cells.

A receiver operator characteristic (ROC) curve is a graphical plot of the sensitivity vs (1-specificity) at all possible test cutoffs. The ROC curve is therefore a plot of the true positive proportion against the false positive proportion at each cutoff. The (1,0) point (top left hand corner of graph) represents the perfect test because here there are no false positives and no false negatives (100% sensitivity, 100% specificity). When comparing 2 tests represented by ROC curves that do not overlap, the better test is the one closest to the (1,0) point. The area under the curve (AUC) is calculated based on ROC curves. A perfect test (as described above) would have an AUC of 1.0, whereas an AUC of 0.50 represents a test that is equivalent to flipping a coin so that sensitivity and 1-specificity are equal at each cutoff and the ROC curve is a straight line from (0,0) to (1,1). The higher the AUC the better the test.

ROC curves and 95%CI for AUC were calculated using SPSS v13.0 software. There was no adjustment of the confidence intervals because of ties.

Results

A total of 5842 biopsy events were identified, of which 2683 (46%) were from the public sector and 3159 (54%) from private. There were 4516 first-time biopsies after excluding those with an initial diagnosis by transurethral resection of the prostate, those where the biopsy was a repeat biopsy, and those in whom there was insufficient information to be certain if this was their first biopsy.

Of these 4516 first-time biopsies, the histology result was missing in 200. The remaining 4316 biopsies form the study group for analysis in this paper. Within this group data was missing on age in 21 patients, PSA in 360, DRE in 364, and number of cores in 1025 patients respectively. Complete data was available in 3139 patients.

Table 1 summarises patient characteristics in our cohort. Just over half of all biopsies showed malignancy (2346/4316, 54.4%, 95%CI 52.9–55.8) and just over half of those with malignancy had a Gleason score of 7 or more (1177/2248, 52.3%, 95%CI 50.3–54.4).

Men found to have prostate cancer were, on average, 3.9 years older than those found not to have cancer (95%CI: 3.4–4.4 years). There was a strong relationship between age, increased PSA level and DRE findings (T stage) and the likelihood of a positive biopsy result.

There was no difference in the likelihood of finding cancer in those with 6 cores taken when compared with more than six cores. Those with less than six cores had a greater likelihood of a cancer diagnosis, but tended to be over represented by those with a higher risk of prostate cancer as indicated by higher PSA levels and greater T stage (results not shown but available on request).

A logistic regression model predicted biopsy outcome based on age, PSA and DRE finding.

Table 1. Patient characteristics

Characteristics	Total	Men without cancer	Men with cancer	Proportion with cancer	P
Age					
N with age & outcome	4295	1951	2344	0.54	
Mean age in years (SD)	67.8 (8.7)	65.7 (8.0)	69.6 (8.8)	-	<0.0001 +
<65 years [N(%)]	1508 (35.1)	843 (43.2)	665 (28.4)	0.44	<0.0001 ++
65–74 years [N(%)]	1787 (41.6)	833 (42.7)	954 (40.7)	0.53	
75+ years [N(%)]	1000 (23.3)	275 (14.1)	725 (30.9)	0.73	
DRE					
N with DRE & outcome	3952	1764	2286		
TIC [N(%)]	2487 (62.9)	1428 (85.3)	1059 (46.5)	0.43	<0.0001 ++
T2A [N(%)]	741 (18.1)	210 (12.5)	531 (23.3)	0.72	
T2B/T2C [N(%)]	331 (8.4)	26 (1.6)	305 (13.4)	0.92	
T3/T4 N(%)]	393 (9.9)	10 (0.6)	383 (16.8)	0.97	
PSA					
Number with PSA	3956	1670	2286		
0.0–4.0	195 (4.9)	140 (8.4)	55 (2.4)	0.28	<0.0001 ++
4.1–10.0	2041 (51.6)	1107 (66.3)	934 (40.9)	0.46	
10.2–20.0	916 (23.2)	339 (20.3)	577 (25.2)	0.63	
>20	804 (20.3)	84 (5.0)	720 (31.5)	0.90	
Cores					
N with # of biopsy cores	3291	1138	2153		
<6 [N(%)]	287 (8.7)	52 (4.6)	235 (10.9)	0.82	<0.0001 ++
6 [N(%)]	2686 (81.6)	962 (84.5)	1724 (80.1)	0.64	
>6 [N(%)]	318 (9.7)	124 (10.9)	194 (9.0)	0.61	

+ t-test for two independent samples; ++ Chi-squared test for contingency tables.

Table 2 summarises odds ratios for various PSA ranges, age ranges, and DRE findings using this model. Being under 65 is associated with a slightly reduced risk of malignancy (OR=0.85, p=0.05). Otherwise age is not an independent risk factor for malignant biopsy. PSA level and DRE finding are strong predictors of a malignant biopsy in this multivariable model with approximately a 15-fold change in OR from the lowest to the highest risk category (p<0.0001). Table 2 also shows odds ratios for various age ranges, PSA levels and DRE findings in predicting patients with higher Gleason grade tumours detected on biopsy. Being 75 or older is associated with a small increased risk of having a Gleason score of 7 or more, even taking into account DRE and PSA. Both DRE and PSA are independently associated with the risk of a Gleason score of 7 or more on biopsy.

Using logistic regression, a nomogram was created for predicting a patient's likelihood of having a positive biopsy based on their DRE finding and PSA level. This is shown in Table 3. Separate nomograms were calculated for each age group and are available from the author but because they differed so little, only one overall nomogram is provided here. Model estimates rather than direct observations were used for this nomogram to stabilise values in cells with only small numbers of observations. This provides more accurate estimates in the absence of any evidence of lack of fit for the main effects model (Hosmer-Lemeshow goodness of fit, χ^2=8.92, df=8, p=0.35).

As PSA and DRE were independently associated with higher Gleason grade tumour, we produced a second nomogram for predicting patients with Gleason 7 or greater tumour on biopsy (Table 4).

Table 2. Odds ratios and 95% confidence intervals for age group, serum PSA, and digital rectal examination (DRE) for prediction of malignancy in a joint model (N=3825)[3] and for prediction of a Gleason score of 7 or more (N=3775)[3]

Clinical variable	Level	Malignancy[1]			Gleason ≥ 7[2]		
		OR	95%CI	p	OR	95%CI	p
Age	<65	0.8	0.7–1.0	0.05	0.8	0.7–1.0	0.12
	65–74	1.0	–	–	1.0	–	–
	75+	1.1	0.9–1.3	0.61	1.3	1.1–1.6	0.01
DRE	T1C	1.0	–	–	1.0	–	–
	T2A	4.2	3.4–5.1	<0.0001	4.5	3.7–5.6	<0.0001
	T2B/T2C	10.9	7.1–16.8	<0.0001	7.8	5.9–10.5	<0.0001
	T3/T4	24.2	12.0–48.8	<0.0001	11.7	8.3–16.6	<0.0001
PSA	0.0–4.0	0.2	0.1–0.3	<0.0001	0.3	0.1–0.4	<0.0001
	4.1–10.0	1.0	–	–	1.0	–	–
	10.1–20.0	1.5	1.3–1.8	<0.0001	2.1	1.7–2.5	<0.0001
	20.1+	3.4	2.6–4.6	<0.0001	4.8	3.7–6.2	<0.0001

[1] Malignancy model c=0.77 (c = Area Under the Curve for predictions based on this model).

[2] Gleason≥7 model c=0.84.

[3] 4316 with biopsy outcome but only 3825 also had age, DRE and PSA documented and only 3775 had Gleason score available.

Table 3. Nomogram relating PSA and DRE to positive prostate biopsy. (N=3825)

PSA levels	DRE			
	T1C % (95%CI)	T2A % (95%CI)	T2B or T2C % (95%CI)	T3 or T4 % (95%CI)
0–4.0	12 (9–16)	36 (29–45)	60 (47–72)	77 (61–88)
4.1–10.0	40 (38–43)	74 (70–77)	88 (83–92)	94 (89–97)
10.1–20.0	51 (48–55)	82 (78–85)	92 (88–95)	96 (93–98)
>20.0	71 (65–76)	91 (88–93)	96 (94–98)	98 (97–99)

Table 4. Nomogram relating PSA and DRE to biopsy with Gleason score 7 or greater (N=3775)

PSA levels	DRE			
	T1C % (95%CI)	T2A % (95%CI)	T2B or T2C % (95%CI)	T3 or T4 % (95%CI)
0–4.0	3 (2–4)	11 (7–18)	18 (11–28)	25 (15–38)
4.1–10.0	10 (9–11)	34 (29–38)	46 (39–54)	57 (48–65)
10.1–20.0	19 (17–22)	53 (48–58)	66 (59–72)	75 (67–81)
>20.0	37 (32–43)	73 (68–78)	83 (78–86)	88 (84–91)

Table 5 demonstrates the sensitivity and specificity of using a PSA cutpoint of 4.0 compared to using age adjusted reference ranges and PSA density using a cutpoint of 0.15 for predicting a malignant biopsy. The age-adjusted reference range varies between 2.5 and 6.5 depending on the patient's age. There were 1269 (30%) patients in our cohort who had PSA values between 0.0–6.5. Of these 73% had a benign rectal examination.

Sensitivity, specificity, PPV, NPV, and AUC are all similar for both a single PSA cutpoint and for age adjusted reference ranges. The AUC for PSA density with a cutpoint of 0.15 is substantially higher at 0.72, suggesting that this may be better than PSA alone.

Table 5. Comparison of PSA with single cutpoint (4.0), age-adjusted PSA reference ranges, and PSA density for predicting a positive biopsy result

Measure	PPV (%)	NPV (%)	Sensitivity (%)	Specificity (%)	AUC
PSA ≤4/>4 ng/ml	59.3	71.8	97.6	8.4	0.53
PSA age adjusted[+]	59.7	61.4	93.9	12.2	0.54
PSA density* <0.15/≥0.15	73.8	70.4	76.7	67.0	0.72

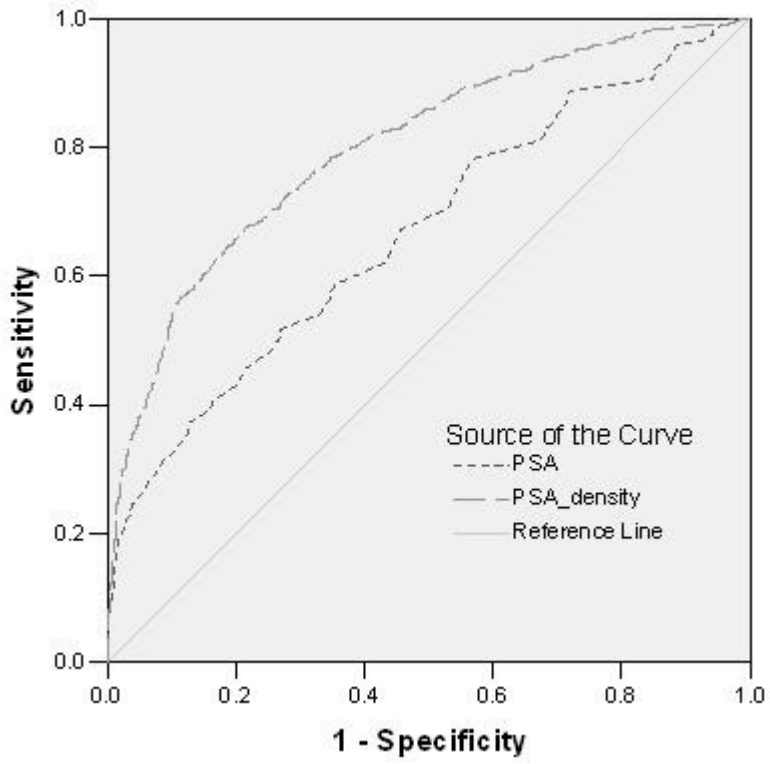
[+] Age adjusted ranges used:

- Age<50, PSA ≤2.5; Age 50-59, PSA ≤3.5; Age 60-69, PSA ≤4.5; Age 70+, PSA ≤6.5
- N=2009 for PSA density data (see text)

Receiver Operating Characteristic (ROC) curves were produced to see if the superior performance of PSA density compared with PSA held across multiple cutpoints or if it arose from the choice of a specific cutpoint. Figure 1 shows a clear advantage for PSA density regardless of cutpoint. The Area Under the Curve for PSA density is 0.80 (0.78–0.82) and for PSA it is 0.67 (0.64–0.69).

There has been no recorded death from a TRUS biopsy in Christchurch over the 15 years that the procedure has been used. The morbidity of the biopsy procedure includes minor bleeding, urinary retention, and sepsis. The bleeding secondary to this procedure has not been formally assessed, but is usually minor and the authors can remember only 5 admissions in the last 15 years, all of whom were managed conservatively without sequelae. Urinary retention secondary to this procedure has not been assessed.

Figure 1. ROC curves for PSA and PSA density (N=2009 for both)



Sepsis has been formally assessed in a separate cohort of 1161 prostate biopsies performed between 1 May 2004 and 30 April 2006. During this period, 28 patients (2.4%) were admitted with sepsis following their biopsy. *E. coli* was isolated on culture from 16 patients, coagulase-negative *Staphylococcus* in 3 patients and *Klebsiella* in 1 case. No organism was able to be isolated in 8 cases. In 6 (21%) of these cases the isolated organism was resistant to ciprofloxacin.

Discussion

There are several publications that contain nomograms relating age, PSA, and DRE to likelihood of a positive prostate biopsy.[7–11] However no nomogram exists for a New Zealand population. Prostate cancer rates vary between different ethnicities.[14,16]

To help New Zealand men in making a decision as to whether to have a prostate biopsy, New Zealand based data would be helpful.

In our cohort, median PSA was 9.1 and 36.4% of patients had a rectal examination suspicious for prostate cancer. Comparable studies[8–11] have a median PSA of 7–10 ng/ml and 27–37% patient's with suspicious rectal examination.

Our rate of positive biopsy is relatively high at 54% compared with 36–52% reported elsewhere. This probably reflects differences in the utilisation of PSA testing in New Zealand.

This study has produced a nomogram relating PSA and DRE to risk of prostate cancer for a New Zealand population. We elected to use a model rather than actual figures

because some cells contained only small numbers of cases and so could easily be skewed. When PSA and DRE were included in a model with age, age was no longer an important predictor of malignancy. Initially we produced 3 different nomograms for varying age ranges but found that the figures in corresponding cells were almost identical in each age grouping. Therefore we elected to leave age out of our nomogram for simplification.

The AUC for our model was 0.77. The AUC for reported nomograms for predicting positive biopsy results varies from 0.69–0.75.[7–11] The AUC for such nomograms can be increased to 0.82–0.91[8,11] by adding in additional predictive factors such as free PSA to total PSA ratios and PSA density. We have presented our model as a look-up table. Most published nomograms are in the form of a graph. We believe a look-up table is simpler to use especially as our nomogram only utilises 2 factors, DRE and PSA.

In our nomogram we have put T2B and T2C digital rectal examination findings in the same group. During the period this data was collected there have been two updates to the TNM staging system for prostate cancer.[17,18] These changes affected T2 tumours with T2A being designated as palpable abnormality on one side of the prostate only, T2B on both sides of the prostate and T2C being removed from the staging classification from 1997–2002.

Prior to and subsequent to this period, T2A was a palpable abnormality involving less than a half of one lobe, T2B involving more than half of one lobe and T2C involving both lobes. The staging for T2B and T2C tumours were corrected to the TNM classification in a retrospective manner, but the T2A tumours were too many to correct. Thus, amongst the T2A DREs may exist a number of T2B DREs, which could potentially overestimate the number of cancers found in patients with this T stage.

Prostate cancer tends to have a protracted course with many of the patients diagnosed with prostate cancer dying of other causes.[14,19] Therefore not all patients warrant active treatment and the clinical decision making process around management must take into account a number of factors, including age, general health, PSA, stage, and the grade of the cancer. All of these factors except grade are readily apparent prior to a biopsy.

As a higher Gleason grade is correlated with an increased chance of a cancer being clinically significant,[20,21] knowledge of the likelihood of a higher Gleason grade could inform the decision to proceed to biopsy or not. In our analysis we have shown that PSA and DRE are not just associated with a positive biopsy but also with higher Gleason grade on biopsy. Therefore we have used our model to produce a second nomogram that predicts likelihood of a patient having a biopsy with Gleason grade 7 or higher prostate cancer.

The number of cores taken at biopsy may have an impact on the number of cancers diagnosed. The false negative rate of sextant biopsy is 30-40%[22]. A recent systematic review[22] found that taking 8 cores increased the number of cancers detected by 19%, compared to sextant biopsy. Taking 12 cores increased the detection rate by 31%.

In our cohort there was no difference in the rate of a cancer diagnosis in men having 6 biopsies when compared to men having more than 6 biopsies. Nevertheless, it is

common practice in New Zealand to perform more than 6 biopsies, and given that sextant biopsies were performed in 81.6% of our patients, it is probable that our nomogram underestimates the proportion of men who will have a positive biopsy, when compared to other regions. Furthermore, these nomograms predict results based only on a first-time biopsy rather than overall rates of prostate cancer diagnosis.

A proportion of men who have a negative biopsy will have prostate cancer diagnosed on subsequent biopsies. It is interesting that in our population there is a greater likelihood of cancer being diagnosed if they had less than 6 cores taken. We assumed this was due to a selection bias with less than 6 cores being taken if cancer was clinically obvious. Statistical modelling confirmed this. It was more likely that a small number of cores were taken if the patient was over 75 years of age, had higher DRE, or high PSA.

PSA testing using a single cutpoint has high sensitivity but low specificity for predicting positive prostate biopsy.[7–10] In our study the AUC for a single cutpoint of 4.0 was 0.53. This is consistent with other reports.[7–10] The very low specificity with such low cutpoints is not surprising, given the clear and strong relationship found between PSA level and biopsy outcome when multiple cutpoints are used (AUC=0.69 when 4 PSA groups are used).

In our cohort 28% of men with PSA less than 4.0 had a positive biopsy. Other papers[9,10,14,23,24] have reported rates between 25–30% leading some to suggest the threshold for biopsy should be reduced to 2.5 ng/mL.[23,24] It is estimated that this would double the number of men undergoing a prostate biopsy.[14] The clinical significance of these tumours detected at a lower PSA is unknown. In this study only 3% of men with normal DRE and PSA less than 4.0 had a tumour with Gleason grade 7 or more.

Several methods have been suggested to improve the AUC for PSA testing. In 1997 Oesterling et al[12] proposed age-adjusted reference ranges as a method for improving the specificity of PSA testing. These were based on the knowledge that PSA levels in all men rise as they age. This paper demonstrates that age-adjusted ranges do not improve the AUC of PSA testing in a New Zealand population. Other studies have also shown limited benefit for age-adjusted ranges.[14,15] It is the belief of the authors that age-adjusted ranges should not be quoted for New Zealand patients.

PSA density measurement using a cutpoint of 0.15 has been shown to improve the AUC compared to using a single PSA cutpoint.[25,26] We have been able to verify this for a New Zealand population. As the specificity is improved, however, the sensitivity reduces. Also, to calculate PSA density requires measurement of the prostate volume which is impractical prior to counselling a patient about prostate biopsy.

The morbidity from prostatic biopsies is low. Our hospitalisation rate for sepsis post TRUS biopsy of 2.4% compares well to reported rates of between 0.6%–4.0%.[27–30] There is likely to be a degree of under-reporting as our study only captures men admitted to Christchurch Hospital but as this is the only local hospital for acute admissions patients would have to have moved out of the region.

