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This issue in the Journal

Older patients with hip fractures: evaluation of a long-term specialist orthopaedic medicine service in their outcomes
J Thwaites, F Mann, N Gilchrist, J McKie, R Sainsbury

All patients over the age of 65 years with orthopaedic injuries are now admitted under the shared care of both a physician and orthopaedic surgeon in Christchurch—the Ortho-Medicine Service which was implemented in 2002. An initial study by the same authors found shared care resulted in improved inpatient mortality with the majority of patients returning home and improved treatment of osteoporosis compared with previously. This paper describes the outcomes of those same patients who were discharged home and (where possible) followed-up at 12 months with a questionnaire. Shared care appeared to be associated with a reduced 1-year mortality (although still 18.8%), improved treatment of osteoporosis, and ability to return and remain at home. Many patients, however, continue to be significantly impaired in performing activities of daily living and have impaired mobility 12 months following their injury.

Management of scleroderma in a New Zealand tertiary rheumatology centre: emphasis on pulmonary complications
K Ng, P Gow

Scleroderma is a rare, chronic disease characterised by excessive deposits of collagen in the skin or in other organs. There are two forms of scleroderma: diffuse and limited. Diffuse scleroderma is the most severe form—it has a rapid onset, involves more widespread skin hardening, will generally cause much internal organ damage (specifically the lungs and gastrointestinal tract), and is generally more life-threatening. In this study we determined the current local practice for managing scleroderma patients, with emphasis on screening patients who have lung disease associated with this condition. All scleroderma patients in Middlemore Hospital (South Auckland) were identified from 1999 to 2004. We found that a higher proportion of diffuse scleroderma patients had investigations for lung disease compared to the limited group, although lung disease can occur in both groups. Scleroderma lung disease has generally been poorly screened in our group of patients—largely due to lack of availability of specific treatments in New Zealand. A standardised screening and monitoring protocol may help identify patients with progressive lung disease.

Exploring general practitioner identification and management of psychosocial Yellow Flags in acute low back pain
C Crawford, K Ryan, E Shipton

Over the past decade, psychosocial issues have been increasingly identified as risk factors that are associated with the development of chronicity and disability. These psychosocial risk factors are known as *Yellow Flags*. In New Zealand, in 1997, the Accident Compensation Corporation (ACC) published the *Acute Low Back Pain Guide* and (together with the National Advisory Committee on Health and Disability) the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain*. This study tried to understand the experiences of general practitioners (GPs) in the identification and management of psychosocial *Yellow Flags* in patients with acute low back pain. For various reasons, it was found that GPs did not use the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain* or the screening questionnaire to identify psychosocial risk factors in their patients with low back pain. More investment of resources in GPs to empower them to be effective gatekeepers and guard against the development of chronic illness becomes imperative. The current way that ACC disseminates and implements Guidelines need to be changed as well.

Practitioners, patients, and their visits: a description of accident and medical (A&M) clinics in New Zealand, 2001/2
P Hider, R Lay-Yee, P Davis

Accident and medical clinics are a recent development in New Zealand. This research provides unique information about the characteristics of the accident and medical clinics, the practitioners who work in the clinics, as well as data about the patients and their visits based on information gained in the National Medical Care Survey undertaken in 2001/2.

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Hip fracture management—we need to do better

Geoffrey Horne

The article in this issue of the *Journal* serves to emphasise the increasing problem of hip fractures in our already stretched public healthcare system (Thwaites et al. *Older patients with hip fractures: evaluation of a long-term specialist orthopaedic medicine service in their outcomes*; http://www.nzma.org.nz/journal/120-1254/2535)

Any condition with a 1-year mortality rate approaching 20% and a very significant morbidity rate demands that we make every effort to reduce the impact of the condition on society.

Hip fracture mortality has fallen in the last two decades (probably as a result of better perioperative management), but families are often startled when advised that there is a minimum 20% risk that their loved one will not survive beyond 12 months.

Unfortunately with the ageing population we may not be able to reduce the mortality rate. However many units report a higher mortality rate[1] and New Zealand (NZ) units with a 12-month mortality rate higher than 15–20% need to examine their perioperative protocols .

Perhaps the greater challenge is to address the issue of the morbidity that results from hip fractures. In Thwaites' study only 48% of patients regained their pre-fracture level of mobility by 12 months and, at the time of discharge, 56% needed a walking frame.

In a study of 208 hip fracture patients followed for 4 months, only 18% of surviving patients had reached their pre-injury functional level.[2] The impact of the fracture on subsequent rehabilitation is not gender-specific[3] although these fractures are approximately 2–2.5 times more common in females than males in NZ.[4]

The value of a team approach to the rehabilitation of hip fracture patients to reduce morbidity has been demonstrated by many authors.[5,6] So Thwaites' study of a hip fracture population under the shared care of orthopaedic surgeons and geriatricians establishes a dataset that others working in this field in NZ may use as a benchmark to gauge the effectiveness of their management protocols.

The role of post hip fracture assessment and treatment of osteoporosis in reducing the subsequent risk of a second fragility fracture is well recognised[7,8] but may not be widely utilised in NZ.

Issues preventing the widespread adoption of such assessment and treatment include a lack of "ownership" of osteoporosis in NZ, concerns regarding access to (and funding of) Dexa scanning, and funding of appropriate treatment should osteoporosis be diagnosed.

Accident Compensation Corporation (ACC) might have been expected to have shown leadership in this area to reduce the subsequent fracture burden and thus their costs, but as yet this is not evident. Considerable effort has been made under the guidance of the Bone and Joint Decade to increase awareness of this issue, but it is likely to be some time before all patients are appropriately screened and treated.

Hip fracture numbers in NZ were predicted to double between 1987 and 2011,[4] although more recent analysis suggests that the increase may be slightly less than predicted.[9]

Of concern, the annual cost of this increasing burden has been estimated to be almost $NZ37 million by 2011.[10] Thus it is essential that all hospitals treating patients with a hip fracture look carefully at their management methods with a view to improve the perioperative and rehabilitation process and thus minimise the mortality ,morbidity, and cost.

Failure to do so will mean an increasing percentage of "Vote Health" will be consumed by this group of patients, and patients may be at risk of not achieving their full potential following the fracture.

Competing interests: None.

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Fees in primary care

James Reid

The letter in this issue of the *Journal* from Dr Bill Douglas of Wanganui outlines very real difficulties that are arising in the fees-setting process (or should this read fees-regulation process?) in New Zealand—Douglas B. *General Practice Fees Review Committees* [letter]; http://www.nzma.org.nz/journal/120-1254/2540.

The current LECG* committee has recommended a maximum annual fees increase of 2.4%, based on a current total "ordinary" fee of $57. This begs the question—what if the total "ordinary" fee is below $57? What happens if a practice that is not charging for under 6s suddenly charges $5? This process comes at a time of unprecedented increase in practice overheads.

The letter and the outcome of the review were sent to this *Journal* on 1 April 2007. The Editor's first comment was "is this a joke". Unfortunately it is not.

The proposed increase from Dr Douglas was not seen as fair and reasonable for the patients. There is no comment about what is fair and reasonable for the doctor. They saw no evidence of unusual cost increases in spite of an 18% increase in staff wages. As wages make up the majority of practice expenses, it is extraordinary that 18% is not regarded as unusual.

The LECG recommendation makes no allowance for any increase in a doctor's remuneration. Sadly, in comparison with their hospital colleagues' remuneration, general practitioners (GPs) are the poor cousins. Currently with 5 weeks' holiday a year, provision for study leave, often superannuation, and a salary in the vicinity of $160,000, senior hospital doctors are, in comparison to GPs, "on the pig's back". And a further wage round is impending.

There is currently a recruiting drive from across the Tasman in Australia to recruit GPs. For instance, this author has received a number of brochures, and personal contact from a recruiting agency offering a very attractive package of salary of $300,000, assistance with housing, and a provision of a vehicle. In comparison to this, New Zealand GPs can only dream.

With the increase in TEC funding for medical schools, universities are being extolled by Government to increase the number of GPs and rural doctors. But surely it is a two-way street. While it is the responsibility of the universities to produce graduates for all disciplines, it is also the responsibility of Government to make a discipline (including general practice) attractive enough to attract young doctors and to retain those already in practice.

New Zealand graduates are of an excellent calibre and are keenly sought on the international market. While it is frequently stated that New Zealand cannot afford salaries that are competitive with international rates, this seems to be a selective statement with respect to medicine. The same does not apply to other vocations.

The *Primary Health Care Strategy* arrived in 2001, and with it came a significant increase in funding which quite reasonably was required to be passed on to patients.[1] Unfortunately, up until then, some GPs had been working on ridiculously low incomes. Current data suggests that due to low morale in general practice 20% intend to leave to workforce within 5 years.[2]

According to the Auditor General's report of 2003, "Government has been trading on the goodwill of primary care doctors".[3] Many still work in this way, personally providing subsidy to under 6-year-olds by seeing them without fee, thus maintaining the "free to under 6s" introduced about 10 years ago. This essentially subsidises the primary health care system.

Fees should not be a barrier to consulting a doctor but, on the other hand, a GP needs to have an adequate financial return for being in the high achiever group at university, for the 11 years (at least) of study before being able to practice independently, and for providing a service that patients perceive as being at least highly satisfactory. As general practice supplies a subsidised service, it is up to Government to quantify this. If the subsidy is insufficient, it should say so, and should not pass on the blame.

Government feels that constraint is necessary with primary care fees, yet in the past the sector has acted responsibly—the example of provision of free care to under 6s is a continuing example of this. One must ask, therefore, is it really necessary to negatively single out a sector of the healthcare workforce that so regularly scores high satisfaction ratings from patients? To do so will have a very negative effect on recruitment and retention.

Surely commonsense must prevail and if a practice's fees are grossly out of line with the recognised "norm" then a review is appropriate. But the current "routine" review for all rises above the recommended 2.4% on total fee, no matter if the original fee was much below the norm for the region, is bureaucracy gone mad.

The cost to practices will be considerable. In this author's region (the southern South Island), the author knows of four GPs who have walked away from their practices in recent months. Without a change of policy from the DHBs and the Ministry, there will be more—this author included.

*LECG is a global expert services firm that provides expert analysis, testimony, authoritative studies, and strategic advisory services to clients.

Competing interests: The author's general practice is currently undergoing a fees review.

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Fees reviews of New Zealand general practices—a New Zealand Medical Association (NZMA) perspective

Peter Foley

The NZMA absolutely supports the right of general practitioners (GPs) to set fees commensurate with the services they offer. As government subsidies do not cover 100% of the fee, neither should the Government have 100% control over levels of fee increases.

We have welcomed the increased focus on the role of primary care in our New Zealand health scene, but not the increasing desire of the Government to control the business of general practice. The current year has created particularly high inflationary pressures that GPs must take into account when reviewing their financial viability.

Last year we talked of a more mature relationship with the Ministry of Health (MoH) and district health boards (DHBs), based on ideals such as 'good behaviour' and 'trust'. The insistence of DHBs, at MoH direction, to decline to exercise commonsense and discretion when considering invoking a fees review makes a mockery of any intention to move to this more mature and less hostile environment.

The LECG report does not allow for many of the factors that are creating inflationary costs in general practice. Our GPs must not feel compelled to adhere to any guideline that compromises the ability of their practice to continue to provide high-quality care to their patients.

Obviously, good business practice requires that the setting of patient fees occur in the context of fully reviewing all costs, including the ability to reduce some of these costs.

We are surprised that more practices are not yet going through this fees review process. It seems that the MoH's inflexible approach has surreptitiously achieved its aim—that of suppressing legitimate fee increases. This is not what the NZMA agreed to, as part of the GP Leaders' Forum, last year.

Our GP workforce has a proven track record of passing on increases in patient subsidies, so do we really need this expensive and soul-destroying distraction?

I would hope that the Government will recognise that the provision of general practice services in New Zealand is a partnership in which the private GPs are not subservient to our DHB funders. These DHBs should be striving to endorse a positive relationship with their primary care providers, not alienating them.

Competing interests: None.

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Older patients with hip fractures: evaluation of a long-term specialist orthopaedic medicine service in their outcomes

John Thwaites, Fazal Mann, Nigel Gilchrist, John McKie, Richard Sainsbury

Abstract

Aims To evaluate the long-term outcomes of a specialist orthopaedic medicine service in older patients up to 12 months after hip fracture.

Methods All patients over the age of 65 years admitted with hip fracture under the shared care of geriatricians and orthopaedic surgeons over a 6-month period were identified in an initial audit. A follow-up postal questionnaire was sent to those patients asking about their place of domicile, level of functioning, compliance with osteoporosis treatment, and whether they had sustained further fractures in the 12 months following discharge from hospital. Mortality was also recorded.

Results The 1-year mortality of the 149 patients discharged from hospital following their hip fracture (who were identified in the initial audit) was 18.8%. There were 69 (46.3%) responses to the questionnaire. The mean age of respondents was 81.3 years (range 66–98 years).

At discharge, only 5 of 69 (7.2%) patients were independent in their walking, 13 (18.8%) walked with the aid of a stick, 39(56.5%) with a frame, 7(10.1%) required supervision, and 5 (7.2%) were immobile. Excluding those who were immobile prior to their hip fracture, 31 of 64 (48.4%) of patients regained their pre-morbid level of mobility at 12 months.

At discharge, 27 of 69 (39.1%) patients were independent with activities of showering, dressing, and toileting—with 42 of 64 (65.6%) independent at 12 months.

At discharge, 57 of 69 (82.6%) patients were on calcium and vitamin D, and 5 (7.2%) on alendronate. At 12 months, 50 of 64 (78.1%) remained on calcium, 40(62.5%) on vitamin D, and 26(40.6%) on alendronate.

Five of 64 (7.8%) patients experienced a total of 11 further osteoporotic fractures at 12 months, but no further hip fractures.

Of respondents discharged home, 44 of 50(88%) remained at home at 12 months.

Conclusions Shared care between geriatricians and orthopaedic surgeons for older hip fracture patients appears to be associated with a reduced 1-year mortality, improved treatment of osteoporosis, and return to home. Many patients, however, continue to have impaired function and mobility.

Hip fracture in older people significantly impacts on morbidity and mortality, and increases the likelihood of subsequent hospitalisation[1] and long-term institutional care.[2] The 12-month mortality for hip fracture patients is over 25%,[3–5] with significantly decreased mobility and function compared with pre-fracture status.[4,6,7]

To address the needs of older patients with hip fractures and other orthopaedic injuries in Christchurch, shared inpatient care between geriatricians and orthopaedic surgeons ('the Ortho-Medicine Service') was implemented for all older people admitted with orthopaedic injuries in December 2002.

All patients over the age of 65 years with orthopaedic injuries are now admitted under the shared care of a physician and orthopaedic surgeon, with a full-time medical registrar based on the acute orthopaedic wards in addition to the orthopaedic resident medical officers. The physician's 'ownership' of the patient's medical care in conjunction with the orthopaedic team, and good interdisciplinary care are seen as critical elements of the new service.

A retrospective case records audit of all (150) patients over the age of 65 years with hip fractures admitted during the first 6-month period (following implementation of the Ortho-Medicine Service in December 2002) showed a low inpatient mortality, with the majority of patients returning to their pre-morbid place of domicile. Most patients were discharged on treatment for osteoporosis.[8]

We conducted a further retrospective study to determine whether the positive short-term effects of a specialist orthopaedic medicine service resulted in improved long-term mortality, morbidity, and treatment of osteoporosis.

Methods

All patients over the age of 65 years with hip fracture admitted and discharged to the Ortho-Medicine Service at the Canterbury District Health Board during the 6-month period from December 2002 to June 2003 were identified from the initial retrospective study.[8]

Ethical approval was granted by the Upper South B, South Island, Regional Ethics Committee for the questionnaire and research.

Mortality data was accessed through the national database, with the assistance of the hospital medical records department, to determine who had died within this 12-month period.

A questionnaire—including questions covering place of domicile, level of functioning, mobility status, compliance with osteoporosis treatment, and whether further fractures had occurred in the 12 months following discharge from hospital—was sent to all those patients still living. This questionnaire was also sent to relatives/caregivers/whānau (extended family) of those who were identified as being unable to provide consent. All were asked to give their informed written consent.

Patient information was entered into a dedicated database using Microsoft Access software, including patient demographics, place of domicile, mobility and function at 6 and 12 months, osteoporosis treatment, the occurrence of further fractures, and mortality.

Results

Mortality—Of the 149 patients discharged from hospital following their hip fracture identified in the initial audit, 19 (12.8%) had died within 6 months and a further 9 (6.0%) had died by 12 months, with an overall 1-year mortality of 18.8%. The 12-month mortality by gender was 8 of 34 males (23.5%) and 20 of 115 females (17.4%).

Patient characteristics—Of the 149 patients identified from the initial study, 69 (46.3%) responded to the questionnaire. Five patients of the 69 had died within the 12-month period post-discharge (3 by 6 months and 2 by 12 months after discharge), with relatives/caregivers/whānau completing the questionnaire on their behalf. Therefore, follow-up data was available for 69 patients at discharge, for 66 after 6 months, and for 64 after12 months respectively.

The mean age of the 69 respondents was 81.3 years (range 66–98 years); comprising 51 (74%) females and 18 (26%) males.

Place of domicile—Of the 53 respondents who lived at home pre-fracture, 51(96.2%) had been discharged back home, and 2 (3.8%) went to rest-home care. Of the 12 patients admitted from rest-home and 4 patients from hospital-care pre-fracture, all were discharged back to their same level of care.

Of the 51 patients discharged home, 44 (86.3%) remained at home at 6 months and 44 of 50 (88.0%) at 12 months, with 1 patient having died. The remaining 6 patients were discharged to a higher level of care.

Of the 14 patients discharged to rest-home care, 3 had died by 6 months. Of the remaining 11, 7 remained in rest-home care at 12 months with 3 having gone to hospital level of care. One patient had returned home.

Of the 4 patients discharged to hospital level of care, 3 remained at hospital level of care at 12 months and 1 patient had died.

Of those patients who were living at home pre-fracture, 44 of 53 (83.0%) were still living at home after 12 months.

Mobility—Level of mobility from before and after hip fracture is shown in Table 1.

Excluding those who were immobile, only 31 of 64 (48.4%) patients regained their pre-morbid level of mobility after 12 months.

Table 1. Mobility compared before and after hip fracture

Mobility	Pre-admission N=69	Discharge N=69	6 months N=66	12 months N=64
Independent	39 (56.5%)	5 (7.2%)	18 (27.2%)	21 (32.8%)
Stick	11 (15.9%)	13 (18.8%)	13 (19.7%)	10 (15.6%)
Frame	15 (21.7%)	39 (56.5%)	25 (37.9%)	22 (34.4%)
Supervision	1 (1.5%)	7 (10.1%)	5 (7.6%)	3 (4.7%)
Immobile	3 (4.4%)	5 (7.2%)	5 (7.6%)	8 (12.5%)

Activities of daily living (ADL)—At discharge, 27 of 69 (39.1%) patients were independent in activities of showering, dressing, and toileting. After 6 months, 39 of 66 (59%) patients were independent in activities of showering, dressing, and toileting. After 12 months, 42 of 64 (65.6%) patients were independent with activities of showering, dressing, and toileting.

At discharge, 14 of 69 (20.3%) were independent in showering, dressing, and toileting plus at least one instrumental activity of daily living, at 6 months 33 of 66 (50%) and at 12 months 37 of 64(57.8%) were independent in showering, dressing and toileting, plus at least one instrumental activity of daily living.

Further fractures—In the first 6 months 2 patients sustained a vertebral fracture, 1 patient sustained a wrist fracture, and 1 patient a vertebral and wrist fracture. At 12 months, 2 patients (who had already sustained fractures) had a further wrist and vertebral fracture respectively. One further patient sustained a wrist fracture and another a wrist, vertebral, and clavicle fracture. No patient, however, suffered a

further hip fracture in the 12-month follow-up period. This represents a total of 11 new fractures in 64 patients (17.2%), with vertebral fractures the most common.

