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community referrals—to reduce inequality of inpatients and outpatients, reduce unnecessary workload, and improve waiting times for community patients.

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New Zealand rheumatology in 2004: realising the potential?

John Highton

One of the things that influenced me as a registrar to choose a career in rheumatology was the perception that this was a field in which there was potential for new developments. In particular, it seemed likely that advances in immunology could make a substantial difference to practice. It has taken a long time coming, but it is now happening at last. New investigations have been introduced allowing earlier and more specific diagnosis. This has been complemented by concurrent development of promising new treatments. Together, these developments provide the potential for substantially improved treatment outcomes. In New Zealand, our challenge is to ensure that these advances can be brought to fruition for the benefit of New Zealand patients with rheumatic diseases.

New Zealand rheumatologists have most recently welcomed the introduction of testing for antibodies to CCP (cyclic citrullinated peptides). Antibodies to CCP have developed from a quirky piece of obscure research laboratory information into a practical serologic test: from antibodies reactive with oesophageal slices to an ELISA test available as a commercial kit. Anti-CCP antibodies are present in about 68% of patients with rheumatoid arthritis and are highly specific.[1] This new serologic test can now be added to antibodies to dsDNA offering specific diagnostic information in SLE, and ANCA for vasculitis. Antibodies to CCP help with early recognition of rheumatoid arthritis and give added confidence in early initiation of disease modifying therapy, which we know is more effective and gives better outcomes.[2]

MRI scanning has opened up a whole new world for evaluation of patients with musculoskeletal diseases due to the ability to image details of soft tissues not visible on X-rays. At some stage, we hope that MRI scanning will be sufficient to image changes in cartilage for early diagnosis of osteoarthritis, and that it might be accompanied by some treatments able to modify the course of this common condition. In the meantime, MRI scanning shows substantial promise for early detection of damage due to rheumatoid arthritis. Indeed, work from New Zealand has made a significant contribution in this area and has shown that MRI scanning provides valuable prognostic data in patients with early rheumatoid arthritis.[3]

New treatments have become available to match the improvements in early diagnosis and determination of prognosis. For example, leflunomide has recently been introduced in New Zealand for treatment of selected patients with rheumatoid arthritis.[4,5] This was the first introduction of a new disease modifying treatment for a long time and as such an encouraging development. This has been followed by the development of tumour necrosis factor (TNF) antagonists, which have brought new hope to patients with a number of rheumatic diseases. It is encouraging that these agents are based on at least some understanding of the pathogenesis of joint inflammation and destruction—they are also very effective.[6,7] Patients treated with these agents experience a rapid improvement in well-being due to removal of cytokines which contribute to the malaise and general features which are a miserable

part of these systemic inflammatory diseases. Anti-TNF agents are also very effective interventions for joint destruction in patients with rheumatoid arthritis.[8] Furthermore, they offer new treatment options for patients with more severe psoriatic arthritis,[9] and ankylosing spondylitis.[10]This may prove to be one of the most valuable applications of anti-TNF treatments as current disease modifying agents have little impact in ankylosing spondylitis. Thus, currently available anti-rheumatic treatments have proven ability in improving well-being, reducing joint destruction, and reducing the mortality associated with diseases such as rheumatoid arthritis.[11]

What do we need to do to ensure that these benefits are available to New Zealand patients with rheumatic diseases? In this issue of the journal, Dr Andrew Harrison's investigations show that we have some substantial homework to do. In 1994, the basis for a good national plan for delivery of care to patients with musculoskeletal diseases was achieved.[12] This envisaged delivery of care to patients with rheumatic diseases through regional networks of clinics. To ensure adequate services, it was recommended that there should be approximately 1 rheumatologist per 100,000 population.

Moreover, close linkage was suggested between rheumatology and orthopaedic services to ensure the coordinated medical and surgical care of patients with rheumatic diseases. Specifically, paediatric rheumatology services would be coordinated at a national level with perhaps 2 paediatric rheumatologists in the country. This was a good plan. However, Dr Harrison's paper indicates that nearly 10 years later regional services are highly variable—with provision of rheumatologists ranging between 1 per132,000 population and 1 per 860,000 population. Indeed, the overall level of service provision is well below target, and we have not made the progress in delivery of care that has been evident in the UK and Australia.

Why has the rheumatology workforce not expanded? This is not due to lack of trainees. At the end of last year, we had 10 rheumatologists in training—including two who completed training at that time and a further two so far in 2004. However, the problem is finding posts for these trainees in New Zealand. Consequently, a not inconsiderable number of our former trainees are now working long-term as consultants in Australia and the UK when their services are greatly needed here in New Zealand.

Some of New Zealand's best medical graduates are amongst our recent rheumatologist trainees. Furthermore, several have pursued research as part of their training, and several are PhD graduates. Unfortunately, however, those remaining in New Zealand have not found conditions favourable to continuing their research careers in rheumatic diseases. Others, meanwhile, have left New Zealand having given up trying to extract substantive funding for their research from our major medical research organisations.

Overall, in New Zealand in 2004, we now have the talent, technology and treatments to make an impact on rheumatic diseases well beyond what was possible 10 years ago. We have a very reasonable plan as to how clinical services should be delivered. But what we need now is the recognition of this potential by those who hold the purse strings for clinical services, pharmaceuticals and research. Indeed, with relatively modest funding, we could catch up with Australia and the UK to the great benefit of

New Zealand patients with rheumatic diseases. Now, ‘in the bone and joint decade’, would be a very good time to do this.

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Avian influenza: a public health risk for New Zealand

Lance Jennings

Introduction

Avian influenza A/H5N1 is currently spreading through domestic poultry and a variety of other birds in Asia. This epidemic is unprecedented, both for its immense geographical scale and for its human health implications. Since its onset late in 2003, eight Asian countries have been affected. Human cases with a high fatality rate have been reported in Vietnam and Thailand, both countries with widespread outbreaks in poultry.

The avian influenza A/H5N1 2004 viruses associated with this epidemic are believed to be very volatile. They are antigenically and genetically distinct from the 1997 viruses, which caused the 'Chicken Flu' outbreak in Hong Kong and are thought to have evolved from the A/Goose/Guangdong/96 virus through both a number of reassortment events with other avian viruses, and through the accumulation of stepwise single mutations.[1]

Although 34 human H5N1 cases have been confirmed, there is no direct evidence of human-to-human transmission. One family cluster of four cases in Vietnam has been extensively investigated for this possibility; however, if person-to-person transmission was occurring, larger clusters of disease might be expected. The sequence analyses of the H5N1 viruses recovered from this family have shown them to be similar to the avian virus, providing no evidence of any genetic change.

The emergence of a novel influenza A virus able to be spread efficiently from person to person has occurred three times in the last 100 years (in 1918, 1957, and 1968) and the resulting pandemics have been associated with high morbidity and mortality. The most recent estimates of mortality during the 1918 Spanish (H1N1) Pandemic suggest between 50–100 million people died—2–5% of the world's population. With the 1957 Asian (H2N2), and 1968 Hong Kong (H3N2) Pandemics, the pig is believed to have acted as a 'mixing vessel' for the reassortment of the genes from an avian virus and animal influenza A virus.[2] H3N2 viruses are present in pigs throughout Asia, so infection of pigs with the H5N1 2004 virus is another possible route for the emergence of a genetically modified virus with pandemic potential.

Human influenza A viruses are endemic in tropical and sub-tropical Asian countries, so the risk of a co-infection with H5N1 2004 in a human also exists. Workers engaged in culling operations (which can involve intensive exposure to infected poultry) are at considerable risk, and the use of personal protective equipment and vaccination (with the current trivalent human influenza vaccine) are recommended procedures to decrease the risk of possible dual infections.[3] However, as long as this virus continues to circulate in the poultry population, the possibility of further human H5 infections, the risk of a double infection, and the emergence of a new virus strain through genetic reassortment (with the capacity to spread easily among humans) remains.

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Virus spread

Little is known about the origin of this current epidemic and the mechanism for its rapid spread, however initial seeding of the H5N1 2004 virus through Asia may have been by wild migratory bird species in which most avian influenza viruses cause mild or asymptomatic infections. Some of the recent H5N1 viruses have been highly pathogenic for a wide range of avian species, so other dissemination mechanisms may be implicated. Since the 1980s, the poultry industry has undergone substantial expansion, and the commercial and other movement of live poultry between Asian countries (along with the live bird and wet market practices) may have provided the fertile environment for virus amplification and dissemination in this epidemic.[4]

Control

The control of this avian epidemic is essential to prevent continuing human outbreaks and avert a human influenza pandemic. Control through culling (of infected or potentially exposed flocks), quarantine, and movement restriction are standard World Organisation for Animal Health (OIE) recommended measures aimed at preventing the spread of highly pathogenic avian influenza viruses. To date, in excess of 100 million chickens have died or been slaughtered. Some countries are using strategic vaccination of poultry, however this practice is controversial.

Unlike human influenza viruses, avian influenza viruses can survive in the environment for long periods. Prolonged survival in faeces in the poultry house environment has been shown for up to 5 weeks. However, the lessons learned following the 1997 Hong Kong outbreak clearly show that rapid culling, followed by the introduction of biosecurity measures (including basic hygiene measures) were pivotal to the control of that outbreak and subsequent virus circulation.

Surveillance

The initial control measures of an outbreak are very reliant on disease surveillance. Rapid reporting is required for both national response interventions and for international collaborative support. As this epidemic continues, of considerable concern is the insidious spread through small groups of poultry in widespread rural communities. In China, it is estimated that 80% of its 13 billion chicken population occurs in backyard farms. Outbreaks in rural communities are extremely difficult to identify and difficult to control.

Virological surveillance is as equally important. Virus isolates are required for the confirmation of outbreaks, for understanding the epidemiology of the virus, and for prototype vaccine strain selection. The sharing of viruses and information within the WHO global influenza network has immense benefits to the World community; however, commercial and political considerations in the current epidemic have often taken priority—leading to the poor international transparency of some countries.

Pandemic planning

As this epidemic continues to evolve in Asia, leadership must be taken from the Food and Agriculture Organisation (FAO), OIE, and the World Health Organisation

(WHO) for the delivery of control strategies to the region, and from WHO for preparation for the worst case scenario, the emergence of a virus with human pandemic potential.

New Zealand must continue to be aware of these international efforts and place an increased emphasis on its national influenza awareness programme and pandemic planning activities.

As we enter the New Zealand 2004 influenza season, we can be confident that there is an increased awareness of the seriousness of influenza amongst the public and healthcare professionals. Through the efforts of the National Influenza Strategy Group (NIISG), influenza vaccine coverage has increased to an estimated 63% of the general population aged 65 years and older.[5]

New Zealand has an advanced Pandemic Action Plan. It is one of only five countries in the Asian-Pacific region to have a plan, and one of only four countries globally to have their plans formalised and endorsed by Government. This level of preparedness has been achieved through initiatives such as Exercise Virex[6] and the use of the Pandemic Plan as the framework for the Ministry of Health's 2003 SARS Outbreak Response. The lessons being learned from the current Asian avian influenza outbreak must be used to strengthen New Zealand's public health disaster preparedness.

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Provision of rheumatology services in New Zealand

Andrew Harrison

Abstract

Aims To measure equity of access to rheumatology services and to assess the adequacy of the rheumatology workforce—compared with published recommended levels, United Kingdom levels, and previous New Zealand data; and to provide data to assist in workforce planning.

Methods Data from of the central North Island of New Zealand were obtained during a review of rheumatology services in the Midland region. Complete national public-sector rheumatology workforce data were obtained in a separate survey.

Results This study demonstrates wide geographical variation in outpatient (more than three-fold variation) and inpatient (more than six-fold variation) service volumes *per capita* between the District Health Boards of the Midland region. Outpatient waiting lists were not higher in underserviced areas. Nationally, there were 251,211 people for each full time equivalent (FTE) rheumatologist, compared with the 1999 level of 235,593 population per FTE, and well behind the UK recommendation of 85,000 people per FTE.

Conclusions This study has demonstrated marked variation in access to rheumatology services based on geographical location, as well as evidence of a worsening shortage of rheumatologists in New Zealand.

Advances in pharmacotherapy have potentially improved the outcome of patients with rheumatoid arthritis and other rheumatic diseases.[1] By virtue of their training, experience, and familiarity with these conditions, rheumatologists are ideally suited to advise on the management of patients with inflammatory arthritis and systemic autoimmune disease.[2,3] Recommendations for the ratio of full time equivalent (FTE) rheumatologists to population have varied (1:85,000 to 1:150,000)[4,5] Although the influence of geographic and demographic variables on access to rheumatology services is yet to be formally evaluated, socioeconomic factors are thought to be important.[6]

A review of rheumatology service provision in New Zealand in 1999 highlighted a shortfall of rheumatologists and rheumatology service volumes.[7] Furthermore, longitudinal data from the United Kingdom have shown that the gap between recommended and actual rheumatology workforce levels has been steadily closing over the past decade.[6,8] To date, there have been no published data on changes in rheumatology service provision in New Zealand over time.

In this study, equity of access is evaluated using data from a review of rheumatology services undertaken in 2002/2003 on behalf of the five District Health Boards (DHBs) that comprise the Midland region of the central North Island—Bay of Plenty DHB, Lakes DHB, Tairawhiti DHB, Taranaki DHB, and Waikato DHB. National rheumatology workforce levels are presented to compare equality of access between

DHBs. Current New Zealand workforce levels are then compared with a 1999 New Zealand survey, with published recommendations, and with service levels in the United Kingdom.

Methods

Data on service volumes and waiting times were collected during a review of rheumatology services in the central North Island (Midland region) of New Zealand undertaken between June 2002 and June 2003.[9] Outpatient and inpatient service volumes for the most recent 12-month period available were obtained from all publicly funded rheumatology service providers in the Midland region. Outpatient waiting times were estimated by rheumatologists based on the interval between receipt of the referral letter and first assessment in clinic. A national survey of the rheumatologist workforce was subsequently undertaken by email in September 2003.

Full data for public rheumatologist FTEs from all (21) DHBs in New Zealand were obtained, either directly from individuals, or from Rheumatology Units. DHB population data were obtained via individual DHB websites. Census data and population projections were obtained from Statistics New Zealand.[10] Workforce data are expressed as full time equivalents (FTEs), where one FTE is a full time position. Service volumes are expressed as patient units (PUs), where one new patient visit is equivalent to two follow-up visits, on the basis of relative allocation of time. Inpatient volumes are expressed as numbers of admissions to allow comparison of the data in the format supplied by the different providers.

Results

Outpatient volumes Using data from the Midland region, outpatient consultations provided to the residents of each DHB were compared (Table 1). There is disparity in the level of service provision across the Midland region. Levels of service provision are highest in the DHBs in which regional rheumatology services are based (Lakes and Waikato DHBs) and lowest in DHBs where rheumatology is provided by a visiting rheumatologist (Tairawhiti DHB), or a combination of visiting rheumatologist and resident physician with rheumatology training (Taranaki DHB).

Table 1. Patient units (PUs) per annum, based on DHB of residence

District Health Board (DHB) of residence	Patient Units	PU per 1000 population
Bay of Plenty	2018	11.17
Lakes	2151	21.50
Tairawhiti	362	7.91
Taranaki	664	6.44
Waikato	4390	14.03
Totals	**9459**	**12.91**

Outpatient visit waiting times Waiting times for new patient and follow-up visits in the Midland DHBs are compared in Table 2. New patient waiting times are similar across the region and are within the Ministry of Health recommendations of no greater than 6 months for a first specialist assessment. The expected inverse correlation between service provision (PU/1000 population from Table 1) and waiting time for the various categories of new patient was not seen. In other words, outpatient waiting times were not increased in the DHBs with relatively low outpatient volumes.

Table 2. Outpatient visit waiting times (weeks) in each DHB

District Health Board (DHB)	Urgent	Semi-Urgent	Routine	Follow-up
Bay of Plenty	4	12	24	8–24
Lakes	4	12	24	4–12
Tairawhiti	6	12	20	24
Taranaki	0–4	0–4	0–4	4
Waikato	2	8	14	12

Inpatient volumes The numbers of inpatient admissions are given in Table 3. Among the five Midland DHBs, there was a wide variation in the numbers of residents per 1000 population being admitted to hospital for a rheumatological indication. Admission rates were higher in the DHBs in which the two rheumatology services in the Midland region are based, and lower in the more remote DHBs.

Table 3. Midland DHB inpatient admission numbers per annum by DHB of residence

District Health Board (DHB) of residence	Total inpatient admissions (number of persons)	Admissions per 1000 population (number of persons)
Bay of Plenty	273	1.51
Lakes	183	1.83
Tairawhiti	37	0.81
Taranaki	41	0.40
Waikato	806	2.58
Totals	**1340**	**1.80**

National workforce data Public rheumatology service FTEs in each of the 21 DHBs are given in Table 4. For comparison, data have been expressed as the ratio of population to one FTE, revealing a wide variation between DHBs. At the time of the survey, the Nelson-Marlborough DHB did not have a rheumatologist, and Northland, Waitemata, and Otago DHBs are underserviced relative to the national mean. Data from the Midland DHBs, Wairarapa/Capital and Coast/Hutt Valley DHBs and the Canterbury/West Coast DHBs data have been presented in blocks, as there is considerable cross-boundary movement of i) patients travelling to clinics outside their DHB of residence, and ii) rheumatologists undertaking satellite clinics in neighbouring DHBs. The survey also found that the total number of rheumatologist FTEs in the private sector was 8.2.

Table 4. Provision of rheumatologist full time equivalents (FTEs) by DHB as at 31 October 2003

District Health Board (DHB)	Population*	FTEs	Population per FTE
Northland	146,750	0.17	863,235
Auckland	388,404	1.8	215,780
Waitemata	445,470	0.7	636,386
Counties-Manakau	399,800	1.9	210,421

Midland DHBs: - Bay of Plenty - Lakes - Tairawhiti - Taranaki - Waikato	742,473	4.45	166,848
Hawkes Bay	143,000	0.5	286,000
Midcentral	160,800	0.8	201,000
Wanganui	66,360	0.5	132,720
Wairarapa Capital and Coast Hutt Valley	415,780	2	207,890
Nelson-Marlborough	122,540	0	
Canterbury West Coast	456,000	2	228,000
South Canterbury	52,781	0.3	175,937
Otago	170,739	0.5	341,478
Southland	103,377	0.4	258,443
Totals	**3,814,274**	**16.0**	**238,095**

*Data from individual DHB websites; generally based on 2001 Census data.