Of concern is the large number of ciprofloxacin-resistant organisms that were discovered (21%). Flouroquinolone resistance has been reported elsewhere. Feliciano et al[30] found that 50% of patients who were re-admitted following TRUS biopsy with

sepsis, grew ciprofloxacin-resistant organisms on culture. TRUS biopsy done in an outpatient setting under local anaesthetic without the use of sedation has been shown to be well tolerated by New Zealand men.[31]

Conclusions

This study presents the findings in a large cohort of New Zealand men undergoing TRUS guided biopsies of the prostate. Nomograms are formulated to help inform New Zealand men of their risk of not only having a positive first-time biopsy, but also of having a higher grade cancer detected on this biopsy.

Age-adjusted reference ranges did not improve prediction of cancer in this population, but the use of PSA density may enhance prostate cancer diagnosis.

Competing interests: None known

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Accuracy of prenatal diagnosis in a tertiary fetal medicine unit

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Abstract

Aims To establish the accuracy of prenatal diagnosis in a tertiary referral fetal medicine unit by comparing those diagnoses made prenatally with diagnoses made at birth until discharge, and with postmortem information from cases that resulted in termination, intrauterine, or neonatal death.

Methods All cases seen in the Fetal Medicine Unit between 1 June 2004 and 30 November 2005 were collected prospectively and sorted according to diagnosis. Relevant outcome data for these pregnancies were collected including postmortem information.

Results 681 cases seen which accounted for 1219 visits. 198 were classified prenatally as a major abnormality, 46 cases minor, 56 with raised nuchal translucency, and 381 no abnormality. Outcome details were not available for analysis in two cases. Therefore 679 (99.7%) cases were available (711 out of 713 fetuses). Of the liveborns, 93.6% of the prenatal diagnoses were confirmed, 5.1% were resolved (predominantly soft markers), and 1.3% resulted in an additional major abnormality that had a significant clinical effect. Postmortem examinations were performed on 52% fetal or neonatal deaths with a normal or unknown karyotype. There was one new finding at postmortem that changed the fetal medicine diagnosis significantly.

Conclusions Accuracy of prenatal diagnosis in a tertiary fetal medicine unit is high. Parents and staff need to be aware that not all abnormalities will be detected prenatally, but inaccurate diagnosis is uncommon. Clinical indicators for benchmarking need to be developed.

The offer of prenatal screening and diagnosis for fetal abnormality is now a standard component of routine maternity care. The aim of fetal medicine is to provide accurate diagnosis and information regarding prognosis and management. This accuracy is important to minimise unnecessary anxiety in those with normal or likely normal findings and to streamline ongoing care with accurate information, support, planning and possible therapy, for those with a significant fetal abnormality.[1]

Increasing availability of both ultrasound and overall prenatal screening and diagnosis in pregnancy, has led to concerns related to the amount of information given to women prior to these investigations.[2–4] Furthermore, it has been suggested that anxiety is induced when an abnormality is found but is not confirmed. Although this discussion has particularly related to the diagnosis of soft markers and chromosomal screening,[5,6] it is almost certain that for the woman and her partner, overall accuracy of diagnosis is important to minimise anxiety and to engender confidence in their medical carers. Accuracy of prenatal diagnosis directs care and resources efficiently to those who most require it.

Multiple studies have assessed the accuracy of prenatal ultrasound in unselected low-risk populations,[7–10] with varying results, reflecting the underlying population mix and the quality of ultrasound.

Few studies have assessed the accuracy of ultrasound in a tertiary referral setting.[1–13] These studies had limitations, including a large number of undiagnosed associated abnormalities[11] and a low concentration of abnormalities (36/420 8.6%) less typical of a regional fetal medicine unit,[12] but all show improved detection rates when compared to nonspecialist units.[13]

Two other studies have reported a high degree of accuracy for prenatal diagnosis of fetal cardiac abnormalities within a referral fetal echocardiography service, but these tend to involve lower numbers focusing on the fetal heart alone.[14,15]

We therefore wanted to address the issue of accuracy of diagnosis when a woman is referred to a tertiary fetal medicine unit, so that this information would be available for the women being referred to the unit, and to the referrers themselves.

The aim of this study was to establish the accuracy of prenatal diagnosis by comparing those diagnoses made prenatally to those made after birth until discharge or comparison to postmortem findings following termination or intrauterine or neonatal death.

Methods

All cases seen in the Fetal Medicine Unit in Christchurch, New Zealand, over an 18-month period (1 June 2004 to 3 November 2005) were identified. The Unit is a tertiary referral centre for both its local population and for the majority of the South Island (10,000 maternities total). Tapes from cases with significant cardiac abnormalities were reviewed by a paediatric cardiologist (in another centre) to confirm the diagnosis.

The Unit has strong links with genetics, radiology, neonatology, paediatric surgery, and perinatal pathology. The cases were identified from the Fetal Medicine database (Viewpoint v5.5, GE, Germany), and were sorted according to the prenatal diagnosis made within the Fetal Medicine Unit. Singleton and multiple pregnancies were analysed separately, and then combined. All liveborn outcomes were retrieved from the maternal neonatal notes, neonatal intensive care (NICU) discharge summaries, information from the lead maternity carer (LMC), and/or information from hospitals within the South Island.

The study was submitted to the local ethics committee, but as it conformed to the standards established by the Ethics Guidelines for Observational studies, Observational research, Audits and related activity published by the Ministry of Health, ethics approval was not considered to be required.16

Results

Data—During the 18-month period, 681 cases were seen in the Fetal Medicine Unit, which included 650 cases of singletons, 30 twins, and 1 triplet pregnancy. These accounted for 1219 visits, and 400 procedures including 148 chorionic villus samplings (CVS), 208 amniocentesis, 9 fetal blood samplings (FBS), 17 intrauterine transfusions (IUT), 3 shunts, 6 embryo reduction, and 9 other drainage procedures.

The majority of cases were seen only once in Fetal Medicine, but the median and range of the number of visits was 1 (1–5). The first diagnosis made in Fetal Medicine was used for this analysis, and the majority of these were before 21 weeks gestation. In New Zealand termination of pregnancy for fetal abnormality is up to 20 completed weeks of gestation.[17]

The diagnoses were divided into 4 categories: major, minor, increased nuchal translucency, and normal. The proportions for these categories and their outcomes are shown in Table 1. The major cases included significant structural abnormalities of each organ system (Table 2), and minor cases, referred mainly to soft markers but not solely (Table 3).

Table 1. Classification of type of case seen in 18 months

Variables	All cases	Singleton cases	Singleton outcome					
			Live	TOP	NND	Miscarriage	IUD	Not Known
Major	198	190	106	58	11	6	9	
Minor	46	46	45	0	1	0	0	
Increased NT	56	52	50	1	0	1	0	
Normal	381	362	354	0	2	2	2	2
Total	681	650	555	59	14	9	11	2

NT=nuchal translucency; TOP=termination of pregnancy; NND=neonatal death; IUD=intrauterine death.

The high number of cases classified as normal (56%) in the Fetal Medicine Unit can be explained by referral reasons such as; history of a significant abnormality in a sibling or previous pregnancy, karyotyping for increased maternal age, genetic testing for a positive family history, and monitoring situations such as for complicated twin pregnancies, or high levels of maternal red blood cell (RBC) antibodies.

Overall rate of follow-up available for analysis was 679 out of 681 cases (99.7%); 2 were lost to follow-up as they delivered outside of New Zealand. Including singletons and multiples, this meant follow-up was available for 711 out of 713 fetuses. There were 625 liveborns, which included cases until discharge, either from the maternity ward or NICU, or neonatal death, and 86 fetal losses including miscarriage, intrauterine death, or termination.

Survivors—Of the liveborns, 93.6% of the prenatal diagnoses were confirmed, 5.1% resolved, and 1.3% resulted in an additional major abnormality that had a significant clinical effect (Table 4). Minor diagnoses such as skin tags, skin haemangiomas, and small ventricular septal defects (VSD) that were clinically insignificant at neonatal follow-up, were not included.

The 5.1% of cases that resolved were mainly soft markers, although there was one case of an abdominal cyst which disappeared when a follow-up scan was performed in the third trimester, and after birth there were no clinical consequences. Of the significant additional major abnormalities, these 8 cases are listed in Table 5 and can be divided into 3 categories. The first category (cases 1 & 2), reflect diagnoses made after birth which have no relation to the indication for assessment or findings when seen during pregnancy. These reflect abnormalities which evolve and present either later in gestation or at some time after birth. We would not expect to predict these outcomes.

Table 2. Major abnormalities diagnosed in fetal medicine and outcome in singleton pregnancies (N=190). All confirmed at outcome except for one abdominal cyst)

VARIABLES	Number	Live	TOP	NND	Miscarriage	IUD	Not Known
CNS							
Anencephaly	4		4*				
Holoprosencephaly	2		1	1*			
Ventriculomegaly	6	3	3				
FACE							
Cleft Lip	5	5					
Cleft lip and Palate	3	3					
Micrognathia	1	1					
NECK							
Cystic Hygroma	9		7			2	
Cystic swelling/mass	2	2					
HYDROPS FETALIS	7	2	3*	2			
SPINE							
Myelomeningocoele	10	3	7*				
Hemivertebra	1	1					
Diastematomyelia	1	1					
sacrococcygeal teratoma	1	1					
CARDIAC							
Atrioventricular Septal Defect	1	0	1				
Tetralogy of Fallot	3	2	1*				
Transposition of the Great Arteries	1	1					
SVT	2	2					
Myocardial Hypertrohpy	1	1					
Hypoplastic L heart	1		1				
Hypoplastic RV +TV +VSD	1	1					
Truncus arteriosus	2	1	1*				
Truscupid regurgitation	1	1					
Multiple echogenic foci + other defect	2	1	1				
Situs Inversus	1	1					
Bradyarrhythmia	1	1					
VSD	1	1					
Pericardial effusion + other	2		2*				
THORACIC							
Pleural Effusion	2	2					
Diaphragmatic Hernia	3	0	2*	1			
Thoracic cyst + upper abdo cyst	1	1					
CCAM or Pulmonary sequestration	2	2					
ABDOMINAL WALL							
Exomphalos	8	4	2*	1		1	
Gastroschisis	7	7					
Bladder Exstrophy	1	0	1				
GIT							
Likely oesophageal atresia	1	1					
Abdominal cyst	1	1					
Dilated Bowel	1	1					
Hepatic AVM	1	1					

RENAL							
renal agenesis - bilateral	2		1*	1			
Cyst	1	1					
Hydronephrosis	4	4					
Multicystic kidneys	5	3	2				
Megacystis /urethral obst	2	0	1	1			
Bilat small dysplastic kid + anhy	1	1					
SKELETAL							
Extremities: Talipes Bilateral	11	11					
Extremities: Abnormal arm (Amniotic Band or Raplasia)	2	2					
Skeletal Dysplasia - Likely Lethal	1			1			
OTHER							
Fetal Growth Restriction: Possible Fetal Abnormality	4	2	1*	1			
Fetal Growth Restriction: Likely Placental insufficiency	4	2	1*			1	
Anhydramnios: Unknown Cause	2			1		1	
Severe Polyhydramnios	2	2					
Cord Abnormality: Large complex Cord Cyst	2	2					
Amniotic membrane / band	1					1	
Fetal Anaemia	13	13					
Fetal Demise	7				6	1	
Multiple abnormality unknown karyotype or N	5	2	1	1			
TEST ABNORMALITY							
Multiple abnormality ABN Karyotype	4		2*			2*	
Raised NT or soft marker ABN Karyotype	7	2*	5*				
No marker ABN Karyotype	6	4*	2*				
DNA testing abnormality	4		4				
TOTAL	**190**	**106**	**58**	**11**	**6**	**9**	**0**

*Abnormal karyotype; ABN=abnormal; AVM=ateriovenous malformation; CCAM=congenital cystic adenomatoid malformation; CNS=central nervous system; GIT=gastrointestinal tract; NT=nuchal translucency; RV=right ventricle; SVT=supraventricular tachycardia; TV=tricuspid valve; VSD=ventricular septal defect.

Table 3. List of minor abnormalities and increased nuchal translucency and their outcome in singleton pregnancies. Of the minor abnormalities, 31 resolved and the remainder were confirmed

Variables	Number	Live	TOP	NND	Miscarriage	IUD	Not Known
Enlarged Cysterna Magna	1	1					
Echogenic Bowel	10	9	1*				
Choroid Plexus Cysts + Age >35	12	12					
Echogenic Focus + Age >35	8	8					
Premature Atrial Ectopics	3	3					
Persistent R umbilical vein	1	1					
Talipes Unilateral	3	3					
Fetal growth restriction: likely constitutional	2	2					
Oligohydramnios	3	3					
Cord Abnormality: 2 vessel cord	3	2		1			
Renal pelvic dilatation	2	2					
TOTAL	48	46	1	1	0	0	0
Increased NT	56	50	6*	0	0	0	0

*Abnormal karyotype

Table 4. Accuracy of Fetal Medicine diagnosis compared to outcome according to singleton, multiple and total livebirths (N=625), including neonatal deaths (N=14) (11 additional minor abnormalities were not included as did not make a clinically significant difference on neonatal follow-up assessment)

Singletons	**Number**	**%**
Total Diagnoses Confirmed	530	93.1
Total Resolved by Birth	32	5.6
Total Additional Major	7	1.2
Total singleton Live Births	569	100
Multiples		
Total Diagnoses Confirmed	55	98.2
Total Resolved by Birth	0	0
Total Additional Major	1	1.8
Total multiple Live Births	56	100
TOTAL		
Total Diagnoses Confirmed	585	93.6
Total Resolved by Birth	32	5.1
Total Additional Major	8	1.3

The second category (cases 3 & 4) includes 2 cases where concerns were raised, but a diagnosis was not made. In both cases the mother declined chromosomal testing, of which one was a borderline chromosomal risk assessment, but in the other, highly suspicious case for fetal abnormality, a very mild form of Tetralogy of Fallot was diagnosed postnatally.

The final category (cases 5, 6, 7, 8) include cases in which additional significant abnormalities were identified after birth, which converted the cases from a likely good prognosis to one which was much more guarded due to a syndromic diagnosis. Particularly cleft palate and oesophageal and anal atresia were abnormalities that were difficult to diagnose prenatally.

Case 5 died from Smith-Lemli-Opitz syndrome, but also was challenging for prenatal assessment, because the referral was made in the third trimester, dates were uncertain, and oligohydramnios was present. Cases 6–8 are progressing well, but for Case 8, as yet at 12 months of age, a specific syndrome has not been identified despite considerable genetic input.

Table 5. List of liveborn cases (including neonatal deaths) in singletons and multiples in which an additional abnormality was found following birth. (Divided into additional major and additional minor abnormalities. Cases 1–8 are discussed in detail within the text.)

Case	Fetal Medicine diagnosis	Category	Additional abnormalities found after delivery	Category
1	Choroid Plexus Cysts; Echogenic focus L ventricle	Minor	Craniosynostosis. Premature delivery, surgery planned for 3 months.	Major
2	No obvious signs of fetal anomaly were observed.	Normal	Duodenal web Day 4. Only prenatal scan in Fetal Medicine was at time of CVS.	Major
3	Small fetus, increased NT, no karyotyping	Major	Tetralogy of Fallot. Small VSD 2-3mm with minimal overriding of aorta	Major
4	Borderline Nuchal Translucency. Declined testing.	Normal	Trisomy 21 - Hirschprungs.	Major
5	IUGR, olioghydramnios 3rd trimester referral.	Major	Smith Lemli-Opitz syndrome. Baby died. 3rd trimester scan only in Fetal Med	Major
6	Micrognathia	Major	Likely syndromic. Cleft palate, microgathia confirmed.	Major
7	Hemivertebrae	Major	VATER syndrome. Hemivertebra , anal atresia, tracheo-oesophageal fistula.	Major
8	Talipes - bilateral	Major	Pierre Robin sequence (cleft palate, micrognathia, microstomia), arthrogryposis.	Major
	Fetal growth restriction: possible fetal abnormality	Major	Bilateral polydactyly, L extra digit tied off, R side may require plastic surgery.	Minor
	Gastroschisis	Major	ASD confirmed (4-5mm). Gastroschisis repaired day 1	Minor
	Increased nuchal translucency	NT	Bilateral hydroceles.	Minor
	No obvious signs of fetal anomaly were observed.	Normal	Haemangioma on lip.	Minor
	No obvious signs of fetal anomaly were observed.	Normal	Periauricular skin tag on L ear.	Minor
	No obvious signs of fetal anomaly were observed.	Normal	Bilateral hydrocele.	Minor
	Normal growth and fluid	Normal	Unilateral talipes.	Minor
	Normal growth, fluid and dopplers(UARI)	Normal	Haemangioma on R buttock.	Minor
	normal intrauterine pregnancy	Normal	Membranous VSD 3-4mm. No surgery required.	Minor
	MCMA twins	Normal	Small VSD not requiring closure at 6 months in Twin 2	Minor
	Increased NT Twin 1	Increased NT	Unilateral positional talipes	Minor

ASD=atrial septal defect; IUGR=intrauterine growth restriction; MCMA=monochorionic monoamniotic.

Fetal and neonatal loss—The number of fetal losses (miscarriage, termination, intrauterine death), and neonatal deaths are listed in Table 6. Six of the miscarriages were diagnosed at the time of the first visit, and 6 occurred before 20 weeks gestation (3 fetal loss from multiple pregnancies). Six cases of increased nuchal translucency were shown to have a chromosomal abnormality and underwent termination. The remainder continued their pregnancies with normal liveborns.

The majority of intrauterine and neonatal deaths could be anticipated due to the presence of significant fetal abnormalities, however, several occurred from an unrelated diagnosis, separate to the original fetal medicine referral, including

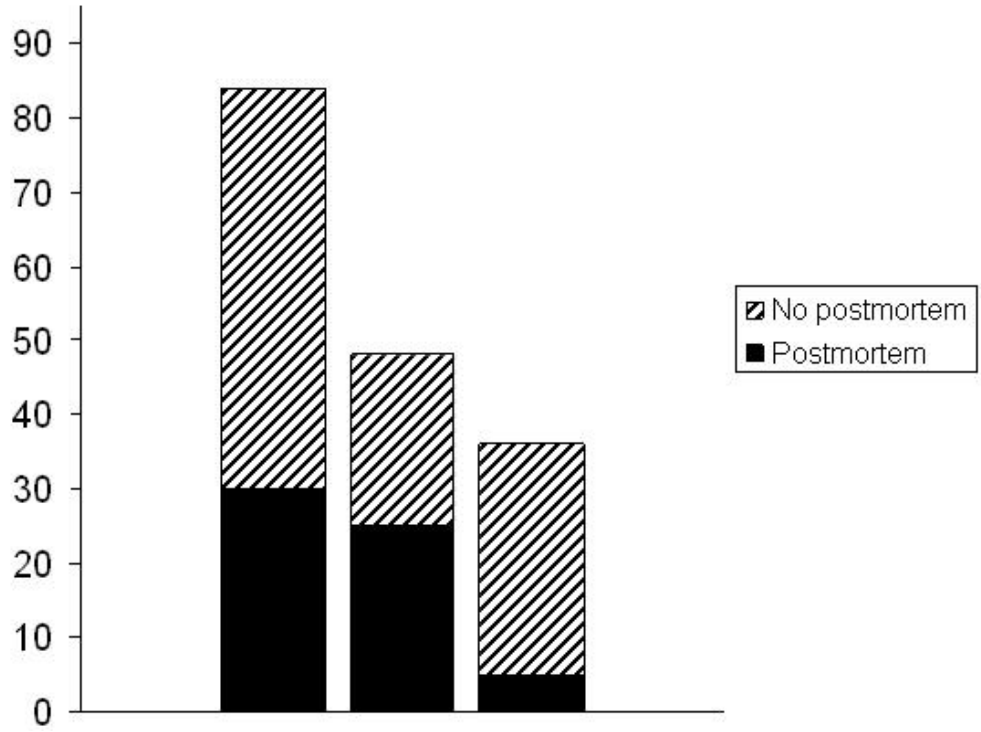
prematurity in multiple pregnancy, and an unexpected acute Herpes simplex virus infection.

