

CONTENTS

This Issue in the Journal

- 3 A summary of the original articles featured in this issue

Editorial

- 6 Outcomes for differentiated thyroid cancer in New Zealand:
comparison of South Island and Auckland data
James H F Shaw

Original Articles

- 13 Deaths due to differentiated thyroid cancer: a South Island, New Zealand experience: 1984–2009
Bevan Brownlie, John Turner, Al S Abdelaal
- 22 The demographics and prevalence of youth (15–24 year olds) with type 1 diabetes in the Canterbury District Health Board catchment area in 2010: has the prevalence changed since 2003?
Balsam Obaid, Eileen Britt, Mark Wallace-Bell, Shelley Johnson-Elsmore
- 29 Biosocial profile of New Zealand prosthetic eye wearers
Keith R Pine, Brian Sloan, Robert J Jacobs
- 39 Gout in women: differences in risk factors in young and older women
Sunil Kumar, Rajiv Gupta, Ravi Suppiah
- 46 Bariatric surgery makes dramatic difference to health-related quality of life
Briar McLeod, Grant Beban, Jill Sanderson, Ann McKillop, Andrew Jull
- 53 Undetected rheumatic heart disease revealed using portable echocardiography in a population of school students in Tairāwhiti, New Zealand
Geoffrey Cramp, Mary Stonehouse, Rachel Webb, Diane Fuller, Gina Chaffey-Aupouri, Nigel Wilson
- 65 A decade of serious non-fatal assault in New Zealand
John Langley, Pauline Gulliver

Clinical Correspondence

- 77 Non-traumatic spontaneous spinal subdural haematoma
Nur A B Haji Mohd Yasin, Daniella Donato-Brown, Ahmad Taha

- 81 Right atrial mass—a venous thrombosis in transit
 Jonathan W Hinton, John Lainchbury, Ian Crozier
- 84 Medical image. The malign face of atrial fibrillation
 Suleyman Ercan, Adnan Dogan, Vedat Davutoglu, Gokhan Altunbas
- 86 Medical image. Persistent fever in ulcerative colitis
 Pazhanivel Mohan, Rahul Anil Kothari, Krishnaveni Janarthanan

Letter

- 88 Premature failure of artificial joint components
 Geoffrey Horne

100 Years Ago in the NZMJ

- 89 Presidential Address: Quackery and chemists

Methuselah

- 90 Selected excerpts from Methuselah

Obituaries

- 92 Thomas Derisley (Derry) Stuart Seddon
- 95 John Burd Carman

Notices

- 97 Medical Benevolent Fund
- 98 University of Otago Faculty of Medicine: Postgraduate Scholarship in
 Obstetrics and Gynaecology

This Issue in the Journal

Deaths due to differentiated thyroid cancer in New Zealand: a South Island, New Zealand experience: 1984–2009

Bevan Brownlie, John Turner, Al S Abdelaal

We assessed differentiated thyroid cancer (DTC) deaths from the northern half of New Zealand's South Island by reviewing Christchurch Hospital Thyroid Clinic and Oncology Department clinical records of patients who died of differentiated thyroid cancer of follicular cell origin over the 25-year period 1984–2009. During the 25-year study period, 25 patients died from differentiated thyroid cancer. All patients (17 female, 8 male) were Caucasian, with average age 65 years (47–86 years) at presentation. The average survival from diagnosis was 5.5 years (0.2–22 years).

The demographics and prevalence of youth (15–24 year olds) with type 1 diabetes in the Canterbury District Health Board catchment area in 2010: has the prevalence changed since 2003?

Balsam Obaid, Eileen Britt, Mark Wallace-Bell, Shelley Johnson-Elsmore

There was no significant increase in the prevalence of type 1 diabetes in youth in the Canterbury District Health Board catchment area between 2003 and 2010. However, due to the population increase, the absolute number of adolescents and young adults with type 1 diabetes have increased, which implies an increased demand on health care associated with diabetes compared to seven years ago.

Biosocial profile of New Zealand prosthetic eye wearers

Keith R Pine, Brian Sloan, Robert J Jacobs

This study sought to address the lack of information about prosthetic eye wearers in New Zealand. Based on figures from Northland we estimated that approximately 1 in every 1440 people wear artificial eyes in this country and that most eye loss was through accident. In the past 20 years, however, medical conditions have become the main cause of eye loss due to a decline in accidents. This decline is consistent with decreasing workplace and traffic accidents in the general population which is due to improved workplace safety standards, safer roads and better medical management. An additional finding of this study was that in spite of good healthcare provision, mucoid discharge is prevalent amongst prosthetic eye wearers in New Zealand. No standardised treatment exists for this distressing condition and we plan further research to establish an evidence-based protocol for its management.

Gout in women: differences in risk factors in young and older women

Sunil Kumar, Rajiv Gupta, Ravi Suppiah

Gout is a common medical problem in men and postmenopausal women. Its prevalence in the community is rising and we are seeing younger women presenting with gout. This study was to look at risk factors for gout in women and see if there was any difference in the risk factors between young and older women. It showed that women who develop gout are usually over the age of 50 years and tended to have several underlying medical conditions like, hypertension, obesity, kidney disease, diabetes and use diuretic medicines. Main difference between the two groups were that younger women who developed gout had higher body mass index (more obese) and were of Māori or Pacific ethnicity.

Bariatric surgery makes dramatic difference to health-related quality of life

Briar McLeod, Grant Beban, Jill Sanderson, Ann McKillop, Andrew Jull

We aimed to explore the impact of bariatric surgery on health-related quality of life. Patients completed questionnaires at their first preoperative clinic and at their 6-month follow-up appointment after surgery. Twenty-three patients underwent laparoscopic Roux-en-Y gastric bypass and five underwent laparoscopic sleeve gastrectomy. The patients were predominantly New Zealand European, female, with a body mass index greater than 40 kg/m². Significant improvements in health-related quality of life were observed after surgery.

Undetected rheumatic heart disease revealed using portable echocardiography in a population of school students in Tairāwhiti, New Zealand

Geoffrey Cramp, Mary Stonehouse, Rachel Webb, Diane Fuller, Gina Chaffey-Aupouri, Nigel Wilson

A school-based echocardiography scanning programme in 2009 in Tairāwhiti (Gisborne and surrounding region) of nearly 700 school students showed that 1.61% of the students scanned had established rheumatic heart disease. Just over 1% had previously undetected rheumatic heart disease. The place of echocardiograph screening for Rheumatic Heart Disease is still to be established in New Zealand. However the current programmes of primary prevention by sore throat swabbing in schools in areas at high risk of rheumatic fever is advocated particularly in Tairāwhiti where anyone aged between 5 and 45 years with a sore throat should be swabbed and treated for Group A streptococcus.

A decade of serious non-fatal assault in New Zealand

John Langley, Pauline Gulliver

We studied the distribution of, and trends in, the characteristics of serious non-fatal assault injury for the period 2000–2009. Males, 15–24 year olds, Māori, Pacific Islanders, and those from deprived neighbourhoods had significantly higher assault rates. There is a disturbing level of serious assault in New Zealand and the situation is getting worse. We need to review current efforts to prevent these incidents.

Outcomes for differentiated thyroid cancer in New Zealand: comparison of South Island and Auckland data

James H F Shaw

Brownlie and colleagues¹—in this issue of the *NZMJ*—have reviewed the outcomes of the bulk of the New Zealand South Island cases of differentiated thyroid cancer (DTC) seen between 1984 and 2009; a total of 411 cases. Twenty-five patients or 6% of the total died, compromising twice as many women as men, with a mean age of 65 years. The patients were operated on by 19 different surgeons and no patient less than 45 years of age died. All deaths were in Caucasian patients, and 15 of the 25 were incurable from the outset as a result of locally advanced unresectable disease or metastatic disease at diagnosis.

According to Table 1, local failure in the neck occurred in 9 or 36% of their patients. The ratio of females to males who died was similar to the gender ratio for all differentiated thyroid cancer (DTC) patients: 2.1:1 favouring females.

Table 1. Prognostic effects of extra-thyroid extension and regional disease on outcome

Variables	T4A N0/n1a	T4A N1b	T1-3 N1b
A&W	56% 11/18	40% 10/25	77% 17/22
N1 Rec	66% 9/18	13% 2/25	18% 4/22
AWD	22% 4/18	50% 9/25*	9% 2/22
DOD	0	20% 5/25*	5% 1/22

N0 no regional disease, N1a central compartment disease; N1b lateral neck disease, A&W alive & well; N1 Rec recurrence in lateral neck; AWD alive with disease; DOD dead of disease. *Significantly different to both the other groups $p < 0.04$ or better; T4A extra-capsular spread.

The editorial author has reviewed his personal experience of over 2000 thyroidectomies performed in Auckland between 1984 and 2012; 300 patients had differentiated thyroid cancer.

The patients treated between 1984 and 2007 have been analysed. These data have been presented both nationally and internationally but never published. Statistical analysis utilised Chi-squared analysis. Patients were staged in accord with The Mayo Clinic staging MACIS,² and also using the guidelines from the Joint American Staging System for Thyroid Cancer.³

Lymph node status (levels 1–7) was as described by Memorial Sloan Kettering Cancer Centre.^{4,5} There were 127 DTC patients aged <45 years (97 papillary, 30 follicular), and 123 aged >45 years (94 papillary, 29 follicular).

The ethnic distribution was similar for the two age groups with Europeans making up 61%, Māori 13%, Asian 15%, and Pacific 14%. Longest follow-up (FU) is 25 years with mean FU 13 years.

The vast majority of patients were treated with total thyroidectomy. Over recent years central node dissection (level 6) has been almost universally performed for papillary cancer or follicular variant of papillary cancer in order to achieve lower recurrence rates in the central compartment and to have a higher percentage of patients with post-op thyroglobulin levels approximating zero.⁶

In addition for patients with lateral neck disease, neck dissection of levels 2–5 (after level 6 the commonest sites of involvement are in order levels 4,3 and 2 with level 5 being involved in >20% of patients) was employed for most patients.⁵

There are a number of differences in this Auckland experience when compared with the South Island report of Brownlie and colleagues;¹ in particular the following.

- In Auckland, major prognostic factors governing outcome were similar to those from The Mayo Clinic,² these included patient age, the presence of locally advanced disease, adequacy of resection, the status of regional disease, the completeness of the resection performed.
- In addition, in Auckland the ethnicity of the patient was significant with Asian patients having the best outcomes while Pacific patients had the worst outcome especially male Pacific patients aged >45 years.⁷
- Age was a strong prognostic indicator for both recurrence and death in accord with Mazzaferri and Jhiang.⁸ Recurrence was frequent in young patients and in older patients with the lowest rate of recurrence in middle aged patients. In contrast, DOD and the presence of unfavourable bone mets were largely confined to patients over 60 years of age. See Figure 1.

Abbreviations used in the following figures and tables

N0 no regional disease, **N1b** lateral neck disease, **N1a** central compartment disease

Rec recurrence, **N1 Rec** Recurrence in lateral neck

Mets metastatic disease, **M1 Rec** develop metastatic disease

DOD dead of disease

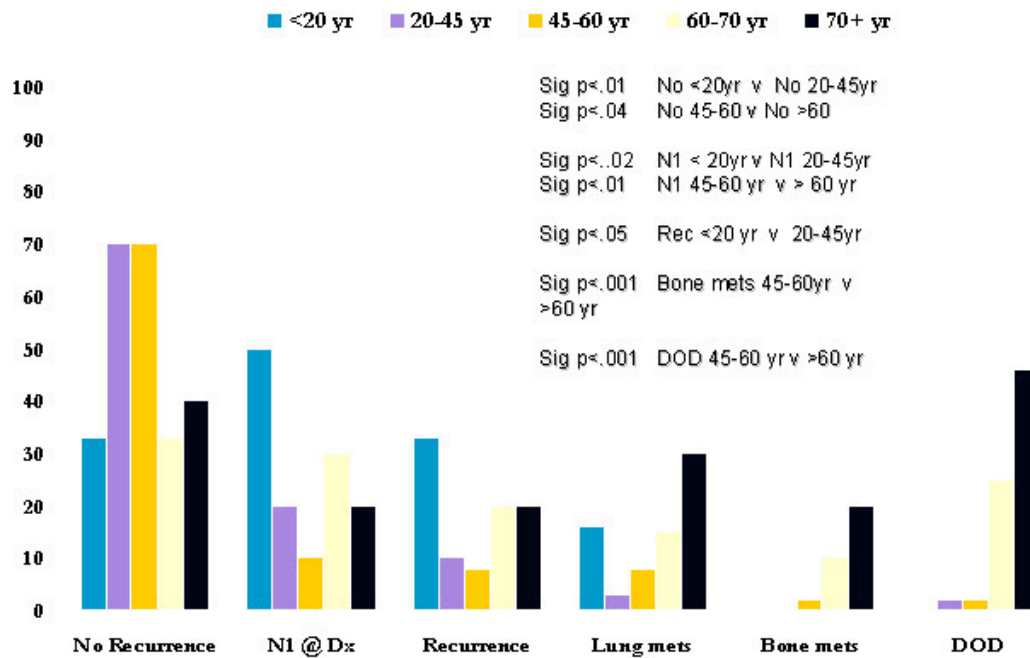
@ **Dx** at diagnosis

NS not statistically significant

AWD alive with disease, **A&W** alive & well

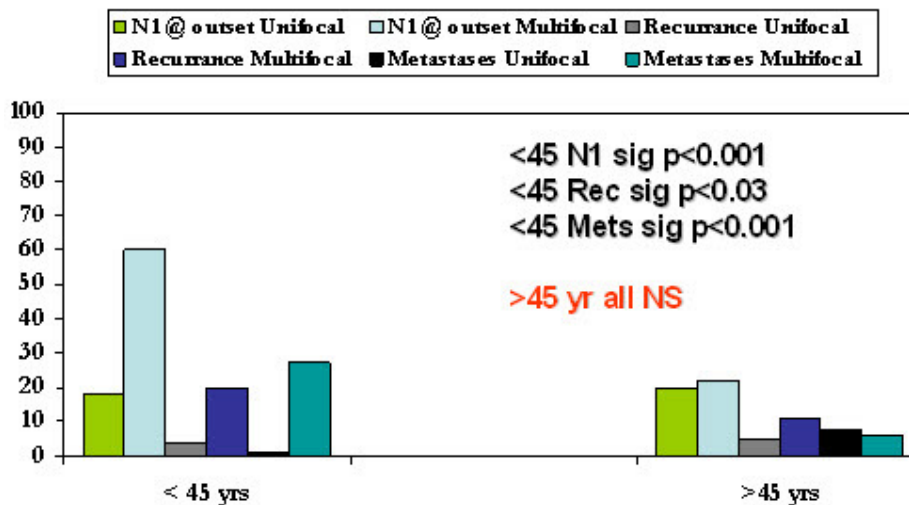
* significance $p < 0.05$ ** significance < 0.03 or better

Figure 1. Effect of age on outcome of papillary cancer



- Multifocal tumours were associated with less good outcome in patients < 45 years, but multifocality did not influence outcome in older patients. See Figure 2.

Figure 2. Multifocal papillary primary: influence of age



- Progressive increase in tumour size had only a minor effect on outcome, but extra-thyroid spread (T4a) had a major effect on outcome. See Table 2.

Table 2. Effect of tumour size and extra-capsular spread (T4A) on outcome

Size CM	T1 <2 cm	T2 2–4 cm	T3 >4 cm	T4A
A&W	96% 67/71	91% 78/80	80% 22/28	49% 21/43**
AWD	1% 1/71	0% 0/80	7% 2/28	30% 13/43**
DOD	0% 0/71	1% 1/80	0% 0/28	12% 5/43**

** Significant difference from other groups $p < 0.04$ or better.

- In addition if there was lateral neck disease coupled with a locally invasive primary (Stage T4A N1B) then the outcome was significantly worse. See Table 2. This is in accord with the findings of Hay et al from The Mayo Clinic.²
- Also Table 2 demonstrates the relatively good outcome for regional disease when there is no locally invasive primary, the relatively good outcome when a locally invasive primary was not coupled with regional disease.
- For younger patients, the number of nodes involved impacted on recurrence. See Table 3. This is in accord with Sugatani et al.⁹

Table 3. Papillary cancer in patients aged <45 years of age: effect of >5 involved nodes

Variables	Nil or <5 Nodes	>5 Nodes
A&W	93 % 75/81	57% 8/14 $P < 0.001$
N1 Rec	6% 5/81	29% 4/14 $P < 0.01$
M1 Rec	1% 1/81	36% 5/14 $P < 0.001$
DOD	1% 1/81	0% 0/14 NS

- In patients aged >45 years the size of nodes impacted significantly on both recurrence and survival. See Table 4. This finding is in agreement with Kitajiri et al.¹⁰

Table 4. Papillary cancer in patients aged >45 years: effect of nodes >3 cm diameter

Variables	No or nodes <3 cm	Nodes >3 cm
A&W	79% 59/75	38% 6/16 $p < .001$
N1 Rec	9% 7/75	19% 3/16 NS
M1 Rec	11% 8/75	43% 7/16 $p < .001$
DOD	1% 1/75	25% 4/16 $p < .003$

- Completeness of resection was a highly significant prognostic factor, as was the case at Mayo [Clinic].² Patients with extensive disease necessitating borderline resection of cancer involving trachea, larynx, oesophagus did significantly less well than when complete resection could be performed. See Table 5.

Table 5. Outcome: incomplete versus complete resection

Variables	Complete resection	Incomplete resection
A&W	74% 147/204	12% 3/26 p<0.01
LR Rec	1% 2/204	31% 8/26 p<0.01
AWMets	1% 2/204	62% 16/26 p<0.01
DOD	0.5% 1/205	27% 7/26 P<0.01

- Outcome as a function of race is shown in Table 6. The outcomes for Europeans, Māori, and Asians were similar irrespective of age, and the outcomes of Pacific patients aged <45 years were also similar to the above three ethnic groups.
- Asians were relatively over-represented (Asian population over the study period would have been much less than 50% of the Māori population⁷), and Asians had the best outcomes of any race with no patients either AWD or DOD.
- In contrast Polynesians aged >45 years fared significantly worse than other races largely due to the high proportion of males aged >65 years with T4A, N1b disease (6 patients), along with 4 Polynesian males with T4b disease, 3 of whom died of disease.

Table 6. Differentiated thyroid cancer (DTC) in all ages: ethnicity versus outcome

Variables	European	Asian	Māori	Pacific <45	Pacific >45
A&W	88% 127/144	100% 37/37	77% 14/18	73% 11/15	30% 6/20 p<0.03
AWD	10% 15/144	0%	22% 4/18	27% 4/15	45% 9/20 p<0.05
DOD	1% 2/144	0%	0%	0%	25% 5/20 p<0.03

- MACIS score (based on presence of Metastases, Age, Completeness of resection, Invasion and Size²) stratified patients for outcome. See Table 7.

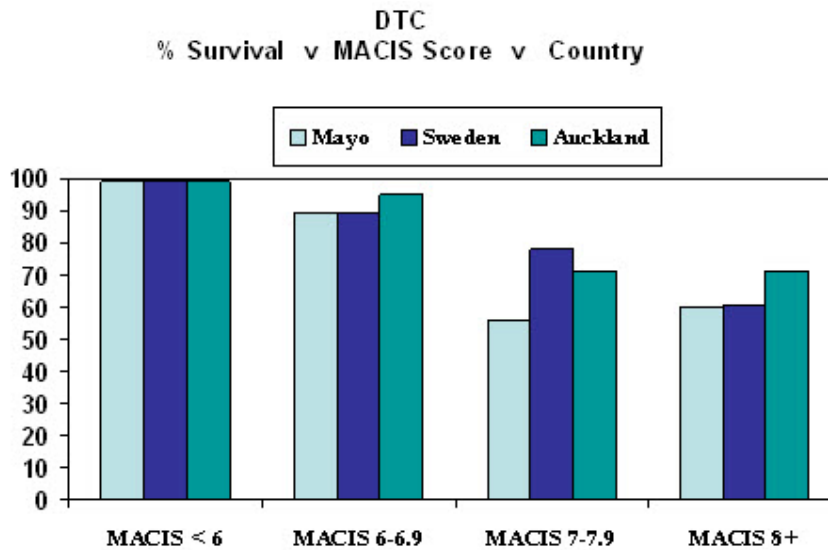
Table 7. Outcome of DTC in all ages versus MACIS score

MACIS Score	<6	6-7	>7
A&W	99% 185/187	66%* 16/24	21%* 5/24
AWD	0.5% 1/187	29%* 7/24	50%* 12/24
DOD	0.5% 1/187	4%* 1/24	29%* 7/24

* Significantly different to preceding MACIS value p<0.04 or better.

- When survival as a function of MACIS score for patients treated in Auckland, Mayo [Clinic],² and Sweden¹¹ were compared, the survival data were similar. See Figure 3. In addition, the percentage of patients with high MACIS score seen in Auckland was double that seen at Mayo.

Figure 3. DTC Outcomes of Mayo Clinic (USA) versus Sweden versus Auckland (NZ)



- Gender was unimportant in survival for any patient group. When all females were compared with all males the outcomes were virtually identical. See Table 8 below.

Table 8. DTC effect of gender on outcome: all female patients versus all male patients

Variables	Females	Males
A&W	88% 168/192	83% 40/48
AWD	8% 15/192	10% 5/48
DOD	5% 9/192	6% 3/48

Overall there are a number of differences in outcome for South Island patients versus North Island patients. This is largely explained on the one hand by the higher percentage of good prognosis Asian patients in the North Island coupled on the other hand with a higher proportion of poor prognosis Polynesian patients seen in Auckland, many of whom were referred from Samoa.

There is little doubt that most patients with differentiated thyroid cancer are best treated by total thyroidectomy and for papillary cancers a central neck dissection is also appropriate.⁶

An assessment of the roles of adjuvant radioiodine (RI) and external beam radiation (EBR) in the management of DTC is beyond the scope of this editorial. However it is this author's view that while RI is useful for treating metastatic thyroid cancer, data supporting its use as an adjuvant therapy in young patients with good prognosis lesions, is limited.¹²

Available World data indicate that RI may diminish recurrence in older patients with unfavourable prognostic factors.¹² In contrast, the data supporting the use of EBR to diminish recurrence in patients with unfavourable central or lateral compartment disease is more compelling,¹² and this modality is currently probably under-utilised in New Zealand for managing patients with locally advanced central and/or lateral neck disease.

Competing interests: None known.

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Deaths due to differentiated thyroid cancer: a South Island, New Zealand experience: 1984–2009

Bevan Brownlie, John Turner, Al S Abdelaal

Abstract

Aim To assess differentiated thyroid cancer (DTC) deaths from the northern half of New Zealand's South Island.

Methods Retrospective review of Christchurch Hospital Thyroid Clinic and Oncology Department clinical records of resident patients who died of differentiated thyroid cancer of follicular cell origin over the 25-year period 1984–2009.

Results During the 25-year study period 25 patients died from differentiated thyroid cancer. All patients (17 female, 8 male) were Caucasian, with median age 65 years (47–86 years) at presentation. Most (24/25) patients presented with advanced (15 Stage IV, 9 Stage III) disease. Three patients initially presented with cervical lymphadenopathy and four patients with distant metastases—three patients with bone metastases, and one with a pleural effusion. The pathological classification of the tumours included 14 papillary cancers (four were follicular variants), six follicular cancers and five Hürthle cell cancers.