Discussion

The data from the Midland region demonstrate a wide variation in the rate of use of outpatient and inpatient rheumatology services there, with residents of the DHBs where the rheumatology services are based having a higher use *per capita* than residents of satellite DHBs. It was notable that relative underservicing in the DHBs peripheral to the service centres did not result in longer outpatient waiting lists than in the DHBs with a high rate of use. Assuming a similar level of need, it seems likely that the variation in the use of services is related to a higher rate of referral to rheumatology clinics in DHBs with easily accessible services, compared to those with limited local services.

Waiting lists are frequently used as surrogate indicators of adequacy of service provision, perhaps because they are easier to measure than true unmet need. Waiting lists, however, do not take account of the unmet need of patients who, due to lack of access to rheumatology services, are referred to a less appropriate specialty or managed in general practice. A significant number of rheumatology patients are being seen in private practice, judging by the 8.2 private sector rheumatologist FTEs that comprise one-third of the national total.

The ratio of population to each FTE rheumatologist varied greatly among the 21 New Zealand DHBs. This variation did not seem to be explained by population size of the DHB or proximity to a main centre. Nationally, there were 16 FTE rheumatologists in October 2003. Using 2001 Census data this equates to 238,095 New Zealanders per FTE rheumatologist. The official population estimate for 31 September 2003 is 4,024,40010, which equates to 251,211 people per FTE rheumatologist.

In a workforce survey in 1999, there were 16.2 rheumatologists in New Zealand, which equated to 235,593 people per FTE rheumatologist. The rheumatologist FTEs

have remained static over the last 4 years, during which time the population has increased by 15,618 per FTE rheumatologist.

In 1994, the National Advisory Committee on Core Health and Disability Services considered 100,000 per FTE to be the optimal population to rheumatologist ratio for New Zealand.[11] In 1995, the British Society for Rheumatology estimated that one FTE rheumatologist is required per 85,000 population, based on clinical need.[4] Since these recommendations were made, there has been a steady improvement (ie, lowering) in population per FTE in the United Kingdom, with a contrasting deterioration in New Zealand (Table 5).

Table 5. Population to rheumatologist full time equivalent (FTE) ratios in New Zealand and the United Kingdom

Source	Population per FTE
1994 New Zealand Core Health Committee recommendation[11]	100,000
1995 BSR epidemiologically based estimate for UK[4]	85,000
New Zealand October 1999[7]	235,593
New Zealand October 2003	251,211
United Kingdom 1997[6]	191,913
United Kingdom 1999[6]	181,707
United Kingdom 2001[6]	164,165

The shortfall of rheumatologists in New Zealand appears to be due to a lack of posts rather than a lack of applicants. Anecdotally, there are currently several New Zealand-trained rheumatologists waiting for consultant posts to become available, and any vacancies that arise are filled quickly.

In conclusion, there is significant variation in the level of rheumatology service provision in New Zealand, both within the region studied and nationally. The rheumatology workforce level is lower than published recommendations, lower than the current level of service provision in the United Kingdom, and lower than the level found in a previous New Zealand survey.

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Skin infections of the limbs of Polynesian children

Florian Finger, Melissa Rossaak, Richard Umstaetter, Udo Reulbach, and Rocco Pitto

Abstract

Aim The aim of this study was to obtain information regarding the incidence of cellulitis or cutaneous abscess in children of Polynesian ethnicity (including New Zealand Maori), and to calculate the relative risk increase versus other ethnicities.

Methods We reviewed all patients aged between 1 to 14 years who were admitted at our tertiary care institution during the year 2000. Ninety-one children (of 10 different ethnicities) with skin infections were identified.

Results The most common diagnosis was cutaneous abscess (46 of 91 cases, 50.5%), followed by cellulitis (45 of 91 cases, 49.5%). The most common location of infection was the lower limb (79.1%). The major pathogenic organisms were *Staphylococcus aureus* and *Streptococcus pyogenes*. All but one of the children had an uneventful recovery. The incidence of infection in the Polynesian children was 137.7 per 100,000, and the incidence in European children (and children of other ethnic groups) was 35.4 per 100,000. In addition, we calculated a relative risk increase of 3.89 (95% confidence interval of 2.33 to 6.52, $p < 0.05$), which underlines the increased risk that Polynesian children suffer from skin infection.

Conclusion This is the first study showing (in detail) how Polynesian children are affected by a high incidence and increased relative risk of skin infections in their limbs (arms and legs). However, further research (to identify whether genetic disposition or social and environmental circumstances are involved) is required.

Soft tissue infections, and particularly infections of the skin, are a well-known problem in the South Pacific area.[7] Polynesian children are prone to bacterial infections, but little is known about the epidemiology of cellulitis and cutaneous abscess of the limbs in this high-risk population.[1] Cellulitis is a diffuse spreading infection of the skin involving deeper tissues than erysipelas [3]—and characterised by pain, erythema, swelling, and heat. Severe forms of skin infections can be limb- or even life threatening.[9] Infection may start from superficial lesions of the skin providing a portal of entry, but in some patients the cause remains unidentified. The most common causative pathogens are *Staphylococcus aureus* and group A streptococci.[2,4,9]

Common complications of soft-tissue infections are bacteraemia, lymphangitis, local abscess formation (or superinfection) with Gram-negative or gas-forming organisms, necrotising fasciitis, myonecrosis, and osteomyelitis[2,3,11]

The objective of this retrospective audit was to calculate the incidence and the relative risk of skin infections among Polynesian children versus children of European origin.

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Methods

In this study, all children aged from 1 to 14 years, and with a soft-tissue infection requiring inpatient treatment at Middlemore Hospital in the period between 1 January.2000 and 31 December.2000 were included—and reviewed using the Plato® computerised audit system.

The following data was recorded for each patient involved: age, gender, ethnic group, and duration of hospital stay. The following diagnoses (listed in International Classification of Diagnoses [ICD]10) were used for selection: cellulitis of face (L03.2), cellulitis of lower limb (L03.10), cellulitis of toe (L03.0), cellulitis of upper limb (L03.10), cutaneous abscess (L02.0-L02.9), and local infection of skin (L08.8).

Fever (>37.5°C), white blood cell count (WBC), serial erythrocyte sedimentation rate (ESRs), and C-reactive protein (CRP) were recorded from all patients. In addition, diagnostic specimens for laboratory evaluation included swab samples from wounds and aspirates. Tissue specimens were Gram-stained.

The method of treatment was noted either as conservative or surgical. The surgical procedure included incision and drainage with washout. Demographic and epidemiological data of the population under the care of our institution were obtained from the Health Profile of Counties Manukau, Auckland.[12]

For statistical evaluation, current software was used to calculate baseline data (SAS 8.02, SAS Institute Inc., Cary, NC, USA). The relative risk interval has been calculated using the Mantel and Haenszel-Method, with asymptotic 95% confidence interval.

Results

Ninety-one cases of skin infection in 91 children were recorded. The most common diagnosis was cutaneous abscess, and the most common site of infection was the lower limb (Table 1). Fifty-three of 91 cases (58.2%) were diagnosed with swab-samples or abscess aspirates.

Table 1. Demographic data and diagnoses of 91 children with skin infection

Age	7.6 years (mean); range, 1 to 14 years
Gender:	
Males	64 (70.3%)
Females	27 (29.7 %)
	Ratio of males/females = 2.37
Hospital stay	3.4 days (mean); range, 1 to 20 days
Diagnosis:	
Cutaneous abscess	46 (50.5%)
Cellulitis	45 (49.5%)
Localisation:	
Upper limb	**19 (20.9%)**
- cutaneous abscess	8 (8.8%)
- cellulitis	11 (12.1%)
Lower limb	**72 (79.1%)**
- cutaneous abscess	38 (41.8%)
- cellulitis	34 (37.3%)

The most common causative pathogen was *Staphylococcus aureus* (33 of 53 cases, 62.2%), followed by *Streptococcus pyogenes* (12 of 53 cases, 22.6%). Methicillin-resistant *Staphylococcus aureus* (MRSA) was identified in 5 cases (9.4%). Gram-negative or sporing organism caused infection in the remaining 3 cases (5.7%). In 38 cases (41.8%), antibiotic therapy was started before admission to hospital, and the

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diagnosis of soft-tissue infection was made on the basis of clinical evaluation and laboratory investigation. Fever on admission was noted in 16 of 91 patients (17, 58%) and there were 75 of 91 cases (82, 4%) with an abnormal white blood cell (WBC) count (mean value 13, 31 x 10^9 cells/L). Fifty of 91 cases (54, 9%) had a raised serial erythrocyte sedimentation rate (ESR) with a mean value of 28 mm in 1 hour.

Only 25 of 91 patients (27,5%) were tested using C-reactive protein (CRP)—with the mean value being 52 mg/L (range: 5 to 123 mg/L). Forty-eight of 91 cases (52, 7%) underwent surgical treatment (incision and drainage) followed by antibiotic therapy. In 43 cases (47, 3%), conservative therapy was employed using antibiotics. The infection resolved uneventfully in 90 children. Osteomyelitis (requiring surgical treatment and prolonged antibiotic therapy) occurred in 1 case (1.1%).

During the observation period, Middlemore Hospital was responsible for the healthcare of a total of 103,900 children.[12] The ratio of Polynesian children to New Zealand European children was 1.04. During the study period, 8251 children required inpatient treatment. 2096 of 4885 Polynesian children (42.3%) were of New Zealand Maori ethnicity. The ratio of Polynesian children to New Zealand European children was 1.45.

Comparison of ethnical group distribution is depicted in Table 2. There was a statistically significant higher number of Polynesian children suffering from cellulitis and skin abscess (73 of 91 children, $p < 0.05$). Thirty-eight (41.8%) of these 73 children were of New Zealand Maori ethnicity. The estimated incidence of skin infection in the Polynesian population was 137.7 per 100,000 (73 out of 53,000). In contrast, the estimated incidence of infection in European children was 35.4 per 100,000 (18 out of 50,900).

Table 2. Admissions according to ethnicity of children living in the urban and suburban area served by Middlemore Hospital

Ethnicity	Population served by Middlemore Hospital	Admissions (for any reason)	Admissions (for skin infections)
Polynesian	53.000 (51%)	4885 (59.2%)	73 (80.2%)*
European	50,900 (49%)	3366 (40.8%)	18 (19.8%)
Total	**103,900**	**8251**	**91**

*The difference is statistically significant ($p < 0.05$).

The calculated relative risk (referring to the total population of children under the healthcare of the hospital) was 3.89 (95% confidence interval of 2.33 to 6.52, $p < 0.05$). The calculated relative risk (referring to the total number of children requiring in-patient treatment) was 2.79 (95% confidence interval of 1.67 to 4.67, $p < 0.05$).

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Discussion

The aim of this study was to obtain data regarding the incidence of skin infections in Polynesian children (including New Zealand Maori), and to calculate the relative risk increase versus children of other ethnicities.

Hill et al[6,7] have shown that people from Pacific Island countries frequently suffer from bacteraemia caused by *Staphylococcus aureus*, and consequently are at a higher risk of developing bone and joint infections. Moreover, children of Polynesian ethnicity (including New Zealand Maori) show a higher incidence of meningitis and pneumonia—the reasons remain unclear, but it has been hypothesised that the high infection rate could be attributed to genetic disposition. Social circumstances are also likely to be involved.[1]

In the present study, the incidence of skin infection of the limb in the Polynesian children was rated 137.7 per 100,000. In contrast, the incidence in European children was rated 35.4 per 100,000. We calculated that the relative risk of acquiring skin infections in Polynesian children was 3.89 times greater than European children. This value refers to the whole population of children under the healthcare of a tertiary hospital. The calculated relative risk increase (referring to the number of all children requiring inpatient treatment) was 2.79. The discrepancy of the values (3.89 versus 2.79) is related to the higher ratio of Polynesian children versus New Zealand European children requiring inpatient treatment (ratio = 1.45:1), when compared to the ratio of the whole population of children under the healthcare of the hospital (ratio=1.04:1).

Two different scenarios could explain this discrepancy: i) the general health condition of Polynesian children is poorer than that of European-ethnicity children, and consequently they require more inpatient treatment, and ii) some children of European ethnicity are also treated in other hospitals. In our opinion, the reality is probably a combination of both scenarios. From the statistical point of view, both calculated relative risk values in this study are correct (with respect to the data they are generated from)—ie, children with cutaneous infection versus all children requiring inpatient treatment, or versus total children population under healthcare in one hospital. Nevertheless, data regarding children ethnicity under hospital care are estimated. In contrast, data of inpatient children ethnicity are accurate.

Cutaneous abscess was the most frequent soft-tissue infection (64%). Specifically, the lower limb appeared to be the most common site of infection, which is supported by findings of other authors.[3-9] It can be assumed that the skin of lower limbs is more often injured during daily life, especially the foot and knee area, thereby providing portals of entry. In our patients, the most frequent pathogens were *Staphylococcus aureus* and *Streptococcus pyogenes*; this finding is also supported by other authors.[4,9]

The low rates of infection caused by Gram-negative bacteria can be explained by the young age of the population studied.[8] Cellulitis is expected to occur more often in association with systemic diseases like diabetes or other immunodeficiencies.[4] Surprisingly, only one child on steroid medication was found in our patient population.

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This is the first study showing (in detail) the increased relative risk of skin infections that Polynesian children experience (versus children of other ethnicities). However, the present study has some major disadvantages. Firstly, this is a retrospective audit regarding a single-hospital experience. Secondly, the observation period was too short to pull together all potential complications following the skin infection, particularly regarding bone and joint involvement. Thirdly, diagnosis of infection has been given without microbiology testing in 42% of children.

In conclusion, this study shows that there is a marked difference in incidence and relative risk between Polynesian and European children in how they suffer from skin infections. Further research is required to identify whether genetic disposition or social and environmental circumstances are involved in this phenomenon.[13]

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Incomplete primary excision of cutaneous basal and squamous cell carcinomas in the Bay of Plenty

Simon Talbot and Brandon Hitchcock

Abstract

Aim To investigate factors associated with pathologically reported incomplete primary excision of squamous and basal cell carcinomas.

Methods All Medlab Bay of Plenty histology reports were obtained for all primarily excised cutaneous basal and squamous cell carcinomas of the skin for the Tauranga and Western Bay of Plenty regions covering the period 1 January through 30 June 2001. Data were analysed according to surgical training, site of lesion, pathology, and location of positive margin involvement.

Results 1833 non-melanoma skin cancer excisions occurred during the 6-month study—including 1126 basal cell carcinomas, 705 squamous cell carcinomas, and 2 basosquamous carcinomas. 257 (14%) were reported as incompletely excised. There was no difference in rates of positive margin involvement for gender or histology. Proportionately, excisions from the nose and ear revealed the highest incomplete excision rates. General practitioners excised 1003 lesions, with a 16% incomplete excision rate. Consultant surgeons excised 695 lesions, with a 12% incomplete excision rate. Surgical registrars excised 123 lesions, incompletely excising 8%. These data are statistically significant ($p < 0.01$). Tumour was most often found at lateral (rather than deep) margins.

Conclusion The incidence of non-melanoma skin cancer is known to be very high in the Bay of Plenty. Pathologically reported incomplete excision rates are nevertheless comparable with other studies. Of all skin cancers, those on the head and neck are most commonly associated with incomplete excision. Trained surgeons have significantly higher complete excision rates.

New Zealand is known to have one of the highest incidences for basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) in the world. A 1982 study in the Waikato, Bay of Plenty, and Taumaranui areas found an incidence of 306 per 100,000 for non-melanoma skin cancers.[1] The baseline incidence increases 10-fold for those patients over 75 years-of-age and is known to double with each 8-10 degree decrement in latitude.[2] A 1991 New Zealand study reports an age-standardised mortality rate for 35 to 74-year-old males of 2.3 per 100,000 for 1980–86, up 44% from 1966-72.[3] As a comparison, *melanoma*-related deaths in New Zealand account for 3.3 and 4.7 deaths per 100,000 in females and males respectively.[4]

Ultraviolet (UV) radiation is a major aetiological factor—producing DNA alterations as well as immune modulation, with a higher risk for those persons exposed to excessive UV light in childhood, or receiving sporadic, heavy exposure.[5]

BCCs account for around 60% of skin malignancies, while SCCs compose about 25%.[6] Non-melanoma skin cancers predominate in the head and neck. In fact, 70–80% of BCCs, and half of SCCs, occur above the clavicle (collar bone).[1,5]

Several treatment methods for BCC and SCC have been described: curettage, primary surgical resection, Mohs' micrographic surgery, radiotherapy, cryotherapy, and laser excision. Surgical resection is most common in the UK (and NZ) with 80% being primarily closed—although healing by secondary intention; skin grafting, and local, regional, or free flaps are possible. A cure rate of 90-95% is usually quoted.[5,6]

Guidelines have been formulated as to which modes of therapy are acceptable for low- or high-risk lesions. Excision with postoperative margin assessment is usually acceptable. Mohs' surgery has been recommended—where excision margins are positive, where 4-mm margins for BCC and 6-mm margins for SCC are not attainable (or 10-mm margins for lesions ≥20 mm), or for other high risk lesions and sites.[7,8] The very high incidence of skin cancer in the Bay of Plenty means that Mohs' techniques are seldom feasible due to the cost, time, training involved, and the inherent lack of resources.

Furthermore, skin cancer represents an ideal model for cancer prevention, early detection, and treatment—since we know the high risk groups, the aetiology is generally well-understood, and tumours are usually able to be detected and treated curatively.[9]

Numerous studies have shown positive margins to be predictive of non-melanoma skin cancer recurrence (about four times as likely to recur),[10–12] although this is not a universal finding for BCCs.[13] The goal of this study was to step back and investigate some of the factors that result in excisions with positive margin involvement.

Methods

Medlab Bay of Plenty provides all private and public sector histology services in the Bay of Plenty. All histology reports of skin basal and squamous cell carcinomas submitted through Medlab were obtained for a 6-month period from 1 January 2001 to 30 June 2001, inclusive. Inclusion criteria were all squamous and basal cell carcinomas submitted for histology following primary excision during the first 6 months of 2001. Residual tumour resections, Mohs' micrographic surgery, staged procedures (in which closure is not performed until margins are known to be clear), and incision or punch biopsies were excluded as much as possible, based on the histology reports and operation notes.

The study population was all people in the Western Bay of Plenty and Tauranga regions (comprising the Tauranga Public Hospital catchment area). The estimated resident population as of 30 June 2000 was 128,300 with a higher proportion of Maori and people aged over 65 years than the New Zealand average.[14]

Operation notes were obtained for all in-hospital procedures to gain additional information not available from the histology report (for example, surgeon performing excision).