Table 6. Number of fetal and neonatal losses listed as miscarriage (Misc), terminations (TOP), intrauterine death (IUD), and neonatal death (NND), postmortem rate (PM)

Pregnancy	Misc	IUD	TOP	NND	TOTAL loss	% all cases	PM rate %
Singletons	9	11	59	14	93	14.3	32.3
Multiples	3	2	2	3	10	15.9	0.0
TOTAL	**12**	**13**	**61**	**17**	**103**	**14.4**	**29.1**

Postmortem examinations were performed on 29% of all the fetal and neonatal losses. However, none of the miscarriages nor multiple pregnancy losses, and only 5 (15%) of the abnormal karyotypes cases had a postmortem performed. This meant that in a singleton pregnancy with a fetal abnormality and a normal or unknown karyotype which resulted in a termination, intrauterine or neonatal death, the post-mortem rate was 52% (Figure 1).

Figure 1. Singleton losses (excluding miscarriages) and proportion of postmortems according to the presence of a chromosomal abnormality or not. Postmortem rates for the 3 groups respectively were 36%, 52%, and 15%



Note: Columns represent total, normal or unknown karyotype, and abnormal karyotype from left to right respectively.

There was one new finding at postmortem that changed the fetal medicine diagnosis significantly; this was the case of Smith-Lemli-Opitz syndrome mentioned above. In addition, there were 2 other new findings from postmortem but which did not change the fetal medicine diagnosis .

The first was a case of acute Herpes simplex infection, 6 weeks subsequent to referral for a different reason, and in another, which underwent termination for multiple abnormalities, additional findings were identified at postmortem including oesophageal and anal atresia. This additional information led to confirmation of VATER syndrome. Overall, the postmortem examinations were important for the finer detail of diagnosis from both macroscopic and histological information, and clearly were important for confirmation of the prenatal findings.

In the cases that did not undergo postmortem examination, there were no obvious missed or incorrect diagnoses. These included:

- All of the multiple pregnancy losses could be explained including early demise of one twin for a particular reason, miscarriage, or early preterm delivery leading to neonatal death,
- 34 out of 93 singleton losses had a chromosomal abnormality diagnosed prenatally, and although only 15% underwent postmortem examination, the chromosomal abnormality was consistent with the initial ultrasound findings,
- The neonatal deaths were examined and investigated prior to death so we can be confident of these diagnoses.

The only group for which further confirmatory information is not available, is for terminations or intrauterine deaths with a fetal abnormality and normal karyotype. Nevertheless the structural abnormalities were so marked in this group that a diagnosis was not in doubt, although postmortem examination would have been helpful both to confirm the abnormality but also to shed light on the underlying cause, particularly in two cases of unexplained fetal hydrops fetalis.

Discussion

These data show that the accuracy of diagnosis in a tertiary fetal medicine unit is high. In this study almost complete follow-up was obtained (99.7%), so that there can be considerable confidence in the results.

The missed additional diagnosis rate of 1.3% is low, and the accuracy of the primary diagnosis is high with few false positive cases. The missed additional diagnoses can be explained by several factors, which include:

- Some abnormalities evolve or appear later in gestation or even after birth—i.e. the natural history of the abnormality may preclude diagnosis at 20 weeks gestation'
- Incidental findings can occur later in pregnancy'
- Parents decline additional testing to make a diagnosis prenatally, and

- Technical limitations make not all abnormalities amenable to detection by ultrasound such as oesophageal or anal atresia, and cleft palate.

Other factors that also limit accurate prenatal diagnosis include third trimester examination, oligohydramnios, and increased maternal body mass index.

The accuracy of diagnosis for fetal and perinatal loss could be considered to be more difficult to assess as the overall postmortem rate was only 29%. However, the rate was 52% in the group where additional investigations or confirmatory evidence were most required.

Of the postmortems that were performed, they confirmed the high degree of accuracy of the prenatal diagnosis, in that there was only one new finding that led to a significant poor outcome that had not been suspected prenatally. There was one additional intrauterine death that was diagnosed at postmortem but due to a new incidental cause, unrelated to the initial fetal medicine referral.

In the cases that did not undergo postmortem examination, there were no obvious missed or incorrect diagnoses. Other studies have confirmed the high accuracy of prenatal diagnosis when correlated with postmortem results,[18–20] but also highlighted the reduced rate of postmortem investigation over the last 10 years.[18]

The latter retrospective study performed in an unselected population showed additional information from postmortem examination was important for genetic counselling in up to 25% of cases.[18] This rate is likely to be less in the present study, where the cases came from a single specialised unit. Nevertheless, ongoing strategies to emphasise the importance of postmortem examination in fetal and perinatal loss continues to occur in our unit, including the introduction of formal guidelines.[21]

We recognise that there are several limitations to this study, including only a moderate sample size, and neonatal follow-up only to the time of discharge. However, as the follow-up rate was high, particularly in the liveborns (99.7%), we consider the results are reliable. Longer term follow-up was not possible without access to a congenital malformation database, which unfortunately does not exist in New Zealand.

Although the results of accuracy from this tertiary fetal medicine unit, is likely to be representative of most tertiary fetal medicine units, individual units will vary in quality. This raises the question of whether all units should produce clinical indicators of quality of results and care, both for their referring population and for benchmarking between units. However, the collection of pregnancy outcomes, especially when the referral population is derived from a wide geographical area with women birthing in many hospitals, is a formidable task, involving time and manpower and cost, and is usually unfunded.

It would be helpful if there were some simple universally accepted standards of clinical outcomes (clinical indicators) that would provide information for the individual unit's referring population, but also to benchmark between units.[22] An alternative approach with large populations which have established fetal medicine, perinatal, and congenital malformation databases is to link these, but clearly this is unlikely to be available for all units, and is easiest to establish in those that have good

geographical boundaries. Certainly electronic records and databases are the only way such information on a large scale can be collected and correlated.

Conclusion

The accuracy of prenatal diagnosis in a tertiary fetal medicine unit is high. However, parents and staff need to be aware, that not all abnormalities will be detected by prenatal assessment, but this is uncommon. Clinical indicators to compare outcomes and results between units need to be developed.

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Obstructing the goal? Hospitalisation for netball injury in New Zealand 2000–2005

Pam Smartt, David Chalmers

Abstract

Aim The last descriptive epidemiological study of netball injuries in New Zealand was published in 1993 (data from 1988). The current study examines the changes in the injury profile of hospitalised netball cases since then.

Method Records of public hospital inpatients discharged in the period 2000–2005 were searched for netball injury cases using a novel search procedure. Data were analysed by demographics, body region, nature/severity of injury, and medical procedure.

Results 1126 cases were identified; 81% were female. The average age was 29 years (5–82 years); 26% of cases were Māori. The highest incidence rate was 325 per 100,000 participants 35–49 years. The population injury rate was 5.0 per 100,000. Forearm fractures predominated in the 0–14 year age-group and Achilles tendon injury in the 15+ age-groups. Six cases (0.5%) sustained a "serious" injury (ICISS score≤0.941), >50% of cases had an injury that would require a "lay-off" period of 3–6 months.

Conclusion The differences highlighted in this study, between hospitalised netball injury in the late 1980s and the present, suggest that (a) the average age of hospitalised netball injury cases may be increasing, (b) forearm fractures in young netball players are a cause for concern, (c) surgical repair of Achilles tendon injuries appears to have increased while knee ligament injuries requiring hospitalisation/surgical repair appear to have decreased, and (d) the indoor version of the game and male players may be important targets for injury prevention.

Netball is a fast, demanding team game characterised by sudden direction changes, rapid acceleration, pivots, and elevating leaps.[1,2] Injury is common and is reported to be one of the main reasons for young people dropping out of the sport.[3,4] Reducing the risk of injury in netball is therefore an important goal for the sport, for New Zealand's Accident Compensation Corporation (ACC) and, more generally, for public health.[5]

For the sport, injury affects performance and participation, for ACC it results in compensation claims, and for public health injury can negate the health benefits of physical exercise.[6]

Netball is the most popular team sport for women in New Zealand (NZ) and is played widely by women of all ages. Participation continues well into the childbearing years when active involvement by females in other sports is generally declining.[7] Netball is popular with Māori who make up 26% of netball participants.[7] The game is also played by a growing number of males,[8] with 81% of male participants involved in the indoor game.[7]

In 1989–1990 there were an estimated 100,000 netball participants in NZ.[9] According to Netball New Zealand, there are now an estimated 200,000 participants: 120,000 registered members and a further 80,000 playing in social competitions nationwide.[8] This estimate is likely to be conservative as in 2003 SPARC[7] estimated that 255,700 New Zealanders (club and non-club members/informal participation) aged 5 years and over played netball.

The last descriptive epidemiological study of netball injuries in NZ was published in 1993 (data from 1988).[9] There have been several significant changes in the sport in that period. Netball became a recognised Olympic sport in 1995 (making its inclusion in future Olympics possible), and has grown in popularity in NZ both as a participant and a spectator sport. It is acknowledged that netball today is a faster, more physical game and that there is a closer contest for the ball. Attitudes toward sports injury have also changed and there has been a national netball injury preventive programme *NetballSmart* in place since 2005.[10]

The aim of this study was to examine the current profile of hospitalised netball injury and identify any the changes that have occurred since 1988. We have limited the study to injury treated in the public hospital system because our main focus is serious acute injury.

Methods

Study design and definitions—This study was undertaken as part of a larger study of hospitalised injury across 187 sporting and recreational activities.[11] A novel search procedure was developed to identify injury cases associated with specific activities. The procedure comprised a search of both coded and narrative information in a linked hospital discharge and ACC entitlement claim dataset.

Sporting and recreational activity was defined as any pastime or game requiring physical effort undertaken for "amusement, diversion or fun" (Concise Oxford Dictionary). NZ's National Minimum Dataset (NMDS) changed from ICD version 9 to ICD version 10-AM in 1999, with the first full year of implementation in 2000. There were subsequent updates in 2001 and 2004. With no single year likely to be free from the effect of coding changes, all of the available data coded to ICD version 10-AM was used to obtain proportions for comparison with the earlier study.

Datasets—NZ's National Minimum Dataset (NMDS) for 2000–2005, coded to three editions of ICD-10-AM (ICD-10-AM-1-3), was the primary data source. The ability to identify sport/recreational injury cases directly from these data varied according to the edition of ICD-10-AM applying at the time (e.g. individual sports were not recognised in ICD-10-AM-1[12]), and the hospital providing the data (e.g. narratives describing the circumstances of injury were not available for some hospitals).

To improve case ascertainment and sport-specific injury information, the NMDS data were linked to ACC sport/recreational injury entitlement claims data (2000-2005). The probabilistic linkage program AUTOMATCH[13] was used to link the two datasets. Data were blocked into subsets of similar records and the records in each block matched on common variables which included National Health Index number, surname, first name, injury day and second initial. After each pass, successful matches were removed and the procedure repeated using a new set of match variables on the residual.

Approval for the use of these datasets in this way was provided by the Lower South Island Regional Ethics Committee (ref. OTA/99/02/008 & OTA/01/07/049) and the ACC Research Ethics Committee (ref.45).

Cases—Cases comprised discharges from public hospitals in the period 2000–2005 with an e-code in the discharge record. For the purposes of injury prevention, reporting is commonly restricted to diagnoses in ICD-10-AM Chapter XIX "Injury poisoning and certain other consequences of external causes" (diagnoses S00-T98).[12, 14, 15] Because a number of sport and recreational injury diagnoses fall outside this chapter (e.g. musculoskeletal injuries, M00-M99), this restriction was not applied. However, injury diagnoses in the range A00-R99 (Chapters I-XVIII) are reported separately.

Although our original intention was to include injury arising from sports-specific paid-work, limited recording of occupation in the NMDS dataset precluded this and so cases coded as "paid work" were excluded. Other exclusions included day-patients (these are inconsistently recorded in the NMDS), readmissions for the same injury, cases of poisoning, self-harm, assault, and complications of medical and surgical care. ICD-10-AM classification groupings were used in the reporting of cases by body region, diagnosis and procedure. So for example, cases with "knee and lower leg" injury comprised all injuries with an ICD-10-AM diagnosis code in the range S80-S89. Where cases were reported according to medical procedure type, the first recorded procedure was used to group cases according to the invasiveness of the procedure. This classification separates cases undergoing ligament and tendon "repair" from those undergoing more invasive "reconstruction" procedures.

Identification and assignment of cases—The linked NMDS/ACC dataset, comprising 328,802 first-time admissions for injury, was the starting point for the search.

Separate searches were undertaken which identified cases with:

- An ICD-10-AM-1-3 sport activity code or an e-code likely to be associated with sport or recreation,
- A sport or recreational activity-specific keyword appearing in the NMDS narrative,
- An ACC sport code, or
- A relevant keyword appearing in the ACC narrative.

A total of 56,144 new cases of sport and recreational injury were identified using these procedures. Cases were assigned to sport/recreational activities according to ICD-10-AM-3 activity codes U50-U72.[14] Two netball codes are available in this classification: "Indoor Netball" (six players and excluding traditional netball played indoors) and "Netball, other and unspecified".[14]

For the current study, cases classified to these two codes were extracted from the master dataset of 56,144 new sport and recreational injury in-patient cases. Conflicts between the datasets in the identification and assignment of cases were resolved by applying a hierarchy of preferences with (a) as the highest priority and (d) as the lowest.

Injury rates—Population estimates were obtained from Statistics New Zealand for the calculation of age-specific population rates. There were no reliable estimates of current participation in netball by age and gender. Participation was therefore estimated by applying the proportions of netball participants reported in the New Zealand National Surveys of Sport and Physical Activity (NZPAS), 1997–2001,[16] to the average usually-resident population for 2001. These surveys were carried out by the National Research Bureau (NRB) for SPARC and gathered information about participation in sport and leisure activities through household surveys of NZ residents.

Thus the estimates used for calculating incidence rates were for participation in netball activity, not membership of netball clubs/associations; the former being likely to have come from the same population as the hospitalisation data which makes no distinction between members and non-members of clubs/associations.

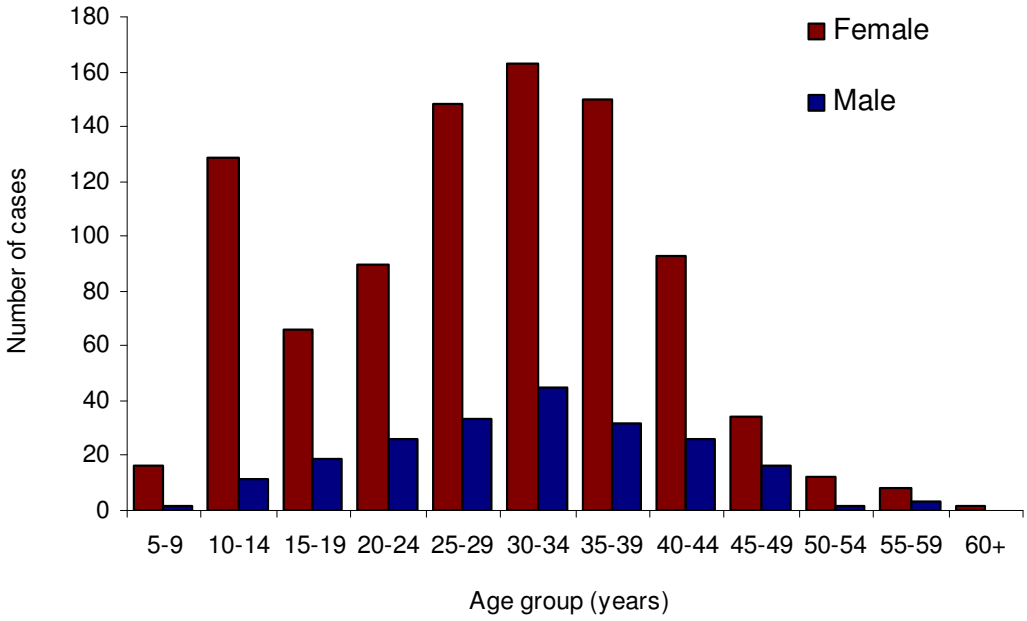
Injury severity—For the purpose of this study a serious non-fatal injury case was defined as a case with an ICD-based Injury Severity Score (ICISS) of less than or equal to 0.941,[17] i.e. cases whose injuries at admission give them a survival probability of 94.1% or worse (probability of death in hospital of at least 5.9%). The ICISS score for each case was computed as the product of the survival risk ratio (using survival probabilities based on 1999–2001 mortality data) for each of their injuries individually derived.[18]

Results

56,144 (17%) of all injury cases were attributable to sport or recreation; 1126 (3%) of these cases were attributable to netball: 911 (81%) female and 215 (19%) male. The average age at injury was 29 years (range 5–82 years); males were slightly older (31 years, 7–59 years) than females (29 years, 5–82 years). Europeans comprised the largest ethnic group (61%), followed by Māori (26%). Pacific Island peoples accounted for 5% of cases.

Almost all cases (99%, N=1113) occurred in the 5–54 year age-range, with frequencies peaking in the 30–34 year age-group for both sexes (208 cases: 163 female, 45 male); see Figure 1. There was also a notable peak for females in the 10–14 year age-group (129 cases).

Figure 1. Incident hospitalisations due to netball by age-group and gender (New Zealand 2000–2005)



Assuming a participation rate of 7% and an average annual population of 3,974,483 for the period (2000–2005), the crude participant injury rate for regular players (i.e. adults and children who had played netball in the last 4 weeks) was 123 per 100,000 per year.

The highest injury rate was 325 per 100,000 participants per year for the 35–49 year age-group. The participant injury rate increased with age. The population injury rate was 5.0 per 100,000.

Body region and nature of injury—Across all diagnoses, the lower leg/knee (683 cases, 61%), forearm/elbow (151 cases, 13%), wrist/hand (91 cases, 8%), and the ankle/foot (73 cases, 6%), were the most commonly injured regions; see Table 1.

As far as could be determined from the coded data, injuries coded as lower leg/knee (S80-S89) included 73 cases (6% of all 1126 cases) with primary diagnoses relating to the knee.

