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Vol 118 No 1222 ISSN 1175 8716

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The MaGPIe Research Group

The MaGPie study is a study of the prevalence, detection, and management of mental disorders in general practice attenders in New Zealand. It included a cross-sectional survey of GPs in the lower part of North Island, New Zealand. GPs completed a questionnaire about aspects of their provision of mental healthcare. At the time of the survey, GPs reported that consultations with patients with mental health problems took longer and could lead to increased waiting times for other patients. Many GPs subsidised mental health consultations either by not charging for longer consultations or writing-off fees. The subsequent establishment of primary health organisations provides potential for improving primary mental healthcare through specific contracts for mental healthcare, allowing variation in consultation length and the addition of mental health professionals to the general practice team.

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A newly recognised cause of vertigo: horizontal canal variant of benign positional vertigo

J Hornibrook

Vertigo is a frequent presenting symptom in general practice, the most common cause being benign positional vertigo (BPV). Proof that its cause is a “loose body” (detached calcium carbonate crystals) in a semicircular canal of the inner ear remains as one of the most unheralded medical discoveries of the late 20th century. In at least 10%, the “loose body” is in a horizontal canal, causing symptoms and signs that are often confused with other causes of vertigo. When the patient is upright there are no abnormal findings, which is why every vertiginous patient without nystagmus should undergo a provocative positional test. This paper describes 49 subjects with horizontal canal BPV; it details the mechanism and its simple treatment.

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Mental health treatment at the New Zealand GP

James Reid

Mental health services are an essential element of healthcare services. Moreover, promotion of mental health and the diagnosis and treatment of mental illness are an essential part of general practice.[1,2] Indeed, the continuity of care of general practice makes early recognition of problems possible.

In this issue of the *NZMJ*, the MaGPIe Research Group of the Wellington School of Medicine and Health Sciences report on general practitioners' perceptions to their provision of mental healthcare (http://www.nzma.org.nz/journal/118-1222/1654) in New Zealand.

Mental health issues are frequently unrecognised, and even when diagnosed, they are often not treated adequately.[3] Diagnosis and treatment of mental illness are significant issues for general practitioners, who provide the majority of mental healthcare.[4]

As outlined in the paper, although GPs are major providers of psychiatric care, they are discriminated against by methods of payment that create a disincentive to provide thorough and comprehensive mental health screening and treatment.[5] This also occurs outside New Zealand.[6]

However since the time of their survey, conditions in primary healthcare have changed with the development of the *Primary Health Care Strategy*.[7] With this model there has been a change from subsidised fee for service to capitation resulting in a more equitable fee structure. The addition of *Care Plus* to the model has further increased access. *Care Plus* is a new service that was introduced through Primary Health Organisations from 1 July 2004 onwards. It is aimed at people who often need to visit their general practitioner or nurse because of significant chronic illnesses (such as diabetes or heart disease), acute medical or mental health needs, or a terminal illness.

When considering the costs associated with mental illness, it is important to remember that mental health interacts with physical health, and that many patients with mental illness have comorbidities. For example, there is evidence that elderly depressives have a higher prevalence of ischaemic heart disease and subsequent death as compared with non-depressives.[8] Furthermore, patients with mental disorders have higher utilisation rates for general medical services and higher related medical costs compared with patients without mental disorders.[9]

Time constraints and workload, as outlined in this MaGPie study, are major barriers for busy general practitioners in providing longer consultation time. Multiple symptoms and problems are managed by family physicians. A visit to a psychiatrist typically can last a minimum of 30 minutes and is focused on mental health. In contrast, a general practice of 15 minutes may include multiple problems and follow a biopsychosocial model[10]—often moving from one presenting problem to another.

While psychiatrists are essential to the health system, they, along with a number of other disciplines in New Zealand, are in short supply—including general practice

itself with estimates of a shortage of 1400 general practitioners in this country.[11] However the majority of patients with mental health issues will continue to access the healthcare system through general practice and be managed there.

The desire of patients to receive treatment from their doctors, or at least to have their general practitioners more involved in their care, has been repeatedly documented. Improving mental health treatment requires enhancing the ability of the GP to treat and be appropriately reimbursed for that care.

Reimbursement mechanisms should recognise the importance of primary care in the treatment of mental illness as well as the significant issues of comorbidity that require nonpsychiatric care.

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Vol 118 No 1222 ISSN 1175 8716

Lawyers and letters

The palliative care and motor neurone disease article in this issue of the *New Zealand Medical Journal* by McKenna and MacLeod[1] would have gone largely unnoticed by many (including myself) if it wasn't for the interesting tug-of-war that has been going on around its publication. A letter from a lawyer, Mr McClelland,[2] in the letters to the editor section gives a clue as to what has been happening.

The *Journal,* as with most publications, at times finds itself drawn into a situation in which it must decline or withdraw publication because of legal action. The most public recent experience was in 2001 when the then editor Professor Gary Nicholls withdrew a paragraph in an article and left part of the page blank, except for the comment 'the paragraph was withdrawn for legal reasons'.

When the *Journal* receives an article, which (as editor) I am concerned that possible legal action may result from if published, a legal opinion is sought and discussion with the Journal Management Committee is undertaken. Recently such a series of events led to withdrawal of a letter to the editor without publication.

Today we publish a letter, which although does not threaten legal action, clearly is meant to intimidate the editorial staff. The editorial staff from time-to-time receive abuse from irritated authors about rejection of manuscripts, or about some issue related to publication of their article. However this sort of correspondence raises matters to another level.

In this case, an interested party who had not seen the manuscript made an initial compliant of plagiarism. An investigation was then undertaken which showed no evidence of this. Subsequently there was a claim that the questionnaire was developed from their original work, which the authors agreed was true. The authors were happy to acknowledge it, however the complainant did not want to have their name associated with the manuscript in any way. Then the letter arrived from Mr McClelland (a Wellington lawyer) on behalf of his client. A reply was sent, but alas no further correspondence to date.

Several issues raised by this letter to the editor are covered in *the Uniform Requirements for Manuscript Submitted to Biomedical Journals* (http://www.icmje.org/). These requirements continue to evolve and are updated regularly. There is a very good part on publication ethics, which I draw readers' attention to.

The *New Zealand Medical Journal* is one of the group members who develop the policy and uniform requirement statements. The most recent notable change related to the registration of clinical trials.[3] To others who are uncertain about the sort of issues raised by Mr McClelland, can I suggest that you review the uniform requirements.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

General practitioners' perceptions of barriers to their provision of mental healthcare: a report on Mental Health and General Practice Investigation (MaGPIe)

The MaGPIe Research Group

Abstract

Aim To explore GP attitudes and perceptions of barriers to providing mental healthcare.

Methods The MaGPie study included a cross-sectional survey of a random sample of 78 GPs in the lower part of North Island, New Zealand. GPs completed a questionnaire about aspects of their provision of mental healthcare including consultation fees, perceived barriers to providing mental healthcare, and factors likely to increase detection of mental illness in general practice patients.

Results Seventy (90%) GPs completed the questionnaire. GPs reported that consultations with patients with mental health problems took longer and could lead to increased waiting times for other patients. Many GPs subsidised mental health consultations either by not charging for longer consultations or writing-off fees. GPs thought that funded longer consultation times and more training in interviewing techniques would increase recognition of mental health problems in general practice.

Conclusions Structural aspects of general practice at the time of this survey presented a barrier to the provision of primary mental healthcare. The subsequent establishment of primary health organisations provides potential for improving primary mental healthcare through specific contracts for mental healthcare allowing variation in consultation length and the addition of mental health professionals to the general practice team.

The MaGPie study is a study of the prevalence, detection, and management of mental disorders in general practice attenders in New Zealand. General practitioners (GPs) in the MaGPie study thought that 54% of female and 46% of male patients had at least some psychological problems in the past year. An in-depth interview, using the Composite International Diagnostic Interview (CIDI), found that more than one in three general practice attendees had had a diagnosable mental disorder during the past 12 months.

The most common mental disorders were depressive, anxiety, and substance-use disorders. These disorders were more prevalent amongst younger than older general practice attenders. Over half of those with a disorder in the last 12 months also had another comorbid disorder.[1]

In New Zealand, general practice is the first point of contact with the health system for most, and 80% of adults report seeing a GP at least once in a year.[2] GPs act as both the gateway and gatekeepers to secondary and tertiary care, including mental healthcare, with the exception of emergency presentations. Primary care, including general practice as well as pharmacological and laboratory charges, is funded through

a mix of public subsidy and patient fees—with greater subsidies currently for people on low incomes enrolled with primary health organisations serving predominantly low income populations; for children; and for those aged over 65. Other users pay the full costs of seeing their GP. Specialist mental healthcare is fully subsidised and currently serves 1.6% of the population.[3] Psychological services for other patients are also available in the private sector and through non-governmental social and community groups.

The GP's role in mental healthcare is therefore very important and up to three quarters of all mental healthcare in New Zealand is delivered from a primary healthcare context.[4] Data from the MaGPie study has shown high rates of GP recognition of mental health symptoms in patients, at least in patients with whom they have an ongoing continuous care relationship.[5]

As part of the cross-sectional phase of the MaGPie study, GP's attitudes to mental healthcare, and the way in which they delivered mental healthcare, were explored to identify GPs' perceptions of barriers to the provision of that care. A greater understanding of GPs' perceptions of the barriers and challenges they face in delivering primary mental healthcare will contribute to the development of initiatives (such as service structures and funding arrangements) to facilitate the provision of effective primary mental healthcare.

Materials and Methods

Data collection—Data were collected as part of the cross-sectional phase of the MaGPIe study—a study of the prevalence, outcomes and management of common mental disorders in New Zealand general practice. Methods are described in detail elsewhere.[1]

GPs were selected at random from a list of all 299 known GPs in a geographical area encompassing the administrative health districts around and between Wellington City and Palmerston North in the lower part of the North Island of New Zealand (a mix of urban, small town and rural practices). GPs were eligible to participate in the study if they were currently practicing at least half time without restriction (e.g. due to ill health or compulsory supervision).

GPs were asked to complete a brief questionnaire before participating in the MaGPie Study, and the results presented in this paper are drawn from responses to that questionnaire.

The questionnaire included both open and closed questions and covered the following topic areas:

- GP demographic profile, mental health training, and experience;
- Time allowed for mental health consultations;
- The costs of mental health consultations and the billing system used by the GP; and
- The GP's perception of a range of barriers to the provision of mental healthcare.

The questionnaire was based on questions used in the St Louis Epidemiologic Catchment Area Study[6] and the Christchurch Psychiatric Epidemiology Study.[4]

The Wellington and Manawatu-Whanganui Ethics Committees approved the methods and procedures used in the study.

Statistical methods—Statistical analyses were carried out using Statistical Analysis Software (SAS) version 8.2. Prevalence estimates and 95% confidence intervals (CI) were derived using the SAS procedure SURVEYMEANS.

Results

Seventy of the 78 (90%) randomly selected eligible GPs participated, comprising 56 males and 14 females. Participating GPs were approximately equally distributed between the 25 to 44 years age group (36 GPs) and the 45 and over age group (34 GPs). Twenty-two (31.4%) GPs had at some time worked in posts in a mental health

field. There was no significant difference between the proportion of male (32.1%) and female (28.6%) GPs who had held posts in a mental health field (chi-squared=0.07, p=0.80). While more younger (38.9%) than older (23.5%) GPs reporting having held posts in a specific mental health field, this difference did not reach significance (chi-squared=1.27; p=0.26). Twenty GPs (28.6%) had undertaken training courses in mental health. A greater proportion of younger (36.1%) than older (20.6%) GPs (chi-squared=1.37; p=0.24) and of female (42.9%) than male (25.0%) GPs (chi-squared=0.98; p=0.32) reported such training, but these differences were not significant.

Approximately one-third of GPs (37.1%) reported their practices held contracts or agreements for the provision of mental health services. Patients with mental health problems could be identified from disease registers by one third of all GPs (34.3%). Published guidelines were reported by almost two-fifths (38.6%) as being used for the care of patients with mental disorders. Guidelines prepared by a local Independent Practitioners Association (IPA) were frequently cited as used.

GPs were asked to indicate on a three-point scale (very much, somewhat, not at all) the extent to which they agreed with statements about attitudes to mental healthcare. There was agreement 'very much' by 47.5% with the statement 'I am interested in providing care for patients with mental health problems' and 47.1% agreed they had no personal difficulties in dealing with mental health patients (Table 1). GPs were also asked about how confident they felt in the diagnosis, treatment and referral of patients with mental health problems. Approximately one-quarter of GPs agreed 'very much' that they were confident in diagnosis and management. More (62.9%) agreed 'very much' that they were confident in referral.

Table 1. GPs' attitudes to the provision of mental healthcare

Statement	Extent of agreement with statement					
	Very much		Somewhat		Not at all	
	n	%	n	%	n	%
I am interested in providing care for patients with mental health problems	33	47.1	35	50.0	2	2.9
I have no personal difficulties in dealing with mental health patients	33	47.1	32	45.7	5	7.1
I am confident in the diagnosis of mental health problems	18	25.7	51	72.9	1	1.4
I am confident in the treatment of patients with mental health problems	16	22.9	54	77.1	0	0.0
I am confident in the referral of patients with mental health problems	44	62.9	23	32.9	3	4.3

GPs were asked which of four options they considered the most important in increasing a doctor's detection of psychological disorders (Table 2). The most frequently selected options were 'more time in the consultation' (44.3%) and 'better interviewing techniques' (37.1%).

Table 2. GPs' perceptions of the factors most likely to increase their detection of psychological disorders in patients

Statement	GPs agreeing with statement	
	n	%
More time in the consultation	31	44.3
More formal training in psychiatry	9	12.9
More appropriate interviewing techniques	26	37.1
Use of structured questionnaires about psychological disorders	3	4.3
Total*	**69**	

*One GP did not complete this part of the survey.

When GPs were asked to consider the extent to which a range of factors limited the ability of mental health patients to come to see them: cost and a reluctance to seek help for mental health were perceived as barriers to a greater extent than patient concerns about confidentiality (Table 3).

Table 3. GPs' perceptions of barriers to providing mental healthcare for their patients

Statement	Extent of agreement with statement					
	Very much		Somewhat		Not at all	
Barriers to patients coming to see the GP:						
- The cost of coming*	19	27.1	41	58.6	9	12.9
- A reluctance to seek help for mental health problems	15	21.4	53	75.7	2	2.9
- Concerns about confidentiality	0	0.0	20	28.6	50	71.4
Barriers to GP provision of care:						
- Availability of mental health services	20	28.6	39	55.7	11	15.7
- Accessibility of mental health services	24	34.3	43	61.4	3	4.3
- The cost of private services such as counsellors/psychiatrists/psychologists	43	61.4	24	34.3	3	4.3
- The length of consultations	14	20.0	45	64.3	11	15.7

*One GP did not answer this part of the survey

GPs were also asked to indicate the extent to which four factors were barriers to them in providing care for their patients with mental health problems (Table 3); 61.4% of GPs considered that the costs of private mental health consultations were a substantial barrier. The availability and accessibility of mental health services were also perceived as substantial barriers by 28.6% and 34.3% of GPs respectively. Slightly fewer GPs (20.0%) considered the length of the consultation as a substantial barrier to providing mental healthcare.

Almost all GPs surveyed (92.9%) reported varying the amount of time they spent on consultations for patients with mental health problems. GPs explained that patients with mental health problems 'almost inevitably have longer consultations, often involving the practice nurse, receptionist, and often telephone follow up.' A problem arising from these, usually unexpected and unplanned, longer consultations was 'delays for other patients.'

Approximately half of the GPs (54.3%) varied their consultation fees for patients with mental health problems with some writing off charges: '[I] take it on the nose and write it off,' and others reporting that 'longer consultations may attract a higher fee but it is unrealistic to charge on a strictly time spent basis.' Most (90.0%) practices had a system to help patients who may otherwise have difficulty in the payment of consultation fees. Options available for patients included deferred payment systems, automatic payment systems, not charging for longer consultations, and writing off fees. The need to help regular patients was emphasised. One GP expressed some frustration at a forced choice between seeing patients with mental health problems at his own expense or having to give less time to their problems than was appropriate.

At the end of the questionnaire, GPs were asked if they had any comments about the provision of mental health services in their locality. The overwhelming number of comments related to difficulty in accessing public mental health services, especially specialist services for counselling, postnatal care, and child and adolescent services. As one GP said 'MH services seem under-resourced and over-stretched to a certain extent, but give a good service in relation to the nil service offered when I started in practice.' Patients most affected by limited access were those with non-acute problems and 'complicated presentations that cross disciplines.'

There were locality-specific differences in opinions regarding the quality of public mental health services, the merits of community mental health teams, and the ability of crisis teams to respond to urgent cases. There was also confusion about the criteria for accessing publicly-funded specialist mental health services: '[it is] constantly changing and difficult to know exactly what is available and where the boundary between appropriate general practice and secondary services is.'

Discussion

This paper reports part of a study of the recognition and subsequent management of common mental disorders in general practice attendees; the MaGPIe Study. The 90% response rate from GPs provides some assurance that our results are broadly representative of New Zealand GPs.

GPs have an important role in the provision of mental healthcare in New Zealand. Mental illnesses such as anxiety, depression, and substance-abuse are prevalent amongst general practice attendees,[1] and effective primary care management of mental illness requires identification of patients with mental health problems and then ongoing management of the patient.