The majority of primary tumours were large (>4cm) and 11 were locally invasive. However one patient had a small (1.3cm) papillary cancer and presented with a pleural effusion. Surgical removal of the primary tumour was attempted in 24 of the 25 patients, 18 received postoperative radioiodine ¹³¹I therapy, and three had external beam radiation therapy. The median survival from diagnosis was 5.5 years (0.2–22 years) with two Stage IV patients (both with Hürthle cell cancers) dying within 4 months. The majority of patients died of metastatic disease but seven died of local disease.

Conclusions During the 25-year study period, 25 patients died of differentiated thyroid cancer which approximates to one DTC death per year in our region. The median age at diagnosis was 65 years with no patients <45 years of age, and the female to male ratio was 2.1:1. Most patients presented with advanced disease—7 patients (28%) had distant metastases. Hürthle cell cancers were over-represented (20%) in our series.

Differentiated thyroid cancer (DTC) of follicular cell origin is usually an indolent tumour and with adequate initial surgery the prognosis is good. However a small proportion of patients with DTC die as a result of local invasion of cervical structures or distant metastases. Papillary thyroid cancer, the most common histological type, has a more favourable prognosis than follicular and Hürthle cell cancers.

Adverse prognostic indicators include age greater than 45 years, distal metastases, extrathyroidal invasion, and primary tumour greater than 4cm in diameter.¹ Several

prognostic indices have been developed, but the most generally used is the TNM (tumour, nodes, metastases) classification which has been upgraded.²

Thyroid cancer is relatively uncommon and is responsible for some 1% of New Zealand cancer registrations.³ In New Zealand as in other developed countries the incidence of differentiated thyroid cancer is increasing and it is uncertain whether this is due to better diagnostic testing, improved diagnostic criteria or environmental factors.

The New Zealand incidence rate has risen between 1971 and 1996 from 3.7 to 6 per 100,000 females and 1.7 to 2.6 per 100,000 males. The mortality rate in the same period in contrast has shown a steady decline from 1.1 to 0.7 per 100,000 females and from 0.7 to 0.4 for males.³

There have been few reports of thyroid cancer deaths, with most from large northern hemisphere referral centres.^{4–8} In this study we document 25 New Zealand South Island patients dying of differentiated thyroid cancer (disease-specific mortality) in the 1984–2009 interval—a 25-year experience. This review of our experience should lead to better initial management, and hopefully improved prognosis.

Patients and Methods

The Thyroid Clinic and Oncology Services at Christchurch Hospital are the referral centre for the northern half of the South Island of New Zealand, population 553,000 (2001 Census). Review of departmental files identified resident patients who died of differentiated thyroid cancer (DTC) of follicular cell origin in the 1984–2009 period—patients with anaplastic and poorly differentiated tumours, medullary thyroid cancer and lymphoma were excluded. Clinical data was collated and the initial histological slides of earlier patients were reviewed. Only patients dying as a direct consequence of cancer (disease-specific mortality) have been included.

Twenty five patients dying of DTC were identified from our records. The study population of 25 subjects, all Caucasian, consisted of 19 patients from the North Canterbury district, 3 from South Canterbury, and 3 from the West Coast.

Pathology (see Table 1)—Current WHO criteria were applied for thyroid histological classification. The original histological slides for patients diagnosed prior to 2000 were reviewed by senior pathologists, but slides were not available for Patient 3 diagnosed in 1967. Three of the earlier patients originally classified as follicular tumours were re-classified as papillary thyroid cancer-follicular variant (Pf).

Results

Clinical—The age, sex, year of diagnosis and clinical presentation is summarised in Table 1 where the patients (1 to 25) are listed in order of date of death—late 1984 to December 2009. The series of 25 patients (17 female, 8 male) had a median age at diagnosis of 65 years (47–86 years). Ten of the 25 patients had a long-standing history of goitre, and one goitre was very large (Patient 5). Three patients had previous treatment for cancer—two for breast cancer and one for endometrial cancer.

Prior neck irradiation in adulthood had been received by two patients—one patient had radiotherapy for ipsilateral breast cancer 19 years earlier, and one patient had received three radioiodine ¹³¹I doses (total 25mCi) 30 years earlier for contralateral toxic thyroid nodule. No patient had a family history of thyroid cancer but six had a family history of goitre.

Table 1. Clinical data, histological classification and TNM staging using revised (2002) classification.

Patient	Age/Sex	Year of Diagnosis	Presentation	Histology	Stage	T	N	M
1	52M	1973	unilat. goitre	P	II	2	0	0
2	70F	1983	cervical nodes	P	IVC	4a	1b	1
3	48F	1967	unilat. goitre	F	III	3	0	0
4	62M	1984	cervical nodes	P	IVC	3	1b	1
5	52F	1984	huge goitre, dyspnoea	F	IVC	4a	0	1
6	54F	1986	unilat. goitre, dysphonia	P	IVA	4a	0	0
7	47M	1983	stridor, dyspnoea	P	IVA	4a	0	0
8	70F	1993	pleural effusion	P	IVC	1	1b	1
9	66F	1986	nodule	F	III	3	0	0
10	62F	1988	unilat. goitre	P	IVB	4b	0	0
11	54F	1973	unilat. goitre, dyspnoea	H	III	3	0	0
12	61F	1994	unilat. goitre	F	III	3	0	0
13	86F	1995	bone 2 ^o	F	IVC	x	x	1
14	56F	1996	bone 2 ^o	Pf	IVC	x	1b	1
15	66M	1986	nodule	F	III	3	0	0
16	65F	1990	nodule	Pf	IVA	4a	0	0
17	68M	1989	unilat. goitre, dyspnoea	Pf	III	3	0	0
18	81F	2001	nodule, dyspnoea ◊	H	IVA	4a	0	0
19	67F	1996	unilat. goitre	H	III	3	0	0
20	76F	2000	nodule	P	IVA	4a	1a	0
21	75M	2004	bone 2 ^o	H	IVC	3	0	1
22	71F	2001	nodule	Pf	III	3	1a	0
23	60F	2000	cervical nodes	P	IVB	4b	1a	0
24	68M	2006	unilat. goitre	P	IVA	4a	1a	0
25	59M	2002	unilat. goitre ¥	H	III	4a	0	0

Key:

F: female

M: male

Unilat: unilateral

2^o: secondaries.

◊ ¹³¹I therapy 30y previously for contralateral toxic nodule

¥ lithium therapy

Histology:

P: papillary

Pf: papillary – follicular variant

F: follicular

H: Hürthle

Staging of primary tumours using TNM classification
(American Joint Committee on Cancer 2002)

The presenting features of thyroid malignancy are shown in Table 1. Unilateral goitre or recent nodule was the most common presentation, with five patients complaining of respiratory obstructive symptoms (dyspnoea in Table 1), and three patients presented with cervical lymphadenopathy.

Significant goitre or nodule was not initially noted in three patients with advanced disease—Patient 7 presenting with stridor due to intratracheal tumour, Patient 8 with unilateral pleural effusion, and Patient 21 with back pain due to spinal metastases. Diagnosis of thyroid malignancy in these three patients followed biopsy of intratracheal tumour, mediastinal lymph nodes, and bone respectively.

Staging (see Table 1)—The primary tumours were staged using the revised TNM classification (American Joint Committee on Cancer 2002). Primary tumours 2cm or less are now classified as T1, and T4 tumours extending beyond the thyroid capsule are now divided into T4a, and T4b when tumour invades prevertebral fascia or encases the carotid artery or mediastinal vessels. Nodal staging has been revised with '1b' for bilateral, contralateral or mediastinal nodes.

Most patients presented with advanced disease - 15 Stage IV, nine Stage III, and one Stage II. The majority of primary tumours were large (> 4cm in diameter) and 11 were locally invasive. Only one patient (Patient 8 with papillary cancer) had a small T1 tumour (1.3cm), and presented with a pleural effusion. Distant metastases were present at diagnosis in seven patients - lung in five, bone in four, and liver in one (three of seven patients had multiple sites of metastasis).

Surgery—Surgical removal of the primary thyroid tumour was attempted in 24 of the 25 patients. One patient (Patient 13) had no thyroid operation—she had a long-standing multinodular goitre and later presented with a large vascular metastasis in the sternum and had a bone biopsy only. The initial thyroid surgery was performed in smaller peripheral hospitals in five patients who were subsequently referred for radiation treatment. Nineteen different surgeons performed the initial thyroid operations with only four having extensive experience in thyroid surgery—three general-endocrine surgeons and one ENT surgeon.

Recent patients have been treated by total thyroidectomy, and two patients had modified cervical node dissections. The patient with intratracheal invasion had tracheal resection and anastomosis.⁹ Eight of the earlier patients were treated by lobectomy only, and three patients with extrathyroidal invasive cancers had debulking thyroidectomies. Postoperative complications included permanent hypoparathyroidism in one patient, permanent recurrent nerve palsy in one patient, and one patient with locally invasive disease had a temporary oesophageal fistula.

Radiation therapy—18 patients received postoperative radioiodine therapy—11 had inpatient ablative ¹³¹I (100–152mCi, median dose 108), and seven received smaller outpatient ¹³¹I therapies (20–49mCi, median dose 30). Seven patients received no postoperative ¹³¹I.

Postoperative external beam radiation therapy was administered to three patients with locally invasive disease—Patient 19 received a radical treatment (60Gy), and two patients received palliative radiotherapy following debulking thyroidectomy.

In subsequent years 10 patients received ablative ¹³¹I (100–154 mCi) for recurrent disease—three for local recurrence and seven for distant metastases. Progression of recurrent disease was probably delayed in five of these 10 patients.

Palliative external radiotherapy was administered to eight patients for local neck recurrence, and eight patients received radiotherapy to bone metastases for pain relief.

Outcome (see Table 2)—Most patients remained clinically well for some years after initial treatment, but two Stage IV patients (both with Hürthle cell cancers) died within 4 months of diagnosis. Recurrent disease occurred relatively early (< 4years) in nine patients, but occurred much later in a proportion of patients—in four patients

local neck recurrence occurred after 7 years, and in two patients distant metastases occurred after 10 and 14 years, respectively.

The most frequent site of distant metastasis was the lung, with pleural effusions usually a late preterminal feature (three patients). Bone metastases could be large, and were hypervascular (pulsatile) in three patients. The median survival from diagnosis of distal metastases was 2.3 years (0.3–7.3 years).

Thyroglobulin data was incomplete for earlier patients, and elevated levels were most helpful in the detection of recurrent disease in one patient with solitary bone metastasis, and in one patient with neck recurrence. However thyroglobulin levels were not elevated despite extensive metastases in six patients with high antithyroglobulin antibody titres.

Table 2. Clinical outcome and mode of death.

Patient	RECURRENCE		Years Post Diagnosis	Mode of Death	DEATHS	
	Local	Mets				Years Post Diagnosis
1	+		10	carcinomatosis, paraplegia		11
2				carcinomatosis, paraplegia		1
3		L,B	14	carcinomatosis,		19
4				carcinomatosis, paraplegia		4.5
5				carcinomatosis		5.5
6	+		1	local disease		6
7	+		7	intratracheal bleed		10
8				carcinomatosis		1.5
9		B,L	3.8	lung 2 ^o , effusions		8
10				carcinomatosis		2
11	+		7.8	local disease		22
12	+		1	lung 2 ^o , & emboli		2
13				carcinomatosis		2.3
14				carcinomatosis – lung		3
15		B	10	carcinomatosis		12
16	+		7.5	local disease		8
17		L	3	carcinomatosis & local disease		10.3
18				local disease		0.2
19	+		2.7	local disease/cranial nerve palsies		6.7
20		L	3	carcinomatosis – lung		4
21				carcinomatosis		0.33
22		L	2	carcinomatosis - brain & local disease		3
23				carcinomatosis & local disease		6.9
24		L	1	carcinomatosis - brain		1.8
25		L	2.7	local disease & carcinomatosis		7

L: lung metastases; B: bone metastases; 2^o: secondaries

The median survival from initial diagnosis was 5.5 years (0.2–22 y) with the majority of patients dying of metastatic disease, with major weight loss being a late manifestation. However seven patients died of local disease—with airways obstruction and dysphagia; and Patient 7 who had presented initially with an

intratracheal tumour died 10 years later of massive intratracheal haemorrhage. During the terminal illness, four patients required tracheostomy.

Discussion

This retrospective study of 25 patients who died of thyroid cancer of follicular cell origin summarises a 25 year regional experience. It approximates to one DTC death per year and can be contrasted with the average 16 newly diagnosed DTC patients seen by our service in the late 1990s.¹⁰

Earlier published series of patients dying from DTC have also been accrued over several decades. It is important to note that the earliest patients in our series were initially treated back in 1967 and 1973, and diagnostic testing and treatment have advanced over the last 40 years. Our 25 patients were all Caucasian and this South Island experience may differ from that experienced in northern New Zealand centres with much larger Māori/Pacific Island, and Asian populations.

The female to male ratio of patients dying of DTC was 2.1:1 and this is similar to the ratio for our newly diagnosed patients with differentiated thyroid cancer.¹⁰ Forty percent (¹⁰/₂₅) of our patients had long-standing goitres, and one was receiving a goitrogen (lithium) - delayed diagnosis is more likely in these patients. Many of our cohort were elderly when first diagnosed (median age 65 years), and the oldest patients would not have received iodised salt during childhood.

During the 25-year study period no patient under 45 years of age at diagnosis died of thyroid cancer of follicular cell origin. (The only young patient dying of thyroid cancer during the study period was a 36 year old female with Stage IV medullary cancer - of C-cell origin.) Our experience is similar to published reports of DTC deaths which show that most deaths occur in patients > 45y at the time of diagnosis.⁴⁻⁸ The reasons for the very low mortality rate in young DTC patients remains unexplained.

At initial presentation all but one of our cohort had advanced DTC (Stage IV or III). Most of the primary tumours were large (>4cm), and 11 were locally invasive—this extrathyroidal invasion may result in incomplete surgical excision leaving macro or microscopic disease. Twenty-eight percent (⁷/₂₅) of patients had distant metastases with metastases causing the presenting symptoms in four patients.

In other malignancies the presence of distant metastases at initial diagnosis has a very bad prognosis but DTC metastases may prove to be indolent (especially in lung) but multiple organ involvement has a bad prognosis.¹¹ Although lung is the most common site of DTC metastasis, pleural effusions are uncommon but may occur in the terminal illness.¹² However one of our cohort with a small primary tumour, presented with a pleural effusion which is a rare presentation for a papillary cancer.

The optimal initial treatment of thyroid cancer remains the subject of debate but United Kingdom and American consensus guidelines for the management of thyroid cancer have been published.^{13,14} A multidisciplinary approach is recommended with patients with large tumours treated by specialist surgeons to minimise surgical morbidity. When possible, large primary tumours should be excised to minimise persistent or recurrent local disease with total thyroidectomy to facilitate postoperative radioiodine ablation therapy.¹⁵

By today's standards many of our earlier patients would have benefited from more radical initial surgery and high-dose radioiodine therapy. External beam radiation has a minor role in the initial management of thyroid cancer except for palliation of unresectable disease, and for patients with extrathyroidal invasion and high likelihood of residual microscopic tumour.¹⁶ Following surgery and radiation treatment high risk patients need 'thyroxine suppression therapy' to suppress thyrotrophin (TSH) below 0.1mU/L to reduce the risk of recurrent disease.

Papillary thyroid cancer, which is by far the most common type of DTC, was responsible for 56% (¹⁴/₂₅) of our DTC deaths. Hürthle cell (oncocyctic) cancer caused 20% (⁵/₂₅) of the deaths, which is a much higher proportion than is found in newly diagnosed DTC patients. In earlier reports Hürthle cell cancers were included with the much commoner follicular cancers, but Hürthle cell cancers are probably best classified separately as they behave more aggressively and are less likely to concentrate radioiodine.^{17, 18}

During follow up several of our high-risk patients had early recurrent cancer but recurrence occurred later in some patients—including two patients with distant metastases diagnosed after more than 10 years. Much later (>20y) distant metastases have been reported in patients with papillary thyroid cancer.¹⁹ The incidence of second primary tumours in thyroid cancer patients is increased, however none of our cohort developed new malignancies during follow up (one patient had a large meningioma diagnosed 18 years post thyroidectomy).²⁰

Surgical re-exploration is the treatment of choice for recurrent disease in the thyroid bed or cervical nodes. Distant metastases are usually not amenable to surgery and should be treated with ¹³¹I therapy. However recurrent disease may prove to be non-avid for ¹³¹I because of de-differentiation of the tumour.

High-dose external beam radiation therapy may control soft tissue deposits and palliate bone pain. Hypervascular bone metastases should be considered for embolisation.²¹ Palliative chemotherapy with cytotoxics such as doxorubicin has proved disappointing, but several new therapeutic agents such as tyrosine kinase oncogene inhibitors are being evaluated in drug trials.²²

This community based study has shown that differentiated thyroid cancer deaths are uncommon and occur in older patients that presented with advanced disease. Our experience is similar to published reports of DTC deaths.⁴⁻⁸ The reason for the very low mortality rate in young DTC patients remains unexplained. Hürthle cell cancers were over-represented (20%) in our series with fatal thyroid cancer.

In recent years ultrasensitive TSH assays to monitor thyroxine suppression therapy, and improved thyroglobulin assays for the early detection of recurrent disease may improve DTC prognosis. In future it is hoped that earlier diagnosis and more radical initial surgery and radiation treatment of thyroid cancer will reduce the number of patients dying of thyroid cancer.

Competing interests: None known.

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The demographics and prevalence of youth (15–24 year olds) with type 1 diabetes in the Canterbury District Health Board catchment area in 2010: has the prevalence changed since 2003?

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Abstract

Aim The aim of the present study was to provide up-to-date descriptive information in relation to youth (15–24 years) with type 1 diabetes, residing within the Canterbury District Health Board (CDHB) catchment area. This included calculating the prevalence of type 1 diabetes in youth, and investigating whether there was an increase in the prevalence since a previous study reporting the prevalence of type 1 diabetes in youth in the CDHB in 2003.

Methods Data were collected from multiple clinical and research sources. Descriptive information and demographic characteristics, including age, gender, ethnicity, deprivation level, and diabetes duration were gathered. The prevalence, stratified by age and ethnicity, was calculated using the 2006 population census data.

Results There were 248 people with type 1 diabetes aged between 15 and 24 years residing within the CDHB area at the time of present study, giving a prevalence of 426 per 100,000 European youth with type 1 diabetes. The prevalence is found to have increased by 45 per 100,000 (12%) since 2003, but was statistically insignificant.

Conclusions There was no statistically significant increase in the prevalence of type 1 diabetes in youth in the CDHB catchment area between 2003 and 2010. However, the absolute figures of adolescents and young adults with type 1 diabetes have increased, which implies an increased demand on health care associated with diabetes compared to 7 years ago.

Type 1 diabetes is the most common endocrine disorder in children and adolescents.¹ Type 1 diabetes in youth may have profound long-term complications, if poorly managed. Long-term complications may contribute to life-lasting disabilities and chronic diseases, such as blindness, kidney failure, nerve damage, circulatory problems, heart disease, and early death.^{2,3}

The onset of long-term complications can be prevented or delayed if good diabetes management is achieved,⁴ thereby reducing the costs for the individual and their family, and to the healthcare system.

The incidence and prevalence of type 1 diabetes is increasing nationally and internationally.^{5–7} For example, over a 30-year period in the Canterbury geographical region, the incidence of type 1 diabetes in children and adolescents (0–19 years) was found to have significantly increased with time.⁶

The incidence rate of type 1 diabetes over time was significant in children (0–14 year olds).⁶ Whereas there were insignificant changes in the rate of new presentations of type 1 diabetes with time in adolescents (15–19 year olds).⁶ The last study of the prevalence of type 1 diabetes in youth (0–24 year olds) in the Canterbury District Health Board (CDHB) indicated a prevalence of 227 per 100,000 population.⁸ The present study provides up-to-date data on the number and prevalence of youth with type 1 diabetes. The aims of this paper were to

- Describe the demographics of youth (15–24 year olds) with type 1 diabetes in the CDHB catchment area and to compare this data with previous research.⁸
- Calculate the prevalence of youth with type 1 diabetes in the CDHB catchment area.
- Investigate whether there has been an increase in the prevalence of youth with type 1 diabetes residing within the CDHB catchment area.

Methods

The study comprised two main phases – data collection and data analysis. The project involved collating information for youth with type 1 diabetes aged between 15 and 24 years, residing within the CDHB catchment area. This included Christchurch, the town of Ashburton, and Northern rural Canterbury. South Canterbury District Health Board (SCDHB) entries were excluded from the study.

The number of youth residing in the identified catchment area based on an anchor date of 1 November 2010 was calculated. This date was aligned with the previous study's anchor date,⁸ yielding a 7-year gap between the two studies.

The search criteria (i.e. valid entries) used in the previous study was followed.⁸ Thus, those with newly diagnosed and patients with secondary or type 2 diabetes were excluded from the study.

CDHB records were searched in multiple electronic and physical sources. The electronic data sources included the CDHB inpatient and outpatients lists; the Diabetes Youth Canterbury database; and records from the previous study.⁸

The data collected comprised descriptive information and demographic characteristics of the youth. This included gender, ethnicity, age, residential area deprivation level,⁹ and diabetes duration.

Missing data-point entries were updated by searching the physical files held at the CDHB Diabetes Centre based on the National Health Index (NHI), and contacting the individual youth's General Practitioner (GP).

The collated data was entered in raw format using Microsoft Excel 2007 and IBM Statistical Package for Social Sciences (IBM SPSS 19). These software packages were used in the first (data collection) and second (data analysis) phases of the study. Age data was divided into two age bands (15–19 years and 20–24 years), representing adolescents and young adult developmental stages.

In the second phase, the prevalence of youth with type 1 diabetes was calculated, and then this was compared to the results of the last audit's study that was conducted in the CDHB in 2003.⁸ In the present research, the prevalence calculation was based on the 2006 census data, whereas the 2003 prevalence calculation was based on the 2001 census data.¹⁰

Prior to comparing the two results, the studies populations were checked for adequate comparability. The checking for this revealed two potential points of difference. The first potential point of difference point was related to maintaining the unity of the source of the obtained census lists. It was established that both of the census population figures were drawn from the same source of either Total Responses or Prioritised lists.¹¹

The second point was related to the different data collection and entry system of the 2001 and the 2006 census. For example, the 'New Zealander' entry was grouped with the European entry in the 2001 census data; however, in 2006 a separate classification under 'other ethnicities' was created to sum the New Zealander entries.¹¹ To overcome this issue, the total number of the New Zealander subcategory

in the 2006 census data was added to total number of the European category, thus making the 2001 and 2006 census population totals comparable to each other.

After ensuring the populations comparability, the 2010 and 2003 prevalences were compared. This process involved calculating the difference between the prevalences, and the 95% confidence interval (CI) of this difference.^{12–14} This difference was then statistically tested for significance.^{12–14}

Results

The number of youth with type 1 diabetes residing within the CDHB catchment area on the anchor date 1 November 2010 was 248. The demographics of these youth are depicted in Table 1.