Data from these full reports was extracted and entered into a Microsoft Excel spreadsheet based on NHS number, patient age, patient sex, histology of lesion, site of lesion, size of lesion, complete/incomplete resection (and margin involved), and level

of surgical training. Computer analysis was then undertaken using the Analysis Toolpak add-in software for Microsoft Excel 2000.

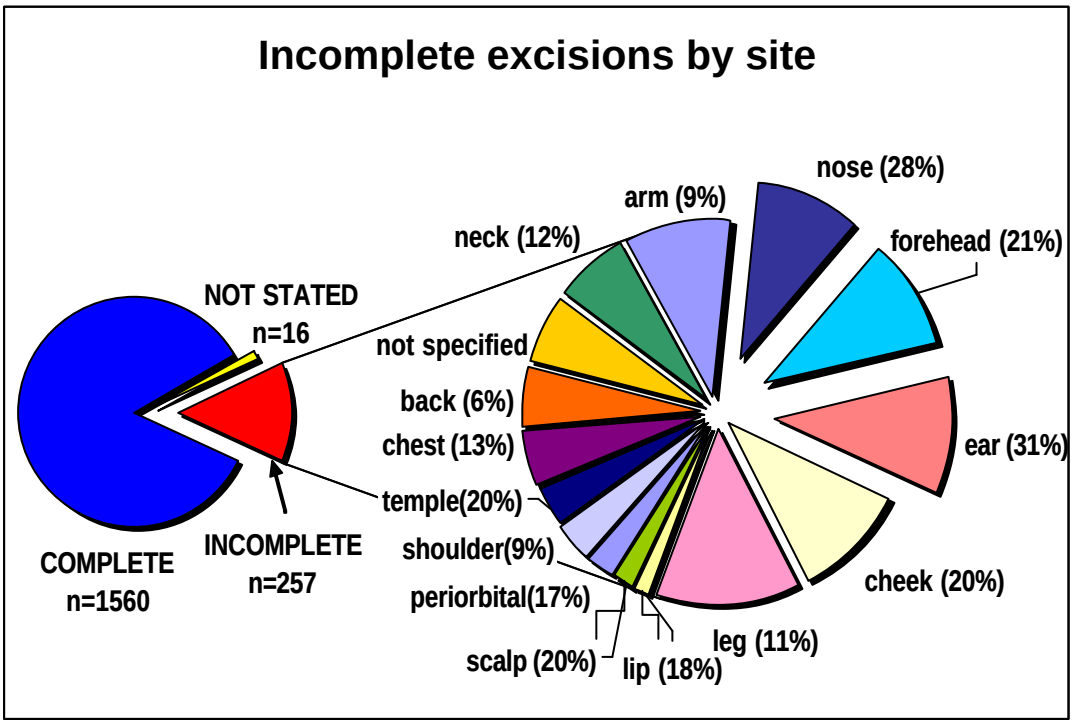
Results

1833 non-melanoma skin cancer excisions from 1320 patients occurred during the 6-month study period. This included 1126 (61%) basal cell carcinomas, 705 (39%) squamous cell carcinomas, and 2 basosquamous carcinomas. Of the total, 1560 (85%) lesions were reported as completely excised, 257 (14%) incompletely excised, and 16 histology reports failed to state the completeness of excision (and thus were excluded for statistical analysis). Of the patients undergoing excisions, their average age was 70 years and 61% were male.

Gender and histology had no influence on rates of margin involvement. However, due to the retrospective nature of this study, and inconsistent reporting, we were unable to accurately assess the subtypes for both squamous and basal cell carcinomas. This may represent a confounding factor for incomplete excision, especially in the basal cell carcinoma group where morpheaform/infiltrative subtypes may predominate.

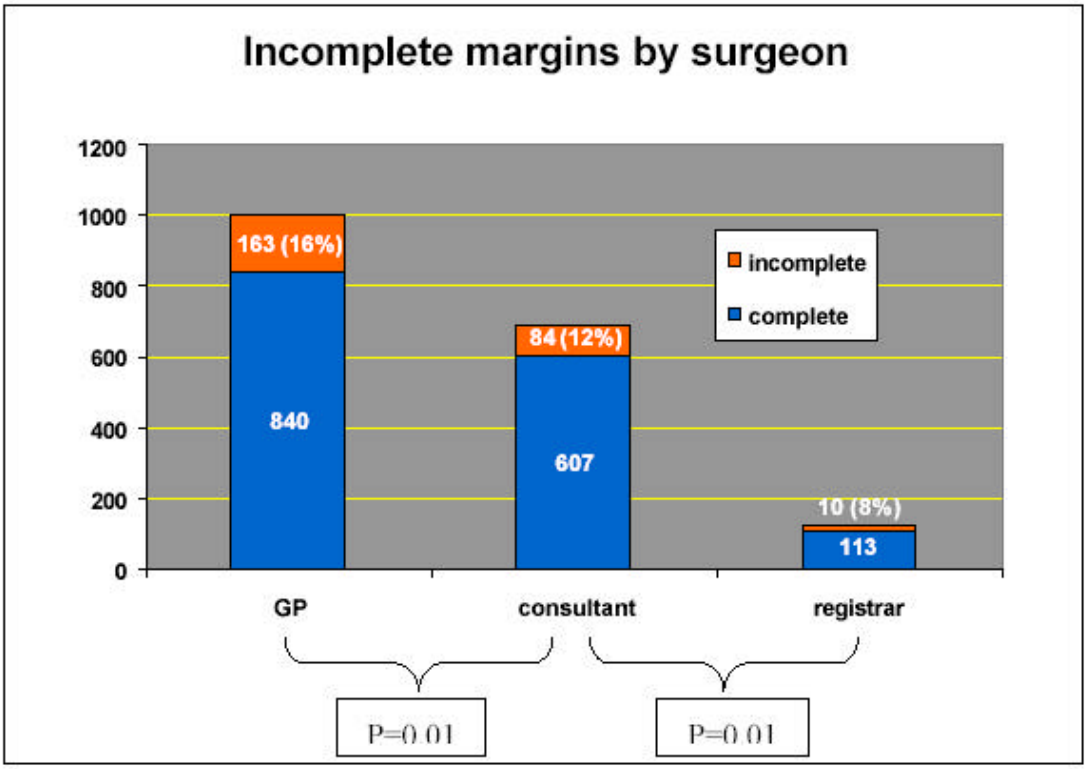
Analysis was initially performed by site of lesion. When proportions of excisions from each site were taken into account, excisions from the nose and ear revealed the highest incomplete excisions rates (28% and 31% respectively)—with all other sites 21% or below. Stated more simply, 40% of BCC and SCC excisions in the study were from the head and neck, but well over half of the incomplete excisions came from these regions (Figure 1).

Figure 1. The left pie shows 14% of excisions as incomplete—broken down as predominating in the leg, cheek, and so on. (Numbers in parentheses refer to the percentages of incomplete excisions for each site.)



Analysis by surgical training revealed 1015 excisions by general practitioners, with a 16% incomplete excision rate. Consultants (including general surgeons, otolaryngologists, plastic surgeons, and dermatologists) excised 695 lesions, with a 12% incomplete excision rate. Registrars excised 123 lesions, incompletely excising 8%. These data were statistically significant ($p < 0.01$) using a chi-squared test with 2 degrees of freedom (Figure 2).

Figure 2. Numbers of excisions and percentages of incomplete excisions by various surgeons. P values refer to differences in rates of incomplete excisions only—with $p < 0.01$ overall.



No comment is possible regarding the influence of the size of lesion on positive margin involvement, as very few lesions were greater than 20 mm in diameter.

Lastly, data was calculated to show which margin was most commonly positive for residual tumour. Specifically, 73% of incomplete excisions were of one or more lateral margins. The deep margin alone was involved in 13%. Eleven percent of incomplete excisions had tumour present at both the deep and lateral margins.

Discussion

A surprisingly small number of studies have investigated the statistics of non-melanoma skin cancers—possibly because data for squamous and basal carcinomas are not gathered by the various national databases. Therefore (combined with the very

high incidence of skin cancer in this region) these data represent one of the largest databases amassed to date.

Our study was undertaken to identify the excision rates of the three large groups: consultants, registrars, and general practitioners. It should be recognised, however, that (within each group) there are subgroups that achieve better (or worse) rates than average.

Our epidemiological data agree with most other studies—these cancers are most common in males in the 56–74 age group.[1,12] The accepted epidemiology is stated by Schuller et al[15] who looked at non-melanoma skin cancers from 1938 to 1973—1796 patients with BCC, 736 with SCC, and 33 with basosquamous carcinomas. These carcinomas predominate in the sixth and seventh decades of a person's life, with a similar age distribution for all types. 11.9% of BCCs recurred after initial treatment, compared with 15% of SCCs and 12% of basosquamous carcinomas—not a significant difference. Recurrence of carcinomas decreased until 5–10 years post-treatment when another surge occurred; the reason for this surge is unclear. However, metastases occurred at a very high frequency: 4–6% in the SCCs and BCCs ($p > 0.05$), which was statistically significantly different from the 0.40% frequency of metastasis for BCCs.[15]

In Northern Hemisphere studies, squamous- and basal-cell carcinomas are most common on the head and neck—specifically, between half to three-quarters of BCCs and SCCs occur in this region of the body in Northern Hemisphere residents.[5] This area of the body also contributes significantly to the mortality associated with non-melanoma skin cancers.[3] However, our data show that only 40% of carcinomas occur on the skin of the head and neck in Bay of Plenty residents. This may reflect a climate (in the Bay of Plenty) in which more body surface (in addition to the head and neck) is exposed to UV radiation.

Depending on the study quoted, incomplete excision rates vary widely; and it should be noted that we are not addressing the issue of what distance is considered a safe margin in this paper. Eleven percent of 723 BCCs excised at Middlemore Hospital in 1986 were reported as incompletely excised (including 70 primary and 12 recurrent lesions).[13]

A paper by Corwin et al[16] looked at excisions performed by 28 Christchurch general practitioners, which included 61 malignant skin lesions—29 BCCs, 28 SCCs, 3 malignant melanomas, and 1 lymphoma. The total incomplete excision rate was 31%. Hallock et al[17] followed non-melanoma skin cancers over 3½ years beyond an initial 4-year study period of excisions. 65 (15.7%) were margin-positive on histology but only 3.1% recurred. SCCs and BCCs were equally likely to be incompletely excised, and body region did not appear to impact on frequency of positive margin involvement. Rates of incomplete excision are often quoted as being highest in the head and neck (16.5% versus 3% elsewhere [$p < 0.001$]), and our data make similar conclusions.[13]

Prevention of tumour recurrence with minimal functional and cosmetic compromise is the goal of any treatment, and the following paragraph summarises the very varied studies on basal cell carcinoma recurrence. Koplin et al[12] states that 93% of BCCs occur on the head and neck, with a 95% cure rate (regardless of mode of treatment). Recurrence rates range from 0.5–14% with an average of 4% and with recurrence

occurring within 15.6 to 30 months. The only factor apparently associated with recurrence in his series was positive margin involvement—with most data supporting the view that complete excision has a 99% cure rate, tumour within 1 high-power-field giving a 12% recurrence rate, and positive margin involvement giving around a 30% recurrence rate.

A systematic review of studies from 1970 through 1997 to establish the true recurrence rates for primary BCC treatment after different therapies quotes a cumulative 5-year recurrence rate for surgical excision ranging from 3.2–8.0% (mean 5.3%).[18] At Middlemore Hospital, 30% of incompletely excised BCCs clinically recurred after observation alone—with a median time to recurrence of 18½ months (during a median follow-up time of 60 months). Recurrence rates were not statistically significantly related to site, sex, prior treatment, involved margin, or histological variant of BCC.[13]

In Dixon's[10] study of 30 recurrent tumours, compared with 74 non-recurrent BCCs with follow-up ranging from 5 to 11 years, the only apparent clinical predictor of recurrence was location—with all recurrent tumours coming from the head (most frequently nose, ear, and temple). Numerous histological factors were associated with recurrence; including, close resection margin. Interestingly, 60% of those persons with recurrent tumours had positive resection margins at the initial resection versus a 13.5% positive margin rate in the non-recurrent group—suggesting that, although positive margin involvement is important, it is not the only factor.

Moreover, Freidman et al[19] investigated 447 BCC resections, all in the head and neck region—8.6% were margin positive. All 15 patients (who did not undergo a re-excision) developed a recurrent tumour, within an average time of 12.06 months—while no recurrences occurred during an average 2-year follow-up of re-excised cases.

Squamous cell carcinoma recurrence has been much less investigated. Recurrence after surgical excision is quoted as 15.7% for SCCs over 2 cm and 5.8% for smaller lesions, while well-differentiated SCCs recur at a rate of 11.8% compared with 25% for poorly differentiated lesions.[2]

The site of the lesion is clearly a major factor influencing the likelihood of an incomplete resection, and few other papers mention the importance of this. From our data, it is clear that a lesion in the head and neck is considerably more likely to be incompletely excised. This seems most likely related to the more technically difficult surgery—with the frequent need for reconstruction in these sites to obtain a cosmetically and functionally acceptable result, thus making it appealing to use narrower margins than elsewhere in the body.

It is clear that incomplete excision is not absolutely related to recurrence. Indeed, several factors may complicate reports of 'incomplete excision'. Firstly, surgery may devitalise residual tumour (mechanically or immunologically). Secondly, marginal extension may sometimes be an artefact conferred by tissue processing. Thirdly, it is plausible that excision occurs on (or very near) the true edge of the lesion, thus causing tumour to appear on the free edge but leaving none behind. Fourthly, formal histology has a degree of subjectivity; and specimens are processed using a bread-slicing technique with intrinsic limitations on the ability to analyse all margins fully. Finally, recurrence is time-dependent and may often not be noticed within a patient's lifetime.[20]

Our data show a significant difference in positive margin involvement depending on the surgical training involved. Only one other paper was found on this topic, which showed that 20 of the 85 general practitioners (versus 13 of the 36 specialist surgeons) had submitted incompletely excised lesions for histology. However, there was no breakdown based on the numbers of excised lesions, making this data incomparable to ours.[21]

Being retrospective and non-randomised, it is important to study which confounding factors may have influenced the selection of patients to each 'surgeon'. We found that general practitioners most often excise arm, back, and leg lesions; while consultants excised most of the nose, scalp, chin, lip, and periorbital lesions. Furthermore, consultants excise larger lesions on average—with 61% of excisions of lesions greater than 20 mm in diameter being performed by consultants. Thus, although more senior surgeons excise larger lesions from more difficult sites, their incomplete excision rates remain relatively low. It seems likely, therefore, that consultants are less constrained in their excisions because of their ability to reconstruct defects. This is particularly so in head and neck lesions where function and cosmesis are critical (and where the less experienced practitioners tend to take narrower margins).

In tumour resection, *which* margin remains positive is also of interest. We found about three-quarters of incomplete margins were lateral—similar to data obtained by Sussman et al[13] and Hauben et al.[20] From a surgical perspective, deep-margin involvement is often more concerning—as re-excision of deep margins is more difficult and recurrences are only detectable in the later stages. However, Hauben quotes a paper by Pascal stating that BCC recurrence is more common when lateral (rather than deep) margins are involved.[20]

Cutaneous basal and squamous cell carcinoma have a very high incidence in the Bay of Plenty. Although acceptable excision (with clear margins) is not the only factor associated with recurrence, it is certainly very important. This study demonstrates that lesions of the head and neck are more likely to be incompletely excised and that those practitioners with increased levels of surgical training are more likely to achieve a complete excision.

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Audit of acute referrals to the Department of Dermatology at Waikato Hospital: comparison with national access criteria for first specialist appointment

Amy Stanway, Amanda Oakley, Marius Rademaker, and Mark Duffill

Abstract

Aim This audit was designed to compare current referral practice with the Ministry of Health elective services National Access Criteria for first Specialist Assessment (ACA) guidelines, to identify specific problems, and (if possible) to improve the use of acute dermatology services.

Method Information regarding referral source, information provided, urgency and diagnostic accuracy, time interval between referral and consultation date, and follow-up arrangements was collected via data sheet on each referral received. We confined the audit to acute referrals—ie, 'immediate and urgent cases' from general practitioners (GPs) that had been discussed with the dermatologist by phone, and internal referrals when an urgent consultation had been requested.

Results More acute referrals came from other hospital departments (74%) than from general practitioners (26%). Acute referrers, especially hospital teams, tended to overestimate the urgency with which a dermatological condition needed to be seen. Information about inpatients was often considered inadequate for triage. GP referrals contained more useful information. GP referral diagnostic accuracy is in keeping with other studies (approximately 50%) but the diagnostic accuracy of hospital doctors is well below this level. All acute referrals were seen within the recommended timeframe. Follow-up patterns were similar (whether referrals came from general practitioners or hospital teams) but for both groups there was a relatively high failure to attend rate.

Conclusions Inappropriate referrals are time-consuming and reduce our capacity for seeing community patients on the waiting list. To improve referral triage, we recommend that a referral letter that clearly specifies the information that should be provided. The majority of acute referrals did not comply with the ACA guidelines. We recommend applying the ACA guidelines to internal acute dermatology referrals (as well as those from GPs) to reduce unnecessary inpatient reviews, and to provide a better urgent service for those persons who truly require it.

Like other specialist services, the Department of Dermatology at Waikato Hospital receives a large number of acute referrals each week from inpatient teams and primary care. However, our impression is that many of these acute referrals to our service are inappropriate. Indeed, many do not comply with the Ministry of Health elective services' National Access Criteria for first Specialist Assessment (ACA) guidelines (at http://www.nzgg.org.nz or Surgical Unit policy, which requires that the referral is made by a consultant when it impacts on the acute admission. Furthermore, other referrals provide insufficient information, or are illegible.

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The National ACA guidelines identify criteria and conditions that help determine how urgently a patient should ideally be seen by a hospital specialist service. Whilst they are primarily developed for referrals from primary care, the same criteria can be used for inpatient referrals. The guidelines state that Category 1 (urgent) cases should be seen within 1 week (eg, erythroderma, eczema herpeticum, pemphigus, toxic epidermal necrolysis), Category 2 (semi-urgent) within 4 weeks (eg, melanoma, acute contact dermatitis, toxic erythema, impetiginised eczema), and Category 3 (routine) within 16 weeks (eg, basal cell carcinoma, acne, eczema, congenital naevus). Category 4 cases include venous ulceration, sexually transmitted diseases, minor cosmetic conditions, and bee-sting allergy and are not seen. Our departmental policy is to comply with these waiting times, although routine cases are currently waiting for more than 16 weeks and up to 26 weeks. The guidelines state—'immediate and urgent cases must be discussed with the specialist or registrar in order to get appropriate prioritisation, and then a referral letter sent with the patient, faxed or emailed *(there may be local variations to this)*'.