Table 1. Incident hospitalisations due to netball injury by body region and nature of injury (New Zealand 2000–2005)

Nature of injury	ICD-10-Am Body region codes (S00-S99)													
	Abdomen, lower back, lumbar spine, pelvis S30-S39	Ankle, foot S90-S99	Elbow, forearm S50-S59 †	Head S00-S09	Hip, thigh S70-S79	Knee, lower leg S80-S89 ‡	Multiple regions T00-T07	Neck S10-S19	Shoulder, Upper arm S40-S49	Thorax S20-S29	Wrist, hand S60-S69	Unspecified, , NOS, other	Grand Total	% of all injuries
ICD-10-AM Chapter XIX: "Injury poisoning and certain other consequences of external causes" (Injury diagnoses)														
Muscle, tendon injury	1	2	0	0	3	509	0	0	2	0	1	0	**518**	**46**
Fracture	1	18	147	7	15	91	0	0	9	1	74	0	**363**	**32**
Sprain/strain *	4	21	1	0	1	40	1	4	0	0	4	0	**76**	**7**
Dislocation	1	20	1	0	0	3	0	0	6	0	9	0	**40**	**4**
Other	2	3	0	7	4	3	0	3	0	0	0	0	**22**	**2**
Concussion	0	0	0	18	0	0	0	0	0	0	0	0	**18**	**2**
Contusion, superficial injury	2	1	0	5	0	0	0	2	0	1	1	0	**12**	**1**
Internal injury	2	0	0	0	0	0	0	0	0	1	0	0	**3**	**0**
Unspecified injury	0	0	0	0	0	0	0	0	0	0	0	3	**3**	**0**
Open wound	0	1	0	1	0	0	0	0	0	0	0	0	**2**	**0**
Nerve injury	1	0	0	0	0	0	0	0	0	0	0	0	**1**	**0**
Vein	0	0	0	0	0	1	0	0	0	0	0	0	**1**	**0**
Total	**14**	**66**	**149**	**38**	**23**	**647**	**1**	**9**	**17**	**3**	**89**	**3**	**1059**	**94**
ICD-10-AM Chapters I-XVIII: A00-R99 (Non-injury diagnoses)														
Diseases of the musculoskeletal system [a]	4	6	2	0	1	16	0	0	3	0	2	0	**34**	**3**
Diseases of the circulatory system	0	0	0	0	0	12	0	0	0	0	0	1	**13**	**1**
Diseases of the skin and subcutaneous tissue	1	1	0	0	0	8	0	0	0	0	0	1	**11**	**1**
Diseases of the genitourinary system	2	0	0	0	0	0	0	0	0	1	0	1	**4**	**0**
Endocrine, nutritional and metabolic disorders	0	0	0	0	0	0	0	1	0	0	0	0	**1**	**0**
Mental and behavioural disorders	0	0	0	0	0	0	0	0	0	1	0	0	**1**	**0**
Diseases of the respiratory system	0	0	0	0	0	0	0	0	0	1	0	0	**1**	**0**
Disease of the digestive system	0	0	0	0	0	0	0	0	0	0	0	1	**1**	**0**
Symptoms and signs [b]	0	0	0	0	0	0	0	0	0	0	0	1	**1**	**0**
Total	**7**	**7**	**2**	**0**	**1**	**36**	**0**	**1**	**3**	**3**	**2**	**5**	**67**	**6**
Grand Total	**21**	**73**	**151**	**38**	**24**	**683**	**1**	**10**	**20**	**6**	**91**	**8**	**1126**	**100**
% of all injuries	**2**	**6**	**13**	**3**	**2**	**61**	**0**	**1**	**2**	**1**	**8**	**1**	**100**	

† This group includes injury to the distal areas of the radius and ulna; ‡ This group includes injury to the maleolus or 'ankle bone" and injury to the Achilles tendon; * Including avulsion and traumatic rupture of joint cartilage, capsule, and ligament; [a] Includes connective tissue; [b] Includes abnormal clinical and laboratory findings NEC.

The most common diagnoses were ACL rupture (22 cases), various disorders of the patella (17 cases), derangement and tears of the meniscus (12 cases), sprains and strains of collateral and other unspecified ligaments of the knee (9 cases), and chronic instability of the knee (5 cases). The remaining 8 cases had general or nonspecific conditions. Lower leg/knee injuries also included 500 cases of injury to the Achilles tendon (S86.0). A number of other strains and sprains and ruptures (22 cases) of the ankle area were coded as ankle/foot (S90-S99).

The distribution of cases by body region was similar for males and females except for the forearm/elbow (4% of all male vs. 15% of all female cases) and the ankle and foot (9% of all male vs. 5% of all female cases). For cases with a diagnosis in the range S00-T98, muscle and tendon injuries were the most common (46%, 518/1126 cases), followed by fractures (32%, 363/1126 cases); see Table 1.

Injury to the Achilles tendon (S86.0) was the most common individual diagnosis (500/1126 cases, 44.4%); followed by fractures of the proximal phalanx (34/1126, 3.0%), lower end of the radius (29/1126 cases, 2.6%) and lateral malleolus (29/1126 cases, 2.6%).

Five of the top 10 primary diagnoses were fractures of the distal radius. For cases with primary diagnosis codes in the range A00-R99 (67 cases), 35 cases (52%) had musculoskeletal injuries (M00-M99) including chronic instability and derangements of the ligaments of the knee, recurrent dislocations, nonunion/malunion of fractures; a further 12 cases (18%) had post trauma deep vein thrombosis of the lower extremities.

Age and the nature of injury—The distribution of injuries across age-groups by body region (detailed for injury diagnoses, S00-S99) is shown in Table 2. Injury to the forearm/elbow was the most common injury in the 0–14 year age-group (83 of 158 cases, 53%), while knee/lower leg injury was the most common injury in the 15–24 year age-group (98 of 201 cases, 49%), 25–54 age-group (523 of 753 cases, 69%) and the 55+ age-group (6 of 13 cases, 46%).

Wrist/hand injuries were relatively constant throughout the age-groups at 8–9%. The proportion of "non-injury" primary diagnoses varied across the age-groups, and (leaving aside 2 cases in the 55+ age- group), peaked at 9% in the 15–24 age-group.

The distribution of injury types across age-groups is shown in Table 3. Fractures dominated in the 0–14 year age-group (123/158 cases, 78%), in particular, fracture of the forearm (82 cases). Injury to the muscle and tendon dominated in the 25–54 year age-group (455 of 754 cases, 60%) and these were predominantly to the Achilles tendon (439 cases). Injury in the 15-24 year age-group was more evenly distributed between fractures (59 cases, 29%), muscle/tendon injury (57 cases, 28%) and strains/sprains (34 17%).

Overall, the most common diagnoses were Achilles tendon injury (S86.0, 470 cases) with a median age of 34 years, and forearm fracture (S52, 147 cases) with a median age of 13 years.

Table 2. Incident hospitalisations due to netball by age group and body region (New Zealand 2000–2005)

Body region	Age group (years)								all ages	%
	0-14		15-24		25-54		55+			
Head (S00–S09)	10	6%	10	5%	17	2%	1	8%	38	3%
Neck (S10–S19)	2	1%	4	2%	3	0%	0	0%	9	1%
Thorax (S20–S29)	0	0%	0	0%	3	0%	0	0%	3	0%
Abdomen, lower back, lumbar spine, pelvis (S30–S39)	1	1%	8	4%	5	1%	0	0%	14	1%
Shoulder, upper arm (S40–S49)	3	2%	1	0%	13	2%	0	0%	17	2%
Elbow, forearm (S50–S59)	83	53%	11	5%	54	7%	1	8%	149	13%
Wrist, hand (S60–S69)	12	8%	18	9%	58	8%	1	8%	89	8%
Hip, thigh (S70–S79)	9	6%	2	1%	11	1%	1	8%	23	2%
Knee, lower leg (S80–S89)	20	13%	98	49%	523	69%	6	46%	647	57%
Ankle, foot (S90–S99)	10	6%	29	14%	26	3%	1	8%	66	6%
Multiple body regions, complications of trauma	1	1%	1	0%	2	0%	0	0%	4	0%
Non-injury primary diagnoses (A00-R99)	7	4%	19	9%	39	5%	2	15%	67	6%
All regions	**158**	**100%**	**201**	**100%**	**754**	**100%**	**13**	**100%**	**1126**	**100%**

Table 3. Incident hospitalisations due to netball by age-group at admission and nature of injury (New Zealand 2000–2005)

Nature of injury	Age group (years)								All ages	
	0-14		15-24		25-54		55+			
Injury to muscle and tendon	0	0%	57	28%	455	60%	6	46%	518	46%
Fracture	123	78%	59	29%	178	24%	3	23%	363	32%
Sprain strain	6	4%	34	17%	35	5%	0	0%	75	7%
Non-injury diagnoses †	7	4%	19	9%	39	5%	2	15%	67	6%
Dislocation	5	3%	14	7%	20	3%	1	8%	40	4%
Intracranial	6	4%	5	2%	6	1%	1	8%	18	2%
Other and unspecified	8	5%	8	4%	11	1%	0	0%	27	2%
Superficial/contusion	2	1%	3	1%	7	1%	0	0%	12	1%
Open wound	0	0%	1	0%	1	0%	0	0%	2	0%
Nerve injury	0	0%	1	0%	0	0%	0	0%	1	0%
Injury to internal organs	1	1%	0	0%	2	0%	0	0%	3	0%
All injury	**158**	**100%**	**201**	**100%**	**754**	**100%**	**13**	**100%**	**1126**	**100%**

† Primary diagnoses in the range A00-R99

Operations and procedures—168 cases (15%, and including 31 cases of injury to the Achilles tendon) had no coded procedures (standard procedures such as X-rays and plaster applications are not usually coded)

The remaining cases (958) underwent 166 different procedures.[12,14,15] The type of *first recorded procedure* (usually the procedure performed for treatment of the principal diagnosis), by body region is shown in Table 4.

Table 4. First recorded procedures performed in new inpatient cases hospitalised for netball injury (New Zealand 2000–2005)

First recorded ICD-10-AM coded procedure type (grouped according to the invasiveness of the procedure)	Injury diagnoses (Chapter XIX ICD-10-AM, codes S00-T98)													
	Head (S00-S09)	Neck (S10-S19)	Thorax (S20-S29)	Abdomen, lower back, pelvis (S30-S39)	Shoulder, upper arm (S40-S49)	Forearm, elbow (S50-S59)	Wrist, hand (S60-S69)	Hip, thigh (S70-S79)	Knee, lower leg[a] (S80-S89)	Ankle, foot (S90-S99)	T - diagnoses	Other diagnoses A00-R99	Total all diagnoses	%
Repair [b]	2	0	0	0	0	0	1	1	446	1	1	0	452	40%
Open/closed reduction, +/- fixation[c]	5	0	0	0	11	134	72	8	67	23	0	4	324	29%
Other procedures	0	0	1	1	2	5	7	4	28	9	1	19	77	7%
Reconstruction[d]	1	0	0	0	0	0	0	0	32	2	0	7	42	4%
Arthroscopy/washout/debridement	0	0	0	0	1	0	0	2	10	0	0	4	17	2%
Discectomy	0	0	0	1	0	0	0	0	0	0	0	2	3	0%
Immobilisation/stabilisation	0	0	0	0	0	1	0	0	5	2	0	3	11	1%
Imaging only (CT/MRI)	8	3	0	5	1	1	2	1	3	5	1	2	32	3%
No coded intervention (blank ops)	22	6	2	7	2	8	7	7	56	24	1	26	168	15%
Total all cases	**43**	**9**	**3**	**14**	**28**	**283**	**161**	**31**	**714**	**89**	**4**	**71**	**1126**	100%

[a] The ICD-10-AM classification includes injury to the Achilles tendon in this group; [b] 97% (437 cases) of repairs were to the Achilles tendon; [c] 52% (160 cases) open reduction with or without fixation, 42% (130 cases closed reduction, 5% (14 cases) fixation only, unspecified 2 (1%).; [d] 27 reconstructions of the knee (22 with a primary diagnosis involving the ACL), 11 reconstruction of the Achilles tendon. Note: average number of procedures in cases with operations is 2 (1-8), (958 cases), 168 cases had no **coded** procedures; application of plaster is not a coded procedure.

Surgical repair was the first recorded procedure in 452 (40%) cases, mostly (437 cases) to the Achilles tendon. A further 324 (29%) cases had open or closed reduction and/or fixation of a fracture to the forearm (134 cases), lower leg/ankle (90 cases), wrist/hand (72 cases) or other areas (28 cases). "Other procedures" (77 cases) included wound drainage, removal of loose bodies in the joint, aspiration and other open procedures. Forty-two cases (4%) received more invasive reconstructive surgery to the knee (27 cases), Achilles tendon (13 cases), and hand and orbit (2 cases).

Of the remaining 140 cases, 32 had a CT or MRI scan, 17 had an arthroscopic washout and/or debridement, 11 had immobilisation or stabilisation procedures, and 3 cases had a discectomy.

Mechanism of injury—"Overexertion and strenuous or repetitive movements" was the most common mechanism of injury (517 cases, 46%), followed by a fall (314 cases, 28%); see Table 5. Narrative information suggested that falls were often the result of a heavy or bad landing after a jump and that "overexertion" was also associated with leaping, jumping and landing; there are no ICD-10-AM codes that adequately capture these "pre-fall" incidents or underlying mechanisms of injury.

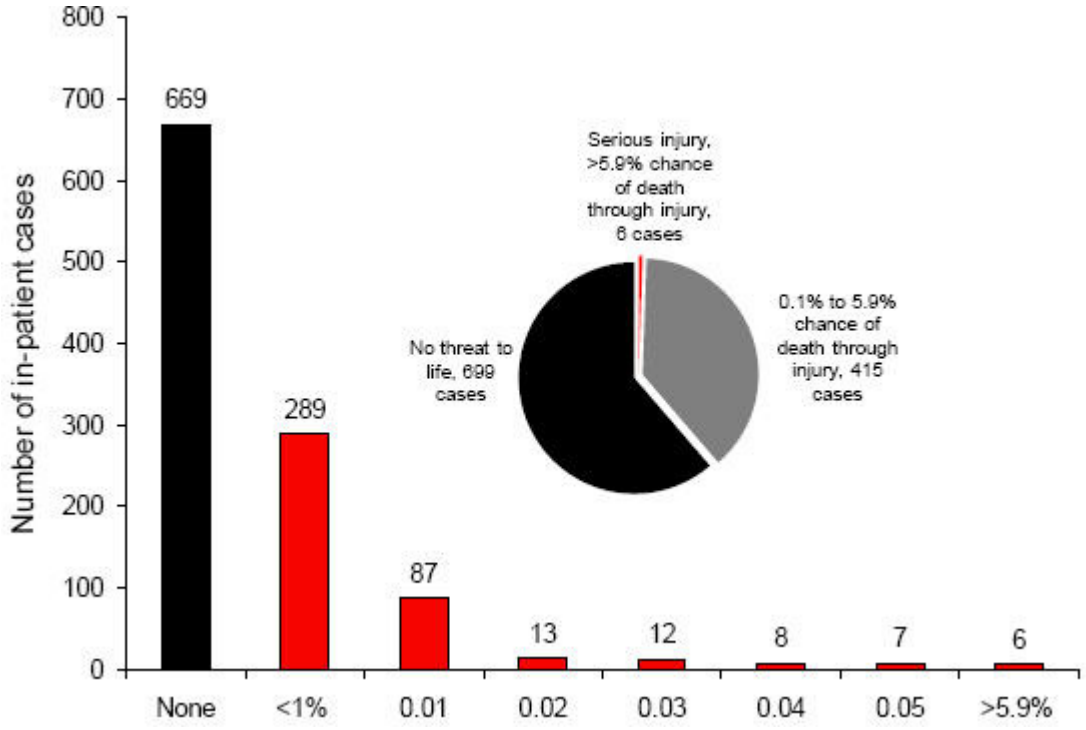
Table 5. Incident hospitalisations due to netball injury by mechanism of injury (New Zealand 2000–2005)

Activity	Cases	%
Overexertion/strenuous /repetitive movements	517	46%
Falls, all types	314	28%
Falls - Collision, Pushing, or Shoving	51	
Falls - One Level to Another	6	
Falls - Slipping, Tripping, or Stumbling	127	
Other Falls	130	
Struck By/Against	161	14%
Other mechanisms	134	12%
All cases	1126	100%

Injury severity and length of stay (LOS)—Netball injury posed no "threat-to-life" (ICISS=1) in 669 cases (59%), while in 422 cases (37%) there was a threat-to-life of between 0.05% and 13.44% (ICISS scores 0.995-0.942); an ICISS score could not be calculated for 35 cases; see Figure 2.

The "threat-to-life" was less than 5.9% (ICISS score >0.941) in 416 cases. In the remaining 6 cases the injury sustained was deemed as a "serious threat to life" (ICISS score <=0.941 i.e. cases whose injuries at admission gave them a survival probability of 94.1% or worse or a probability of death in hospital of at least 5.9%), using the NZIPS definition of serious injury.[17] These 6 cases all sustained fractures, 3 to the head/neck region and 3 to the femur; 5 were the result of a fall. The average age of players with a "serious injury" was 41 years, the gender split was even and the average length of stay (LOS) was 4.8 days. The average LOS for all 1126 cases was 2.3 days (range 1–21 days).

Figure 2. Incident hospitalisation due to netball injury by ICISS score (New Zealand 2000–2005)



Discussion

While injury incidence rates are lower in netball than in many other sports, the popularity of the game and its ability to keep females actively engaged in sport make it an important target for injury prevention.

The last descriptive epidemiological study of netball injuries in NZ, for injury data in 1988, was published in 1993.[9] In that study, hospitalised injury per 100,000 population per year was 4.3, the hospitalised injury per 100,000 participants per year was 143, the gender split was 89.5% female to 10.5 % male, the commonest sites of injury reported were the ankle (52.4%) and knee (18.9%), and the commonest ages at injury were 26, 25 and 17 years. The modal length of stay in hospital was 2 days and the proportion of serious injuries (AIS≥3) was 9%. Netball accounted for 3.1% of hospitalised sports injury and cost ACC $1.7 million. These results were based on day and inpatient cases hospitalised in 1988.

There are several notable points of difference between the earlier study[9] and the current report. The proportion of male netball players hospitalised increased from 10% (1988) to 19% (2000-2005). This increase is consistent with an increase in the proportion of male participants; the SPARC physical activity survey of 1997–2001 reported that 21% of netball participants were male. A recent study of netball hospitalisation in Australia[19] reported that 12% of hospitalised injury cases were male (participation rate 13.4%).

The importance of differentiating indoor from outdoor netball injury was acknowledged in the earlier study by Hume but injury coding at the time did not allow this distinction to be made. ICD-10-AM-3 (introduced July 2004) distinguishes "indoor" 6-player netball from "other netball" (including "traditional" netball played indoors). In 2005, the first full year of discharges coded to ICD-10-AM-3 which distinguished different types of netball, "indoor" netball accounted for 27% of cases, with an even gender split and an average age at injury of 34 years. In "other netball" females predominated (84%) and the average age of injury was 26 years.

Hume reported that 17, 25, and 26 year-olds were the most frequently hospitalised ages (5.6%), whereas in the current study 30, 31, and 32 year-olds were most frequently hospitalised ages (3.8-4.4%). One possible explanation for the apparent difference is the inclusion of day-cases in the earlier study—a recent Australian study,[19] however, reported the peak age for netball hospitalisations as 25–34 years lending support to the impression that the average age of hospitalised injury cases may be increasing.

Netball has been characterised as a game prone to knee and ankle injury, with knee injury highlighted because of the high cost and associated disability.[5,9,20,21] Only 6% of hospitalised injury in the current study involved the knee, compared to 19% reported by Hume. It is not possible to determine if knee injury (and in particular ACL rupture) has become less common or if differences between studies are due to changes in treatment and management since 1988. There is anecdotal evidence to suggest that same-day arthroscopic reconstruction of the knee carried out in private hospitals under ACC cover may account for the difference.

There also appear to be large differences between the two studies in the proportion of ankle injuries reported. The most likely explanation is that Achilles tendon injury was classified as "ankle injury" in the earlier study and as "Injury to the knee and lower leg" in the current study. Because the mechanisms of injury are likely to be very different for Achilles injury and ankle sprain/strain, reporting specific diagnoses is arguably more important than general "body areas" or injury "type" for targeted injury prevention.

Forearm fractures were much less frequent in the earlier study (5 cases, 3.5%) than the present study (149 cases, 13%); with the young median age (12 years) of cases in the current study being of concern. There was also a notable overall injury peak (129 cases) for females in the 10-14 year age-group in the current study that was not observed in the earlier study. There are a number of possible reasons for the increases. It has been argued elsewhere that children are starting competitive sport too young, increasing their risk of injury;[22] 70% of children presenting to Australian emergency departments with a netball injury sustained the injury during formal competition.