In the Magpie study, when comparing GP recognition with assessment of patient symptoms using the Composite International Diagnostic Interview (CIDI), there was a higher recognition of mental health problems if there was more continuity of care in the GP's relationship with the patient.[5] In the survey reported in this paper, GPs perceived structural aspects of general practice as barriers to identifying patients with mental health problems. In particular, GPs highlighted the (short) length of the consultation as a barrier.

Having more time available in the consultation was thought by a greater number of GPs to increase recognition than either the use of structured questionnaires about psychological disorders or more formal training in psychiatry. The value of increased

consultation time as an enabling factor facilitating high quality outcomes has been recognised in other aspects of general practice.[9]

Many GPs also highlighted the need for additional training in appropriate interviewing techniques, a theme which has been discussed in other papers.[7,8] Identification of the need for additional interviewing techniques may reflect difficulty in accessing counselling for patients, especially cognitive behavioural therapy.[11]

In this survey, GPs were asked about factors that might affect their ongoing management of patients with mental health problems. GPs reported difficulty in accessing specialist services for patients with non-acute conditions and some lacked confidence in the quality of the services. Given that specialist mental health services deliver care to only 1.6% of the population,[3] general practice will continue to have an essential role in the provision of mental healthcare, particularly for patients with high prevalence conditions. GPs certainly consider mental healthcare to be an integral part of their role.[7,8] The challenge for policymakers is to facilitate the provision of mental healthcare through general practice.

The length of the consultation was also perceived to be a barrier to providing care for patients with mental health problems as these patients often required a longer consultation time than the standard 10 to 15 minutes. As longer consultations were often unscheduled, they presented problems for general practices as they extended the waiting time for other patients. This work is complimentary to recent studies exploring patient awareness and concerns about time constraints with mental health consultations, in which patients displayed self-imposed restraints in taking up the doctor's time.[10] Scheduled longer consultations were also problematic, as GPs often felt unable to charge patients for some or all of the additional consultation time.

After the completion of this survey, Primary Health Organisations have been established in New Zealand consistent with the New Zealand primary care strategy.[12] Primary Health Organisations have the potential to provide a vehicle for the development of different ways of providing primary care services. A recent request for proposals by the Ministry of Health[13] for extending and improving primary mental healthcare has seen a tremendous response by primary health organisations. A wide range of initiatives have been suggested including proposals for additional consultation time, practice-based counselling services, community services, and mental health facilitators to co-ordinate care between primary and secondary services.

General practitioners in this survey identified several barriers preventing them achieving the full potential of their involvement in primary mental healthcare. Barriers to diagnosis included the length of the consultation and a need for more training in appropriate interviewing techniques. Barriers to ongoing management included a lack of time, access issues connected with the payment structure, their own training needs, and challenges associated with the successful liaison between primary and secondary care.

Identification of the consultation time as a barrier to both diagnosis and management, combined with MaGPie study evidence that GPs are more likely to recognise the symptoms of mental illness in patients they know,[5] suggests that a focus on increasing the length of the consultation has the potential to improve primary mental healthcare provisions. Additional funding for mental health consultations is one way of addressing this problem. There is also potential to improve the ongoing management

of patients with mental illness by improving access to counsellors, psychiatrists, and psychologists. The recent primary mental healthcare initiatives will hopefully go some way towards addressing these issues.

Author information: The 'MaGPIe' (Mental Health and General Practice Investigation) Research Group consists of a management committee and an advisory committee. The management committee that undertook day-to-day oversight and management of this study consisted of John Bushnell, Deborah McLeod, Anthony Dowell, Clare Salmond, and Stella Ramage. The Advisory Committee consisted of Sunny Collings, Pete Ellis, Marjan Kljakovic, and Lynn McBain.

Members of both committees were involved in the detailed planning of the study and have reviewed this paper. Deborah McLeod drafted and revised the paper and is the correspondent.

Acknowledgements: The Health Research Council of New Zealand funded the project with supplementary funds contributed by the New Zealand Alcohol Advisory Council. We are also grateful for the support of participating general practitioners, patients, other practice staff, and our research staff.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Access to palliative care for people with motor neurone disease in New Zealand

Christine McKenna, Rod MacLeod

Abstract

Aim To identify the services available through hospices for people with motor neurone disease (MND) in New Zealand; to find out about the type of care and support available in each service; and to identify any barriers to access to care for these people.

Method A postal questionnaire was sent to hospices and palliative care services who were listed in the Hospice New Zealand directory (N=41) in March 2004.

Results Thirty-five services returned completed surveys (85% response rate). All services responding offered care for people with MND, with a wide range of services being offered. Results indicated that for a small number their service contract was a barrier to accessing services. One identified the issue of bed availability for respite care and another expressed concern about long-term care. Other challenges noted included the high level of time and resources needed to care for people with MND; the complexity of multidisciplinary care; the knowledge of the disease process needed; the duration of the condition and the uncertainty of prognosis; the different focus of care compared to the care of people with cancer; and the ability to find appropriately skilled carers.

Conclusion Palliative care services have much to offer in the care of people with MND particularly in symptom management, respite care, and in addressing the psychological and spiritual issues that have been shown to have a greater bearing on quality of life than physical functioning. Co-ordination of service provision and timely referral to palliative care services are essential if the optimum care is to be provided. The development of the knowledge base required for effective care, a systematic approach to providing support, and effective coordination are all essential to improve the quality of life for people with MND and their families.

Motor neurone disease (MND) is a rare neurodegenerative disease which occurs in about 1–3 people per 100,000 population per year; it has a prevalence of about 5–9 per 100,000 (depending on the survival of those affected). The New Zealand Palliative Care Strategy[1] states that the palliative care needs of people with conditions such as motor neurone disease need to be recognised. (In this paper, the terms MND and amyotrophic lateral sclerosis [ALS] will be used synonymously.)

The care of people with MND is described by a number of writers as palliative from the time of diagnosis, notwithstanding that some treatment options, which may delay the progression of the disease but are not curative are now available.[2–6]

The literature identifies significant concerns about symptom management in MND, especially in the terminal phase,[4,5,7] that almost all symptoms in MND/ALS are

amenable to palliation[8] and that hospices may be underutilised in the care of people suffering from MND.[9]

Furthermore, there is a high level of commonality of symptoms experienced by people with MND and people with a wide variety of cancers, with whom palliative care professionals are generally much more familiar,[7,10–15] and hence there is transferability of knowledge and skills. However, O'Brien et al cite the examination of a sample of patients referred to a hospice which found that whereas a high proportion of patients with cancer were referred specifically for symptom control only 15% of admissions of people with MND were referred for symptom control, despite the fact that they all suffered multiple symptoms (Table 1).[11]

A study of respite admissions to hospice for people with MND[10] found that the median number of problems identified per patient was 10, and that 81% of respite admissions resulted in a change of medications.

Table 1. Comparison of cancer patients' symptoms versus motor neurone disease patients' symptoms (on their admission to a hospice)

Symptom	PwMND	Cancer
Constipation	65%	48%
Pain	57%	69%
Cough	53%	47%
Insomnia	48%	29%
Breathlessness	47%	50%

Source: O'Brien et al[11]; PwMND=people with motor neurone disease

In 2000, an investigation of the involvement of specialist palliative care services in supporting people with MND in the UK showed variable involvement, with many services only involved in providing respite and terminal care.[16] The suggestion is made that early involvement is advantageous, as there is the opportunity for easier communication with the patient and therefore to obtain a clearer understanding of the patient's views on their care. The potential for late involvement to adversely affect communication and end-of-life care is reflected in the literature,[7,8,17] and in anecdotes from some palliative care services and people with MND.

Methods

The survey was designed and presented to hospices and palliative care services who were listed in the Hospice New Zealand Directory (N=41) in March 2004. This survey was based on unpublished work undertaken on behalf of the Motor Neurone Disease Association of New Zealand in 2001 to enable comparisons over time. A reminder was sent to the services that had not responded in April 2004. Thirty-five services returned completed surveys (85% response rate).

Results

The survey asked for a range of qualitative and quantitative information. To ascertain whether the level of experience and the issues identified were related to the size of the service, results were analysed by the number of new patients seen in 2003.

The following groupings reflected the number of new patients the service had seen in 2003 (Tables 2 and 3).

Table 2. Size of New Zealand hospice and palliative care services in 2003

Number of patients in service	Number of services that size
500+	5
250–500	10
100–250	5
< 100 (including unknown)	15

Table 3. Proportion of new patients with motor neurone disease in New Zealand hospice and palliative care services in 2003

Total number of new patients	New patients with motor neurone disease
500+	14+
250–500	31
100–250	9
< 100 (including unknown)	10
7373	64 (0.86%)

At the time of the survey, palliative care services were supporting 38 people with motor neurone disease (it is estimated that at any given time there are likely to be 250–300 people in New Zealand living with MND). These figures also suggest that at the time of the survey approximately 12–15% of people in New Zealand with MND were receiving support from palliative care services.

Every service that responded to the survey indicated that they offer care and support to people with MND—a small number of these services indicated that this would largely be confined to support in the community at the terminal stage only.

Services were asked to identify circumstances under which they would not provide care for people with MND. Four services identified that their service contract or the definition of palliative care as being for the last year of life was a barrier, one service identified issues around bed availability (particularly for respite care), and one service expressed concern about long-term inpatient care.

Both the numbers and the percentage of services willing to offer each form of support have increased since 2001. At every point from diagnosis to terminal care, hospices indicated a higher level of willingness to accept referrals of people with MND than in 2001, and that their willingness to accept referrals was greater than the level of referrals they actually received. Additional questions in the 2004 survey identified that services offered increased support (compared with 2001) for decision-making, advice, family/carer support, and bereavement support. Moreover, the data suggest that there was an increased level of involvement at every stage and in all aspects of care.

Respondents identified other challenges and issues which are annotated in Table 4 below.

Table 4. Other challenges and issues faced by respondents to the survey

High level of need—time and resource issues, funding	7 (20%)
Complexity of multidisciplinary and multi-agency care	3 (9%)
Knowledge	3 (9%)
Duration of condition and ability to estimate prognosis	3 (9%)
Different focus—finding appropriate carers especially when speech involved	1 (3%)
Contract—precluding shared care	1 (3%)
Contacting MND Association	1 (3%)
Community issues and issues for MND person/family	
Carer burden, support for carers	2
Equipment and housing modifications	2

Strategies

Three strategies for improving access and care for people with MND were identified by palliative care services responding to the survey:

- Earlier referral.
- Advisory systems.
- Shared care.

Earlier referral—Earlier referral is needed for the development of a relationship of trust while communication is generally easier. However, for some people, referral 'too early' can cause resistance to the concept of hospice.

Only one service has established guidelines for referrals in the form of information about its services for the person with MND and their family. The Palliative Care Strategy recommends that the introduction of palliative care or referral of a person to palliative care services should be guided by referral protocols. The development of these may be even more important in the case of MND because of greater issues around knowledge, familiarity, and confidence. Such guidelines have been developed in the United States by the American Academy of Neurology in their document reviewing the evidence base of the major management issues in patients with MND,[18] but the focus is largely on changing from working to maximise function to providing terminal care.

If the philosophy of palliative care is to assist people to have the best possible quality of life or to 'make the most of life,' earlier involvement is needed to achieve this objective, as well as to establish a relationship while communication is easier.

A 2000 study[19] found that quality of life, as assessed by the person with MND, does not correlate with measures of strength and physical function, but appears to depend

most on psychosocial and spiritual or existential factors—things that are very much within the ambit of palliative care.

Advisory systems—Advisory systems and shared care are supported by Dharmasena and Forbes[20] along with the suggestion that training schemes in palliative medicine include opportunities to gain experience in a variety of non-malignant conditions, such as MND.

No single group of health professionals has all the knowledge and skills required to support people with MND. A survey undertaken in 1995 of health professionals in the Wellington region aimed at identifying the responses of health professionals to working with people with MND and multiple sclerosis (MS)[21] found that those health professionals were significantly more negative about MND than MS in terms of the amount they felt able to offer patients and in their confidence in managing people with motor neurone disease.

They also felt generally less able to convey hope to people with motor neurone disease. The main reasons expressed for difficulty in conveying hope was the fact that MND is incurable, the inevitable and rapid progression of the disease, the high level of disability, and issues of identification with the person with motor neurone disease.

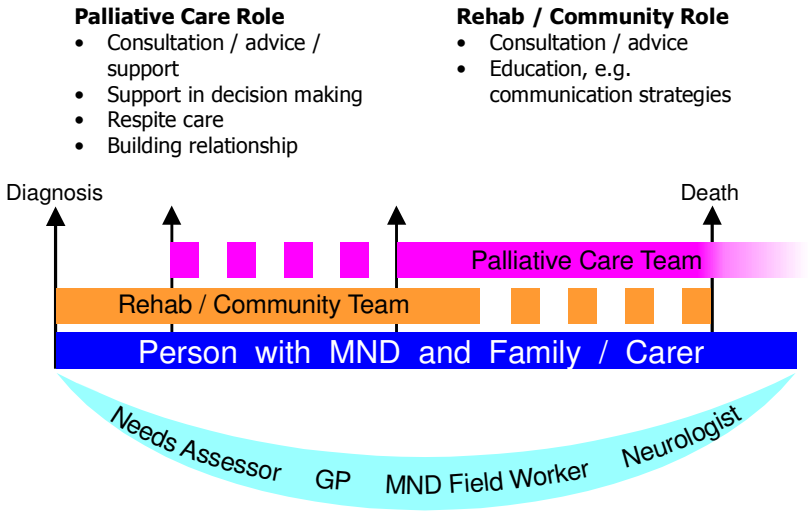
From this current survey there appears to be an increase in experience particularly in services that have dealt with a significant number of people with MND. Health professionals also identified a range of strategies that support them in working with people with MND. At the top of this list were strategies around supportive and well coordinated teamwork. This teamwork needs to be both within the palliative care service, and with other providers.

Shared care—While working with other providers poses some challenges, bringing together the range of skills required is likely to reduce the stress on health professionals and provide learning opportunities for all involved, particularly for hospice staff in relation to communication difficulties and for community teams in issues around dying. The involvement of hospice services should be complementary to the roles of the health professionals who have built relationships with people with MND both in hospitals and in the community, and not seen as a replacement for them.

An approach to the care of people with MND, adapted from the work of Mary Harmer from Te Omanga Hospice and Louise Rees, the Wellington MND Association Field Worker, which provides continuity is set out schematically in Figure 1.

Figure 1 illustrates the journey of the person with MND and their family/carer, and how it is proposed that care might be delivered. The key question is on what basis decisions for transitions in care should be made.

Figure 1. Schematic diagram of available services for a motor neurone disease patient and their family/carer in New Zealand



Specialist palliative care services

The Palliative Care Strategy recommends that specialist palliative care services be established in Auckland, Hamilton, Palmerston North, Wellington, Christchurch, and Dunedin, with particular responsibility for providing the specialist palliative care advice for the region.

Palliative care services in some of these areas already have significant experience in caring for people with MND, and are likely to have established a skill base. There is scope to enhance the knowledge and skills of other specialist services, to enable them to become a resource for palliative care services in their area regarding the care of people with MND. Perhaps as an interim measure, the services with experience and expertise might become 'centres of excellence' in palliative care for people with MND and provide a 'super-advisory' service.

The MND Association has a National Health Professional Special Interest Group covering a wide variety of health professionals. A specific MND Palliative Care Special Interest Group or Working Party may have a variety of useful roles:

- Exploring the need for additional funding to meet the high level of need that is common in MND.
- Identifying more precisely where specialised knowledge exists in the palliative care of people with MND.
- Sharing specific knowledge and expertise in education programmes and on a ongoing basis.
- Developing guidelines for referral to palliative care.

Conclusion

Palliative care services have a great deal to offer people with MND, particularly in symptom management, respite care, and in addressing the psychosocial and spiritual issues that have been shown to have greater bearing on quality of life than physical functioning. There is, unsurprisingly, significant commonality in the issues identified in this survey and our earlier study. Bringing together the individual issues identified the 1995 study and the organisational issues identified in this 2004 survey makes a systematic approach to the way forward clearer.

To provide effective services while minimising the risk of staff burnout (or an unsatisfactory experience for the person with MND and their family), it is important to:

- Ensure that the relationship with the person and their carer begins at an appropriate time,
- Have effective continuity with other services that have complementary skills,
- Support staff well. (This needs to involve the development of the knowledge base, a systematic approach to providing support and advice to services that do not have experience in managing MND, and effective coordination with providers with complementary expertise.)

These strategies should make it possible to work towards generally earlier (but carefully timed) referrals to make a real difference to the quality of life of people with MND and their families/carers.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Depression and compliance with treatment in a sample of Northland diabetes patients

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Abstract

Aim The purposes of the study were to compare self-ratings and clinicians' ratings of compliance with treatment for diabetes among New Zealand European and Maori diabetes patients living in Northland Province, New Zealand; and to examine the unique relationships of demographic factors, diabetic history, and depression to the ratings.

Method A questionnaire containing demographic items, a depression inventory and a compliance rating scale devised in consultation with clinicians was completed by 99 diabetes patients attending the Northland Health retinopathic clinic. Clinicians independently rated the patients on a similar scale.

Results Patients' ratings were unrelated to (but significantly higher than) clinicians' ratings. Patients were more depressed than a New Zealand normative sample; were more concerned than clinicians about treatment inconveniencing their lifestyle; and wanted more control over treatment options. Maori who strongly identified with Maori culture were as compliant as European patients, who in turn were more compliant than Maori whose identification was weak. Controlling for demographics and diabetic history, multiple regression analyses showed that depression uniquely accounted for variance in patients' ratings, but not in clinicians' ratings.

Conclusion Strategies to increase compliance include monitoring psychological wellbeing, establishing Maori case management clinics, negotiating individual treatment plans with patients and problem-solving training.