Aim

This audit was designed to compare current referral practice with the Ministry of Health elective services' National Access Criteria for first Specialist Assessment (ACA) guidelines, to identify specific problems, and (if possible) to devise solutions to these problems.

Method

When an acute referral was received, a handwritten standard tick-box data sheet was completed by reception staff and/or the registrar (see Appendix 1 at the end of this paper). Additional information was added after the patient was seen in the department. Over a 5-month period (October 2002 to April 2003), the following information was collected on the referrals:

- Time of day and date referral received.
- Date first seen by dermatologist.
- Referral source (general practitioner [GP], inpatient service).
- Referral method (fax, mail, telephone).
- Referral urgency as marked by referrer.
- Referral urgency as judged by dermatologist on basis of referral.
- Referral urgency as judged by consultant if and when patient was seen.
- Referral diagnosis.
- Dermatologist's diagnosis.
- Follow-up arrangements.
- Comments (including adequacy of referral information and reporting previous referrals).

To reduce bias, the referring services were not advised in advance that this audit was being carried out.

Results

There were 200 acute referrals during the 23-week period—17 October 2002 to 1 April 2003. Using data collected in the full months of November, December, January, February, and March, this was an average of 44 referrals per month (range 26-54) and 9.8 referrals each week. Most referrals were received on Mondays, Tuesdays, and Fridays (Figure 1). Forty-nine percent of acute referrals came before midday and 51%

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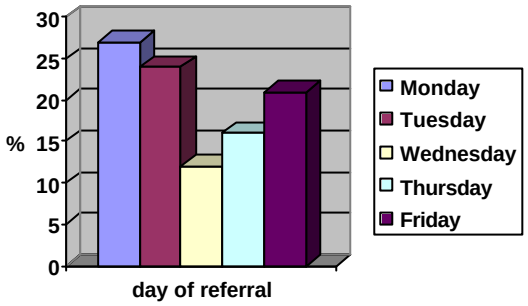
after midday. Ninety-eight (49%) referrals were initially made via telephone, 86 (43%) by fax, 8 (4%) by mail, and 8 (4%) in person. A written referral was eventually received for all patients. The registrar had an additional 15 telephone consultations that did not require the patient to be seen face-to-face. All except six (3%) referrals were seen by the registrar in the first instance.

Figure 1. Percentage of referrals by day of the week



Seventy-four percent of all acute referrals were made by other hospital teams, and 26% by GPs. Thirty-nine percent of all referrals came from the Department of Medicine (52% of hospital referrals). Figure 2 illustrates referral source according to speciality.

Figure 2: Referral source by speciality

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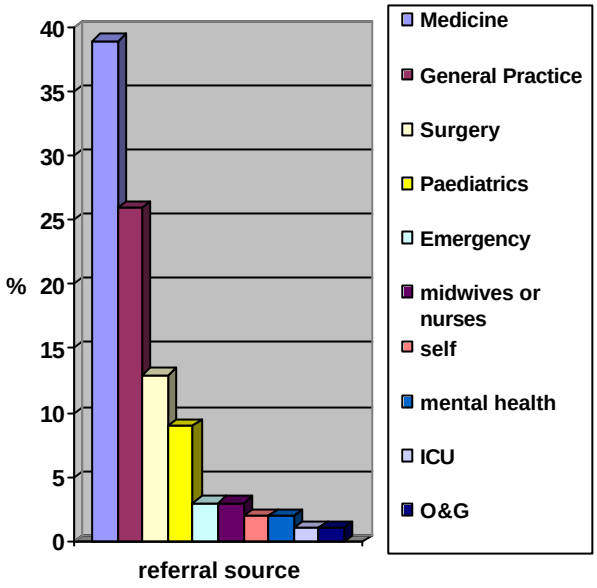
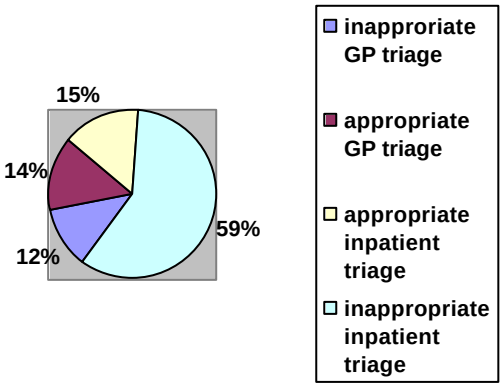


Referral prioritisation based on the referral diagnosis was retrospectively compared with ACA guidelines. If no referral diagnosis was given, categorisation was based on the information given. If there was insufficient information, the referral was categorised as not complying with the guidelines. Overall, 29% of referrals were prioritised in accordance with ACA guidelines and 71% were not (Figure 3). Fifty-five percent of GP referrals were prioritised appropriately by the referrer compared with 20% of inpatient referrals. Sixty-six percent of all acute referrals were marked by the referrer as 'urgent'. However, only 20% of the referrals were correctly categorised as urgent, as 40% should have been marked semi-urgent, and 38% routine. Two percent would not have been seen as they were category 4 referrals.

Figure 3. Overall triage appropriateness



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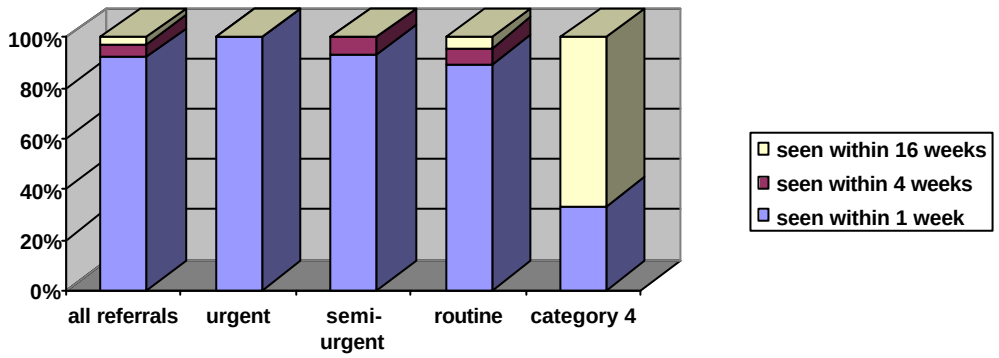
Referral adequacy was assessed according to whether triage was possible for the dermatologist receiving the referral. In most (but not all) circumstances, this required the patient's age, sex, a brief description of their skin complaint, its duration, previous treatment—and other illness, medications, occupational, or social circumstances. Ninety-seven percent of GP referrals and 61% of inpatient referrals supplied adequate information. Thirty percent of all referrals contained insufficient information for adequate triage. Overall, 32% of referral diagnoses were the same as the diagnosis made by the dermatologist who saw the patient. Forty-five percent of GP and 28% of inpatient referral diagnoses were considered correct.

Ninety-two percent of all acute referrals were seen within 1 week, and all acute referrals were seen within the timeframe recommended by the ACA guidelines (Figure 4). All (100%) of ACA Category 1, 93% of Category 2, 89% of Category 3, and 33% of Category 4 patients were seen within 1 week. Of all the acute referrals, 11 patients were not seen (5.5%). These were all inpatient referrals. Four referrals were cancelled on the same day as they were referred 'because the rash had gone', six referrals were returned to the referrer with the recommendation that they bring the problem to the attention of the GP on discharge, and one patient died of an unrelated complaint before being seen.

Figure 4: Time to review according to urgency



Follow-up appointments were arranged for 127 (73%) of patients seen acutely. Sixty-one patients (35%) were booked for review in Dermatology Outpatients, 50 (29%) were advised to see their GP, and 16 (9%) were referred to another speciality such as Plastic Surgery. Two patients (1%) were admitted to hospital. The percentage of patients who received follow-up appointments was similar, regardless of referral source (Figure 5). Of the 61 patients that received follow-up appointments at dermatology outpatients, 24 (34%) did not attend. During the same period, only 22 of the 774 non-acute patients booked to see the registrar (3%) failed to attend. The hospital-wide rate for non-attendance is 14%.

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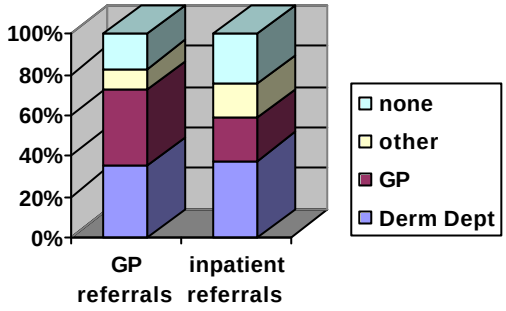
Figure 5. Follow-up arrangements



Discussion

There are few skin diseases that must be seen immediately by a specialist dermatologist. In fact, most patients with an acute dermatological condition are systemically well and present to a primary care physician in the first instance. Generally, GPs prefer to manage dermatological conditions themselves, referring (to specialists) only when in difficulty.[1] Indeed, although they can be very distressing, very few dermatological conditions are acutely life-threatening. In fact, a large proportion of the general public has a skin condition of some type (approximately 25% worthy of medical attention in most studies, and up to 50% of people have skin disease on examination).[2] It is widely known by doctors and the general public that a rash can indicate serious underlying systemic illness. Therefore, the appearance of an undiagnosed rash or skin lesion can be very alarming.

In our department, the acute referrals were nearly all seen by the registrar and (including follow-up visits) accounted for 25% of her total number of consultations each month. The workload included telephone discussions preceding about half of the referrals. Approximately 10% of acute referrals were seen on the ward. Referrals were usually directed at the registrar due to greater availability—but the majority were also seen by a consultant and the remainder were discussed with a consultant. If the referrer preferred to discuss the issue with a consultant, they were redirected to an available consultant.

Up to eight acute referrals are booked into a specific half-day acute clinic each Friday afternoon. Others are double-booked onto existing clinics, or seen at lunchtime. Clerical, nursing, and medical staff must find the medical records, vet the referral forms, take phone calls, and see the patients. These are often complex cases that require lengthy consultation with one or more medical staff and time-consuming procedures such as skin biopsies. Our aim is to reduce the number of inappropriate acute referrals so we can give urgent specialist care to those that truly require it.

Almost three-quarters of referrals in this audit did not comply with ACA guidelines—almost without exception, the referrer had overestimated the urgency. Many referrals are non-specific (for example ‘rash’), so are seen urgently in case the patient has a serious condition. GPs appeared to refer appropriately and their assessments were

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consistent with the guidelines more than 50% of the time, compared with only 20% for hospital doctors. All acute referrals were seen within the recommended limits. GPs were generally satisfied with the 1-week timeframe for urgent conditions, whereas inpatient teams often indicated that they felt the patient should be seen the same day. This was frequently agreed to, as it was just as convenient to double-book them immediately as to do so 4 weeks later. (Waikato Hospital does not get any funding for non-urgent conditions seen while patients are in hospital.)

Why are we getting so many inappropriate referrals from hospital doctors?—inpatient teams most often referred a non-urgent skin condition urgently because they wanted to discharge the patient that day, or because the patient was from a remote area, or simply because the patient had brought the skin condition to their attention. Some inpatients had received perfectly suitable management of their skin condition under the care of their GP. On several occasions, the referrer appears to have failed to read the case notes (as the patient had already been seen and treated by the Dermatology Department for the same condition).

Commonly, patients were unaware that a referral had been made—or the reason for it. We considered many would have been suitable for GP management, including those with longstanding inflammatory skin diseases such as mild acne, seborrhoeic dermatitis, venous eczema, solar keratoses, and small non-melanoma skin cancers. This is consistent with the objectives of the New Zealand Government's strategy titled *Reduced Waiting Times for Public Hospital Elective Services* (released March 2000). The ‘one-stop-shop’ approach (where as many problems as possible are dealt with while patients are in hospital) may be an admirable ideal—but it is not always practical. To reduce inappropriate referrals, we recommend the ACA guidelines should be used for inpatient referrals as well as those from general practice.

In community practice, the more common reasons for urgent referral of a non-urgent skin condition appeared to relate to potential loss of employment for the patient, pressure from the patient to refer to a specialist service, or failure of a trial of treatment. However, these referrals were appropriate to be seen by the Dermatology Department on a routine basis—that is, they had been managed as far as possible by the GP before referral.

It is impossible to categorise referrals if referral information is inadequate. This audit has confirmed the tendency to offer these patients an urgent appointment in case they have a very serious condition. However this process is inequitable, and means that those patients with well-described (but clearly elective) complaints must wait months to be seen. Frequently, we are not informed of the patient’s past medical history, current illness, and medications although these frequently impact on diagnosis and management of their skin complaint. Almost all acute referrals from GPs were made via telephone, which, although time-consuming, allowed the registrar to directly ask for relevant information.

In this audit, referral information from GPs and inpatients was superior to those in a study of outpatient referrals to an Irish general medical service (where information and pre-referral management was inadequate in more that 50% of referrals)[3]—but inpatient referrals, in particular, were still less than ideal. The Ministry of Health’s *Reference Manual for Managing Elective Services; Summary for Outpatient Clinics.*

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(Draft; February 2003) lists minimal information to be included in standard referral forms. Waikato Hospital's referral form complies with these recommendations and is clearly laid out. Should these forms also be used for inpatient referrals?

Inappropriate categorisation may arise from incorrect diagnosis made by the referrer. However, an experienced dermatologist may infer diagnosis from a carefully composed referral letter even when the referrer has been incorrect. Previous studies have shown that approximately 50% of referrals to a dermatology service have the correct diagnosis.[4,5] GP diagnostic accuracy in this study was close to this figure, but only 28% of inpatient referrals had the correct diagnosis. As we may not be able to teach non-dermatologists how to diagnose skin diseases, we should encourage referring practitioners to provide us with comprehensive and careful historical data. A slowly increasing number of referrals are accompanied by printed clinical images, and these are proving very helpful for categorisation.

After first assessment, follow-up appointments were arranged for three-quarters of the patients with our department, another department, or their family doctor. Follow-up with our department was arranged to ensure efficacy or safety of recommended treatment. The high follow-up non-attendance rate of the acute patients may suggest that the patients did not view the skin condition as particularly problematic or that their skin condition may have significantly improved.

The main problems identified by this audit are as follows:

- Inappropriate selection of a significant number of inpatients by hospital medical staff for referral.
- Inappropriate triage of acute referrals, particularly by other hospital departments.
- Inadequate acute referral information from other hospital departments.
- Overbooked registrar clinics due to acute patients and their follow-up needs.
- High non-attendance rate for acute patient follow-up.

In general, GP referral practices were superior to those of hospital teams and frequently complied with ACA guidelines and Government elective services recommendations.

What can be done to improve the situation? Options:

- Educate other doctors about skin diseases.
- Reduce the number of non-urgent patients seen acutely by refusing inpatient referrals except when these clearly impact on current hospital admission (consultant referrals only).
- Reduce follow-ups.
- Increase the number of junior and/or senior medical staff.

To reduce the workload, we need to increase the number of junior and/or senior medical staff or see fewer patients. Perhaps fewer and more appropriate cases might be referred if we provided an intensive educational campaign. Medical practitioners

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have very little training in diseases of the skin and understandably find diagnosis mysterious and management confusing. With about 1500 diseases, it is not surprising that referral diagnoses are inaccurate. Evidence also suggests that training might instead increase the number of referrals.[6]

To refuse to see inpatients with minor skin complaints would be unpopular with hospital staff and will cause friction with our colleagues. Instead, if we reduce the number of appointments allocated to patients referred by GPs, we will aggravate the delay to see patients who have important and disabling skin complaints and we will be unable to comply with Government's strategy to provide first specialist assessment within 6 months. (The elective services' website states 'The Government's strategy [in a document] titled *Reduced waiting times for public hospital elective services* set out the following objectives: a maximum waiting time of six months for first specialist assessment [this is the fourth objective]'.)

It seems unlikely that we will be able to reduce the number of follow-up visits—as a departmental review conducted in April 2003 has indicated these are necessary for diagnosis, procedures, serious skin disease management, monitoring second-line drugs, and for teaching. Dermatology is not considered a priority for increased health spending, and repeated requests have not resulted in additional medical staff.

We plan to place the following notice on the Waikato District Health Board intranet, and send it to all medical staff:

> 'The Department of Dermatology has very limited capacity to see acute inpatient referrals. We are delighted to do so when the skin condition is affecting the management of the acute condition for which the patient is in hospital or if the need of a specialist dermatology assessment is genuinely urgent. In the latter case, please refer to the National Access Criteria for First Specialist Assessment (attached). We expect a consultant to request or approve of all such referrals. Please complete a yellow referral form and include the following information:
>
> - Site, severity, and duration of condition.
> - How it affects inpatient management.
> - Previous treatments by GP or specialist(s).
> - Other medical conditions and medications.
> - Relevant social history.
> - How urgent is the request.
>
> Inadequate referrals will be returned to the consultant of the referring team for completion'.

These recommendations aim to improve the adequacy of referrals from inpatient teams and hence reduce unnecessary load on clinics, thus allowing us to provide a better service to patients referred from the community. We plan to repeat the audit (after 12 months) to determine if these actions have had an effect.

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Appendix 1. Acute referrals to Waikato Hospital's Dermatology Department

Time of day of referral **Day of week of referral**

am pm Mon Tues Wed Thurs Fri

Month of referral

Jan Feb Mar Apr May Jun
Jul Aug Sep Oct Nov Dec

Date seen

Referral Source **Referral Medium**

GP fax
Inpatient (please specify consultant) telephone
Other (eg. midwife, nursing staff) mail

Referral urgency as marked by referrer

Today Urgent Semi-urgent Routine

Referral urgency as judged by dermatologist on basis of referral

Today Urgent Semi-urgent Routine

Referral urgency as judged by consultant once patient reviewed

Today Urgent Semi-urgent Routine

Referral diagnosis **Dermatologist diagnosis**

Management of condition

Topical applications Oral antimicrobials Hospital admission
Oral immunosuppressives Advice only Other (please specify)

Follow-Up Arrangements

GP Derm OPC Other

Comments (incl. adequacy of referral information and any previous referrals).

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Therapeutic challenges in gout

Kamal Solanki and Peter Moller

Gout has a prevalence in New Zealand of 13.9% in Maori men and 5.8% in European men.[1] Maori have stronger family history, earlier age at onset, and higher frequency of multiple tophi and polyarticular gout.