Changes in the tempo and physicality of the game since 1988, and the increase in indoor netball and the type of game played on indoor courts, may also have increased the risk of injury. However, it is also possible that the success of the national netball team, the silver ferns, in the 2000s has increased the popularity of the sport with young females and the observed number of injuries in this group.

Injury rates varied between the studies but not appreciably. Census data were used in both studies to calculate population injury rates. Participant injury rates were

calculated using estimates from two physical activity surveys—i.e. the Life in New Zealand (LINZ) study and the New Zealand Sport and Physical Activity Survey (NZSPAS). A slightly higher population injury rate (5.0 vs. 4.3 per 100 000) was reported in the current study but a slightly lower participant injury rate (123 vs. 143 per 100 000).

Netball injury accounted for only 3% of all sports injury in both studies. There were few cases in either study where injury posed a high threat to life and the most frequent LOS was 2 days in both studies. The proportion of "serious" injuries was 9% in the earlier study compared to <1% in the current study, however, different severity measures and thresholds were used in each study. A case-by-case examination of injuries in the current study suggested that approximately 90% of players with lower leg injuries could be "out of action" for 3-6 months. In the sports injury setting, a severity measure based on a "threat of disability" rather than "threat to life" is required.

There were a number of study limitations. In restricting eligible cases to in-patients our population is not representative of all netball-related injury cases; however, it is likely to represent cases with the greatest consequences for the health system and the individual. Routinely collected census and sport participation data were used to estimate injury incidence rates in both studies. However, although the sport participation surveys (LINZ, NZSPAS) were conducted in a similar manner and were funded by the same organisation (Hillary Commission/SPARC) they were conducted at least 10 years apart and were subject to the limitations associated with such surveys;[23, 24] the reported rates therefore should be interpreted with caution.

Conclusion

Netball is a relatively safe sport;[2] however, the physical demands of the game and its popularity have grown. The differences highlighted in this study, between hospitalised netball injury in the late 1980s and the present, suggest that (a) the average age of hospitalised netball injury cases may be increasing, (b) forearm fractures in young netball players are a cause for concern, (c) Achilles tendon injuries and/or the surgical repair of these injuries appears to have increased while knee ligament injuries requiring hospital admission/surgical repair appear to have decreased, and (d) the indoor version of the game and male players may be important targets for injury prevention.

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(Non)regulation of marketing of unhealthy food to children in New Zealand

Caroline Shaw

Abstract

Three and a half years ago an editorial in the *NZMJ* called for restrictions on marketing of unhealthy food to New Zealand children. This paper discusses progress since then. There has been a seemingly relentless documentation of adverse health consequences of the obesity epidemic in the intervening years, increasing evidence that marketing of unhealthy food contributes to the epidemic, growing knowledge about New Zealand children's exposure to marketing of unhealthy food, and evidence of public support to decrease children's exposure to marketing of unhealthy food. Yet there is still a lack of substantive action on the restriction of marketing of unhealthy food to children in New Zealand.

New Zealand (NZ) continues to be saturated by research documenting the presence of overweight and obesity in adults and children, and by research looking at the serious impacts of these conditions on individual and population health.

Three and a half years ago, in an editorial in this journal, Carolyn Watts and Rob Quigley issued an election year challenge to regulate marketing of unhealthy food to children, as a step towards addressing obesity.[1] In the interim we have had two elections, a Select Committee Inquiry into these issues, and a Government response to the Select Committee report,[2,3] so it seems an appropriate time to revisit these issues and mark progress.

Child overweight and obesity statistics in NZ are so often cited that they now cause little reaction, but the facts still bear repeating. In NZ, approximately 61,800 children aged 2–14 years are obese (8.3%) and 155,000 (20.9%) are overweight.[4] Like most conditions in NZ, overweight and obesity are not equitably distributed among different groups of children.[4,5]

International longitudinal studies suggest that 40–80% of children who are obese at adolescence will remain so into adulthood.[6] If this is correct in NZ, then up to 49,500 (of the 61,800) children will experience obesity throughout their life. This is in addition to expanding waistlines that develop in adulthood.[7,8]

The health consequences of overweight and obesity are better described than they were 3 years ago, particularly in relation to increased cancer risk.[9–11] Less well understood are the economic and social effects of this population weight increase. In addition, the current generation of children are not yet old enough for the full extent of their body mass index (BMI) related health issues and healthcare needs to be experienced.[12]

Marketing of unhealthy food to children is harmful

The evidence of harm from food marketing has become even more compelling in the last 3 years. There have now been four, independently-funded, systematic reviews of the evidence looking at the evidence of a relationship between marketing and children's dietary beliefs, dietary choices, and diet-related health.[13–16] All of these reviews have concluded that there is an adverse effect.

The 2003, United Kingdom (UK) Food Standards Agency-funded, Hastings' review concluded that food promotion is having an effect, particularly on children's preferences, purchase behaviour, and consumption—and that the effect is independent of other factors, and operates at both a brand and a category level.[14]

The Hastings' review was subjected to a rigorous peer-review process,[17–20] and was judged sound enough to inform policy change in the UK around television advertising to children. While the precise magnitude of the effect of marketing is unclear, the Institute of Medicine systematic review noted that even a small effect accrued over an entire population of children substantially increases in the number of obese children.[15] This population effect is supported by modelling work in Australia which showed that interventions with a small effect on population mean BMI translated into substantial population gains.[21]

Additionally many debates over the harmful effects of children's exposure to marketing fail to acknowledge a crucial difference between children and adults in this area. Young children do not understand the intent of marketing. Children are not cognitively developed enough, probably until early adolescence to understand the persuasive intent of marketing.[15,22] It is not ethical to expose children, especially very young children, to something that we know is harmful and that they are not capable of understanding. This was the rationale for the restrictions of advertising to children under the age of 12 in Sweden.[23]

Why do companies market to children? There are three main reasons:

- Firstly, the 1.1 million children and young people (aged 18 and under) in NZ have spending power of their own, through pocket money,[24] lunch money,[5] and/or (in older children) employment. Overseas research (and parental experience) shows that unhealthy food is a common item purchased with that money.[15]
- Secondly, children are an important 'influence market' (i.e. they influence household spending). In 1993 in the USA this 'influence' was estimated to be worth $295 billion.[15]
- Finally, today's children and young people are tomorrow's consumers; brand awareness and loyalty are a strategic investment by companies.

The power of marketing is impressive. For example in 2007 research was published showing that pre-school children have specific beliefs about brands by age 4, thinking that food wrapped in branded McDonalds' wrappers tastes better than identical plain wrapped food.[25] Despite extensive research on the effects of marketing activities, some members of the marketing and food industries continue to muddy the waters, for example by commissioning their own research[26] (with methodological issues which

limit the findings[27,28]) or by casting doubt on the issue in other forums.[29] The irony of this position must surely be self evident: $2.2 billion is spent on marketing annually in NZ,[30] so it seems untenable to be arguing that it does not affect consumer behaviour.

New Zealand children are exposed to marketing of unhealthy food

So what do we know about NZ children's exposure to marketing of food? Children's exposure to marketing occurs in numerous ways including television, Internet, within the school environment, product placement, sponsorship, and sales promotions.[31] Food marketing is ubiquitous; much of it may be reaching children while being largely unnoticed by caregivers. At this point there is no systematic monitoring, so our understanding is based on a number of ad-hoc research projects, mainly looking at television advertising during advertising industry designated 'children's viewing hours'. These projects have not used identical methodology so assessing trends over time is difficult.

Television marketing

- Television is probably the most common medium of exposure to marketing of unhealthy food, with two out of three children watching 2 or more hours of television per day.[4]
- Food advertising during after-school television is common, with 25–29% of products advertised being food.[32–34] Seventy percent of this food advertising is for foods that are counter to healthy nutrition messages.[35]
- Research in 2005 showed that children who watched 2 hours of the TV2 channel each weekday afternoon and 2 hours each morning in the weekend would see a total of 7134 food advertisements per year.[36]
- Similar to international patterns the 'Big Five' (soft drinks, pre-sugared breakfast cereals, confectionary, savoury snacks, and fast food outlets) dominate food products advertised to children on television in NZ.[32–36]
- While it is difficult to draw conclusions about trends in the amount of food marketing during children's viewing hours in the last decade it does not appear to have decreased.[34,36]

Other forms of marketing

- Whilst there has been no systematic assessment of the extent to which food companies market into schools in NZ, there is evidence that it does occur.[37,38] It has been noted overseas that companies with controversial practices or products tend to be the companies that place sponsored educational material in schools, which can allow them to influence discourses and discussions.[37]
- Access to and use of the Internet is common in NZ children, and this is a growth area in terms of marketing spend.[30] The extent of children's exposure to marketing of food through this medium is currently not quantified.
- Product placement, which is embedded marketing, occurs in television programmes, films, advergames, music videos.[15] (For example, in 2007, Pepsi-Cola had product placement in the following movies: *30 Days of Night*,

American Gangster, *Blades of Glory*, *I Now Pronounce You Chuck and Larry*, *Resident Evil: Extinction, Superbad*, *Transformers*[39]) Product placement is a growth area: it is often not apparent as advertising to viewers, changing technology means it is easy to insert and viewers are increasingly able to avoid advertising breaks.[40] Product placement occurs in NZ, including in programmes such as *Shortland St*, but how much of this is food related is not clear.[41]

- Sponsorship is also common, although not well quantified. One NZ study identified that junior sport was 14 times more likely to be sponsored by companies that produce unhealthy food compared to all other sponsorship.[42]

Arguments against marketing restrictions

Debate is vigorous around the issue of marketing restrictions aimed at reducing children's exposure to marketing of unhealthy food. Debate is largely grouped into two areas: firstly, the theoretical justification of any restrictions and, secondly, details of any restrictions (for example how to define 'unhealthy' or the type of regulatory framework).

Table 1. Objections and counter arguments to restrictions on marketing unhealthy food to children

Objection	Counter arguments
There is no causal proof between marketing and the obesity epidemic in children	Evidence from a health perspective is now sufficient for action to protect children.[13–16] At least $2.2 billion per year is spent on advertising in NZ.[30] It seems hard to believe that this is not effective at influencing children's diets. Marketing is one of a number of important factors that need to be addressed, part of larger picture of obesity prevention. A small effect of marketing on each individual translates to a large effect on the population (due to the large exposure).
Parents are responsible for their children and have control of the family budget	Children have access to pocket money and, sometimes, their own income and frequently purchase their own food. NZ parents report that advertising influences their children likes and requests for specific food and drink.[43] International and national surveys show that parents want restrictions on marketing of unhealthy food to children.[44,45]
Restrictions on marketing would violate the freedoms of speech set out in the Bill of Rights Act 1990 and the right to advertise legal products as set out in the Fair Trading Act 1986 and Commerce Act 1986	There are national precedents for regulation of marketing activities, for example Smokefree Environments Act and regulations (including international treaty requirements for various controls on tobacco marketing and for warnings[46]). There is at least one legal precedent in a sub-national jurisdiction that supports a state's right to protect children from marketing (Supreme Court of Canada judgement on Irwin Toy Ltd v Quebec Attorney General 1989)
An advertising ban would be ineffective/the 'forbidden fruit' argument.	Similar arguments were made for tobacco advertising, but as part of a comprehensive package marketing restrictions are probably contributing to declining smoking rates in New Zealand. Marketing restrictions alone are likely to be of limited effectiveness; they need to be one of a number of anti-obesity interventions.

Table 1 outlines some of the key objections and counter arguments to the theoretical justification of restrictions on marketing of unhealthy food to children. These objections are not necessarily specific to food marketing: similar themes are traversed in discussions about tobacco and alcohol marketing restrictions.

International developments around marketing restrictions

The increasing international concern around the role of marketing to children in the obesity epidemic has translated into some action. For instance, the World Health Organization's (WHO) *Global Strategy on Diet, Physical Activity and Health* calls for Governments to make changes in this area.[47]

WHO have also commissioned two reports on the global regulatory environment around marketing to children.[31,48] The first of these reports is a stocktake of the regulatory environment, and the second documents changes since the release of the Global Strategy. Broadly these reports note some increase in regulatory activity, although efforts are largely confined to developed countries, are not comprehensive, and mainly rely on self-regulation.

Attempts to regulate are being strongly opposed by some industry groups. Most activity is focused on television advertising but there is increasing interest in other media.[31,48] There is some suggestion that, at least in Europe, advertising spend is being diverted into other areas (such as in-school marketing) as television advertising becomes restricted.[49]

The UK has a system of co-regulation of television advertising of unhealthy food through their Office of Communications (Ofcom) and Communications Act 2003. While there are different permutations to co-regulatory systems, the basic premise of co-regulation is that it involves a mix of a statutory framework (giving Government some control) and self-regulation.

Under the UK co-regulatory model, food is defined as 'healthier' or 'less healthy' using a Nutrient Profiling Model developed by an independent body the Food Standards Agency.[50] Any 'less healthy' food is not permitted to be advertised on television when the proportion of children watching a programme is more than 20% higher than their proportion of the general population audience. Brand advertising is currently permitted but may be reviewed.[51,52]

The UK Advertising Standards Authority regulates all other advertising, including 'healthier food' under it's codes and complaints system. There are also content restrictions on food being advertised, including no celebrity endorsements, health claims, use of cartoons, or promotions (such as toys).[53]

While the Ofcom system has not yet been fully implemented or evaluated, it is already apparent there are serious limitations. Ofcom's preliminary review 6 months after the first phase of restrictions was implemented noted that while there was some decrease in exposure to advertising of food during 'children's airtime', there was an increase in exposure during adult airtimes.[53] Independent analysis by UK consumer advocacy group *Which?* shows that 8 out of 10 programmes most commonly watched by children are still permitted to advertise 'less healthy' food. Even once the system is fully implemented this will remain largely unchanged.[54] *Which?* calls for restrictions to cover all programmes up until 9pm at night instead of the use of the proportion-

based assessment of the audience.[54] The UK Government is reviewing the system due to concerns around the effectiveness of it.[55]

The Australian Media and Communications Authority is currently drafting new Children's Television Standards.[56] The draft standards did not recommend a change to the rules around food and beverage advertising, although it signalled a willingness to review this area in the future.[57] Submissions on the draft standards were divided on this issue. Interestingly modelling work done by the Victorian Government in Australia, shows that a modest but sustained decrease in mean BMI of 0.17 units per child, as a result of restricting television advertising of unhealthy food, was one of the most cost-effective interventions available to Government to prevent obesity.[21]

There are three jurisdictions that have banned all advertising directed at children: Sweden, Norway, and the Canadian province of Quebec have all had statutory bans in place for up to 20 years to protect children from commercial interests.[31] Opponents cite the level of obesity in these countries as evidence that regulation of advertising is ineffective. However regulation of advertising in these countries was not initiated to combat obesity and thus was not part of a comprehensive anti-obesity programme; and children in all these jurisdictions are subject to cross-border advertising that is unaffected by the regulations.

New Zealand: A laggard in the control of marketing?

NZ has an almost entirely self-regulatory system for marketing. Consumer protection legislation, such as the Commerce Act, applies to all advertising and the Broadcasting Act 1989 restricts advertising on television on certain days of the year and certain times. For all other matters relating to television advertising the Broadcasting Act devolves responsibility to the Advertising Standards Authority (ASA)—a voluntary industry body.

The ASA has codes of practice relating to advertising of food and to children, which members are required to follow, and a complaints process, in case of perceived breaches to those codes.[58] These codes apply to advertising in it's 'broadest sense', although some types of marketing, such as product placement, are not specifically mentioned in the codes or in the definition of marketing.[58]

In terms of a response to obesity, a codes and complaints based self-regulatory system is fundamentally flawed. A codes and complaints based self-regulatory system is not designed to be a public health policy tool; it is designed to identify advertising 'outliers' who breach acceptable standards, rather than reduce large volumes of effective advertising that inundate children everyday.[59] Under a codes and complaints system it is difficult to argue that a single advertisement is inconsistent with a healthy diet, but given that 70% of food advertising in 'children's viewing hours' is for food that is counter to healthy nutrition,[36] television advertising does not support and promote healthy diets.

One comparison of international regulatory systems of television advertising of food to children identified that NZ is one of the few developed countries in the world that is entirely self regulatory.[23] From the evidence available it appears that the current self-regulatory system has thus far failed to decrease children's exposure to advertising of unhealthy food.[34,36]

In the last three years the issue has been on the policy agenda in NZ to some extent including:

- In May 2007 a '5-point plan' by the Ministers of Health, Education and Broadcasting and New Zealand Television Broadcasters Council (which includes TVNZ, CanWest, and Māori Television). This plan has a children's food rating system and only food products that receive this rating will be able to advertise in programmes directed at children from October 2008.[60,61] While this is a start, it will be of limited effectiveness as children watch television more commonly outside the industry designated 'children's viewing hours' than they do within.[62]
- In August 2007, the report of the Health Select Committee Inquiry into obesity and Type 2 diabetes was released. This called for clear targets around advertising and marketing of food to children and a commitment to regulation depending on progress.[2] In their response to this inquiry, Government committed to reviewing the industry's progress towards reducing advertising unhealthy foods and beverages to children and developing targets in this area (in conjunction with the food industry) by June 2008.[3] At the time of publishing this article (January 2009) targets had yet to be announced.
- The draft Public Health Bill, which underwent consultation in 2008, attempted to establish a generic framework, and some tools, for dealing with non-communicable diseases and their causes. This section was opposed by media organisations and the food industry who perceived it as a Trojan horse to regulate food advertising.[63] The Public Health Bill was not passed before the election.

What the new Government intends to do in relation to these issues is not yet clear.

What does New Zealand need to do?

The rationale for why we need to address this issue has been outlined above. In order to advance the agenda in NZ a number of things need to occur. Ideally these would include the following:

- Government needs to articulate a vision about what it wants to achieve in this area. The outcome needs to be of clear, measurable, and time-specific. (For example, a 90% reduction in exposure of children aged under 16 to any marketing of unhealthy food by 2012, from 2007 baseline.) This process should be independent of the advertising and food industry to have public credibility. Additionally there needs to be a broad definition of marketing to encompass the range of current activities as well as to allow flexibility to deal with the dynamic changes in media technology and useage.[40]
- Once a vision is developed, methods to achieve it can be considered. Options for the legal framework include co-regulation or full Government regulatory systems. A self-regulatory system is simply not capable of addressing this issue, based on current evidence or practice. Any framework could be incorporated within current or planned legislation (e.g. the Broadcasting Act or the Public Health Bill) or could be a stand alone Act.

- While a specific NZ system needs to be developed, there are principles from the International Obesity Task Force and Consumers International that could be used to underpin a framework.[64] Additionally there are exemplars from other jurisdictions that have been successful (e.g. the FSA nutrient profiling model) or less successful (e.g. the use of a proportion based measure of audience) that NZ can learn from.
- There needs to be independent systematic monitoring of all forms of food marketing activities, so that progress (or lack of it) can be measured and policy interventions can be evaluated for effectiveness.
- NZ should start taking leadership on the possibility of using an international treaty approach (similar to the Framework Convention for Tobacco Control) to dealing with cross border marketing.

Conclusions

The obesity epidemic is a serious threat to public health in NZ. NZ children deserve appropriate protection from marketing of unhealthy food as part of a response to this issue. Despite the need for action limited progress in policy in the control of food marketing has occurred in the last 3 years in this country.

Fortunately NZ is in a position to learn from other countries as they implement regulatory systems. This agenda needs to be commenced now; this is a complex and time-consuming process. NZ is already lagging behind other jurisdictions, we need to advance this work quickly and decisively.