According to the Ministry of Health's projections to 2011, diabetes mellitus in the New Zealand population will increase dramatically, especially among Maori and Pacific Island peoples.[1] In addition to increases in mortality rates, effects will include increases in healthcare costs and deteriorating quality of life for sufferers. Health authorities provide medical and educational services for diabetes patients, but compliance with treatment is important for managing diabetes and its complications. With diabetes, the patient confronts the facts that treatment will not result in a cure of the illness, and that the treatment regime is likely to be complex, life-long, and require the patient to make many behavioural changes.

Compliance with diabetes treatment has been defined as the extent to which a diabetic's behaviour reflects or coincides with the health advice given.[2] It excludes failure due to memory lapses or to misunderstanding of treatment instructions, but occurs when patients intentionally follow the recommendations including self-management behaviours, changes in lifestyle and adherence to medication.

From a behavioural perspective, the learned helplessness model of depression[3] suggests that when an individual is confronted with a continuous, uncontrollable,

aversive situation, the person's ability to act in future stressful situations that *are* controllable is impaired. The resulting sense of helplessness might lead to depression and hence poor self-management and compliance with treatment. A cyclical result might occur: the diabetic who feels depressed is not motivated to comply with treatment, leading to further complications, which in turn make the person more depressed and even less compliant.[4]

The purposes of the present investigation were to compare self-ratings and clinicians' ratings of compliance with treatment for diabetes among New Zealand European and Maori diabetes patients living in Northland, and to ascertain the unique contribution of depression to the ratings. It was predicted that:

- Patients' ratings of compliance will be higher than clinicians' ratings;
- Maori with strong Maori cultural identification will be more compliant than Maori with weak identification;
- Diabetes patients' depression will be greater than a New Zealand normative sample; and
- Depression will be strongly associated with lower compliance, after controlling for relevant demographic variables and self-reported diabetic history.

Method

Sample—In the 4 months of April to July 2003, 130 diabetes patients attending the Northland Health retinopathy clinic were invited to complete a self-report compliance questionnaire; 99 of them were rated independently by three non-Maori nurse educators responsible for their respective patients, giving a response rate of 76.2%.

Table 1 shows the demographic characteristics and self-reported diabetic history of the sample. Their ages ranged from 28 to 88 years, with a mean age of 59.2 (*SD*=14.96) years. The average number of years diagnosed with diabetes was 10.9 (*SD*=8.77), ranging from 6 months to 38 years. Compared with the total population of diabetes patients being treated by Northland Health Limited from April to July 2004 (N=652); the sample was not significantly different for sex (chi-squared=0.02), average age, *t*-test (749)=1.40, or proportions of New Zealand European and Maori (chi-squared=2.85), *p*s>0.05.

Measures—A compliance rating scale consisting of behavioural, attitudinal, and lifestyle components was derived from a review of the relevant literature[5] and from focus groups including the doctors and other clinicians at the diabetes clinic. (See Table 2 for items.) Because none of the previous studies of compliance with diabetes treatment compared patients' with clinicians' perceptions of compliance, two parallel forms of the scale were constructed, with appropriate wording: a patient self-report scale and a clinicians' scale.

For example, "My treatment plan is working well for me" corresponded with "The patient's treatment plan is working well for this particular person". Each scale consisted of 20 items, and each item was rated on a five-point Likert scale from "never" (0) to "always" (4), with a maximum score of 80. Internal consistencies measured by Cronbach's alphas were high (0.82 and 0.91, respectively).

In addition to demographic items and the compliance rating scale, the patients' questionnaire included a self-report inventory of depressive symptoms for which there are some normative data on town and rural samples in New Zealand.[6] The inventory of 18 items, each rated on the same scale as the compliance measure, had a maximum score of 72 and an internal consistency of 0.93 for the present sample.

At the suggestion of the Apiha Maori / Maori Officer for Northland Health Mental Health Services, the questionnaire asked respondents who indicated New Zealand Maori for ethnicity to select the importance of the Maori language, culture, and spirituality to their daily life from four categories. Maori who selected "crucial" or "important" were classified as having a strong Maori identification. Those who indicated "aware of, but does not influence my daily life" or "not that significant" were considered to have weak identification.

Table 1. Sample characteristics, means and standard deviations of compliance scores and depression (N=99)

Variable	n	Self-report compliance scores			Clinicians' compliance ratings			Self-report depression scores		
		Mean	*SD*	*t/F*	Mean	*SD*	*t/F*	Mean	*SD*	*t/F*
Male	50	54.5	9.13	0.22	42.5	9.99	0.35	25.9	13.71	0.45
Female	49	54.9	9.55		43.1	8.67		27.1	14.01	
Strongly Maori	15	51.7	9.04	1.51	41.3	7.22	5.83‡	31.1	14.68	0.83
Weakly Maori	31	53.4	9.52		38.6	8.65		27.2	13.30	
European	43	56.8	8.92		46.8	8.96		24.7	13.48	
Other	10	53.7	9.90		40.9	9.67		25.1	15.73	
Single/separated	40	56.0	8.55	1.20	43.1	9.57	0.23	25.9	13.61	0.34
Married/de facto	59	53.8	9.73		42.6	9.23		26.9	14.03	
NZD$0–20,000	40	54.1	9.31	0.25	41.9	7.23	0.61	25.8	14.18	1.46
NZD$20–30,000	30	54.5	8.32		42.5	10.38		29.9	13.33	
NZD$30,000+	29	55.7	10.41		44.3	10.77		23.9	13.52	
Type 1 diabetes	44	53.0	9.43	1.61	41.0	8.98	1.70	29.7	12.82	2.08*
Type 2 diabetes	55	56.0	9.05		44.2	9.43		24.0	14.15	
Knows type	52	56.2	10.26	1.74	45.7	9.23	3.46‡	21.8	14.00	3.79‡
Does not know	47	53.0	7.86		39.6	8.39		31.7	11.67	
Family history	64	52.8	8.78	2.85†	41.3	9.21	2.27*	29.1	12.71	2.64†
No family history	35	58.1	9.32		45.6	8.98		21.7	14.58	
Complications	48	52.7	8.79	2.14*	42.1	9.55	0.73	29.6	13.94	2.23*
No complications	51	56.6	9.45		43.5	9.14		23.5	13.13	
Other illnesses	52	54.1	9.62	0.68	41.9	9.54	0.97	29.1	13.74	2.00*
No other illnesses	47	55.3	8.98		43.7	9.08		23.6	13.42	

*$p < 0.05$; †$p < 0.01$; ‡$p < 0.001$; NZD=New Zealand dollars.

Table 2. Mean scores* for compliance items (N=99)

Item	Patients'compliance ratings	Clinicians' compliance ratings	*t*-test
Most appointments are regularly attended	3.2	2.5	5.72
Always remember to take medication	3.1	2.3	6.84
Coming to clinic is difficult and/or inconvenient	2.9	1.9	7.72
Treatment plan is working well	3.0	2.4	5.06
Blood tests indicate that treatment appears to be working	2.9	2.3	4.37
Have to cancel appointments at short notice	2.8	2.0	5.57
Substitute suggested foods in diet with own choices	2.2	1.7	3.64
Staff often have to repeat treatment instructions	2.8	1.8	8.00
Understand why need special diet and exercise plan	3.1	2.5	5.28
Often in good mood when attending appointment	3.1	2.6	4.86
Open and accepting of guidance from staff	3.3	2.5	7.78
Suggested diet is working well	2.8	2.1	5.28
Asked questions about medication, risks, benefits †	2.2	2.1	0.53
Told staff that diet and medication is intrusive to lifestyle	2.7	1.7	7.77
Tend not to tell staff when forget medication†	1.6	1.9	-2.37
Would like more help from staff†	1.8	2.0	-1.66
Uncomfortable to tell staff when not managing condition	2.6	2.0	4.76
Patient should have more control over treatment	2.8	2.0	6.70
Think that too much fuss is made over having diabetes	2.7	2.2	4.72
In general, compliant with treatment	3.4	2.3	12.79

*Five-point Likert scale: 0=never to 4=always. Standard deviations are omitted for brevity and clarity. All *t*-values are significant ($p < 0.001$), except for items marked†

Procedure—Approval was granted by the Massey University Human Ethics Committee and Northland Health Limited. Because all diabetes patients are scheduled to attend the retinopathy clinic, it seemed the best source for the sample. The second author employed at the mental health unit was informed by retinopathy staff when patients arrived for appointments during times that she was available. She asked each patient if she or he was willing to complete the questionnaire and to return it in a sealed envelope. Three non-Maori nurse educators responsible for the patients independently rated their respective patients on the parallel compliance rating scale. To minimise the effect of social pressure, the patients were assured that their responses were confidential to the researchers and would not be seen by the clinicians.

Statistical methods—Data were entered into an SPSS data file, and analysed with SPSS-11 software. The significance of differences between mean self-ratings and mean clinicians' ratings of compliance for each of the demographic characteristics was examined by independent samples *t*-tests (two categories) or one-way analysis of variance *F*-tests (more than two categories). Coefficients of correlations among duration of diabetes, depression and compliance ratings were computed.

Results

Table 1 shows the mean scores of both compliance measures for the various groups. There were no significant differences in average ratings on either scale for sex, age, marital status, household income, type of diabetes, or other illnesses. Mean clinicians' ratings were significantly different for ethnicity, $F(3,95)=5.83$, $p<0.001$.

Post-hoc *t*-tests revealed that the average rating for Maori who strongly identified with Maori culture (mean=41.3, *SD*=7.22) was not significantly different from the rating for European patients (mean=46.8, *SD*=8.96)—but the latter was significantly higher than the rating for Maori whose identification was weak (mean=38.6, *SD*=8.65), *t*-test (72)=3.97, $p<0.001$.

Patients with no family history of diabetes had significantly ($p<0.05$) higher self-ratings, *t*-test (97)=2.85, and clinicians' ratings, *t*-test (97)=2.27, than patients who did have a history. Patients who knew their type of diabetes had higher clinicians' ratings than those who did not know, *t*-test (97)=3.46, $p<0.001$. Patients without severe complications rated themselves higher than those with complications, *t*-test (97)=2.14, $p<0.05$.

Compared with the average depression score for the New Zealand rural and town normative sample (mean=23.4, *SD*=12.12), the diabetic patients' average score (mean=26.5, *SD*=13.80) was significantly higher, *t*-test (422)=2.15, $p<0.05$. From Table 1, there were no significant differences within demographic groups on depression. Significantly higher depression scores were obtained by Type 1 diabetes patients, those who did not know their type, and those with a family history of diabetes, complications or other illnesses.

None of the scores for the compliance rating scale items correlated significantly with the corresponding scores on the clinician rating scale. On average, patients' self-ratings (mean=54.7, *SD*=9.30) were significantly greater than clinicians' ratings (mean=42.8, *SD*=9.32), *t*-test (97)=10.00, $p<0.001$. Paired samples *t*–tests (Table 2) revealed that the patients were significantly ($p<0.001$) more concerned about difficulties in coming to the clinic, diet and medication being intrusive to one's lifestyle, and treatment instructions being repeated often. They also gave significantly higher ratings to being open and accepting of guidance from staff, and to wanting the system to let them have more control over treatment.

Table 3. Results of hierarchical regression analyses for the prediction of compliance scores from demographic variables and depression

Predictor	Patients' compliance ratings			Clinicians' compliance ratings		
	Step 1	Step 2	Step 2	Step 1	Step 2	Step 2
	Beta	Beta	Partial r^2	Beta	Beta	Partial r^2
Ethnicity				0.40‡	0.40‡	0.17
Duration				-0.06	-0.06	0.01
Knowing type	0.12	-0.03	0.00	0.28†	0.29†	0.10
Family history	-0.27†	-0.16	0.03	-0.08	-0.09	0.01
Complications	-0.19*	-0.12	0.02			
Depression		-0.46‡	0.19		0.05	0.00
R^2	0.14†	0.30‡		0.29‡	0.30‡	
Adjusted R^2	0.11†	0.27‡		0.25‡	0.24‡	
R^2 change	0.14†	0.16‡		0.29‡	0.01	

*p < 0.05; †p < 0.01; ‡p < 0.001.

Self-ratings correlated significantly and negatively with depression scores (r=-0.51, p<0.001), but not with duration of diabetes (r=-0.16) or with age (r=-0.01), ps>0.05. Clinicians' ratings correlated with duration (r=-0.22, p<0.05), but not with depression scores (-0.10) or age (-0.06). Duration did not correlate significantly with depression (0.14).

With the significant correlates and the depression scores, hierarchical regression analyses were performed on the data to assess the independent contributions of predictors of self-ratings and clinicians' ratings of compliance. The significant demographic and diabetes variables were entered first. After accounting for the variance due to them, the depression scores were entered in the second step. In Table 3, the R^2 statistic gives the percentage of variance accounted for by each regression model, and the semi-partial r^2 the proportion of unique variance explained by each component.

In the first regression, knowing one's type of diabetes, family history and complications accounted for 14% of the variance in self-rating scores, F(3,95)=4.94, p<0.01. When depression was incorporated, the total amount of variance accounted for increased significantly by 16% to 30%, F(1,94)=22.02, p<0.001, and the betas for the demographic variables became non-significant. Depression uniquely explained 19% (semi-partial r^2) of the variance in self-ratings. For multiple partial correlations, Cohen[7] has tabled a standardised measure of association (f) which is independent of the original measurement units. An f^2 of 0.15 is considered a medium effect size, and 0.35 a large effect size. For the second regression, an R^2 change of 0.16 yields f^2 of 0.19. Hence, after controlling for demographic and diabetes variables, the singular effect of depression on self-ratings was in the medium range. The statistical power to detect significance at the 0.05 level was at least 80% for medium effect sizes.

For clinicians' ratings, ethnicity (Maori versus European), duration of illness, knowing one's type of diabetes and family history accounted for 29% of the variance in scores, F(4,69)=3.99, p<0.001. The addition of depression did not significantly account for additional variance in scores, F(1,68)=0.19, p=0.66. The R^2 of 0.29 gives f^2 of 0.41, a large effect size, with ethnicity (17%) and knowing one's type of diabetes (10%) accounting for significant proportions of variance in scores.

Discussion

Even though a convenient sample was obtained for this study, it was similar (for sex, age, and ethnicity) to the diabetes population serviced by Northland Health Limited over a comparable 4-month period 1 year later. There were more Maori than Europeans in the sample, reflecting the prevalence of diabetes among Maori in the general population.[1]

Diabetes-related factors significantly associated with low ratings of compliance were duration of the illness; family history of diabetes; patients not knowing their type of diabetes; and severe complications such as cardiovascular disease, kidney disease, blindness, and amputations. Consistent with overall findings from other studies using self-report adherence or metabolic control measures,[5,8,9] demographic factors did not account for differences in compliance or depression.

Because blood tests for the present sample were taken on different days and at places other than at the retinopathic clinic, the accuracy of the compliance measures was not validated against objective measures. Both objective measures and self-report questionnaires have methodological problems.[10] For example, with electronic monitoring and blood tests, the patients are forewarned, so they might alter their adherence behaviour. Objective tests also do not distinguish between intentional and non-intentional compliance.[9] Self-report responses are subject to faulty recall and may reflect a need to please clinicians. However, they can provide a convenient, unexpected check on patients' compliance, incorporate perceptions of compliance behaviour, and be tailored to the situation for the population studied,[11] as in the present investigation.

The first three hypotheses were supported. Patients' ratings of compliance with treatment were higher than the corresponding clinicians' ratings. However, they might have overrated their compliance because they answered according to what they believed was expected of them.[5] The correlation coefficients between the pairs of items revealed that there was no significant agreement between patients and staff on any of the behavioural, attitudinal and lifestyle components of compliance. Patients were more concerned about treatment inconveniencing their lifestyle than the clinicians were. As in the South Auckland household survey of diabetics,[12] the disadvantages of adherence to medication and diet outweighing the benefits of better health predominated in the perceptions of the Northland patients.

For the second hypothesis, the clinicians' ratings were similar for both European and Maori who strongly identified with Maori culture, but lower for Maori who identified weakly. This result confirms the finding from qualitative research that non-Maori doctors attribute Maori compliance with any treatment to identification and acculturation.[13] However, the identification might be a reflection of the clinicians' beliefs rather than differences of any biological or cultural importance.

For the third hypothesis, depression was higher than in the New Zealand normative sample,[6] consistent with findings from large scale studies in the United States[14] and other countries where clinical depression is twice as common among diabetics as non-diabetic comparison groups.[15] Depression is clearly a risk factor for the development of the severity of diabetes' complications, functional disabilities, and early mortality.[14,16] Those especially at risk for depression are diabetes patients who are

Type 1; do not know their type; have a family history of diabetes; have severe complications; or have other illnesses.

For the fourth hypothesis, after controlling for relevant demographic variables and self-reported diabetic history, depression uniquely accounted for variations in patients' ratings of compliance, but not for variations in clinicians' ratings of compliance. Compliance decreased with increasing depression, but this relationship was not found with the clinicians' ratings. It is possible that the clinicians were not aware of underlying symptoms of depression among their patients. Conversely, compliance self-ratings increased with decreasing numbers of symptoms of depression.