Inpatients with gout often have a wide variety of co-morbidities[2]—including chronic renal failure, ischaemic heart disease, congestive heart failure, peptic ulcer disease, and diabetes mellitus. In addition, gout is often complicated by the concurrence of sepsis, thus presenting a diagnostic challenge and management problem.[3]

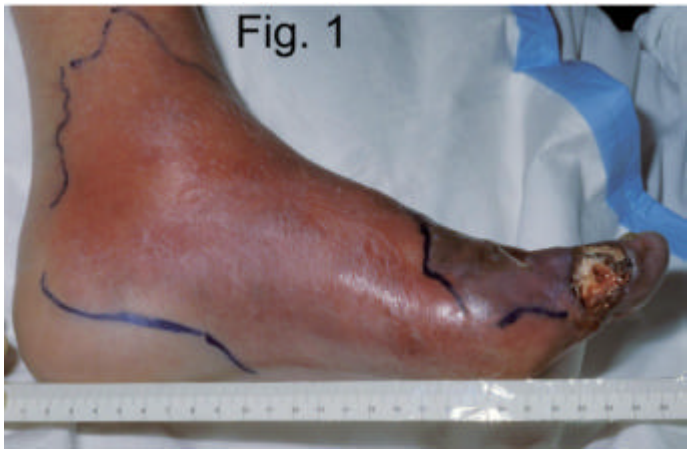
Case report

We present two cases:

Case 1

An 89-year-old Polynesian man with cellulitis of his left foot and ankle (Figures 1 and 2) secondary to an ulcer present for 14 months over the left metatarsophalangeal joint. He also had an acute flare of polyarticular gout with multiple tophi in his elbows and fingers. His co-morbidities included ischaemic heart disease (with previous myocardial infarction), permanent pacemaker, heart failure, atrial fibrillation, chronic renal failure, and moderately severe peripheral vascular disease. His medication list included diuretics, aspirin, warfarin, enalapril, digoxin, and diclofenac—and allopurinol 300 mg daily prior to admission.

Figure 1. Cellulitis in the left foot of an 89-year-old Polynesian man (big toe in foreground)



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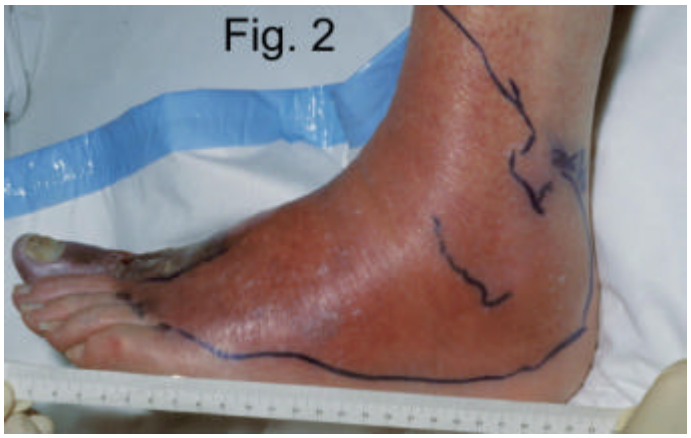
His haemoglobin was 112 g/L, white blood count (WBC) 31.2 x 10^9, neutrophil count 28 x 10^9, erythrocyte sedimentation rate (ESR) 68 mm/hr, international normalisation ratio (INR) 11.3, and creatinine clearance 20 ml/min. Blood cultures grew invasive corynebacterium and *Citrobacter diversus* species requiring intravenous antibiotics. His allopurinol medication was reduced to 100 mg daily and he was commenced on oral colchicine 0.6 mg twice daily along with prednisone 10 mg daily.

Figure 2. Cellulitis in the left foot of an 89-year-old Polynesian man



Despite multidisciplinary team involvement, his condition did not improve. Once his INR was satisfactory following vitamin K administration, he had an angiogram which demonstrated significant vaso-occlusive disease warranting a below-knee amputation as opposed to femoro-popliteal bypass grafting.

Case 2

A 62-year-old Maori man on chronic ambulatory peritoneal dialysis (CAPD) was admitted to the intensive care unit (ICU). He was hypothermic, and hypotensive secondary to *E. coli* peritonitis and chest infection. He had indurated exuding lesions on both knees (Figure 3)—and a background of chronic tophaceous gout, end-stage renal disease (secondary to hypertensive nephrosclerosis), peptic ulcer disease, and previous excessive alcohol intake.

Figure 3. Knee of a 62-year-old Maori man, showing an exuding lesion

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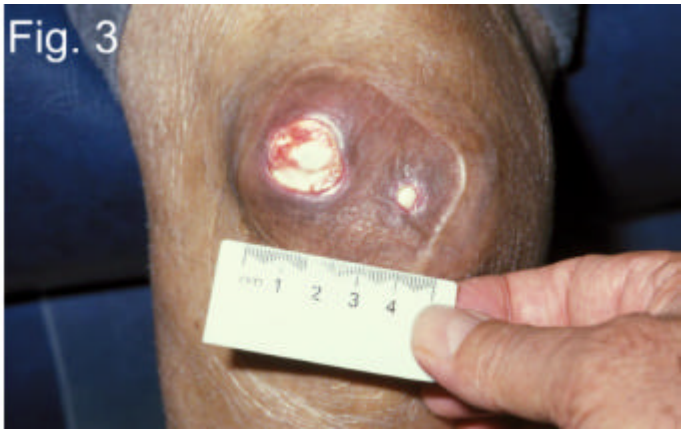

Apart from his renal medications, he was receiving aspirin, amiodarone, and 50 mg of allopurinol daily. In addition, his acute flare of gout was treated with prednisone 20 mg daily. He was not given any colchicine as his stay was complicated by *Clostridium difficile* enterocolitis.

Discussion

Both patients presented in an unstable state with complicated multiple co-morbidities.

It is important to consider acute gout as well as sepsis, to aspirate if any doubt exists, and to treat each condition as soon as possible.

Non-steroidal anti-inflammatory agents (NSAIDs) and cox-2 inhibitors are best avoided in moderate or severe renal impairment or in active peptic ulcer disease.

Colchicine is preferred in the presence of significantly decreased renal function, but the dose will need adjustment if the creatinine clearance is less than 10 ml/min,[4] otherwise there is an increased risk of neuromyopathy (especially with intravenous [IV] doses). Colchicine is also an alternative choice in patients with normal renal function; but it is seldom well tolerated at a dose greater than 0.6 mg thrice daily, so the traditional recommendation of 2 tablets *stat*, followed by 1 tablet hourly (maximum of 6 to 8 tablets or until diarrhoea ensues) has fallen out of favour.

Prednisone often may be the only choice of for acute oligo/polyarticular gout (as it was in our second case). Intra-articular steroid (methylprednisolone or triamcinolone acetate injection) can be considered provided there is no intra-articular and cutaneous sepsis and the joint is accessible. Intramuscular adrenocorticotropic hormone (ACTH) 40 IU is also an option but (very frequently) the patient requires a repeat dose at 12 hours.[5]

For prophylaxis of gout, allopurinol (which lowers serum urate levels by about 20%) is most commonly used. However it should not be started (or stopped if the patient is already on it) during an acute attack. Just as any fluctuations in the urate levels may tend to precipitate an acute attack, an inflammatory reaction (already in progress) may be made worse by a large change in the serum urate concentration.[6] It is a common practice to add a NSAID (provided there is no contraindication) or colchicine (eg, 0.6

mg *bid*) for 4–6 weeks on starting allopurinol—its dosage is gradually built up over 7–10 days to the maximum, which is adjusted to the degree of renal impairment.[5]

In the presence of normal renal function, doses usually range from 200–800 mg with the aim to decrease the serum urate level well below the upper range. From our experience, the lower the level achieved, the better the control of gout and the diminution of tophi. On average, most of our patients have required 200–300 mg of allopurinol to achieve this. However, allopurinol has its own inherent problems of hypersensitivity rash including Steven Johnson Syndrome, and intolerance and cross-interaction with other drugs (especially warfarin, azathioprine, cyclosporin, ACE-inhibitors).

There is a search for alternatives for gout prophylaxis. Recent trials have shown that losarten, an angiotensin-II receptor blocker, may be useful (especially for elderly patients with gout and peptic ulcer disease and hypertension as it promotes urate diuresis). Moreover, a study in hypertensive renal transplant patients on cyclosporin, and treated with 50mg of losarten, resulted in a 17% increase in the fractional excretion of uric acid and a 8% decrease in plasma urate level.[7]

An open-crossover study of fenofibrate[8] (an anti-hyperlipidaemic medication) in patients on allopurinol has shown an additional 19% reduction in serum urate levels and 36% rise in the urate clearance.

Benzbromarone, a uricosuric agent, has been used in the presence of renal impairment (provided creatinine clearance >20 ml/min) for prophylaxis of gout. It reduces the serum urate levels by 33% to 59% in a dose-dependent manner and it has an additive effect if used in conjunction with allopurinol.[9–11]. In New Zealand, benzbromarone has been approved for renal transplant patients with gout and hyperuricaemia.

In cases of allopurinol sensitivity, rasburicase,[12,13] a uricase enzyme which oxidises urate to allantoin (highly soluble and readily excreted in urine) has been used in haematology units for tumour lysis syndrome. A single intravenous dose significantly decreases the serum urate levels within 24 hours.

With regard to surgical options for tophaceous gout, the retrospective study of Gow and Kumar[2] has shown an associated high rate of complications (especially in the presence of sepsis in patients with increased co-morbidities).

Finally, it is important for us to highlight that allopurinol very rarely (if ever) needs to be discontinued in a patient well-established on it during an acute exacerbation , and who is treated in a standard manner with either NSAID, colchicine ,prednisone, or intra-articular injection.

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Case of myelofibrosis with hypertrophic osteoarthropathy: the role of platelet-derived growth factor in pathogenesis

Binu John, Heraganahally Subhash, and Kurien Thomas

Hypertrophic osteoarthropathy (HOA) is a condition characterised by clubbing of digits—with periostitis, plus articular and periarticular pain. We report a patient with hypertrophic osteoarthropathy, who presented with severe anaemia, and was found to also have myelofibrosis (agnogenic myeloid metaplasia). This extremely rare association is particularly significant since it lends credence to the theory implicating platelet-derived growth factor (PDGF) in the pathogenesis of both of these conditions (myelofibrosis and hypertrophic osteoarthropathy).

Case report

A 27-year-old man presented with history of breathlessness (on exertion) and fatigue of 2 years' duration. There had been gradual painless enlargement of both hands and feet (of 3 years' duration) with mild pain and swelling involving ankles, wrist, and knee joints. There was no history of cough with expectoration, haemoptysis, wheeze, abdominal pain, alteration in bowel habits, melaena or bloody diarrhoea. He had no history of fever, palpitations, or cardiac disease. There was no family history of similar illness.

On examination, he had a pulse rate of 80/min and a blood pressure of 120/80 mmHg. He had pallor with marked clubbing. There was swelling of distal extremities; both hands and feet were grossly enlarged. Feet appeared 'elephant like' (Figure 1). There was no icterus, lymphadenopathy, or pedal oedema. Chest was clear. The liver was palpable 2 cm below the costal margin. Spleen was not palpable. There was no free fluid. Examination of the cardiovascular system was normal.

There was no evidence of effusion of any joint and no evidence of acromegaly such as prognathism, coarse facial features, change in voice, or proximal myopathy. Investigations showed haemoglobin 4.7 grams/dL, with a reticulocyte count of 4.6%. Blood picture revealed ovalocytes and teardrop cells. The total leucocyte count was 3100/cu mm, with the differential count showing 68% polymorphs, 16% lymphocytes, and 16% eosinophils. The platelet count was 182,000/cu mm. The mean corpuscular volume was 73, and erythrocyte sedimentation rate (ESR) 87mm/hr. Serum calcium, phosphate, alkaline phosphatase, liver function tests, growth hormone level, and thyroid function tests were normal.

Radiograph of the chest was normal. The forearms showed linear periosteal reaction involving radius and ulna bilaterally (Figure 2), and the lower limbs showed periosteal thickening of bilateral tibia and fibula and distal soft tissue enlargement (Figure 3). Ultrasonography of the abdomen revealed hepatosplenomegaly. Attempted bone marrow aspiration revealed a dry tap. Cellular imprints of the bone marrow trephine biopsy fragment revealed normoblastic erythroid maturation, diffuse

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lymphocytosis, and increased osteoblastic activity. The bone marrow trephine biopsy was consistent with the cellular phase of myelofibrosis.

Figure 1. Photograph of both lower limbs of a 27-year-old man showing marked enlargement of both legs with swelling of both ankle joints and clubbing of toes

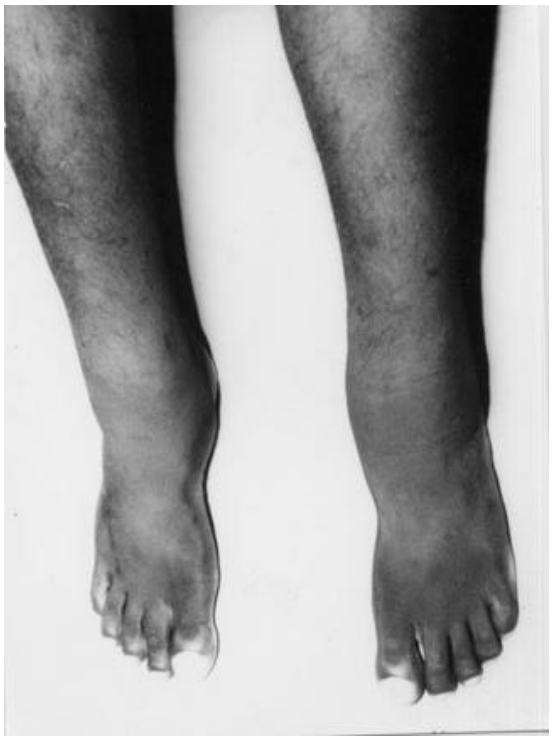

Figure 2. X-ray of a 27-year-old man showing linear periosteal reaction involving radius and ulna bilaterally

Figure 3. X-ray of a 27-year-old man showing exuberant periosteal reaction of bilateral tibia and fibula, and generalised soft tissue enlargement

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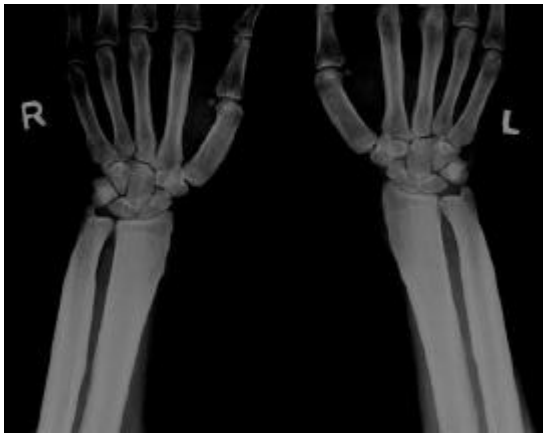

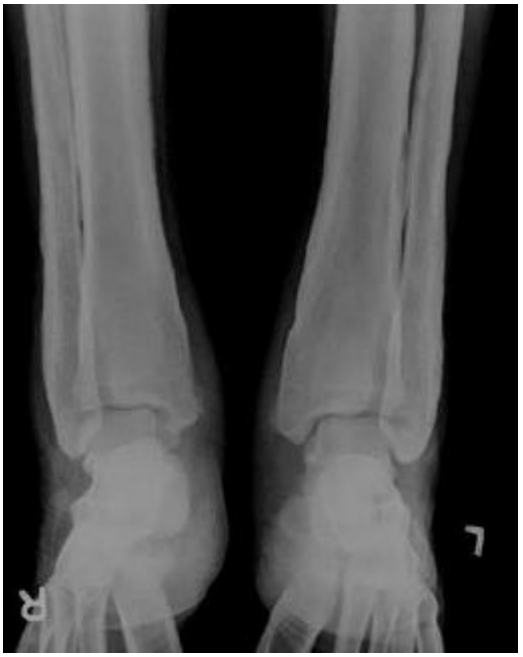

Discussion

This patient presented with hepatosplenomegaly, peripheral cytopaenias, teardrop poikilocytosis, and marrow fibrosis suggestive of myelofibrosis (AMM). The other disorders that may lead to this clinical picture (including infections such as tuberculosis—and metabolic disorders such as renal osteodystrophy, hypoparathyroidism, and hyperparathyroidism) were ruled out in our patient.

He also had features of hypertrophic osteoarthropathy (HOA), a clinical syndrome characterised by marked clubbing of the digits; specifically, enormous hands and feet, arthralgia, and periosteal overgrowth. There were no cutaneous manifestations of primary hypertrophic osteoarthropathy (pachydermoperiostosis), such as furrowed forehead, broad nose, prominent nasolabial folds, and thickened eyelids. There were no clinical or laboratory evidence of other conditions that are commonly associated with secondary hypertrophic osteoarthropathy—such as suppurative lung disease, pulmonary neoplasm, inflammatory bowel disease, colonic neoplasm, congenital cyanotic heart disease, infective endocarditis, or thyrotoxicosis.

Multiple mediators (including PDGF, prostaglandins, ferritin, bradykinin, and oestrogen) have been implicated as causes of clubbing or hypertrophic osteoarthropathy.[1] The most popular theory is that megakaryocytes or large fragments of megakaryocytes in the systemic circulation, preferentially lodge in the tips of the digits, because of the prevailing patterns of blood flow. Once stuck there, the cells release PDGF and other substances that increase endothelial permeability and activate fibroblasts and other connective tissue cells.[1] The exact aetiology of myelofibrosis is not clear, but one of the most widely accepted theories is that it results from the liberation of excessive amounts of growth factors (including PDGF, transforming growth factor-ß [TGF-ß], and epidermal growth factor [EGF], which are each

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contained within platelet and megakaryocyte a-granules). These can lead to marrow fibroblast expansion and collagen synthesis. The PDGF content of platelets from AMM patients is known to be decreased, indicating a release or leakage of such growth factors.[2]

There are only three other case reports describing the association of myelofibrosis and hypertrophic osteoarthropathy.[3–5] In one of these reports, the authors demonstrated a high proliferative potential of bone marrow derived fibroblasts *in vitro* as well as an increased expression of PDGF-BB binding sites.[3] Thus, the concurrent occurrence of these two uncommon conditions may be a pointer to the role of PDGF in the pathogenesis of these conditions.

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Kiwis in America: reflections on the US primary care system

Peter Crampton and Ngaire Kerse

What can New Zealand learn from the United States' system of primary care? We offer a perspective on primary care in the US after spending 12 months in America during 2002-03 as Harkness Fellows in Health Care Policy, a programme funded by the Commonwealth Fund (a US philanthropic foundation). Our work involved researching aspects of primary care policy in the US. We were also, along with our young families, consumers of primary care services. At first glance, the health systems in the US and New Zealand appear so fundamentally different that seeking useful comparative insights might seem overly ambitious. Nevertheless, the opportunity to observe the strengths and weaknesses of the US system has helped highlight for us what we think is most valuable in New Zealand primary care, and prompted us to think critically about aspects of New Zealand's system that could benefit from experience in the US.