Osteoporosis treatment—At discharge, 57 of 69 (82.6%) patients were on calcium and vitamin D (in the form of calciferol or multivite tablets containing vitamin D as per the local osteoporosis treatment protocol); 5 (7.2%) on alendronate; and 3 (4.3%) on etidronate.

At 6 months, 52 of 66 (78.8%) respondents were on calcium, 40 (60.6%) on vitamin D, and 26 (39.4%) on alendronate, 3 on etidronate, and 2 on hormone replacement therapy (HRT).

At 12 months, 50 of 64(78.1%) patients were on calcium, 40 (62.5%) on vitamin D, 26(40.6%) on alendronate, 3 (4.7%) on etidronate, and 2 on HRT.

Thirty-three of 64 (51.6%) respondents had undergone a bone density scan in the 12 months following discharge.

Discussion

In this retrospective audit of 149 patients following hip fracture, overall mortality was 18.8% at 12 months. This compares favourably with the previously reported 1-year mortality of over 25% in hip fracture patients in Christchurch over the age of 79 years.[3]

A recent study by Young et al,[9] also in Christchurch, showed a 12-month mortality of 32%—higher than our study. This possibly reflects the different study population, retrospective identification from operating lists, age groups, and timeframe of that study. In our study, all patients over the age of 65 admitted to the Ortho-Medicine Service with a hip fracture within the defined 6-month period were followed up with respect to mortality. The higher mortality (23.5%) for males in our study is consistent with other studies showing a higher 1-year mortality in men.[2,7,10]

The response rate to the questionnaire was lower than expected at 46.3%, although this is higher compared with a recent Australian postal questionnaire to elderly patients with fractures where only 23% responded.[11] The response rate may have been improved by direct patient contact.

Hip fracture has a significant impact on mobility. Almost all patients experienced a significant decline in mobility after hip fracture, with only 7.2% of patients independently mobile at time of discharge from hospital.

At 12 months, only 32.8% were independent compared with 56.5% pre-admission. Less than half (48.4%) of the respondents had regained their pre-morbid level of mobility at 12 months.

The negative impact on mobility following hip fracture is well described.[6,10] For instance, Edelstein et al[12] found only 45% of patients regained their pre-fracture ambulatory ability while Kitamura et al found 67% recovered to their pre injury ambulatory status.[13] In a younger group of patients with hip fracture, those able to walk without aids fell from 59% to 26% at 1 year.[4]

Following hip fracture there is a decline in ability to perform personal cares. In this study, only 39.1% were independent in personal cares at discharge but this had improved to 65.6% at 1 year.

Several studies have shown the negative impact on functional activities of daily living at 12 months following a hip fracture.[4,12,14] For instance, Edelstein et al[12] found only 69% had regained their pre-fracture level of independence in basic activities of daily living at 12 months. Although our questionnaire recorded other instrumental activities of daily living (including cooking, cleaning, and shopping) these were often covered through external assistance (including personal and domestic assistance and the 'meals on wheels' service) and were therefore excluded from analysis.

The majority of patients in this study had been discharged back to their pre-morbid place of domicile, with the majority going home. Of those patients who were living at home pre-fracture, 44 of 53 (83.0%) were still living at home at 12 months. This compares with Rosell and Parker's[4] study, where 68.7% of those admitted from their own home had maintained that residential status at 12 months (88.3% of survivors). In a large Japanese study which included younger patients, 86% of patients were residing at home at 12 months.[13]

Treatment of established osteoporosis decreases the risk of fracture. Daily supplementation with cholecalciferol and calcium was found to substantially decrease the risk of hip fractures in elderly women living in rest homes,[15,16] although efficacy remains an issue.[17,18]

Furthermore, hip fracture risk can be significantly reduced with oral bisphosphonates; namely, alendronate[19] and risedronate.[20] Recent pooled data from risedronate studies demonstrated efficacy in women aged 80 and older, with an 81% reduction in new vertebral fracture within 12 months with the effect remaining consistent over 3 years.[21]

In our study 82.6% patients were discharged on calcium and vitamin D in the form of calciferol or multivite tablets containing vitamin D (as per the osteoporosis treatment protocol in Christchurch) with 7.2% on alendronate and 4.3% on etidronate. In a recent study of older patients in Western Australia following discharge with a fracture (mostly hip fractures), only 37% were on osteoporosis treatment at time of discharge.[11]

Importantly in our study, 78.1% were on calcium at 12 months, 62.5% on vitamin D, 40.6% on alendronate, and 4.7% were on etidronate. This compares very favourably with previous local data where (at 12 months) only 12% were on calcium; 9% on bisphosphonates, HRT, or calcitriol; and 79% on no treatment.[4] This improvement in treatment and compliance probably reflects the osteoporosis treatment protocol for hip fracture patients resulting in greater awareness of the management of osteoporosis.

The higher percentage of patients on alendronate at 12 months reflects access to special authority approval by having had a bone density scan. Indeed, subsequent to this study, PHARMAC now allows some patients with a significant osteoporotic fracture to receive approval for alendronate without a bone density scan if they are elderly; or if major logistical, technical, or pathophysiological reasons make it difficult for a patient to have a scan. This change should allow a higher percentage of patients being commenced on alendronate at time of discharge from New Zealand hospitals.

Limitations of this study include the delay in sending out the questionnaire to patients, a smaller percentage of respondents than had been anticipated, and overall small

numbers. The delay in questionnaires being sent out may have resulted in some inaccuracy of self-reporting due to the time elapsed. In addition, the number of respondents was too small and the data collated insufficient to identify any factors predictive of outcome for mobility, functional status, or mortality.

Many hip fracture studies have different aims and measure different outcomes.[22] This is in some way being addressed with the establishment of the Hip Fracture Pathway and Database in Christchurch which will allow a consistent approach to collation of data on all patients with hip fracture admitted to the Ortho-Medicine Service.

However a much larger prospective community-based study is needed to accurately identify outcomes locally for this group of patients for whom a hip fracture results in significant morbidity. This will enable effective future planning and resourcing to ensure these patients are being provided with the necessary support, rehabilitation, and care in our community.

In summary, this audit provides some long-term data on outcomes of older hip fracture patients managed by the Ortho-Medicine Service in Christchurch. Mortality appears to be reduced at 1 year. Treatment for osteoporosis at 12 months has improved. Hip fracture in older patients results in considerable impairment of mobility and function seen at time of discharge. Despite showing improvement at 6 and 12 months, many patients remain impaired with function and mobility. The majority of patients remain at their discharge place of domicile at 12 months.

Competing interests: None.

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Management of scleroderma in a New Zealand tertiary rheumatology centre: emphasis on pulmonary complications

Kristine Ng, Peter Gow

Abstract

Aims To determine the current local practice of managing scleroderma (SSc) patients, in particular screening of SSc-related lung disease in a tertiary rheumatology centre.

Methods SSc patients were identified from our inpatient (July 1999 till June 2004) and outpatient (January 2002 till June 2004) databases. Patient demographics and relevant investigations performed to monitor for pulmonary, renal, and cardiac complications related to SSc were sought from computerised clinical and laboratory records.

Results Nine of the 39 (23%) limited SSc (lcSSc) patients and 5 of the 10 (50%) diffuse SSc (dcSSc) patients had lung involvement. A higher proportion of diffuse SSc patients had investigations for SSc lung disease compared to the lcSSc group (90% vs 67% had pulmonary function tests and 70% vs 56% had high resolution chest CT scans respectively). About half of the patients in both groups had echocardiographs (50% lcSSc vs 46% dcSSc) for assessment of pulmonary arterial hypertension.

Conclusion SSc lung disease has been generally poorly screened in our cohort of patients with SSc. Limited SSc patients were not screened as rigorously as dcSSc patients for SSc lung disease. In large part, this was because of the lack of availability of treatments in New Zealand when the lung disease was identified. The generation of a standardised screening and monitoring protocol may help identify patients with progressive lung disease so that early treatment could be considered as this becomes more readily available.

Systemic scleroderma (SSc) is a rare autoimmune rheumatic disease with the potential for multiorgan system involvement. It can be categorised to two major groups, limited (lcSSc) and diffuse (dcSSc) cutaneous SSc, depending on the extent of skin involvement. A recent meta-analysis has found that the involvement of gastrointestinal, lung and renal organ systems are important predictors of mortality.[1]

Lung disease occurs in more than 70% of SSc patients and is the second most frequent organ system involved. The two main clinical manifestations of lung disease in SSc patients are interstitial lung disease (SSc-ILD) and pulmonary vascular disease leading to pulmonary arterial hypertension (PAH).

Lung disease is now the leading cause of death in patients with SSc.[2] The prognosis of these patients is poor with a 70% mortality at nine years.[3] The prevalence of PAH in SSc varies from 10-40%[4,5] and is more common in lcSSc. PAH is an independent poor prognostic marker in dcSSc regardless of the presence of SSc-ILD.[6]

A large proportion of SSc patients have sub-clinical pathology, and symptoms are often an unreliable marker in determining the presence of pulmonary disease.

Pulmonary function tests (PFT), chest X-ray (CXR), and high resolution computed tomography (HRCT) chest scans are routine investigations used to diagnose and monitor lung disease in SSc patients. PFT, in particular diffusing capacity for carbon monoxide (DLCO) is the most sensitive test to detect a functional decline in SSc patients.[7]

A reduction in DLCO in ILD (< than 65% normal) is usually associated with a restrictive pattern on PFT. However, PFT and CXR are relatively crude tests to detect SSc-ILD. HRCT chest is useful to identify early lung disease and to distinguish reversible ground- glass opacification and irreversible fibrotic disease.

Bronchoalveolar lavage (BAL) has shown to be of prognostic value in several studies[8,9] but there is a wide variability in technique and reporting of BAL differential cell counts depending on the training of local personnel.[10] Specialised SSc units generally recommend that PFT and HRCT chest be done at baseline and PFT to be repeated periodically, at more frequent intervals in early disease, and this can be extended to yearly if results are normal and the patient is asymptomatic.

Echocardiograph remains the primary screening tool for PAH, especially if the DLCO is extremely low with near normal forced vital capacity (FVC).[11] However, the sensitivity and specificity of this test is variable depending on the patient population studied and the definition of a "positive" echocardiography result.[12] Right heart catheterisation remains the gold standard to diagnose PAH. Annual echocardiography is recommended as a routine systematic screen for PAH in SSc patients.[13]

The early diagnosis of SSc-related pulmonary involvement is essential for prompt treatment before irreversible damage occurs especially with recent major pharmacotherapeutic advances in the treatment of PAH.[12]

This audit was undertaken to determine the local practice of managing scleroderma patients at Middlemore Hospital, Auckland with emphasis on the screening strategies used to detect pulmonary, renal and cardiac organ involvement. We also discuss the relevance of available evidence based practice for the management of patients with SSc lung disease in New Zealand.

Methods

Middlemore Hospital is a tertiary rheumatology referral centre for the South Auckland population in New Zealand. The SSc patients were identified from our outpatient rheumatology database at Middlemore Hospital from January 2002 till June 2004. The rheumatology outpatient database included all patients with the read code "collagen tissue not otherwise specified". We also identified patients from the inpatient admission database from July 1999 till June 2004. Patients with overlap syndromes, Sjogrens disease, undifferentiated connective tissue disease, and myositis were excluded from analysis.

Patients who were known to our local rheumatology service were identified. Patient demographics; and tests performed by the attending clinician for the presence and extent of lung, cardiac, and renal organ involvement; were retrospectively sought from computerised clinic records and laboratory data. Disease duration and the use of disease modifying anti rheumatic drugs (DMARDS) were noted.

Results

Seventy-four patients with the diagnosis of SSc were identified. The majority of patients (72%) had lcSSc (Table 1).

Table 1. Types of SSc disease

Systemic sclerosis	Patient numbers
Limited	53
Diffuse	11
Localised	3
Not specified	7

As expected, most of the patients were females (88%) and of European ethnicity (Table 2).

Table 2. Patient ethnic groups

Ethnicity	Patient (%)
European	80
Asian	11
Pacific*	3
Māori	1
Other	5

*Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Of the 53 lcSSc patients, 39 were followed up regularly at our rheumatology service. Ten of the 11 dcSSc patients were known to our service. The other SSc patients who were not known to our service were primarily admitted under the inpatient care of the plastic surgical or vascular services for surgical management of ischaemic digits.

The mean age at last review of the lcSSc patients was 61 years compared to 44 years in the dcSSc group.

We analysed the following data from the SSc patients who were known to our service (39 lcSSc and 10 dcSSc patients).

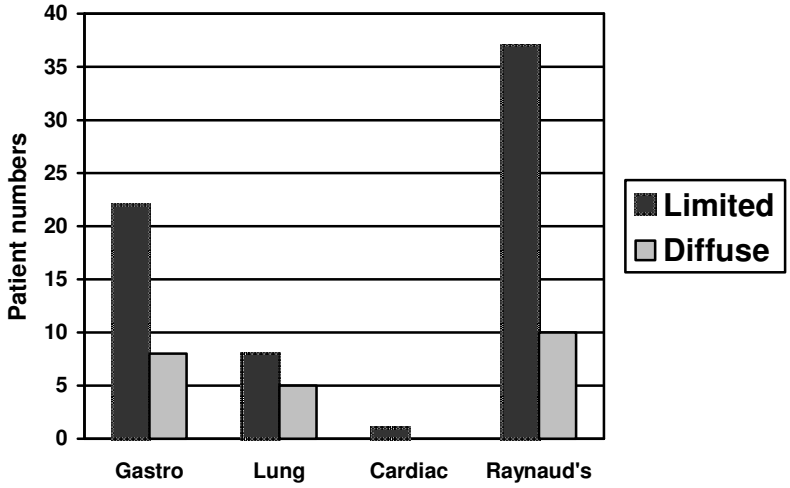
The majority of dcSSc patients (80%), and slightly more than half (58%) of the lcSSc patients, had the disease for more than 5 years. There were only two diffuse SSc patients with disease duration of less than 5 year—one patient was asymptomatic for lung disease but both patients had the same lung screening tests except one did not proceed to have an echocardiography.

Raynaud's phenomenon was the most frequent clinical feature observed followed by gastrointestinal and lung organ involvement in both groups (Figure 1).

Nine of the 39 (23%) lcSSc patients and 5 of the 10(50%) dcSSc patients had lung involvement. Approximately half of these patients (54% lcSSc and 60% dcSSc) had no respiratory symptoms. Of the patients who were symptomatic, the majority (92%) complained of dyspnoea or a dry cough (26%).

All patients with lung involvement had abnormal PFT with an obstructive or restrictive ventilatory defect, decreased DLCO, or a combination of both abnormalities. The types of lung disease observed are ILD (non specific interstitial pneumonitis), bronchiectasis and PAH.

Figure 1. Frequency of major organ involvement



Of the 9 lcSSc patients with pulmonary involvement, 5 (56%) had PAH; 1 patient also had pulmonary embolic disease. Only 1 of the 5 dcSSc lung patients had PAH associated with ILD. None of the patients with underlying SSc-ILD and PAH had specific drug treatments for their lung disease, because treatment was not available at that time. No patients in either the lcSSc or dcSSc groups developed renal crisis. Three lcSSc patients have died. The causes of death were pulmonary hypertension complicated with right heart failure, peritonitis and metastatic oesophageal carcinoma.

Table 3 shows the frequency of patients who have had investigations for their respective organ involvement. In general, a higher proportion of dcSSc patients had investigations for pulmonary (except for BAL), renal, and cardiac involvement compared to the lcSSc group. The majority of the investigations were instigated by the attending physician only when the patient was symptomatic.

Table 3. Frequency of investigations performed for respective organ systems

Investigation	Limited (%); n=39	Diffuse (%); n=10
Pulmonary		
Lung functions	26 (67)	9 (90)
CXR	26 (67)	8 (80)
HRCT chest scan	22 (56)	7 (70)
BAL*	2 (5)	None
Cardiac		
ECG†	6 (15)	2 (20)
Echocardiograph	18 (46)	5 (50)

Renal		
BP‡	30 (77)	9 (90)
Dipstick urine analysis	13 (33)	7 (70)
Electrolytes	25 (64)	10 (100)

*BAL: bronchoalveolar lavage; †ECG: electrocardiograph; ‡BP: blood pressure.

Nail fold capillaroscopy examination was frequently not performed or documented in the clinic letters (67% lcSSc and 70% dcSSc).

The prevalence of common autoantibodies associated with SSc in each group is outlined in Table 4.

Table 4 Frequency of SSc auto antibodies

Auto antibody profile	Limited (n=27) (%) §	Diffuse (n=7) (%) ‖
Anti centromere antibody positive	22 (81)	None
Scl 70 positive	2 (7)	5 (71)

§No records in 12 patients; ‖No records in 3 patients

Table 5 lists the various DMARDS used. The majority of patients were on calcium channels blockers for Raynaud's phenomena (22/39 lcSSc patients and 6/10 dcSSc patients).

Table 5 Disease modifying anti rheumatic drugs (DMARDS) used

DMARDs	Limited (n=39)	Diffuse (n=10)
Penicillamine	1	2
Methotrexate	1	2
Azathioprine	1 ¶	1

¶ For autoimmune hepatitis.

Discussion

This audit reviews our local practice of screening and diagnosing pulmonary, renal and cardiac related diseases in patients with SSc. A review of SSc patients in Auckland 25 years ago showed that the involvement of renal and cardiac organ systems were main adverse prognostic factors associated with a poor survival rate.[14] This has changed over the years with the advent of angiotensin–converting enzyme inhibitors. Scleroderma renal crisis now only accounts for 8% of scleroderma related deaths.[12] This is reflected in this audit where no deaths were related to scleroderma renal crisis.

This audit shows that lcSSc patients were not screened as rigorously as the dcSSc patients for lung disease, although visceral lung involvement can occur in both groups. A higher proportion of dcSSc patients had the relevant tests to monitor for pulmonary, renal, and cardiac SSc-related diseases.

The initial standard respiratory tests used at our centre are PFT and CXR. Some of these patients proceeded to HRCT chest and BAL (in a minority of patients, n=2) if there was a strong clinical indication of SSc–ILD, although BAL was not routinely performed.

We found no consistency in the frequency of PFT request. Although it is recommended that PFT and HRCT chest be repeated at periodic intervals, there has been no Cochrane or published systematic review addressing this aspect of management in patients with SSc.

None of the patients with underlying SSc-ILD in this audit received cyclophosphamide as previously, the evidence for this potentially toxic immunosuppressive has mainly been based on uncontrolled retrospective studies.

The Scleroderma Lung Study group has recently demonstrated that oral cyclophosphamide has a modest benefit in SSc-ILD in a controlled trial.[15] One would need to balance the potential toxicity of cyclophosphamide versus benefit when initiating cyclophosphamide therapy in SSc-ILD. We recommend the generation of a standardised protocol for regular interval respiratory investigations which may help identify patients with progressive lung disease so that early treatment could be considered.

Most of the patients in this audit did not have the recommended annual screening echocardiography. In the majority of cases, an echocardiography was requested only when patients were symptomatic. However, it is important to identify "early" PAH patients for early intervention as the disease can be irreversible when patients become symptomatic. The optimal screening method is yet to be determined. A recent prospective study showed that an algorithm based on dyspnoea, echocardiograph and right heart catherisation can detect mild PAH at an early stage.[16]

In the last 5 years, there have been major advances in the treatment of PAH. Prostacyclin analogues (epoprostenol, treprostinil, and iloprost) can have short term benefits in improving the New York Heart Association function and cardiopulmonary haemodynamics.[17] Endothelin receptor antagonists (bosentan, sitaxsentan) can improve exercise capacity and cardiopulmonary haemodynamics in PAH.[18] Recent studies suggest that these agents may also improve survival in PAH associated connective tissue diseases.[19, 20] Sildenafil (phosphodiesterase inhibitor) may be of benefit in reducing pulmonary vascular resistance.[21] A recent study has confirmed that sildenafil improves the exercise capacity and haemodynamics of symptomatic PAH patients.[22]

Treatment options for pulmonary hypertension in New Zealand are currently very limited. Most of the agents mentioned above are currently not funded in New Zealand and the difficulties of obtaining these agents were highlighted in a recent review.[23] This probably has an impact on our practice as highlighted in this audit where only half of the patients with lcSSc and dcSSc had echocardiographs performed. Thus, the recommended annual echocardiography screen for an early diagnosis of PAH in SSc may not be applicable in New Zealand. Another advantage in performing annual echocardiography screening is for the detection of primary cardiac involvement related to SSc.