The use of self-reports measuring both compliance and depression might have inflated the strength of the relationships because of common method variance and the patients' attempts to be consistent across the questionnaire. Also, because the current findings were based on a cross-sectional study, the directions of any association between compliance and depression need to be tested in longitudinal and prospective designs with larger representative samples of at-risk groups across New Zealand. Depression might lead to less compliance, but not complying for other reasons such as interference with lifestyle might result in depression.[4]

Other variables not included in the present study also need to be incorporated into the designs, such as patients' and clinicians' beliefs,[8] and the effects of social support. For example, beliefs that diabetes is a cyclical illness and caused by poor medical care in the past has been correlated with less adherence to diet and medication among New Zealand European and Tongan diabetics.[17] In contrast, diabetics' perception that they have control over their diabetes has been related to seeking treatment and engaging in healthcare behaviours.[18]

The patients in the present sample wanted more control over their treatment, yet according to the clinicians, they were less diligent in following the treatment plans. Trying to improve patients' knowledge about diabetes (so that they know the seriousness of their condition and the treatment regimens) through education is likely to have little effect on their compliance with treatment.[19] However, strategies which include the following have been effective for increasing empowerment [20] and hence compliance among adolescent and adult diabetics:

- Establishing Maori case management clinics;[21]
- Developing positive patient-clinician relationships and social supports;[22,23]
- Negotiating contracts for treatment options and regular contact;[24,25]
- Training in problem solving, social skills and controlling negative thought patterns;[26] and
- Breaking the cycle of depression and poor compliance through cognitive-behavioural therapy[27] and by routinely monitoring and discussing psychological wellbeing;[28]

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Acknowledgements: This report was based on a thesis by the second author in partial fulfilment of the requirements for the degree of Master of Arts in Psychology at Massey University. The authors are grateful to the Northland Health patients and clinicians who made the study possible.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

A newly recognised cause of vertigo: horizontal canal variant of benign positional vertigo

Jeremy Hornibrook

Abstract

Aims To describe the presentation, causes, treatment, and outcomes of patients presenting with the newly recognised horizontal canal variant of benign positional vertigo (BPV); and to emphasise the importance of performing a positional test on all patients being assessed for vertigo.

Methods The records on 400 patients presenting with BPV were analysed. Two detailed patient histories from 2004 are included.

Results Forty-nine patients (12%) had horizontal canal BPV. The median presentation age was 59 years. In 17 patients, it presented *de novo* during repositioning treatment for 'classical' posterior canal BPV. Repositioning is in the horizontal plane, with the direction depending on whether the mechanism is canalithiasis or cupulolithiasis.

Conclusions Horizontal canal BPV explains nearly all variations on 'classical' (posterior canal) BPV. It accounts for at least 10% of BPV and has been frequently misdiagnosed. Repositioning is usually curative if the symptomatic ear is correctly identified.

Dizziness and vertigo may account for 10% of general practice consultations and for up to 20% of referrals to otolaryngologists and neurologists.[1] Positional vertigo is the most common cause of vertigo. One of the earliest references to it may have been in Shakespeare's play 'Romeo and Juliet': Act 1, Scene II. Bevolio: 'Tut man, one fire burns out another's burning. One pain is lessen'd by another's anguish; turn giddy, and be holp by backwards turning....'.[2]

In 1952, Dix and Hallpike[3] described the 'typical' features of the condition they named benign paroxysmal positional vertigo, now abbreviated to benign positional (or positioning) vertigo (BPV). The only symptom is vertigo induced by a change in head position, particularly turning in bed, but also getting in and out of bed, looking up, and stooping.

Diagnosis is by the Hallpike positional test. When the symptomatic ear is undermost, after a brief delay the patient experiences vertigo and the examiner observes brisk torsional nystagmus whose fast phase is towards the affected ear. While the nystagmus is 'geotropic' it is anatomically upwards. When the patient sits up there may be brief vertigo with the nystagmus reversing to torsional down-beating.

Dix and Hallpike studied the post mortem ear histology on one BPV subject. The utricular macula had lost its otoconia, and they believed that an abnormal macula was the cause of the condition. In the 1960s, at the Harvard Temporal Bone Laboratory, Schucknecht examined 391 temporal bones from 245 patients and found basophilic structures attached to the semicircular canal receptors, including the horizontal canal

receptors.[4,5] In three subjects there was a well documented history of benign paroxysmal positional vertigo. Schuknecht assumed they were the remnants of otoconia which had become detached from the utricular macula, and called the mechanism 'cupulolithiasis' (heavy cupula). Two logical treatments were based on that assumption.[6,7] However, it did not explain all the features of 'typical' BPV.

Others[8,9] made models showing a piston-like effect of a loose particle (canalith) better explained the clinical features. John Epley, a solo private-practice otolaryngologist from Portland, Oregon, designed a five-stage repositioning treatment named the canalith repositioning procedure (CRP), which is now widely used.[10]

Posterior canal BPV is now recognised as the most common cause of vertigo in middle-aged and older adults. An episode of vestibular neuritis, a head injury,[11] and a period of bed rest[12] are recognised antecedents. A survey in a home for the elderly discovered that 9% of residents had the condition.[13] It can be caused by and be a cause of falls in the elderly. It is now clear that BPV has been significantly under-diagnosed. Omission of a simple clinical test can result in patients undergoing unnecessary investigations and inappropriate treatments.[14]

Previously, unusual 'non-typical' forms of positionally induced nystagmus were thought to be of central origin, although rarely proved. While performing CRPs, Epley observed in some subjects a sudden change from 'typical' BPV to another with horizontal direction-changing nystagmus and more violent vertigo. He deduced that BPV could be caused by otolith particles in any of the semicircular canals.[15]

In 1985, McClure[16] published the electronystagmographic (ENG) traces of seven subjects who had positional vertigo with horizontal direction-changing nystagmus when lying supine. The fast phase of the nystagmus was towards the undermost ear (geotropic). McClure assumed this was due to a 'viscous plug' in the horizontal canal causing a piston effect on the receptor.

An ampullopetal (towards the vestibule) deflection is known to cause the most intense nystagmus and vertigo (Figure 1). Horizontal canal BPV was then reported by others,[17–20] and the intensity of the vertigo noted. Early attempts at repositioning failed.[18] A 270° 'barbecue' rotation was trialled.[21,22] Epley suggested a 360° rotation away from the symptomatic ear.[15] (Figure 1).

Methods

The author performs a Hallpike positional test on all patients with a current or past history of vertigo. Between 1995 and 2004, 400 patients had BPV. Forty-nine (12%) had the horizontal canal variant (Table 1).

Where the mechanism was canalithiasis, the patient was assisted in performing a 360° (contralateral) rotation away from the symptomatic ear, with the head as flexed as possible. If the nystagmus was ageotropic, a reverse direction rotation may be required. Follow-up was between 2 and 5 weeks to retest, and later by telephone to confirm that there had been no recurrence. In some patients, a combined eye and body video recording was made for teaching materials.

Two illustrative patient histories are described in Figure 2.

Figure 1. Geotropic direction-changing nystagmus implies a moving particle in a horizontal semicirclar canal. If it is the left ear, rotation of the head to the left causes an ampulopetal deflection of the receptor resulting in a brisk horizontal nystagmus to the undermost (left) ear. Rotation of the head towards the opposite ear causes the particle(s) to move towards the open end of the canal and an ampulofugal deflection of the receptor with weaker horizontal nystagmus towards the (right) ear. The logical treatment is a 360° head and body rotation away from symptomatic ear to allow the particle (s) to exit through the open end of the canal.

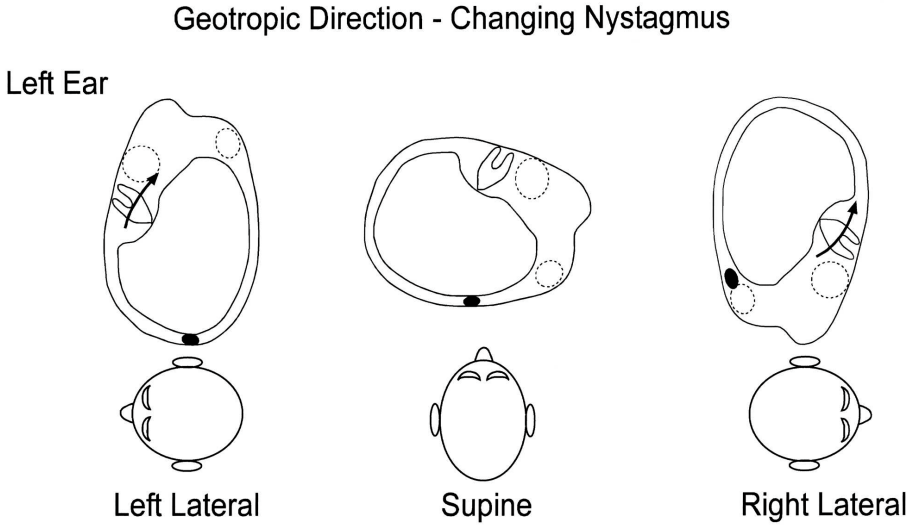

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Figure 2. Two illustrative patient histories from 2004

A 37-year-old female presented complaining of vertigo. She had woken with vertigo and vomiting 7 days previously. Subsequently, vertigo would start every time she lay down or stooped. She would rather be standing or sitting than lying down. While sitting upright, her eyes were observed through Frenzel glasses, and there was no nystagmus. On a Hallpike positional test, there was sudden onset of vertigo, and there was horizontal nystagmus. When lying supine, the nystagmus reversed in direction as her head was turned from right to left. The most intense vertigo and fastest nystagmus occurred with head left (left geotropic nystagmus). She vomited and IM Stemetil was given. The diagnosis was left ear horizontal canal BPV (canalithiasis). She was assisted in performing two 360° 'barbecue' rotations in the horizontal plane and asked to continue this at home by rolling on the floor in a left to right direction. Two days later, the patient reported no vertigo. On lying down, no vertigo or nystagmus occurred. After 5 months, there has been no recurrence. This case illustrates how the diagnosis had been missed by the omission of a positional test.

A 36-year-old female was first seen in 2001 with a 6-year history of recurrent vertigo. She said that with the first episode she awoke with vertigo and cause and 'I couldn't move' for a week. There were no associated ear symptoms. Five months later, it recurred for a week and she was given IM Stemetil. Three similar episodes followed. No abnormalities were found. A MRI scan of posterior fossa was normal. She was requested to present with her next episode, but did not when it occurred in October 2003. Again there were no vestibular abnormalities, including a negative positional test. Insistence on seeing her with symptoms was repeated. She mentioned a past history of migraine. In January 2004 the patient presented with another 'attack.' She said she had had a 'migraine headache' and vertigo for two days. While sitting she had no nystagmus, but a positional test elicited vertigo and direction-changing horizontal nystagmus maximum to the right. The diagnosis was right ear horizontal canal BPV (canalithiasis). It ceased following two 360° 'barbecue' rotations. It has not recurred after 9 months. This case illustrates all the difficulties of reaching an accurate diagnosis in patients with recurrent vertigo. Firstly, the importance of seeing the patient with the 'attack.' Secondly, the interposition of the patient's (or other doctors') theory about the cause. Thirdly, failure to have a positional test when the patient is complaining of vertigo.

Results

The results are presented in Table 1. There were 49 patients with horizontal canal BPV. The age range was 29–87 years (median 59 years). Seventeen patients undergoing repositioning for posterior canal BPV 'converted' to the horizontal canal variant. Twenty-five had primary canalithiasis. Eight had cupulolithiasis. One male had canalithiasis and cupulolithiasis at different times. Seven vomited during their treatment. Nine had a recurrence (1 week–1 year).

Table 1. Data on 49 patients with horizontal canal benign positional vertigo treated between 1995 and 2004. The age range was 29 to 87 years (median 59 years). One male patient had canalithiasis and cupulolithiasis on different occasions

Variable	Conversions	Canalithiasis	Cupulolithiasis
Number	17	25	8
Sex	M 11, F 16	M 10, F 15	M 6, F 2
Onset	Immediate	1 day–years (median 2 weeks)	1 day–7 months (median 3 days)
Ear	R 10, L 7	R 15, L 10	Nystagmus maximum R 5, L 3
Preceded by	Head injury (3) Acute vertigo (2)	Head injury (2) Acute vertigo (2)	Nil
Treatment	Standard barbecue (17)	Standard barbecue (25)	Standard barbecue (3) Reverse barbecue (5)
Vomited	4	1	2
Recurrence	0	8 (1 week–1 year)	1 (8 months)
Follow-up without recurrence	2 months–8 years (median 5.5 years)	2 months–8 years (median 4 years)	2.5–7 years (median 4 years)

Discussion

Since Schucknecht's temporal bone studies in the 1960s, it was widely accepted that the features of 'typical' BPV were generated by the posterior canal receptor. Although

the histology suggested a similar involvement of the horizontal canal receptor, Schucknecht did not predict that a horizontal canal form of BPV could exist. At that time, any variation from 'typical' BPV was assumed to have a central cause, but rarely proved. It is now well established that BPV can be caused by otoconia in any of the semicircular canals. In all large series, posterior canal BPV is the most common, followed by horizontal canal,[19,23] and superior canal variants.[24]

Horizontal canal BPV is likely to be discovered when a patient is undergoing a Hallpike positional test. Typically there is intense vertigo and there is horizontal nystagmus, usually towards the undermost ear (geotropic). The patient is asked to lie supine, and the head is turned from side to side. An ampullopetal (towards the vestibule) movement of the horizontal canal receptor will cause the most intense nystagmus and vertigo, identifying the symptomatic ear.

The logical treatment is a head and body rotation away from the symptomatic ear (Figure 1). It is likely that subjects with horizontal canal BPV inadvertently treat themselves by rolling over in bed, as long as it is in the desirable direction, but aggravate the condition by rolling in the 'wrong' direction. Occasionally the nystagmus is horizontal but away from the undermost ear (ageotropic). This implies a different mechanism or cupulolithiasis where the particle (s) could be lying on either side of the horizontal canal receptor in either ear.[25] It may be impossible to determine the symptomatic ear. If a standard 'barbecue' rotation makes the vertigo worse, a reverse rotation is usually effective.

Horizontal canal BPV is still a newly recognised cause of vertigo, and often missed. Previously its symptoms were attributed to central pathology and to other otological causes of vertigo. It is easily diagnosed and treated if a positional test is performed on every vertiginous patient who does not have nystagmus in the sitting position. If the diagnosis is BPV (from any canal), the subject *must* experience vertigo on the positional test. If not, a central cause should be sought.[26]

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Clinical indications for the investigation of porphyria: case examples and evolving laboratory approaches to its diagnosis in New Zealand

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Abstract

Patients with porphyria present in a diverse and unusual variety of ways and most clinicians will see only a few cases, if any, during their professional lives. Porphyria may present (1) with acute symptoms, which may be abdominal pain, neurological or psychiatric; (2) with skin rash or photosensitivity; or (3) with a putative family history. Screening for latent porphyria has been greatly facilitated by fluorescence emission scanning of plasma and by mutational analysis. Our reference laboratory has recently diagnosed several cases of the less common types of porphyria, which we postulate is due to the availability of these methods and to the changing population of New Zealand. Accurate screening and diagnosis of porphyria is important, as an acute porphyric attack is life-threatening and preventable. Retrospective diagnosis may be difficult.

The porphyrias are a diverse group of metabolic diseases affecting haem synthesis. A traditional classification is acute (acute intermittent porphyria [AIP], variegate porphyria [VP] and hereditary coproporphyria [HC]) versus chronic (porphyria cutanea tarda [PCT] and erythropoietic protoporphyria [EPP]). This is of limited practical value, in our opinion, and investigation is best guided by which of three presenting clinical scenarios is present.

Possible clinical indications for the investigation of porphyria:

- Patients presenting with acute symptoms, which may be abdominal pain, neurological, or psychiatric. Some patients presenting with acute abdominal pain may require a general anaesthetic, which could potentially include drugs that exacerbate acute porphyria.
- Patients presenting with vesicles, bullae, hyperpigmentation, milia, hypertrichosis, or increased skin fragility on sun-exposed skin. This photosensitivity is probably the most common indication for investigation.
- Screening of family members where there is a positive family history—testing for latent porphyria.

An acute attack begins with minor behavioural changes such as anxiety, restlessness, and insomnia—and proceeds rapidly to symptoms of autonomic and sensomotoric neuropathy. Abdominal pain (major presenting feature), usually followed by vomiting and constipation is common and severe, mimicking acute abdominal crisis.

Pain in the back or in the extremities is frequently present. Hypertension and tachycardia, which are signs of increased sympathetic activity and are associated with the activity of the disease, should be followed up together with visual assessment

scaling of pain. Pain declines within a week, but if an additional precipitating factor is administered, the progressing sensorimotor neuropathy manifests and can proceed to respiratory paralysis. Central nervous system (CNS) impairment can manifest with convulsions and confusion. The combination of polyneuropathy and focal CNS involvement is unusual for other polyneuropathies and should alert a doctor to look for porphyrias.[1,2]

Random urine porphobilinogen (PBG) is the most important test for the diagnosis or exclusion of acute porphyria. If urine PBG is negative at the time of acute symptoms, then that excludes porphyria as cause of those symptoms. All acute hospitals should have access to urine PBG screening,[3] and a positive PBG test should be followed by further tests on urine, faeces, and blood to differentiate the type of acute porphyria.

For patients presenting with a skin rash or photosensitivity, it is preferable to send (protected from light) a complete set of samples of urine, faeces, and blood.

The commonest porphyria (90% of all cases) is PCT where the diagnosis is made on positive faecal and urine porphyrins, with characteristic patterns on high performance liquid chromatography (HPLC) analysis.

Screening of family members for latent porphyria, where there is a positive family history, is the most difficult clinical scenario to manage. The approach hinges on validating the diagnosis in the index case, which can be difficult when that case may be deceased or living overseas.