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Heart health in New Zealand

Robert Beaglehole, Ruth Bonita

Abstract

Global health is increasingly dominated by chronic diseases—cardiovascular diseases, cancer, chronic respiratory diseases and diabetes. New Zealand cardiovascular disease death rates have fallen by over 60% since their peak in the late 1960s. Most of the decline can be explained by favourable trends in the main risk factors. Despite these gains, there will be an increasing burden in elderly, Māori, and economically disadvantaged people. There are two major challenges. Firstly, to implement a balanced public health approach to the prevention and control of vascular disease and to ensure a reduction in overall population levels of risk and a reduction in risk levels of high-risk people. Secondly, to promote health equity in New Zealand, initially by encouraging a rapid reduction in tobacco use in Māori and Pacific Island people

The 40th anniversary of the Heart Foundation of New Zealand is an opportunity to place New Zealand's experience in the global context, review progress in the prevention and control of heart disease and stroke in New Zealand, and consider prospects for further health gains. There are lessons for New Zealand in the international experiences with disease control, and lessons from the New Zealand experience of relevance to many other countries.

Global context

Current status—Global health data are increasingly dominated by chronic (noncommunicable) diseases such as heart disease, stroke, cancer, diabetes, and chronic respiratory diseases. These conditions have common causes and there is a move, especially in low-and middle-income countries, to focus attention on the integrated prevention of chronic diseases rather than the specific diseases.

Ageing of populations will increase total chronic disease deaths substantially in all regions, even though age-sex-specific death rates are projected to decline for most conditions other than lung cancer.[1]

Chronic diseases cause approximately 60% of the 59 million deaths that occur worldwide each year. 80% of these deaths occur in low-and middle-income countries where the death rates are now greater than in high-income countries, with great economic consequences for countries. There is considerable potential for the prevention of chronic diseases since they share common risk factors: unhealthy diet, tobacco consumption, and physical inactivity. Furthermore, the underlying determinants such as poverty and limited educational opportunities are also common.

The World Health Organization (WHO), despite its focus on infectious diseases and child and maternal health, is giving increasing attention to chronic diseases although there is still a major misalignment between its budget and the global disease burden with only about 12% of its budget devoted to chronic diseases.[2]

Recent WHO initiatives in the prevention of chronic diseases include: the Framework Convention on Tobacco Control;[3] the Global Strategy on Diet, Physical Activity and Health;[4] and the Bangkok Charter for Health Promotion in a Globalized World.[5] There have been many global and regional resolutions on chronic disease prevention and control although in most countries, action lags behind the stated intentions.

It is noteworthy that New Zealand has consistently played an important role at the World Health Assembly and WHO Western Pacific Regional Committee meetings in promoting the chronic disease agenda.

A global goal for chronic diseases—In recognition of their importance to Member States, a global goal for the prevention and control of chronic diseases was proposed to complement the Millennium Development Goals.[6] This goal comes with a target: an additional 2% per year reduction in death rates attributable to the major chronic diseases. Achievement of this global goal would delay 36 million deaths by 2015 and, because most of these deaths would be in low-income and middle-income countries and about half would be in people under the age of 70, it would have major economic benefits in low- and middle- income countries, extending productive life by approximately 15 years and reducing the need for expensive health care.[7] Progress in all countries is threatened by the obesity and diabetes pandemics[8] and the efforts of multinational tobacco companies to increase tobacco consumption in low-and middle-income countries.[9]

A package of cost-effective and feasible interventions for many chronic diseases is available to achieve the goal.[10] For example, implementation of four measures of the WHO Framework Convention on Tobacco Control and reducing population levels of salt consumption by a modest 15% (to reduce blood pressure levels) could delay, over 10 years (2006–2015), 13.8 million deaths in just 23 low- and middle-income countries. The cost of these interventions would be less than $0.40 per person per year in low-income and lower-middle income countries, and $0.50–$1.00 per person per year in upper middle-income countries (2005 US dollars).[11]

In these same 23 high burden countries, opportunistic screening and treatment with an appropriate set of medicines in people with cardiovascular disease or a 15% or greater probability of dying from heart disease or stroke over 10 years would delay 18 million deaths between 2006 and 2015.[12] This would require an average annual investment of $1.10 per capita (ranging from $0.43 to $0.90 across low-income countries and from $0.54 to $2.93 across middle-income countries), with medicines accounting for around two-thirds of this cost.

Based on the unequivocal evidence that major and rapid health and economic gains are possible with only modest investments, intensified action is required from all stakeholders to achieve the chronic disease goal.[13] Tobacco control remains a top global priority—only 5% of the world's population is covered by effective tobacco control policies. Without urgent and sustained action, tobacco products will kill approximately 1 billion people this century, 80% of whom will be in low- and middle-income countries.[14]

Recent developments in New Zealand

Although contributing only a tiny amount to the global burden of disease, there are important lessons to be learned form the New Zealand vascular disease experience. Chronic diseases cause over 75% of all deaths in New Zealand with coronary heart disease and stroke responsible for 22% and 10% of deaths respectively.[15] Coronary death rates reached their peak in the late 1960s and since then death rates have fallen by over 60% in all age and sex groups although the decline has been slower in Māori.[16]

Most of the decline can be explained by favourable trends in the major risk factors,[17] especially blood pressure and cholesterol levels, and in tobacco use.[18] Adverse trends have occurred in the prevalence of obesity and Type 2 diabetes and these may now be increasing the rates of coronary heart disease in young men as is happening in the USA, Australia, and Scotland.[16]

Stroke mortality trends have followed a similar trajectory with the decline accelerating in the mid 1970s. Explanations for the decline include improvements in incidence and case fatality. A long-term population based study of stroke occurrence in the Auckland region[19] suggests that stroke incidence has declined by approximately one-fifth in New Zealand Europeans during the period 1981 and 2003, but other ethnic groups, particularly Pacific Island people, have not fared so well.[20] Divergent trends in major risk factors were associated with these long-term patterns. Overall, case fatality has also declined, but to a lesser extent.

Chronic diseases are a priority for the Government and health targets have been specified for nutrition, physical activity, obesity, and tobacco control although the indicators are limited and do not include intermediate or definitive outcome measures.[21]

It is expected that the new government will continue with chronic diseases as a general priority area. In order to make substantive improvements in the health of all New Zealanders and to promote health equity, the Government will need to emphasise a comprehensive approach to prevention with a focus on the environmental determinants of population risk. Avoiding a primary focus on individual responsibility for risk status and entrenching a regulatory option for reducing risks for chronic diseases through the Public Health Bill will give the Government the tools to strengthen—if needed—voluntary actions by the food, advertising, and marketing industries.[22]

Priorities for action

Despite the recent progress (as measured by falling death rates and increasing life expectancy) there is still a considerable potential for effective prevention actions especially those which focus on health equity. Advances have been made in reducing levels of tobacco use, as well as blood pressure and cholesterol levels in New Zealand over the last 2 decades. However, progress has been slow and striking ethnic inequalities remain, especially with tobacco use and obesity levels; tobacco use is an important preventable factor involved in the higher death rates in adult Māori.[23]

Salt consumption is much higher than recommended and animal fat consumption is excessive. New national goals and targets for reducing population levels of risk and death rates will encourage effective action.

Population-wide interventions—The immediate priorities for tobacco control include a significant increase in taxation of all tobacco products and harmonisation of the tax rates between manufactured and "roll your own" products which make up about half the tobacco consumed in New Zealand. There is a strong case for banning point of display advertising of tobacco products as recommended by the Health Committee.[24]

Plain packaging of tobacco products will go a long way towards reducing the appeal of tobacco products, and there is a need to for increased support for quitting since most smokers still want to stop. The longer-term goal, now that New Zealand is essentially smokefree, is for a tobacco free nation; Te Reo Mārama—Māori Smokefree Coalition—is leading the way.

Salt consumption remains excessively high in New Zealand at approximately 9 grams per person per day; although there are no data on recent levels. The Heart Foundation's salt reduction project with the major manufacturers of bread has removed approximately 150 tonnes of salt per year from the food supply but this is only a small start towards achieving the desired level of intake of less than 4 grams per person per day.[25]

Continuing work is required with industry to reduce the amount of salt added in the manufacturing process, especially in bread, processed meat products, sauces, fast foods, and snack foods.

The most important underlying cause of vascular disease in New Zealand is the excessive intake of saturated fat, especially butter. Reducing this intake will require continuing work with industry and small businesses to reduce the saturated and trans fat content of dairy products, meat, processed foods, and fast and snack foods.

As recommended by the Health Committee, the efforts of industry to reduce population levels of risk should be guided by a set of timed and quantitative targets for product reformulation and to reduce the exposure of children to the advertising and promotion of products likely to be harmful to health.[26]

These targets should be mutually agreed by government and industry and be independently monitored. If voluntary actions are sufficient to achieve the targets they should periodically be reassessed and strengthened. If progress is slow, the Government should introduce regulations to reduce risks, as specified in the Public Health Bill. A user friendly and uniform food labelling system would encourage healthy consumer choices.

According to recent surveys, almost 50% of the population report activity levels consistent with health promoting guidelines.[27] The goal for increasing the physical activity levels of all New Zealanders should be to increase life long activity as part of everyday life and to reduce the emphasis on competitive sports. This will require greater attention to public transport and opportunities for walking and cycling as well as increased attention to school and community programmes. The looming

environmental and financial crises will support these trends indicating, once again, the importance of intersectoral actions for health.

Individual-based interventions—Although the prevention of the chronic disease epidemics is the only effective long-term method of reducing the disease burden in New Zealand, there is much to be gained by the early identification of people with disease and people at high risk of developing disease because of their overall risk factor status.

Priorities for early detection of disease and management of high risk include: extension of the *Get Checked* and *One Heart Many Lives* programmes for identifying people with or at risk of developing diabetes; encouraging health workers to ask all patients if they are smokers, and offering advice and support to all smokers for quitting;[28] and the training of practice nurses in the assessment and management of overall risk of chronic disease.

Prospects and challenges

It is likely that there will be a continuing slow downward trend in coronary heart disease and stroke mortality accompanied by an increasing life expectancy, especially for middle aged and older people. It is possible that the adverse effects of obesity and diabetes will lead to increasing rates of heart disease death rates in younger people. An increasing burden in elderly, Māori, and economically disadvantaged people is also expected. Vascular disease will continue to place an increasing strain on all aspects of the health system.

There are two major challenges ahead.

Firstly, to implement a balanced public health approach to the prevention and control of vascular disease and to ensure a reduction in overall population levels of risk and a reduction in risk levels of high-risk people. Progress on this challenge will be facilitated by a comprehensive set of goals and targets for chronic disease, including both indicators of risk and outcome measures, together with an appropriate evaluation and monitoring system. In addition, it will be necessary to confront allegations of unnecessary government intervention since this will be required to protect vulnerable population groups, especially children.

The second major challenge is to promote health equity in New Zealand initially by encouraging a rapid and major and rapid reduction in tobacco use in Māori and Pacific Island people.

Nongovernmental organisations (NGOs) such as the Heart Foundation, Cancer Society, Stroke Foundation, and Diabetes New Zealand have key roles in promoting health; the Chronic Disease Peak Group for the Prevention of Chronic Diseases is an important agency for coordinating and strengthening action on the common risk factors for the leading causes of death and disease in New Zealand.

The Heart Foundation, initially under the leadership of Sir David Hay, Medical Director for 15 years,[29] has contributed greatly to heart health and health promotion generally. It now has the opportunity to build on its achievements and increasingly focus on public health approaches to health improvement and health equity.

The Heart Foundation, working with government agencies (led by the Ministry of Health), other NGOs, and community groups can significantly reduce the burden of disease in New Zealand at the same time as promoting health equity.

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Ruptured tubal ectopic pregnancy with negative serum beta hCG—a case for ongoing vigilance?

Joseph K-S Lee, Vincent P Lamaro

Abstract

A 25-year-old female with a history of recent miscarriage presents with haemodynamic shock and a negative serum beta hCG. She presents to six different healthcare facilities within a single metropolitan area, during which a pelvic ultrasound scan showed an empty uterus with a subnormal rise in serum beta hCG. Suspected ruptured tubal ectopic pregnancy was confirmed following laparoscopy and salpingectomy, with histopathological confirmation of chorionic villi in the extirpated fallopian tube. This case report highlights the ongoing clinical diagnostic challenges that are associated with ectopic pregnancy; illustrates the importance of teamwork; and perhaps also draws attention to the need for a robust protocol to facilitate consistent, good-quality early pregnancy care for all women.

The sudden rupture of a tubal ectopic pregnancy can lead to hemorrhagic shock and death if not diagnosed and treated in a timely fashion. Ectopic pregnancy was the eight most common cause of maternal death in the most recent Confidential Enquiry into Maternal Deaths (CEMD) in the United Kingdom 2003–2005; accounting for 10 out of 14 (71%) early pregnancy deaths.[1]

In the most recent Australian CEMD 1997–1999,[2] there was only one direct maternal death attributable to ectopic pregnancy, although more recent figures are not yet available.

For women presenting with early pregnancy problems, the general practitioner or emergency physician are usually the first to be called upon for diagnosis and coordinate timely, effective intervention. The first step in the diagnosis of ectopic pregnancy is the demonstration of pregnancy by means of a rapidly performed and sensitive qualitative urine test for the beta-subunit of human chorionic gonadotropin (beta hCG).

A negative urine pregnancy test result will generally be used to exclude ectopic pregnancy from further consideration. The following is a report of a patient presenting to an emergency department with hypovolaemic shock in conjunction with two negative urine beta hCG analysis results and a quantitative serum beta hCG level of 4 IU/L, a value less than the lower limit of detection for the highly sensitive qualitative urine and serum tests.

This case report demonstrates the importance of further consideration of the diagnosis of ectopic pregnancy in the setting of a negative urine pregnancy test result.

Case report

A 25-year-old gravida 1 para 0 presented to her local medical centre having a regular cycle and missed her last period, with serum beta hCG of 1120 IU/L. She was uncertain of her exact menstrual dates. She represented 2 weeks later with vaginal bleeding and passage of "tissue". Unfortunately, this was discarded without histopathological examination. Her serum beta hCG was then 1400 IU/L with a pelvic ultrasound (USS) demonstrating a bulky endometrial stripe, no evidence of intrauterine pregnancy, and normal ovaries. Her serum beta hCG was quantified 2 days later at 1350 IU/L.

A week later she was seen at a pregnancy clinic at her local hospital with serum beta hCG of 700 IU/L. A diagnosis of intrauterine miscarriage was suggested. She was subsequently referred by her GP to a local gynaecologist some 8 weeks after her last menstrual period. Her serum beta hCG was then 149 IU/L; she remained clinically well and was told that the likely diagnosis was intrauterine miscarriage.

Around 12 weeks after the original menstrual period, she presented to a nearby hospital with lower abdominal pain and fainting episodes, having had a "period" 3 days prior. She required fluid resuscitation, had abdominal and cervical tenderness with a serum beta hCG of 4 IU/L (<10 considered negative), and haemoglobin (Hb) level of 120 g/dL.

She was transferred to our hospital, as it offers tertiary gynaecological and surgical care. She remained hypotensive despite intravenous fluid resuscitation. Her Hb decreased to 80 g/dL, and she experienced a postural blood pressure drop of 30 mmHg systolic. Abdominal examination revealed guarding in her lower abdomen. A bedside ultrasound in the ED demonstrated fluid in the abdomen. Two large bore canullas were sited together with an indwelling urethral catheter. She was commenced on 4 units of packed red blood cells transfusion. A working differential diagnosis was ruptured haemorrhaging ovarian cyst or a missed ectopic pregnancy.

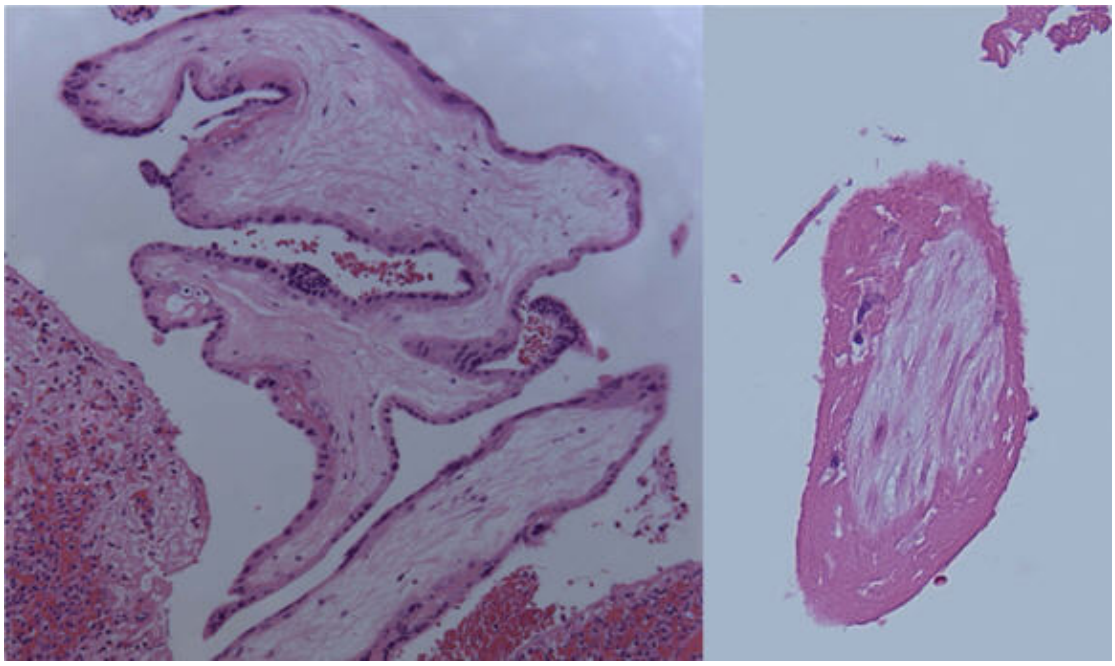
She was seen by a surgical registrar, who following discussion with his consultant arranged a chest X-ray and CT abdo/pelvis scan. No free air was seen, however it confirmed the presence of free fluid. A pelvic USS showed free fluid, empty uterus with an adnexal mass of 6 cm.

She was transferred to theatre with involvement of specialist gynaecologist and anaesthetist. Optimal multidisciplinary specialist involvement, familiar and prompt laparoscopy set-up, a dedicated theatre team, together with a stabilised patient contributed to a decision for laparoscopy with early recourse to laparotomy.

Laparoscopy showed approximately 2 litres of haemoperitoneum, with a ruptured left tubal ectopic, over the ampulla region. Prompt surgical control was executed with monopolar diathermy, and endoloop leading to salpingectomy. Suction irrigation was performed for peritoneal toilet and a blake drain was left *in situ*. She had a total of 6 units of red blood cell blood transfusion.

Her postoperative recovery was unremarkable, with stable Hb and negative serum beta hCG. She remained well at her 6 weeks postoperative follow up and was scheduled for a tubal patency test at 3 months postoperation.

Figure showed sections from histopathological examination demonstrating fragmented and disrupted fallopian tube with blood clot, containing chorionic villi. Some of the chorionic villi appear viable (left), while others are seen as 'ghost-outlines' (right)



Discussion

Despite reported incidence of ectopic pregnancy having stabilised or declined over the past 30 years in some countries, current research suggests that the incidence of ectopic pregnancy is once again on the increase.[3]

Correct diagnosis of ectopic pregnancy can often be made on history, examination, serum beta hCG estimation, and pelvic USS. Typically, there is a period of amenorrhoea associated with lower abdominal pain, adnexal tenderness, a small amount of vaginal bleeding, positive serum beta hCG and pelvic USS revealing no intrauterine gestational sac.