Table 1. Demographics of youth with type 1 diabetes, who are residing within the CDHB catchment area in 2010

Variables		Count	N%
Gender	Female	120	48.4%
	Male	128	51.6%
Age groups* (years)	(15–19)	131	52.8%
	(20–24)	117	47.2%
Ethnicity	European	227	91.5%
	Maori	9	3.6%
	Pacific Peoples	3	1.2%
	Other	9	3.6%
Diabetes duration** (years)	between 1 and 5	55	22.2%
	between 6 and 10	82	33.1%
	between 11 and 15	68	27.4%
	between 16 and 20	33	13.3%
	more than 20	10	4.0%
Deprivation quintiles (1 = least deprived areas)	1	77	31.2%
	2	55	22.3%
	3	55	22.3%
	4	35	14.2%
	5	25	10.1%

* Mean age was 19.2 years old (standard deviation (std. dev.) =2.6 years)

** Mean length of diagnosis was 10 years (std. dev. =5.4 years; minimum =0.9 year (11 months); maximum=22 years). The prevalence calculation only included entries of established diabetes. This included diabetes duration that is around 1 year or more.

Based on the total number of youth with type 1 diabetes residing within the CDHB catchment area in 2010, the prevalence was calculated using the 2006 census data.¹⁰ The calculation was stratified according to ethnicity. Additionally, the European ethnic group had sufficient numbers to allow for the stratifying of data into two age bands (15–19 years and 20–24 years), which correspond to the census age categories. The results are shown in Table 2.

Table 2. Number and prevalence of European New Zealanders with type 1 diabetes (stratified by age) residing in the Canterbury District Health Board Catchment Area in 2010 and 2003

Anchor date	(15–19 years old)	(20–24 years old)	Total
1 November 2010			
Number	126	101	227
Prevalence per 100,000	443	406	426
(95%CI X to Y)	(372 to 527)	(334 to 493)	(374 to 484)
Total population ¹⁰	28,452	24,891	53,343
Anchor date	(15–19 years old)	(20–24 years old)	Total
1 November 2003			
Number ⁸	92	95	187
Prevalence per 100,000	369	394	381
(95%CI X-Y)	(301 to 452)	(322 to 481)	(330 to 440)
Total population ¹⁵	24,951	24,126	49,077

The total prevalence from the present study was compared with the prevalence obtained by the previous study (Table 2),⁸ after ensuring the comparability of data as previously described. The comparison yielded an increase of 45 per 100,000 (12%) population. However, the 95% confidence interval for this prevalence difference ranged from -33 to +122 indicating that this increase was not statistically significant.

From 2003 to 2010 there was a prevalence increase of 74 per 100,000 (20%) in adolescents (15 to 19 year olds), and 12 per 100,000 (3%) in young adults (20 to 24 year olds) with type 1 diabetes. However, the prevalence increase was statistically insignificant in each age band, from 2003 to 2010, according to the confidence interval of the difference: (95%CI -35 to 182) and (95%CI -101 to 125) per 100,000 adolescents and young adults, respectively.

Comparing the total populations in Table 2 suggests that the adolescents' age band had an increase of 14% compared to a 3% increase in the young adults' total population. Additionally, the number of adolescents (15–19 year olds) with type 1 diabetes exceeded the number of young adults (20–24 year olds) with type 1 diabetes by 25% in the present study. This is in contrast to the previous study,⁸ where the young adults' population was slightly (3%) larger.

Discussion

Type 1 diabetes is a chronic illness that is associated with multiple challenges. In addition to the patient's diabetes-management challenges, there are challenges for the health system. These include the planning and accessibility of resources, such as medication, equipment, and access to appropriately trained healthcare professionals. Moreover, additional health costs stem from long-term complications associated with poorly-controlled type 1 diabetes, such as kidney failure and nerve damage.

The burden on the health system is greater if these long-term complications arise at an early age. Thus, it is important for health care planning to gather data on the demographics, prevalence and trends of diabetes in specific populations, such as the youth population.

The present study aimed to gather this data for youth (15–24 year olds) with type 1 diabetes residing within the CDHB catchment area. The results indicate that the majority of CDHB youth who have type 1 diabetes were European New Zealanders, with males slightly outnumbering females.

These results are similar to the previous study.⁸ However, in the present study, the number of adolescents (15–19 year olds) with type 1 diabetes was greater (by 25%) than the number of young adults (20–24 year olds) with type 1 diabetes. Whereas, in the previous study,⁸ the difference was 3% in favour of the young adults population.⁸

This difference may be explained by a 14% increase in the total population of the adolescents in the CDHB catchment areas according to the 2006 census data, compared to only a 3% increase in the young adults' total population.

The total prevalence of CDHB youth with type 1 diabetes was increased by 45 per 100,000 (12%) from the previous audit,⁸ but was not statistically significant. This result is similar to the results of a previous study for the age range of 15 to 19 years old.⁶ The prevalence in the present study was greater in each age band; however, this was not statistically significant.

The prevalence increase in adolescents was greater than that of young adults. Again, this increase could be expected given the increase in the overall adolescent population from the 2001 to 2006.^{10,15} That is, according to the 2006 census there was a greater number of adolescents residing in the CDHB catchment area overall, when compared to the 2001 census, which in turn is likely to result in a greater number of adolescents with type 1 diabetes.

The results also indicated that a high percentage of youth with type 1 diabetes in the CDHB catchment area resided in the least deprived areas (53.5% in deprivation quintiles 1 and 2). This result may simply be representative of the wider Canterbury population. That is, youth of European descent in Canterbury (which includes most of the cases of youth with type 1 diabetes in this region) may be more likely in general to reside in least deprived areas.

Without further indepth analysis to calculate the estimated prevalence of the CDHB youth residing in the different deprivation quintiles, and linking of residential mesh blocks to the different deprivation levels specific to the CDHB area and the youth population in the target age range (which were not possible in the current study), it is not possible to determine if this is a statistically significant result. It is, therefore recommended that future research explores this finding in more depth.

It should be noted that the present study may not have identified all youth with type 1 diabetes within the CDHB catchment area. That is, there may be missing entries, which could have been checked using additional capture-recapture methods. Capture-recapture was applied in the present study using the previous study records.⁸

Another limitation to the study is the search date that was used to download records. This was set to about 1 year prior to the anchor date, and included the month of December 2010. It is expected that youth with diabetes get checked at least once a year. However, records may be missed if they have not attended their annual check in 2010. More entries may have been captured, if the search period was extended to, for example, 2 years from the anchor date.

Although the aforementioned limitations may have resulted in missing entries, the number of missing entries is believed to have been minimised because multiple sources were checked for youth entries, including the inpatient and outpatient hospital discharges and youth databases.

Additionally, the composition of demographic factors for youth with type 1 diabetes in the present study (e.g. the 91.5% Europeans, and 48.4% female and 51.6% males), were similar to the total CDHB youth population according to the 2006 census. For instance, according to the 2006 census records, the total population of European youth in the CDHB was 83% and the percentage of males was higher than females.¹⁰ Hence, this gives confidence that the results of the present study are an accurate representation of youth with type 1 diabetes in the CDHB catchment area.

To conclude, the absolute figures obtained in the present study suggest an increased demand on health care resources associated with youth with type 1 diabetes compared to 7 years ago.

It is recommended that the results from the present study be used to inform planning and decision-making related to diabetes health services both in the short and longer term. For example, if youth receive and engage in adequate health services in the short term, this may delay or prevent the onset of long-term complications, and therefore reduce future health care costs.

Competing interests: None.

Ethical approval: The study was reviewed and approved by Te Komiti Whakarite (CDHB Research Consultation with Maori). Additionally, the study was reviewed and approved by the University of Canterbury Human Ethics Committee (HEC 2010/183).

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Biosocial profile of New Zealand prosthetic eye wearers

Keith R Pine, Brian Sloan, Robert J Jacobs

Abstract

Aim To describe the biosocial profile of New Zealand (NZ) artificial eye wearers and establish a basis for future research and international comparison.

Methods This retrospective study surveyed 431 NZ artificial eye wearers to investigate their ethnicity, gender, age, causes of eye loss, age of current prosthesis, ocular prosthetic maintenance regimes and the extent and severity of discharge associated with prosthesis wear.

Results Approximately 3000 people wear artificial eyes in NZ. Accidents were the main cause of eye loss prior to 1990 and medical conditions have been the main cause since. In the 1960s, the ratio of men to women losing an eye from accidents was 5:1, but during the past decade the ratio was 1.4:1. Socket discharge occurred at least twice daily for one-third of the study group.

Conclusions Approximately 1 in 1440 people wear artificial eyes in NZ. Decline of eye loss due to accidents is consistent with decreasing workplace and traffic accidents and may be due to improved medical management, workplace safety standards and safer roads. Mucoïd discharge is prevalent in the anophthalmic population of NZ and an evidence based treatment protocol for discharge associated with prosthesis wear is needed. Research into this distressing condition is planned.

The prosthetic eye literature has a limited number of published studies describing artificial eye wear over time,¹⁻³ however, with the exception of a study carried out in Dallas, Texas from 1973 to 1977 and repeated in 1990 to 1994,⁴ no information about the epidemiology of eye loss appears to be available. Furthermore, mucoïd discharge is wearers' second highest concern after health of the remaining eye,⁵ but the incidence and severity of this problem in the anophthalmic population is unknown.

This retrospective study is designed to address this lack of information about prosthetic eye wear in New Zealand and to establish a basis for future artificial eye research and international comparison. The study investigated artificial eye wearers' ethnicity, gender, age, causes of eye loss, age of current prosthesis, ocular prosthetic maintenance regimes and the extent and severity of discharge associated with artificial eye wear.

Methods

Background—The New Zealand Artificial Eye Service is the only provider of artificial eyes that offers a local service in Northland, a region which has a mixed rural/urban population, roughly representative of New Zealand's overall population. The estimate of the total size of the anophthalmic population of New Zealand was calculated by extrapolating the number of Northland domiciled patients on the New Zealand Artificial Eye Service database to the estimated residential population of New Zealand.⁶

The participants were then asked whether they felt that having their artificial eyes professionally re-polished improved discharge and if so, how long the improvement lasted. A further section contained an open invitation and space to comment on prosthetic eye wearing experience.

Results

The 109 patients on the Northland database of the New Zealand Artificial Eye Service made up 0.07% of the 157,300 population of the Northland Regional Council.⁶ This percentage, extrapolated to the total population of New Zealand in 2010 (4,367,700)⁷ resulted in an estimated total of 3026 anophthalmic people.

A total of 431 artificial eye wearers (31% of 1373) returned the completed study questionnaire. An analysis of these returns by regional institution is shown in Table 1.

Table 1. Returns of the questionnaire by regional institution

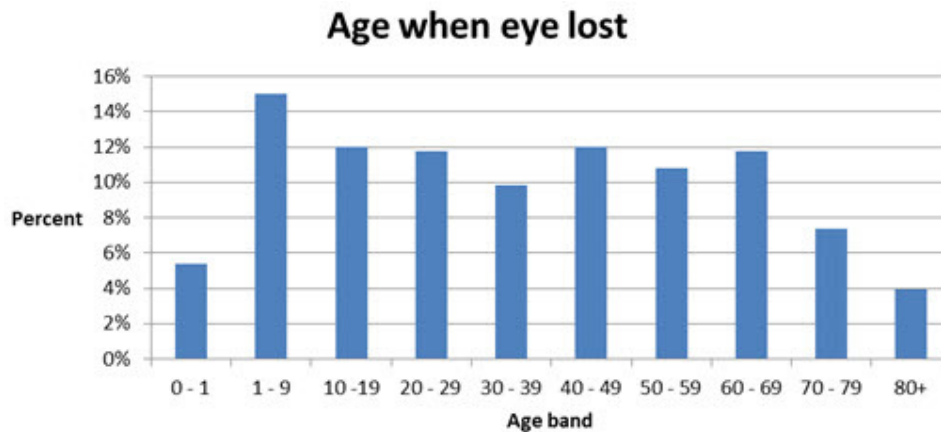
Institution	Questionnaires posted	Returned	Percentage (%) returned
Capital & Coast District Health Board	50	25	50
Lakes District Health Board	53	15	28
Waikato District Health Board	90	20	22
Auckland District Health Board	380	96	25
Royal NZ Foundation of the Blind	Canvassed online	19	unknown
Accident Compensation Corporation	280	83	30
NZ Artificial Eye Service	420	146	35
Canterbury District Health Board	100	27	27
Totals	1373	431	31

Ethnicity—A comparison between ethnicities in the study population and the New Zealand population⁶ was made. Europeans were the only ethnicity to be over represented (79% of the study population compared with 70% of the NZ population). Māori people made up 13% of the study population compared with 14% of the NZ population. Pacific peoples (4% study, 7% NZ), Asian peoples (3% study, 9% NZ) and others (0% study, 1% NZ).

Gender—Of the 334 participants who provided personal details, 41% were women and 59% were men.

Age—Participants were represented across all ten age bands chosen for the study (Figure 2). Eighty-two percent (82%) were 40 years of age or over.

Figure 2. Eye loss as a function of age (using 10 age bands)

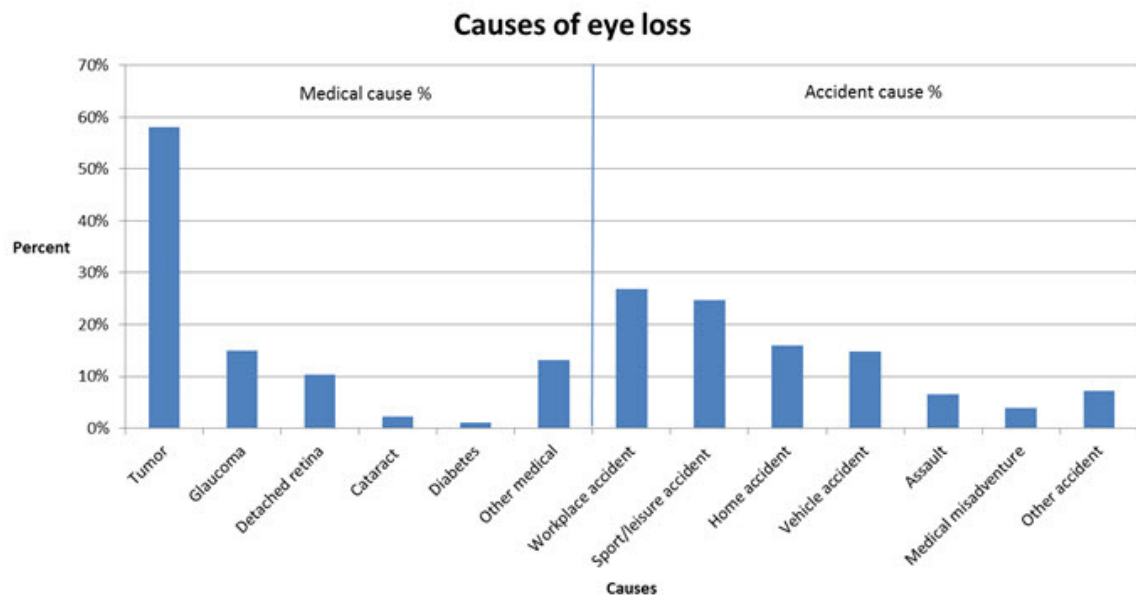


Eye loss—The reported causes of eye loss were: accident (50%), medical (43%) and congenital (7%).

Eye loss due to tumours of various kinds was the most prevalent medical cause followed by glaucoma, detached retina, cataract and then diabetes.

Workplace accidents were the most common type of accident followed by sporting/leisure accidents, home, motor vehicle, assault and lastly medical misadventure (Figure 3).

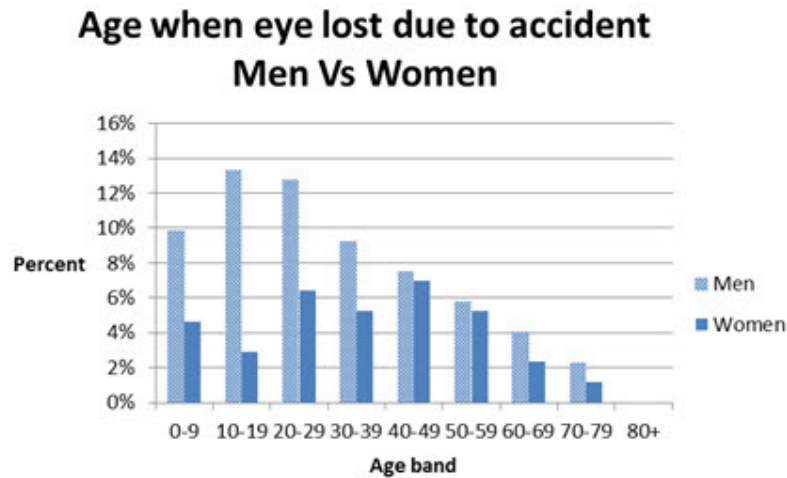
Figure 3. Main causes of eye loss (medical and accidental causes are shown separately)



The highest proportion of eyes lost from any cause occurred between ages one and nine years inclusive (15% of all eyes lost). Between 10 and 69 years eye loss was evenly distributed over the decades (varied between 10% and 12% each decade).

Eye loss due to accident as a function of gender is shown in (Figure 4). For ages less than 40 years eye loss due to accident was significantly greater in men than women ($P=0.002$) but women and men over 40 lost eyes to accidents in similar numbers.

Figure 4. Gender mix of eye loss due to accident



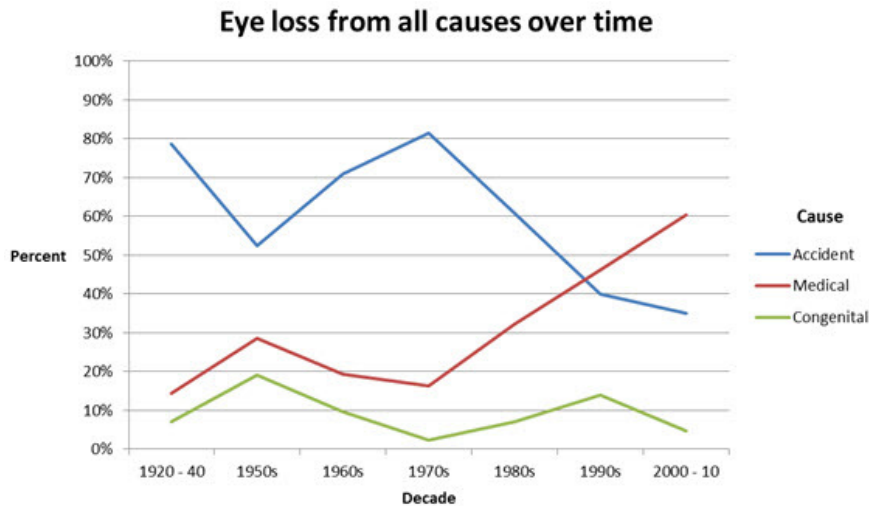
Changing causes of eye loss—Accidents were the main cause of eye loss before the 1990s but since then medical causes of eye loss have predominated (Figure 5).

Table 2 illustrates how the ratio of men to women whose eye loss was due to accident has varied over time.

Table 2. Ratios of men to women whose eye loss was due to accident from 1960 to 2010

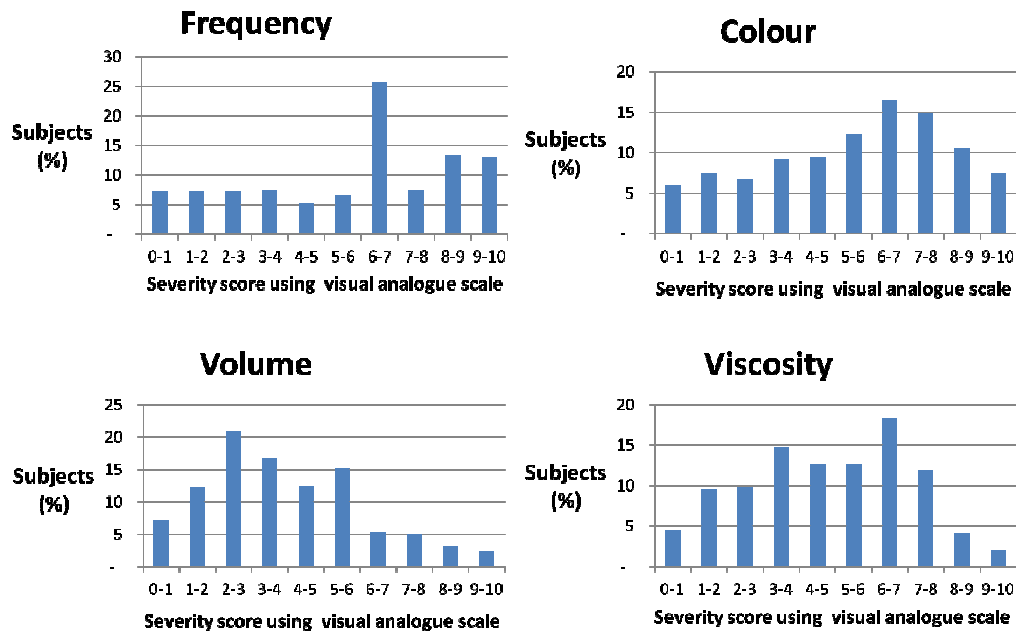
Decade	1960–69	1970–79	1980–89	1990–99	2000–2010
Ratio: men to women	5 : 1	2.3 : 1	1.8 : 1	2.4 : 1	1.4 : 1

Figure 5. Cause of eye loss in New Zealand over time



Discharge associated with artificial eye wear—The average severity score (from 0–10 on the visual analogue scale) for discharge frequency was 5.55 (SD 2.8), discharge colour 5.33 (SD 2.6), discharge volume 3.68 (SD 2.19) and discharge viscosity 4.59 (SD 2.28) (Figure 6).

Figure 6. Self measured visual analogue scale scores for severity of 4 discharge characteristics



Thirty-three percent of the study population marked a VAS score of ≥ 7 for frequency (discharge occurred at least twice daily) and 59.3% of the random comments about discharge were made by participants who scored ≥ 7.0 for discharge frequency. Ten percent of the study population marked a VAS score of ≥ 7 for discharge volume (moderately profuse), 32% for colour (creamy yellow) and 18% for viscosity (thick).

Age of current artificial eye—The majority of participants (64%) had worn their present prosthetic eye for four years or less, 21% for between 5 and 9 years, 8% between 10 and 19 years, and 8% for more than 20 years.

Frequency of professional re-polishing—51% of the participants had their artificial eyes re-polished every year, 9% more often than yearly and 40% less often.

Artificial eye removal and cleaning regimes—48% of people in the study population removed and cleaned their artificial eyes daily but 26% left their artificial eyes in place for more than a month. Twenty-six percent removed their prostheses overnight.

Hand washing behaviour—The majority of wearers (58%) always washed their hands before removing their artificial eye, 25% mostly washed their hands, 12% washed sometimes and 5% never washed their hands.

Removal difficulty—Eight percent (8%) of wearers had difficulty removing their artificial eye including 6% who needed this to be done by others.

Discussion

While the 431 artificial eye wearers recruited to this study represented 31% of the 1373 letters that were mailed out they probably made up 14% of the total anophthalmic population in New Zealand which is estimated by the authors to be approximately 3000 or 1 anophthalmic person for every 1443 in the general population.