A valid diagnosis in an index case enables the diagnostic tests to be focused to those pertinent to the porphyria in question. In the case of AIP, for example, tests for latent disease include measurement of PBG deaminase activity (see below) or mutational analysis.

For VP, fluorescence emission spectrophotometry is gaining acceptance,[4] although it has limited sensitivity. Mutational analysis is the definitive way to confirm or refute a diagnosis of latent porphyria, but depends on the identification of a mutation in the index case. This approach can also identify asymptomatic carriers who may never have clinical manifestations of the disease.

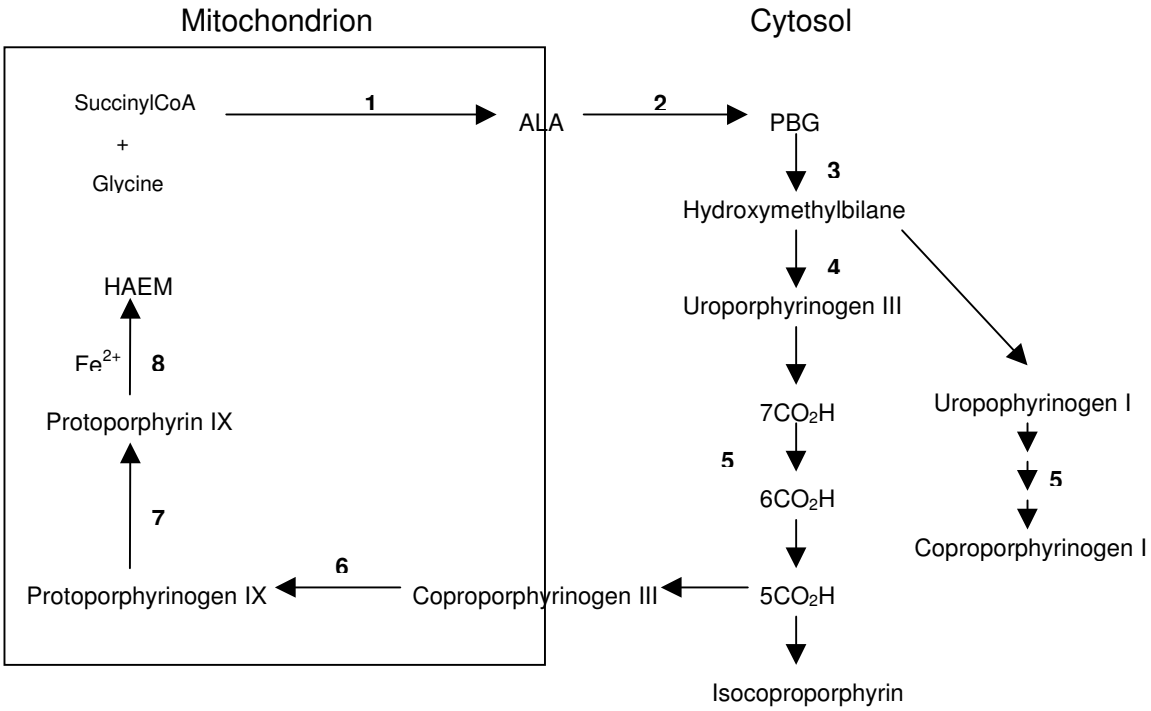
We have witnessed a significant increase in the number of less common types of porphyria. This may, in part be due to the changing population of New Zealand, but may also be attributed to evolving methods of investigation, particularly the introduction of plasma fluorescence scanning.[4]

This review provides background information on the different clinical types of porphyria, some explanation of the diagnostic tests available, and finally some case examples encountered through our practice as a reference laboratory.

Biosynthesis and metabolism of haem precursors

Each type of porphyria results from decreased activity of one of the enzymes of haem biosynthesis as outlined in Figure 1. The porphyrinogen intermediates are unstable and readily autoxidise to porphyrins within tissue and after excretion. In normal circumstances most of the α-aminolaevulinate (ALA) produced is converted to haem, with only small amounts of intermediates being lost from the pathway and excreted either unchanged or after oxidation to porphyrins.

Figure 1. The pathway of haem biosynthesis. The reactions are catalysed by α-aminolaevulinate (ALA) synthase (1), porphobilinogen (PBG) synthase (2), PBG-deaminase (3), uroporphyrinogen-III synthase (4), uroporphyrinogen decarboxylase (5), coproporphyrinogen oxidase (6), protoporphyrinogen oxidase (7), and ferrochelatase (8)



The route of excretion is largely determined by the solubility of the compound. Generally, intermediates from ALA to coproporphyrin are water soluble, and are excreted in urine, while those from coproporphyrin to protophyrin are lipophilic and excreted in bile (faeces). Decreased enzyme activity (at any one point) results in a build-up of the preceding intermediates, while intracellular accumulation and subsequent excretion of the substrate produces a characteristic pattern of plasma, erythrocyte, and excretory abnormalities for each enzyme deficiency.

The different types of porphyrias (in order of enzyme defect in haem biosynthesis) are:

Acute intermittent porphyria (AIP) or Swedish porphyria

- Inherited in an autosomal dominant fashion.
- 1–2 carriers per 100,000 people, of which >80% do not develop symptoms. AIP is latent before puberty; symptoms are more frequent in females than males. Hormonal, drug (lists of safe and unsafe drugs are available)[5] and nutritional factors may aggravate the disorder.
- There are latent and acute stages.

- Symptoms are neurological without skin photosensitivity. Acute abdominal pain occurs in 85–95% of cases. Symptoms may include: abdominal pain, nausea, vomiting, diarrhoea, constipation, ileus, dysuria, muscle hypotonia, respiratory insufficiency, sensory neuropathy, seizures.
- As for all acute porphyrias, urine ALA and PBG are increased in the acute state, slightly elevated or normal in the latent state. Urine porphyrins are often raised, while faecal and erythrocyte porphyrins are normal.

Congenital erythropoietic porphyria (CEP) or Gunther disease

- Inherited in an autosomal recessive fashion.
- Very rare with only 130 cases reported prior to 1997.
- The age of onset and clinical severity is highly variable, in most cases photosensitivity develops soon after birth. Blistering and scarring of exposed areas may lead to mutilating deformity. Symptoms may include: bullae, crusts, scar formation, sclerodermoid, hyper- and hypopigmentation, hypertrichosis, erythrodontia, haemolytic anaemia, splenomegaly.
- Urine ALA/PBG are not raised; urinary, faecal, and erythrocyte porphyrins are raised and are predominantly of the I isomeric form. Protection of the skin from sunlight is essential. Repeated blood transfusions can suppress erythropoiesis and may decrease porphyrin production, thus reducing photosensitivity.

Porphyria cutanea tarda (PCT)

- Two clinically indistinguishable types: Type I is sporadic and occurs as a result of inhibited or inactivated uroporphyrinogen decarboxylase (UROD) activity only in the liver due to: excessive hepatic iron, ethanol use, hepatitis C, HIV infection, or oestrogen administration. Type II is less common and inherited in an autosomal dominant fashion and results in systemic UROD deficiency.
- PCT type I is the most common form of porphyria and is estimated to occur at 1 in 25,000 people.
- Chronic blistering lesions (bullae) develop on sun-exposed areas of skin. Symptoms may include: skin fragility, crusts, sclerodermoid, hyper- and hypopigmentation, hypertrichosis.
- There are no neurological features.
- Urine ALA/PBG are not raised; urine and faecal porphyrins are raised, with a typical pattern on High Pressure Liquid Chromatography profiling.

Hereditary coproporphyria (HCP)

- Inherited in an autosomal dominant fashion.
- Prevalence is less than 1 in 250,000 people.
- HCP symptoms may include those of AIP and PCT. HCP is precipitated by the same factors as AIP.

- As for other acute porphyrias, urine ALA and PBG are increased in the acute state and may be slightly elevated or normal in the latent state. The most predominant finding is the increased urinary and faecal coproporphyrin III, which may be slightly raised in the latent state.

Variegate porphyria (VP) or South African type

- Inherited in an autosomal dominant fashion.
- Especially common in South African whites (approximately 3 per 1000) and can be traced to a common founding couple who emigrated from Holland in the seventeenth century. As many as 20,000 South Africans may carry this trait due to a founder effect.
- The disease is termed variegate because it can present with a variety of symptoms, these may be neurological and or photosensitivity, and are the same as those seen in AIP and PCT. Provoking factors are similar to those of AIP.
- As for all the acute porphyrias, ALA and PBG are increased in the acute presentation and may be slightly elevated or normal in the latent state. Urine and faecal porphyrins are increased, mainly as coproprophyrin III, with a large amount of protoporhyrin IX in the faeces. Plasma has a specific fluorescence peak at 626 nm.

Erythropoietic protoporphyria (EPP) or protoporphyria

- Inherited in an autosomal dominant fashion.
- Prevalence is 1 in 250,000 people, with no racial predilection.[6]
- The major clinical features of EPP are cutaneous photosensitivity, which begins in childhood, with sun-exposed areas developing itching and swelling within minutes of sun exposure, often mimicking very severe sunburn. Symptoms may include: burning sensation, oedema, erythema, itching, scarring, vesicles.
- Protoporphyrin accumulates primarily in erythroid cells in the bone marrow, and appears in excess in plasma, circulating erythrocytes, bile, and faeces. Plasma has a specific fluorescence peak at 632 nm.
- Zinc oxide cream and avoidance of sunlight are essential.[7]

Laboratory investigations

Specimen requirements

Specimens should be protected from light and received by the laboratory within 24 hours.

Urine—Fresh random urine is preferred to 24-hour collections, and is ideally collected during an acute episode if acute porphyria is suspected.

Faeces—A random 10 g sample of faeces is required.

Blood—Whole blood (EDTA or heparinised).

Laboratory tests

Urine porphobilinogen (PBG) screen[8]—PBG is raised in patients with acute attacks of hepatic porphyrias AIP, VP, and HCP. It is not raised in PCT or the erythropoietic porphyrias. Absence of PBG in the urine collected when a patient has abdominal pain excludes porphyria as a cause of abdominal pain. PBG, however, may disappear from the urine between acute attacks (latent phase).

All patients undergoing porphyria testing presenting with abdominal pain should also be questioned with regard to their previous (and family) history of skin and neurological symptoms.

Urine total porphyrins—Concentrations will be increased in patients with current symptoms of PCT, VP, HCP, AIP, and CEP.[9]

Faecal total porphyrins—Faecal porphyrin concentrations are increased in hepatic porphyrias (except AIP), EPP, gastrointestinal bleeding, and very high meat diets.[9]

High pressure liquid chromatography (HPLC) profiling—When there is a elevated excretion in urine or faeces, HPLC separation of the isoforms must be undertaken to differentiate the type of porphyria. The specific pattern of the different porphyrins (in both the urine and faeces) is diagnostic of the type of porphyria, and it is not usually necessary to quantitate the individual porphyrins.

Blood porphyrins

Plasma

The plasma of patients with overt and latent VP contains a porphyrin protein complex (thought to be a porphyrin molecule attached to a short amino acid chain) that has a specific fluorescence emission peak for the excitation wavelength of 405 nm.[10] An emission peak at 626 supports VP and one at 632 supports EPP.[11] An emission peak at 619 nm may occur with AIP or PCT, although it is frequently non-specific.[3]

Whole blood

Red blood cell porphyrin levels are raised in EPP, CEP, HEP, VP, lead poisoning, iron deficiency, and anaemia. In EPP and CEP, there is an excess production and accumulation of free protoporphyrin. Lead poisoning, which can mimic a porphyria, may also present with abdominal pain and increased urine porphyrins. The ferrochelatase activity is inhibited and results in the formation of zinc protoporphyrin. This is also produced in iron deficiency anaemia as a result of the lack of available iron.

Red cell enzyme levels

The measurement of the enzymes of the haem biosynthesis are rarely essential for the accurate diagnosis of overt porphyria. Enzyme assays, however, are useful for the detection of latent porphyria in family members of an index case, especially where the family members have either no symptoms, or are pre-puberty and often thus biochemically normal. In particular, it is useful to measure PBG-deaminase for the diagnosis of AIP (90% of AIP patients have mutations that decrease red cell PBG-

deaminase activity, with some overlap of enzyme activity between the normal and affected population).

Mutation analysis

Similar to the blood enzyme studies, mutational analysis can be used to confirm clinical/biochemical diagnosis or for family studies. For VP, there is the well studied *p.R59W* founder mutation in South Africa. There are also founder mutations in Finish (*p.R152C*) and Chilean (*c.1241_1245del*) populations, as well as 60 novel mutations identified in a recent study of 104 unrelated patients from Western Europe.[12] For suspected latent AIP and VP, mutation analysis offers a means of making a definitive diagnosis in families of an index case, where the gene defect is known.[13,14] Mutation analysis for AIP and VP is now readily available through our laboratory.

Clinical and biochemical studies of specific porphyrias

Variegate porphyria

Case 1 (atypical presentation)—A South Africa-born 52-year-old female presented with severe migraine to an emergency department, she was admitted for observation overnight. The next day her migraine had improved and she was discharged into her GP's care, no diagnosis of cause was made. Her GP, also South African, considered VP as a possible diagnosis. High levels of urinary PBG, ALA, and total porphyrins along with a specific plasma fluorescence peak indicated acute VP, and this was confirmed by detection of the *p.R59W* mutation. Follow-up found that this acute episode occurred several days post-anaesthetic for dental surgery. Her mother and two sisters had been previously diagnosed in South Africa. Mutation analysis was performed for her two symptom-free daughters (aged 17 and 20). and one daughter was found to be heterozygous for the *p.R59W* mutation. Neither daughter had the characteristic plasma fluorescence peak.

Case 2 (typical presentation)—A 48-year-old male presented with abdominal pain and constipation of 7 days' duration. The pain was colicky, central, and associated with anorexia, bloating, and vomiting. One month earlier, he had been started on phenytoin following his first generalised seizure, and after this developed insomnia, anxiety and urethral discharge. Infection, vasculitis, and thromboembolism were excluded. The patient's wife noticed that the urine in the 24-hour collection bottle darkened on standing. Laboratory tests identified raised urinary PBG and total porphyrins, raised faecal porphyrins with a low CI:CIII ratio on HPLC, and a specific plasma fluorescence peak—thus indicating acute VP. Family studies were offered to his three symptom-free sons (aged 14, 19, and 21). The father and eldest son were found to be heterozygous for the *c.461_483del* mutation of the PPOX gene. The eldest son also had raised total faecal porphyrins and an abnormal plasma fluorescence peak.[15]

Case 3 (atypical presentation)—A 25-year-old female of Pacific Island descent was referred to a dermatologist with blistering lesions on sun exposed skin. Raised urinary PBG, ALA, and total porphyrins; raised faecal porphyrins; and a specific fluorescence peak indicated acute VP. Family studies were offered to the extended family, some of whom had been assigned a diagnosis of PCT (probably erroneously without biochemical porphyrin studies), although this offer was not accepted.

Discussion—While VP is a relatively common type of porphyria in South Africa, there has been a significant recent increase in cases in our local experience. This can, in part, be attributed to the significant increase in the New Zealand population of South African immigrants; 14,727 new immigrants between 1996 and 2001, a 130% increase, (Census 2001 statistics). Other *non-p.R59W* cases have also been diagnosed; this has mainly been through the use of the plasma scanning technique on all samples with slightly raised erythrocyte porphyrins; where in some cases the more traditional screening tests have been normal.

Some groups have found this plasma scanning technique to be 100% specific, with a sensitivity of 86% (95% confidence interval: 71-98%) for asymptomatic adults with VP[11]. In contrast, the sensitivity of faecal porphyrin analysis as a test for adult gene carriers was 36%. Plasma porphyrin analysis has been suggested as an adequate substitute for faecal porphyrin measurement as a front line test, and is particularly useful for distinguishing VP from other bullous porphyrias, of which PCT is by far the most common.[16] Neither test is as sensitive in children, where if the mutation can be detected in an index case, DNA analysis remains the preferred diagnostic tool.[4]

Erythropoietic protoporphyria

Case 1 (typical presentation)—A 5-year-old male presented with sudden severe photosensitivity on sun-exposed skin. Symptoms included itching, painful erythema, and swelling. Urinary PBG and total porphyrins were normal; erythrocyte protoporphyrins were raised at 81 μg/100 mL; a specific plasma fluorescence peak at 632 nm indicated acute EPP. Two siblings were diagnosed with EPP on the grounds of their raised erythrocyte porphyrins, plasma fluorescence peak, and reduced ferrochelatase activity. The mother did not have a history of photosensitivity, which was consistent with her normal red cell and plasma porphyrins (despite a very low ferrochelatase activity).

It has been reported that clinical expression of EPP requires a defective ferrochelatase (FECH) gene on one allele and a common low expression FECH variant on the other. Thus they are compound heterozygotes for a mutation and a polymorphism.[17] Her husband was found to carry the commonly occurring low-expression FECH polymorphism, thus explaining his wife's lack of symptoms, and the clinical expression of EPP in their child.

Case 2 (atypical presentation)—An 80-year-old female with history of photosensitivity and ulceration; according to he physician she had 'always had porphyria.' Normal urinary PBG and total porphyrins, a specific plasma fluorescence peak, and raised erythrocyte porphyrins indicated EPP. Family studies were offered to the extended family, but were not accepted.