Significant cardiac abnormalities have been found in more than half of SSc patients at autopsy suggesting the sub clinical nature of SSc-related cardiac disease.[24] The cost-effectiveness of routine screening with more expensive investigations such as HRCT and echocardiography also needs to be taken into account.

This audit highlighted that nailfold capillaroscopy examination was infrequently performed. Abnormalities in nailfold capillaroscopy may be an early finding in SSc-ILD. One study showed that severe nailfold capillaroscopy changes have a sensitivity of 100% for ground-glass opacities on HRCT in patients with disease duration of less than 5 years.[25] This abnormality should be looked for to identify patients needing more intensive investigations for the presence of SSc-ILD.

The overall treatment of patients with SSc is generally lacking. Initial studies suggested that D- penicillamine was effective for the skin and possibly lowers the incidence of systemic involvement.[26] Since then, a double blind randomised trial has shown that there is no benefit of high dose penicillamine compared to low-dose penicillamine.[27] It is still unclear if low dose penicillamine has any beneficial effect. There were only three SSc patients in our audit who were on penicillamine.

Interestingly, the demographic results from this audit suggest a low prevalence of SSc in the Māori and Pacific population (4%) in comparison to a higher prevalence rate of systemic lupus erythematosus in similar ethnic groups.[28]

In conclusion, this audit has reviewed the current local practice of screening SSc patients in a tertiary rheumatology centre in New Zealand. The generation of a local screening and monitoring protocol may improve the management of SSc-ILD in light of the recent Scleroderma Lung Study findings. The access to new therapeutic agents for PAH associated with SSc remains an ongoing issue in New Zealand which has implications on the practice of early detection of pulmonary hypertension in SSc patients.

Competing interests: None.

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Exploring general practitioner identification and management of psychosocial *Yellow Flags* in acute low back pain

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Abstract

Aim Over the past decade, psychosocial issues have been increasingly identified as risk factors that are associated with the development of chronicity and disability. These psychosocial risk factors are known as *Yellow Flags*. In New Zealand, in 1997, the Accident Compensation Corporation (ACC) published the *Acute Low Back Pain Guide* and the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain*. The aim of this qualitative study is to understand the experiences of general practitioners (GPs) in the identification and management of psychosocial *Yellow Flags* in patients with acute low back pain.

Method A qualitative research approach was used. GPs were purposively selected and semi-structured interviews were undertaken.

Results The doctor-patient relationship created the key element for the GPs in approaching any psychosocial factors that were identified. The management of psychosocial factors depended on an individual GP's worldview and orientation to the biopsychosocial model of pain. Problems with time management were composed of multifactorial facets. Funding, lack of appropriate training, and the GPs' perception of ACC's rehabilitation model, all formed components of the meanings that the GPs constructed from their experiences.

Conclusion GPs did not use the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain* or the screening questionnaire to identify psychosocial risk factors in their patients with low back pain. Investment of resources in GPs is needed to empower them to be effective gatekeepers guarding against chronicity. This demonstrates a need to alter the current ACC Guideline dissemination and implementation.

The increasing prevalence of back pain has been described as an epidemic.[1–6] Most back pain becomes manageable within a relatively short timeframe. Serious difficulties arise when the pain persists. These consequences affect not only the person suffering from the pain but their families and workplace as well. Pain impacts upon society in terms of work loss and provision of welfare benefits.[7,8] Recently, psychological and social factors have been recognised as playing a key part in the development of chronic disability.[9–13] This premise led to the development of the *New Zealand Acute Low Back Pain Guide* (ALBPG) and the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain*.

The *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain* (*'Yellow Flags' Guide*) was developed by the National Advisory Committee on Health and

Disability of the Ministry of Health together with the Accident Rehabilitation and Compensation Insurance Corporation.[14]

The *Yellow Flags Guide* outlined the need for the identification of the psychosocial risk factors by healthcare providers. The *Yellow Flags* monograph comprised a clinical assessment, a screening tool, and recommendations for early management.[15] Linton and Hallden[15] developed the *Acute Low Back Pain Screening Questionnaire* (ALBPSQ). The *Yellow Flags Guide* recommends use of the ALBPSQ after no progress 2 to 4 weeks following initial presentation. This is to discriminate between 'at risk' and 'not at risk' patients.

For those 'at risk' individuals identified, a clinical assessment of psychological factors is advised. The healthcare provider is asked to consider referral to a suitable clinician unless they themselves have the skills and resources to develop and implement a management plan, targeting specific issues to prevent long-term distress, reduced activity and work loss. Appropriate early intervention could thus be provided resulting in the prevention of persistent pain and disability.[16,17]

Most New Zealand general practitioners (GPs) have been repeatedly exposed to the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain ('Yellow Flags' Guide).* The screening instrument is *The Acute Low Back Pain Screening Questionnaire*.

The aim of this study was to qualitatively explore (using a social constructionist approach) the experiences of selected GPs in the identification and management of the psychosocial risk factors (*Yellow Flags*). This was performed with reference to the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain* as well as to the theoretical underpinnings involved. Related features included: - What did the GP's make of these experiences? What were their beliefs or worldviews? What were the consequences of the meanings that were constructed from the experiences, for themselves, and for their patients?

There has been rapid international uptake of the construct of *Yellow Flags* as being psychosocial factors that increase the risk of long-term disability.[18] The setting (the where and how of the information gathering) needs to be described. In addition, the screening instrument needs to be validated for each particular context.[18]

Methods

The qualitative paradigm was chosen as the most appropriate means of exploring the experiences of GPs' identification and management of psychosocial *Yellow Flags* in sufferers of low back pain. This due to the fact that it is concerned with describing patterns of behaviour and processes of interaction as well as revealing the meanings, values and intentions of a person's life experience. The chosen perspective, *social constructionism*, fits the aim of the study—to provide an understanding (as experienced by physicians) of their reality in regard to the use of *Yellow Flags*.

Purposeful sampling was employed to create depth to the emerging knowledge. This allowed for (and permitted changes to) strategy and adaptability of the researcher to the unique circumstances of the study. The characteristics of the sampling process were viewed as follows—indigenous to the study; not completely under control of a sampling design; and an ongoing activity involving the interviewer and the participants. Participants were selected for their relevance to the aim of the study, not for their representativeness.

The interview was designed to incite narrative production. GPs were selected, who (because of their experiences with *Yellow Flags*) could provide eloquent and indepth insight into the research question (so called 'information-rich' informants) as opposed to representatives of populations.

The participants needed to be GPs who saw patients with back pain and who (via the RNZCGP and ACC) had been exposed to guidelines. It was calculated that 11 interviews were required to reach the point of variation in data and diversity in participants for a study of this nature. The GPs were informed that the study would explore their experiences regarding identification and management of psychosocial risk factors in low back pain.

Semi-structured audiotaped telephone interviews were conducted with 11 GPs. The basic structure of the interview is presented in Table 1. Brief notes were made during the interviews to ensure that all topics were discussed. Each GP was interviewed only once and interviews lasted between 25 and 45 minutes. The differences in time allowed provided space for open-ended questions that interviewees answer differently, their varying experiences with the guidelines, and their varying lengths of introspection and eloquence.

The audiotapes were transcribed and printed. Interviewing took place over several months allowing the interviewer to develop an understanding that enabled him to facilitate and permit the participants to shift their perspectives during the interview process. Participants were offered different ways of conceptualising an issue. The participants were made aware of the interviewer's background in pain medicine. The interviews were viewed as an interpretive, active practice and process that examined the *how's'* and *what's* of the GPs experiences. The GP's constructions were explored with them at the time of the interview as well as later by the researcher from the interview transcripts.

Table 1. The basic structure of the interview process

Nature of practice	Number of clinicians Multidisciplinary Urban/rural Percentage of business that is Accident Compensation Corporation (ACC)
Nature of participant	Male/female Bandwidth of years in practice Bandwidth of age
Orientation to guidelines	General *New Zealand Acute Low Back Pain Guide* *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain*
Orientation to models	Medical model Biopsychosocial model
Identification and management strategies for biopsychosocial issues in low back pain	
Experiences of success and failure	
Approach to rehabilitation	
Other issues raised during the interview	Should there be incentives for GPs to be able to spend more time with those patients have biopsychosocial issues? What is the level of influence that ACC exerts on clinical practice? Is there a better way of doing things in future?

The interview data was analysed to render recurring themes and phrases, achieving primacy of the subjective data, and permitting understanding of the GP's construction of a worldview of their experiences.

Inductive analysis was used to explore the details and specifics of the emerging understanding. This was achieved by working from the data of the specific cases towards a more general conclusion, while simultaneously seeking to fashion a creative synthesis. The transcripts were read and read again, examining them for broad themes and phrases. This initial phase involved working with the more 'concrete' themes that came directly from what the participants had constructed during the interview process. These were coded from each interview.

An inductive and iterative process then followed in which the information was grouped by the coding that had been developed. Examining the issues identified in the way they related to the main focus

refined it further. These phases evolved over a period of several months. The process was iterative and required consideration and repeated exposure to the data in order to permit the recognition of the primary themes.

Results

The analysis of the data revealed three key themes: cultural perspectives; orientation to guidelines; and orientation to ACC.

Cultural perspectives—The relationship between the GP and their patient was described as being of key importance to the identification and management of any symptoms, particularly if psychosocial components were present.

> We have a relationship with them (from a GP in large group practice).
>
> Knowing the person, that's probably the most important path

Here, the GPs take the initial position that what they are told by their patient is real. The GPs described this as the most satisfactory way of approaching any possible biopsychosocial issues. This gave them the confidence to then start examining any *Yellow Flags* aspects. The consultation follows the patient's agenda and thereby maintains a doctor-GP relationship.

The GP participants consistently stated that their initial approach was biomedical. Some doctors felt that there could be repercussions if they examined the patient from any other perspective than a biomedical one. The GPs valued their relationships with other health professionals involved in their patients' care.

Time issues generated the greatest dialogue. Time was considered a barrier to both the identification and management of *Yellow Flags*. In every case, it was simply not possible to schedule any additional patient time at the initial appointment.

> It takes ages and then you're half an hour late sort of thing…I mean to do a really good physical examination, to take a really good musculoskeletal history and then a psychosocial history is a time-consuming process

The time required for identifying and managing any presenting *Yellow Flags* impacts financially upon both the patient and GP. The GPs consistently identified that a greater amount of time needs to be devoted to patients who have biopsychosocial issues. Longer or earlier follow-up appointments are booked by some physicians for patients with *Yellow Flags*. Compounding the impact of time on the consultation dynamic is the ever-present paperwork that comes with ACC claimants (GPs described the amount of work that ACC represented in their businesses was between ten and fifteen percent. A disproportionate amount of administration occurred with ACC-based work). The limited availability of time became a barrier to GPs reading the guideline in depth and in detail. GPs whose practices were orientated towards the biopsychosocial model felt that education for GPs on the guideline was lacking as well.

> There's probably a lack of understanding with the recognition of the implications of *Yellow Flags* amongst general practitioners

Physicians are obligated to examine and screen for biophysical red flags before undertaking any other approach.

> Many are very split…, there's mind things over there and physical things over here and they're not often thinking about the connections and doctors just simply aren't given the training to deal with it

Most GPs continued with the biomedical approach until there was a lack of progress. Once past the biomedical parameters, strategies were used by GPs to start exploring the possibilities of non-physical signs being a component of their patient's problems.

> If it's like a story that just doesn't fit or you know it's like a very minor thing that all of a sudden, you know

> I've got stress mood graphs and physical symptom graphs that I graph for

Showing the patient that their problems are not purely biomedical may create the risk of losing that patient from their practice. To identify *Yellow Flags*, and communicate this to the patient takes understanding, trust, and time. Biopsychosocial model-orientated GPs felt that patients needed to start to make the connections themselves. This required their facilitation, a skill for which they felt under-trained in.

> Doctors simply aren't given the training to deal with that

The personal orientation of the GP as well as their clinic helped shape the patient's attitude towards exploration of the psychosocial aspects.

> Cause that's the way they're used to us operating really

Orientation to clinical guidelines—Participants held definite views and feelings about the character of the guidelines. The guidelines were seen as an artificial, mechanistic approach to medicine. They were viewed as highly structured, categorical, and prescriptive, subjugating clinical judgement by reducing medicine to algorithms.

> It's pigeon-holing patients in here, there and everywhere

> Some of it is a wee bit insulting…it reduces medicine to…all these algorithms

> Too often it was almost too prescriptive

> I would have been incredibly resistant to someone sort of pushing me through an algorithm.

In identifying and managing psychosocial problems in low back pain, the GPs relied on past experience and clinical judgement over and above the use of guidelines. An intuitive, experiential approach—with the ability to contextualise the problems through the existing doctor-patient relationship—was prized ahead of clinical guidelines.

> They're quite sort of structured aren't they, and I think that eighty percent is missing. Eighty percent is your gut feeling

> The idea of a guideline…it just isn't really useful

> …I use those things that make up *Yellow Flags* intuitively and from experience 'cause I know that those things influence what happens'.

> Given the constraints of the ten to fifteen minute appointment, I personally believe the issues are largely ignored

> I get the feeling that they're unhappy...and they would sort of flag things in my mind, but I don't sort of routinely go down the list and check them all out

> I get the feeling in my gut as soon as they come through the door

The usefulness of the guideline, and associated screening questionnaire, was described as poor with limited clinical value. The advice they provided was perceived as ineffective.

> You know the guidelines from ACC. I'm afraid I'm a little bit jaundiced

> I can never put my hand on it and people seem to fall outside algorithms
>
> They're not a lot of use basically
>
> By the time I need to look out for the guidelines—where are they?
>
> To be honest I don't even refer to them

The guideline presumed consultations to be always doctor driven and operating in a linear fashion. Consultations were, however, described as dynamic and did not necessarily follow the "ACC agenda".

> Some things that ACC might like attended to end up being relegated further down the list…that's actually appropriate

The guidelines were not suitable to the 'real' patient situations with which GPs deal on a day-to-day basis. This contributed to some GPs choosing not to use them.

> A guideline tends to look at the difficult patient or and the patients never quite fit
>
> The guidelines are constructed in a very mechanistic and objective way and what you're doing with your patient so often is a lot different from that

Available time to read them was limited.

> To be honest I don't get the time to read them all

Reading the guideline was often the only educative method used by GPs.

> I read them when a new one comes out but then I don't pay much attention I must admit

The initial reading determined relevance to their clinical practice.

> I would cast my eye over a guideline…and it might modify my behaviour

There was a general mistaken assumption that the ACC guideline was evidence-based.

> The guidelines are evidenced-based aren't they?'

The volume of all the guidelines with which GPs are faced was overwhelming in terms of having time to read them and assimilate into their clinical practice.

> If everything that came across our desks we were meant to read, we wouldn't be doing any work
>
> If you put them all in a pile it comes to about forty-five centimetres. It's another nice glossy document which goes more or less on the shelf…it very rarely comes down

The ACC-created paperwork and process barriers became disincentives to guideline compliance.

> You've got this great big piece of paper and you've got to fill it out
>
> Just paper work gone mad

What was accepted practice amongst their peers superseded any guideline recommendation.

> The judges may look at your peers more than they would the guidelines

GPs queried the clinical value of guidelines.

> ACC rolls out these guidelines with screening questionnaires but clinically they have little value

The short-term benefits of guidelines were not readily identifiable by most GPs. Guideline recommendations were seen primarily as a system of ACC cost-saving.

> This is just to save money for ACC

Orientation to ACC—Three aspects from the GP's perspective affected their outlook on ACC and their guidelines, namely: the process behind the model; funding; and the GP's opinion of the rehabilitation model of ACC. A disproportionate amount of paperwork and ongoing administration was linked to ACC work.

> At any time ACC ask things, they want all the information again. So when you go back over a few years worth and photocopy all these things

It was perceived that the quantitative values measured did not necessarily reflect quality of care.

> You and I both know that (it) doesn't always equate to quality just because someone's off their books

The identification of *Yellow Flags* might even work against the patient and their rehabilitation.

> It's going to attract the ACC Case Manager who may want a copy of my old notes...so I quite often deliberately don't write down those things

Participants argued that financial support was needed to explore the psychosocial dimensions and assist with the dissemination of effective implementation strategies.

> There should be financial drivers
>
> ACC actually has to release funding…because it takes time

Counteracting this was the opinion that the present system (fee for service) gave the GP a financial incentive for medicalising the treatment.

> …an incentive to continue to medicalise a problem by getting people back
>
> It never fails to amaze me…why do ACC fund, you know, all sorts of things when we think there is no evidence that it actually works

Participants saw no self-directed incentive for patients to return to independence. Patients are encouraged to demonstrate to ACC (in an ongoing fashion) that they are unwell in order to obtain continued support.

> For people to continue to receive support they have to demonstrate they are sick. And what does that do for rehabilitation?'

The attitudes of the GPs were partially related to personal experience or practice experience with the local ACC branch.

> Because too many patients have been irritated by the ACC process. That gets in the way of the rehabilitation

Discussion

Low back pain (LBP) is expensive for society and for the individual affected. A small number (5–10%) of LBP sufferers are responsible for the majority of costs (70–90%).[19–26] The total costs vary from country to country, but are measured in billions of dollars.[2,20,22] Many of the costs are medically-related costs, with surgery and clinical investigations being the most expensive followed by physical therapy.

Approaching the treatment of back pain via the disease model does not allow for the complex sensory and emotional human response to pain and disability. The biopsychosocial model allows for the interaction between the person, their social environment, and physical dysfunction and illness behaviour, beliefs, and coping strategies. It represents the clinical presentation of LBP at a point in time and recognises the behavioural and psychological factors that could help explain a person's levels of pain and disability.[2,14]

The World Health Organization (WHO) has used this biopsychosocial model as the basis for the international classification of functioning, disability, and health.[27] Most risk factors in LBP that result in chronicity are psychological, behavioural, environmental, or social in nature.[28,29] The relevance of these risk factors in acute and subacute LBP has been demonstrated.[14,30–39]

Many clinical guidelines have been produced and promoted. However, there is little evidence in the literature showing that using guidelines results in better outcomes. For instance, in a case-controlled study, 547 patients with LBP from urban and rural clinics throughout Australia were compared to groups that were subject to either usual medical care or evidence-based care for LBP.[39] The results indicated that those who received the evidence-based care had significantly better symptom relief, a significantly greater rate of full recovery, experienced greater satisfaction regarding their care and outcome, and were significantly less expensive to treat.

Sheehan[40] looked at LBP in countries where guidelines for LBP had been published and disseminated nationally several years ago. The study found that there was in fact no overall reduction in the number of back pain cases referred to healthcare providers.

The GP participants identified that their interpersonal relationships with their patients allowed them to manage *Yellow Flags* as they felt appropriate. A patient-centred approach facilitated the patient's involvement in the decision-making process. The GPs in this study used a collaborative or partnership approach, where the doctor was the service-provider. They described strategies that permitted them to work with their patient to understand how psychosocial factors may form part of their problem. In doing this, the GPs avoided an antagonistic "tug-of-war" situation.

The doctor-patient relationship has been influenced by several factors: group type practices; the establishment of after-hours clinics where no appointment is required; and a redistribution of medical work to nurses and non-medical staff. This often reduces the work of a GP to a set of biomedical tasks. Yet failure to meet their patient's expectations could result in the patient changing to another GP.

Time is central to medicine. Longer waiting times heightened anxiety and increased patient vigilance in monitoring their consultation time.[41] The GPs in this study all identified time management to be challenging in patients that present with psychosocial elements. The strategies used to manage in these situations included extending the initial consultation time and/or scheduling subsequent appointments.