Thirty-six percent (36%) of study participants lost their eye(s) within the past eleven years indicating that individuals were more likely to participate if their experience of eye loss was more recent. This bias may have increased the number of participants whose current artificial eye was under 11 years old relative to those who lost their natural eyes more than 11 years ago. However, it is unlikely to have affected the main conclusions of the study.

Europeans who might be more comfortable than other ethnic groups completing the English language questionnaire may have biased ethnicity representation and accounted for the finding that Europeans were more highly represented in the study population than in the general population.

Another limitation of this study is that different surgical techniques, socket and eyelid problems, or unsuitable prostheses were not investigated. Discharge may be more severe in the presence of these problems, but there is no reason to suspect that such problems were more or less prevalent in our study population than in the general anophthalmic population. Future studies are planned to try to elucidate some of the mechanisms of increased socket discharge.

The literature on the characteristics of anophthalmic populations is sparse but some information can be found on related topics. For example, Chang et al⁸ describe

aetiologies and clinical characteristics of corneal opacities leading patients to seek cosmetic treatments at the Department of Ophthalmology at Seoul National University Hospital. They examined 401 patients with corneal opacities and report characteristics of age and gender that were similar to the anophthalmic population in this study.

A notable exception was the considerably younger age when injury occurred in the Korean study. The Eye Injury Snapshot Data Summary, 2004–2008 from the USA.⁹ also contained characteristics of age, gender and accident type that were reflected in this study although the ratios of accidents resulting in eye injury and eye loss are quite different. In particular, the most common place to injure an eye was in the home (44.1%) but relatively few eyes (16%) were actually lost through home injuries.

The causes and gender mix reported in a study of eye loss carried out in Dallas County, USA from 1990 to 1994⁴ were broadly in line with this study except that the percentage of eye loss due to accident was higher (59.8% compared to 54%).

Comparisons with the literature are summarised in Table 3. While the studies are very diverse they suggest that gender mix (more young males) and causes of eye loss (more accidents) may be common to most present day anophthalmic populations.

Table 3. Comparisons of eye loss in New Zealand with related injuries in Korea and America

Characteristic	Corneal opacities in Korea	Eye injury in the USA	Eye loss in Dallas County 1990–1994	Eye loss in New Zealand
Cause	Trauma: 50.6% Medical: 43.9% Congenital: 5.5%		59.8% 33.3% 6.9%	54% 46% 8%
Gender (all causes)	Men: 60.7% Women: 39.3%		64.2% 35.8%	59% 41%
Gender (accident only)	Men Women	73% 27%		65% 35%
Age when accident occurred	0–15 yrs: 69.5% 15–55 yrs: 28.6%	0–18yrs: 25.4% 18–45yrs: 47.6% 46+ yrs: 26.9%		0–19 yrs: 31% 20–49 yrs: 49% 50+ yrs: 20%
Accident type	Home Sport/recreation Workplace Roads Other	44.1% 14.7% 15.6% 11.4% 14.2%	7.1% 15.8% (gunshot)	16% 25% 27% 15% 18%

Eye loss—Young men who lost their eyes because of accidents strongly altered the gender, age and cause of eye loss characteristics of the study population. This group was the reason that workplace and sporting/leisure accidents were the most prevalent. However, the dynamics of eye loss appear to be changing over time as eye loss due to accident has declined relative to medical causes and the gender mix of accident victims has changed with a decreasing ratio of men to women.

This study was not designed to uncover the reasons for these dynamics but the trend towards less accidents resulting in eye loss is consistent with the decrease of work

related head and neck injuries between 2003 and 2010 reported by the NZ Accident Compensation Corporation⁷ and the decrease of traffic injuries from a peak of 23,385 in 1973 to 14,541 in 2009.¹⁰ Improved medical management of eye injury is likely to also play a part in the reduction of eye loss from accident.

Discharge and artificial eye maintenance—The observation that 59.3% of the random comments referring to discharge were made by participants who scored ≥ 7.0 on the 1-10 range of the visual analogue scale for discharge frequency suggests that these people were more motivated to write a comment about discharge than those with less severe scores. This in turn suggests that severity scores of ≥ 7.0 for frequency (discharge occurred at least twice daily) are likely to impact on the quality of life of prosthetic eye wearers.

It is disturbing therefore that one-third of the study population reported severity scores of ≥ 7.0 for discharge frequency and as many as 9% experienced severity scores of ≥ 7.0 for both volume and frequency. The high incidence and severity of this problem occurred even though access to professional prosthetic eye services was good. Unfortunately, a standardised treatment protocol for discharge associated with artificial eye wear is lacking¹¹ and further research into the cause and treatment of this prevalent and distressing condition is needed.

Conclusions

This study has sought to address the lack of information about prosthetic eye wearers in New Zealand. We estimated that approximately 1 in every 1440 people wear artificial eyes in this country and that most of the anophthalmic population lost their eyes through accident. Men under 40 years were the most ‘at risk’ group. The gender mix and cause of eye loss appears to be changing over time.

Accidents were the main cause of eye loss in the decades prior to 1990 and medical conditions have been the main cause since. The decline of accidents resulting in eye loss is consistent with decreasing workplace and traffic accidents in the general population and may be due to improved workplace safety standards, safer roads and better medical management. An additional finding of this study was that in spite of good healthcare provision, mucoid discharge is prevalent in the anophthalmic population of New Zealand with 33% experiencing discharge at least twice a day.

Further research is needed to establish an evidence based standardised treatment protocol for discharge associated with artificial eye wear.

Competing interests: Keith Pine owns and operates a private practice in ocular prosthetics, the NZ Artificial Eye Service.

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Gout in women: differences in risk factors in young and older women

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Abstract

Aim To describe the clinical characteristics of female patients with gout, assess risk factors in this group and to identify any differences between pre- and postmenopausal women with this diagnosis.

Methods We retrospectively reviewed the case records of all women who were seen with gout in a secondary care setting (inpatient and outpatient) at Counties Manukau District Health Board between July 2007 and July 2008. Demographic data, risk factors for gout and information on urate-lowering therapy was collected. A cut-off of ≤ 50 years was used to estimate pre-menopausal status.

Results 122/509 (24%) of patients seen with gout were female. Fourteen female patients were ≤ 50 years of age; all of these patients were either Maori (43%) or of Pacific Island ethnicity (57%). Comorbidities in those ≤ 50 years old were renal impairment (78.6%), hypertension (64.3%), congestive heart failure (43%) and diabetes mellitus (42.9%). Comorbidities in women > 50 years old were similar: hypertension (77%), renal impairment (70%), dyslipidemia (53%) and diabetes mellitus (50%). Ischemic heart disease was more common in older women (43% vs 7%), $P < 0.01$. Mean body mass index (BMI) was significantly higher in the younger women (43.5 vs 33.1), $P < 0.01$. Half of all the female patients were on diuretics, and medication used to lower uric acid level was prescribed in 35.7% of women ≤ 50 years of age, and 42.59% of women > 50 years of age.

Conclusion Women who develop gout are more likely to be over the age of 50, have other comorbidities and be on diuretics. In comparison, younger women who develop gout have similar risk factors but tended to have a higher body mass index and are more likely to be of Māori or Pacific Island ethnicity.

Gout is a form of inflammatory arthritis caused by uric acid precipitation in and around joints. This painful condition was first identified in ancient times by Egyptians around 2640 BC. Hippocrates first wrote about this disorder in 400 BC.^{1,2} Prolonged hyperuricemia is the main risk factor for gout.

Hyperuricemia can be caused by either excessive intake of a purine rich diet or inadequate excretion of uric acid by the kidneys. Human beings lack the enzyme uricase which converts uric acid into water soluble allantoin which is more readily excreted.³ Genetic studies have identified association between polymorphism in the GLUT9 (SLC2A9) gene and URAT1 (SLC22CA12) gene to be key regulators and transporters of uric acid.

Genetic variations in these enzymes are identified as risk factors for hyperuricemia and gout.^{4,5} Other well recognised associations for hyperuricemia and gout include

age, diabetes mellitus, hypertension, metabolic syndrome, renal disease, cardiovascular diseases and medications like diuretics.

Gout is predominantly a disease of males, but in recent times, there has been an increase in prevalence in women. The reported male to female ratio is approximately 7:1 to 9:1 but in people over the age of 65 this ratio is reduced to 3:1.⁶⁻⁸ Gout is considered rare in premenopausal women as the estrogenic hormones have a mild uricosuric effect and therefore protective against hyperuricemia and gout. During menopause the estrogen levels drop and women with risk factors become more likely to develop hyperuricemia and gout.⁹ However, in our clinical practice we have noticed an increasing frequency of young women presenting with acute flares of gout.

The objective of this study was to describe the clinical characteristics of female patients with gout, assess risk factors for gout in this cohort and identify any differences between pre- and postmenopausal women with this diagnosis who present to secondary care in South Auckland.

Methods

We retrospectively reviewed the records of female patients with a diagnosis of gout using the International Classification of Diseases-9 (ICD-9 codes 274.0, 274.8, 274.9), who were seen at Counties Manukau District Health Board (CMDHB), South Auckland, New Zealand, between July 2007 and July 2008.

Demographic data, information on relevant comorbidities and diuretic use were collected. Comorbidities of interest were obesity, hypertension, congestive heart failure, dyslipidemia, diabetes mellitus and renal impairment. Obesity was defined as body mass index (BMI) $>30\text{kg/m}^2$; renal impairment was defined as estimated glomerular filtration rate (eGFR) of $<60\text{mls/min}$ using the Modification of Diet in Renal Disease (MDRD) formula.¹⁰

The menstrual status of women was not usually recorded in the hospital medical charts therefore we used an age cut-off of 50 years (average age of menopause in our population) as a surrogate marker for pre- and postmenopausal status.

We collected laboratory data on lipid levels, serum urate and glomerular filtration rate. The use of urate-lowering therapy was determined from the electronic medical records.

Statistical analysis—Statistical analysis was conducted using OpenEpi (version 2.3.1) software. Paired t-test (2 sided) was used to compare differences among means. Chi-squared was used to compare differences in proportions. One-way ANOVA used to compare differences in means when there were more than 2 samples. All reported p values less than 0.05 were considered statistically significant.

Results

Between 1 July 2007 and 31 July 2008, 509 patients presented to CMDHB for management of gout. This included inpatient as well as outpatient visits. Table 1 shows the gender and ethnic distribution of the study population. Among the 509 patients, 122 (24%) were female.

Fourteen female patients (11.5%) were either 50 years or younger and 108 (88.5%) were over 50 years of age. Table 2 shows a comparison of the ethnicity, the traditional risk factors and prescribed treatment between the two age groups.

Table1. Gender and ethnic distribution of patients with gout presenting to secondary care at Counties Manukau District Health Board in the period 1 July 2007 and 31 July 2008

Ethnicity	Total n (%)	Males n (%)	Females n (%)	M:F ratio
Māori	129 (25)	87 (22)	42(34)	2:1
Pacific people	237 (47)	190 (49)	47 (39)	4:1
Europeans	115 (23)	86 (22)	29 (24)	3:1
Others	28 (6)	24 (6)	4 (3)	6:1
Total n (%)	509 (100)	387 (100)	122 (100)	3:1

Table 2. Description of women with gout stratified by age

Characteristics	Age ≤50 years (n=14)	Age >50 (n=108)	P value
Mean age (range)	41 (23–50)	71.5 (51–95)	
Ethnicity			
Māori n (%)	6 (43)	36 (33.3)	0.67
Pacific n (%)	8 (57)	39 (36.1)	0.22
European n (%)	0 (0)	29 (26)	0.035
Others n (%)	0 (0)	4 (3.7)	1.00
Risk factors			
Mean BMI	43.5	33.1	0.002
Hypertension no (%)	9 (64.3)	83 (77)	0.24
Diabetes mellitus	6 (42.9)	54 (50)	0.41
Renal impairment	11 (78.6)	76 (70)	0.39
CHF	6 (43)	40 (37)	0.44
Dyslipidemia	5 (35.7)	60 (55)	0.13
Ischemic heart disease	1 (7.0)	46 (43)	0.01
Three or more comorbidities	9 (64)	70 (64.8)	0.97
SUA mean (range)	0.54 (0.4–0.8)	0.48 (0.22–0.92)	0.17
Treatment			
Allopurinol no (%)	3 (21.4)	45 (42)	0.12
Uricosuric agent	2 (14.3)	1 (1.0)	0.03

Table 3. Risk factors for the three major ethnic groups

Characteristics	European	Māori	Pacific people	P value
Numbers (%)	29 (24.58)	42 (35.59)	47 (39.83)	
Average age (range)	78.9 (52–95)	60.5 (23–85)	60.93 (27–83)	0.10
Mean BMI (range)	31.6 (20.6–40)	34.1 (24–51.2)	37.1 (21–77)	<0.001
Hypertension No. (%)	19 (65.5)	33 (78.6)	35 (74.5)	0.47
Diabetes mellitus No. (%)	7 (24.14)	21 (50)	23 (48.90)	0.06
Renal impairment No. (%)	18 (62)	27 (64.3)	39 (83)	0.07
CHF no (%)	11 (37.9)	18 (42.9)	17 (36.2)	0.80
IHD no (%)	16 (55)	17 (40.5)	13 (27.7)	0.06
Dyslipidemia No. (%)	20 (69)	19 (45.2)	25 (53.2)	0.14
Diuretic use No. (%)	16 (55)	23 (54.8)	20 (42.6)	0.42
Mean SUA (range)	0.45 (0.22–0.71)	0.51 (0.28–0.92)	0.50 (0.27–0.81)	0.42
Treatment				
Allopurinol No (%)	10 (34.5)	16 (38)	21 (44.7)	0.65
Uricosuric agent No (%)	0 (0)	0 (0)	3 (6.4)	0.10

In the 'pre-menopausal' age group (≤ 50 years), 43% were Māori and 57% were Pacific people. There was no European or people of other ethnic group represented in this age group. In the older age group (> 50 years), 26% of the population were Europeans, 33.3% were Māori, 36.1% were Pacific People and 4% were of other ethnic group (predominantly Asian).

Every patient in the 'pre-menopausal' age group had a comorbidity that predisposed them to gout. Renal impairment (78.6%) and hypertension (64.3%) were the two most common co morbidities in the ≤ 50 age group. Renal impairment was attributed to hypertensive disease in 57% and glomerulonephritis in 21%.

Congestive heart failure and diabetes mellitus were present in 43% of patients who were ≤ 50 years old. The underlying cause for congestive heart failure was either rheumatic valvular heart disease or non-ischemic cardiomyopathy.

Comorbidities seen in the older age group were hypertension (77%), renal impairment (70%), dyslipidemia (55%), diabetes mellitus (42.95%) and ischemic heart disease (43%). The presence of ischemic heart disease was more common in older women (43% vs 7%, $P<0.01$), whereas the mean BMI was significantly higher in the younger age group (BMI 43.5 vs. 33.1, $P<0.01$).

Diuretic use was similar between the age groups (approximately 50% in both groups). The mean serum uric acid was not statistically different between the 2 age groups. Medication to lower uric acid was prescribed in 5 (35.7%) of patients in the younger age group and 46 (42.59%) of patients in the older age group. Allopurinol was the drug of choice and only 3 (2.68%) received uricosuric agents.

When risk factors for different ethnic groups were compared, statistically significant difference was noted in the mean BMI. Obesity, defined as a BMI of greater than 30 was noted in 64 % of the less than 50 age group and 39% in the older age group, with the Pacific People having highest mean BMI (37.1) followed by Māori (34.1) and European (31.6). There was a suggestion that European women were older (mean age 78.9 years) compared to Māori and Pacific women (60.52 and 60.93 years respectively), but this did not reach statistical significance.

Hypertension and renal impairment were the two most common comorbidities in all three ethnicities. Diabetes mellitus occurred more commonly in Māori (50%) and Pacific people (48.9%) compared with European (24%), $P=0.06$. The mean serum uric acid (SUA) concentration and diuretic use was similar in all three ethnic groups. Therapy to lower uric acid levels was similar in all three ethnicities with no significant statistical difference noted.

Discussion

The Counties Manukau District Health Board (CMDHB) has an estimated population of 464,000 of which 46% are Europeans, 21% Pacific people, 17% Māori and 16% Asians. It has the third highest district health board (DHB) population in New Zealand and has the highest number of Māori and Pacific people. Gout is a common metabolic condition present in this DHB population (11).

This study shows that 24% of patients presenting with gout to secondary care in South Auckland are women, giving an overall male to female ratio of 3:1. The ratio for Māori was 2:1, European 3:1 and Pacific people 4:1. This compares to epidemiological survey conducted by Klemp and colleagues in 1996 which showed that the sex ratio of Māori men to women with gout was 5:1 and European men to women were 8:1.¹²

A potential reason for this discrepancy between our findings and Klemp could be that women are more likely to seek medical attention and attend appointments in secondary care, but alternatively it could mean that the prevalence of gout in women is rising.

Approximately 89% of women with gout in our study were over 50 years of age, confirming that this disease that mainly affects postmenopausal women, but a significant number of women under the age of 50 are now presenting with gout.

Māori and Pacific women have a higher prevalence of gout, similar to their male counterparts, suggesting genetic factors to be involved. Genetic variation in SLC2A9 is shown to increase the risk of gout by 500% in New Zealand Māori and Pacific Island Polynesians as shown in a study by Hollis-Moffatt et al.¹³

European women with gout in our study were on average eighteen years older than Māori and Pacific women. The main traditional risk factors identified for gout were obesity, hypertension, renal impairment and diuretic use similar to those identified in other studies.^{14,15}

All the women in the 'pre-menopausal' age group had at least one risk factor for gout and 64% had more than three comorbidities. Similar finding were also noted in women over 50 years of age. The main differences between the risk factors for gout between young and older women were increased body mass index (BMI) in the younger age group and Māori and Pacific Island ethnicity. The Nurses health study showed that women with BMI between 23 and 24.9 kg/m² had a 1.55 relative risk of gout.

The risk continued to increase with increasing BMI, so that RR was 2.86 for those with BMI of 25-29.9 kg/m², RR of 4.69 for BMI of 30 to 43.9 kg/m², and RR of 7.25 for BMI exceeding 35kg/m². The same study showed hypertension was associated with a RR of 2.26 and diuretic use had a RR of 2.63.¹⁴

Other important risk factors identified in the women population with gout were diabetes mellitus, congestive cardiac failure and to a lesser extent, ischemic heart disease. A study to determine the prevalence of gout in patients with diabetes mellitus showed a high prevalence (22%) in patients with Type 2 diabetes.¹⁶

Our study showed that 50% of Māori and Pacific, and 25% of European women with gout had diabetes mellitus. This study highlighted that treatment to lower uric acid was inadequately administered in women with only 40% receiving any form of therapy. This would explain why the mean serum uric acid was elevated in all the study groups.

There are limitations to this study. Firstly only patients who presented to secondary care were included. A large percentage of patients with gout are cared for by their

primary care physicians and it is likely that patients with difficult to treat gout or with multiple comorbidities were more likely to be referred to secondary care.

The prevalence of comorbidities in our study may not be the true reflection of our wider gout population. We only had a small number of patients in the young age group which makes it difficult to interpret the risk factors in this subgroup. Menstrual data was not available and therefore we used an age cut-off as a surrogate marker for menopause. Alcohol and diet history was not reliably recorded, hence the influence of these important risk factors were not included in the study.

In conclusion, women who develop gout are more likely to be over the age of 50 (i.e. postmenopausal), have one or more comorbidity and are more likely to use diuretics. In comparison, younger women who develop gout have similar risk factors but tended to have a higher body mass index and are more likely to be of Māori or Pacific Island ethnicity.

Women with gout seem to be under-treated despite being seen in secondary care. This study highlights important demographic feature of women with gout which can be used as a starting point for larger epidemiological studies and also provides valuable information which can be used in power calculations for future interventional studies to test preventative or therapeutic strategies.

Competing interests: None known.

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Bariatric surgery makes dramatic difference to health-related quality of life

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Abstract

Aim To explore the impact of bariatric surgery on health-related quality of life (HRQoL).

Methods An audit of patients referred for bariatric procedures. Patients completed Short Form-36 questionnaires at their first preoperative clinic and at their 6-month follow up appointment after surgery. SF-36 scores were compared with standard parametric tests.

Results 40 patients completed baseline SF-36 questionnaires and underwent bariatric surgery, 28 were surveyed again 6 months post-procedure during the audit period between December 2008 and December 2010. Twenty-three patients underwent laparoscopic Roux-en-Y gastric bypass and five underwent laparoscopic sleeve gastrectomy. The patients were predominantly New Zealand European, female, with a body mass index greater than 40 kg/m². Significant improvements in health-related quality of life were observed. The physical and mental component summary scores were initially well below the population norms, but increased to the norm 6 months after surgery.

Conclusion The HRQoL of morbidly obese patients significantly improves after bariatric surgery. Services including the SF-36 in their measurement armamentarium can demonstrate the Service's impact on patient-perceived outcomes in addition to clinically-focused outcomes.

Obesity is a major problem in New Zealand with the direct-care costs estimated at \$460 million.¹ Although the growth of obesity may be slowing, its prevalence remains high (26.5%) and 9.1% of the population are morbidly obese.² Obesity is associated with increased risk of mortality from cardiovascular disease and some cancers, a higher incidence of numerous conditions including diabetes, hypertension, joint disorders and obstructive sleep apnoea.³

Bariatric surgery is a safe and effective way of managing morbid obesity for selected individuals and a recommended approach in the New Zealand guidelines for weight management.^{3,4} Weight associated comorbidities frequently resolve after bariatric procedures.⁵

Bariatric surgery has been offered by the Auckland District Health Board (ADHB) since 2008. Mainly Roux-en-Y gastric bypass or sleeve gastrectomies are carried out. The effect of bariatric surgery on clinical outcomes has been investigated in New Zealand,⁶ but no estimate of the effect of the surgery on patient-oriented outcomes has been reported in New Zealand.

The ADHB Bariatric Service has been routinely collecting self-reported perceived health using the SF-36 and this data offered the opportunity to measure the impact of bariatric surgery on the patient's perception of their health-related quality of life (HRQoL).

Methods

Background—The ADHB Bariatric Service has selection criteria for bariatric surgery aligned with that proposed by the National Institute of Health.⁷ Patients must be aged between 18 and 60 years, have a body mass index of greater than 40 kg/m², or greater than 35 kg/m² with obesity-related comorbidity, and have been obese for more than five years with failed non-surgical attempts at weight loss. The Service is not available if a patient's weight is greater than 170 kg or their body mass index (BMI) of is greater than 55 kg/m² or if they are considered a high operative risk due to end stage disease.

Participants and study design—An audit was carried out at Auckland District Health Board (ADHB) between January and December 2010. Participants were included in the audit if they met the above selection criteria for bariatric surgery and had completed baseline questionnaires prior to bariatric surgery at any time forward from December 2008 to December 2010.

Participants completed the baseline questionnaire at a preoperative information seminar and returned this at their first preoperative clinic visit. Following a bariatric intervention patient's repeated the SF-36 questionnaire post-procedure at their 6-month follow-up clinic appointment through to the end of the audit period.