Context

The US is huge—it would be hard to overemphasise its scale, diversity, and complexity. The population in 2000 was over 281 million, spread over 50 states (plus the District of Columbia), 26 of which had more people than New Zealand. The US population is ethnically diverse, however Native Americans comprise only about 1% of the population (2.5 million), despite there being around 25 million, according to one source, at the time Columbus made his trans-Atlantic voyage in 1492.[1]

Compared with New Zealand, it was our impression that debates about indigenous health in the US are muted. Over past years, the US government made over 400 treaties with various Native American tribes but systematically violated them all.[1] By contrast, New Zealand has just one treaty, the Treaty of Waitangi, which also was systematically violated by New Zealand's colonial and post-colonial governments.

However, the Treaty has an important contemporary role—providing both an ongoing basis for a public dialogue between indigenous and non-indigenous people, and a constitutional basis to assist the daunting task of addressing the country's colonial historical legacy. Although public dialogue has occurred in the US, along with reinstatement of many of the terms of treaties, the most interesting example being self-determination,[2] this public dialogue was not readily apparent to us in our role as Harkness Fellows or in the course of everyday social debate.

An outstanding feature of healthcare in the US is its cost—nearly three times the amount spent per capita in New Zealand (US$4631 and US$1623 respectively).[3] This high level of expenditure can be accounted for in various ways. For example, the US system is oriented towards technologically intensive specialty care. It is likely, too, that administrative costs are substantially higher in the US, due to the complexity of the system and the presence of multiple funders.[4] Important, also, is the fact that no organisation in the US holds the purse strings for healthcare—there are many funders and purchasers of care. While about 25% of Americans have government health

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insurance (Medicaid or Medicare), about 60% of Americans obtain health insurance via their employer.[5] By contrast, in New Zealand, the government fulfils the role of funder for the bulk (about 80%[6]) of healthcare expenditure, thus having the capacity to exert control over the overall level, and rate of growth, of expenditure via its annual budget setting process (albeit currently only about 60% of primary care funding is public[6]). Despite the high level of healthcare expenditure, overall health outcomes in the US are, at best, mixed, and often compare unfavourably with other developed countries, including New Zealand.[7,8]

For us, one of the most striking features of the US healthcare system is that, despite the high levels of expenditure, 14.6% of the population (over 41 million people) do not have health insurance, and nearly one-third of the non-elderly population lack health insurance for some part of the year.[9] The uninsured are mainly low-income employed people whose employment contracts do not include health insurance and who do not qualify for government health insurance. It should be noted, however, that financial barriers to access is not a problem unique to the US. In a five-country survey of people with below-average income, New Zealand came second to the US with 20% of respondents who reported problems paying medical bills in the past year (compared with 35% in the US).[10]

The final point we wish to make about the context for healthcare in the US is that the US public do not look first to government to solve pressing healthcare problems,[11] and are ambivalent (at best) about redistributive social and economic policies.[12] This lack of confidence in government, reflecting a combination of ambivalence and occasional hostility, is most apparent in social and health programmes, with a notable exception being the widespread public support for the Medicare health insurance programme for the elderly. While public opinion is primarily a product of historical and cultural forces, the influence of special interest lobby groups is also powerful and pervasive in the US. The ability of interest groups, such as insurance industry lobbies, to sway public opinion and government policy became most apparent during the failed attempts by the Clinton administration to reform aspects of the US healthcare system.[13,14]

Primary care in the US

The complexity of primary care in the US reflects the overall complexity of its healthcare system. The US does not rely on primary care generalists to provide the majority of primary care, rather the majority of primary care is provided by specialists who provide ‘generalist’ care within the limitations of their age-group and/or body-part specialty areas. For example, internists care for adults of both sexes, paediatricians care for children of both sexes, and obstetricians/gynaecologists care for adult women; these three specialties provide respectively 25%, 21%, and 13% of ‘primary care’ consultations.[15]

Family physicians, on the other hand, constitute about 31% of primary care doctors in the US,[8] provide 41% of primary care consultations,[15] and have a similar role to general practitioners in New Zealand. About 36%[15] of primary care doctors in the US practice single-handedly, compared with 9.8%[15] in the UK, and 20% in New Zealand (personal communication, Sarah Tallboys, RNZCGP, 2003). Some nursing in family

physician offices is carried out by nurse practitioners, often in independent practice, and some is carried out by practice nurses working in a more traditional role in support of the doctor. Medical-assistants and physician-assistants are also common non-doctor members of the primary care team. Most hospital inpatients in the US are managed by office-based physicians and not by doctors on hospital staff. Therefore, family physicians often have an additional role providing care for their hospitalised patients. On average, US family physicians visit 10 patients in hospital per week.[16]

Family physician training in the US occurs during a 3-year residency programme (following immediately after a 4-year postgraduate medical degree course), although such residency programmes are more plentiful on the West Coast than in the East (indeed, two of the most prestigious East Coast medical schools, Harvard and Johns Hopkins, do not have departments of family medicine). Organisations for primary care are well developed, such as the American Association of Family Physicians (with 53,674 active members, about 45% of all family physicians), and academic development is fostered by organisations such as the Society for the Teachers of Family Medicine (over 4000 members) and the North American Primary Care Research Group.[17]

In 2000, as a result of managed care, about 40% of US people attended a primary care doctor who fulfilled some elements of a gatekeeper role.[18] Gatekeeping, however, has been unpopular with the public and specialists (who largely continue to be paid on a fee-for-service basis). As a result, managed care organisations are becoming less keen on using primary care doctors as gatekeepers.[15] Not surprisingly, given the specialist-rich environment in the US, the proportion of patients per year referred to specialists is about double the proportion in the UK (30–37% versus 14%, respectively).[19] There are no directly comparable figures available for New Zealand, however survey data suggest specialist utilisation to be closer to the US figure than the UK figure.[20]

Primary care utilisation is much lower in the US than in New Zealand (about 1.3[21] and 4-5[22,23] visits per year, respectively), partly because people frequently go directly to specialists in the US. The uninsured receive primary care mainly from ‘safety-net’ providers such as public hospital emergency departments, doctors’ offices, and community health centres (CHCs).[21] The CHC movement in the US started in the 1960s, motivated by the political will to address social and health inequalities.[24] CHCs are funded under the Public Health Service Act to provide primary care to ‘medically underserved’ areas, and the programme is administered by the Bureau of Primary Health Care of the US federal government’s Department of Health and Human Services.[25] Today, health centres care for 8.3 million people—40% of them uninsured, 34% Medicaid recipients, 85% low income, and 65% being people of colour.[26] CHCs cater to about one in six persons who otherwise would lack access to medical care.[27] The term Community Health Centre now subsumes Community Health Centres, Migrant Health Centres, Health Care for the Homeless, and Health Services in Public Housing Projects, and involves about 700 organisations in 3000 communities.[26] The Bush Administration has pledged a very substantial increase in funding to support CHCs.[28,29]

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What can New Zealand learn from the US?

The organisation of primary care in the US is strikingly different from that in New Zealand. In many respects, we believe the US provides case studies of what would be, or in some cases *is*, undesirable in New Zealand's primary care—for example, fragmentation of the workforce, provision of primary care by specialists, inequitable resource allocation, financial barriers to access, uncontrolled costs, and expensive reliance on specialist care.

Lack of systematic transfer of information between doctors caring for the same patient means that coordination of care is severely hampered in the US. Using Starfield's framework for measuring and comparing the strength of primary care systems in different countries, New Zealand's score is intermediate between strong primary care countries such as Denmark and the UK, and weak primary care countries such as the US and Germany.[8,30] However, it would be foolish to overlook initiatives in the US that New Zealand could learn from.

First, community health centres provide an excellent working model of comprehensive, team-based primary care. Their funding and governance arrangements, in particular, are noteworthy: they receive the bulk of their funding from state and federal sources but their governance boards are dominated by patients and service users. The US Public Health Service Act stipulates that each CHC should have a governing body which is composed of a majority of individuals being served by the centre and who, as a group, represent the individuals being served by the centre.[31] While a number of centres in New Zealand, including those belonging to the Health Care Aotearoa network, are required by their constitutions to have community representatives and/or consumers on their governing boards,[32] the US model is stronger in that it legislatively mandates community participation in governance of CHCs. The current primary care reforms in New Zealand require that Primary Health Organisations have some form of community involvement in their governance but, so far, this requirement has been much less clearly spelt out than is the case for CHCs in the US.

Second, while US primary care as a whole is expensive and fragmented, some managed care organisations provide instances of fully integrated, cost-effective care. For example, a comparison of the Kaiser Permanente managed care organisation (which cares for over 6 million people in California) and the British NHS concluded that overall healthcare costs in Kaiser were similar to within 10% of those in the NHS, and that Kaiser's performance was considerably better using measures such as diabetes management, post-myocardial infarction care, access to specialists, and hospital waiting times.[33] The lessons for New Zealand primary care in this study include the value of a truly integrated system where primary and secondary care mesh together seamlessly, the value of treating patients at the most cost-effective level of care (note that Kaiser patients spent one third of the time in hospital compared with NHS patients), and the value of comprehensive and integrated information technology.

Third, while not always earmarked specifically for primary care research, plentiful health research funding in the US has been used to promote, evaluate, and disseminate

information about innovations in primary care. Such innovations include, for example, the chronic care model, which is an expanded primary care model incorporating self-management, decision support, delivery system redesign, and clinical information systems.[34] Evaluation of aspects of the model highlight, for example, improvements in doctors' performance,[35] although final evaluation of the model is awaited. Other successful innovations have focused on information technology, group medical visits, and collaborative care (a version of the patient-centred consultation).[36]

Fourth, US family physicians maintain close contact with their patients through episodes of hospital care, although this role is declining with the increasing use of hospitalists (hospitalists are doctors who take care of primary care physicians' patients while they are in hospital[15]). It is highly likely that this continuity of care improves sharing amongst carers of social and medical information, quality of discharge planning and coordination of community services post-discharge, and patients' sense of being well cared for and supported. Although we are not aware of research into the effects of primary care involvement in hospital care on health outcomes, quality, or efficiency, the US experience raises for consideration an extended role for New Zealand general practitioners in hospital care.

Conclusions

We are relieved to have our families back in New Zealand where relatively holistic and non-interventionist primary care is the norm. Our observations of the fragmented and specialist-oriented primary care system in the US have emphasised for us the importance of New Zealand's commitment to generalist, team-based primary care that provides continuing, comprehensive, and coordinated services. We value New Zealand's capacity as a nation to systematically attempt to improve the quality and equity of its primary care system via the collective efforts of the Ministry of Health working with community and provider groups. However messy such efforts are, they provide a stark contrast with the US, which lacks such capacity for collective action at a national scale.

We have learnt from the US that community involvement in primary care can be strengthened using legislation, and that there is potential for strengthening continuity and coordination through enabling general practitioner involvement in hospital care. While there is much in the US primary care system that we believe New Zealand should not emulate, nevertheless we think that New Zealand can benefit from ongoing selective and critical evaluation of the US experience.

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Rheumatoid Arthritis

This extract is taken from an article by Dr D Colquhoun that was published in the New Zealand Medical Journal 1904, Volume 3 (12), p385–9.

No disease has had more names given to it than rheumatoid arthritis, these names chiefly representing theories as to its causation, description of its anatomical peculiarities, and clinical course. It has been described as a chronic rheumatism, as a form of gout, as a mixture of gout and rheumatism, as a specific disease, as a disease of nervous centres, as a disease of toxic origin, as due to microbes, as due to changes in the uterine and ovarian tissues, &c. From the anatomical standpoint the nodosities and the deformities of joints, the bony outgrowths, the eburnation of the ends of bones, the wasting of muscles, have influenced nomenclatures and diagnosis.

A study of the clinical features of the disease shows that we have at least three types--a chronic, a subacute, and an acute form--not always separate, but often bleeding. Confused and confusing as are the names and theories of this disease, they only correspond with the condition or conditions we meet in practice, and there is a growing feeling that " rheumatoid arthritis " must be looked on as a name which is applied to several forms of disease of joints which are in no way related to each other except by superficial resemblances.

I do not propose to discuss an aspect of the subject which has been dealt with of late years by Bannatyne, Garrod, Hale, White, and others, but to-day only mean to refer to some cases which have come under my notice which strongly suggest that there is one class of cases usually described under " rheumatoid arthritis " which have a distinctly ovarian or uterine origin, and to suggest still another theory--viz., that they may be due to tissue - changes (allied to those which give us mysoedema in deficiency of the thyroid secretion.

I do not offer these cases for your consideration as anything more than suggestive. The rheumatoid conditions and the ovarian operations may be more than coincidences. But I think there can be no doubt that the case of Mrs F. is one of a type that is constantly occurring in practice--viz., a rheumatoid condition following or accompanying the change of life in women. I have treated several such cases with ovarian tabloids, and, I think, always with advantage.

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Proceedings of the Scientific Meeting of the Christchurch Medical Research Society, Friday 17 October 2003

The role of perineural invasion in long-term survival in patients with pancreatic carcinoma.

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Most pancreatic carcinomas demonstrate perineural invasion, however its prognostic significance is unclear. The aim of this study is to grade the severity of perineural invasion and correlate this with survival.

A prospective analysis of all resected pancreatic carcinomas (n = 44) in a UK teaching hospital was carried out from 1997–2001. Perineural invasion was graded 0 (absent) to 3 (severe). Factors predictive of hospital mortality and 5-year survival were determined (patient age, gender, pre-operative albumin/ bilirubin, operation blood loss/ duration, Townsend score, nodal status, vascular/ perineural invasion, tumour size and grade).

Hospital mortality (18%), was significantly higher for patients with albumin <35 g/L, operating time >5.25 hours, blood loss =5 litres, lowest quartile Townsend score and re-exploration. Multivariate analysis identified albumin <35 g/ dL, (odds ratio [OR] 22.5, p = 0.043), re-exploration (OR 30.3, p = 0.029) and lowest quartile Townsend score (OR 39.8, p = 0.018) as independent predictors of hospital mortality. Actuarial survival at 1, 3 and 5-years was 56%, 29% and 24%. Perineural invasion was present in 31 (70%) patients. Survival was significantly better for perineural invasion grade 0–1 than grade 2–3: median survival 37 versus 17 months respectively, p = 0.007. Five-year survival was significantly less with vascular invasion and tumours >2 cm. Cox proportional hazard analysis identified grade 2-3 perineural invasion as the only significant independent indicator of poor prognosis, hazard ratio 2.8 (95% CI 1.1–7.2) p = 0.031.

Grade 2-3 perineural invasion is a strong prognostic indicator of poor outcome in pancreatic cancer. Grading should become standard practice in reporting of pancreatic cancer.

Diencephalic amnesia and the contribution of different thalamic nuclei.

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Diencephalic amnesia occurs following thalamic injury, due to tumour, stroke or the alcoholic Korsakoff syndrome, but the neural basis of this disorder remains uncertain. Clinical and animal evidence has implicated either the mediodorsal, anterior, or intralaminar thalamic nuclei. We hypothesised that these medial thalamic nuclei should be grouped into three regions, which contribute to functionally segregated

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circuits implicated in different aspects of memory. To investigate this hypothesis using a rat model, we performed highly localised, neurotoxic thalamic lesions followed by memory tests that are sensitive to hippocampal, amygdala or dorsal prefrontal cortex damage. Nine rats received lesions to the anterior region (anterior thalamic nuclei), part of a cortico-hippocampal-thalamic circuit. Ten rats had lesions to the lateral region (rostral intralaminar, central medial, and lateral mediodorsal nuclei), which contributes to a cortico-striatal-thalamic circuit. Ten rats received posterior region lesions (the remaining mediodorsal nuclei), to disrupt a cortico-amygdalo-thalamic circuit. Evidence for normal temporal order memory, which is sensitive to prefrontal cortex damage, was found in both the Anterior group and the 11 sham rats (p <0.001 and p <0.01, respectively), but not the Lateral and Posterior groups (p's >0.40).

The Anterior group was the only group impaired on a spatial memory task, consistent with hippocampal circuit dysfunction (p <0.0002), whereas only the Posterior group was impaired on a reward magnitude memory task, consistent with amygdala circuit dysfunction (p <0.002). These lesion-behaviour dissociations encourage a more comprehensive and inclusive approach to our understanding of diencephalic amnesia.

Effect of hypoxia on the vascular targeting agent, Combretastatin A-4-P. G Dachs[1,2], A Steele[2], C Coralli[2], C Kanthou[2], Gillian M Tozer[2]. [1]Angiogenesis Research Group, Department of Pathology, Christchurch School of Medicine and Health Sciences, Christchurch; [2]Tumour Microcirculation Group, Gray Cancer Institute, Mount Vernon Hospital, Northwood, UK.

Combretastatin A-4 phosphate (CA-4-P) is a tubulin-binding agent in clinical trials as a tumour vascular targeting agent. *In vitro* CA-4-P causes microtubule depolymerisation, actin stress fibres and cell cycle block due to interference with spindle formation during mitosis. In experimental tumours, CA-4-P causes a rapid and catastrophic shutdown of the established tumour vasculature leading to necrosis and secondary tumour cell death. However, a narrow viable rim of surviving tumour tissue remains, allowing a rapid regrowth of tumours.

Little is known of the effect of inherent tumour hypoxia on CA-4-P activity, and conversely, what effect CA-4-P-induced hypoxia has on tumour progression. We analysed protein accumulation of the main hypoxic transcription factor, hypoxia-inducible factor 1 (HIF-1) in two human carcinoma cell lines and two primary endothelial cell types following stimulation by severe hypoxia (anoxia). The cells were treated with CA-4-P at clinically relevant doses (0.001–1 μM). Trypan-blue exclusion assays were used to determine cell survival following anoxic CA-4-P treatment.

Western blot analysis of treated cell extracts showed a drug dose dependent reduction in HIF-1 accumulation in hypoxic cells. This effect, although somewhat variable, was evident both when the cells were hypoxia-stimulated at the start or end of the CA-4-P treatment period. Cycling cells showed a reduced number of viable cells following CA-4-P treatment, whereas non-cycling cells, either due to cell density or long term anoxic incubation, did not respond.

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In conclusion, it appears that CA-4-P treatment modulates HIF-1 accumulation. The subsequent effect on down-stream gene expression is currently under investigation.