In the literature, the relationship between consultation length and quality of care is unclear.[42–44] The GPs in this study had appointment times of similar length to one another. They would schedule more time if required. When there are psychosocial aspects to patient's problems, the consultation becomes more complex and takes more time.[45,46] The study demonstrated that appropriate education relevant to identifying and managing *Yellow Flags* in general practice is needed. Thoughts expressed on evidence-based medicine in general and the ACC guidelines in acute low back pain in particular, confirmed these added demands on GPs.

How do GPs use evidence-based medicine to attain 'best practice' in a resource-constrained environment? Simply supplying clinicians with information has not bridged the gap between evidence-based best practice and actual clinical care. The *Yellow Flags* guideline is a consensus document not directly supported by evidence.

None of the GPs were aware of this. They assumed that this guideline was supported by good evidence.

Participants expressed criticisms and concerns about these *Yellow Flags*. A New Zealand study[47] surveying 448 GPs concluded that low morale and high stress continue to affect New Zealand general practice. Paperwork and bureaucracy scored as the highest sources of stress. Indeed, a sense of frustration was detected in the GPs interviewed. High demands and low locus of control affected their work—data from this study confirms bureaucracy to be a stressor for New Zealand GPs.

There are many theories and models relating to the dissemination and implementation of clinical guidelines and the challenges that they present.[48–52] Grol and Wensing[52] found that knowledge relating to barriers and incentives for change occurred at various levels (innovation, individual professional, patient, social context, organisational context, economic context, political context). No advantage to clinical practice by the use of this guide was expressed by the GPs. There were mixed levels of enthusiasm for guideline implementation. This was partially related to their previous experience of clinical guidelines, the consultation dynamic and their orientation towards ACC.

ACC's primary dissemination strategy had been mail-outs or secondary a 'road show' format. Selected clinicians presented these 'road shows', to which GPs and other health providers were invited. In addition to ACC-led initiatives regarding *Yellow Flag* dissemination, contributions were made from the independent practitioner organisations (IPOs), small group meetings, and a GP's individual worldview. There was a lack of evidence of any communication or strategy operating between these different factions to coordinate the process of guideline dissemination and implementation.

Arroll[50] reported that in New Zealand too little emphasis is placed on guideline implementation with the bulk of resources being allocated to the development of the guidelines. This is reinforced by the experiences of GPs in the study. None were aware of the level of evidence used in developing the guidelines. All described a marked lack of utility and saw the guidelines as mechanistic, prescriptive, and as having little relevance to the context of their daily work.

A previous publication readily identifiable as being evidence-based (the ALBPG) was merged with the *Yellow Flags Guideline*, a guideline not subjected to the same level of development and scrutiny. The New Zealand Guideline Group felt the *"information in the document was misleading, in that the Yellow Flags section of the Guideline is a consensus document"* (New Zealand Guidelines Group Information Manager—personal communication, April 2005).

GPs are overwhelmed by information but are unable to find it when they need it. Gray[53] called this information paradox the two laws of dissemination. Firstly *"... the probability that a disseminated document will arrive on someone's desk the moment it is needed is infinitesimally small, and secondly the probability that the same document will be found three months later when it is needed, is even smaller"*. The GPs in this study exemplified this statement with their comments on their difficulty in initially reading the guidelines and then accurately locating them when they were needed.

This study exposed the lack of usefulness of the guideline. One participant described the stack of guidelines he was supposed to be aware of, as "sitting over 6 inches in height on a desk". None of the GPs referred back to the *Yellow Flags* guidelines. GPs manage 90% of presenting problems without referral elsewhere.[54] The challenge remains to effectively disseminate and integrate guidelines to improve everyday clinical behaviours yet without imposing a further burden. The participant GPs focus is on providing the best possible care for their patients.

Conclusion

This qualitative study examined the experiences of a selected group of New Zealand GPs in identifying and managing psychosocial risk factors in their patients with acute LBP. The instrument published to assist GPs with this task, the *Guide to Assessing Psychosocial Yellow Flags in Acute Low Back Pain*, was evaluated. The criticisms and concerns articulated by the participants of these *Yellow Flags* are expressed. In New Zealand today, GPs are expected to follow guidelines, work within the biopsychosocial model, and comply with paperwork from ACC. These factors result in a mismatch between the availability of time, and the expectations of care.[55]

The GPs stated that their existing relationship with their patient allowed them to discuss psychosocial risk factors. Yet to first reach that point a biophysical approach was needed. The identification and management of psychosocial risk factors depended on the individual GP's worldview and culture of practice. The barrier of time was multifaceted and affected all GPs alike. Constructions were drawn from their experiences with the ACC process, and their lack of adequate training and funding. The GPs orientation to the *Yellow Flags* guideline related to their experiences of guidelines in general, and was not linked to methodological status. The GPs did not refer to or make use of the guideline for identification or management of any psychosocial risk factors in patients with acute LBP.

Identification of psychosocial risk factors requires a combination of a suitably skilled clinician, adequate time, and a screening system. Since 1997, the concept of *Yellow Flags* being indicators of psychosocial risk for long-term disability has been rapidly adopted in the medical literature. From a systems perspective, the publication of the *Yellow Flags Guidelin*e aimed at secondary prevention has been considered visionary.[18]

The guideline's construction incorporated the belief that the setting, timing, and screening were key elements. Most GPs chose not to use it to acquire further knowledge. More prospective studies testing other variables are unlikely to alter the GP's choice. Does the situation need a new perspective as has happened in other areas of healthcare? In the world of back pain, there appears to be a need to invest resources at GP level, focus on their needs, and then work towards evidence-based medicine and 'best practice'. In this way, engagement with change that is sustained and progressive may be achieved.

Based on this study's findings, for such an approach to succeed, the emphasis would need to alter from the current orientation of guideline dissemination and implementation, to one based around the GP, taking cognisance of their organisational and behavioural mechanisms of change, as well as their binding within a local

context. GPs operate in a pressured environment. With time, patients may shift from acute and sub-acute states towards persistent (or chronic) status.

This study suggests that investment of resources in GPs is needed to empower them to be effective gatekeepers guarding against chronicity. This is particularly important if GPs are to be involved during the acute stage and remain engaged as active participants throughout the entire course of their patient's rehabilitation.

Competing interests: None.

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Practitioners, patients, and their visits: a description of accident and medical (A&M) clinics in New Zealand, 2001/2

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Abstract

Aims The National Primary Medical Care survey was undertaken to describe primary health care in New Zealand, including the characteristics of accident and medical (A&M) clinic providers, their practices, the patients they see, the problems presented, and the management offered.

Methods Data were collected from a 50% random sample of all A&M clinics in New Zealand as part of the National Primary Medical Care survey carried out in 2001/2.

Results Data were obtained from 12 A&M clinics throughout New Zealand between usual hours (Monday–Friday 8am–6pm) and at other times. A&M clinics were staffed by an average of 2.7 full-time equivalent (FTE) A&M practitioners . Most clinics operated as a limited liability company. The majority of A&M practitioners were male and aged between 35–44 years. On average, A&M doctors had been in practice for over 10 years and had been in the sampled practice for only 2.9 years. More than a third of doctors had not trained in New Zealand. The doctors worked, on average, 6.3 half days and saw nearly 90 patients per week. The findings suggest that young patients and a diverse ethnic range attend A&M practices. Community Services Card holders were not usual patients. Few patients had an ongoing relationship with the practices. Most visits related to a single, new, and short-term problem that was often an injury or a respiratory illness. About a fifth of visits were associated with an order for an investigation or an X-ray, fewer investigations were arranged outside usual hours. About half the visits resulted in a prescription but more visits outside normal hours received pharmacological treatment and the number of items was higher. The most frequently prescribed items were antibiotics and analgesics. Follow-up was arranged for between a third to a half of visits, depending on the time of day. Referrals were often made to non-medical destinations. While patient and visit characteristics were generally similar regardless of whether the visit occurred during usual working hours or at other times, some differences were apparent in the type of problems that were presented out of hours and their management.

Conclusions The main impression is that the medical A&M clinics provide episodic treatment for relatively young patients mainly related to a new, short-term problem, particularly an injury or a respiratory illness. This picture is consistent with previous research and the role of similar clinics overseas. Further work is needed to compare A&M clinics with established general practice in relation to the services that are provided as well as the acceptability and quality of these services.

In the late 1980s, New Zealand primary care witnessed the development of a unique form of general practice—the accident and medical (A&M) clinic. These commercial clinics were usually located in central urban areas and offered extended opening hours, consultations without an appointment, and limited links to traditional general

practice.[1] The clinics likely arose from international trends that included rising patient demands for out-of-office hour care[2] and an increasing willingness by the medical profession to find new collective solutions to providing after-hours medical care.[1]

Since the 1980s, many Western countries have witnessed the rise of cooperative after-hours centres[3] where general practitioners have joined together to provide their after-hours care from one centre thereby reducing their isolation while on-call and rationalising their workload.[4] In addition, deputising services have also flourished especially in the United Kingdom.[5]

Unlike co-operatives, these services function as commercial organisations that effectively provide locum services to GPs out-of-hours thereby relieving them of this task. Medical A&M clinics are, however, unique to New Zealand with their closest parallel being provided by walk-in clinics in Canada.

In common with New Zealand clinics, the Canadian version operates out of convenient, central city locations and provides extended hours with no-appointment schedules.[6] These clinics have developed in parallel with general practitioners and do not attempt to minimise their overlap with family practices or routinely organise follow-up with the established primary care-giver.[7] Surveys have suggested that these clinics are primarily attended by younger people with predominantly minor infections and injuries.[8, 9]

Only one study has previously attempted to describe the characteristics of patients, practitioners, and practice features associated with medical A&M centres in New Zealand.[1] That study was conducted in the Waikato region between 1991–2 and included three clinics and 70 "orthodox" general practitioners. Patients at the clinics were more likely to be younger, employed, and new to the practice. Patients usually visited with single, new, acute, and relatively minor problems such as injuries and respiratory infections. Although important, that research was limited by the small and regional nature of the sample.

The National Primary Medical Care survey (NatMedCa) was undertaken to describe primary health care in New Zealand. Study methods and an overview of results, particularly those related to traditional general practice, have been described.[10] This study followed the methods employed by the previous Waikato-based research[1] and describes the characteristics of practitioners, patients, and patient visits.

Methods

A&M clinics were defined by the following criteria:

- Having X-ray equipment on site;
- Extended hours at least until 8pm, and open 7 days a week;
- Community-based rather than hospital-based.

A list of 52 A&M clinics was compiled from White Pages telephone book listings and supplemented by data from the Accident & Medical Practitioners' Association (AMPA), which provided a supporting letter, and other sources.

A&M practitioners are typically salaried and are not listed in the White Pages. Clinics were distributed throughout the country, with particular concentration in cities. A 50% random sample of all A&M clinics (n=26 clinics) were invited to participate. Data collection was spread over a year and around the geographical areas. Data were collected in relation to two time periods: either between usual hours (Monday–Friday, 8am–6pm) or at other times.

Practitioners from each clinic were asked to provide data on themselves and their practice; they were also asked to report on their patients over a week-long period. The log questionnaire, completed for all patients seen during the data collection period, recorded demographic data. The visit questionnaire recorded data about the patient's problem(s) and management. The practitioner questionnaire obtained data on practitioner background and activities.

Reason-for-visit and diagnosis were coded using READ version 2 software, while drugs were classified using the anatomically based Pharmacodes/ATC system.

Approval was obtained from ethics committees in all areas represented in the survey. The study received advice from an advisory and monitoring committee that included representation from consumers and relevant professional groups.

Results

Data were obtained from 12 clinics (response rate=55%), predominantly based in Auckland (8/12) with the remainder equally shared between the rest of the North and the South Islands. Compared with data describing the national distribution of A&M clinics in New Zealand at the time of the survey, Auckland and the South Island were slightly over-represented among participating clinics—at the expense of the rest of the North Island (Table 1).

Table 1. Distribution of all available accident and medical (A&M) clinics in 2001/2, sample, and location of clinics participating in NatMedCa survey

Location	Total number (%) of A&M clinics in 2001/2	Number (%) of A&M clinics included in sample	Number (%) of A&M clinics that participated in NatMedCa survey
Auckland	31 (59.6)	13 (59.1)	8 (66.7)
Rest of North Island	14 (26.9)	7 (31.8)	2 (16.7)
South Island	7 (13.5)	2 (9.1)	2 (16.7)
Total (New Zealand)	**52 (100.0)**	**22 (100.0)**	**12 (100.0)**

6205 visits were logged, and detailed data were obtained from 1,430 attendance—590 (41%) of which occurred during usual working hours (Monday–Friday, 8am–6pm).

A&M practices were staffed by a mean number of 2.7 FTE A&M practitioners, assisted by an additional 0.8 FTE general practitioner—but no community worker participation (Table 2). Slightly more than half of the doctors provided maternity care, but health promotion was less common. Only 25% of practices utilised computerised patient records. Most practices were governed by a separate management structure and were operated as a limited liability company. No practice needs assessment was undertaken, but a written policy on complaints was a uniform feature of all practices.

The characteristics of 67 participating doctors are described in Table 3. A quarter were female, with a mean age overall of 40 (over half the practitioners being in the 35–44 age group).

On average, doctors had been in practice for just over 10 years, and had been in the sampled practice only 2.9 years. Over a third of doctors had not trained in New Zealand, with the majority—a quarter of the total—from a country other than the United Kingdom or Australia.

On average, doctors saw nearly 90 daytime patients a week, and to do so worked 6.3 half days with an average of 13.7 patients per half day.

Table 2. Characteristics of A&M clinics

Characteristics	A&M clinics (N=12)	
Personnel (mean number)		
Full-time equivalent (FTE) doctors	2.7 A&M practitioners, 0.8 rostered GPs	
FTE nurses	3.2	
FTE community workers	0	
Access		
Hours open per week (mean)	118.1	
Offering evening surgery hours (%)	100	
Offering weekend surgery hours (%)	100	
Offering booking system (%)	8.3	
Services provided (%)		
Doctors providing maternity care	58.3	
Group health promotion	16.7	
Community worker services	0	
Computerisation (%)		
Computerised patient records	25.0	
Governance (%)		
Separate or external management structure	83.3	
Patient representation in management	0	
Legal practice structure (%)		
Sole trader	0	
Partnership	16.7	
Community trust	0	
Other trust	0	
Incorporated society	0	
Limited liability company	83.3	
Other	0	
Practice needs (%)		
Formal community needs assessment	0	
Locality service planning	0	
Inter-sectoral case management	0	
Quality management		
Written policy on complaints	100	
Written policy for quality management	58.3	
Standard fees (mean $)	**Card†**	**No card**
Child (0–5 years)	6.90	6.90
Child (6–17 years)	21.80	26.70
Adult (18 years and over)	34.40	47.50

† Community Services Card (CSC) or a High User's Health Card (HUHC)—card holders receive subsidised healthcare due to their lower incomes or frequent use, respectively.

Table 4 presents the age- and gender-specific log and visit information as a ratio to the corresponding national population data. Overall, there were more than three times as many patients under the age of 5 years visiting the sampled A&M clinics than national figures would indicate, a slight elevation in the 15–24 years age group, and an under-representation of between a third to three-quarters in the groups aged 35 years and over.

This age-related pattern was most marked for "out of hours" visits, with the under 5s more than four times the national indication, and those over 45 generally a half or less than expected. Visits in the 8am–6pm period were in an intermediate position. A slight difference in gender distributions was also evident, with males disproportionate in the youngest age group, and females over the age of 15 "out of hours" over-represented.

Table 3. Characteristics of participant A&M practitioners

Variables	A&M practitioners (N=67)
Gender	
% Female	26.9
Age (years)	Mean=40.0
<35	23.1
35–44	55.4
45–54	18.5
55–64	3.1
>64	0
Total	100%
Years in practice	Mean=10.1
<6	42.6
6–15	32.8
16–25	21.3
>25	3.3
Total	100%
Years this practice	Mean=2.9
<6	86.2
6–15	13.9
16–25	0
>25	0
Total	100%
Place of graduation	
New Zealand	61.2
United Kingdom	9.0
Australia	4.5
Other	25.4
Total	100%
Mean daytime patients/week	86.6
Mean half-days worked per week	6.3
Mean daytime patients per half-day	13.7

Data on the ethnicity and card status of A&M clinic patients are presented in Table 5. For about 60% of visits, the recorded ethnic group was New Zealand European. The next largest single group was Māori (9.0%), followed by Samoan (6.1%). Taken together, Chinese and Indian accounted for just under 10% of all patient visits, second only to New Zealand European. This, overall ethnic distribution was relatively stable between normal and "out of hour" periods.

Nearly three-quarters of all patients did not have a benefit card of any kind—i.e. either a Community Services Card (CSC) or a High User's Health Card (HUHC). Little over a fifth had a CSC. This pattern was not so marked for visits in normal

hours, where a quarter of patients had a CSC. There was no information on card status for nearly 5% of patients visiting out of normal hours.

Table 4. Ratio of A&M visits to national population, by age and gender (logs and visits)

Gender	All ages	0–4	5–14	15–24	25–34	35–44	45–54	55–64	65–74	75+
Total (All hours)*										
Male	1.01	3.56	0.97	1.22	1.05	0.76	0.56	0.44	0.32	0.47
Female	0.98	3.31	0.95	1.37	1.04	0.66	0.65	0.50	0.32	0.58
Mon–Fri, 8am–6pm †										
Male	1.03	3.09	0.75	1.26	1.30	0.90	0.65	0.63	0.33	0.83
Female	0.94	2.07	1.11	1.53	1.04	0.65	0.58	0.57	0.51	0.57
Other hours †										
Male	0.95	4.73	1.05	0.93	0.83	0.64	0.40	0.22	0.08	0.17
Female	1.03	3.94	0.93	1.51	0.90	0.70	0.59	0.48	0.28	0.82

*Logs; †Visits.

Table 5. Patient characteristics: percentage distribution of A&M patients, by ethnicity, card status, and NZDep2001 decile (log and visit)

Variables	Total (All hours) *	M–F, 8am–6pm †	Other hours †
Ethnicity ‡			
(N)	(5419)	(570)	(666)
New Zealand European	59.6	59.7	58.0
Maori	9.0	11.2	8.0
Pacific	10.9	8.8	13.2
Asian	10.0	11.1	10.7
Other	10.5	9.3	10.0
Total	100%	100%	100%
Card status			
(N)	(6032)	(571)	(803)
No card	75.6	72.3	75.1
Any card (Community services/ High user)	24.4	27.7	24.9
Total	100%	100%	100%
NZDep2001 decile §			
(N)		(534)	(784)
1–3 [poorest]		30.2	31.8
4–6		27.9	28.6
7–10 [richest]		42.0	39.6
Total		100%	100%

Note: missing data have been excluded; *Logs; date and time were not collected; †Visits; ‡Ethnicity was self-reported, with multiple categories allowed. One ethnic category was assigned per patient according to prioritisation of Māori and Pacific people; § NZ Deprivation decile is a measure of deprivation based on the area of residence.

Aspects of the visit characteristics are described in Table 6. For most patients, the clinic was not their usual source of care especially among those patients who presented after-hours. For about two-thirds of patients outside of normal hours, the current visit was the first visit to the clinic over the preceding 12 months.

Although the majority of patients presented with an urgent condition, relatively few were judged by the practitioners to be severe. Self-limiting conditions were more common after-hours. For at least a third of visits, the practitioners did not identify any disability (the proportion was higher for visits out-of-hours). On average, visits lasted slightly more than a quarter of an hour regardless of when they occurred. Patients identified just over one reason per visit at any time during the week.