A possible cause for delay in diagnosis could be uncertain menstrual dates and declining beta hCG with a history consistent with a complete miscarriage (passage of products). On the basis of subnormal progression of beta hCG and empty uterus on USS, it would appear our reported patient had a chronic ectopic pregnancy.

Chronic ectopic pregnancy classically has a different presentation with mild pelvic pain and irregular vaginal bleeding over several weeks. It is postulated that this symptomatology may reflect repeated episodes of small bleeding from the tubal gestation, in contrast with a single major bleed from an acute ectopic pregnancy.[4] Ectopic pregnancy with negative beta—hCG would be a particularly rare form of a chronic ectopic pregnancy.

Although Lonky[5] may have reported the first case of ruptured ectopic pregnancy with an undetectable beta hCG level, it was Taylor[6] who described ruptures of chronic ectopic pregnancies with active ectopic trophoblastic tissue using immunocytochemical localisation of placental proteins. Trophoblast degeneration with cessation of hormone production, an extremely small mass of chorionic villi (producing little if any hormone) or defective beta hCG biosynthesis have been postulated as theoretical mechanism to explain unexpectedly low or absent beta hCG in chronic ectopic pregnancies. Since then, medical literature have been littered sporadically with reports of ruptured ectopic pregnancies presenting with negative beta hCG.[7–14]

Our patient initially presented with what was thought to be intrauterine miscarriage. It would be prudent to have further supportive evidence to reduce the risks of missed ectopic pregnancy by ongoing beta hCG surveillance—with the expected halving of beta hCG in 48 hours and to follow it to zero.

Ideally the patient should be counselled regarding the potential of an ectopic gestation with the view to early presentation to ED in the event of abdominal pain or vaginal bleeding. As shown in our case, a recent history of presumed miscarriage should not deter a clinician from considering the possibility of an ectopic pregnancy should patients present with consistent symptoms and signs, even if the serum beta hCG is zero. This also serves to remind us not to rely solely on laboratory criteria for diagnosis, and further underscores the need of ongoing vigilance for women presenting with early pregnancy problems.

Modern management of ectopic pregnancies has evolved, most evidently with the advent of medical management with the use of systemic methotrexate,[15] or in selected cases expectant management.[16] Several studies have shown that initial serum hCG level is the best predictor of successful expectant management of ectopic pregnancy, with the probability of spontaneous resolution decreasing with increasing hCG levels.

One report[17] found that 88% of ectopics with an initial hCG level of less than 200 IU/L resolved spontaneously compared with only 25%, when the hCG was >2000 IU/L. A similar report[18] demonstrated a spontaneous resolution rate of 96% in women with low initial hCG levels of <175 IU/L. A recent prospective cohort study has also reported the use of hCG ratios (hCG 48h / hCG 0h) in the prediction of spontaneous resolution of pregnancies of unknown location.[19]

An hCG ratio of <0.87 was found to outperform absolute serum hCG levels with a sensitivity of 92.7% and a specificity of 96.7%.Nevertheless, despite advances in modern management of ectopic pregnancies, it should not detract us from providing timely diagnosis and optimal management for women who suffer from early pregnancy problems, including ectopic pregnancies. “Traditionally” a typical patient journey for women with early pregnancy problems can include initial attendance at the GP/ED, referral to an acute gynaecology team, canvassing senior opinion prior to investigations, and pelvic USS most likely the following day. Further, potential delays in accessing emergency operating slot can culminate in excessive burden of suffering physically, mentally and financially on the part of patients, healthcare workers, and healthcare providers.

Professional colleges have introduced a guideline recommending setting up dedicated early pregnancy units in all hospitals accessible by GPs, women, and other hospital departments. Potential benefits include rapid diagnosis, a sympathetic environment, prompt gynaecological input, dedicated scanning facilities, reduced unnecessary admissions, reduced costs, and preservation of women's dignity.[20–22]

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Carbamazepine and falsely positive screening tests for Cushing's syndrome

Keith Tiong, Henrik Falhammar

Abstract

A case of markedly elevated 24-hour urine-free cortisol levels, mainly due to interference with carbamazepine when measuring with high performance liquid chromatography but also due to increased fluid intake, is presented. Moreover, the 1 mg overnight dexamethasone suppression test was positive and the midnight serum cortisol level was slightly increased, probably as a result of carbamazepine-induced activity of CYP3A4 and increased levels of cortisol binding globulin.

Endogenous Cushing's syndrome is a rare disorder with high morbidity and mortality if undiagnosed. It is therefore important to have a high index of suspicion and investigate with appropriate screening tests. However, this can sometimes be difficult as the following case report demonstrates.

Case report

A 26-year-old female was referred to the Department of Endocrinology and Diabetes for investigation of possible Cushing's syndrome with markedly elevated urine-free cortisol levels (UFC). Her presenting symptoms were hirsutism, polyuria, and polydipsia. She revealed a history of complex partial seizures since the age of 6 years and was on carbamazepine 400 mg twice daily. She denied smoking, excessive alcohol intake, and other medications including oral contraceptive pills. There seemed to be an excessive intake of water and she was always carrying a water bottle. There was no family history of endocrine disorders.

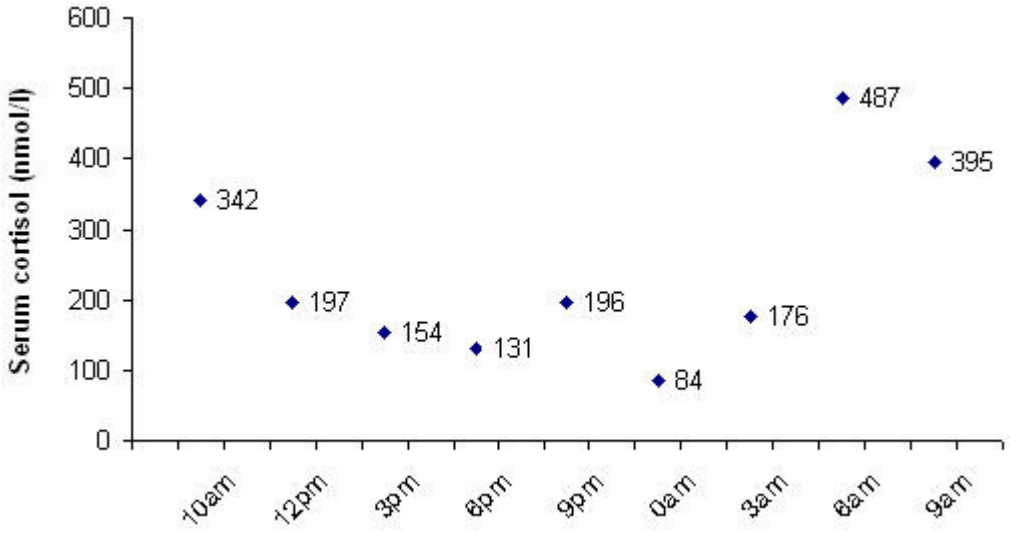
Clinical examination did not reveal any signs of central obesity, facial plethora, buffalo hump, fat pads in the supraclavicular fossae, proximal myopathy, acne, striae, or bruises. Facial hirsutism was present and it was considered secondary to her carbamazepine medication. Her weight had been stable at 53 kg for many years and supine blood pressure was 110/70 mmHg.

Two 24-h UFC collections were persistently elevated at 8426 and 1680 nmol/L (reference range 25–180) and the urine volumes were 2.9 and 2.1 litres respectively (reference range 0.5–2.0). Biochemical analysis of serum/plasma full blood count, urea, electrolytes, liver function tests, fasting glucose, TSH, DHEAS (dehydroepiandrosterone sulphate), testosterone, androstenedione, and 17-hydroxyprogesterone were all within normal range.

Serum osmolality was 287 mmol/L (reference range 280–297) with a urine osmolality of 189 mmol/L (reference range 50–1450) further strengthening the suspicion of mild psychogenic polydipsia. A 1-mg overnight dexamethasone suppression test (DST) was performed demonstrating inadequate suppression (first morning s-cortisol: 391 nmol/L; second morning: 286; normal <50).

A 24-h s-cortisol rhythm was performed in hospital demonstrating an almost normal diurnal rhythm (Figure 1). However, the midnight sleeping s-cortisol was slightly elevated (84 nmol/L; normal <50). It was concluded that the failed biochemical tests were mainly due to the carbamazepine medication and subsequent reviews later have not reveal any further signs of Cushing's syndrome.

Figure 1. Diurnal cortisol rhythm



Discussion

Cushing's syndrome can be very difficult to diagnose and there are many pitfalls.

Initial test is commonly UFC or DST and sometimes in a later stage midnight s-cortisol. Our patient failed to demonstrate normal results on all three different tests although she did not reveal any symptoms of Cushing's except mild hirsutism.

Interference with carbamazepine has been reported when measuring UFC by high performance liquid chromatography,[1] the method used in this case, and thereby causing falsely elevated levels. Moreover, high fluid intake can increase UFC,[2] which was also present in the case. Interestingly the UFC decreased on the second measurement, but was still markedly elevated, when her urine output decreased.

Dexamethasone is primarily metabolised by the hepatic cytochrome P450 CYP3A4, an enzyme complex responsible for the metabolism of many drugs.

Carbamazepine and other antiepileptics,[3–5] barbiturates, rifampicin, troglitazone,[6] aminoglutetimide, anti-psychotics,[7] and benzodiazepines[8] induce the activity of CYP3A4 which may lead to false positive DST.

Serum cortisol has a diurnal rhythm in healthy people with stable conventional sleep-wake cycles. The levels begin to rise at 3–4 am, and then fall to a nadir when the individual is unstressed and asleep at midnight.[9] However, this usually requires at least 48-h admission, as the first midnight cortisol may be elevated due to the stress of

being in a hospital. This could be the reason why our patient did not decrease below 50 nmol/L at midnight, although the diurnal rhythm otherwise seemed normal.

She complained of sleep disturbances which also may have affected the midnight cortisol. Moreover, the majority of s-cortisol is bound to cortisol binding globulin (CBG) and albumin. Chronic use of antiepileptic (but also oestrogen-containing medications) increases the levels of CBG and thereby the s-cortisol level[4] which also may have influenced the slightly elevated midnight value.

We believe our patient does not have Cushing's syndrome as no classical symptoms were present, we have good explanations for the elevated cortisol values, and subsequent yearly follow-ups have failed to demonstrate any further symptoms or signs. However, there is still a remote possibility that Cushing's syndrome is actually present.

In conclusion, this case highlights the difficulties encountered in exclusion of Cushing's syndrome during carbamazepine medication.

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Cephalgia in a young woman

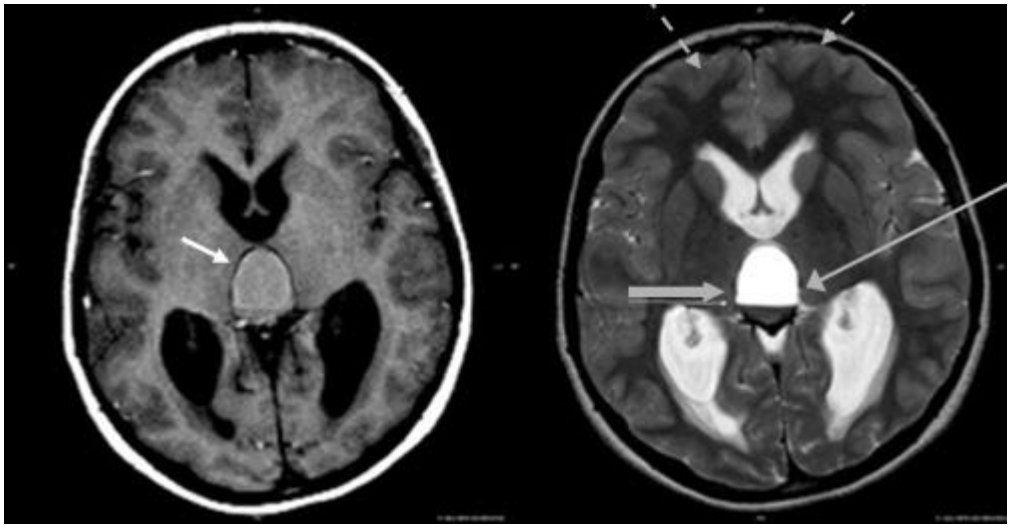
Rahmi Cubuk, Nuri Tasali

Clinical

A 19-year-old female patient with headache and progressive balance disorder attended our neurology clinic. Neurological exam showed ataxia. A cranial magnetic resonance imaging (MRI) examination was performed. There is a cystic lesion in the third ventricle at axial T1-weighted (Figure 1A, short arrow). The lesion caused expansion in the third ventricle.

The T2-weighted sequence showed a fluid/fluid level (Figure 1B, long arrow) in the posterior part of the lesion, whereas the other parts were hyperintense at all sequences because of protein content (bold arrow). The lateral ventricles were dilated and transependymal cerebrospinal fluid passage was detected (dashed arrows).

Figure 1A **Figure 1B**



What is the diagnosis?

Answer

A colloid cyst in the third ventricle. Third ventricular colloid cysts are rare benign lesions accounting for 0.5–1% of primary brain tumours. The developmental origin is unclear, though they may be of endodermal origin, which would explain the mucin-producing, ciliated cell type. As the patient ages, these cells secrete a gelatinous material, resulting in cyst formation and expansion. They are typically located in the anterior third ventricle, attached to its roof between the columns of the fornix.

Presentation may include headache, vertigo, memory deficit, seizures, and behavioural disturbance. Sudden neurological decline and sudden death from acute obstruction of the foramen of Monroe have been documented, especially with cysts larger than 1 cm.

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Cupolic asymmetry

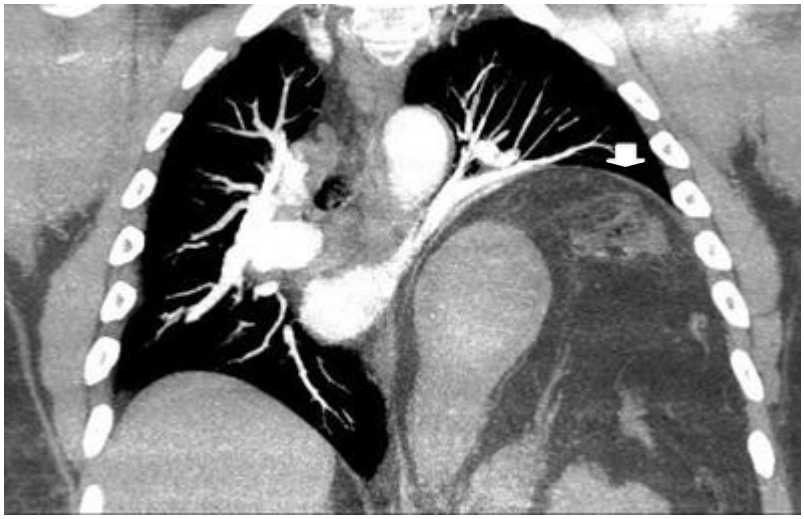
Gerson D Valdez, Roger D Smalligan

Clinical

A 60-year-old paraplegic man came to the emergency department with a 1-day history of chest pain and shortness of breath. Physical exam revealed tachycardia, heart sounds (heard best on the right), and the presence of bowel sounds in his left hemithorax.

The patient's intermediate probability for pulmonary embolism prompted a computed tomography of the chest (Figure 1). He was subsequently diagnosed with a non-ST-segment elevation myocardial infarction.

Figure 1. Computed tomography of the chest, coronal view



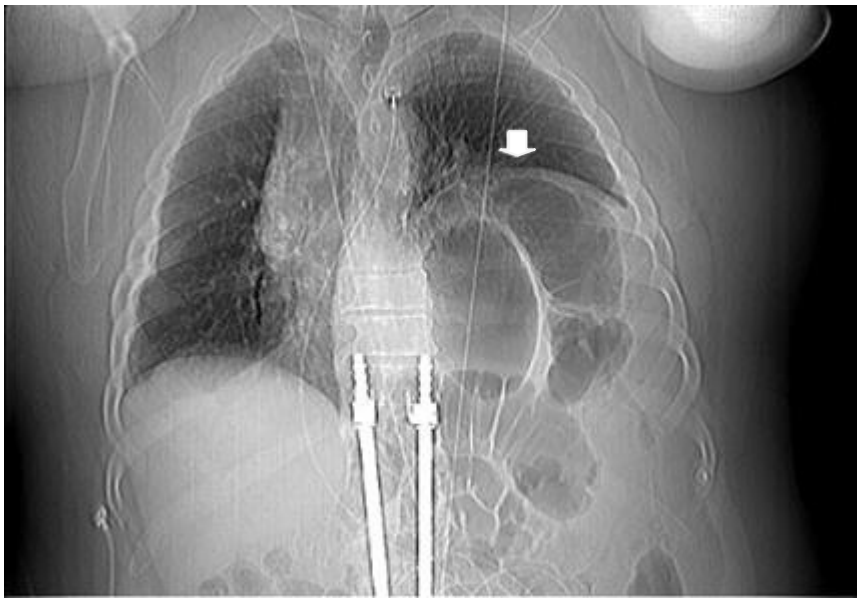
What is the non-cardiac diagnosis seen on the CT scan?

1. Morgagni's hernia.
2. Diaphragmatic paralysis.
3. Diaphragmatic rupture.
4. Bochdalek's hernia.

Answer

2. Unilateral diaphragmatic paralysis.

Figure 2. Chest radiograph



Discussion

Unilateral diaphragmatic paralysis is usually asymptomatic in sedentary individuals and is frequently discovered incidentally. The most common causes are trauma, surgery, tumours, aortic aneurysm, herpes zoster, pleurisy, and diabetic neuropathy though occasionally it is idiopathic. More active patients may present with orthopnoea or exertional dyspnoea once they develop the unilateral paralysis.

Since our patient was wheelchair-bound due to his paraplegia, he was asymptomatic until he developed another condition which caused dyspnoea. The diagnosis can be made with the conventional chest radiographs (Figure 2) and confirmed with a fluoroscopic or ultrasound sniff test with a sensitivity of 90%.

Unilateral diaphragmatic paralysis usually does not require treatment unless it causes significant symptoms and its prognosis depends on the underlying cause. The only treatment available is surgical or thoracoscopic plication of the paralyzed diaphragm.

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British Medical Association: Thirteenth Annual Meeting of the New Zealand Branch

Excerpt taken from the President's Address at the Meeting of the New Zealand Branch of the B.M.A. held in Napier February 22 to 27, 1909 (Dr. T. C. Moore, President, in the chair) and published in NZMJ 1909;7(29):1–3.

Gentlemen,—I have to thank you for the honour you have conferred upon me by selecting me for the office of President for the ensuing year. The British Medical Association, of which we are one of the youngest branches, consists of over 22,000 members and is still growing. Its income is £51,000 per annum. It is an Imperial institution co-extensive with the Empire, a bond of union between medical men in all parts of the King's dominions.

This is the first annual meeting to be held in Napier. We had, it is true, in March, 1894, a meeting of the now defunct New Zealand Medical Association here, a small but very pleasant and successful gathering, on which occasion I had the honour of acting as secretary. The late Dr. Spencer made a dignified and impartial chairman.

The New Zealand Medical Association, as most of you remember, was the nucleus of our present branch, which was formed in 1898. The first annual meeting was held in Nelson in March, 1897, the late Dr. Cleghorn being in the chair—a man whose cornparatively early death was a loss not only to the profession but to all New Zealand.

In the last eight years the members of the Branch have doubled, and it is estimated that two-thirds of the profession in the Dominion now belong to it. But although our membership is fairly satisfactory, I am afraid our meetings are not attended as well as they might be, and it is left to a very small minority to take an active part in promoting the objects of the Association. I trust that this apathy and want of appreciation of the benefits of an Association like ours will soon cease, and that in future we will all endeavour to make our Branch even more useful to the profession and to the public that we have done in the past.