The Northern Regional Ethics Committee X approved the audit (NTX/10/01/EXP/001).

The SF-36 is a widely used generic instrument to measure HRQoL. The SF-36 contains 36 questions, 35 of which measure perceived health using a 0–100 scale across eight domains – physical functioning, role limitations due to physical functioning, bodily pain, general health, vitality, social functioning, role limitation due to emotional problems and mental health and the last item measures health transition, which is not reported here.⁸

Higher scores reflect better perceived health, with 100 being the best possible score for a domain. The eight domains can also be summarised into two component summary scores, namely the physical and mental component summary scores (PCS and MCS respectively).⁹ Both scores were normalised to have a mean of 50 and a standard deviation of 10. Scores below 50 represent scores below the population mean. As the standard deviation is 10, each point below 50 represents 0.1 of a standard deviation.

Statistical analyses—Student's paired t-test was used to examine differences between matched pairs, with statistical significance set at 5%. Missing data were handled as per standard procedures.¹⁰ Briefly, where greater than 50% of items in a multi-question domain (e.g. role physical and vitality) were answered, a person specific estimate or average score of completed questions within the same domain was used. To assess the size of any changes between baseline and post-procedure scores at 6 months, standardised response means (SRM) were calculated.

SRMs are the change in outcome divided by the standard deviation of the change and thus remove the noise of variance of the change leaving only the signal. Cohen's criteria for effect sizes were utilised when assessing the size of the SRMs.¹¹ SRMs of greater than 0.8 were considered large, 0.5 to 0.79 were considered moderate, 0.2 to 0.49 were considered small and SRMs less than 0.2 were considered trivial.¹²

All analyses were carried out in SPSS (version 18) software.

Results

Forty patients completed baseline SF-36 questionnaires prior to bariatric surgery. Twelve patients had either not received bariatric surgery or had not completed the 6 month follow up by the end of the audit period. Twenty-eight patients underwent bariatric surgery and completed SF-36 questionnaires at 6 months post-procedure during the audit period.

Twenty-three patients underwent laparoscopic Roux-en-Y gastric bypass and five underwent laparoscopic sleeve gastrectomy. These patients were predominantly New Zealand European, female, and class III obese (Table 1). Fifteen patients were diabetic.

Table 1. Patient characteristics and comorbidities at baseline and 6 months after the bariatric procedure

Variables	Baseline Number (%)	6 months Number (%)
Age (mean, SD)	42.8 (9.9)	
Female	22 (78.6)	
Ethnicity		
NZ European/Other	19 (67.8)	
Maori	4 (14.3)	
Pacific	3 (10.7)	
Asian	2 (7.1)	
Weight (kg, mean, SD)	131.2 (19.6)	94.4 (13.6)
BMI (kg/m ² , mean, SD)	45.3 (6.1)	32.6 (4.1)
Obesity class		
Overweight	–	12 (42.9)
Class I (BMI 30–34.99)	1 (3.6)	6 (21.4)
Class II (BMI 35–39.99)	4 (14.3)	2 (7.1)
Class III (BMI ≥40)	23 (82.1)	8 (28.6)
Type 2 diabetes mellitus		
Diet controlled	–	1 (3.6)
Oral hypoglycaemics	12 (42.9)	2 (7.1)
Insulin	3 (10.7)	1 (3.6)

The mean SF-36 domain and component summary scores are provided in Table 2. The baseline PCS and MCS reflect scores well below the population norms. There was significant improvement in all SF-36 scores across all domains at 6 months. The mean differences ranged from a low of 15.3 on the mental health domain to a high of 45.8 in the general health domain.

The change in the PCS would indicate that the patients moved from scores 1.5 standard deviations below the population mean prior to surgery to the population mean 6 months after surgery. The change in the MCS was not as dramatic, but still represents a shift of one standard deviation to slightly better than the population mean 6 months after the surgery.

Table 2. Difference between SF-36 domain scores at baseline and 6 months after bariatric procedure (n=28)

Domain	Baseline Mean	6 months Mean	Mean difference (95% CI)	P value
Physical functioning	53.1	86.1	33.0 (21.4-44.6)	<0.001
Role physical	45.5	81.3	35.7 (15.4-56.1)	0.001
Bodily pain	45.5	77.1	31.6 (20.0-43.3)	<0.001
General health	41.5	84.6	45.8 (37.0-54.6)	<0.001
Vitality	38.8	72.9	34.1 (24.8-43.4)	<0.001
Social functioning	58.9	87.1	28.1 (17.0-39.2)	<0.001
Role emotional	67.9	83.3	17.3 (2.5-32.1)	0.024
Mental health	67.4	82.7	15.3 (7.3-23.2)	0.001
PCS	36.0	50.1	15.3 (10.3-20.2)	<0.001
MCS	42.5	52.8	12.9 (8.2-17.7)	<0.001

PCS = Physical Component Summary score, MCS = Mental Component Summary score.

The SRMs for the mean differences were between 0.5 for the role emotional domain to 2.2 for the general health domain (Table 3). Although the change in the role physical, role emotional and mental health domains is a moderate sized change, the change in the remaining five domains is large. As would be expected from the large changes in some of the constituent scores, the changes in the PCS and MCS scores could also be considered large.

Table 3. Standardised response means to measure size of change SF-36 Health Related Quality of Life

Domain	Mean difference	SE	SRM	Effect
Physical functioning	33.0	5.7	1.1	Large
Role physical	35.7	9.9	0.7	Moderate
Bodily pain	31.6	5.7	1.1	Large
General health	45.8	4.3	2.2	Large
Vitality	34.1	4.6	1.4	Large
Social functioning	28.1	5.4	1.0	Large
Role emotional	17.3	7.2	0.5	Moderate
Mental health	15.3	3.9	0.7	Moderate
PCS	15.3	2.4	1.3	Large
MCS	12.9	2.3	1.2	Large

PCS = Physical Component Summary score, MCS = Mental Component Summary score.

SE = Standard Error, SRM = Standardised Response Mean.

Discussion

This is the first New Zealand study to report on the effect of bariatric surgery on HRQoL. Morbidly obese people qualifying for bariatric surgery have lower HRQoL

than the population norms and bariatric surgery appears to improve HRQoL to such an extent that these patients are returned to levels of HRQoL equivalent to the population norm.

Obese patients have been shown to have significantly lower HRQoL than the population in both New Zealand and overseas investigations,^{13 14} including bariatric surgery studies that have compared preoperative HRQoL to population norms.¹⁵⁻¹⁷

The baseline scores in the audit are of a similar order to the preoperative scores in other investigations, despite the small size of the audit cohort. The mean PCS and MCS scores were 31.6 and 42.9 in the Utah Obesity Study,¹⁸ which had 308 participants, and 36.8 and 45.2 in the Melbourne study, which had 459 participants.¹⁹

The significant improvements across all domains of the SF-36 are supported by international literature.^{16 18 19 17} This literature has rightly focused on longer term outcomes, but only one study has reported at 6 months in addition to longer term data.¹⁶ There were significant improvements across seven of the eight domains with role emotional being the only domain in which there was no significant improvement although there was a positive change in the domain score.

The change in scores ranged from 8-30 point improvements, with the PCS improving 1.3 standard deviation points to 50.2. These changes were all sustained over the two-year post-operative follow up with little change from the 6-month scores. Ten-year follow up in the Swedish Obese Subjects study suggested that peak changes in HRQoL occur in the first year,²⁰ which is consistent with the 6-month HRQoL improvements documented here. However the longitudinal measures at 12 months post-bariatric intervention would be valuable to support the international literature with further New Zealand perspective.

One other study explored the effect of bariatric surgery on HRQoL at two years, using Cohen's d (an effect size) in order to determine the size of the change in HRQoL as measured by various instruments.¹⁸ This study found 15 to 47 point changes in the SF-36 domain scores from baseline, with effect sizes ranging from 0.6 to 2.0. The effect sizes would have been considered large in seven of the eight domains compared to five of the domains in our audit. These effect sizes are consistent with our findings. However effect size uses the standard deviation of the baseline score rather than the standard deviation of the change and thus does not standardise response to between-subject variability over the course of change. Consequently, effect size may simply reflect the variance of the baseline scores rather than a true change in patient status.²¹ Thus our choice of using SRM rather than effect size.

This audit is subject to two main limitations. First, the size of the sample was small with only 28 patients having had bariatric surgery during the audit period. Although 12 patients could have potentially been included, they had either not received surgery or sufficient follow up to be added to this analysis. The audit represents 1 year of activity in the Service and improvements in HRQoL are of such an order that this small sample can show statistical significance. Second, only 6-month follow up data was available. However, it is intended to undertake longer term follow up to explore whether improvements are sustained.

Conclusion

Bariatric surgery makes a dramatic difference to HRQoL in morbidly obese patients. The SF-36 is an excellent tool to objectively measure the impact bariatric services have on patient-perceived outcomes. Services using the SF-36 can demonstrate the Service's impact on the whole of the patient's life across mental and physical health spectrums rather than just focusing on clinical outcomes.

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Undetected rheumatic heart disease revealed using portable echocardiography in a population of school students in Tairāwhiti, New Zealand

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Abstract

Aim The aim of this programme was to find undetected rheumatic heart disease (RHD) in students from selected schools in the Tairāwhiti region (eastern part of the North Island) of New Zealand.

Method Portable echocardiography was used to scan students in 5 urban and rural schools in Tairāwhiti where the population is predominantly Māori. The age range of students in the urban schools was 10–13 years and in the rural schools 5–17 years. Those with abnormal echocardiograms were referred for a paediatric consultation, with hospital-based echocardiography if required for the clarification of diagnoses and further management.

Results A total of 685 students, representing over 95% of the schools' students, consented to having echocardiographic scanning. After repeat hospital based echocardiography for 11 students, a total of 52 scans were regarded as abnormal. In this population definite (n=4) or probable (n=7) RHD was found in 11 students a prevalence of 1.61% (95%CI 0.80–2.85). Possible RHD was found in 19 students. Previously undetected confirmed (n=1) or probable (n=7) RHD was found in 8 students a prevalence of 1.17% (95%CI 0.51–2.29). Congenital heart defects (CHD) were found in 22 students a prevalence of 3.21% (95%CI 2.02–4.83).

Conclusion Echocardiography was a popular modality and detected a significant burden of previously unknown RHD in this young Māori population who are now receiving penicillin. However, echocardiography detected a greater prevalence of possible RHD for which optimum management is at present uncertain. Echocardiography also detected students with a range of severity of CHD. Screening with echocardiography for RHD would involve a significant use of public health, paediatric and cardiac resources with 7.6% of students and their families requiring clinical consultations and ongoing management of the abnormal echocardiographic results.

Many children affected by rheumatic fever have no history of the classical symptoms of arthritis but have episodes of undetected inflammation of the heart leading to permanent rheumatic heart disease (RHD).^{1,2} Untreated RHD leads to progressive cardiac valve changes, heart failure in early adulthood and decreased life expectancy.^{1,2}

Many adults present in their 3rd or 4th decade with heart failure due to RHD, many with no recorded history or memory of having had ARF.³ There are 150–180 deaths annually from RHD in New Zealand.² However, if RHD is detected early, progression

to serious RHD related morbidity can be prevented by penicillin.⁴ A recent innovative approach to allow rapid, detection of RHD for childhood populations is this use of portable ultrasound (echo) screening.^{5,6}

The first New Zealand study using portable echocardiography was undertaken in South Auckland in 2007–08.⁷ This study revealed a prevalence of 2.4% RHD in a population of 1174 10–13 year old children mainly of Pacific ethnicity from education decile 1 and 2 schools.

An opportunity arose in Tairāwhiti District Health to undertake a programme of echocardiographic scanning for undetected RHD for a limited period of two weeks in schools with high numbers of children already known to have had episodes of ARF. The programme was entitled *Let's get rid of rumatiki in Tairāwhiti* and was based on the evidence from the South Auckland Study.⁷

Methods

The echocardiography or scanning programme was planned and coordinated by the Public Health Service of Tairāwhiti District Health Board and the Paediatric Department in Gisborne Hospital in conjunction with the cardiology department of Starship Children's Hospital in Auckland.

The programme of scanning was undertaken over a two week period in March 2009. Consultations took place with schools and Māori health providers. As the project proposal was based on the South Auckland protocols, which had received ethics approval the Northern Y Regional Ethics Committee stated that no separate ethics committee review was required for this project. Of the childhood population just under 8% were screened.

Due to time and resource constraints the programme could not be offered to all the intermediate schools in Gisborne City. For the first week of the programme of the three intermediate schools (age range from 10–13 years) the school that was situated in an area known to have a high number of children who have had ARF was selected.

For the second week, the decision was made to scan a predominately Māori coastal community also with a high number of children who have had ARF. The four schools in this township community comprised two full primary schools for students aged 5–13 years and 2 composite schools for students aged 5–18 years.

The project team liaised with school staff and families and made educational presentations to the school children in their classrooms. A locally devised and detailed information sheet and consent form were sent home from school, outlining information about ARF and RHD, the scanning procedure, consent for the procedure and allowing parents/caregivers the option to be present for the procedure. Public Health and Rural Health nurses assisted with this process by conducting home visits and telephoning parents if consent forms were not returned.

School scanning procedures—The programme was based on echocardiography alone as it had been shown that auscultation was not sensitive in the South Auckland study⁷ or internationally.⁵ Vivid™ (GE Healthcare) portable cardiac ultrasound machines were used. Gain settings were optimised by the sonographers as the large variation in body habitus precluded the use of standardised settings. The echocardiography protocol consisted of 2-dimensional and colour Doppler images in parasternal and apical four-chamber views, with careful attention to record colour sweeps across any mitral or aortic regurgitant jets.

Pulse-wave Doppler interrogation of regurgitant jets was undertaken. Valve leaflet morphology was assessed in parasternal long axis (PSLA) (mitral valve and aortic valve) and parasternal short axis (PSSA) views (aortic valve). A variable range 2.5–5 MmHz probe was used for all studies. When indicated, echocardiographic measurements were interpreted according to body surface area. Considerable effort was undertaken to preserve modesty especially for adolescent girls. Private areas were available to change into gowns. All scanning was performed by female echocardiographers and the students were behind screens lying down on plinths. The echocardiograms were reported by the Starship Paediatric Cardiology Department. Criteria for the diagnosis of RHD is shown in Appendix 1, this process was the same as used in the South Auckland study.⁷

The parents or guardians of all the students were sent a letter after the scanning. If the results were normal this was stated on the letter, if the results were abnormal the letter stated "Your child ... will be seen with you by the children's doctors from Gisborne to discuss this result and to see if there is a need for any further tests or treatment". For each child a letter was also sent to the family doctor stating either that there was a normal result or an abnormal that required further paediatric assessment.

The programme took place over a 2-week period with a week in the urban school and a further week in the rural schools.

Any scans that were abnormal were reviewed by two paediatric cardiologists with a third opinion available and the classification made available to the project paediatrician.

Paediatric consultation and assessment—Clinical consultation allowed a full history, in particular any history of episodes of arthralgia or arthritis or other illness that could have been an episode of ARF. Examination was used to exclude abnormal body habitus or mitral valve clicks which would indicate that mitral regurgitation was congenital rather than rheumatic in aetiology. The presence of a diagnostic murmur enabled a clinical case definition of definite RHD rather than probable RHD. Repeat echocardiography was only utilised where there was diagnostic doubt about the echocardiogram.

Students with definite or probable RHD were recommended to commence long-acting intramuscular benzathine penicillin G (BPG) prophylaxis whereas observation with repeat consultation and echocardiography was recommended for those with possible RHD.

Results

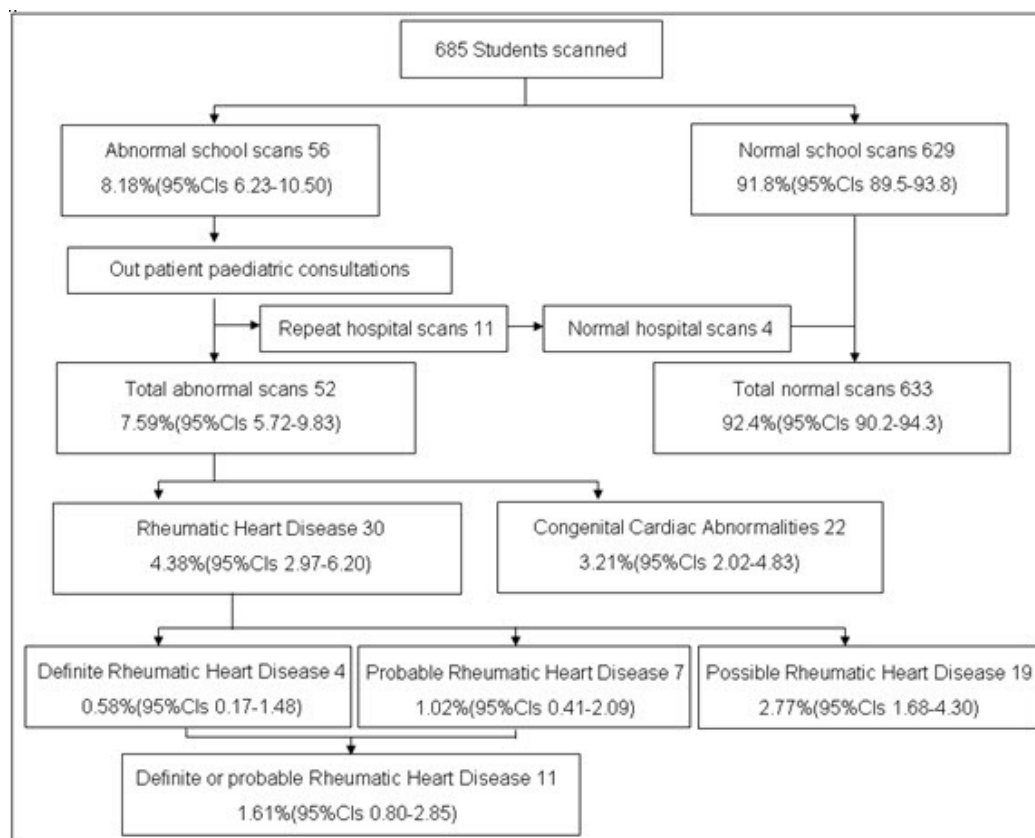
For the urban school the return rate of forms was 92% (437/474), with 96% consenting for echocardiography an overall consent rate of 88%. In the rural region the consent rate was 99% (366/369) with all those consenting wishing to be scanned.

Over the 2 weeks of the programme 362 echocardiograms were performed in the urban school and 323 in the rural locations, giving a total of 685 scans. The two echocardiographers performed on average 37 scans each per day, equating to one scan every 8 minutes.

Of the 685 echocardiograms performed 629 (91.8%) were classified as normal and 56 (8.2%) echocardiograms were classified as abnormal. Of the students with abnormal echocardiograms 11 (1.6% of the total performed) also underwent repeat hospital based echocardiography as the portable echocardiogram was not of sufficient quality to be diagnostic.

Students with abnormal scans were subsequently seen for a full consultant paediatric clinical assessment based on the echocardiographic findings. One child with probable RHD remains lost to follow up. After consultation by the paediatrician and repeat echocardiography a total of 52 students were found to have a cardiac abnormality, 30 due to RHD and 22 due to congenital heart disease (CHD). (Figure 1)

Figure 1. Flow diagram showing the number of students scanned, the numbers and percentages with 95 % confidence intervals (CIs) of normal and abnormal scans found at the schools and at subsequent repeat hospital scans and for the final diagnostic classification after clinical consultation



Of the 30 students with rheumatic valvular changes 11 had probable (n=7) or definite (n=4) RHD requiring prophylactic penicillin. Three of these 11 children were already known to have confirmed RHD on previous echocardiography and were under follow up and on BPG. Thus, the programme identified 8 students with previously undetected RHD, with a prevalence in this population sub group of 1.17% (95%CI 0.51–2.29).

Only one of these had a typical murmur of mitral regurgitation. Overall, the RHD prevalence for the cohort was 1.6% (95%CI 0.80–2.85). There was no statistical difference between the prevalence of RHD in the urban compared to the rural regions.

Nineteen children had valvular changes consistent with possible RHD representing 2.77% (95%CI 1.68–4.30). Of these the cardiac lesions observed were Mitral regurgitation (n= 11), isolated aortic regurgitation (n=5) and structural RHD without significant regurgitation (n=3).

These families have been counselled about the importance of sore throats, and their general practitioner advised about the importance of acute Group A streptococcal

(GAS) sore throat management. Repeat echocardiography is also recommended after 2 years to ascertain if there is progression to definite RHD and this work is currently being undertaken.

Twenty-two children had a CHD (Table 1) ranging from minor lesions such as bicuspid aortic valve to a child with a symptomatic large atrial septal defect who underwent semi-urgent cardiac surgery. The six children diagnosed as congenital mitral valve prolapse did not have a mitral valve click on auscultation but three of them were tall with thin body habitus. Overall the prevalence of CHD was 3.21% (95%CI 2.20–4.83).

Four thought to have abnormal echocardiograms were classified as normal after paediatric consultation and repeat echocardiography. One child had difficult echocardiographic windows due to pectus excavatum.

Table 1 Classification and number of congenital heart defects (N=22) discovered during the programme

Congenital heart defects	n
Congenital mitral valve prolapse	6
Bicuspid aortic valve ± aortic regurgitation or aortic annular dilatation	8
Atrial septal defect	1
Patent ductus arteriosus	4
Pulmonary stenosis	2
Tricuspid regurgitation	1

Discussion

We found 11 students with definite or probable RHD out of 685, a prevalence of 1.6% (95%CI 0.80–2.85) in 5 urban and rural schools in the Tairāwhiti region. Of these 11 students with RHD 8 had previously undetected RHD with a prevalence of 1.17% (95%CI 0.51–2.29).

The prevalence of RHD was lower than the 2.4% detected in South Auckland region but statistically the difference is insignificant.⁸ In Tairāwhiti the population is mainly Māori whereas in South Auckland the population is mainly of Pacific Island ethnicity with known higher rates of ARF than Māori.⁹

The RHD prevalence in this population was also lower than some international reports^{5,6,11} but direct comparisons are problematic due to the variations in case definitions used for rheumatic heart disease.

There was a high consent rate in all schools reflecting the high level of awareness of the consequences of ARF and well known cases of early death from RHD experienced in these close communities. In the rural region, all families were known to the Rural Health nurse who empowered the families to participate. Offsetting this was that consented students only underwent scanning if they were at school on the days when echocardiography team were present.

There was a lower rate of need for repeat hospital based echocardiography in this programme compared to that in the South Auckland study (1.6% vs 8%) reflecting

the accuracy of the portable echocardiography based on the experience of the South Auckland study.⁷

Echocardiography not only detects RHD but also detects CHD and in this programme 22 students were found with CHD which is 3.21% (95%CI 2.02-4.83). Some of the students with CHD required medical and even surgical intervention but many did not. Thus, considerable paediatric outpatient workload is created for those with abnormal echocardiography results, with the need for clinical assessment of the child and counselling of the families. Should echocardiographic screening be more widely implemented in New Zealand, an increase in paediatric clinical workload will occur.

The natural history of possible RHD (usually mild mitral regurgitation) without morphological changes of RHD is unknown, particularly what percentage of possible RHD on echocardiogram progresses to definite RHD.