An innovative system for rapidly reporting the FM 100-hue colour vision test. R Hidayat[1], R Hidajat[2], J McLay[2], D Goode[3], M Elder[2]. [1]Applied Computing Department, Lincoln University, Lincoln; [2]Department of Ophthalmology, Christchurch Hospital, Christchurch; [3]Department of Medical Physics and Bioengineering, Christchurch Hospital, Christchurch.

The Farnsworth-Munsell (FM) 100-hue test is globally accepted as one of the most sensitive and specific methods for assessing colour vision. It can detect ophthalmic disease (glaucoma, diabetic retinopathy, optic nerve lesions) at an early stage and has the added advantage of being non-invasive. However a major shortcoming of the test is the laborious and time consuming effort needed to calculate the results and to plot them on a chart for interpretation.

We have developed a computer program for reporting the FM test which also allows the patient's data to be read with a bar code scanner. This new system has been in routine use in eye clinics at Christchurch Hospital for one year and has proved to be of great assistance both in saving time and eliminating arithmetic errors in the scoring calculations. It produces the two reports, one for each eye, in four minutes which contrasts with the 60 minutes required by the conventional manual reporting system. Such a substantial saving in time will encourage clinicians to make greater use of this valuable diagnostic test.

Significantly our computer generated report duplicates the conventional manual report, the report clinicians are familiar with, and in addition indicates whether the results fall within the normal range appropriate for the patient's age.

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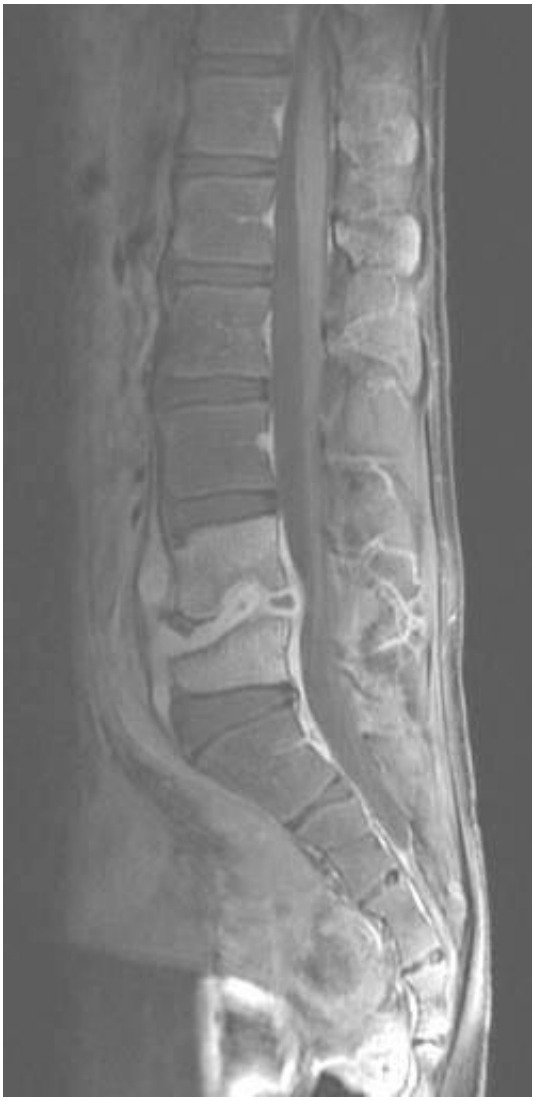
Adolescent Lumbago

A 16-year-old boy presented with back pain and pyrexia.

An MRI scan of his lumbar spine was performed (Figure 1).

Figure 1



What is the diagnosis?

See below (next page) for the diagnosis.

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Figure 1. Abnormal signal in the L 4/5 disc with destruction of the adjacent vertebral end-plates

Diagnosis:

Lumbar discitis which is usually secondary to blood-borne bacterial invasion (eg, from urinary tract infection). The pathogen in this case was *Staphylococcus aureus*, but in the immunocompromised patient it is often secondary to a Gram-negative organism. *Mycobacterium* is another possible pathogen.

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Aids treatment with generics

The former US president Bill Clinton last week took a swipe at the Bush administration's close relationship with American pharmaceutical giants by announcing a deal to enable poor countries to buy cheap generic drugs and testing equipment for Aids, rather than the US companies' more expensive wares. The deal with five generic drug companies will bring the cost of Aids drugs down to $140 per person per year and cut the cost of testing equipment by 80%.

The UN's Global Fund – which grants money to poor countries to buy drugs – the World Bank and Unicef signed an agreement with the Clinton Foundation to provide the cash and assistance.

The move runs counter to the thrust of the Bush administration's $15bn anti-Aids plan. It has become increasingly clear in recent months that the administration wants to pay only for drugs made by the big US-based companies. It has been accused of trying to undermine confidence in generic copies.

President Bush's anti-Aids supremo, the former Eli Lilly chief executive Randall Tobias, recently told Congress that there were doubts over the quality of cheap generic Aids drugs made in India and China, even though they have been approved by the World Health Organisation.

Guardian Weekly (UK), 15–21/4/04, p4.

Can't face up to it

Attempts to perform the world's first face transplantation were thwarted on March 2 by the French National Ethics Advisory Committee (CCNE), which ruled that inherent risks of the procedure make it unethical. Plastic surgeons led by Laurent Lantieri at Hôpital Henri-Mondor, near Paris, had been waiting for the response since 2002, after they put together a list of people with facial disfigurements in whom traditional procedures had failed.

After an extensive survey of French plastic surgery and burns units, Lantieri found a candidate who was interested in undergoing the procedure. However, the operation poses several ethical dilemmas because the donor face must be removed shortly after death and before the family farewell. If CCNE's opinion had been favourable, a clinical trial involving five face transplantations would have started this year.

However, CCNE stressed that "face transplants must not be performed until more complete investigations about the procedure are done and the precise risks are better known".

The report noted, for instance, that it would be "illusory" to talk about "informed consent" since a failure would make the recipient's situation worse.

Lancet 2004;363:871.

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Mein Gott!

A new study of the advertising material and marketing brochures sent out by drug companies to GPs in Germany has shown that about 94% of the information in them has no basis of scientific evidence.

The study, carried out by the Institute for Evidence-Based Medicine, a private independent research institute in Cologne, evaluated 175 brochures containing information on 520 drugs, which were either sent by post or handed out to 43 GPs since last June. The study was published in this month's issue of the drugs bulletin, *Arznei Telegramm* (2004;35:2-3; www.di-em.de/data/at_2004_35_21.pdf).

About 15% of the brochures did not contain any citations, while the citations listed in another 22% could not be found. In the remaining 63% the information was mostly correctly connected with the relevant research articles but did not reflect their results. Only 6% of the brochures contained statements that were scientifically supported by identifiable literature.

BMJ 2004;328:485.

Medical editors to take tougher line

EDITORS of medical journals should agree to expose dubious or unethical research submitted to them for publication, according to a proposed code of conduct. Editors signing up to the code would be obliged to blow the whistle on all types of bad practice and misconduct that in the past might simply have led to papers being rejected.

Published by the Committee on Publication Ethics (COPE), which represents 178 mainly British medical journals, the code goes further that any other in obliging editors to blow the whistle. For instance, they would have to inform institutions where offending research was carried out, or funding bodies.

Publication of the code comes a week after *The Lancet* alleges that Andrew Wakefield, the main author of the report in 1998 linking autism to the MMR triple vaccine for measles, mumps and rubella, had failed to declare to it a possible conflict of interest prior to publication of his report.

The new code would oblige editors to press authors hard to declare conflicts of interest.

New Scientist 6/3/04, p11.

On the other hand

The Lancet routinely publishes papers by employees of drug companies and by researchers who are paid to testify in legal cases relating to their work. Wakefield's speculation about MMR was not well grounded, and many experts in public health will be delighted that this prestigious journal is now distancing itself from this part of

his paper. It's unclear, however, whether Wakefield was any more compromised by conflicting interests than are many other of *The Lancet's* authors.

Then there is the question of whether Wakefield really did hide his alleged conflict. He made no statement when submitting the original paper, but in correspondence published in the *The Lancet* some two months after the original publication, he acknowledged evaluating a few children on behalf of the Legal Aid Board. Wakefield did not state the funding he received—now reported to be pounds 55,000 (US$103,000)—and there is some ambiguity as to whether he was referring to patients in the controversial paper. But his actions hardly seem those of someone determined to conceal a fatal conflict. And his belated disclosure did not prompt any public reaction from *The Lancet's* editors at the time.

The problem is that many observers of *The Lancet's* disavowal of Wakefield's speculation about MMR in the face of a journalist's inquiry will now assume that researchers' conflicts of interest inevitably undermine their integrity. But if these conflicts are managed properly, and disclosed, this needn't be the case. Few journals are in a position to aggressively police their conflict-of-interest policies, so further allegations are inevitable.

Nature 4/3/04, p1.

Better breast cancer management?

A large international randomized trial, which included 4742 patients, compared the standard adjuvant treatment for early breast cancer in postmenopausal women, five years of therapy with tamoxifen (an inhibitor of the estrogen receptor), with two to three years of tamoxifen followed by therapy with exemestane, an aromatose inhibitor, for the balance of the five-year period. Disease-fee survival was significantly better in the exemestane group than the tamoxifen group, however, overall survival was not significantly different in the two groups.

This trial indicates that the sequential use of antiestrogen compounds with different mechanisms of action has the potential to obviate the problem of resistance that has been encountered in women treated only with tamoxifen.

N Engl Med J 2004;350:1081–92.

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Suicides among elderly men

The recent suicide of my elderly father reflects an important public health issue. The majority of suicides are adult suicides and, in many countries, there is a surprisingly high rate of suicides amongst elderly men [1,2]. However, the focus in 'developed' countries is on youth suicide [2,3]. The danger of this focus is that the risk of suicide in other age groups may not be recognised. Indeed, although there may be a difference in 'years lost', suffering does not distinguish between the young and the old.

My father visited his general practitioner (GP) 2 days before he ended his life, however depression and suicidal risk were not detected. Indeed, the majority of older people who end their lives visit their local doctor in the week preceding their death without their depression and suicidal risk being detected [2,4,5]. A number of reasons for this have been suggested.

First, signs of depression in older people may be misinterpreted and dismissed as age-related changes—or related to another illness or disability that the person is suffering from.[1] Second, older persons often do not present with the classic symptoms of depression.[1] Third, it can be very difficult for many people, and particularly men, to disclose to a GP or other people how they are feeling, and they may actively hide this.[4]

My father had a number of known risk factors for suicide and was experiencing symptoms that could be attributed to depression. Even his suicide note said that he was suffering from depression. Furthermore, family members had noticed a number of changes in his behaviours and attitudes over the previous year; however, these were not known collectively by any one person.

As many older people visit GPs, an opportunity exists for the identification of depression and suicidal ideation and appropriate treatment.[1,5,6] There are guidelines for GPs in New Zealand regarding the recognition, treatment, and management of both depression and youth suicidal behaviour. Similar guidelines could be developed that address other age groups—and that information could then be re-presented to GPs every few years to refresh their understanding and to capture new and locum staff. It is important that the differing presentations of elderly people are recognised, and that specialist questioning skills are used.

The signs of suicide amongst adults are often not recognised by the relatives and friends of older people and by the general community.[6] Public awareness raising with regards to elder suicide should be considered. Finally, research is required that helps to illuminate the causes and treatment of self-inflicted death by older people.

Name withheld by request

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Prostate screening

It took about 20 years for the cervical screening of women to be proved of value to women. Smear campaigns do not diagnose cancer. This will never happen for men's prostate because it is being proscribed. (Perhaps saving the Government's money could be the main reason—biased research has always been suspect.)

What has happened to the principle of the early diagnosis of malignancy? Let the facts of epidemiological science be as they say. The need for men, to have freedom and support for their health needs, should be obvious. Let the patient find whether there is cancer, and then decide any treatment.

Do we now have an ethic which says 'the Government has found that the diagnosis and the various treatments are so onerous that it will not permit (pay for) this established clinical condition to be decided by the patient/doctor'—it, the Government, will decide what is best.

Perhaps the power and success of political correctness has gone to their heads so they can use it to control the thinking, experience, and freedoms of the doctor and patients. They plan to 'educate both the people and the doctors'. Will we have a report on how successful the campaign has been, and what it cost?

Will our medical profession, and our Association lie down, agreeing that this is yet another place were the Government can save money for more important things in the health budget?

I not only cringe but feel like crying for those men and their families who will find they have ongoing cancer (diagnosed only when signs and symptoms appear), when it could have been found much earlier—but for the actions of the controlling authorities.

Bruce Conyngham
Retired specialist in obstetrics and gynaecology
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New Zealand guideline for management of stroke

The New Zealand guideline for management of stroke[1] has just been released. Two pivotal messages of these guidelines are that all District Health Boards (DHBs) should provide an organised stroke service, and that all people who have a stroke should expect to be admitted to a stroke unit.

Unfortunately, most DHBs fall far short of these guidelines (at present) with only one acute stroke unit[2] and one dedicated stroke rehabilitation unit[3] in New Zealand. Why has New Zealand been so slow to develop stroke units, when it has been clearly shown that they save lives, reduce dependency, and increase the chance of patients returning home to live?[4]

The reasons are not clear to us, although inertia within large organisations and resistance to changing existing services may be some of the reasons.[5] Another reason that has been put forward is that stroke care in generic Assessment, Treatment, and Rehabilitation (ATR) wards, such as commonly occurs in New Zealand, is not significantly different from that provided in stroke units.[4]

Our experience of changing an existing ATR ward to a dedicated stroke rehabilitation ward, however, does not support this suggestion—as it resulted in a significant reduction in total length of stay (LOS) in hospital (8.0 days) with similar discharge domicile outcomes.[5] Such data reinforce the importance of implementing these guidelines. In this letter, we would like to present additional patient outcome data from this change to a stroke specific rehabilitation ward.

Two cohorts of patients, discharged with a diagnosis of an acute stroke, were followed prospectively at 3 and 12 months post-discharge. The control group was a random selection of patients discharged from any hospital in 2000 (“Before”). Our stroke rehabilitation ward (SRW) opened on 19 February 2001, so the intervention group (“After”) was a similar random sample, but discharged after 19 February 2001. Originally, a more radical reorganisation of stroke services was envisaged, with an acute stroke unit, a community based rehabilitation team, and the inpatient rehabilitation ward. Only the latter has so far come to fruition, so only those patients who needed further in-patient rehabilitation in an ATR ward were affected by the changes. The evaluation project was therefore modified to include only these patients.

Postal questionnaires were sent to patients and included the following assessments—London Handicap scale (LHS),[6] Barthel Index (BI),[7] Nottingham Extended ADL (E-ADL) scale,[8] Carer Strain Index (CSI).[9] Total length of stay (LOS) in hospital (both acute and rehabilitation) was also collected. The total number of patients (response rates in parentheses) for the before and after cohorts were 60 (35%) and 59 (32%), respectively. Of these, 21 and 30 patients, respectively, came to an ATR ward or SRW and their data were further analysed. Univariate analysis of variance was used to correct for the significant age and gender imbalances. The results are shown in Table 1.

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Table 1

		Before (mean)	After (mean)	F statistic	P value between group comparison
Total Length of Stay in acute and rehabilitation hospitals (days)		46.4	31.0	6.09	0.02
Barthel Index*	3 months	16.5	15.4	0.003	0.96
	12 months	15.0	14.8	0.003	0.96
Nottingham Extended ADL†	3 months	38.4	31.7	0.48	0.49
	12 months	32.0	29.9	0.003	0.96
London Handicap Scale‡	3 months	12.5	15.8	1.46	0.24
	12 months	13.6	17.2	1.27	0.27
Carer Strain Index§	3 months	2.6	2.6	0.09	0.77
	12 months	4.01	2.9	0.07	0.79

*Higher scores reflect greater independence; †higher scores reflect greater independence; ‡lower scores reflect lower handicap; §higher scores reflect more carer strain.

This small prospective study shows that patients in both groups had similar functional outcomes, but following the introduction of the SRW, patients left hospital significantly earlier. Discharge domiciles were similar.[5] In other words, patients got to the same level of functioning, faster.

Implementation of the guidelines with organised stroke services around the country is vital. This small study shows that 'win-win' scenarios are possible. Patients 'win' with similar outcomes (but get home sooner), and the health system 'wins' through shorter time spent in hospital. It is feasible, therefore, to implement some of these guidelines in New Zealand. Let us all rise to the challenge.

Acknowledgement: This small study was funded (in part) by 'Canterbury Health Care of the Elderly Education Trust' and 'Elder Care Canterbury'.

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Dr 'M' versus the Medical Council: Dr 'M' wins—and the Medical Council's conduct is heavily criticised by the Auckland District Court

Last October, Judge GV Hubble, in the Auckland District Court, delivered a judgement upholding an appeal against an adverse competence review by the Medical Council of an Auckland doctor, Dr M. In his concluding remarks, the judge said this:

"The end result is in my judgement a miscarriage of justice, and the only appropriate step is to set aside the decision of the Council to embark on a competence review".

The case began five years before when the Council received letters of complaint against Dr M from two apparently independent doctors. The letters arrived within two days of each other, and were of substantially similar content. The Council immediately referred the letters to the Health and Disability Commissioner who carried out a full investigation of Dr M's practice, and found that he did not in any way breach the code of consumers rights.

The report of the HDC however, failed to reassure the Council. As the judge noted:

"The next, very surprising, development is that in a letter from the Medical Council to [Dr M], he is advised that a competence review should be done…despite the fact that…the complaints had been shown to be, if not vexatious, certainly without foundation".

And earlier:

"What then followed were numerous attempts by [Dr M] to obtain a copy of the letters which were complaining about his practice…The Medical Council refused to part with this information. This in my judgement, was a fundamental breach of natural justice, the Bill of Rights Act, and the Council's own code. This required that a complainant be advised that a letter of complaint would be 'made available to the particular practitioner complained about, so that that the practitioner would at least have the opportunity of knowing what he or she needed to answer'. The letters of complaint were, of course, ultimately provided, but only after many months of pressure and threats of legal proceedings".

It is inconceivable that all members of the Council conspired to deny Dr M copies of the original complaints. Who then was responsible for withholding the copies? Was it a member of the Council? Or was it a member of the Professional Standards Committee? Whoever it was, that individual or individuals displayed a total lapse of judgment and fairness that has no place in the Medical Council of New Zealand. It could fairly be questioned whether disciplinary or any other action should be taken against this person or persons, as such failures bring the Medical Council into disrepute and undermine the profession's confidence in that body.