Discussion—In both these cases, traditional (urine and faecal porphyrins) screening strategies would have missed the diagnosis of EPP. While erythrocyte porphyrins are raised, this test is not specific, as they can be raised in conditions such as; high blood lead levels, chronic infection, malignancy, or iron deficiency anaemia.[18] However, by following up raised levels with plasma fluorescence, a clear diagnosis can be achieved. This characteristic fluorescent peak at 632±2 nm, has been found to be particularly useful for the diagnosis and screening for this type of porphyria. Plasma porphyrins may be increased in conditions where porphyrin excretion is impaired,

such as renal failure and cholestasis, but the specific peak at 632±2 nm has only been reported in association with EPP.[9,16] Furthermore, the identification of the two polymorphic variant sequences associated with this low expression allele now enables improved predictive counselling for couples where one partner has EPP.[19]

Conclusion

The porphyrias are relatively uncommon disorders that require specialised investigation for precise diagnosis. Every effort must be made to establish an accurate diagnosis during presenting illness—as retrospective diagnosis may be difficult. All laboratories should be able to identify acute porphyria, with ready access to urine PBG testing. Confirmation of the type of porphyria will require more specialised assays including HPLC profiling, plasma fluorescence scanning, and mutational analysis. For definitive investigation and diagnosis, a full set of blood, urine, and faecal samples should be sent to a reference laboratory.

Although PCT is the most common type of porphyria in New Zealand, the other less common types (especially AIP, VP, and EPP) must be considered when the appropriate clinical symptoms are present. Additionally, with the recent increased immigration rate from a country with a high known prevalence of VP (South Africa), there is likely to be an increased rate of presentation here in New Zealand.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Clinical considerations of antidepressant prescribing for older patients

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Abstract

Depression may affect up to 50% of people in older age groups. The increasing numbers of patients over 65 years of age may lead to higher prevalence rates of this debilitating condition. The diagnosis of depression in older patients is complex because symptoms may present as somatic concerns or anxiety. Comorbid conditions need to be investigated, evaluated and, if necessary, treated. Prescribing antidepressants to older patients requires consideration of the pharmacokinetic parameters of the medications used in this population, as these affect drug selection, duration of treatment, and suitability when comorbidity is present. This paper provides a review of current literature and makes recommendations for the pharmacological management of depression in older patients within the New Zealand context.

Depression is the most common psychiatric illness suffered by older patients, and is considered a leading cause of disability worldwide.[1] Considering that the number of people over the age of 65 has increased significantly in the past decade,[2,3] a high prevalence of depression is expected, particularly in hospitalised older patients and those in long-term care facilities.[4]

Depression in later life can be serious, and has been associated with suicidal ideation and attempts. Indeed, it is proposed that suicides and suicide attempts among the elderly will increase as the numbers and proportion of older people in the population rises.

Depression may affect up to 50% of people in older age groups[5] and those with less social support. Older people comprise 12% of the New Zealand population and use approximately 48% of prescription medicines dispensed each year.[6] This paper reviews some of the important considerations and pharmacotherapy treatment options for older patients with depression.

Diagnosis of depression in older people

Studies suggest that the diagnosis of depression in older people is poor, and that management is less than optimal.[7] Depression in older patients is likely to be comorbid with other physical and psychiatric disorders which makes the diagnosis more complex. Symptoms may be vague, presenting as somatic concerns rather than depressive symptoms. It is important to remember that not all older people are 'sad' and sadness is neither a necessary nor a sufficient criterion for depression.

Comorbid conditions—Depression may manifest as a psychosomatic illness, a pseudo-dementia, or a late-onset anxiety disorder.[8] Subjective memory complaints may require exclusion of dementia which can initially present as an apparent depressive illness. Depression is also associated with Parkinson's disease and chronic

pain, two conditions commonly found among older patients. Alcohol misuse can mask depression and may be 'hidden' and undiagnosed among the elderly;[9] careful questioning may be required to elicit information on alcohol misuse.

Types of depression

Assessment includes the type, severity, and duration of the depression, and any coexisting conditions. Symptoms of major depression (according to DSM-IV criteria) are listed in Table 1.

Table 1. DSM-IV diagnostic criteria for major depression

Five or more of the following symptoms must be present for at least two consecutive weeks:
• Depressed mood most of the day;
• Decreased interest and pleasure in nearly all activities;
• Changes in appetite or weight loss without dieting;
• Disruptions in sleep patterns nearly every day;
• Psychomotor retardation or agitation;
• Feelings of worthlessness or inappropriate guilt;
• Diminished ability to concentrate; and
• Recurrent thoughts of death or suicide.

Adapted from DSM-IV (Diagnostic and Statistical Manual of Mental Disorders DSM-IV. 4th ed, Text revision. Washington DC: American Psychiatric Association; 2000).

There is more functional impairment associated with 'moderate' than 'mild' episodes of depression. Moderate to severe depression is as disabling as congestive heart failure.[5] In 'severe' episodes there is marked interference in social and occupational functioning; referral to a specialist (geriatric psychiatrist) may be required.

Other types of depression for which consultation with a specialist is recommended include:

- Major depression with psychotic features which may present with hallucinations and/or delusions, as there may be other reasons for the psychotic symptoms.
- Atypical depression, characterised by mood reactivity.
- Depression with melancholia. An essential feature is loss of pleasure in almost all activities, but depression with melancholia can also be accompanied by loss of reactivity with marked neurophysiological features of anorexia and poor hydration, diurnal variation, and early morning wakening. This severe form of depression can be life-threatening in the elderly, and referral is necessary. Electroconvulsive therapy (ECT) may be required.
- Treatment resistant depression. This may require augmentation therapy which is discussed in more detail later in this article.

'Dysthymia' is marked by a chronically depressed mood (of at least 2 years' duration) that is not severe enough to meet the diagnostic criteria for major depression. The chronic nature of dysthymia imparts significant functional impairment. 'Seasonal affective disorder' has a characteristic pattern of depressive episodes and should be considered during the winter months in New Zealand.

Practical prescribing issues for older patients

It is reported that less than 30% of elderly depressed patients receive adequate antidepressant treatment.[10] Non-adherence to treatment, sub-therapeutic dosing, ineffective choice of agent, and inadequate duration of treatment[11,12] contribute to poor outcomes of antidepressant therapy.

Non-adherence may be intentional and can be influenced by cost factors. For example, patients are required to pay two co-payment fees if fluoxetine 10 mg tablets are prescribed to initiate treatment for a few days, followed by fluoxetine 20 mg capsules for maintenance treatment.

Older patients may experience difficulty in swallowing, and consideration of the size and shape of dosage forms is important. Patients who suffer from arthritis may struggle to open medicine containers, especially those with 'childproof safety caps'. Some antidepressants are dispensed on a three-monthly basis and patients may 'decant' their medication into smaller, improperly-labelled containers. It is important to discuss these issues with the patient to ensure adequate patient education and to allay fears and promote adherence.

Pharmacological issues when treating the elderly

Changes in drug absorption of antidepressants tend to be clinically inconsequential despite an age-related decrease in small-bowel surface area and an increase in gastric pH. Consideration of the drug's pharmacokinetic characteristics in ageing individuals is more important, and age-related changes in drug distribution, metabolism, and elimination of individual antidepressants should be considered.

Side effects

The choice of drug must include consideration of the side effects. Increased receptor activity, decreased lean body weight, reduced renal function, and decreased protein binding result in higher than expected blood levels of 'free' drug. This results in more pronounced side effects. As the side-effect profile is a major determinant in the choice of antidepressants (particularly in the older patient), a review of important side effects in this age group with each class of medication is presented below.

Selective serotonin reuptake inhibitors (SSRIs)—Cases of clinically significant hyponatremia in older patients treated with the SSRIs have been reported.[13–17] The incidence in one study was 12% of patients. Although severe hyponatremia can be fatal, symptoms associated with mild to moderate hyponatremia are nonspecific (e.g. anorexia, nausea, fatigue, lethargy, and confusion). Without proper monitoring of sodium levels in elderly patients using SSRIs, these symptoms may be dismissed as age-related weakness or malaise, thereby putting these patients at increased risk of hyponatremia and its consequences (e.g, seizures, coma, and even death).

If hyponatremia develops and continuation of SSRI therapy is desired, long-term restriction of daily fluid intake (e.g. 800–1000 mL) has been suggested, although patient compliance is often poor.[18] Failure to respond to fluid restriction warrants discontinuation of the causative medication until sodium levels normalise.

The clinical presentation of SSRI-induced hyponatremia may resemble the syndrome of inappropriate antidiuretic hormone (SIADH) secretion.[19] Hyponatremia can also

occur with tricyclic antidepressants (TCAs) and other medications (it is common with carbamazepine). Thus, monitoring of serum electrolytes may be required with antidepressants, as low sodium is also associated with depressed mood.

SSRIs frequently cause agitation, anxiety, and insomnia, and have been associated with restless leg syndrome (RLS).[20] These factors need to be considered especially in the elderly who may already be experiencing disturbed sleep patterns.

Sexual dysfunction is associated with loss of libido and delayed ejaculation in men, and orgasmic dysfunction in women.[20] It is more apparent with SSRIs than TCAs.

For older patients, the choice of an antidepressant that is not generally associated with weight change may be beneficial. Paroxetine caused weight gain in patients of various ages after long-term treatment, while fluoxetine showed appetite-suppressing effects in the short-term, producing a greater degree of weight-loss than paroxetine or citalopram.[20]

Anticholinergic side effects such as dry mouth, blurred vision, constipation, and difficulty with micturition may be more apparent in elderly patients taking paroxetine than other SSRIs.[20] This is because paroxetine has an *in vitro* affinity for the muscarinic cholinergic receptor.

Current exposure to SSRIs was found to be associated with upper gastrointestinal bleeding at a rate of approximately one case per 8000 prescriptions. Concurrent use of nonsteroidal anti-inflammatory drugs (NSAIDs) or aspirin greatly increased this risk.[21]

SSRIs with short half-lives (such as paroxetine and citalopram) may be beneficial for patients with multiple comorbidities and complex multidrug regimens. However, an SSRI with a short half-life may cause withdrawal reactions on discontinuation of treatment, and doses must be tapered with care. The half-life of the parent compound and all active metabolites is an important consideration when deciding on a dosing regimen. Paroxetine and fluoxetine have non-linear pharmacokinetics, and dose increases produce greater changes in plasma concentration. Citalopram has linear pharmacokinetics making dose titration easier.

Fluoxetine, which has a half-life of 6 days, has an active metabolite (norfluoxetine) with a half-life of 14 to 21 days. It may take 3 to 4 weeks to achieve steady-state after initiation of therapy or modification of doses.

Tricyclic antidepressants (TCAs)—Symptoms of cardiotoxicity and confusional states are more apparent in the elderly than in younger adults using TCAs. Because of the potential prolongation of QTc interval in the elderly, a baseline ECG is recommended prior to commencing TCA treatment. A repeat ECG once a therapeutic dose is achieved is sound clinical practice. TCA sedative effects are more noticeable in older patients, and the use of alcohol and drugs affecting the central nervous system (CNS) must therefore be more carefully monitored.

Anticholinergic side effects such as dry mouth, tachycardia, blurred vision, urinary retention, constipation, sedation, and delirium may occur more frequently among elderly patients. These side effects appear to be more pronounced with the tertiary amine TCAs, amitryptiline and imipramine. Secondary amines such as nortryptiline and desipramine are the safest TCAs to use in older people.[22] Dry mouth, which contributes to dental decay, can lead to denture problems and dysphagia. Pupillary

dilatation causes blurred vision and increases the risk of falls and angle-closure glaucoma.

The TCAs produce pronounced sensitivity to postural hypotension which can lead to an increased risk of falls and associated fractures. Additive anticholinergic effects may be experienced when using TCAs with antihistamines, antipsychotics, and some 'antiparkinsons' agents.

Reversible inhibitors of mono-amine-oxidase (MAO)—Moclobemide is a reversible inhibitor of MAO type A. MAO type B is not inhibited, therefore it is still available for metabolising tyramine and other similar vasopressive compounds such as those present in many foods; thus drug-food interactions leading to hypertensive crises with moclobemide are rare. Moclobemide is usually recommended for treating mild to moderate depression. The benign side effect profile of moclobemide, its low potential for adverse drug interactions, as well as the fact that its pharmacokinetics are not age-dependent, offer advantages in the treatment of elderly depressed patients.[23] However, there are relatively few clinical trials evaluating the efficacy of moclobemide in older people.

Irreversible mono-amine-oxidase inhibitors (MAOI)—Trancylpromine is a MAOI which causes irreversible MAO inhibition of type A and B. Thus, it can induce hypertensive crisis after ingestion of tyramine-rich foods, the so-called 'cheese-reaction.' Dietary control (avoidance of tyramine-containing foods) is essential, and the possibility of drug interactions must be carefully monitored. Postural hypotension is of particular concern among the elderly because of the risk of falls. Concomitant use of sympathomimetic agents, narcotics, and serotonergic agents must be avoided.[5]

Serotonin and noradrenaline reuptake inhibitors (SNRI)—Venlafaxine does not produce sedative and anti-muscarinic side effects. However, there are safety concerns with elevations in blood pressure which may diminish over time.[5] Abrupt withdrawal must be avoided; doses should be tapered over 1 week.

First- and second-line treatments for depression among older patients—For uncomplicated depression, a SSRI should be the first choice antidepressant for the elderly. If the patient does not respond even after optimising the dose and ensuring proper duration of treatment, a TCA should be considered. If the patient does not respond despite adequate dose and duration, referral to a specialist is prudent.

In most cases, antidepressant therapy should be initiated with half the maintenance dose for the first five days of treatment. In the case of fluoxetine, for example, this may incur additional cost for the patient, as two different dosage forms (tablets and capsules) may need to be prescribed to initiate treatment. Table 2 provides a summary of the subsidy information and dosing guidelines for older patients for selected antidepressants. It is recommended that doses for the elderly are half that of normal adult doses.[5]

Treating mixed anxiety and depression—Anxiety is a common symptom in older patients and should be treated concurrently if it occurs with depression. The diagnostic category of mixed anxiety and depressive disorder (MADD) is outlined in the DSM-IV. Several antidepressants have shown efficacy in treating both anxiety and depressive symptoms in patients of various ages, including moclobemide, venlafaxine, and fluoxetine.[24] Imipramine and clomipramine have been recommended

for the treatment of depressed patients with comorbid panic disorder.[25] Benzodiazepines, although effective in treating anxiety and depression-related insomnia, have little efficacy in treating the core symptoms of depression.

Table 2. Dosages, indications for use and subsidy information on selected antidepressants

Class	Drug	Recommended dose in the elderly	Dosage form available in NZ	Subsidy information
SSRIs	Fluoxetine	S=5-10mg, M=20mg (morning)	Capsule 20mg Tablet 20mg, dispersible, scored	Fully subsidised Use tablet+capsule for incremental doses
	Paroxetine	S=10mg, M=20mg (morning)	Tablet 20mg	Endorsement required
	Citalopram	S=10mg, M=20 mg (morning)	Tablet 20mg	Endorsement required
SNRI	Venlafaxine	S=37.5mg (not available in NZ), M=225mg	Capsule 75mg Capsule 150mg	Retail pharmacy-Special authority
MAOI Type A	Moclobemide	S=150mg daily (preferably morning), M=up to 300mg *bd* (morning and evening)	Tablet 150, 300mg	Retail pharmacy – Specialist
MAOI nonselective	Trancylpromine	S=10mg *bd* (morning and lunch) M=10-30mg daily	Tablet 10mg	Fully subsidised
TCAs	Amitryptiline	S=10–25mg, M=100mg if tolerated (bedtime)	Tablet 10, 25, 50mg	Fully subsidised
	Dothiepin	S=50–75mg/day M=75mg/day divided doses or at bedtime	Capsule 25mg Tablet 75mg	Fully subsidised
	Doxepin	S=10–50mg/day M=30–50mg/day (divided doses or bedtime)	Capsule 10,25,75mg Capsule 50mg	Fully subsidised
	Imipramine	S=10mg/day, max 100mg (preferably bedtime)	Tablet 10, 25mg	Fully subsidised
	Trimipramine	S=30–50mg/day, max 100mg (preferably bedtime)	Capsule 25, 50mg	Fully subsidised

S=Starting dose; M=Maintenance dose; bd=twice daily; SNRI=Serotonin and noradrenaline reuptake inhibitors; MAOI=Mono-amine-oxidase inhibitor; TCA=Tricyclic antidepressants;
Adapted from British National Formulary, South African Medicines Formulary, Pharmac Schedule 2005

Drug interactions

All SSRIs inhibit (in various degrees) the metabolism of many other medications by the hepatic cytochrome P450 (CYP) isoenzymes.[26] Therefore, care must be exercised when adding an SSRI to a multidrug regimen, or when adding any new medication to a regimen that already includes an SSRI. The SSRIs (especially paroxetine and fluoxetine) have a high rate of protein binding which can lead to altered therapeutic or

toxic effects of other highly protein-bound medications. Citalopram is less highly protein-bound than the other SSRIs.[27]

Concomitant use of SSRIs, MAOIs, lithium,[28] St John's wort, and other serotonergic medicines can give rise to serotonin syndrome. This condition is characterised by confusion, agitation and hyperthermia, and medication should be discontinued.

TCAs are extensively metabolised in the liver and can be potentiated by cimetidine, fluoxetine, paroxetine and steroids.

Antidepressant drug interactions with commonly prescribed medications for older people are summarised in Table 3. Potential interactions between antidepressants and 'complementary medicines,' which are frequently used by older patients, are also reflected.