In New Zealand we are currently recommending follow up in the first instance to see if there is any progression of RHD changes. The Australian gECHO programme is following up those with possible RHD as a case control study.¹⁰ A recent study in Nicaragua suggests that half of those defined as possible RHD may progress to definite RHD by echocardiography a decade later.¹¹

Since this programme, a decile 10 school in New Zealand in a region without any cases of ARF was studied. No cases of definite or probable RHD were found in 397 children but 2 had mild pathological mitral regurgitation meeting the criteria of possible RHD.⁸

The use of screening for RHD using echocardiography challenges the traditional model of the epidemiology of RHD where symptomatic ARF is the precursor of RHD.

There are many clinical scenarios where RHD presents without a history of ARF: Indolent carditis and Sydenham's chorea in childhood¹² and in adulthood symptomatic mitral stenosis, heart failure with mitral or aortic regurgitation, arrhythmia with underlying RHD, endocarditis and strokes with underlying RHD.² Thus, GAS infection causing asymptomatic Recurrent Rheumatic Fever and RHD may be as common as episodes of clinical ARF.

The considerable academic and clinical endeavour to combat ARF and RHD in New Zealand over the past three to four decades has recently been summarised.² Despite these endeavours, the incidence of ARF and prevalence of RHD has not fallen since the 1980s. Indeed, ethnic disparities are actually widening,⁹ and Māori and Pacific rates may be increasing.

Primary prevention of GAS pharyngitis is very important to reduce cases of ARF.¹² For many reasons this has not altered ARF rates in New Zealand. Children with a GAS sore throat may have difficulty accessing medical care, antibiotics may not be prescribed or a full course may not be taken. Importantly, ARF occurs without a history of sore throat in over a third of cases.¹³

There has been a renewed effort to reduce ARF rates through the production of New Zealand guidelines to raise awareness of ARF and for its appropriate management.^{12,13} There are also recent innovations using GAS sore throat school clinics in the primary prevention of ARF¹⁴ which can be effective when combined with community and

health professional awareness of the importance and correct treatment of GAS pharyngitis.^{15–17}

Effective secondary prevention has been the mainstay of ARF control in New Zealand with well run ARF registers,^{13,18,19} well audited in the Auckland region^{19,20} which has 60% of the disease burden of ARF in New Zealand.¹³

It is known that regular intramuscular long acting benzathine penicillin (BPG) reduces the recurrence of ARF by 87–97%.²¹ In Auckland ARF recurrences fell from 20% in 1980 to 5% in the period 1993–9.²⁰ Secondary prevention is currently considered to be the most cost effective clinical intervention for the control of RHD.²²

The place of echocardiography screening in RHD disease control is still to be established in New Zealand and globally. Portable echocardiography as a utility was demonstrated to be both fast (an average of an echo every 8 minutes) and also acceptable with 97% of students willing to have this form of test. This programme demonstrated that previously undetected RHD and clinically significant CHD can be detected using methodology that would be similar to a screening programme.

Echocardiographic screening is likely to be complementary to the primary prevention of ARF and secondary prevention of RHD. There is little doubt that it is better to diagnose early RHD changes so that BPG can be commenced to reduce the progression of the RHD and the burden of heart failure and valve replacement for individuals and the health service. We found the echocardiographic scanning was very acceptable to the students and parents with consent rates of over 95%.

Limitations of the programme described include not meeting all the requirements of an ideal screening test.²⁴ In particular the natural history of subclinical RHD detected by echocardiography is unknown and required. Treatment thresholds are still based on best clinical practice for RHD following an episode of ARF.

There are no randomised controlled studies that prove that echocardiography will result in reduced mortality and morbidity for RHD. Cost-effectiveness was not addressed in this study.

Competing interests: None known.

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Appendix 1

Criteria for Diagnosis of Rheumatic Heart Disease

Students were classified as *definite*, *probable* or *possible* RHD, congenital heart disease (CHD) or normal according to the combination of their clinical and echocardiographic findings. RHD classification was developed during a 2005 WHO expert consultation ²³ and modified with experience with RHD screening by the Paediatric Cardiology Department, Starship Hospital.

Definite RHD

- pathologic murmur of mitral and/or aortic regurgitation
- AND echocardiographic features of rheumatic heart disease:
 - pathologic grade mitral and/or aortic regurgitation
 - AND / OR valve morphologic changes of mitral and/or aortic valve consistent with RHD
 - OR Mitral stenosis (mean gradient \geq 4 mmHg)
- OR ARF with echocardiographic features of rheumatic heart disease

Probable RHD

- no murmur
- echocardiographic features of definite rheumatic heart disease

Possible RHD

- no murmur
- echocardiographic features of rheumatic heart disease
 - pathologic grade mitral and/or aortic regurgitation without morphologic changes of RHD
 - OR morphologic changes of mitral and/or aortic valve consistent with RHD without pathologic grade mitral or aortic regurgitation

Pathologic-grade regurgitation was defined as a colour Doppler jet meeting all the following minimum criteria.

Doppler criteria for pathological valve regurgitation

Mitral regurgitation	Aortic Regurgitation
Substantial colour jet seen in 2 or more planes	Substantial colour jet seen in 2 or more planes
Extending >2 cm beyond mitral valve leaflets in at least one plane (unless markedly posterior jet hitting atrial wall)	Extending >1 cm beyond aortic valve leaflets in at least one plane
Holosystolic with well-defined spectral envelope on PW/CW Doppler	Holodiastolic with well-defined spectral envelope on PW/CW Doppler
High velocity >3.5 m/sec	High velocity >3.5 m/sec
PW/CW pulse wave/continuous wave	

Morphological criteria for structural RHD

Mitral valve morphologic features	Aortic valve morphologic features
Thickening** of AMVL mid-point or tip	Thickening of AV leaflets or closure line in PSSA views
Fixed elbow (dog leg) deformity of AMVL mid-point or tip	Rolled AV leaflet edges
Prolapse of AMVL (in absence of clinical features of congenital MVP)	Overt prolapse of AV leaflets
Thickening** or retraction of PMVL	Coaptation defect of AV leaflets
Thickening, tethering, retraction or rupture to chordae of AMVL	

** valve thickening in the absence of other morphologic features of RHD was not classified as RHD

- AMVL - anterior mitral valve leaflet
- PMVL - posterior mitral valve leaflet
- AV - aortic valve
- PSSA - parasternal short axis
- MVP - mitral valve prolapse

A decade of serious non-fatal assault in New Zealand

John Langley, Pauline Gulliver

Abstract

Aim To describe the distribution of, and trends in, the characteristics of serious non-fatal assault injury for the period 2000–2009.

Methods Serious non-fatal hospitalised assault injury for the 2000–2009 period were identified and described by: sociodemographic characteristics, location of incidents, methods used to inflict injury, alcohol involvement, and nature of injury. Trends in assault by age, gender, and method were examined.

Results Males, 15–24 year olds, Māori, Pacific Islanders, and those from deprived neighbourhoods had markedly elevated assault rates. Assault by bodily force and head injuries predominated with the former being the major category of assault that increased the most over time

Conclusions There is a disturbing level of serious assault in New Zealand and the situation is getting worse. We need to review current efforts to prevent these incidents.

Assault resulting in injury is a significant public health problem in New Zealand. The most recently available national data indicates assaultive deaths account for 3.5% of all injury deaths and 3.4% of hospital discharges.^{1,2}

Assault attracts considerable public and media attention, and is one of six priority areas for the New Zealand Injury Prevention Strategy (NZIPS)³. A key focus of NZIPS is to “enhance the infrastructure that supports injury prevention activity to improve injury prevention performance”.

A recent review of that strategy reported that between 2003 and 2008 there was a 50% increase in the age standardised rates of serious non-fatal assaultive injury⁴. However, no further insight was provided concerning the risk factors for, or characteristics of, assaultive injury. Nor has there been any other recent published NZ research that might provide further insight into epidemiological trends of these more serious events.

Accordingly we sought to describe the:

- (1) Distribution of serious non-fatal assault injury for the period 2000–2009 by sociodemographic characteristics, location of incidents, methods used to inflict injury, alcohol involvement, and nature of injury; and
- (2) Trends in serious assault injury by age, gender, and method.

Methods

We used the same methods for defining a serious non-fatal assault case as that are used in the NZIPS chartbooks.⁵ Briefly, this consisted of identifying all cases in the National Minimum Data Set of hospital discharges (NMDS) with:

- An International Classifications of Diseases and Related Health Problems, version 10, Australian Modification (ICD-10-AM) injury related principal diagnosis (ICD-10-AM diagnosis code S00-T78), (b) where the first cause of injury was recorded as assault (ICD-10-AM external cause codes X85-Y09),
- Which resulted in injuries which were high-threat to life (6% or more chance of dying),
- The victim was discharged in the period 2000–2009.

Readmissions for follow-up treatment were excluded. Public hospitals provide 99.5% of inpatient treatment of injury in the acute phase.

For the purposes of providing a measure of treatment resource impact we estimated the cumulative bed days stay associated with each case. This measure is the sum of the days stayed over the first visit and any subsequent readmission related to the incident.

All records which meet the above criteria were searched for ICD-10-AM diagnosis and external cause codes indicative of alcohol involvement using the approach adopted by a US study of non-fatal suicide acts which resulted in hospitalisation.⁶ Records with ICD-10-AM codes indicating alcohol intoxication [Mental and behavioural disorders due to use of alcohol (F10): acute intoxication (F10.0), harmful use (F10.1) dependence syndrome (F10.2) residual and late onset psychotic disorder (F10.7), psychotic disorder (F10.5)] were identified.

Additional records with external cause of injury codes indicative of accidental poisoning by and exposure to alcohol (X45), intentional poisoning by and exposure to alcohol (X65) or alcohol poisoning intent undetermined (Y15) were also flagged as alcohol involved, as were those with diagnosis codes that included problems related to lifestyle—alcohol use (Z72.1), and evidence of alcohol involvement as determined by blood alcohol level (Y90.0–Y90.9). Cases were only counted as being alcohol involved once, irrespective of the number of alcohol related diagnosis or external cause of injury codes recorded in the hospital record.

The NMDS records self-defined ethnicity in which patients may self-report up to three ethnic groups to which they belong. For those who report multiple ethnicities, the concept of ethnicity for this investigation has been defined in line with NMDS reporting: ethnicity is prioritised with Māori ethnicity receiving the top priority, followed by Pacific Islander, Asian and finally European.

Residential area deprivation levels (NZDep2006) were based on the residential address of the assault victim as recorded in the NMDS. NZDep2006 scores are derived from Statistics New Zealand census data and combine measures of income, home ownership, support, employment, qualifications, living space, communication and transport to provide a measure of the relative level of deprivation for census meshblock areas (each meshblock contains approximately 87 people in 2006 and represent the area covered by a single census enumerator). Residential areas are assigned a value on an ordinal score from 1 (least deprived) to 10 (most deprived).⁷ Assault victims were assigned area deprivation scores based on their residential address.

Denominators for rates were obtained from the Statistics New Zealand usually resident population estimates (series 5) for June 30 of the relevant year. Series 5 estimates assume medium fertility, medium mortality and long-run annual net migration of 10,000. For the calculation of ethnic specific rates, Statistics New Zealand population estimates for the relevant ethnic group, as at June 30 of the relevant year was used as the denominator.

Ethnic-specific population estimates for Māori, Pacific Island and Asian populations were only available for the period 2006 onwards. These rates were age standardised. Age standardisation was via the direct method with five year age bands from 0–4 years to 85 and 85+ years. The standard population was the New Zealand population 2006 as at June 2006.

Calculation of crude rates for NZDep2006 decile categories were based on Statistics New Zealand census area unit population estimates, available for the period 2006 onwards, as at June 30 of the relevant years. Statistics New Zealand provides mapping files that allow meshblocks used in the calculation of NZDep scores to be mapped to larger geographical area units, such as census area unit

population estimates. NZDep scores for larger area units are derived from the average NZDep scores of the meshblocks included. 95% confidence intervals for rates assume a Poisson distribution. Ethics approval for this investigation was obtained from the New Zealand Multi-region Ethics Committee (Reference number OTA/99/02/008).

Results

For the period 2000–2009 inclusive there were 8006 serious non-fatal assaults (average annualised rate of 19.56 per 100,000 per year). Overall males accounted for the bulk (76%) of the burden of assault (Table 1). Males aged 15–24 years had the highest rates of assault. This rate was approximately five times the highest age specific rate for females (25–34 year olds).

Table 1. Sociodemographic characteristics of serious non-fatal assaults by gender 2000–2009

Variables	Males (n=6,335)			Females (n=1671)		
	N	%	Rate (95% CI)	N	%	Rate (95% CI)
Age						
0–14 years	239	3.8	5.3 (4.6–6.0)	156	9.3	3.6 (3.1–4.2)
15–24 years	2409	38.0	81.6 (78.4–84.9)	423	25.3	14.7 (13.3–16.2)
25–34 years	1633	25.8	61.6 (58.7–64.7)	463	27.7	16.3 (14.8–17.9)
35–49 years	1530	24.2	34.6 (32.9–36.4)	481	28.8	10.2 (9.3–11.2)
50–64 years	416	6.6	12.7 (11.5–14.0)	98	5.9	2.9 (2.4–3.6)
65+ years	108	1.7	4.9 (4.0–5.9)	50	3.0	1.8 (1.4–2.4)
TOTAL	6335	100		1671	100	
Ethnicity			2006–2009			2006–2009
Māori	992	31.7	77.4 (72.3–82.4)	380	47.8	28.0 (24.1–30.9)
Pacific Island	429	13.7	67.8 (60.8–74.7)	72	9.1	11.0 (8.5–13.6)
Asian	145	4.6	14.8 (12.2–17.4)	31	3.9	3.2 (2.0–4.4)
NZ European/ other	1563	50.0	26.0 (24.7–27.3)	312	39.3	4.8 (4.3–5.4)
NZDep 2006 Score			Crude rates 2006–2009			Crude rates 2006–2009
1 (least deprived)	30	0.5	2.5 (1.5–3.9)	13	0.8	0.8 (0.2–1.7)
2	227	3.9	13.4 (11.1–16.1)	30	1.9	2.4 (1.5–3.7)
3	420	7.1	26.8 (23.3–30.7)	100	6.4	6.4 (4.8–8.5)
4	502	8.5	27.6 (24.1–31.6)	147	9.4	8.1 (6.2–10.3)
5	615	10.4	36.2 (32.3–40.4)	158	10.1	8.2 (6.4–10.3)
6	633	10.7	36.1 (32.1–40.5)	140	8.9	7.9 (6.1–10.0)
7	961	16.3	56.9 (51.9–62.6)	219	14.0	12.9 (10.7–15.5)
8	898	15.2	50.6 (46.2–55.4)	260	16.6	14.1 (11.8–16.6)
9	1095	18.6	64.9 (59.7–70.4)	336	21.4	17.5 (15.0–20.4)
10 (most deprived)	519	8.8	31.1 (27.4–35.0)	166	10.6	8.1 (6.4–10.2)

For males there was a marked and consistent increase in rates among 15–24 year olds over time (Figure 1). The trend for females in this age group was very variable. The trend for the 25–34 year olds showed a more consistent and marked increase over time (Figure 2).

Figure 1. Serious non-fatal assault, annual rates by age group, *males only*

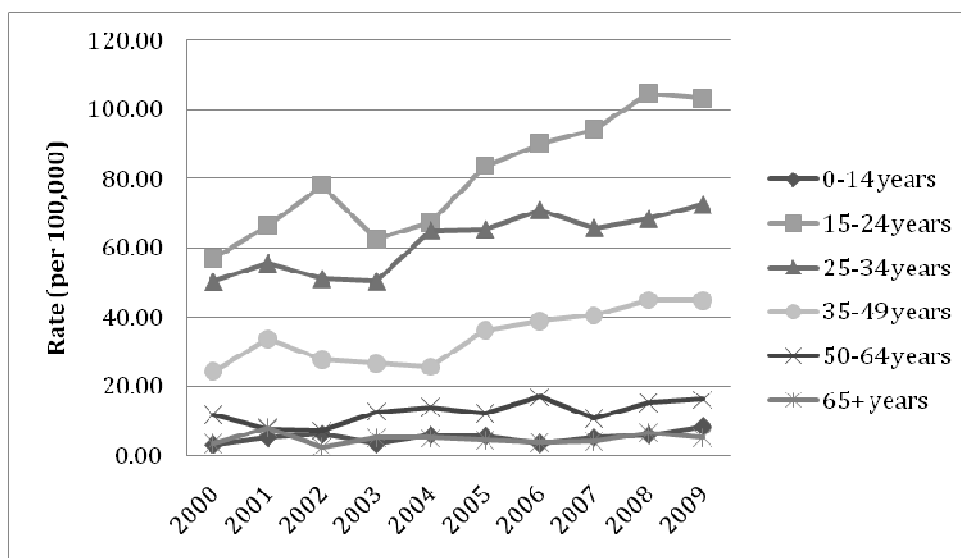
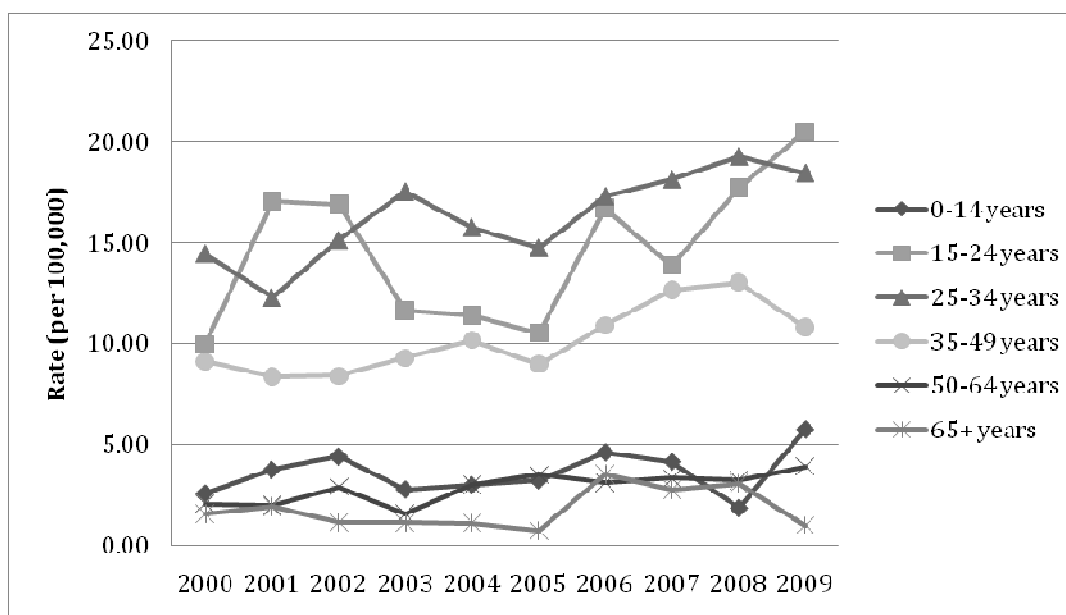


Figure 2. Serious non-fatal assault, annual rates by age group, *females only*



Māori accounted for almost half (48%) of female and one third (32%) of male serious non-fatal assaults (Table 1). Pacific Island people also figured prominently among the male statistics having a rate only slightly lower than Māori males. Between 2006 and 2009 the rates of serious non-fatal assault increased for Māori from 43.6/100,000–62.6/100,000, for Pacific Islanders from 37.3/100,000 to 38.6/100,000, for Asians

from 7.7/100,000 to 9.1/100,000 and for New Zealand European/Other from 13.3/100,000 to 15.8/100,000.

For both males and females the rates for NZDep groups 7, 8 and 9 were 17–26 times higher than that for the least deprived group for females and 32–39 times higher than the least deprived group for the males.

Overall, the home (26%), and the street/highway (18%) were the most common locations of incidents (Table 2). There was a difference in the location of the injury scene by gender, for female victims, 56% of assaultive injury events occurred in the home, in comparison with 20% for male victims; while 24% of assaultive injuries for males occurred in the street/highway compared with 10% for females. There was a high proportion of victims for which no information was recorded about the location of the injury event—35.5% of male cases and 24.5% of female cases (Table 2).

Table 2. Circumstances of serious non-fatal assaults by gender 2000–2009

Variables	Males (n=6335)		Females (n=1671)	
	N	%	N	%
Injury scene				
Home	1188	20.0	855	55.8
Residential institution	162	2.7	15	1.0
School/admin	88	1.5	20	1.3
Sports/athletics area	57	1.0	1	0.1
Street/highway	1397	23.6	156	10.2
Trade/service area	583	9.8	58	3.8
Industrial area	20	0.3	3	0.2
Farm	7	0.1	0	0.0
Other specified	322	5.4	50	3.3
Unspecified	2108	35.5	375	24.5
Method				
Hanging	14	0.2	41	2.5
Firearms	32	0.5	5	0.3
Smoke	18	0.3	8	0.5
Vapours	13	0.2	12	0.7
Sharp object	953	15.0	118	7.0
Blunt object	1284	20.3	214	12.8
Bodily force	2879	45.5	820	49.1
Neglect	125	2.0	213	12.8
All other*	1017	16.1	240	14.4
Alcohol involvement				
Yes	1433	23.6	220	13.1
No mention of alcohol	4902	77.3	1451	86.8
Total	6335	100	1671	100

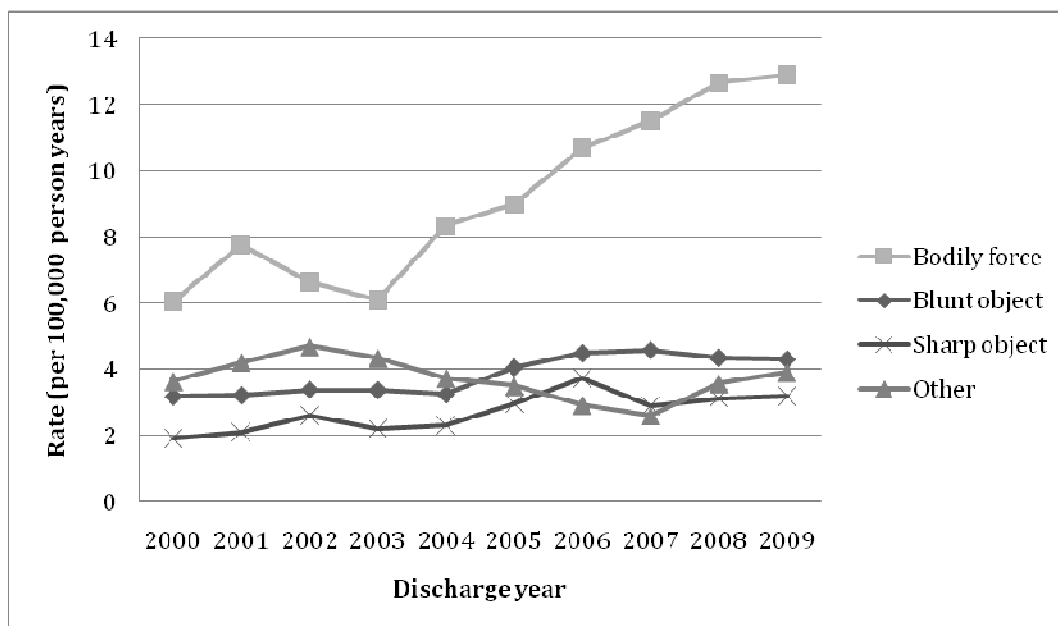
* Includes “other specified” (Y08), “unspecified” (Y09), poisoning by drugs, other substances, gas, and other noxious substances (X85, X86, X87, X88), drowning (X92), explosives (X96), pushed from a height (Y01, Y02), motor vehicle traffic crash (Y03).