A further finding by the Court was the inappropriate appointment of the convenor of the committee which reviewed Dr M's practice, this person being the only practitioner (Dr Z) in Auckland in direct competition with him. The judge commented as follows:

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"Further, and more importantly, she (the convenor) is in direct competition with (Dr M), and it is inescapable that behind the laudable altruism…is an issue of money and income. [Dr Z's] personal interest…could not be described as either indirect or remote. She has a clear conflict of interest. It is not an issue of whether she was actually biased, and it is not suggested that she was. It is an issue of the appearance of possible bias, which in this case is obvious".

Earlier the judge had observed that (Dr Z**) "ought not to have been appointed to the** (review) **committee, and having been invited to sit ought to have disqualified herself".**

In preparation for the appeal, two highly respected Auckland clinicians Associate-Professor Clifford Tasman-Jones and Associate-Professor Tom Marshall) examined Dr M's practice. Both emphatically disagreed with the findings of the Council's competence review committee and with the Council's original determination, verifying instead, that Dr M's practice and methods were of an acceptable and high standard. It is astonishing that the Council remained unpersuaded by the two professors, while preferring the views of Dr Z, a practitioner with lesser qualifications, and "a clear conflict of interest" which was made known to the Council prior to the appeal hearing. This calls into question the objectivity of the Medical Council in its approach to competence reviews where there are conflicting opinions as to the standard of practice of a medical practitioner.

The wider profession can learn from this saga. Any doctor required to undergo a competence review should treat it very seriously. He or she should immediately seek the advice of the MPS. The right to have a support person present at the review should be exercised, and meticulous notes should be taken, remembering that this case extended over a period of five years.

Drs Michael Cooper, David de Lacey, George Hitchcock (all three doctors are past presidents of the Auckland Division of the Medical Association).

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Patient acceptability of home intravenous antibiotic therapy

We have previously reported in the Journal that 20% of patients treated with home intravenous antibiotics experienced some problems.[1] Most problems related to the peripherally inserted central catheter (PICC) or midline catheter. A smaller number of problems were associated with a spring-driven or elastomeric-continuous-infusion device carried in a belt bag and replaced daily either by a nurse or the patient.

To determine patient acceptability of this service, 100 consecutive patients were posted (6 weeks after they finishing treatment) a self-administered questionnaire with a stamped, addressed, return envelope. If a reply was not received within 2 weeks, the patient was contacted by telephone, and if needed, sent an additional questionnaire. An independent person conducted the survey and all responses were anonymous. The study was funded from Canterbury District Health Board sources.

Eighty-eight questionnaires were returned, and answers expressed as a percentage of responses to each individual question. The median patient age was 63 years (range 9–87 years). Seventy-two (82%) patients had a PICC line; 15 (17%) a midline; and 1 (1%) a peripheral catheter. Sixty-four (73%) patient had pumps (Homepumps® 7, Accufuser® 11, Paragon® 46), and 24 (27%) received intermittent intravenous therapy.

Overall, home intravenous therapy was rated as ‘good’ or ‘very good’ by 85 (97%) of respondents, and 2 (2%) would not have it again. When patients were asked to comment freely, 49 (91%) of replies were extremely positive. The commonest reason was that they preferred being at home to being in hospital.

The experience of having a PICC or midline inserted was rated as ‘very good’ by 52 (60%), and ‘good’ by 30 (35%) of patients, respectively. Of the 73 patients who had previously been treated using peripheral cannulas, 69 (94%) preferred a PICC line or midline.

Twenty-three patients (28%) reported some problem with the intravenous catheter—leakage 6 (7%), blockage 5 (6%), discomfort 6 (7%), inconvenience 3 (4%), arm swelling 1 (1%), skin reaction to dressing 1(1%), and connector becoming undone 1 (1%).

Fifty-six (88%) of responders with a pump device rated this as ‘good’ or ‘very good’. Forty-four (70%) of respondents had the bag in bed with them, 5 (8%) reported discomfort or restricted movement, and 2 (3%) reported kinking (reducing antibiotic flow). Twelve (14%) patients self-administered antibiotics—and all would do so again. Of those respondents who did not self-administer, 20 (27%) would liked to have done so—the main reasons offered were freedom from appointment times with nurses.

This survey shows that the patients regarded the problems (they encountered) as manageable and preferable to being in hospital and having peripheral cannulas inserted repeatedly. The infusion devices were accepted and offer the advantage of allowing administration of narrow spectrum agents with a short half-life. Because of

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the preference for self-administration expressed by some patients, we intend to increase this both for replacing infusion devices and giving boluses—but we believe that self administration is inappropriate for many older immobile patients.

Self-administration is particularly suitable for younger patients and those who require temperature-sensitive agents such as penicillin or amoxycillin. It reduces ongoing costs—but requires more patient training, which may delay hospital discharge.

Stephen Chambers
Infectious Diseases Physician
Christchurch Hospital

Kate Gallagher
Nurse Specialist
Christchurch Hospital

Alan Pithie
Infectious Diseases Physician
Christchurch Hospital

Reference:

1. Chambers S, Gallagher K, Metcalf S, Pithie A. Home intravenous antimicrobial service – twelve months experience in Christchurch. N Z Med J. 2002;115:216–8.

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Professional Misconduct – Failure to Refer

Charge:

The Director of Proceedings charged Dr Hauptfleisch with professional misconduct in respect of his care and management of a patient between 26 April and 27 April 2001.

The particulars of the charge alleged:

1. On or about 26 April 2001 he failed to consult with, or refer the patient, to a specialist or other medical practitioner in a timely manner for the purposes of excluding or confirming a diagnosis of intracranial haemorrhage or other abnormality in the brain.

AND/OR

2. On or about 27 April 2001, having been advised by his patient's husband that there was no improvement in his patient's condition he:

    b. failed to refer his patient, or assist referral of his patient, to a specialist in a timely manner.

At the commencement of the hearing Ms Baker withdrew particular (2)(a) of the charge.

Background:

The patient was 47 years of age and had had epilepsy since she was 11 years old. Although she did experience some migraine headaches at the onset of puberty she had not experienced many headaches and had gone for about 18 years, from 1973 until 1990, without a fit.

In November 1992 she experienced pain in her head while at work. She said it felt as though there was a band right around her head. It was very sudden. She remembered that she was in so much pain that she couldn't answer the phone. She remained unwell for a number of days and after having two grand mal seizures she was taken to Palmerston North Hospital where she was discharged after two days. A subsequent CT scan revealed that she had an arteriovenous malformation ("AVM"). There was some suspicion that her experience in 1992 was a brain haemorrhage but because of the time between the event and the scan it could not be confirmed. Following that incident the patient has continued to be monitored by Dr Bala Krishnan of the Neurosurgery Department at Wellington Hospital.

On the 26 April 2001 the patient was at work having returned from a month's holiday in the South Island. The patient described herself as feeling very relaxed and not feeling particularly stressed. At about 4.30 p.m she suddenly felt dreadful. She had an instant headache, starting in the back of her head and spreading around her head. She felt as though she had a tight band around her head and she felt really awful.

The patient sought help from the office manager. Her sister picked her up and on the way to the doctor's surgery the patient vomited. On arrival at the surgery she was placed in the nurse's room. She could not walk without aid and was holding her head because of the pain.

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When Dr Hauptfleisch came to see her she told him that she had had this before. He examined her including feeling the back of her neck and advised her that he considered that she had had a muscle spasm in her neck. The doctor's notes on that date stated:

"Tight band around back of head unable to hold head properly BP l(arm) 100/80 r (120/70) vomited x 1, Voltaren inj im 75/3 mls L(im)."

Dr Hauptfleisch's practice nurse said that the patient appeared to be in pain, had her eyes closed and was holding her head. I found it very difficult to get a good history from her and her sister often answered for her. She told me that she had a headache like a tight band around the back of her neck and it was exactly like that she had with an aneurysm several years ago.

It is not clear whether the advice given to Dr Hauptfleisch about the earlier incident was taken into account by Dr Hauptfleisch. The Tribunal did not have the benefit of hearing directly from Dr Hauptfleisch.

On Dr Hauptfleisch's instructions, the nurse gave the patient a Voltaren injection and was asked to monitor her.

Somewhere between 15 and 30 minutes after the Voltaren injection Dr Hauptfleisch came and asked the patient how she was feeling. Both the patient and her sister, who was with her, gave evidence that she had responded that the pain was alright as long as she didn't move her head. The nurse gave evidence that the patient's response was that her pain was less severe. What was clear was that the patient still required assistance to leave the surgery and to get into her sister's car. She vomited a few times during the night.

In the morning she was still in a great deal of pain. The patient's husband waited for the surgery to open at 8 a.m. as he wanted to talk to Dr Hauptfleisch. He relayed what had happened during the night and asked him to come and see his wife. Dr Hauptfleisch said that he could not visit as he had a surgery full of patients and asked for no further information. The patient's husband wanted Dr Hauptfleisch to ring Dr Bala Krishnan. Dr Hauptfleisch agreed to do so but said he could make no promises as to whether it would be that day or the following day. The patient's husband was somewhat frustrated and asked for Dr Bala Krishnan's telephone number. He was told to ring the surgery again and speak to the receptionist and get the number from her, which he did. On ringing Wellington Hospital the patient's husband was told that Dr Bala Krishnan was on leave. When he rang Dr Hauptfleisch again he was told to bring the patient in to see him. As he could not move her the patient's husband suggested that he bring her in by ambulance. The ambulance arrived and Dr Hauptfleisch came out to see the patient in the ambulance. Dr Hauptfleisch then said that she would have to be admitted to hospital and he wrote a referral letter to Palmerston North Hospital.

On arrival at Palmerston North Hospital a CT scan was performed. It confirmed that the patient had had a brain haemorrhage. She was then transferred to Wellington Hospital by helicopter. During the helicopter flight she had a seizure.

In Wellington Hospital the patient and her husband discussed treatment options and opted for stereotactic treatment. The patient remained in Wellington Hospital for

approximately two weeks, was transferred to Palmerston North for one night and then discharged. She underwent stereotactic surgery in Dunedin in June 2001.

It appeared that following these events there was no contact at all between Dr Hauptfleisch and the patient until the hearing before the Tribunal.

Finding:

The Tribunal found Dr Hauptfleisch was guilty of professional misconduct.

The Tribunal was satisfied that there were serious deficiencies in Dr Hauptfleisch's management of the patient regarding particular 1.

The Tribunal was of the view that Dr Hauptfleisch had before him the patient's notes, he was familiar with her, and she and her sister had both clearly made the point that this was similar to what had occurred in 1992. Dr Hauptfleisch, for reasons not before the Tribunal, chose to put that information to one side and diagnosed muscle spasm or tension headache.

The overwhelming evidence from expert witnesses was that where a patient was presenting with a headache that had begun from the back of the head and was accompanied by vomiting, there was a need for some diagnostic assistance, particularly a CT scan.

On particular 1 alone the Tribunal found Dr Hauptfleisch guilty of professional misconduct.

When considering particular 2(b) the Tribunal was of the view that Dr Hauptfleisch assisted in the referral of the patient at the insistence of her husband. It is of some concern to the Tribunal that without the patient's husband's insistence and, in his own words, "aggressive behaviour" the patient's referral to hospital might have been deferred for a longer period of time.

The Tribunal however did accept that on 27 April 2001 Dr Hauptfleisch, on assessing the patient in the back of the ambulance, did move in a timely manner to assist with her referral to Palmerston North Hospital.

The Tribunal considered that Dr Hauptfleisch could certainly have done more to assist the referral and to that end, the Tribunal considered particular 2(b) of the charge was made out and it amounted to professional misconduct.

Penalty:

The Tribunal ordered Dr Hauptfleisch be censured; pay a fine in the sum of $7,500.00 and pay 40% of the costs and the expenses that were incidental to the inquiry by the Director of Proceedings in relation to the subject matter of the charge, the prosecution of the charge, and the Tribunal's hearing of the charge.

It further ordered that a notice under s138(2) of the Act be published in the New Zealand Medical Journal.

Appeal:

An appeal against the decision has been filed on behalf of Dr Hauptfleisch in the District Court.

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The full decisions relating to the case can be found on the Tribunal web site at http://www.mpdt.org.nz Reference No: 03/101D.

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Barbara Mary Stone

Barbara Stone (nee Dana), OBE, a well-known Wanganui practitioner who held several influential health and disability appointments, died 27 October 2003.

Barbara was born in Surrey, England in 1925 and studied at Kings College London and the West London Medical School. Next she worked in paediatrics at Southampton Children's Hospital and then general practice in London, where she met her husband, Richard, who was completing postgraduate training there.

In 1955, they moved to Wanganui where she worked part-time as Medical Officer for the Department of Health and, after taking time out to raise four children, she became full-time before assuming the important position of Medical Officer of Health for the Manawatu/Wanganui Area Health Board from 1988 until her retirement in 1991.

Her work involved the remoter parts of the district (including Taumaranui, Ohura, Waiouru, Taihape, Rangitikei, and Patea) and she was one of the few Medical Officers in recent decades to occasionally visit patients by dugout canoe when they were on the other side of the Wanganui River. Indeed, the local Maori community spoke of her as 'the grandmother of the river'.

Barbara also served on the Wanganui High School Board of Governors, the Wanganui Hospital Board, and the Area Health Board where she chaired the Services Committee. In addition, she served on the local divisional committee of the NZMA, and she was involved for many years in Marriage Guidance, Plunket Society, Crippled Children's Society, and the IHC.

In 1970, she was invited to join the foundation steering committee of the College of Community Medicine and was made an honorary (and later full) member—until then, the first person to be so recognised. Later, when the College was incorporated into the Royal Australasian College of Physicians, she was elected a Fellow of the Faculty of Public Health Physicians of that College.

In recognition of her services to the community, she was awarded an OBE in 1992 and, after her retirement, served with the Wanganui Hearing Association (including as President), even after she moved to Wellington in 1994.

Barbara's hobbies included gardening (especially growing orchids), embroidery, and travel—and she enjoyed spending time with her five grandchildren (in whom she had an intense and rewarding interest) and her dogs.

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Deeply involved in her profession, she was strongly motivated to help young and old people who lived with some disability, and although possessing a conservative temperament, she could become a vigorous and effective activist if necessary.

Barbara is survived by her husband, Richard, a retired doctor and former medical superintendent of Wanganui Hospital—and their four children: Katherine (a general practitioner); and Richard, Christopher, and Andrew who all followed careers in business. Barbara's memorial service in Old St Pauls Wellington was attended by over 300 friends, colleagues, and representatives of the organisations with which she was so involved.

We are grateful to Barbara’s husband, Dr Richard Stone, for this obituary.

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The Royal Australasian College of Physicians
Adult Medicine Division

Call for Applications for

Foundation Fellowship of the Australasian Chapter of Sexual Health Medicine

The RACP has formed the Australasian Chapter of Sexual Health Medicine within the Adult Medicine Division. Foundation Fellowship will be available to experienced registered medical practitioners who practice in Sexual Health Medicine in Australia and New Zealand.

Those applying for admission will be considered on the basis of the following criteria:

1. Fellowship of the Australasian College of Sexual Health Physicians (FACSHP);
2. Broad experience in all aspects of clinical Sexual Health Medicine;
3. Ongoing contribution to Public Health policy development in the control of sexually transmitted infections on a population basis in Australasia or overseas;
4. Full-time academic position in Health Sciences relevant to Sexual Health Medicine at senior lecturer level or above;
5. Evidence of clinical training in Sexual Health Medicine;
6. Attainment of academic qualifications in Sexual Health Medicine;
7. Evidence of participation in Continuing Medical Education and Quality Improvement in the field of Sexual Health Medicine;
8. Evidence of contributions to the field of Sexual Health Medicine by:
   - participation in research in the field with appropriate supervision and collaboration
   - development of professional or academic activity
   - regular contributions to undergraduate/postgraduate education; and/or
   - publications in scientific journals and/or contributions to scientific meetings.

For specific details concerning eligibility, please refer to the detailed criteria in the *Guidelines for Determining the Eligibility of Candidates for Foundation Fellowship* in the Application Package.

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Applicants must demonstrate a satisfactory practice history (no professional misconduct, or disciplinary issues).

Foundation Fellows will participate in ongoing professional activity in the field of Sexual Health Medicine and are strongly encouraged to supervise trainees and participate in a Maintenance of Professional Standards (MOPS) Program. Payment of the annual subscription for Fellows is a requirement of the Chapter. Continued Fellowship is conditional upon a satisfactory practice history.

Application Process

Application Packages may be downloaded from the RACP Website at http://www.racp.edu.au/public/sexualhealth.htm

or obtained from:

Australasian Chapter of Sexual Health Medicine
Telephone: **+61 (0)2 9382 7457**
Email: sexualhealthmed@racp.edu.au

Closing date for applications: Wednesday 28 July 2004

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An introduction to complementary medicine

Terry Robson (editor). Published by Allen & Unwin Australia, November 2003. ISBN 1741140544. Contains 384 pages. Price $65.00 (Aust.)

This book achieves its title, namely a comprehensive introduction to complementary medicine. It recognises the diversity of complementary medicine, but selects four philosophies, ayurveda, indigenous healing, naturopathy, and traditional Chinese medicine as popular examples. It then describes 11 healing modalities—including acupuncture, aromatherapy, chiropractic, osteopathy, homeopathy, herbal medicine, counselling, and nutrition. It does not pretend to be all encompassing. Each chapter is written by a different practitioner, the vast majority of whom are Australian, with one New Zealander and one North American.

In each chapter, the basis of each philosophy or treatment modality is described, and it soon becomes apparent that the philosophies predate current biomedical medicine by centuries. Each chapter addresses whether there is evidence or research to support the complementary therapy described, discusses contraindications and possible adverse outcomes, and describes how it is currently practised. Some chapters give detailed references alongside each statement. Each chapter is followed by a short list of recommended reading, and at the end of the book there is a list of references per chapter. There is also an index, which appears quite detailed. The final chapter considers how complementary medicine will be integrated with standard health care, and notes that it is challenging to provide an evidence base, and that research to date has been largely based on the pharmacological effects of complementary therapies. It also speculates on training and licensing of practitioners.

My criticisms of the book include that some therapies are supported with anecdotes, and mainly describe patients with illnesses related to stress. Such anecdotes fall far short of evidence-based medicine. The main value of this book is as a resource of information about the various complementary medicines. The editor, Terry Robson, is to be congratulated for including a consideration of quality issues, contraindications and adverse outcomes. The book would be useful for students and practitioners in a range of health professions. Having being written predominantly by Australians it has more relevance for New Zealanders than similar books. There is, however, no consideration of Maori medicine.

It is a good starting place for further reading or research about the various complementary therapies. Although it takes a relatively positive view of complementary medicine, it does not see it as an alternative to mainstream medicine.

Bridget Robinson
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