Table 6. Patient visit characteristics

Variables	Mon–Fri, 8am–6pm	Other hours
Relationship with practice	(n=536)	(n=691)
Not usual source of care [%] (95%CI)	73.7 (70.0–77.4) ‡	84.8 (82.1–87.5) ‡
Number of visits in previous 12 months*	(n=566)	(n=740)
1	48.2	66.4
2	15.2	11.4
3	8.7	4.6
4	4.8	4.5
5	7.4	3.5
>5	15.7	9.8
Total	100%	100%
Maximum	(48)	(67)
Mean (95%CI)	3.3 (2.9–3.7) ‡	2.3 (2.1–2.6) ‡
Urgency (%) (95%CI)	(n=562)	(n=716)
ASAP today	57.2 (53.0–61.2) ‡	72.2 (68.9–75.5) ‡
This week/month	42.9	27.8
Total	100%	100%
Severity [%] (95%CI)	(n=563)	(n=705)
Severe	42.3 (38.2–46.4) ‡	34.5 (31.0–38.0)‡
Self limiting	42.1	54
Not applicable	15.6	11.5
Total	100%	100%
Duration of visit (minutes)	(n=478)	(n=588)
Mean (95% CI)	16.4 (15.0–17.8)	16.0 (15.0-17.0)
Reasons for visit †		
Number of reasons per 100 visits (95%CI)	Male 119 (113–125) Female 115 (109–120)	Male 113 (108–117) Female 119 (114–125)

Note: missing data have been excluded; *Patient-reported; includes the current visit; †Up to 4 reasons could be recorded per visit; ‡Significant difference between normal and out-of-hours visits.

Injury or poisoning was the most common problem identified by practitioners during usual working hours, whilst respiratory illnesses predominated after-hours (Table 7). Nervous system sense organs and infectious conditions were also relatively common after hours. Most problems encountered at the clinics were new or related to short-term follow-up. Visits for preventive care were rare.

Table 7. Frequency of problems (per 100 visits) and percentage of problem status

Common types of problems *	M–F, 8am–6pm	Other hours
	N=590 visits	N=840 visits
	Problems per 100 visits (95% CI)	Problems per 100 visits (95% CI)
Injury/poisoning	32.7 (28.6-36.8) ‡	20.4 (17.5–23.3) ‡
Respiratory	24.9 (21.1–28.8)	32.0 (28.6–35.4)
Nervous system / sense organs	9.7 (7.3–12.0)	13.3 (10.9–15.7)
Skin / subcutaneous tissue	7.6 (5.4–9.8)	7.0 (5.3–8.8)
Infectious/parasitic	6.8 (4.7–8.8) ‡	12.6 (10.4–14.9) ‡
Total problems per 100 visits	119.8 (115.6–124.1)	114.6 (111.8–117.4)
Percentage of problem status (%) (95% CI) †	N=508 visits	N=698 visits
New problem	57.7 (53.4–62.0) ‡	77.7 (74.6–80.7) ‡
Short-term follow-up	30.7 (26.7–34.7) ‡	14.6 (12.0–17.2) ‡
Long-term follow-up	6.7 (4.5–8.9) ‡	2.9 (1.6–4.1) ‡
Long-term with flare-up	3.3 (1.8–4.9)	3.9 (2.4–5.3)
Preventive	1.6 (0.5–2.7)	1.0 (0.3–1.7)

Note: missing data have been excluded; *Up to 4 problems could be recorded per visit; classified according to READ2 chapters; †Status categories are not mutually exclusive; a visit was assigned to a category if any one of its related problems fell into that category; ‡Significant difference between Normal and Out-of-hours visits.

Laboratory and radiology tests were ordered more frequently during normal working hours (Table 8). Visits after-hours were more likely to be provided with a prescription but not other types of treatment (such as health advice, dressings). The most frequent types of medications provided at the clinics were antibiotics and analgesics.

Follow-up or referrals were less frequently arranged for patients who presented after-hours. Referrals were made for about 15% of visits at any time. However, whilst emergency referrals and referrals to medical/surgical specialties were more likely during usual working hours, referrals to non-medical health workers (such as counsellors, physiotherapists, dentists) were more common after-hours.

Table 8. Tests and investigations ordered, treatments and medications provided, follow-up and referral

Variables		A&M: M–F, 8am–6pm (N visits=590) (N problems=707)	A&M: Other hours (N visits=840) (N problems=963)
TESTS AND INVESTIGATIONS (%) (95% CI)			
Any laboratory test		10.3 (7.9–12.8) ¶	4.6 (3.2–6.1) ¶
Imaging		7.8 (5.6–10.0) ¶	3.7 (2.4–5.0) ¶
Other		6.6 (4.6–8.6)	5.5 (3.9–7.0)
Any test/investigation		21.5 (18.2–24.8) ¶	12.7 (10.5–15.0) ¶
TREATMENTS (number) (95% CI) *			
All treatments	Per 100 visits	138 (127–148)	133 (125–142)
	Per 100 problems †	119 (110–127)	120 (112–127)
All script items	Per 100 visits	66 (59–73) ¶	82 (75–88) ¶
	Per 100 problems †	55 (49–61) ¶	73 (67–79) ¶
All other treatment items	Per 100 visits	72 (64–80) ¶	52 (46–57) ¶

	Per 100 problems †	63 (56–70) ¶	47 (42–51) ¶
TYPES OF MEDICATION ‡ (number) (95% CI)			
Antibacterials	Per 100 visits	19.7 (16.1–23.2)	24.5 (21.3–27.8)
Analgesics	Per 100 visits	11.0 (8.4–13.6) ¶	19.5 (16.7–22.3) ¶
NSAIDs	Per 100 visits	3.7 (21.–5.4)	3.8 (2.5–5.1)
Beta-adrenoceptor agonists (tablets)	Per 100 visits	2.7 (1.4–4.0)	2.5 (1.4–3.6)
Corticosteroids topical	Per 100 visits	2.5 (1.2–3.9)	1.5 (0.7–2.4)
Anti-histamines	Per 100 visits	2.2 (0.9–3.5)	2.4 (1.3–3.5)
DISPOSITION (%) (95% CI)			
Follow-up within 3 months		48.0 (43.9–52.0) ¶	36.9 (33.6–40.2) ¶
Referred on ‖		16.1 (13.1–19.1)	14.2 (11.8–16.5)
Emergency		4.2 (2.6–5.9)	2.1 (1.2–3.1)
Medical/surgical specialties		4.2 (2.4–5.9)	2.0 (1.1–3.0)
Non-medical		7.0 (4.9–9.0)	8.8 (6.9–10.7)

Note: missing data have been excluded; *Any number of prescription or other treatments could be recorded; †Up to 4 problems could be recorded per visit; ‡Most frequently prescribed; classified according to Pharmacodes/ATC level 2. ‖Follow-up and referral are not mutually exclusive; one referral is counted per visit; referral types are mutually exclusive; and Emergency referrals are given precedence; ¶Significant difference between Normal and Out-of-hours visits; NSAIDs=Non-steroidal anti-inflammatory drugs.

Discussion

The NatMedCa survey, conducted in 2001/2, provides a rare description of a novel branch of primary care, the A&M clinic, which has arisen in New Zealand over the last 20 years. The clinics are defined by their extended hours, their community location, and the provision of X-ray facilities.

The clinics attract a young, ethnically-diverse clientele but relatively few people who hold CSCs or HUHCs. Most patients do not have an ongoing relationship with the clinic and attend for an urgent but often relatively minor, self-limiting problem. Many attendances relate to new injuries or respiratory problems and antibiotics and analgesics are the most frequently prescribed items regardless of the time of the day.

Follow-up is arranged for between one third to one-half of visits. Referrals are infrequent (about 15% of visits) and relatively stable throughout the week.

The characteristics of patients and visits during usual hours are generally similar regardless of whether they were made during usual hours or at other times. Some differences are evident in the types of problems that people presented with out of hours and either investigations or follow-up were less frequently arranged outside normal hours whereas pharmacological treatment was more frequently provided.

The overall impression is that the medical A&M clinics provide episodic treatment for relatively young patients—mainly related to a new, short-term problem, particularly an injury or a respiratory illness. This picture is fully consistent with the previously published regional description of clinic activities in 1998. It is also compatible with reports from walk-in clinics in Canada that have also documented the preponderance of younger patients, with visits related to infectious illnesses or injuries.[7,8,11]

However, in contrast to these overseas surveys, the users of New Zealand clinics were not more likely to be female or unemployed.[8, 12] Instead, visits by patients possessing

CSCs appear to be less frequent in New Zealand. In addition, attendance rates appear to be fairly constant across all levels of deprivation.

Relatively high local charges for visits at the clinics, regardless of the time of day, are likely to be a significant factor in attracting more affluent clients while subsidised visits for injury-related problems may ensure that significant numbers of lower socioeconomic groups can still attend.

Patients who attend A&M clinics have a wide ethnic diversity. In particular, a high proportion of Māori and Pacific Island people attend the clinics. The presence of a higher proportion of practitioners from a range of ethnic groups underlines some ability of the clinics to provide culturally acceptable health care to a wider ethnic range of patients.

Canadian research based in similar walk-in clinics indicates that the extended hours, central city locations, and the availability of X-ray facilities on-site are widely appreciated by younger patients who disproportionately reside in central city areas, participate in contact sports and work in physically demanding jobs.[13]

Indeed, for over 20 years in Canada, commercial walk-in clinics have sought to occupy a niche between emergency departments and after-hours GP services.[6] Since they first appeared in Western Canada during the early 1980s, walk-in clinics have now become well established in British Columbia, Alberta, Saskatchewan, and Manitoba.[14] Most clinics now offer a range of medical and allied health services including physiotherapy, massage therapy, and chiropractic treatment under one roof.[6, 14] In Canada, many patients attend walk-in clinics in preference to visiting their own GPs, often attracted by the availability of many services under one roof.[9,12]

Some data exist in New Zealand to describe the extent of the expansion in the number of A&M clinics and practitioners. The New Zealand Medical Register indicates that since it was first made a vocational branch of medical practice in 2001, the number of doctors registered as accident and medical practitioners has grown by over 25% per annum to include some 103 doctors by 2004.15 By 2000 it has been estimated that some 2 million consultations (about 9% of consultations in primary care) were provided by A&M doctors.[15]

The arrival of the clinics has not always been welcomed by other primary care practitioners, and claims and counter claims have bounced to and fro about protective practices and unfair competition.[16,17] Empirical evidence about why patients choose to attend the clinics and what they want from primary care providers are uncommon in New Zealand. One rare example documents the preferences of 355 North Shore residents and underlines the importance of continuity of care to many patients.[18]

Among respondents with regular GP contact, some 80% indicated they would attend an A&M clinic after hours—although only 25% suggested they were more convenient and 89% considered that GPs were better value for money. Meanwhile, 78% of patients who had no regular GP welcomed the extended hours and appointment-free schedules at the clinics. [18]

Although the data used in the study was collected in 2001/2, and precedes the Primary Health Care Strategy, it still provides a unique and important snapshot of an area of primary care that has received relatively little research attention.

In Canada, concerns by general practitioners[19] that walk-in clinics may fragment care, increase costs, and compromise the quality of care have not been demonstrated by the limited research that has been conducted.[11,20]

Further work is urgently needed in New Zealand to reliably compare the characteristics of different practice types along with their practitioners, patients, and visits. Additional research should also closely examine the reasons that patients choose different providers and undertake assessments of the relative quality of care that is provided.

Competing interests: None.

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Rat-bite fever: a cautionary tale

Renukadas Sakalkale, Chris Mansell, Deborah Whalley, Kim Wisnewski-Smith, David Harte, Paul Reeve

Abstract

We present the first ever report of streptobacillary rat-bite fever in New Zealand. The patient was a young man who was admitted with systemic sepsis. He presented with a high fever, hypotension, and tender axillary lymphadenopathy. He had been bitten by a rat a week earlier. Blood cultures grew *Streptobacillus moniliformis*, thus confirming the diagnosis. The literature on rat-bite fever is also reviewed.

Rat-bite fever (RBF) is a rare cause of systemic bacterial infection. It can be caused by two organisms, *Streptobacillus moniliformis* and *Spirillum minus*, which are usually transmitted by rat or rodent bites. It is reported sporadically in North America. This is the first reported case of "streptobacillary' RBF and only the third reported case of RBF from New Zealand since the first suspected case was reported in the *New Zealand Medical Journal* in 1919.[1]

Unexplained fever with rash and a history of rodent contact preceding the illness should always raise the suspicion of RBF. Blood culture is diagnostic. Initial therapy with intravenous penicillin followed by tetracycline is recommended. There is no known carrier state. Mortality does occur (albeit at a low incidence), but there are no sequelae in those individuals who recover.

Case report

DR, a 19-old-man, presented to his family doctor with fever, headache, and backache following a bite from his pet rat 8 days earlier. His symptoms had fluctuated over a week, getting worse for 4 days, followed by 3 days of apparent improvement, before recurring. He did not have any respiratory or gastrointestinal symptoms. His temperature was 39.2°C and blood pressure 84/52 mmHg—as recorded by his family doctor who discussed the case with the hospital clinical microbiologist (author: CM); a provisional diagnosis of sepsis was made and the patient was referred for admission. The working diagnosis was that *Staphylococcus aureus* or enterobacteriaceae were the most likely causative organisms.

On arrival in the emergency department he was febrile, with a temperature of 39.8°C, pulse 112/minute, and blood pressure 102/58 mmHg, He had painful left axillary lymphadenopathy. He no longer had a rash but described an appearance suggestive of lymphangitis over his forearm.

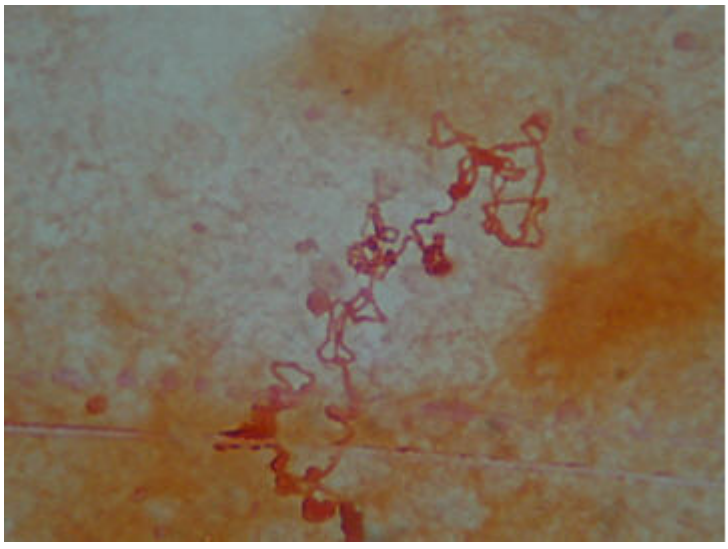
He had been bitten by his pet rat a day before his symptoms started. The bite was between the webspace of his left index and middle fingers, where he had reddish puncture marks with localised cellulitis. His hand and finger movements were normal.

The presumptive diagnosis was rat-bite-related fever. Investigations revealed a white cell count of 17×10^9/L, with neutrophil count of 15.4×10^9/L. His biochemical investigations were within the normal range. Urine microscopy /culture and CSF obtained by lumbar puncture were all normal or negative. His chest X-ray was normal. Because of spinal tenderness and para-spinal muscle spasm, a magnetic resonance imaging (MRI) scan of his brain and spinal cord was performed and this was also normal.

Blood cultures (Bactec Plus Anaerobic/F 442193, Becton Dickinson, MD, USA) signalled positive after 21 hours incubation. The organism grew from 3 of 3 anaerobic bottles and 1 of 3 aerobic bottles. Phenotypic characterisation was performed at the Waikato Hospital Laboratory.

The Gram stain showed filamentous, pleomorphic Gram-negative bacilli with numerous bulbs (Figure 1). Colonies on sheep blood agar were 1–2 mm smooth and grey after 2–3 days incubation. Gram stain morphology from young colonies showed wavy Gram-negative rods up to 5 μm in length, and older plate cultures were more filamentous.

Figure 1. Gram stain appearance from blood culture of patient "DR"; organism identified as *Streptobacillus moniliformis* (×1000 magnification)



The organism was catalase, oxidase, indole and nitrate negative, showed susceptibility to sodium polyanetholsulfonate (SPS), and unlike typical Gram-negative organisms was resistant to nalidixic acid and colistin but susceptible to vancomycin.

Penicillin MIC by E test (AB Biodisk, Solna, Sweden) on sheep blood agar was 0.008 μg/mL. Disc testing by NCCLS methods for non-enterobacteriaceae (M2-A8, M100-S14) indicated susceptibility to erythromycin, tetracycline, ceftazidime, piperacillin, and meropenem, but resistance to norfloxacin and cotrimoxazole. The appearance suggested *Streptobacillus*, and (when the history of rat bite and sepsis was obtained) a provisional laboratory diagnosis of *Streptobacillus moniliformis* was made.

Sequencing of 16s rRNA was performed at Environmental and Scientific Research (ESR), Wellington. The 1086 base pair sequence is available from EMBL, accession number AM503636. Searching the EMBL Prokaryote library using FASTA v3.4t25 found 99.632% identity (a single nucleotide insertion) compared to the *S. moniliformis*-type strain ATCC 14647 and 100% sequence identity to *S. moniliformis* DQ325537. The next closest matches were a variety of *Leptotrichia* spp., at 90 to 91%.

The patient had been started on intravenous flucloxacillin and gentamicin. This was changed to oral amoxycillin after 3 days. He rapidly improved in 72 hours and was discharged home on the 5th day, with 1 week's course of oral amoxycillin. He remained symptom-free at follow-up at 3 months.

Discussion

Rat-bite fever, also called Sodoku,[2] is a systemic illness caused by either *Streptobacillus moniliformis* or *Spirillum minus*.[3,4] A clinical case of RBF was first described in New Zealand in 1919[1] in this journal. The exact incidence or prevalence is not known.[3,5] It is a zoonosis, the rat being the most important reservoir. The bacterium is usually transmitted by a rat bite or by ingestion of contaminated food or water.[3]

Streptobacillus moniliformis is a Gram-negative rod. It is a slow growing organism in culture; the organism is fastidious, requiring 20% serum or 10% whole blood plus 10% CO_2 in a liquid medium for optimal growth. It is also susceptible to SPS detergent in concentrations of 0.0125%.

The presence of this detergent in most commercial blood culture media (0.05% in the medium used in this case) may be why it is seldom isolated in routine medical practice. It is found in the nasopharynx of rat and small rodents. Up to 100% of laboratory and wild rats have been reported as carrying the organism.[6]

Though a normal commensal in the rat mouth, it is unlikely to be isolated by standard methods. Laboratory personnel should have training in the proper handling of the rodents and use protective gloves.[6] *Spirillum minus* is a rarer cause of RBF. It is a spirochete-like organism seen better on a dark-field microscopy.[2,6]

The incubation period of RBF ranges from 1 to 22 days, with a usual onset of 2 to10 days, after a rat bite.[5] In the classical description the original rat bite heals but as systemic symptoms develop, becomes re-inflamed. There is an abrupt onset of fever, which remits in 2 to 5 days and recurs after 3 days.[8] This was similar to the presentation in our case, but due the rarity of the disease in New Zealand and lack of experience of the condition, RBF was not initially suspected.

Indeed, the initial differential diagnosis was of systemic infections of viral or a leptospiral nature and meningitis. However, once the re-inflamed puncture marks between the finger webspaces were evident along with the confirmatory blood culture growing the *sine-qua-non Streptobacillus moniliformis*, the diagnosis was made. A centrifugal rash with erythematous macules, petechial haemorrhages, and sore throat has been reported as concurrent findings.[9] Complications can include abscesses, endocarditis, pericardial effusion, desquamation,[9] and suppurative arthritis.[10]

In untreated patients, the case fatality rate can be as high as 7–10%.[7] Two fatal cases reported by the Center for Disease Control[7] show the importance of including the rat-bite fever in the differential diagnosis of acutely ill patients with rat exposure as well as considering zoonotic infection among persons with occupational or recreational exposure.