The use of arms and legs (assault by bodily force) was the most common means of inflicting injury, accounting for nearly half the female cases and 45% of the male cases. For 12.1% of male and 7.0% of female assault related hospitalisations, the method involved was unspecified.

A higher proportion of males than females were recorded as being alcohol involved (23.6% vs 13.1%, Table 2). For those for whom alcohol was associated with the injury event, 10% (n=146) of males and 6% (n=14) of females had a blood alcohol content recorded on the clinical notes (data not shown).

Figure 3 illustrates that the use of bodily force (e.g. kicking, punching) was the method of assault that increased the most over time.

Figure 3. Serious non-fatal assault, annual rates by method



For the majority of serious non-fatal assaultive hospitalisations, head injury was the principal diagnosis accounting for 72.6% of serious non-fatal injuries (Table 3). The 8006 incidents required a total of 35,186 bed day's stay. There was considerable variation in the days stay by nature of injury (median total length of stay = 1, range 0–443 days).

Although the single most frequently recorded group of diagnoses was a fracture of the skull, the three principal diagnoses that recorded the highest median total length of stay (epidural haemorrhage, traumatic subdural haemorrhage and traumatic subarachnoid haemorrhage) accounted for 12.2% of serious non-fatal assault cases. Injuries to intra-thoracic and intra-abdominal organs also recorded high median total length of stays (median 4 days and 5 days; range 0–56 days, 0–52 days respectively).

Table 3. Nature of injury and diagnosis specific length of stay (LoS)

Diagnosis (3 char)	Diagnosis (4 char)	Description	N	%	First admission		Cumulative total	
					Median LoS	Range LoS	Median LoS (total)	Range LoS (total)
S02	S020	Fracture of the skull	2490	31.0	1	0–111	1	0–414
	S021	Fracture of vault of skull	245	4.8	1	0–37	3	0–235
	S022	Fracture of base of skull	385	7.4	1	0–34	2	0–244
	S023	Fracture of nasal bones	263	5.0	0	0–9	0	0–15
	S024	Fracture of orbital floor	283	5.4	0	0–14	1	0–18
	S026	Fracture of malar and maxillary bones	590	11.5	1	0–111	1	0–414
	S028	Fracture of mandible	447	8.7	1	0–12	2	0–69
		Fractures of other skull and facial bones	205	3.9	1	0–13	1	0–16
S06	S060	Intracranial injury	1980	24.8	0	0–78	1	0–443
	S063	Concussive injury	966	18.8	0	0–25	0	0–35
	S064	Focal brain injury	198	3.9	2	0–51	3	0–122
	S065	Epidural haemorrhage	105	2.0	3	0–78	6	1–443
	S066	Traumatic subdural haemorrhage	399	7.6	3	0–72	7	0–306
		Traumatic subarachnoid haemorrhage	134	2.6	2	0–72	4.5	0–237
S01	S010	Open wound of head	703	8.8	0	0–19	0	0–83
	S018	Open wound of scalp	362	7.0	0	0–19	0	0–48
		Open wound of other parts of head	127	2.5	0	0–5	0	0–83
S00		Superficial injury of head	438	5.4	0	0–10	0	0–10
S27		Injury of other and unspecified intra-thoracic organs	412	5.2	3	0–54	4	0–56
S36		Injury of intra-abdominal organs	284	3.6	5	0–36	5	0–52
S09		Other and unspecified injuries of head	213	2.6	0	0–16	0	0–37
S22		Fracture of rib(s), sternum and thoracic spine	186	2.3	1	0–30	1	0–62
Other*			1300	16.2	1	0–92	1	0–236
Total			8006	100.0				

* No other 3-character ICD-10-AM principal diagnosis category exceeded 2%.

Discussion

General findings—For the period 2000–2009 there were an average of 801 serious non-fatal hospital discharges in New Zealand annually. The rate of these has increased by 63% over the 10-year period reviewed. The variations in rates by age, ethnicity, deprivation, and gender are substantial. For example, males aged 15–24 have the highest rate of serious injury from assault. That rate is 16 times that of male 65+ age group and is more than five and a half times higher than for the 15–25 female age group.

Similarly, while male Māori and Pacific Island people have a similar rate, that is not the case for females, where the Māori rate is more than twice that for Pacific Island people. The findings for deprivation are even more striking, males in the decile 9 having an assault rate 26 times those in decile 1.

It should be emphasised at the outset that this study was concerned with serious assault defined as incidents where the resulting injuries represented a threat of dying of 6% or more. It is thus of considerable concern that we identified 8006 cases in a ten year period which met this threshold and moreover that rate of these serious incidents is increasing with time.

Strengths and limitations—The main strength of the investigation presented lies in the case definition. Publication of trends in physical assault often result in considerable debate as to their meaning. For example, in New Zealand the annual release of police statistics on violence which might show an increase in the incidence is characterised by two distinct responses from Parliament.

The opposition claim it illustrates the Government's failure to control violence, while the Government claim that it illustrates that programmes which promote public intolerance to violence, and thus the need to report it to the police, are working. Both explanations could be valid to varying degrees. However, as police statistics are subject to extraneous factors (such as changes in reporting behavior) we have no way of determining the true explanation¹⁶.

It has been argued that in order to overcome this problem data from emergency departments should be used¹⁶. This ignores the fact that such health service data are also subject to extraneous influences such as changes in service delivery.¹⁷ Similar arguments apply to the unthinking use of hospital inpatient data, a common source of data for monitoring trends in non-fatal injury¹⁸.

This contrasts with trends we have investigated here. We applied a threshold for severity of injury which enables us to have a reasonable degree of confidence that we have captured most of the cases consistently and thus the trends presented reflect the underlying phenomenon of concern, namely serious non-fatal assault injury. In other words it is our contention that assault victims whose injuries represent a threat to life of 6% or more will be invariably hospitalised, irrespective of access and service delivery issues.

It is possible that, even using the threshold employed, variable levels of reporting the cause of injuries may exist. We have previously examined this possibility for serious non-fatal assault and have found that no systematic change in reporting the cause of injury existed.¹⁹ Having said that, in 2006, the Ministry of Health mandated for the

inclusion of emergency department discharges in the national collection of hospital discharge data.

Thus, trends in serious non-fatal assault injury from 2007 onwards may be influenced by increased reporting from some New Zealand hospitals. However, the impact of increased reporting will have been moderated by the high severity threshold we have used. In addition, the trends reported in this investigation began well before 2007 suggesting that increases in the frequencies and rates of serious non-fatal assault are real.

There are a number of limitations with this study. There were a high proportion (34%) of cases for which the location of the injury event was unspecified. In addition, the proportion of unspecified locations varied by gender, indicating that the distributions reported for specific locations needs to be treated with caution. There was also limited information on the perpetrator involved in the assaultive event (the perpetrator was listed in only 31% of cases). As a result, we elected not to include this information in the analysis.

A further limitation of this study is our findings on alcohol involvement. In terms of alcohol involvement, it is important to note that our results relate to the victim. We have no information on alcohol involvement among perpetrators. However, some of the 'victims' in this series may also have been 'perpetrators' in the incident in which they were injured. For example, a fight that is initiated by an intoxicated person who also subsequently is injured.

Moreover, the 'alcohol involvement' we have reported (Table 2) is likely to be a significant underestimate of the proportion of serious non-fatal assault cases who had consumed alcohol prior to their hospital event given that patients are not routinely screened for acute or chronic alcohol problems.

We elected not to disaggregate the alcohol data since in those cases where more than one alcohol code was specified there appeared to inconsistencies in the manner in which acute intoxication was recorded in combination with diagnoses that indicated chronic alcohol problems.

We have no way of knowing whether alcohol involvement played a casual role in the assaultive injuries described in this paper. It could well be, for example, that many of the males just happened to be drinking at the time. On the other hand there is evidence from elsewhere that being intoxicated increases one's risk of becoming a victim of assault⁹. Alcohol misuse is also a distal risk factor for assault, where a pattern of heavy episodic drinking has been associated with a threefold increase in victimisation involving drinking.¹⁰

The definition of serious in this study was based on threat to life, operationally based on acute anatomical damage. By definition this excludes important assault incidents where the main outcome is a mental disorder (e.g. many sexual assault incidents). Regretfully there is no database in NZ that allows one to describe the epidemiology of the more serious of these incidents in a manner similar to that used here.

While the Accident Compensation Corporation maintains a sensitive claims database which records details on such cases, this database is also susceptible to extraneous

influences that vary over time, not the least of which is changes in eligibility and the strictness with which such criteria are implemented.

In their guide to the use and reporting of ethnicity data, Statistics New Zealand recommend using the ‘total ethnicity’ concept in which case all persons are counted in all ethnic groups to which they associate. The result is that the total across all ethnic groups is greater than the whole population. Prioritised recording of Māori ethnicity in the NMDS roughly equates to ‘total ethnicity’ reporting for Māori, as all those who identify themselves as Māori are subsequently reported as such. However, because of the nature of the prioritisation, use of the prioritised ethnicity field is likely to result in an undercount of other ethnicities. For this investigation the prioritised ethnicity field was used. As such, there may be an undercount of ethnicities other than Māori.

Implications—In recent times there has been a concern expressed about the incidence of knife related assaults⁸. While we have not presented statistics on knife assaults our results show that in total, sharp objects (the category a knife is coded under) account for 13% of all assaults that result in serious injury.

While there is some evidence of an increase in incidents involving sharp objects, of greater concern is that the overall increase in serious assault is largely attributable to incidents involving ‘bodily force’. We are not suggesting that initiatives to reduce the risk of increasing knife crime in New Zealand should not be pursued. Rather it is important that, as a community, we do not ignore or under-resource prevention initiatives aimed at the major cause of harm in pursuing such initiatives.

Prior to the analyses presented here the most recent published descriptive epidemiological overview of non-fatal assault, from a victim perspective, in New Zealand was published in 1995 and was concerned with assaults resulting in hospital admission from 1979–1988¹¹.

As emphasized above our series focused on a subset of hospital admissions. Despite this both series identified males, young adult, and Māori, as being at elevated risk. Similarly use of bodily force was the most common mechanism of injury, and the home as a common location of the incidents. There are very few published studies from other countries that have been concerned with all types of assault to all persons.

The literature is dominated by studies of domestic violence and sexual assault with an emphasis on injury to women. The few recent studies we have been able to identify which are similar in scope to our investigation report similar sociodemographic patterns and circumstances of injury.^{12–14}

Typically statistics on assault are based court or police statistics and as such are perpetrator orientated. It is of interest to consider the prevention resource implications that may arise from our analyses and analyses in which the person of interest is the perpetrator. For example, in a recent presentation based on Court and Police data, Judge Becroft was reported as saying “*Firstly youth violence is not out of control, in fact it had been decreasing for the past 10 years, but violent crime was increasing in every age group, though the increase was greatest in those aged over 60*”¹⁵.

In contrast we have shown a substantial and relatively steady increase over time in the rate of serious assault among males aged 15–24 years. Due to smaller numbers, trends

for females aged 15–24 years is more variable. However, there is suggestion of significant increase in the most recent years.

Despite the limitations highlighted above, this study has provided new and recent insights into serious non-fatal assaultive hospitalisations. The trends we have observed are concerning, especially since there is no evidence of any recent abatement. This raises questions about our efforts to reduce such harm.

Are we applying evidence-based interventions? If so how good is our application? Are we targeting the right sectors of the population in our injury prevention efforts? Given the cross-sector role of NZIPS, and the recent addition of alcohol as a crosscutting theme for NZIPS, it would be a most appropriate mechanism through which to pursue these important questions.

Competing interests: None known.

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Non-traumatic spontaneous spinal subdural haematoma

Nur A B Haji Mohd Yasin, Daniella Donato-Brown, Ahmad Taha

Abstract

We are presenting a case of non-traumatic spontaneous spinal subdural hematoma in a patient on warfarin and fluoxetine. This diagnosis should be considered early in patients who are on warfarin or fluoxetine or both presenting with acute neurological abnormalities of the limbs, and early decompression could result in good neurological outcome.

Spinal subdural haematoma is a rare condition that can have devastating consequences if not managed early.¹ There are multiple aetiologies described in the literature. Common causes of spontaneous spinal subdural haematoma include vascular malformations, neoplasm, and coagulopathy (either hereditary or secondary to anticoagulant therapy).¹

In this report, we present a case of non-traumatic spontaneous spinal subdural haematoma in a patient on warfarin and fluoxetine.

Case report

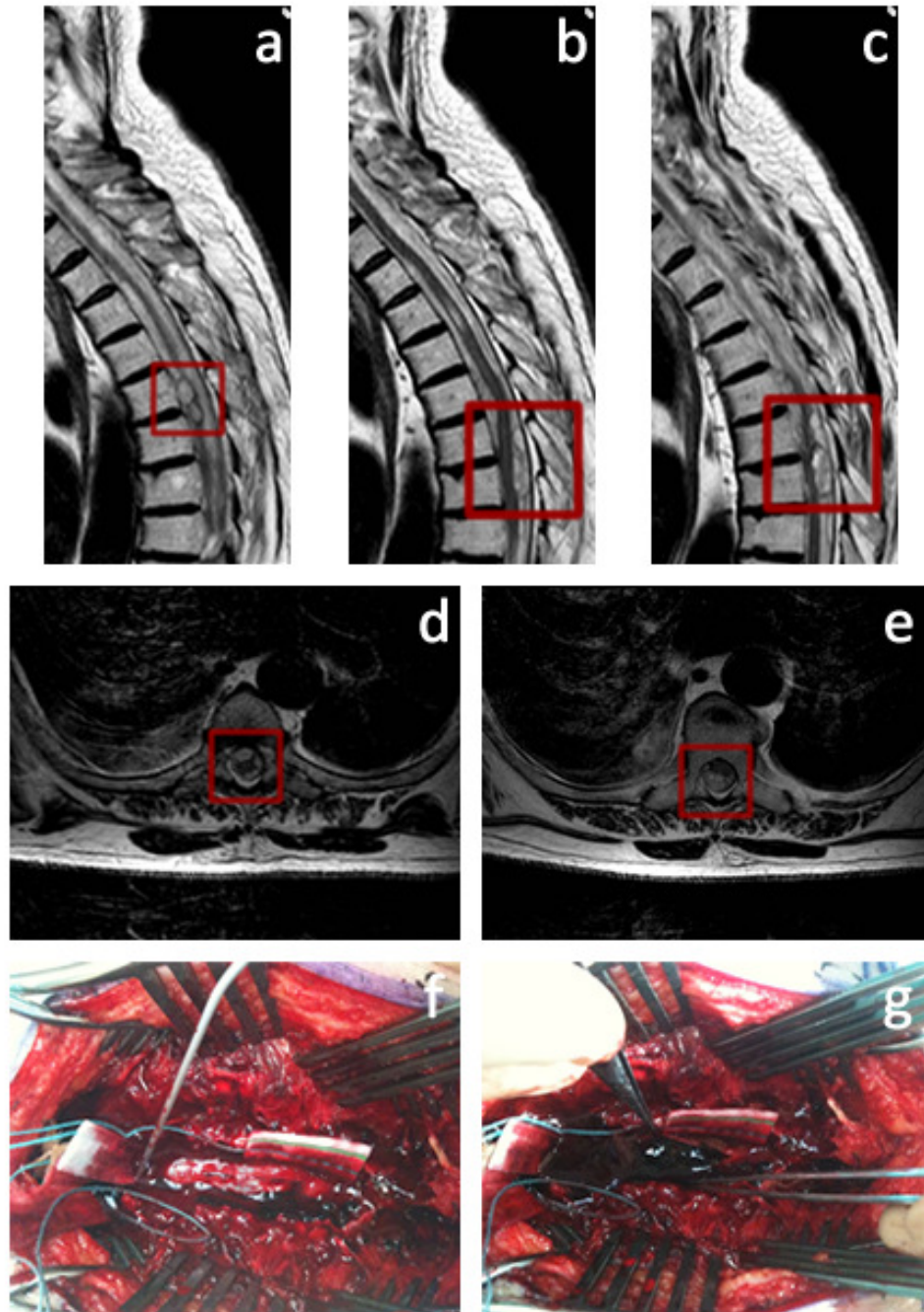
A 73 year old gentleman with a background of atrial fibrillation on warfarin presented with sudden onset of back pain. This initially started in the lumbar area with radiation down both lower limbs, and then evolved to complete paralysis of both lower limbs and urinary retention in 12 hours. His past medical history included depression, coronary artery bypass grafting, previous renal calculi, hypertension, transient ischaemic attack, and gastro-oesophageal reflux. His regular medications were fluoxetine, candesartan, metoprolol, simvastatin and ezetimibe.

On admission to the neurosurgical service at Dunedin Hospital, his lower limb power was MRC grade 0 out of 5 in all ranges of movement bilaterally. Light touch sensation in both lower limbs was normal with the exception of the right foot. The knee, ankle and plantar reflexes were absent bilaterally. Rectal examination revealed poor tone in the anal sphincter. The INR was 2.8.

MRI scan showed loculated areas of increased T2 signal anterior and posterior to the cord from T5–T7.

The patient received prothrombinex, vitamin K and dexamethasone before T5–T8 laminectomy was performed with removal of the large subdural haematoma encircling the spinal cord from T5 to T9. The time interval between the onset of the symptoms and the operation was 16 hours.

Fig a–c. Sagittal left to right T2 scans showing loculated areas of increased T2 signal anterior and posterior to the cord from T5-T7; Fig d. Axial T2 scan showing loculated areas of increased signal anterior to the cord at T5/6; Fig e. Axial T2 scan showing loculated areas of increased signal posterior to the cord at T7; Fig f. Tense dura prior to incision; Fig g. Subdural haematoma after incision of dura



Postoperatively, the patient received daily physiotherapy and recovered some of his lower limb motor function as described in Table 1.

Table 1 Lower limb strength recovery over 8 weeks period

Variables	Admission (Rt)	2 weeks (Rt)	8 weeks (Rt)	Admission (Lt)	2 weeks (Lt)	8 weeks (Lt)
Hip flexion	0/5	0/5	1/5	0/5	3/5	3/5
Knee extension	0/5	0/5	3/5	0/5	2/5	4/5
Ankle dorsiflexion	0/5	2/5	4/5	0/5	2/5	4/5
Ankle plantarflexion	0/5	1/5	4/5	0/5	1/5	5/5
Toe extension	0/5	2/5	3/5	0/5	2/5	3/5

Rt, Right Leg; Lt, Left Leg.

At 8 weeks, he was mobilising with a wheelchair, and still required a suprapubic catheter but was faecally continent.

Discussion

A review by Domeniucci et al, which looked at 106 cases of non-traumatic acute spinal subdural haematoma from 1948 to 1998, found that 35% of the cases were on anticoagulant therapy. A number of them were on the early generations of vitamin K antagonist therapy such as dicumarolic therapy.¹ More recent case reports involve other vitamin K antagonists such as warfarin and acenocoumarol.^{2,3}

Another possible contributing factor for the spontaneous bleed was fluoxetine. A case control study looking at 1848 patients on vitamin K antagonist showed that SSRI usage was associated with increased risk of hospitalisation secondary to non-gastrointestinal bleeding such as intracranial bleed, haemarthrosis, haemoperitoneum etc.⁴ This is due to SSRIs decreasing the serotonin level in platelets which eventually leads to the inhibition of platelet aggregation.

In this case, the definitive diagnosis of spinal subdural haematoma was made after investigating the patient with MRI. We agree with Braun et al that MRI is the modality of choice in diagnosing spinal subdural hematoma.⁵

In conclusion, we recommend that patients who are on long term anti-coagulation therapy, especially if this is combined with concurrent use of selective serotonin reuptake inhibitor, presenting with back pain and acute neurological deficits should be investigated with an immediate MRI scan, as early diagnosis of spinal subdural hematoma and subsequent spinal cord decompression could result in good neurological outcome.

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Right atrial mass—a venous thrombosis in transit

Jonathan W Hinton, John Lainchbury, Ian Crozier

Abstract

Right heart thrombi are unusual complications of pulmonary embolism that are associated with a high early mortality. We present a case and transoesophageal echocardiography of a 65-year-old man who presented with pulmonary embolism, following routine knee replacement and was found to have a type A right heart thrombus. Despite the increased risk associated with this presentation, treatment with heparin alone was successful.

Case report

A 65-year-old man was admitted with an acute pulmonary embolism. He had undergone a routine knee replacement 2 days prior and then presented with syncope and hypoxia. He had no other significant history.

On examination he was unwell, tachycardic and had a swollen left leg. His electrocardiogram showed atrial fibrillation with a ventricular rate of 107 beats per minute.

Computerised tomography pulmonary angiogram showed extensive bilateral pulmonary emboli with borderline right heart strain. He was treated with intravenous heparin and loaded with warfarin (he was not thrombolysed due to his recent surgery).

Transthoracic echocardiography showed a flattened septum with abnormal motion secondary to right ventricular pressure overload and a moderately dilated right ventricle with moderate to severe right ventricular systolic impairment. In addition there was a mobile structure in the right atrium. Subsequent transoesophageal echo showed a large mobile linear mass within the right atrium, which was curled around the Eustachian valve (Figure 1).

A repeat transthoracic echo prior to discharge showed no sign of the thrombus that was previously seen. He recovered well and was discharged a week later.

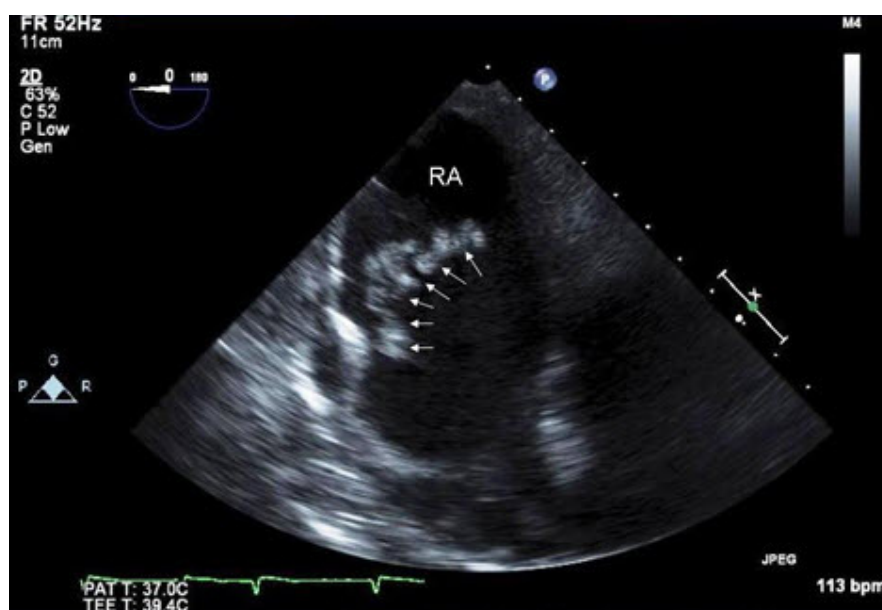
Discussion

Right atrial thrombi in association with pulmonary embolism have been well reported.¹⁻⁴ This complication occurs in less than 4% of unselected patients with pulmonary embolism but is found in 7–18% of patients requiring admission to intensive care with pulmonary embolism.⁵

There are two forms of right heart thrombi: type A (as in our patient) which are long, thin, highly mobile and are associated with a high incidence of deep vein thrombosis; type B resemble left heart thrombi, are immobile and are associated with thrombogenic cardiac abnormalities.⁴

Whilst this is an unusual complication of pulmonary embolism, type A thrombi are important because they are associated with a high early mortality (45% within the first 8 days).⁴ Treatment is controversial owing to the low number of cases and the lack of randomised controlled trials.⁵ Thrombolysis and surgical intervention seem to have preferable outcomes compared with anticoagulation alone.⁵

Figure 1. Right atrial thrombus (RA – right atrium)



We postulate that the right atrial mass seen on transthoracic echocardiography in this patient was a type A right heart thrombus in transit from the peripheral venous system. Subsequently, this may have either embolised into the pulmonary bed (without clinical sequelae) or dissolved.