On behalf of the Hawke's Bay Division, I extend a hearty welcome to all the visitors who honour us by their presence, and I trust they will carry away pleasant memories with them. They will be afforded opportunities of seeing as much as possible of our town and its surroundings, and so will be able to form a good idea of the advantages of Napier and the district as a health resort.

We have to thank our Mayor and member who, with his usual liberality and hospitality, proposes to entertain us and our visitors at his summer residence; also Mr. J. N. Williams and Mr. E. H. Williams, who have invited us to visit them at Hastings.

Frimley is a household word all over New Zealand, and visitors from other parts of the Dominion will be able to see, owing to their kindness, what our soil can produce in the way of fruit. During a residence here of nearly 24 years I have naturally seen many changes and great improvements.

Most of the country between here and Meanee on my arrival, and for many years afterwards, was a swamp, which in summer at times gave off a very evil smelling odour. It is now reclaimed, many acres are under the plough and in the immediate neighbourhood of the town there is ample room for extension, and residences are being rapidly constructed.

Our Parade now brings us every season hundreds of visitors who bask in the sunshine and enjoy their dip in the breakers. Next year we will have baths for those who prefer to bathe in safer and quieter waters. I can remember when there was no parade, when the sea periodically washed away the apology for a road along the beach, sometimes even levelling the fences, bursting in the front doors and flowing through the houses.

Typhoid fever is now almost non-existent, but formerly, every autumn, it claimed many victims. Summer diarrhoea was a scourge to which in the hot months numbers of infants succumbed. It is now much less frequent and less fatal. In the days I speak of Napier was a favourite place to send consumptives to, and these unfortunate cases, in all stages, formed a large proportion of these who sought our help. It is now a comparatively rare disease here. Thanks to better methods we are able to take a more hopeful view of it, and restore any of the sufferers to health.

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Should general practice be nurse led?

A hot debate in the UK. A nursing academic offers the view that in the UK "general practitioners have already yielded considerable ground to nurses in the interests of improving the quality and efficiency of primary care. It is time this trend moved to its logical conclusion, acknowledging nurses to be the true frontline providers of primary care."

A general practitioner's riposte is as follows—"patients prefer to consult with a GP if they think their symptoms are serious. Might this be due to their understanding of GPs' training, and uncertainty concerning the ability of nurses to diagnose "rare but important health problems"?

And she goes on to point out that a GP's training is over a 10-year period and is considerably broader based than that of a specialist nurse. An accompanying paper elaborates on the topic, throwing light, or confusion, on the topic.

BMJ 2008;337:658–9 & 660–2.

Why are nursing home residents sent in to hospital to die?

This provocative question is posed by Dr Judith Partridge a clinician at St Mary's Hospital in London. The problem is set out as follows—such patients are often transferred as an emergency to the acute hospital setting, to receive sometimes formulaic and futile intervention at the end of their life. Commonly such intervention has no effect on eventual outcome but can often be unpleasant and distressing for the patient, their family and the medical team.

Why does this happen? Dr Partridge's list of reasons includes a lack of advanced care planning, poor physician and nursing back up, limited contact with specialist palliative care teams, and widespread uncertainty and lack of training among care home staff.

It all sounds somewhat familiar. On a more positive note, the recommended solutions involve proactive patient and family discussion about their expectations and end-of-life preferences. And, access, when appropriate, to specialist palliative care services. All sounds very reasonable.

Postgrad Med J 2008:84:281.

A vaccine against the human immunodeficiency virus (HIV)?

Development of an effacious HIV vaccine is clearly a high priority. The authors of this report point out that the usual vaccine development from live attenuated virus, whole killed virus, or subunit proteins—are either thought to be too dangerous or are ineffective in generating robust immune responses or protecting against HIV.

So a cell-mediated immunity vaccine provided by Merck Research Laboratories was used in this randomised trial. High-risk subjects were recruited in North America , the Caribbean, South America, and Australia. All were seronegative for HIV-1 at the outset. The seroconversion rate was 3% in both the vaccine recipient cohort and those in the placebo group. So, unfortunately the bottom line is that the cell-mediated immunity vaccine did not prevent HIV-1 infection or reduce early viral level.

Lancet 2008;372:1881–93.

Infection in the pathogenesis of chronic obstructive pulmonary disease (COPD)

It used to be thought that repeated airway infection and hypersecretion of mucus were the cause of COPD and the continuing deterioration. However, this theory was dented by the observation that the frequency of bacterial isolation from sputum was found to be similar in stable COPD and during exacerbations. Subsequently, exposure to tobacco smoke was identified as the predominant cause. Most clinicians think both are relevant.

In this 11-page review article, the evidence is turned over with the help of new molecular, cellular, and immunological techniques used to study the host-pathogen interaction. The authors offer a vicious cycle hypothesis—childhood respiratory disease and smoking impair innate lung defences. This allows microbial colonisation and antigens to damage airway epithelium, etc. The inflammatory response, increased proteolytic activity, and proteinase activity are also incriminated.

So it is as we suspected—both smoking and infection are relevant.

N Engl J Med 2008;359:2355–65.

Poisoned by eyedrops?

It is generally assumed that topical therapy is systemically inactive. This assumption and the fact that many patients may not mention eye drops when asked about their drug history may lead to health problems. How so? Topically administered medications gain access to the highly vascular nasal mucosa via the lacrimal drainage system and are variably absorbed, avoiding first-pass hepatic metabolism. And the laws of pharmacokinetics make ocular drug delivery more akin to intravenous than to oral administration.

The authors on this interesting paper point that there are reports in the literature of sulphonamide allergy after eye drops, possibly aplastic anaemia after chloramphenicol topical usage and a variety of other mishaps. The most well documented appear to relate to usage of topical β-blockers which may precipate or aggravate bronchospasm, congestive heart failure, bradyarrhythmias, sinus arrest, a variety of central nervous system effects and dyslipidaemias. One drop of timolol 0.5% solution in each eye approximates a 10 mg oral dose for treating systemic hypertension or angina. Their advice? “All topical ophthalmic agents should be considered potentially potent systemically”.

Med J Aust 2008;189:356–7.

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Health and safety will be the death of us

We are a bunch of people who like to worry. One of the constant medicolegal conundrums that we scare each other with is what to do at the scene of an accident. Should we risk the wrath of our professional medical bodies, ignore our ethical duty, pass by the wayside and not admit to being a doctor, or should we treat and risk being sued by the accident victim? Maybe there is a third answer to this which an airline provided me with.

On a recent flight from New Zealand to the UK I could see that the airline staff were struggling with a lady who was in distress. I felt that I had a duty to treat so told the staff what I did as a day job and whether I could help. The air steward then asked to see my qualifications before he would let me assist. My medical registration, and college certificates don't tend to be something I carry as hand luggage and therefore I had to go back to my seat. Without proof of being medically qualified I wasn't allowed to assist in an emergency.

Leaving aside the issue of whether an air steward really would be able assess my qualifications and fitness to practice, the airline's policy throws up a strange ethic. From their stance it seems to suggest that it is better to be sure that someone is not pretending to be a doctor rather than allowing a medical person who could potentially save someone's life from treating someone in a medical emergency.

To my mind an airline could be more criticised for the latter policy rather than the former. After all, how do you know that those certificates the doctor produces actually belong to them? I personally would rather be treated by the consultant A and E doctor who may be on my flight rather than the air steward with a first aid certificate.

It also ignores the fact that a search could be done on the line as long as the doctor can remember their number and also have proof of ID (my passport is with me when I travel, unlike my University certificate).

It seems that Health and Safety and risk management culture in trying to protect is actually exposing people who are acutely unwell to more risk.

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Discussing end-of-life issues with Asian patients and their families

The recent article entitled *Telling the truth to Asian patients in the hospital setting* by John Windsor and his team (http://www.nzma.org.nz/journal/121-1286/3378) is a timely reminder of both the need and difficulty of communicating accurately in clinical situations with patients of a different ethnic group and whose first language is not your own.

The issues raised in the article arise similarly in the hospice setting when discussing end-of-life issues with Asian patients and their families. We also share similar concerns about interpreters fully grasping the implications of clinical circumstances and themselves being uneasy talking about end-of-life issues with the risk that the tenor of the consultation is not fully delivered.

The development of the questionnaire is an innovative approach to lessening some of the concerns about who receives and passes on information to the patient

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National Immunisation Register inaccuracies and duplications

For some time we have been concerned about inaccuracies and duplications with the National Immunisation Register (NIR) database. Below are results from a local audit of patients linked to the NIR at a general practice level in the Rotorua region.

The NIR Coordinator from Lakes DHB undertook an NIR download with the following criteria: Using a birth cohort from 1 Aug 2005 to current; children domiciled to Lakes DHB as of 28/10/08; data from one medium-sized General Practice in urban Rotorua with a registered population of 5100 which includes 234 children born on or after 1 August 2005. The practice uses Profile as its Practice Management System.

The results showed: 253 children (born on or after 1 August 2005) had an NIR link to the practice, and of these:

- 55 children (22%) had duplicate messages because they had seen more than 1 provider at the surgery—nominated and associated provider status.
- 7 children (2.7%) had never been seen at the surgery at any time in their lives.
- 26 children (10%) had transferred out of the DHB region.
- 21 children (8%) had transferred to other surgeries within the Rotorua region.
- Only 198 of the 253 (78%) with an NIR link to the practice were currently registered with the practice:
    - Of these 198 children, 43 (21%) were "late" (mostly due to having to backdate entries and catch ups for the introduction of Prevenar™)
    - Only 3 (1.5%) were informed consent declines.
    - In total, 155 (78%) were fully vaccinated or informed consent declines.

Furthermore there were an additional 36 children in the eligible age group who were registered with the practice but had no NIR link from this download.

The above data confirms our concerns about the accuracy of using NIR data as the basis for establishing immunisation rates. It also highlights where some of the errors are occurring. The level of duplicated messages has created a considerable burden for Practice Nurses. As a result, many have stopped routinely using the NIR. There is no NIR facility currently available for practices to notify transfers both from and to the surgery. The current functionality of the NIR is not supportive of General Practice processes. This has created a significant lack of confidence in the effectiveness of the NIR at a provider level. We are currently working with the DHB at addressing some of these issues locally but clearly this is a national issue.

Neil Poskitt
GP, Rotorua

Sue Taft
Child Health and Immunisation Coordinator, Rotorua Area Primary Health Services

THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

Research into the Cartwright Inquiry

The 2008 Nordmeyer Lecture to the Wellington School of Medicine, entitled *Inquiries into health care: learning or lynching?* was delivered by Health and Disability Commissioner Ron Paterson and published in the *New Zealand Medical Journal* on 28 November 2008.[1] It contained some misleading statements about my current research into the Cartwright Inquiry, which I wish to correct.

Mr Paterson stated that, 'The revisionists, most recently in the guise of University of Auckland historian Lynda [sic] Bryder, continue to argue that Judge Cartwright got it wrong'. The implication is that I am part of some unspecified group is entirely mistaken; I know of no such group, and am certainly not part of one.

I am an independent scholar and medical historian of 30-years standing with an international reputation in my field. During this time I have published articles and reviews in all the major English-language medical history journals and have frequently assessed research proposals for the Wellcome Trust for the History of Medicine. I also currently hold honorary professorships at the London School of Hygiene and Tropical Medicine and at Glasgow Caledonian University.

Mr Paterson cites Charlotte Paul's claim that 'not a scrap of empirical evidence has been published by the revisionists'. If he is including me as one of the so-called revisionists, I would point him towards my recent paper on the history of cervical screening, published in the *Journal of Epidemiology and Community Health*.[2] However, I take issue with the 'revisionist' label. As the first academic historian to have extensively reviewed the documentation related to the Cartwright Inquiry I am not revising what previous historians have written. Like any other historian my role is to explain past events, according to the available evidence. My research is based on a wide range of sources, including the Inquiry transcripts, other government archives, and international medical journals.

Mr Paterson claims that I believe 'that the women who were part of Green's study had the same outcomes in terms of invasive cancer as those treated before or after.' This remark is based on hearsay and speculation rather than evidence; I make no such claim in my forthcoming book about the Cartwright Inquiry, which will be published in 2009 by Auckland University Press. I believe that this text will withstand close scrutiny and hope that Mr Paterson and others will read it with an open mind and assess the research on the basis of the supporting evidence.

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1. Paterson R. Inquiries into health care: learning or lynching? New Zealand Medical Journal. 2009;122(1288). http://www.nzma.org.nz/journal/121-1286/3376
2. Bryder L. Debates about cervical screening: an historical overview. Journal of Epidemiology and Community Health. 2008;62:284–7.

THE NEW ZEALAND MEDICAL JOURNAL
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Professional Misconduct – Indecent Assault (Med08/89P)

Charge

A Professional Conduct Committee charged that Dr Theomal Hirantha Joseph Fernando, (the Doctor), medical practitioner formerly of New Plymouth had been convicted of offences that reflected adversely on his fitness to practise.

The particulars of the charge related to 26 counts of indecent assault on 10 complainants while practising as a medical practitioner.

Finding

The hearing proceeded on the basis of an agreed summary of facts, and the Doctor accepted that the convictions reflected adversely on his fitness to practise. After consideration of the convictions and the agreed facts the Tribunal was satisfied that the Doctor's conduct did reflect adversely on his fitness to practise.

Background

In summary, the offending which was admitted:

- involved 10 of the Doctor's female patients;
- occurred while he was practising as a medical practitioner at New Plymouth;
- spanned a period of approximately 21 years from 7 October 1981 to 25 July 2002;
- with the exception of one count (involving a family friend), took place in the Doctor's surgery during the course of examinations and therefore in the context of his professional practice.

The learned High Court Judge, Priestley J, stated in his sentencing notes that the Doctor's offending:

"Constituted a gross breach of trust which patients are entitled to expect from medical practitioners"

The Doctor was sentenced to a total of three years and two months imprisonment for these offences.

Reason for Finding

The Tribunal was satisfied that the offences of which the Doctor was convicted reflect adversely on his fitness to practise for the following reasons:

- all but one of the multiple counts occurred in his surgery;
- there was a gross breach of trust, and of professional boundaries;

- there were multiple offences, over more than 20 years;
- most of the offences involved young women, who were particularly vulnerable;
- the evidence was that the offending affected patients very significantly.

Considered separately, the Tribunal was satisfied that each particular was established, and that each established particular reflected adversely on the Doctor's fitness to practise. Therefore, considered cumulatively, it followed that there had been an extremely serious breach which reflected adversely on the Doctor's fitness to practise.

Penalty

The Tribunal considered the offending was completely reprehensible.

It was recognised that the Doctor had considerable support from patients who regard him highly, and there was no doubt that he had done much for the community. However, the Tribunal considered that it must be primarily concerned about the safety of the public; it had no hesitation in concluding that an order of cancellation of registration should be made in this case. It was satisfied this was the only sensible option given the gravity of the offending.

The Tribunal ordered that the Doctor's registration be cancelled. This was announced at the hearing on 2 July 2008, and that the order of cancellation took effect from that date.

The Tribunal censured the Doctor and expressed in the strongest possible terms that the persistent offending of this nature over many years was completely unacceptable.

The Doctor was ordered to pay $6,750.00 for the costs and expenses incurred by the PCC and $5,750.00 for the costs and expenses as to the conduct of the hearing by the Tribunal.

It was directed that details of the decision be published on the Tribunal's website, and in the New Zealand Medical Journal.

The full decisions relating to the case can be found on the Tribunal web site at www.hpdt.org.nz
Reference No: Med08/89P.

THE NEW ZEALAND MEDICAL JOURNAL
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Maxwell George Goodey

Dr Goodey (FRNZCGP MB ChB) died on 8 March 2008 in Sydney, Australia. Max was born in Te Kuiti, New Zealand near the world-famous Waitomo Caves. As a boy he learned from his Scots grandfather how to grow and store fruits and vegetables and took on a life of amateur horticulture with the greatest enthusiasm.

He began a BSc at Victoria University in Wellington in botany and geology which was interrupted by the outbreak of war. He served with the Royal New Zealand Engineers in the Pacific; at Honiara, Vela-lavela, Guadalcanal, and New Caledonia and returned to medical school at Otago.

He came up to Auckland and Thames to do his house surgeon's time. His first locum was for Dr Charlie Mules of Dargaville. He painted, listened to classical music, acted in plays and became a good cook and jam-bottler; ever collecting and cultivating plants. He opened a general medical practice, in Forest Hill Road and then in the centre of Henderson.

He met Margaret Gleeson in Auckland and they married in December 1954, the beginning of 53 years of wedded happiness .

Max moved to Parnell in 1960 and gave anaesthetics for much of the 1960s. He also served as a doctor for the ports and began his long association with Student Health at the University of Auckland and his role as Police Surgeon in Auckland and Cancer Detection, all of which were to last over 20 years, all the while keeping his general practice in Gladstone Road near the Parnell Rose Gardens, and tending his rare plant specimens. He gave over 300 cultivars to the Auckland Institute of Horticulture and had a special interest in and knowledge of palms and cliveas.

He keenly supported the Medico-Legal Society with his good friend the late Professor Jack Matthews.

Sir Douglas Robb thought him one of Auckland's best diagnosticians;

Max prolonged and saved the lives of many in Auckland and (during locum periods) in Australia by sharp spotting of illness, and great devotion. He gave so generously to his community. He outlived many famous contemporaries and most of his referring specialists and fellow GPs. In his last 8 years he had a very full life of travel, bridge, reading and seeing the films, and remained registered as a general practitioner in New South Wales.

In latter years, Max, ever a keen supporter of medical conferences and reunions, travelled frequently to Asia and beyond. In one last trip to Europe via the Middle East in October 2006 he triumphed over his old arthritic knees to enter the magnificent courtyard of the Hospices de Beaune and several Cathedrals en route to Paris, through the Marne where his uncle had died in the Great War. He did not need any coaxing to sample the wines of Burgundy or elsewhere.

He remained cheerful at all times, and rose early to do everything he had to do and greet all manner of patients with his famous wicked sense of humour. Many would

readily say that no more decent or caring medical man ever walked this earth. Epitomising the solo GP, in a style of medicine not offered by many today, Max always put people before either profitability of practice or senseless bureaucracy. He leaves one son, Rhys and thousands of patients and friends and their gardens.

Max's son, Rhys Goodey, wrote this obituary.

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GRAHAM AITKEN NUFFIELD TRUST

GRAHAM AITKEN NUFFIELD MEDICAL POSTGRADUATE TRAVELLING SCHOLARSHIP

Applications are invited from well qualified New Zealand medical graduates in the 25–35 age group for the above Scholarship.

The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom.

The Scholarship will provide up to three return airfares to the U.K., together with allowances amounting to $3000.

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the U.K. who will provide salary and facilities.

For further information please consult the Deans of the Schools of Medicine or write to Professor A.D.Campbell, Honorary Secretary, Managing Trustees, Graham Aitken Nuffield Trust, C/- Department of Chemistry, University of Otago, P.O. Box 56, Dunedin.

Applications must be submitted to Professor Campbell by 31 March 2009

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Reviewers for the New Zealand Medical Journal in 2008

The Editorial Board (Frank Frizelle, Tim Buckenham, Roger Mulder, Richard Beasley, Jennie Connor, Jim Reid) and Editorial Team (Frank Frizelle, Editor; Brennan Edwardes, Production Editor; Sally Bagley, Administrative Assistant) thank all those who generously gave their time and expertise in reviewing papers for the *New Zealand Medical Journal* in 2008. (We apologise to anyone whose name has been inadvertently omitted from the following list.)

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