Table 3. Antidepressant drug-interactions with medications commonly prescribed for older people

Antidepressant class	Most important interacting drugs/drug class to avoid	Potential interacting drugs/drug class requiring careful monitoring
TCAs	Avoid all agents that strongly inhibit relevant cytochrome P-450 enzymes. Anticholinergic agents Do not use with MAOIs or other TCAs	Antihypertensives (e.g. guanethidine), thyroid drugs, sedatives/hypnotics, sympathomimetic drugs (eg, pseudoephedrine)
MAOIs	Do not use with SSRIs, TCAs (clomipramine, tryptophan), can lead to serotonin syndrome	Sympathomimetics (phenylephrine, pseudoephedrine). Buspirone
SSRIs	TCAs, phenytoin, codeine, flecainide, propafenone, warfarin Do not use with MAOIs Citalopram has the least potential for drug interactions with the CytP450 enzyme system	Other serotonergic drugs (eg. tryptophan, dextromethorphan) Lithium Warfarin, haloperidol, clozapine, alprazolam, triazolam, carbamazepine, beta-blockers
Venlafaxine	MAOIs	SSRIs, serotonergic drugs, lithium
Herbal medicines (e.g. St. John's wort, *Ginkgo biloba*, ginseng)	Do not use with SSRIs or MAOIs (potential serotonin syndrome)	Thiazide diuretics and oral anticoagulants (especially with *Ginkgo biloba*)

Duration of treatment

Antidepressant treatment should be continued for at least 12 months after remission of symptoms in the treatment of uncomplicated mild-to-moderate depression with no history of previous episodes. It is advisable to continue treatment for longer periods in older people especially if there is an increase in the likelihood of relapse. Older patients may have other risk factors for reoccurrence of depressive symptoms such as bereavement and loneliness. Relapse is more likely in patients who take longer to

respond, in those with high concomitant anxiety, and in those experiencing a medical or interpersonal event.[12,29]

Abrupt discontinuation of antidepressant treatment should be avoided because withdrawal symptoms may be precipitated. Doses should be tapered gradually over at least 4 weeks, and for patients who have been on long-term maintenance treatment, the dose should be tapered over 6 months.

Augmentation of antidepressants

Deliberate concurrent use of antidepressants is known as augmentation therapy, and involves the addition of a second antidepressant to existing treatment.[30] This is associated with an increased risk of toxicity and is not routinely recommended, and should only be considered after consultation with a specialist psychiatrist.

Lithium—Although lithium is not classified as an antidepressant, there is a scientific body of evidence supporting it as an effective and generally well-tolerated augmentation agent.

Lithium added to TCA therapy is the most intensively studied augmentation strategy in younger age groups. Consistent and significant responses have been documented. The positive effects of lithium augmentation have generally been achieved at therapeutic plasma lithium concentrations (0.4 to 1 mmol/L), but there does not appear to be a correlation between response and plasma concentration. Adverse effects associated with lithium are more common in the elderly, including renal effects, thyroid abnormalities, and dose-limiting neuromuscular and neurological adverse effects.[31]

Drug-induced depression

The contribution of prescribed medications to depression in the medically ill is poorly understood. Most information on drug-induced depression is derived from case reports, and various classes of medications have been implicated.[32,33]

Examples of drugs commonly prescribed to older patients that can induce or aggravate existing depression include:

- Antihypertensive and cardiovascular drugs: beta-blockers , hydralazine, methyldopa, reserpine, prazosin and clonidine, calcium-channel blockers, digitalis.
- Anxiolytic and hypnotic agents: long-term use of benzodiazepines, especially triazolam.
- Steroids: corticosteroids, oestrogen withdrawal.
- Other drugs such as antiparkinson and antineoplastic agents, cimetidine and ranitidine, metoclopramide, and opioids.

Psycho-stimulants in the treatment of depression

The use of psycho-stimulants (such as methylphenidate) for depressed elderly people is very controversial, and their use in this population may not be as safe as reported in their younger counterparts. They may be effective in medically ill patients, for whom

they should be prescribed short-term, under controlled conditions (while in hospital) and based on specialist recommendations.[34]

Non-pharmacological treatments

Light therapy, psychotherapy, and electroconvulsive therapy (ECT) are alternative or adjunct therapies. Light therapy may be used successfully to treat seasonal affective disorder (SAD).

Psychotherapy may be first-line treatment for mild-to-moderate depression, and is often recommended in conjunction with pharmacotherapy. Cognitive behaviour therapy (CBT) explores the origins of negative thoughts and feelings, and is particularly effective at treating mild depression if medication is not considered necessary. It is a useful adjunct to medication. Services from private psychologists range from approximately NZ$120 per hour.

ECT would only be considered after referral of the patient to a psychiatrist. This treatment is considered effective in severely depressed and suicidal patients, or when pharmacological therapies are contraindicated. Antidepressant therapy is usually required to prevent relapse after ECT. Some patients experience memory loss and there are also risks associated with the anaesthetic required.

Conclusion

It is difficult to diagnose depression among the elderly—it is compounded by the frequent coexistence of comorbid conditions. The choice of antidepressant for an older patient is complex. Individual patient factors such as severity of the depression and pharmacokinetic variables, socioeconomic factors, comorbid conditions, and concurrent medications have to be weighed against side effects, cost-effectiveness, patient adherence to the dosage regimen, and persistence with treatment.

The frequency and severity of side effects is a major factor in selecting an appropriate antidepressant. A simple antidepressant dosing regimen that takes into account other medication the patient may be taking will facilitate adherence to treatment. Duration of treatment must be continued for at least 12 months after remission of depressive symptoms. Other non-pharmacological treatment options that would be beneficial for an older patient should be considered, including referral to a specialist psychiatrist or geriatric psychiatrist if one is available, when appropriate.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Diluting delirium

Michael Croxson, Jennifer Lucas, Warwick Bagg

We present a patient who developed delirium after correction of chronic severe hyponatremia. The delirium was reversed after treatment to lower the serum sodium concentration and allow a more gradual rise in serum sodium concentration.

Case report

A 54-year-old Tongan man with treated miliary tuberculosis and diet-treated type 2 diabetes mellitus was admitted to the emergency department with a 3-week history of increasing nausea, lethargy, and headache. Pulmonary and miliary tuberculosis had been diagnosed 3 months earlier, the CT lung scan showing miliary nodules and minor lower lobe bronchiectasis. Sputum culture was positive.

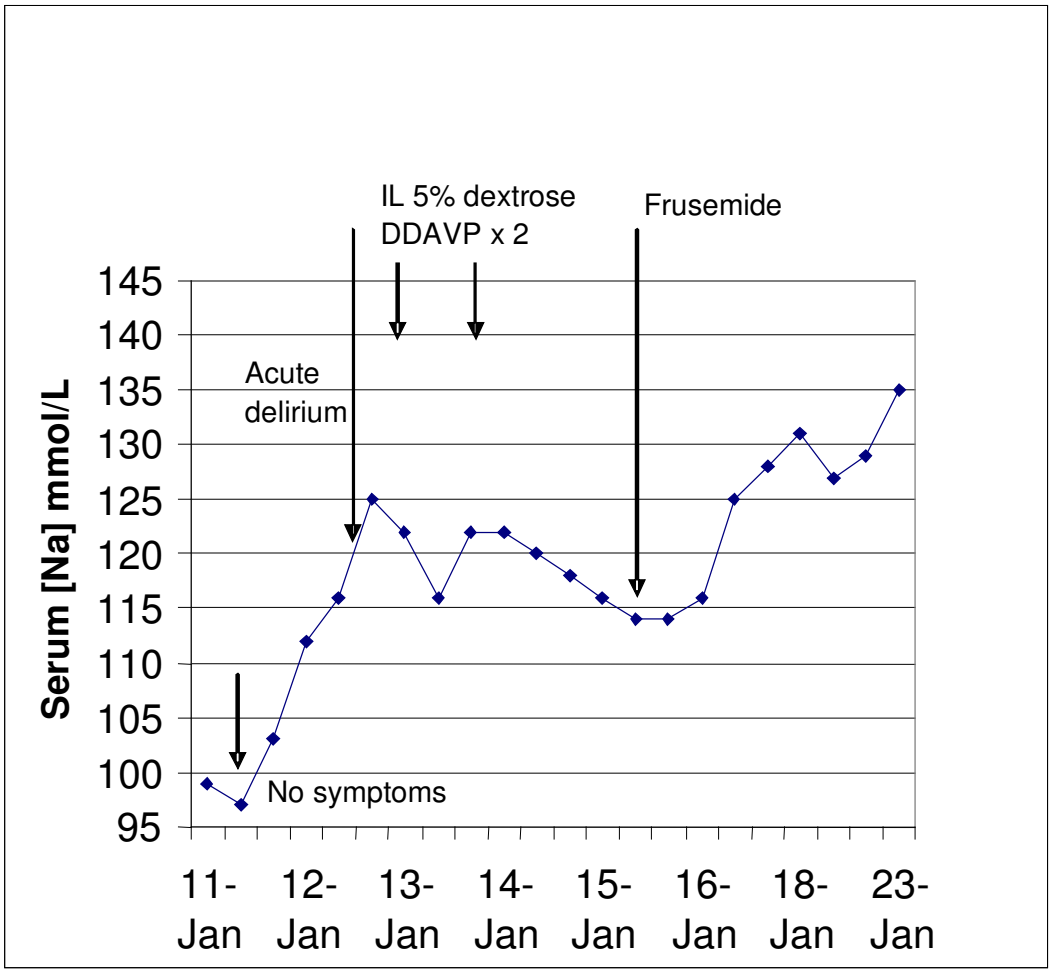
He had been treated with rifampicin 600 mg and isoniazid 300 mg daily together with a 6-week course of prednisone 30 mg daily. He was alert, hyperpigmented, and mildly hypovolemic. His neurological state was normal except for absent or reduced leg tendon reflexes. His blood pressure was 85/50 mmHg; serum sodium 99 mmol/L, potassium 5.5 mmol/L, and serum creatinine 0.12 mmol/L.

We suspected tuberculous Addison's disease with adrenal crisis induced by rifampicin[1] and withdrawal of prednisone. Despite receiving intravenous hydrocortisone (100 mg initially and 8 hourly together with 1 litre of 0.9% saline), his serum sodium fell to 97 mmol/L and he became confused. The blood glucose was 3.7 mmol/L but his confusion resolved rapidly after 50% IV dextrose.

Because of the severe hyponatremia, we elected to increase his serum sodium using 1.8% saline. He was given 240 mmol over 12 hours, and from then his fluid intake was restricted to 600 ml per 24 hours. His serum sodium rose to 112 mmol/L at 24 hours and then 122 mmol/L 48 hours post-admission. The rate of increase of his serum sodium was about 0.5 mmol/L/hour. At that time he developed an acute, agitated delirium with visual hallucinations and behavioural disturbance but without focal neurological abnormalities. Clonazepam sedation was required.

A CT brain scan was normal. Concerned that his acute delirium resulted from too rapid correction of his chronic hyponatremia and fearful of impending myelinolysis[2] we decided to lower his serum sodium again.[3] He was given 1 L of 5% dextrose over 2 hours plus dexamethasone 4 mg intravenously. The serum sodium fell to 116 mmol/L. Moreover, within 18 hours of induced hyponatremia his delirium subsided completely. As this water load was readily excreted with a rise of serum sodium to 122 mmol/L he was then given two doses of DDAVP and free fluid intake until the fifth day when the serum sodium was allowed to rise at approximately 5 mmol/L per day into the normal range. A follow-up MRI brain scan at 4 weeks was normal.

Figure 1. Serum sodium levels during the patient's treatment in 2001



Discussion

Rapid correction of chronic hyponatremia may cause fatal pontine and extra-pontine myelinolysis heralded by delirium.[4] Experimental and clinical studies by Soupart and coworkers showed that myelinolysis can be prevented and even reversed by delaying the rate and magnitude of serum sodium correction.[5] Glucocorticoids lower vasopressin levels and restore the kidney's ability to excrete free water.

The water diuresis resulting from glucocorticoid therapy in this man caused an unexpectedly rapid reversal of his chronic hyponatremia thereby provoking an acute delirium that resolved after relowering of the serum sodium. His Addison's disease was compensated initially then disguised by prednisone therapy until the enzyme-inducing effect of rifampicin led to his presentation with overt adrenal insufficiency. Subsequently primary adrenal insufficiency was confirmed by the findings of aldosterone and cortisol deficiency, markedly raised plasma renin and ACTH values, and the appearance of nodular adrenals typical of tuberculous gland involvement on a CT scan.

Hydrocortisone 100 mg IV immediately and 8 hourly led to a water diuresis. Acute delirium developed when the serum sodium was 122 mmol/L and resolved 18 hours later when the level was 116 mmol/L.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Mesalazine-associated acute tubulointerstitial nephritis in a patient with spondylarthropathy

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Mesalamines (or mesalazines) are slow-release formulations of 5-aminosalicylic acid (5-ASA) and are effective as primary treatment and maintenance therapy in inflammatory bowel disease (IBD).[1] Some concern has recently been raised about the safety of mesalazine, with regards to renal toxicity.[2] Herein we present a case of acute tubulointerstitial nephritis (TIN) due to mesalazine (Salofalk®) in a spondylarthropathy (SpA) patient. The drug was started for his IBD and no additional risk factors were present for renal failure.

Case report

56-year-old Turkish man, with a 6-month history of inflammatory back pain (IBP) was referred from the gastroenterology unit of our hospital (Izmir, Turkey) where he had been diagnosed to have ulcerative colitis and started on mesalazine (Salofalk®) 2 g/day.

On the basis of IBP and presence of IBD, he was diagnosed as having SpA according to the European Spondylarthropathy Study Group criteria.[3] Meloxicam 15 mg/day was prescribed. Three months after the initial presentation, he was hospitalised complaining of fever, malaise, and anorexia which lasted 10 days. He stopped meloxicam because of his concerns over renal toxicity despite of an immediate favourable effect on his IBP initially, but continued receiving mesalazine.

Physical examination was normal. Vital signs revealed an oral temperature of 38.2ºC. Laboratory tests demonstrated a normocytic anaemia (11.5 g/dL), leucocytosis (WBC 14,300/mm^3), elevated renal function tests (BUN 45 mg/dL, creatinine 5.3 mg/dL) and increased CRP (31 mg/l).

Blood, urine and stool cultures were negative. Urine examination revealed a protein value of 0.5 g/day. Chest X-ray and renal ultrasonography with Doppler were normal. The use of mesalazine was discontinued. On day 4, serum creatinine increased up to 6.3 mg/dL and his temperature remained high (39°C).

Renal biopsy was performed and histological findings were consistent with an acute TIN. Over the next few days, the patient improved dramatically. On day 10, serum creatinine had fallen to 1.6 mg/dL. In the follow-up, the patient maintained stable mild chronic renal insufficiency with a creatinine level of 1.8mg/dL.

Discussion

In the last decade, 5-ASA products have largely replaced sulphasalazine (SSZ) in the treatment of IBD because of their comparable efficacy and better tolerability.[4,5] On the other hand, mesalazine has also been suggested as an alternative to (SSZ) in the treatment of SpA.[6,7] A safety problem of nephrotoxicity with mesalazine has been

recently announced in several case reports—including acute TIN,[8] nephrotic syndrome,[9] and chronic TIN[10] (the most frequently reported).[2]

The risk of TIN associated with 5-ASA is unknown. It has been reported that 1 in 100 patients will develop some renal impairment and less than 1 in 500 will develop serious renal damage. Most cases of TIN have been reported in patients who have taken 5-ASA for at least 6 months. If mesalazine nephrotoxicity is diagnosed within 10 months, renal function restoration after withdrawal is observed in 85% of patients. In those cases in which the diagnosis was delayed beyond 18 months after treatment, only partial recovery of renal function occurred.[10] It is interesting that TIN has been rarely reported with SSZ,[11,12] even though it contains 5-ASA too. It may be due to comparably high absorption and urinary excretion of mesalazine.[5,12]

Our patient was diagnosed as having SpA. He was reluctant to take NSAIDs particularly his fear of renal toxicity but stayed on mesalazine which had been prescribed for IBD until he developed renal failure 3 months later. Prognosis in our patient was excellent because of early diagnosis. To prevent serious side effects of mesalazine therapy, it has been recommended that serum creatinine should be measured in any patient treated with 5-ASA at the start of the treatment, monthly for the first 3 months, 3-monthly for the next 9 months, 6-monthly thereafter, and annually after 5 years of treatment, based on the available data.[4]

Patients with SpA are frequently prescribed NSAIDs, which are well-known nephrotoxic agents. When a decrease in renal function develops in a patient who is on NSAIDs and 5-ASA concomitantly, nephrotoxicity due to 5-ASA should also be kept in mind.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

The ownership elephant: ownership and community-governance in primary care

Peter Crampton

Abstract

Ownership of primary care is an often neglected but important health systems design parameter. The New Zealand Primary Health Care Strategy has established Primary Health Organisations (PHOs) as non-profit umbrella organisations, however in most instances their constituent general practices are for-profit small businesses. This viewpoint paper aims to: (a) define ownership and community participation; (b) summarise some of the evidence from the NatMedCa study pertaining to ownership-related differences; and (c) discuss the policy implications of different ownership forms in primary care, and the implications of merging different ownership forms under the umbrella of PHOs. Ownership confers governance responsibility (ultimate control) for an organisation, and accountability for its actions. Community governance involves vesting overall control of resources in users and the community, rather than with health service managers or health professionals. Results from three studies using the NatMedCa survey indicate that community-governed non-profits in New Zealand differ in a number of respects from their for-profit counterparts. As non-profit and for-profit ownership forms have different social roles, and as meaningful community participation in governance is determined in large part by ownership structures, there is a need for ownership frameworks to be used more widely in health policy making. Because of the ownership boundary that exists between non-profit community-governed PHOs and their constituent for-profit general practices PHOs may have little real ability to effectively govern their practices.