Haverhill fever is a streptobacillary bacteraemia due to *Streptobacillus moniliformis* caused by ingestion of rat faeces-contaminated food or water, and two large outbreaks have been reported.[9,10]

Diagnosis of *Streptobacillus moniliformis* is usually established by systemic blood culture or rarely by microscopy of the involved tissue.[2] *Streptobacillus moniliformis* may also be cultured from synovial fluid[6] and can be mistaken for protenaceous debris on microscopy.[11] No serological test is currently available. Due to performance limitations, a previously used slide agglutination test is no longer performed.[5]

Both *Streptobacillus moniliformis* and *Spirillum minus* are susceptible to penicillin; recommended treatment being intravenous penicillin for 5–7 days followed by oral penicillin for a further 7 days.[5] Oral clarithromycin has also been successfully used.[3] The efficacy of any prophylactic antibacterial therapy so far is unknown.[5,12] Most cases resolve in 2 weeks.

Rat-bite fever is not a notifiable disease in New Zealand or other countries but, as an uncommonly recognised pathogen of interest, this case was recorded in the New Zealand Public Health Surveillance Report June 2004. A previous New Zealand case was diagnosed in Auckland in 1980 (J Say, Personal Communication, 2004) but details were not published. A case was also reported in Australia, where the patient had been bitten by a greyhound dog.[13]

We summarise our conclusions from this case as follows:

1. Rat-bite fever is a very rare but important systemic infection.
2. Diagnosis should always be suspected by detailed history and confirmed by early blood culture.
3. The organism has distinctive laboratory features, permitting early presumptive identification.

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Brainstem lesions presenting with nausea and vomiting

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Abstract

Positional vomiting is an important alerting sign for the presence of a brainstem central nervous system (CNS) lesion. Failure to identify another cause of protracted vomiting should prompt consideration of a CNS cause.

Gastrointestinal symptoms have often been associated with lesions of the central nervous system. Hiccups may be associated with lateral medullary lesions.[1] Vomiting may be associated with raised intracranial pressure but may also occur in isolated lesions of the fourth ventricle.[2,3]

A presentation with nausea and vomiting usually prompts a search for a gastrointestinal cause. This path of investigation may be protracted and involve a number of testing modalities. We present two patients who underwent extensive gastrointestinal (GI) investigation before a brainstem cause for their symptoms was identified.

Case 1

A 43-year-old European woman was admitted with a 2-week history of positional nausea and vomiting which was worsened with abrupt head movement. She had no weight loss, no headache, no visual symptoms, and no vertigo. Central nervous system (CNS) examination was normal.

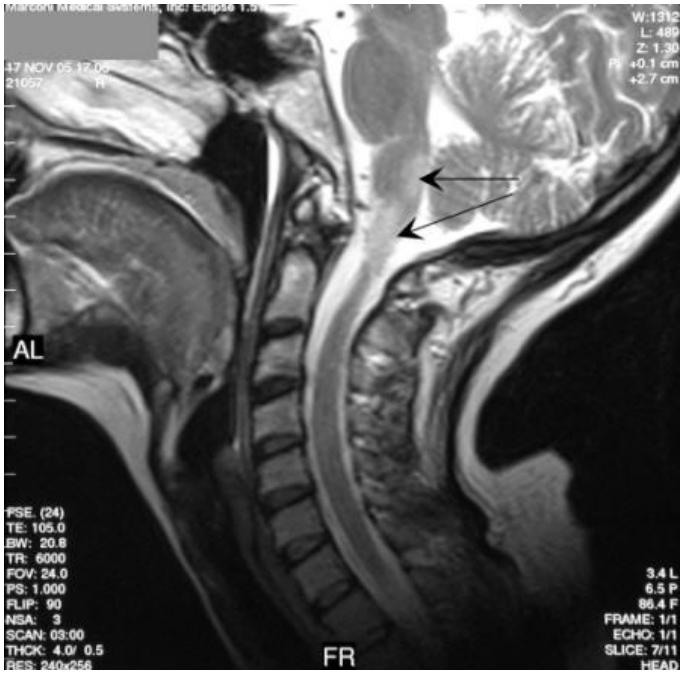
Upper GI tract endoscopy was normal. A non contrast computed tomography (CT) scan of her head and a contrast-enhanced abdominal and pelvic CT scan were also normal. She remained nauseated and was discharged 1 week later with symptomatic improvement with antiemetics.

Three weeks later she developed a variety of head pains—both occipital and frontal—which worsened with change in position. She also reported brief diplopia and positional vertigo as well as skin hyperesthesia over her anterior neck bilaterally.

Review by the neurological service confirmed positional vomiting with additional findings of limitation of terminal neck flexion, ankle clonus, and bilateral extensor plantar reflexes. There was vermal ataxia with difficulty standing unsupported. Examination was severely limited by the patient's vertigo and nausea which was precipitated by movement. However, there was no down beat nystagmus in the primary position of gaze. Pursuit eye movements were normal but there was second-degree nystagmus to the right.

Resource constraints limited access to an immediate magnetic resonance imaging (MRI) scan. A further contrast-enhanced CT head scan remained unremarkable. A subsequent MRI head scan revealed a T2 hyperintensity in the medulla oblongata with some expansion of the brainstem as well as the superior aspect of the cervical spinal cord (Figure 1). There was no hydrocephalus.

Figure 1. T2 weighted MRI scan showing medulla and upper cervical lesions



A cerebrospinal fluid (CSF) sample revealed a white cell count (WCC) of 17 (100% mononuclear); red cell count (RCC) 0, glucose 2.6, protein 0.69 (upper limit 0.60). There was a non-specific increase in IgG antibodies, with no oligoclonal bands present. This lesion best fitted a diagnosis of acute demyelinating encephalomyelitis (ADEM) rather than multiple sclerosis.

The patient had a good response to pulse methyl prednisolone and at 3 months made a near complete recovery apart from a slightly wide-based gait and improving tonic arm spasms.

Case 2

A 45-year-old European woman presented with a 5-month history of nausea and vomiting with a negative gastroscopy. Her symptoms were both spontaneous and positional. The vomiting was worse when leaning forward but not worse with coughing or straining. She also had positional vertigo with a sense of rotation on bending and angular head rotation which improved with lying still. The vomiting was occasionally associated with momentary occipital and frontal headaches. She had a background of rheumatoid arthritis and reactive depression.

On examination, the features appeared to be those of positioning nystagmus of the horizontal semi circular canal. In retrospect, these were really of periodic alternating nystagmus that was brought on by positional change. There was no other central abnormality of eye movement and no downbeat nystagmus in the primary position of gaze or on positional testing. She moved cautiously but gait, Rombergs, and heel toe walking were all normal. The remaining CNS examination was normal.

An MRI brain scan revealed a 3.5-cm fourth-ventricle ependymoma which was successfully resected 2 weeks later (Figure 2 and Figure 3). She gradually improved

over the following months. Unfortunately, however, she developed a post radiation myelopathy after 12 months.

Figure 2. Brain MRI showing enhancing lesion in brain stem

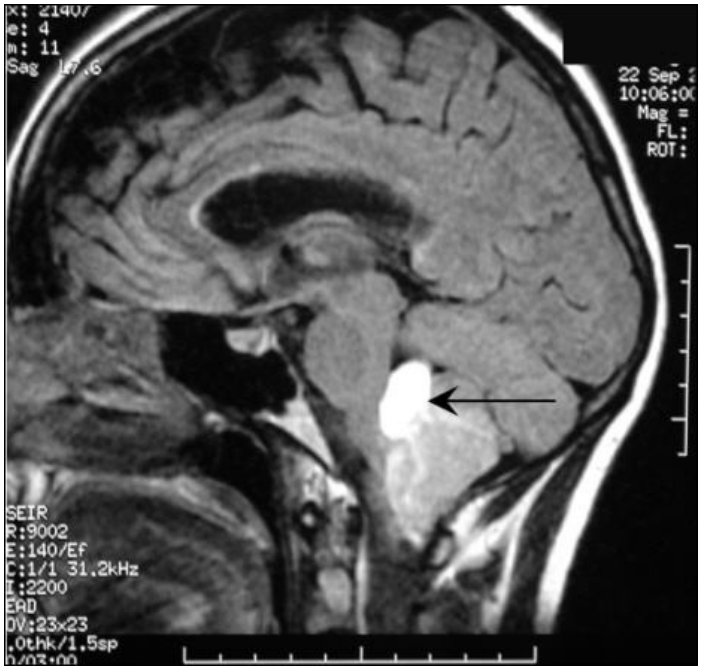
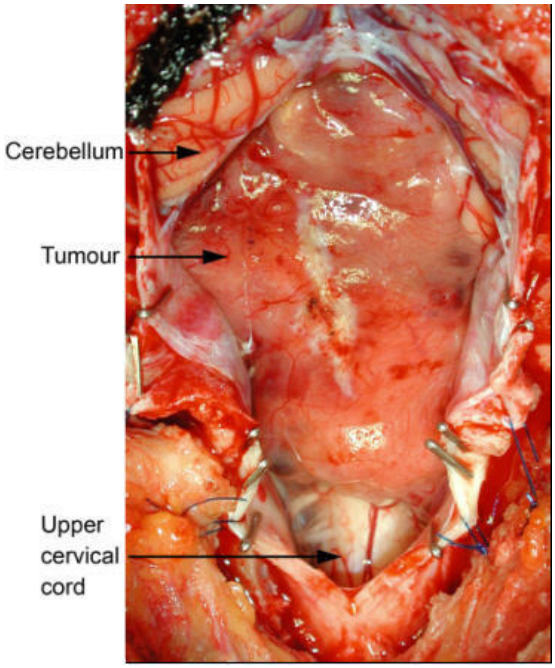


Figure 3. Intraoperative photo with tumour *in situ*



Discussion

While nausea and vomiting are non-specific symptoms, their association with headache or positional vomiting should prompt consideration of a central cause. Failure to correctly identify such an intracranial lesion may subject the patient to unnecessary morbidity and delay appropriate treatment.

Vomiting is produced by both humoral and neuronal stimuli. Within the brainstem, the area postrema in the base of the fourth ventricle contains a zone that is sensitive to blood-borne stimulants such as drugs, toxins, and various peptides. Any lesion that directly stimulates the fourth ventricle or the nucleus tractus solitarius can present with vomiting. Inflammatory lesions in the dorsal component of the medulla have been implicated in presentations with sustained vomiting and hiccups.[4]

There are no controlled trials for the evaluation of chronic nausea and vomiting, and the diagnosis is therefore based on "expert opinion".[5] With isolated spontaneous vomiting, a careful neurological history should be obtained routinely and then reevaluated, especially in the presence of negative gastrointestinal investigations. The presence of positional vomiting is an important discriminator suggesting a CNS lesion.

These two cases demonstrate that a posterior fossa lesion may not always present with a 'full house' of neurological signs. Case 1 had clear cerebellar ataxia with mild eye movement abnormalities. Case 2 had a mild eye movement abnormality, but no

ataxia. This underlines the importance of a careful assessment of associated symptoms with unexplained vomiting.

Truly isolated vomiting without other CNS symptoms is unlikely to be of CNS origin, but if no gastrointestinal cause is found despite adequate investigation, then a head MRI scan should be considered.

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Folic acid and neural tube defects in New Zealand: a cautionary tale?

Joanne Dixon

…Rarely has there been a case where the science has been so unequivocal, uncontentious, and universally accepted, yet the development and implementation of appropriate policy continues to be problematic…

Public Health Intelligence Occasional Bulletin #18
Improving Folate Intake in New Zealand: Policy implications
Ministry of Health
September 2003

History

The impact of diet on human fetal malformation was first systematically documented in Europe after the Second World War. Many countries experienced considerable deprivation both during and immediately after the conflict and there was a concurrent increase in observed "birth defects".[1]

By the 1960s there were already suggestions that folic acid (a B group vitamin) might be involved in a particular group of "birth defects"—neural tube defects (NTDs). However, it was not until 1980–1981 that the first intervention studies were published demonstrating that vitamin supplementation, around the time of conception, in women who had a previous child affected by a NTD, led to a reduction in the risk of recurrence of NTD in subsequent pregnancies.[2–4]

In 1983, the MRC Vitamin Study Research Group launched a randomised double-blind prevention trial (in 33 centres in 7 countries) to determine whether folic acid taken around the time of conception could prevent NTDs in women at high risk because of a previous affected pregnancy.[6]

In 1984, another study was started in Hungary[8] which also sought to establish the efficacy of periconceptional multivitamins in reducing the first occurrence of NTD.

Both of these studies, published in 1991 and 1992 respectively, concluded that there was evidence of benefit (the reduction in incidence of NTD both as a first occurrence and as a recurrence). The MRC trial showed a 72% reduction in NTD incidence amongst the infants of women who took the supplement when compared to the offspring of those who did not.

These developments were discussed in a review article in the *Journal of Paediatrics and Child Health* in 1992.[7] The authors noted that despite the limitations in the studies, the evidence was in favour of some aspects of diet, and of folate in particular, being important in the aetiology of NTDs. They suggested that the challenges would be to find ways of improving the folic acid intake of all women of child-bearing age, and to evaluate the effect of any intervention.

The consensus was that folic acid supplementation in the range 360 mcg–4000 mcg per day, taken periconceptionally, would prevent up to 70% of NTDs. The

intervention appeared safe, cheap, and effective as a method of primary prevention of neural tube defects.

So what then happened in the intervening years?

Observation and recording of birth defects like spina bifida became an international collaboration in 1974 with the establishing of the International Clearinghouse for Birth Defects Monitoring Systems (ICBDMS).

These initiatives facilitated the exchange of data on birth defects worldwide, and encouraged further collaborative research. The ICHBDMS now has more than 40 member programmes across five continents (and includes Australia and New Zealand).

There was an observed steady decline in NTD incidence, not all accounted for by the increasing termination of affected pregnancies in the 1990s. General improvements in nutrition and diet are thought likely to have also contributed.[10]

Around the World

United States (US)—In 1991, the US Government recommendation to women who had a previous pregnancy with NTD was to take 4000 mcg folic acid daily for at least 1 month prior to conception.

In 1992, the recommendation was for all women of child-bearing age to consume 400 mcg folic acid per day, periconceptually, to reduce the risk of NTD occurrence.

In 1998, the US Government introduced mandatory fortification of all cereal grain products with 140 mcg of folic acid per 100 g of flour. In addition, they also recommended periconceptual supplementation with 400 mcg folic acid for all women except those with a history of a prior affected pregnancy (where the recommended supplementation was still 4000 mcg).

After these initiatives, median folate levels in non-pregnant women of reproductive age more than doubled,[10] and the US reported a drop of 30% in cases of neural tube defects.[14]

United Kingdom (UK)—In 1991, the UK Government recommended women with a previous NTD pregnancy take 5000 mcg folic acid periconceptually.

In 1992, this advisory was extended to all women and the dosage was reduced when the tablet availability had been arranged.

In 1995–1996, there was a national media campaign to improve folic acid intake by fortification (consumption of foods fortified with folic acid), supplementation, and dietary change.

In 2000, the COMA report[11] recommended flour fortification (240 mcg folic acid per 100 g of flour), periconceptual supplementation (4000 mcg for the high risk group and 400 mcg for all other women), and dietary consumption of folate-rich foods.

In 2004, the Health Ministry decided not to proceed with the COMA recommendations (due to outstanding concerns about B12 deficiency in older people).

Australia—The number of cases of neural tube defects in Australia has been reduced by voluntary fortification of some foods, but this benefit has not been distributed across all of Australian society.[10] The largest reduction of cases of disability from

neural tube defects has occurred because of antenatal detection (and consequent pregnant termination), to the extent where (in 2005) NTDs were the leading cause for late-stage termination of pregnancy in Australia.[10]

New Zealand (NZ)—NZ has contributed annual figures to the ICBDMS for about 25 years. These figures are collected by the Ministry of Health and include livebirths and stillbirths with a range of congenital abnormalities, including NTDs. However, because NZ has not collect the same data from terminations of pregnancy, the statistics are inevitably incomplete when it comes to the incidence of congenital abnormalities.

For NTDs, this lack of data from terminations is critical, as there is good evidence from many countries that the majority of affected cases are identified on antenatal ultrasound and terminated in the second trimester.[10]

Public health initiatives in NZ

In 1993, in the NZ Sunday Times, there appeared an article noting:

> The Department of Health recommends women eat plenty of green leafy vegetables to reduce the risk of their baby being handicapped

In the same article, they quoted the Principal Medical Officer as saying:

> …the abundance of vegetables in NZ meant that there was no need to recommend 'pills and potions' to pregnant women. Instead they should simply ensure they ate plenty of green vegetables, lightly steamed, and salads

In the same year (1993), a letter to the *New Zealand Medical Journal* drew attention to the results and recommendations of the UK-MRC Trial.[6] The then Public Health Commission issued a "letter to health professionals" and a public statement:

> All women planning a pregnancy should take a daily supplement of folic acid, 5000 mcg, for 4 weeks prior to conception and for the first trimester of pregnancy

In 1995, when smaller 800 mcg tablets became available, the Ministry of Health amended its advisory to include the lower dosage for women planning a pregnancy who had no history of NTDs, and the higher dose (5000 mcg) for women with a prior history. The revised advisory noted that diet alone was not sufficient to provide the levels of folic acid needed to reduce NTD risk. A target was set to "reduce spina bifida live birth incidence to one-third of the 1993 level by 1997".

In 1999, another publication from the Ministry of Health, *Folate, Folic Acid, and Health* again noted that "the proportion of infant deaths due to birth defects remained at 24%". The document further explored the levels of folate in women in the reproductive age group in NZ (low), and the scientific evidence supporting the health benefits of fortifying breads with folic acid.[12]

In 2001, the Ministry of Health reprinted its brochure *Folic Acid and Spina bifida*, with advice on periconceptional supplementation.

In 2003, the Ministry released its Public Health Intelligence Occasional Bulletin Number 18: *Improving Folate Intake in New Zealand: Policy implications*.[13] This document reviewed the (then) recommendations, and noted that NZ women did not have a sufficient daily intake of folic acid to reduce the risk of NTDs.

The options reviewed included the "status quo" (improving dietary folate, periconceptional supplementation and voluntary fortification) and mandatory fortification.

In July 2004, Food Standards Australia New Zealand (FSANZ) issued a consultation document, *Proposal P295—Consideration of Mandatory Fortification with Folic Acid*.[15]

This consultation sought to review the background science, literature, and research as well as the future options for reducing NTDs in NZ. The options included the "status quo" (diet, supplements, and voluntary fortification) or mandatory fortification of a food product (bread-making flour or bread) with folic acid. In conclusion, this document noted that improving folate intake in NZ would involve:

- A comprehensive, ongoing national campaign to increase awareness of and consumption of folate through diet, supplements, and fortification in women planning a pregnancy.
- Consideration of mandatory fortification of either bread or flour with folic acid.
- Continuing the policy of recommending daily folic acid supplements to women planning a pregnancy, either in combination with voluntary fortification (status quo) or with mandatory fortification.
- Continuing to monitor NTDs in NZ.
- Improving the reporting of NTDs in terminations of pregnancy.
- Monitoring folic acid intake and folate status of the NZ population.

After the public consultation there was a 2-year wait (until September 2006) before FSANZ issued their response to the submissions received in the consultation. This response noted that "the Proposal for mandatory folic acid fortification of **bread** had been approved by the Board of FSANZ". It was also noted that this represented a "refinement" of the original proposal (which had been for the mandatory fortification of **bread-making flour**) following "targeted consultations with key stakeholder groups".

In October 2006, the 9th Meeting of the Australia and New Zealand Food Regulation Ministerial Council met. The proposal for mandatory fortification of bread was considered. FSANZ were requested to review the proposal with respect of compliance (at each individual bakery?) and monitoring and to report back to the Council in 6 months.

Personal commentary

So why, despite the science and the public health initiatives, have we not made more progress?

The use of folic acid supplements in NZ is still low.[13] Many women appear unaware that folic acid supplements will reduce the occurrence and recurrence of NTDs—if consumed prior to conception and for the first trimester (the critical time for the formation of the spinal column).

To compound the problem, about 50% of pregnancies appear to be unplanned. Anecdotally, some women start their folic acid supplements once they have a positive pregnancy test (usually 6–8 weeks after the last menstrual period [LMP]). This is too late to impact on spinal column formation which is complete by 56 days (8 weeks) gestation. What about diet? Most evidence suggests that less than half of pregnant women change their diet when pregnant.