Whilst thrombus passing through the right atrium has been previously reported these images show an unusual and clear example of a venous thrombus that became entrapped in the right atrium periodically. This was managed conservatively with anticoagulation and did not go on to cause a further significant embolism.

This case highlights that right atrial thrombi should be considered in patients presenting with pulmonary embolism who are found to have a right atrial mass on echocardiography and the ability of transoesophageal echo to clarify the nature of the mass. Despite increased risk indicated by the clinical presentation and echocardiographic appearances treatment with heparin alone was successful.

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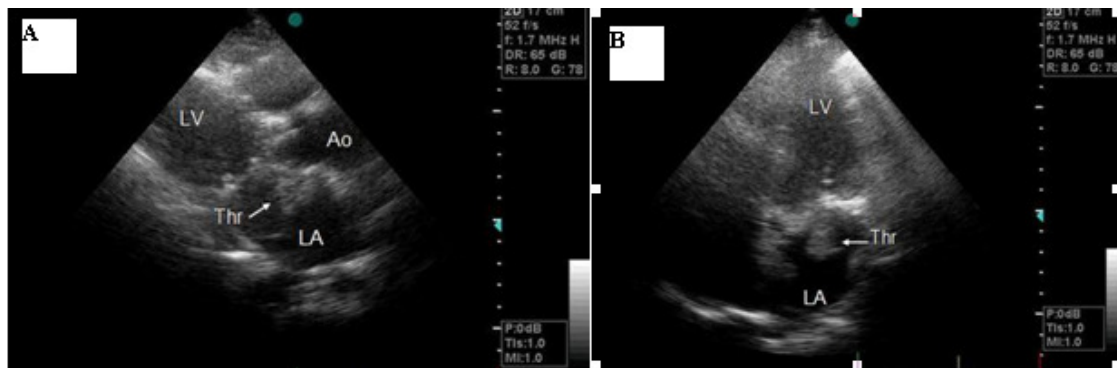
The malign face of atrial fibrillation

Suleyman Ercan, Adnan Dogan, Vedat Davutoglu, Gokhan Altunbas

A 78-year-old female patient presented with a 1-month history of cold pulseless lower limbs consistent with ischaemia. She was diabetic and hypertensive. No popliteal or pedal pulses could be palpated. Electrocardiography revealed atrial fibrillation (AF).

Echocardiography revealed that thrombus in atrial side of the mitral valve (Figure 1), with moderate mitral regurgitation, mild left atrial enlargement (43 mm), and normal left ventricular systolic function. Lower extremity arterial Doppler revealed occluded bilateral superficial femoral arteries. Revascularisation was recommended but the patient refused. Warfarin and enoxaparin was started but she did not use them regularly.

Figure 1. Transthoracic echocardiographic images (A) parastenal (B) apical approach



LV: left ventriculi, LA: left atria, Ao: Aorta, Thr: thrombi.

A month later bilateral necrosis below knee gangrene developed and amputation was performed by orthopaedic surgeons (Figure 2). No thrombus was observed on the echocardiography performed at this time.

The objective for presenting this case was to remind once again that AF is not an innocent disease and may lead to severe limb ischaemia. We recommend that all AF patients should be evaluated about necessity of anticoagulation and those presenting with thromboembolic symptoms to be screened by echocardiography to diagnose left atrial thrombus.

Figure 2. Bilateral necrosis beneath the knee



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Persistent fever in ulcerative colitis

Pazhanivel Mohan, Rahul A Kothari, Krishnaveni Janarthanan

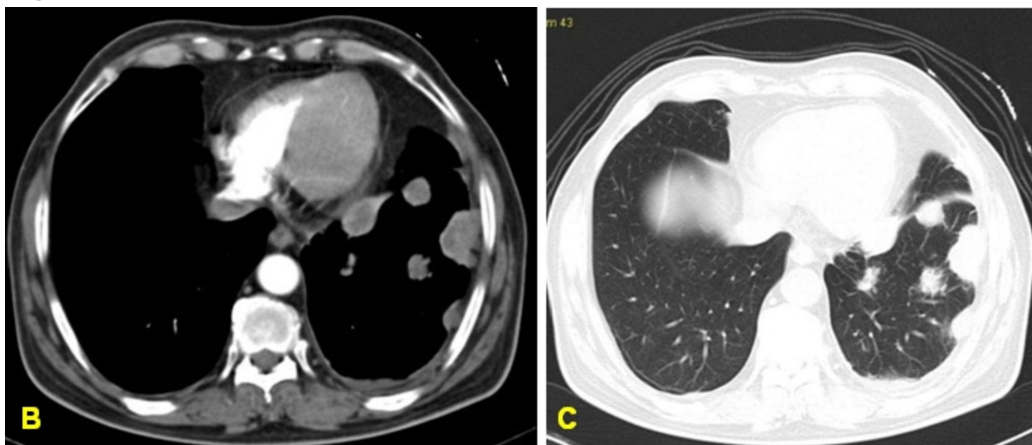
Clinical—A 69-year-old man with ulcerative colitis was admitted with persistent fever for 4 days. There was no history of cough, breathlessness or chest pain. He was on treatment for ulcerative colitis for the past 5 years. He was started on prednisolone for acute exacerbation of ulcerative colitis, 4 weeks prior to the onset of fever. There was no abdominal pain, vomiting, jaundice or distension of the abdomen. Blood investigations revealed haemoglobin: 11.3 gm/dl; total count – 8300 and platelets – 228,000/mm³. His renal and liver function tests were normal. X-ray of the chest (Figure A) and contrast CT scan of the thorax (Figures B & C) were done.

Figure A. X-ray chest



What is your diagnosis?

Figure B & C. Contrast CT of the thorax



Answer and Discussion—X-ray chest showed multiple rounded opacities in the left lung (Figure A). Contrast CT of the thorax showed multiple subpleural and pleural basal nodular densities predominantly in left lung fields (Figures B & C).

Sputum examination showed branching filamentous acid fast bacilli resembling *Nocardia* which was later grown in culture. His fever settled with combination of sulfamethoxazole and trimethoprim and withdrawal of steroids. There was complete resolution of lung shadows at 4 weeks.

Pulmonary involvement is less common in inflammatory bowel disease (IBD) with drug-induced disease responsible for a majority of cases.

Aspergillus fumigatus, *Nocardia asteroides*, *Mycobacterium tuberculosis* and *Pneumocystis carinii* have been reported with cyclosporine, infliximab and corticosteroids.¹

TREAT registry has identified the use of prednisone, narcotic analgesics and severe disease activity as responsible for serious infections and mortality in Crohn's disease.² There is often difficulty and delay in the diagnosis of *Nocardiosis* due to its varied clinical and radiological presentation.

The infection can be localized or disseminated most often affecting the lungs, skin and central nervous system. No randomised control trials are available for the best treatment regimen in nocardiosis.

A combination of SMZ-TMP is preferred for pulmonary and cutaneous involvement. The duration of treatment varies from 6 months to a year depending on the location of lesions and host immunity.³

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Premature failure of artificial joint components

Readers of the popular press may have noted that orthopaedic surgery has been in the news recently with regard to premature failure of artificial joint components. Medical practitioners would be unwise to think that they are immune to this type of publicity.

Medicine in general, and surgery in particular, is a fashion industry. Patients want the latest devices; instrument companies want surgeons to use the latest devices; pharmaceutical companies want practitioners to use the latest drugs; and overseas trends are held in high regard.

Much information gleaned by patients from the Internet is believed to be the last word rather than subtle marketing on behalf of industry. The medical supply industry is rather like the auto industry, devices and drugs have a product life of 3 or 4 years before they are superseded by a new or upgraded model, and in many cases these changes result in products that are not as effective as the ones they replace. This is particularly the case in orthopaedic surgery.

In the medical world, the area of deep vein thrombosis (DVT) prophylaxis is another fashion-driven industry. Most articles on the topic are written by groups compromised by industry funding, and guidelines for the same problem are vary enormously between countries, although this year for the first time there is agreement on prophylaxis between the American Academy of Orthopaedic Surgeons and the American College of Chest Physicians—two groups who for years disagreed.

Although it might seem unfashionable, medical practitioners should stick to proven technologies rather than satisfy the whims of patients who have information from the Internet, or salespeople anxious to achieve sales targets for new devices or drugs.

Geoffrey Horne

Professor and Orthopaedic Surgeon
Wellington Regional Hospital
Wellington

Presidential Address: Quackery and chemists

Excerpt of a Presidential Address by H. V. Drew, F.R.C.S. Annual Meeting of the New Zealand Branch, Timaru, 1912. Published in NZMJ 1912 March;11(41):1–8.

The next subject that deserves scrutiny in the interests of the community is quackery, of which there are many varieties. Many attempts have been made to control this. The B.M.A. has had analyses made of many of the best known quack medicines, in order that the public may learn the simplicity of the frauds which amass millions to the quacks and induce the patient to postpone getting a doctor's opinion and so wasting money and time (which is the more valuable), for when lost the opportunity for successful treatment has often gone forever.

Whilst on this subject, I should like to point out that the Press are large gainers by these quack advertisements, and how they reconcile it with their high standard of public morals I have not yet heard explained. Clearly, the advertising of false statements, often of a disgusting character, should be stopped by the authorities, for it is simply gaining money under false pretences.

Prescribing by chemists should also be made an offence, as being injurious to the community.

Screening for and management of obesity in adults in the USA

The prevalence of obesity in the United States is high, exceeding 30% in adult men and women. Obesity is associated with such health problems as an increased risk for coronary heart disease, type 2 diabetes mellitus, various types of cancer, gallstones, and disability. These comorbid medical conditions are associated with higher use of health care services and costs among obese patients.

Bearing in mind this background data and the fact that weight loss in the obese is associated with a lower incidence of health problems and death, the US Preventive Services Task Force have investigated this matter in depth.

The Task Force's conclusions are a recommendation for screening all adults for obesity: "Clinicians should offer or refer patients with a body mass index (BMI) of 30 kg/m² or higher to intensive multi-component behavioural interventions."

Ann Intern Med 2012;157:373–8.

Another look at the value of the ABCD² score in predicting the likelihood of a stroke after a transient ischaemic attack

The ABCD² stroke risk score was developed to identify transient ischaemic attack (TIA) patients with a high risk of early stroke requiring urgent management. It recommends points for age ≥60 years (A), blood pressure >140/90 mmHg (B), clinical features (C), duration (D) and diabetes (D). The higher the score the more likely a stroke will follow. Since its inception in 2007 it has been highly regarded and achieved widespread usage. Those subjects with a moderate to high score need urgent hospital admission, investigation and treatment.

This study from two Australian hospitals reviews 827 patients who presented to their emergency departments with a TIA. They report that there was no significant difference in proportion of strokes between those with a low or moderate-high ABCD² score at 30, 90 and 365 days. They also note that early stroke risk was low after an emergency diagnosis of TIA and significantly lower in admitted patients.

Int Med J 2012;8:913–18.

Angiotensin converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARB) and the risk of pneumonia

ACE inhibitors have secondary effects on the respiratory system which may protect against pneumonia, although various reports offer conflicting evidence. This systematic review included 37 studies. The researchers report that ACEi significantly reduced the risk of pneumonia compared with the control groups (odds ratio 0.66). However, ARBs did not reduce the risk.

The authors speculate that these data could discourage the withdrawal of ACE inhibitors in some patients with tolerable treatment-related adverse events, namely cough, who are at particularly high risk of pneumonia.

BMJ 2012;345:e4260.

Tofacitinib in active ulcerative colitis

Tofacitinib, an oral janus kinase inhibitor, has recently been reported to be helpful in the management of recalcitrant rheumatoid arthritis (see abstract [NZMJ 24/8/12](#)). This report concerns its use in another autoimmune disease which does not always respond well to the usual treatments—glucocorticoids, azathioprine, mesalamine and anti-tumour necrosis factor agents.

194 adult patients with moderately to severely active ulcerative colitis were randomised to a range of tofacitinib dosages or placebo for 8 weeks. Patients receiving the higher dose (10mg & 15mg twice daily) of tofacitinib were more likely to have a clinical response and remission than those on placebo. Adverse effects included increases in both low and high density lipoprotein cholesterol.

N Engl Med J 2012;367:616–24.

Thomas Derisley (Derry) Stuart Seddon

There is always an easy solution to every human problem—neat, plausible, and wrong (The Divine Afflatus, H L Mencken 1917)

Derry Seddon spent his life trying to influence and implement solutions that were neat, plausible and right.



He was born on 2 May 1932 in Wellington. He was a visionary leader who wanted to see the medical profession with a clearer understanding about both the ‘art of medicine’, that is, being patient centred, having empathy for patients and the vagaries of the human condition. He also wanted us to understand and use the power of ‘science in Medicine’ so that we might measure and reflect on the way we practice medicine and evaluate what we do. He saw medical practice as an ever-changing current of knowledge that is constantly being re-evaluated and re-written.

He wanted medicine’s journey to occur within a framework of self-development, using experience and measures of our practice to form the foundation of continuous improvement to the services we offer our patients.

He had an original mind that was uncluttered by the lesser goods of fame, fortune and other people’s assumptions. He was a free thinker, who felt passionately that the task of medicine was to serve the individuals and our practice populations. He questioned the assumptions of our medical profession and did not tolerate the shallow or the self-seeking.

He did not seek recognition but was driven to use his vision to argue, cajole, influence and create a momentum for change.

He was clear and outspoken in his constructive criticism of the *status quo* and always had useful, pragmatic suggestions for the way forward.

In his professional life, he led by example and confronted and implemented change at both local and national levels. He was highly regarded by his peers in New Zealand, United Kingdom, Canada and the USA.

He graduated MB ChB from Otago University in 1956 and entered solo general practice in Tauranga in 1960. In those first 10 years, he developed his practice, was a GP anaesthetist, and a GP obstetrician.

In 1970, he took on a partner, Dr Pat Hertnon, and between them they set up the Otumoetai Health Centre in 1975, eventually growing to a practice with seven partners.

This was a most unusual practice. Its architecture was determined by two significant pieces of evidence, the 1972 paper by Spitzer and Kergin on the randomised trial of nurse practitioners in Southern Ontario, and Lawrence Weed's seminal book, *Medical Records, Medical Education and Patient Care* 1969. The evidence created the core functionality of the Centre and the architecture housed and facilitated this new functionality by giving it form.

The practice nurses worked as nurse practitioners and the whole practice used type written problem-orientated notes with flow sheets to manage the long-term conditions.

Drs Seddon and Hertnon went on to establish Medical Data Processing, a company that sent in computerised GMS claims from practices. It also developed the capacity to act as a disease register and was used for the introduction of capitation funding at the Otumoetai Health Centre.

It was typical that Derry Seddon not only introduced capitation but also insisted that the new model of funding be evaluated. He obtained funding and evaluated the change over a 3-year period, 1979–1982. The evaluation was published in 1985 as an occasional paper (25) by the Department of Health. (*Capitation Funding in a New Zealand General Practice*—It was co-authored by his daughter, Bridget Daldy and Judy Reinken.)

Twenty years later, capitation became the new funding mechanism for general practice. In combination with enrolment it delivered the denominator for measuring, activity, disease burden, outcomes, referral rates, understanding of population medicine.

In short, having a denominator in the primary sector was the pre-condition required for quality and performance measures and from there to accountability.

From 1986 to 1989, Derry was the Chairperson the Royal New Zealand College of General Practitioners. During his time in office, working with Dr Tessa Turnbull, they set in motion the College's move towards quality and re-accreditation.

He understood the link between, measurement, information, quality and accountability and started the journey towards bringing these together.

Derry Seddon's legacy to the New Zealand health system should not be underestimated. He developed an integrated health centre long before the term itself was thought up; facilitated nurse practitioners in general practice, understood the limitations of a fee-for-service funding structure and introduced a capitation funding mechanism 20 years before the rest of the profession. He was patient-focussed and patient-centred, and importantly he understood the power of measurement for improvement.

He was frustrated, after he retired, by the continuing resistance to change and by the failure to deliver the health system supported by evidence that would support the people of New Zealand.

He wrote to the *New Zealand Herald* in response to an editorial that asked: Why we are not doing better with our health system?"

He responded based on his life's experience of trying to address this question. His observations remain cogent and relevant now.

...The medical professional organisations, including the medical schools, can be described as sclerotic. Other commentators are pointing out the need for them to change, not to another steady state but to become flexible, relevant and fitting the rapidly changing knowledge and technology of today's health sector.

Several of the blocks to change continue to exist because there has been no consistent and continuing effort to collect information on what is happening, what are the needs and what are the effects of changes to the system. Beliefs can be stated, firmly held and introduced; simple solutions are suggested for complex issues; and new services push aside those existing with no realisation of the consequences.

It is essential that the health service has a widespread public information system that allows for continuing evaluation of services and needs.

In addition, he suggests an Academy of Medicine:

...In 1988, an effort was made to establish an academy of medicine, an organisation that would be an open forum for all, suppliers and consumers, where options were debated and hence could supply broad-based advice to its members, the Government and the ministry.

It would be an organisation that could insist on, and commission, data-gathering and its analysis and encourage and measure new methods of healthcare delivery.

Such an organisation would provide the political parties and the Minister of Health with non-partisan advice and information.

There was no interest in the concept. The reaction was defensive and dismissive, typical of the response to any suggestion for changes. The lessons of the 1990s may now be sinking in, and this idea should be revisited.

It would go a considerable way to relieving all the puzzlement, while making the health service one that serves us better.

Derry Seddon's legacy is encapsulated by his actions and influence and maybe by our addressing the issues embedded in these last paragraphs.

He married Jenny in 1953 and they had 7 children, 15 grandchildren, and 6 great grandchildren. His daughter, Mary, is a doctor at Middlemore Hospital and her daughter, Kate, is going to read health sciences and medicine in Dunedin.

Derry Seddon purchased a farm outside Katikati in his retirement from practice. It was managed by his son-in-law, Rick Burke, and became an award-winning farm that excelled in its innovation in the area of land use and conservation.

He planted several thousand kauri and rimu trees, fenced off the waterways and retired established bush into the QEII National Trust.

He was a man with many facets, many talents and many frustrations and will be sadly missed.

Jonathan Simon (GP in West Auckland who worked with Derry in Tauranga from 1982 to 1998) wrote this obituary.

John Burd Carman

8 November 1932 – 11 August 2011; Emeritus Professor, MBChB, BMedSc, DPhil (Oxford); University of Auckland Medical School

John Carman, Foundation Head of Department of Anatomy at the Auckland University School of Medicine, died suddenly on 11 August 2012. He had celebrated his 80th birthday a week earlier in wonderful spirit with family and friends.



John was born in Johnsonville, he and twin Len, two of four boys to Marjorie, and Walter Carman, director of Wright and Carman, printers, of Wellington.

John's love for the science and art of anatomy began at a very young age when he chose a book on anatomy as his prize for becoming Dux at his primary school.

John, and brother Len, were educated at Nelson College where John excelled academically and learned to play the flute and with Len played in the College orchestra, forming his love of music.

John chose to study medicine, also achieving a BMedSc research degree in anatomy and later graduating MBChB at Otago University.

While there, he was awarded a Nuffield Fellowship and spent 3 years at Oxford where in 1961 he gained his D.Phil for his research in neuroanatomy on the cerebral cortex under the supervision of a leading anatomist of the day, Sir Wilfred Le Gros Clark.

He took up his appointment as the Foundation Professor in Anatomy in 1968 at the young age of 35. His impact on the new medical school at Auckland extended beyond anatomy and in those early days he put his abiding interest in engineering and functional design to practical use, playing a leading role in the management and design of the new Medical School buildings.

In 1985 John and Mary were married and thereupon he became a very happy and proud Poppa to Adrian, and Suzi and Peter, and wise Grandpa to Jessica, Tom and Zoe all of whom he loved as his own family.

In 1988 John retired from the headship of the department of anatomy after 20 years and continued to pursue his varied research interests until his retirement in 1998. Before he retired, John and Will Richardson, Renaissance Latinist in the Auckland Classics department, had begun the co-authorship of a 'major opus' to translate from the Latin "*De Humani Corporis Fabrica*" (On the Fabric of the Human Body"), written by the Belgian anatomist Andreas Vesalius and published in 1543. Their work has been recently published in English in five volumes, where John was able to demonstrate his depth of knowledge of anatomy and his understanding of Vesalius himself. *Nature* in 1988, reviewed the first of the five volumes, saying that it was "one of the publishing, scientific and literary achievements of the decade".

John was an inspirational Head of Department who made great contributions to the Faculty and the University by his work on committees and the MRC and also to the Auckland Biomedical Engineering Institute in assisting research in applying engineering principles to the form and function of the human skeleton, and particularly the head and neck. His input was recognised in 2010 by the Biomedical Institute establishing their annual “John Carman Prize” for excellence in biomedical research.

John also contributed to anatomy internationally over the years with many publications, and was inspirational to so many students and staff in a well-recognised world-class department of Anatomy. John had that wonderful combination of scientific logic, literary excellence and human understanding, and students and staff alike over many decades benefited from his mentorship and wisdom.

Many learned from John how to communicate science through clear and expressive writing, concise and accurate yet never dry or stilted. He would remind us that where understanding fails “words rush in”.

John was also an artist able to draw the intricacies of aircraft and car design, while his skill and precision at anatomical drawing and demonstrating with chalk at the blackboard were renowned. Those fortunate enough to be taught by John will long remember and be thankful for his wonderfully insightful lectures.

John leaves behind a grieving family and many friends all over the world with the memory of an endearing man of gentleness, intellect and integrity.

The University community has lost one of its most distinguished members but his teaching and his wisdom will continue through his students.

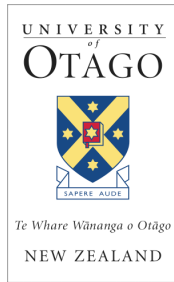
Professor Richard Faull (Neuroscientist, Department of Anatomy and Centre for Brain Research, University of Auckland Medical School) wrote this obituary.

Medical Benevolent Fund

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

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161



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