It is clear that 10 years of public health advice regarding diet, supplements, and voluntary fortification have had limited impact on the true incidence of NTDs in NZ. Indeed, the greatest reduction in livebirths has probably been as a result of terminations of affected pregnancies, as seen in Australia.[10] This experience has mirrored that in Europe.[16]

Equally clear is the science which has demonstrated unequivocally that fortification of flour results in a significant reduction in the incidence of NTDs in the population. Whilst mandatory fortification alone may achieve only some of the estimated 70% reduction seen in the original research trials, the combination of mandatory fortification, periconceptional supplementation, and healthy diets will achieve a very significant reduction in a preventable disability and reduce the number of terminations for major fetal anomalies.

The proposed strategy from FSANZ (October 2006) steps outside the established science (where all the benefit has been demonstrated when folic acid is added to flour at source rather than to bread in the bakery); introduces the further complication of developing standards and monitoring for hundreds of individual bakeries rather than to four flour mills in NZ; and risks a significant impact on the overall effectiveness of the fortification initiative.[13,14,16]

Currently approximately 25 children are liveborn or stillborn with an NTD each year in NZ. An equal or greater number are terminated each year for the same anomalies. Conservatively, over the last 15 years (since 1991), there have been 750 children affected by this preventable disability. At least half of these children were not born but were terminated in the second trimester. The survivors (10–15 per year) face a lifetime of medical intervention and significant physical disability.

Spina bifida is a preventable disability. We are talking here about the primary prevention of "birth defects". Any reasonable person might ask why this seemingly straightforward public health initiative has floundered for so long. A lack of will perhaps? Or, a lack of care?

Recommendations

New Zealand should proceed with a **coordinated program** to reduce the incidence of NTDs in our children. The program should include:

- A comprehensive, ongoing **national campaign** to increase awareness of and consumption of folate through diet, supplements and fortified food in women planning a pregnancy.
- **Mandatory fortification of flour** to a level of 240 mcg of folic acid per 100 g of flour.

- A policy of recommending **daily folic acid supplements** to women planning a pregnancy, at a level of 800 mcg of folic aid per day for the general population and 5000 mcg per day for those women with an increased risk, for at least 8 weeks prior to conception and for the first 12 weeks of the pregnancy.
- The **mandatory monitoring** of all NTD livebirths, stillbirths, and terminations by the Ministry of Health—to evaluate the effectiveness of this public health intervention.
- Continued 5-yearly monitoring of the folic acid intake and folate status of the NZ population.

Conclusion

In 2000, the death of one child playing with a Pokemon toy received international media coverage and led to the recall of 70,000 toys.

In the same year, in Canada, 250 children were liveborn with NTDs and a further 250 were aborted. There was no public outcry, no media attention, and no response from politicians.

> What we need is to feel a sense of responsibility and failure every time a fetus is aborted because of an NTD, or a child is born with this preventable disability
> Dr AGW Hunter, Canada, 2000.[18]

It is our tragedy that the above words still apply in 2007.

Competing interests: None.

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A case of cerebellar haemorrhage

NZMJ 1907;5(23):4–6. Written by H. M. Inglis, M.B., C.M. Edin., and P. Clennell Fenwick, M.B. London., F.R.C.S., Edin., (Christchurch Hospital)

ADMITTED APRIL 21ST. DIED MAY 3RD.

PREVIOUS HISTORY.

Father and mother alive and healthy. No history of Syphilis or Consumption.

Patient has been quite well until recently, when she began to complain of numbness in the left side of the face.

She has often complained of headaches; but these were not very severe. She has also occasionally complained of giddiness. A fortnight ago she became ill. On April 14th she was sick and went to bed remaining there till Tuesday, 16th. She remained fairly well till Saturday, 20th, when she became semi-con-scious arid was sent to hospital.

ON ADMISSION.

Patient is semi-conscious. Speech thick and unintelligible; temperature, 97; pulse, 108. Swallows slowly and with difficulty. Has been constipated, and has occasionally vomited after taking fluids. No vomiting since admission. No discharge from the ears. Patella reflex exaggerated on right side and well marked on left. No ancle clonus. No retraction of the head. Understands what is said to her. Can put out her tongue when asked to do so. The left arm and leg are partially paralysed, and can only be moved with difficulty. No twitchings or convulsions. Shakes her head suddenly as if in play. Urine normal.

This condition remained unchanged till April 24th, when slight ptosis of the left eye was noticed. The pupils are now widely dilated and insensitive on the left side and very sluggish on the right. The pulse is 1.08, weaker, and limbs, are cold. Patient does not move the left arm, but can slowly draw up the left leg. She is menstruating.

April 26th. Examination of eyes by Dr Manning.

Corneae insensitive. Pupils dilated and unequal; both act to light, but right is more active. No optic neuritis. Ptosis of left upper lid. Right eye divergent.

Patient remains in semi-conscious condition, occasionally moaning. She cannot masticate, and food collects in left cheek,

April 30th—Patient is rigid all over. Babinski sign well marked, especially in left foot. The breathing is stertorous. No convulsions. The head and eyes are turned to the side.

May 1st.—Pupils now .are equally dilated. Left insensitive to light, right very sluggish. Breathing quieter. The limbs are not so rigid. Apparently more conscious. Since the 30th she has had incontinence of urine, She is occasionally more conscious.

May 2nd.—Respiration Cheyne Stokes character. Is weaker and cold. The temperature rose at 2 p.m. to 101.8, and she perspired freely. Pulse 140.

May 3rd.—Temperature rose to 104.2. She became cyanosed and died unconscious.

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Arthritis and macroglossia

Varun Dhir, Skanda Shukla, Nigil Haroon, Amit Kumar Chowhan, Amita Aggarwal

A 52-year-old Asian Indian male presented with painful joint swellings (elbows, wrists, fingers) of 2 years' duration, but no morning stiffness. He was self-medicating with prednisone 10 mg and diclofenac 100 mg/day. He recently complained of difficulty in swallowing due to the enlargement of his tongue. He had decreased appetite and had lost 5 kg in weight.

On examination, he had macroglossia (Figure 1) and waxy papules over the medial canthus of his eyes. His wrists, ankles, and knees revealed swelling, with flexion deformity of fingers due to tendon thickening (Figure 2). There was no fluctuation or tenderness. Investigations revealed a haemoglobin level of 8 g/dL, an erythrocyte sedimentation rate (ESR) of 110 mm in the first hour, and a creatinine level of 2 mg/dL.

Figure 1. Macroglossia

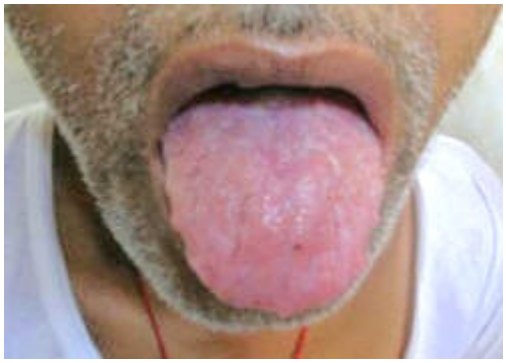
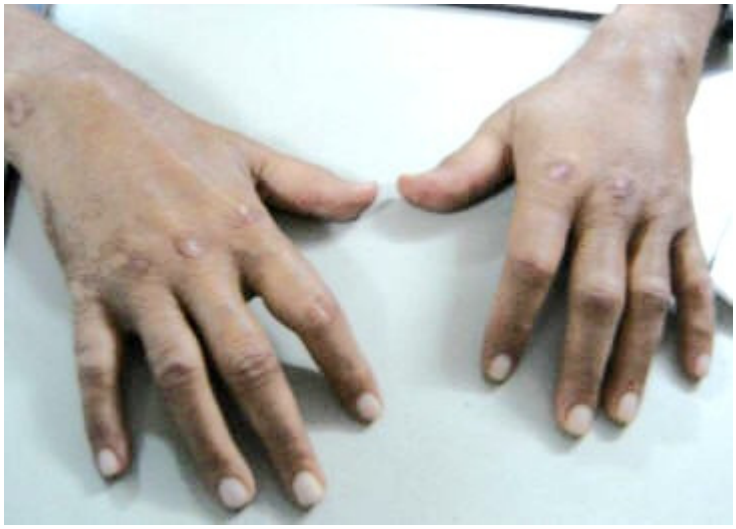


Figure 2. Joint swelling



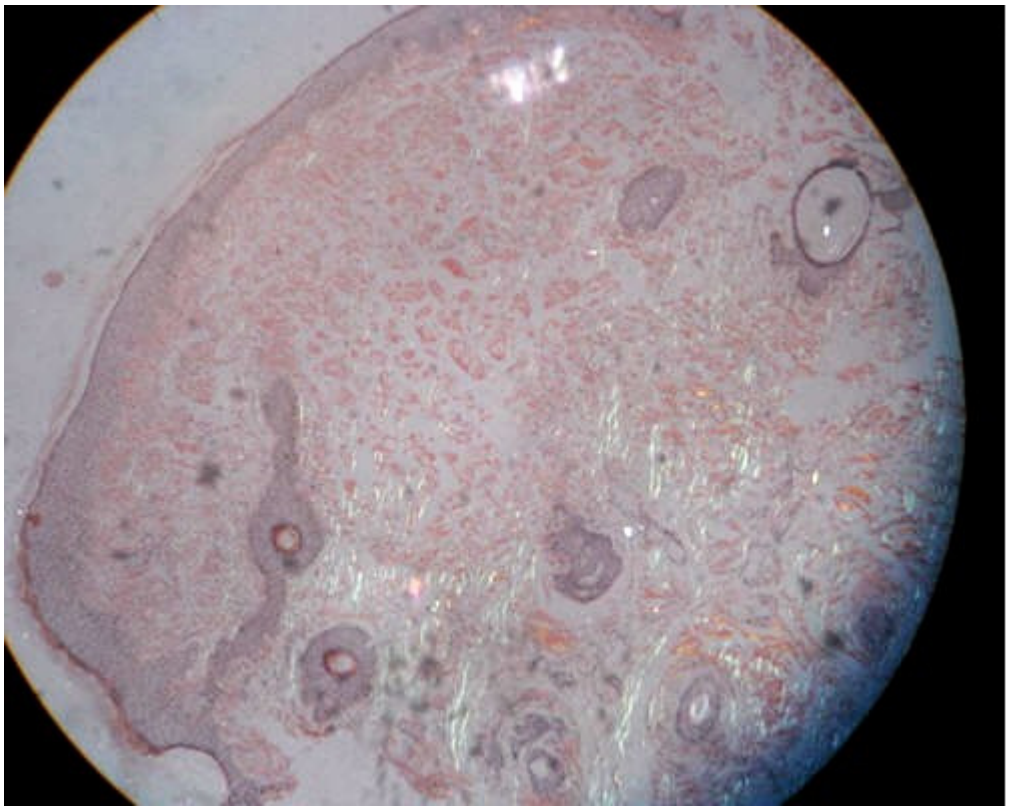
What is the diagnosis?

Diagnosis

Multiple myeloma complicated by amyloidosis causing arthropathy—as confirmed on urine electrophoresis, bone marrow aspirate, and skin biopsy (Figure 3).

Arthropathy is an uncommon manifestation of primary amyloidosis which mimics rheumatoid arthritis. Macroglossia is a more common clinical finding that suggested the diagnosis.

Figure 3. Skin biopsy from facial papules showing congophilic, amorphous deposits with apple green birefringence under polarising light (Congo red stain)



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The demise of nursing (in the United Kingdom)

Two British academic nurses in a provocative paper state that medicine without nursing is an untenable concept: doctors could not practice without highly educated, knowledgeable, and competent nurses as part of the health care team. We certainly agree with that, so what's wrong in the UK? This is what the authors say—in the UK, nursing is haemorrhaging knowledge, skills, and people from all sides. Unqualified health care assistants are taking nursing skills, as cheaper workers with scant education are replacing RNs (registered nurses).

Furthermore, while those workers may be able, for example, make the bed of, and feed, a stroke patient, they cannot assess skin condition, or the effects of facial paralysis while they are so doing, nor assess the effects of the person's illness on the family. A RN does all these, and plans care from this information.

Your scribe also commends this viewpoint (and notes that it also applies to the New Zealand scene). And the solution offered? The public must be told and the politicians should fix it (and nurses should wake up and protest more).

J R Soc Med 2007;100:70–4

Clinical benefit from animal research?

This controversial topic has recently been subject to meta-analysis and editorial commentary in the BMJ. An international assembly of authors conducted meta-analyses of all available animal data for six interventions that showed definitive proof of benefit or harm in humans. For three of the interventions—corticosteroids for brain injury, antifibrinolytics in haemorrhage, and tirilazad for acute ischaemic stroke—they found major discordance between the results of the animal experiments and human trials.

Corticosteroids worked for brain injured animals but not humans, whereas antifibrinolytics reduced bleeding in human trials but not in animals. Tirilazad reduced infarct volumes in the animal scene but had a worse outcome in patients.

So where does this leave us?—confused I'm afraid. To compound the confusion, they found consistent methodological flaws throughout the animal data, irrespective of the intervention or disease studied.

BMJ 2007;334:163–4 and 197–200

More (bad) news about influenza

Neuraminidase inhibitors, such as oseltamivir (Tamiflu), are presently the only reliable antiviral option for treatment of influenza infection, the usefulness of the adamantanes (amantadine and rimantidine) has been virtually eliminated by the development of resistance. However over the last 2 years, the medical community has

been tensely following the emergence of resistance to oseltamivir in strains of influenza A.

And now a report from Japan indicating that the influenza B virus may be learning some new tricks. They noted reduced sensitivity to oseltamivir in a small percentage of children infected with influenza B virus. They conclude that in this population, influenza B viruses with reduced sensitivity to neuraminidase inhibitors do not arise as frequently as resistant influenza A viruses. However, they appear to be transmitted within communities and families, requiring continued close monitoring. An accompanying editorial elaborates on this potential hazard.

JAMA 2007;297:1435–42 and 1492–3

Computer-aided screening mammography

Intuitively one might assume that computer input in the interpretation of mammograms could only be useful. This multicentre study from the United States reviewed data for 222,135 women (a total of 429,345 mammograms), including 2351 women who received a diagnosis of breast cancer within 1 year after screening.

Seven facilities (16%) implemented computer-aided detection during the study period. Diagnostic specificity decreased from 90.2% before implementation to 87% after implementation ($P<0.001$), the positive predictive value decreased from 4.1% to 3.2% ($P=0.001$), and the rate of biopsy increased by 19.7% ($P<0.001$).

We don't need a computer to help us work out that computers are harmful in the interpretation of mammograms.

N Engl J Med 2007;356:1399–409

Dyspepsia—how often is upper gastrointestinal endoscopy indicated

Anne Duggan, an Australian gastroenterologist, notes that gastroscopy has a low diagnostic yield. A review of 22 studies investigating dyspepsia found that, overall, findings in 50% of gastroscopies were normal, 12% revealed reflux oesophagitis, 33% gastroduodenal ulceration, and 1.2% malignancy. She also observes that management guidelines recommend two alternatives to gastroscopy—empiric acid suppression therapy, or *H. pylori* testing and treatment. The first of these involves the "omeprazole test"(a simple trial of omeprazole [40 mg twice daily for a week]).

Duggan quotes a paper that claims that this test diagnoses gastro-oesophageal reflux disease (GORD) more accurately than endoscopy, and with a sensitivity of around 80%. And the second recommendation involves serological or urea breath testing and treatment of patients with a positive result, symptomatic patients can be offered empiric therapy with antacids followed by H_2-receptor antagonists, or proton-pump inhibitors. And she concludes with the opinion that endoscopy should be reserved for those with a familial or ethnic risk of upper gastrointestinal cancer; older patients with alarm symptoms such as dysphagia, haematemesis, or weight loss; and cancer-phobic patients; as its role in patient reassurance is small and not cost-effective compared with other strategies.

Med J Aust 2007;186:166–7

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General Practice Fees Review Committees

The first of the government-imposed General Practice Fees Review Committees has met to deliberate on the first of many recalcitrant general practitioners who feel the need to raise their fees by more than the government-appointed LECG report's level of $1.35 per patient per visit—$1.35 on a total visit fee of $57.00 is a 2.4% rise.

The LECG Committee decided to denominate the rise over the patient co-payment of $30.00 assuming a taxpayer GMS contribution of $27.00 (inclusive of the practice nurse subsidy of $3.00 per head per visit for three visits per year) making it a more inflated 4.5% rise to the patient. (This $27.00 level of patient benefit funding is yet to be provided to many patients in the 25 to 44 year old age band in accordance with the last roll out of patient benefits funding promised for 1/7/2007.)

The first recalcitrant GP did the figures, drew up a budget, and came up with a nominal fee rise of $10.00 per visit being needed to improve the practice viability—and pay for the new staff wage rises of around 20%, the Holidays Act 2003 requirements, pay parity with employed colleagues, superannuation and Kiwi Saver expectations, PHO administration and data input required for every patient contact, upgraded computer equipment, 15 years of deferred maintenance, locum fees and after hours on-call payments, plus a return on investment, and a large accumulated deficit as the result of under-funding, and under-charging for the last 20 years.

The GP looked at the numbers of GP visits, called service utilisations, and found that the average patient aged 6 to 65 has a GMS-equivalent GP visit of 2.3 times a year. So $10.00 a visit equates to an increased cost per patient aged 6 to 65 of $23.00 inclusive of GST per year.

To put this in perspective, this is the same net increased cost per patient per year as a 6 cents rise in the price of a pint of milk per day per patient. The proposed fee increase was also still within the existing fee scales charged by Wellington practices as advised on the Wellington IPA website, so did not seem too excessive.

The doctor thought about this and felt that most patients would not ignore their pint of milk a day for a mere 6 cents and he felt that for trying to look after his patients' most valuable asset (their lives), an increase of 6 cents a day was worth it.

He was then struck by a realisation and fell to the floor rolling with laughter. The Minister and Ministry of Health, with DHBNZ and Treasury, had set up a bureaucratic process of Fees Review Committees to deliberate on whether a fee rise of less than 6 cents a day (under 50 cents a week) was fair to patients, affordable, and justified to help with managing their most valuable asset.

When he regained his composure, the doctor stood up and began to weep.

As he wept, he asked himself "Why?" "Why hadn't the Minister and the Ministry understood the futility and extreme pettiness of this system?" "Why had no-one looked at the dollar values involved?" "Why had IPAC, NZMA, RNZCGP, PHONZ, and DHB NZ leaders not managed to persuade the Minister and Ministry what the Fees Review Committees (costing potentially millions of dollars of taxpayers' money)

were being asked to deliberate on…the equivalent of a 6 cents rise in the price of a pint of milk…is it fair to the patients of New Zealand and can they afford it?"

On 1/12/2006, the doctor notified his PHO of the proposed fee increase of $10.00.

On 15/1/2007, the doctor was notified of the DHB's intention to refer the matter to the local Central Region Fees Review Committee for their consideration—as the fee rise was deemed to be 30–100% and exceeded the LECG Committee's recommended fair rise of 4.5% in patient contributions.

Thereafter, many hours were spent collating data, photocopying annual accounts for 4 years, looking at comparative incomes of doctors in hospital practice, advertised locum fees and incomes from practices looking for GPs, ASMS MECA agreements, and local employed GP salary scales and terms and conditions.

The breakdowns of the Practice's service fees for the last 3 years were sent to the Committee and local PHO, and national utilisations were requested. Practice sizes were compared from available data from various PHO reports and Ministry sources, and information requested on subsidies paid (or required to be paid) to local not-for-profit community-owned practices to maintain viability through either ratepayer contributions, subsidised housing, rooms, equipment, vehicles, and rural subsidies and reasonable roster subsidies.

Various reports on income were obtained and reviewed, including the 2002 IPAC report and the Waikato School of Management report 2006 on general practice expenses and "profits"—these reports and information were forwarded to the Fees Review Committee via the PHO to the DHB to the FRC Secretariat to the Chair of the Fees Review Committee who dispersed them to the Fees Review Committee Members, recently described by a DHB official as senior partners in reputable private accounting firms. (The Committee members could have been emailed directly but that wasn't appropriate bureaucratic procedure, apparently!)

Eventually, a time and date for a conference call was set on 15/3/2007 from 10am to 11am with the Members of the Central Regional Fees Review Committee, the Committee Secretariat, and the doctor, supported by his wife, the practice nurse, a chartered accountant of 50 years standing, and a supportive PHO manager.

The Committee consisted of three chartered accountants from Wellington private practices plus another accountant observer from one of the other Fees Review Committees. The questioning showed little understanding of general practice, income sources, differential subsidy rates (based solely on political whim with no evidential basis) for under-5s, practice nurse subsidy components, ACC subsidies, and Care Plus—nor the history of political manipulation of patient subsidies and increasing compliance costs and legislated cost increases and other costs incurred by general practice over the last 20 years or more.

The written questions which had been forwarded 3 days prior to the teleconference were:

- Validation of expenses?
- Impact of increase on patient numbers and patient visits?
- If the increase is not approved, what will happen to his Practice?

- What initiatives are planned now and in the future to advance the Primary Health Care Strategy?
- What is the supply/demand of doctors in Wanganui? Will patients continue to access his Practice, even if a fees increase is applied?
- What is the patient load like? Are his books closed? How far in advance is he booked for appointments?
- How is he utilising his Practice Nurse and what contribution is she making to the Practice?
- What is the current annual leave available to the Practice Nurse and Administrator?

A question asked by one accountant was whether patients could afford the increase? How can there be a deep and meaningful, worthwhile discussion about whether patients can afford 42 cents a week? It was pointed out that (on 1/4/2007) the minimum wage would rise by $40.00 per week, the youth wage by $32.00 per week, and even superannuitants income was to increase by over $10.00 a week—42 cents a week should be affordable therefore.

For superannuitants and patients on low income with several health needs, disability allowance and Care Plus payments and high needs nurse funding were available to reimburse and reduce direct patient expenditure.

At the end of the hour-long conference, the Committee was asked if they had any GP clients and the reply was in the affirmative (No Comment).

On 22/3/2007, the Committee returned its considered opinion.

It did not recommend the fee increase as it was not seen as fair and reasonable to patients.

The doctor was gob-smacked—42c a week is seen as an unreasonable cost to patients by a committee of four senior private practice chartered accountants.

An appeal was lodged.

Will committees approve fee increases for practices paying their doctors in excess of $200,000 or $150,000 or $100,000 per annum as being fair to patients? If fees reviews are approved above the LECG Committee's recommendations, will Practices be able to seek reparation and costs for the time spent on the process from the DHBs? Will that be at an hourly rate? Will an acceptable hourly rate for a solo self-employed GP be $185.00/ hour (the ACC rate paid for A&M clinics and rural consultations) or the real charge-out rate of $450.00 per hour? ($75.00 for a 10-minute standard consultation).

If the answer is no, will there be a legal challenge in the courts about the whole process? If a charge-out rate of $450.00 per hour is seen as reasonable by a Fees Review Committee, how does it justify a charge out rate of $260.00 per hour as unreasonable for a GP seeing 4 patients an hour at $65.00 a consultation? Many not-for-profit community-owned practices are finding the real cost of General Practice is not covered by cheap $30.00 patient fees. How does the Committee feel those Practices are supposed to pay all their costs, including reasonable salaried GP costs?

Is it reasonable for a committee of professional private accountants to expect a full-time GP—in order to earn a salary and benefits similar to a salaried employee, to need to have a practice size of 2000 patients and to work a 40-hour week actually seeing patients at quarter-hour appointments with study, paperwork, meetings, computer upgrades and crashes, practice education, conferences, home visits, resthome admissions, body identifications, hospital visits, terminal visits—to do all this work outside of normal time?

Has the Minister made provision of perhaps upwards of $20 million to the DHBs for the fees review process and appeals and covering of costs of the review and appeal process?

Is this good use of GP time and taxpayers' money? The possible $20 million that this deliberation is likely to cost if 1000 practices go through the process is more than the cost of 2 years of the roll out of the funding of the 25–44 year olds' increased benefits on 1/7/2007.

Do the Minister, Ministry, Treasury, and the Audit Office see this whole farcical process as an intelligent use of taxpayers' money? What is the point of it all—to control GP incomes, costs, or fees? Is there anyone among the Minister's advisors and the Ministry officials who has an understanding of real General Practice—its costs and business structures and the benefits that it provides for the money paid? What input has there been from the Health Workforce Advisory Committee about job-sizing, and incomes and benefits needed to attract and retain quality practitioners in New Zealand?

If the Minister and his advisors can see past the egg on their faces, this first Fees Review Committee deliberation should also be the last.

Put simply, it is a complete waste of taxpayers' money and a terrible example of governance, as permeating throughout the health system, by trying to micromanage every last detail of practice and service provision (both in the private and the public system), and it is completely losing sight of the bigger picture and what can be achieved with realistically very small increases in funding to Primary Care and General Practice.

Who else would set up a royal commission to decide if a 5 cents price increase on a pint of milk can be justified and is affordable to the nation and its people?

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Why is there a decreasing retention of New Zealand junior doctors?

On my return to New Zealand—after being notified by one of my colleagues and reading in the *Sunday Star Times* recently that there is currently a serious junior doctor shortage in New Zealand[1]—I decided to do some research on the topic to try to answer whether the shortage is being contributed to by a decreasing retention of New Zealand junior doctors (as predicted by a study on the effects of student debt on New Zealand junior doctor's intentions);[2] and if so, why.

The most recent statistics I could find were in the Medical Council of New Zealand (MCNZ) Medical Workforce survey in 2004 which came to the conclusion "there has been little change in retention in the last 10 years which after the 3rd-postgraduate year stabilises at 70–80% of the original final class year as measured by annual practising certificates (APCs)."[3]

As the earliest university tuition fee increase was in 1990, with the passing of the 1990 Education Act, I decided to check previous reports to see if MCNZ had reported any data prior to their 2004 report. To my surprise, their survey in 2000 had reported a "decreasing retention of New Zealand graduates up to 15 years postgraduation compared with 1995" and that in 1999 there had been "an increasing loss of New Zealand postgraduate year 2 and 3 doctors overseas to 58% at postgraduate year 3 for those in the 1997 final year class as measured by APCs."[4]

This trend of decreased retention measured by APCs was also found in their 2001 report.[5] On comparing the data reported, it was noted that the report in 2004 had reported (without explanation) different percentages for graduates with an APC by postgraduate year for those in the final class years of 1996, 1997, and 1998 to the reports in 2000 and 2001. In addition, strangely last year's *Fit for Purpose and for Practice* report (which looked into retention) makes no comment on whether there have been changes in retention with the introduction of student debt.[6]

If we agree that there is decreasing retention, the next question is why? Unfortunately, none of the MCNZ Medical Workforce surveys so far have asked why these doctors are not practising in New Zealand, even though they are in the most ideal position to ask, nor could I find any research anywhere else showing why.

As well, the *Fit for Purpose and for Practice* report[7] makes the comment:

> The extent to which debt influences the work/life decisions of students and young doctors in areas such as vocational training, career choice, work location and deciding if and when to leave or return to New Zealand is unclear

Despite the publication of that report, one draws the conclusion that student debt has certainly had a significant impact on recent retention of junior doctors.[2]

Currently I know of 15 of my New Zealand colleagues working in Australia; for 10 of them, being accepted into vocational training in Australia has been a factor. Personally I am currently planning to go wherever I am accepted into my desired area of vocational training.

I see that the Government has increased the trainee intern grant, however it is a pity that this will not benefit doctors like Bevan Jenkins and myself who have already graduated.[8] Good to see, though, that the Government has decided to make the student loan debt interest-free for those living in New Zealand for at least 183 consecutive days a year regardless of whether or not you are studying[9]—which certainly does increase the financial attractiveness of staying in New Zealand. However for those wanting to go overseas, there will still be an impetus to pay off your loan as soon as possible, which may still tempt them into higher hourly rate locuming jobs.

A study by Moore et al into student debt amongst New Zealand junior doctors found factors encouraging us to stay in New Zealand are increased salaries, employer contributions towards student loans, and training opportunities in New Zealand.[2]

The next step in solving this puzzle should be for MCNZ to research why increasing numbers of junior doctors are working overseas.

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Vincent Trevor Eardly Fernando

Radiologist (18 February 1929 – 7 February 2007)

Trevor was born in Dehiwela, a suburb of Colombo in Sri Lanka. He completed his primary and secondary education at St Thomas' College, an Anglican missionary school. He entered Medical College in 1947, and graduated in 1953.

For the next 10 years, he served in various one-man rural hospitals in Sri Lanka. He always recalled those years with immense satisfaction, as he said that he was able to care for the underprivileged.

In 1963, following a decade of long hours with few breaks, he opted for a career change, and chose radiology as his specialty. He trained in England at Guy's Hospital, St Mary's Paddington, and Mt Vernon Hospital.

In 1970, he accepted the post of Sole Radiologist to the West Coast Hospital Board based in Greymouth. He provided an X-ray service to Grey, Reefton, Hokitika, and Westport Hospitals. Trevor took pride in the fact that he was able to maintain this service without a significant waiting list or private practice. He had a great rapport with his staff, who in turn responded with genuine affection and loyalty.

Trevor retired in 1992 and, as he had grown to love the Coast, remained in Greymouth after retirement.

Trevor was a keen bridge, snooker, and billiard player. He enjoyed gardening and took pleasure in distributing his produce during his daily walks. He was also a miniature railway enthusiast, and dabbled in amateur carpentry.

Trevor had a special love of travel, and extensively traversed the globe until he was diagnosed with terminal prostate cancer in October 2005.

He was happily married for 50 years, and is survived by his wife Tina, two sons, and four grandchildren.

Trevor's values and approach to medicine were an inspiration to his children: Tus, a physician/geriatrician in Whangarei; and Charitha, a radiologist in Christchurch.

Tina Fernando (Trevor's wife) wrote this obituary and supplied the photograph.

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University of Otago Faculty of Medicine Freemasons Postgraduate Fellowships in Paediatrics and Child Health for 2008

The above Fellowships or Scholarships are open to University graduates who intend long term to pursue work in Paediatrics or Child Health within New Zealand. The Fellowships include full-time salary for one year with provision for a further year.

Applications close on **22 June 2007** with the Manager of the Faculty of Medicine,
University of Otago Medical School,
P O Box 913,
Dunedin,
from whom further details may be obtained.

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Erratum

Suzanne Marsh, Sarah Aldington, Mathew Williams, Mark Weatherall, Philippa Shirtcliffe, Amanda McNaughton, Alison Pritchard, Richard Beasley. *Complete reference ranges for pulmonary function tests from a single New Zealand population.* N Z Med J. 2006(27 Oct);119(1244). http://www.nzma.org.nz/journal/119-1244/2281

The authors advise that two errors were in the reference equations at publication:

1. In Table 3 for LogPEF, the height parameter is 0.56 (*not* 0.056).
2. In Table 3 for DLCOAdj§/VA, the height variable is –0.40 (*not* –0.04).

Please refer to the above URL to view the corrected copy of the article.

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Adverse Reactions: The Fenoterol Story

Neil Pearce. Published by Auckland University Press, 2007. ISBN 9781869403744. Contains 200 pages. Price $40.00

This is a great book, by a passionate scientist who, against almost insurmountable opposition, helped expose the dangers of fenoterol. Although the events described in the book took place between 1988 and 1990, it has taken Neil Pearce to this time to bring himself to document this history. He was too angry to do it earlier.

The story is an account of how the Wellington Asthma Research Group of Richard Beasley, Carl Burgess, Julian Crane, and Neil Pearce noticed an apparent association between fenoterol and asthma deaths in New Zealand. The Group then set about examining all the data they had available, using the best epidemiological research methodology of the time. What the Group had not prepared themselves for, and why this was a story that had to be told, was the enormous obstruction that transpired (at a local, national, and international level) to both the progress of the studies, and the publications that arose from them.

There were two main groups involved in this obstruction. Not surprisingly, a major group was that of Boehringer Ingelheim, the manufacturer of fenoterol. More surprisingly, the other group, was the Asthma Task Force, set up in 1978 by the then Medical Research Council. The Task Force was chaired Tom O'Donnell, who at the time was Professor of Medicine, at the Wellington School of Medicine, and subsequently became Dean in 1986. The nucleus of the Task Force was Tom O'Donnell, and three other respiratory physicians: Peter Holst, a senior lecturer in O'Donnell's department; Harry Rea, a respiratory physician at Greenlane Hospital in Auckland; and Malcolm Sears, a senior lecturer in the Department of Medicine at Otago University in Dunedin.

In short, Boehringer Ingelheim, led by their New Zealand Medical Director, Doug Wilson, brought into play the full force of a large multinational company's resources to discredit the research from the Wellington Asthma Group. This extended to lavish international meetings involving chosen experts, huge mail-outs to doctors in New Zealand and even attempts to influence the publication process. The Asthma Task Force, in turn, for reasons known only to themselves, were not only resistant to the implication of the preliminary findings, but continued to thwart the ongoing studies. This was interesting, since they themselves had collected most of the data which led to the fenoterol hypothesis.

To their credit they did allow their data (except that from Dunedin Hospital) to be analysed by the Wellington Research Group. Both the Asthma Task Force, and the experts employed by Boehringer Ingelheim, criticised various aspects of the research which lead to the first paper that appeared ultimately in *The Lancet*.

Of course, epidemiological studies can always be criticised, and alone may not prove cause and effect. They are (by their nature) observational, and are only as good as the data that are available. It is not usually possible, often for ethical reasons, to do a

prospective randomised double-blind cross-over study. Therefore it is always easy to find fault with epidemiological studies (indeed it is easy to find fault with any study, even prospective randomised controlled trials).

The critics of the first of three trials—mainly the Asthma Task Force and Boehringer Ingelheim—latched onto anything they could, even if the faults they enunciated created a bias against implicating fenoterol. The Wellington Asthma Research Group responded to the criticisms as best they could, and designed the second trial with the aim of removing some of these biases. The results supported the findings of the first study. Even then, the opponents refused to let go, and found further fault. So a third study was undertaken, which again vindicated the findings of the first two studies.

Finally the world began to take notice, assisted by independent reviews by prominent New Zealand epidemiologists such as David Skegg and Mark Elwood. It has to be said that Boehringer Ingelheim continued to denigrate all of the studies, though less vehemently than previously.

Two other major studies were then published, from outside the Wellington Research Group. The first was a study by Malcolm Sears, one of the members of the Asthma Task Force. He compared asthma symptoms when patients were receiving fenoterol regularly, versus on an "as required" basis, and showed that patients did much worse when they were using fenoterol regularly. Although Sears described the study as involving "regular use of beta-agonists" rather than "regular use of fenoterol," he did subsequently conclude that fenoterol might be particularly dangerous because it was marketed in too high a dose.

The other supportive study came in 1991 from a Saskatchewan Group, led paradoxically by Walter Spitzer and several other members of the Boehringer Consensus panel. The study was funded by Boehringer Ingelheim and looked at asthma deaths in Saskatchewan between 1980 and 1987. Like the New Zealand studies, this was a case control study, which found that patients prescribed fenoterol were 5 times as likely to die as patients prescribed other beta-agonists. This relative risk was even higher than that shown in New Zealand. Thus, with incredible irony, two groups representing the opponents of the fenoterol story proved themselves wrong! Even then, however, their published papers downplayed the link to the fenoterol hypothesis.

While the battles were raging, and no doubt more patients were dying, the authorities in various countries were slow to act upon the findings. In New Zealand, the Ministry of Health was reluctant to embrace the new findings, probably as a result of pressure from the Asthma Task Force, Boehringer Ingelheim, and the Medicines Adverse Reactions Committee (MARC). Finally, when the results became irrefutable, they were unable to maintain their resistance to the findings and, along with Australian and Japanese medical authorities, took action to restrict the use of fenoterol. Needless to say, the asthma death-rate declined in proportion to fenoterol market share. The epidemic was over.

There are a number of other remarkable ironies in this story. Firstly, Julian Crane, who really was the first to raise the fenoterol hypothesis, was (at the time) employed under a grant from Boehringer Ingelheim. Secondly, Dr Doug Wilson, who (as Medical Director of Boehringer Ingelheim New Zealand) led the drug company attack on the fenoterol hypothesis, had (in 1981) published the first report of the epidemic of

deaths in New Zealand related to asthma. This was when he was Professor of Immunology at Auckland Medical School. In this paper he concluded that "the only reasonable explanation is that the change (in asthma death rate) must be a reflection of changes in the patterns of treatment of asthma in Auckland." Thirdly, the data used by the Wellington Research Group as the basis of their first paper, was largely that of the New Zealand Asthma Task Force. In other words, the Asthma Task Force had the data which carried the signal that they not only ignored, but chose to attack.

As an aside, in a stunning gesture of bloody-mindedness, Dunedin Hospital refused to allow their data to be used in the case controlled study. Finally, Professor Tom O'Donnell, who was Head of the Department of Medicine in the Wellington School of Medicine; and the boss of Crane, Beasley, and Burgess; did his best throughout the sorry saga to prevent these junior colleagues from investigating their hypotheses. The irony here is that all three of his young charges have subsequently become leading Professors at the Wellington School of Medicine.

In the end, this was a win to Neil Pearce and his colleagues, and to the people with asthma whose lives were saved. It was a loss for the New Zealand Asthma Task Force and for Boehringer Ingelheim, whose respective behaviours were reprehensible. While it was reasonable for them to question a new finding, particularly if it seems out of left field, it is not reasonable to use power and money aggressively to suppress data with life-threatening implications.

It would be tempting to believe that this has been a learning exercise assigned to history. However it is a saga that reflects human behaviour and it is therefore as relevant today as then. The recent Vioxx story is merely more of the same. I wonder if the long-acting beta-agonists represent the unfolding of another similar story.

This is a wonderful book. Neil Pearce is to be congratulated, not only for his candour, but for creating a great read. It was a hard book to put down. It was written with great passion, yet his residual anger was held to "'between the lines." I recommend this book unreservedly to anyone interested in the story itself, and to anyone who is interested in the pursuit of the truth.

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Essential Clinical Procedures (2nd edition)

Richard W Dehn and David P Asprey. Published by Saunders (Elsevier), 2007. ISBN 9781416030010. Contains 592 pages. AUD$99.00

This is a "how to do it" book of some common clinical procedures. It is written mostly by physician assistants, with that audience in mind but is just as applicable to doctors, nurses, and any other health professionals who undertake clinical procedures.

Each chapter is devoted to a particular skill and each contains useful revision of the relevant anatomy, physiology as well as listing the indications, contraindications, instructions for follow-up, and suggestions for further reading. Each procedure chapter covers the relevant preparation, equipment, useful diagrams, and step-by-step instructions.

The only idiosyncratic part is what was included and what was excluded as an "essential clinical procedure." The expected skills such as venepuncture, arterial puncture, lumbar puncture, and nasogastric tube insertion etc are there. There are also chapters on treating ingrown toenails, sigmoidoscopy, removal of ear wax, and draining abscesses. A nice inclusion is an outline for obtaining informed consent and sterile technique.

Some of the oddities that have also been included though are examination of male genitalia, measurement of blood pressure, and examination of the foot in someone with diabetes (all of which I would regard as clinical examination skills rather than procedural skills). Also included are outpatient coding and giving bad news. All these sections are still useful but it wasn't quite clear why they were included and other aspects of physical examination or patient communication were excluded. Knee aspiration was the only joint included.

It is very clearly written and nicely laid out. It's the sort of book that is nice to have handy when faced with needing to undertake a procedure and needing to remind oneself of the relevant information. It would also be very useful as an accompaniment to formal clinical skills teaching sessions. As such it would be good to have in any clinical skills laboratory.

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