Organisations within the New Zealand primary care sector are evolving rapidly.[1] The Primary Health Care Strategy has established Primary Health Organisations (PHOs) as non-profit umbrella organisations, however in most instances their constituent general practices are for-profit small businesses.

In the UK, the US, Canada, New Zealand, and Australia private ownership of primary care maintains a dominant position, albeit with important local modifications in each country. The UK, the US, and New Zealand are experimenting both with different ownership arrangements,[2,3] and with increased community involvement in governance in primary care,[4–7] but there is no consensus about what constitutes the ideal ownership configuration. Indeed, there seems to be little agreement that ownership is an important structural variable in health systems design. Ownership is the 'elephant in the living room' that policy makers and practitioners in New Zealand tread carefully around but fail to acknowledge properly in the formulation and implementation of primary care policies.

Recent research evidence from the NatMedCa study sheds light on ownership-related differences between different types of primary care practice in New Zealand. This new evidence provides the opportunity for primary care practitioners and policy makers to explore the implications of different primary care ownership arrangements.

This viewpoint paper aims to:

- Define ownership and community participation,
- Summarise some of the evidence from the NatMedCa study pertaining to ownership-related differences, and
- Discuss the policy implications of different ownership forms in primary care as well as the implications of merging different ownership forms under the umbrella of PHOs.

Ownership

Ownership confers governance responsibility (ultimate control) for an organisation, and accountability for its actions. Primary care organisations can be classed as government owned and operated, or privately owned and operated, with the latter being divided into those responsible to a community-governance board versus those not responsible to such a board.

Community-governed non-profit practices are often referred to as third-sector organisations (indicating non-government non-profit status). As with most organisational typologies, there is an inevitable blurring of organisation forms. Typically, however, government organisations, irrespective of their specific governance arrangements, are primarily accountable to government; private for-profit organisations are primarily accountable to their proprietary owners or shareholders; and private non-profit organisations are primarily accountable to their governing body.

Despite blurring of ownership boundaries, clear differentiation between the public and private spheres is essential if there is to be accountability for the spending of public funds.

Community governance

Most general practices in New Zealand are owned and governed by general practitioners (GPs); in an ownership sense they are private for-profit practices (but it should be noted that this ownership classification does not in itself imply that a general practice actually makes a profit or operates as a successful business). While the classical distinction between non-profit and for-profit rests largely on the non-distribution constraint—a non-profit organisation may not lawfully pay its profits to owners or anyone associated with the organisation[8]—the terms ‘third sector’ and ‘non-profit’ are frequently used as shorthand for a package of organisational characteristics that includes governance arrangements where primary accountability is not to private proprietors or shareholders, but rather to users and community representatives.

The Family Planning Association and the New Zealand Aids Foundation are examples of non-profit/third sector organisations, as are PHOs, which are required by government to have meaningful community participation in their governance.[5]

Community governance seeks to ensure that the communities served by the organisation have control over key decision-making.[9] In New Zealand, community governance has distinguished a subset of primary care organisations located in the private non-profit sector (for example Health Care Aotearoa practices[10]), but

increasingly the Primary Health Care Strategy is introducing the principles of community governance more broadly in PHOs.[5] There are numerous benefits associated with community participation in primary care,[11–13] however the challenges and implications of implementing community governance of PHOs and general practices are considerable given the current predominance in primary care practices of a governance culture associated with private for-profit ownership where accountability and control reside with private proprietors.[13]

The concept (and practice) of community governance is distinct from the concept of clinical governance. Clinical governance refers to the exercise of collective accountability for the management of clinical performance, placing emphasis particularly on the role of clinicians, as well as managers.[14] Clinical governance has been interpreted as a response, in part, to the commercially driven health reforms of the 1990s which reduced the role of clinicians in decision making.[15]

Collective clinical accountability for quality is highly desirable; however, to the extent that clinical governance involves clinicians in the management of resources, there is potential conflict between the principles of clinical governance and the principles of community governance. The latter involves vesting overall control of resources in users and the community, rather than with health service managers or health professionals.

Currently the balance between community control, managerial control and clinical control varies considerably between different types of primary care organisation with apparently little consensus about what the 'right' balance is.

What can be learnt from the NatMedCa study?

Three recent studies comparing for-profit and community-governed non-profit primary care organisations, using data from the NatMedCa survey, highlight a range of important ownership related differences.[16–18]

The National Primary Medical Care Survey (NatMedCa), carried out over 2001/2002, was a nationally representative, multistage, probability sample of GPs and patient visits.[19] The primary purpose of the survey was to collect data on the content of patient visits. For two periods, each lasting 1 week, each selected GP completed a questionnaire for a 25% systematic sample of patient visits. The questionnaire was adapted from the annual US National Ambulatory Medical Care Survey (NAMCS) 2003.[20]

To obtain a nationally representative sample:

- Geographic locations were sampled, and
- GPs were sampled from locations, stratified by organisation type (independent; independent practitioner association; capitated; community-governed non-profit) and rural/urban (metropolis & cities; towns and rural areas).

GP and visit weights were calculated to take account of different sampling probabilities, so that approximately unbiased estimates of proportions, means, and measures of association between ownership status and visit characteristics could be calculated.[21] Practices in the study were categorised according to their ownership status—private for-profit and community-governed private non-profit.

Non-profits had to meet at least two of these three criteria:

- They had a community board of governance,
- There was no equity ownership by GPs or others associated with the organisation, and
- There was no profit distribution to GPs or others associated with the organisation.

The total visit sample consisted of 10,506 records gathered from 246 GPs, 48 (19.5%) of whom worked in non-profit practices and 198 (80.5%) of whom worked in for-profit practices. The overall GP response rate was 71.7% (70.7% in the for-profits and 72.7% in the non-profits).

Currently about 3% of New Zealand GPs work in community-governed non-profit settings.

Patient and visit characteristics[18]—Compared with for-profits, community-governed non-profits served a younger, largely non-European population, nearly three-quarters of whom had a community services card; 10.5% of whom were not fluent in English; and the majority of whom lived in the 20% of areas ranked as the most deprived by the NZDep2001 index of socioeconomic deprivation. Patients visiting non-profits were diagnosed with more problems. The problems presented to non-profit primary care GPs included higher rates of asthma, diabetes, and skin infections. The duration of visits was significantly longer in non-profits. No differences were observed in the average number of laboratory testes ordered. The odds of specialist referral were higher in for-profits when confounding variables were controlled for.

Practice characteristics[16]—Community-governed non-profits had lower financial and cultural barriers to access as measured, respectively, by their lower patient fees and their higher numbers of Maori and Pacific Island staff. Non-profits provided a somewhat different range of services; for example non-profits were more likely to provide community worker and group health promotion services and for-profits were more likely to provide sports medicine and emergency/accident call-out services.

For-profits were more likely to have specific items of equipment, such as cautery machines and proctoscopes, despite being on average smaller practices with fewer full-time equivalent doctors. Non-profits performed better in terms of written policies on quality management; for example, they were more likely to have written policies on complaints, critical events and quality management. The greater percentage of non-profits with these ‘process’ quality indicators may reflect the larger staff that was available to them. In general, these quality measures were not a requirement of their funding contracts. Non-profits were more likely carry out locality service planning and community needs assessments.

The majority of the differences in practice characteristics persisted when comparisons were restricted to:

- Practices serving similar low-income and non-European populations,
- Practices that were capitation funded, and
- For-profits that employed more than one GP. This suggests that the non-profits are not merely adapting to the needs of the patient population, or the incentives

associated with capitation funding, but were operating in a substantially different way compared with for-profits.

Staffing and primary care teams[17]—Primary care teams were largest and most heterogeneous in community-governed non-profit practices; the majority employed doctors, nurses, managers, reception staff, administrative staff, and community workers, and 34.6% employed midwives. Next most heterogeneous in terms of their primary care teams were practices that were members of an independent practitioner associations (IPA) which employed the majority of the country's GPs (71.7%).

'Independent practices' (those that were not members of an IPA) had the most parsimonious practice teams. A majority of both IPA and independent practices employed doctors, nurses, and reception staff—but only a very small percentage employed community workers or midwives. Community-governed non-profits employed a higher proportion of women GPs than did IPA and independent practices.

There were marked ethnicity differences between staff employed at the different types of practices, with community-governed non-profits employing more Maori and Pacific staff, including both doctors and nurses.

Policy implications

Community-governed non-profits in New Zealand serve a poor, largely non-European population who present with somewhat different rates of various problems compared with patients at for-profits. Community-governed non-profits have reduced financial (i.e. lower patient fees) and cultural barriers to access (i.e. more Maori and Pacific Island staff) compared with their for-profit counterparts. They also provide a different range of services, are more likely to have a population-orientation to service planning, are more likely to have quality-management policies, and have larger and more diverse staff teams on average.

These results are consistent with theoretical predictions relating to the social role of the non-profit sector. Various partly complementary and partly competing theories seek to explain the role and scope of private non-profit activities in different countries.[22–27] Theories suggest that non-profit organisations predictably fulfil a range of social functions that may be of great use to policy-makers and communities. In particular, they have a role catering for the diverse needs of minority populations not catered for by the government and for-profit sectors.

Non-profits are able to respond to the needs of minority community interests; for example, minority ethnic groups, because their governance boards are more able to closely represent minority groups compared with their for-profit business counterparts, whose governance structures are likely to reflect the interests of the proprietary owners or share holders. From a theoretical standpoint, this responsiveness to minority needs may reflect a basic motivation arising from the failure of government and for-profits to serve minority populations, but it may also reflect the interdependency of government and the non-profit sector insofar as the non-profit sector, unlike government, is unconstrained by the needs of the 'median voter'.[22] The capacity of non-profits to independently represent the interests of minorities assumes great importance in New Zealand, where Maori have striven to establish primary care services tailored to meet their needs, and have used the non-profit form as a vehicle for increasing self-determination.[10]

Explanations for the observed differences are likely to include a clear mission amongst the community-governed non-profits to serve the needs of vulnerable population groups, and a more managerialist management culture (as opposed to a predominantly health professional management culture). But to what extent does financing explain the observed differences between for-profits and community-governed non-profits?

While differences between non-profits and for-profits are likely to be associated with their ownership and governance arrangements, it is hard to separate out the dual influences of community-governance and the different financial incentives facing GPs in the two types of practice.

In the NatMedCa studies cited above, non-profits were defined partly on the basis of no equity ownership by GPs and no profit distribution to GPs (that is, financial surpluses did not directly represent extra income for GP practice owners), hence it is likely that financial incentives had some influence on such characteristics as patient mix, patient charges, staffing arrangements, and referral patterns. This is due to the fact that, historically, the level of government subsidies for primary medical care has required GPs to charge a co-payment.

While the level of co-payments has been at the discretion of GPs and influenced by patients' ability to pay, co-payment-related income has undoubtedly determined in part the level of investment GPs have been able to make in practice infrastructure and service provision. There are no recent data on the total amounts of government funding going to different types of practice. At the time of the NatMedCa study, all practices were free to determine their level of patient co-payments for consultations.

Government funding of primary care practices was determined within a complex contracting framework. Some Maori and community-governed practices received government assistance for their establishment, as did the independent practitioner associations to which most for-profit practices belonged.[28] The operational funds for all practices were negotiated largely within a framework that did not distinguish between non-profit and for-profit status. Funding of programmes outside of standard medical consultation work was allocated through a modified form of tendering. Most community-governed practices did not participate in referred services budget-holding programmes which, in their early years (1990s), delivered significant new funding to practices for service development.[29,30] More research comparing financing, profits, and GP incomes is required to further elucidate the above issues.

A more complete discussion of ownership frameworks and the pros and cons of different primary care ownership arrangements can be found elsewhere.[31] A few points deserve emphasis here. While private for-profit ownership of primary care facilities is the norm in many countries, including the UK, the US, Canada, Australia, and New Zealand, each country has its own particular mechanisms for encouraging the alignment of private for-profit interests with those of government.

For example, in the UK, GPs are held in a very tight contracting and funding embrace by government, and their opportunities for unchecked entrepreneurialism are severely restricted. The main argument advanced in support of private for-profit ownership is efficiency, but there are few empirical data that provide support. Some insights are provided by the literature on the US hospital sector.

In a review of the US literature, Gray concluded that (in respect of general hospitals, psychiatric hospitals, and nursing homes) non-profits overall tended to care for more uninsured patients, provided a wider array or services, had quality advantages, and had similar or lower costs.[32] Indeed, the quality issue is illustrated in a meta-analysis of studies of mortality in US hospitals by Devereaux et al,[33] who found a 2% increased risk of mortality in private for-profit hospitals in the US (equating to about 14,000 excess deaths[34]).

The disadvantages of private for-profit ownership include the loosening of direct accountability to government; the potential intrusion of private for-profit interests into public good programmes; and the existence of market failures in health and the consequent need for extensive government involvement. In primary care, many of the conditions necessary for perfect markets are violated to some degree. For example, information asymmetries between doctors and service users are frequently huge; primary care is responsible for providing a range of public good services such as immunisations and drug and alcohol treatments; in small towns and rural areas there is frequently a lack of competition; and there are marked positive externalities associated with primary care such as reduced social costs resulting from the provision of preventive care and screening.

Market failure is addressed in various ways. In the UK and New Zealand contracts and funding mechanisms have been important tools for government.

Does it matter whether for-profit primary care organisations are part of a large conglomerate that is listed on the stock exchange, or are smaller proprietary organisations? Proprietary organisations are independent, owner-operated organisations (typical of general practices in New Zealand, Australia, and the UK), and investor-owned organisations are usually part of multi-facility systems whose stock is publicly traded and whose owners therefore have little if any direct contact with the institution.[35]

While it might be expected that ownership-related differences in structure and patterns of treatment between private for-profits and non-profits will be relatively small in countries such as New Zealand and the UK (due to the proprietary style of general practice), this was not the case in the NatMedCa studies: ownership-related differences were marked and are likely to be related, at least in part, to the presence of community governance in the non-profits.

The non-profit sector is diverse and non-profit status does not provide any guarantee that an organisation is focused on non-profit public-good service. Experience from the US and elsewhere suggests that non-profit status can provide an effective vehicle for the pursuit of profit objectives (disguised profit distribution).[36,37]

Taking into account the differences observed in the NatMedCa studies between community-governed non-profit practices and for-profits, and the theoretical literature concerning the different social roles of the non-profit and for-profit sectors, what then are the implications of merging different ownership forms under the umbrella of PHOs: can the objectives of non-profit community-governed PHOs be aligned successfully with their constituent for-profit general practices?

PHOs have no mandate or power to breech the ownership boundary that separates them from their privately owned constituent general practices. Indeed, given that

different general practices are likely to have substantially different cost structures and business objectives from one another, it seems unlikely that a community-governed PHO can exercise real control over its private for-profit general practices—in regard to patient fees and staffing arrangements for example.

Under current funding arrangements (i.e. with partial government subsidies for general practice) such control is both impractical and would violate the ownership rights of private GPs. In essence, both the research evidence and theory suggest that community-governed PHOs may have little real ability to govern their constituent for-profit general practices.

For PHOs to exercise real governance control over their member practices, the ownership boundary between PHOs and practices will have to be eliminated either through the conversion of practices to non-profit community-owned entities with objectives aligned to those of the PHO, or through government ownership of both PHOs and practices.

Conclusions

Historical and contemporary policies have favoured private for-profit ownership of primary care in the UK, the US, Canada, Australia, and New Zealand. However, private for-profit dominance should not be regarded as a constant, but rather as a design variable amenable to incremental policy manipulation. Few observational studies have directly compared characteristics of community-governed non-profit primary care practices with their for-profit counterparts.[11]

The NatMedCa studies cited above provide evidence that ownership and governance have an important influence on the structure and function of primary care practices in New Zealand. These studies provide no insights into the total amount of government funds going to different types of practice, and further research will be required to determine the efficiency of the different ownership models analysed here.

Communities, purchasers, primary care professionals, and policy makers should consider more actively experimenting with different ownership arrangements in order to gain sufficient local experience to enable informed choices to be made regarding ownership. The capacity of community-governed non-profit practices to serve diverse ethnic and low-income population groups highlights for communities, policy makers, and purchasers the role of ownership and governance in shaping the purpose and function of primary care practices.

In conclusion, ownership of primary care is an often neglected but important health systems design parameter. As non-profit and for-profit ownership forms have different social roles, and as meaningful community participation in governance is determined in large part by ownership structures, there is a need for ownership frameworks to be used more widely in health policy making.

Finally, because of the ownership boundary that exists between non-profit community-governed PHOs and their constituent for-profit general practices, PHOs may have little real ability to effectively govern their practices.

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Acknowledgements: The NatMedCa survey was funded by the Health Research Council of New Zealand. I am also grateful to the NatMedCa Research Group led by Professor Peter Davis for their assistance, support, and data access; practitioners and organisations for providing data and supporting the NatMedCa study; and anonymous reviewers for their helpful comments on an earlier draft of this paper.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Long-acting inhaled bronchodilators for COPD—lack of logic continues

David Jones

Abstract

Long acting inhaled bronchodilators—beta agonists and anti-cholinergic—can improve symptoms, enhance quality of life, increase exercise ability, and reduce exacerbation rates in patients with COPD. PHARMAC needs to change policy so that these patients have easy access to this effective group of drugs.

Medications::	Long acting beta agonists (LABAs)—formoterol and salmeterol.
	Long acting anticholinergic—tiotropium.
Indication:	Bronchodilatation, for control of symptoms in chronic obstructive pulmonary disease (COPD).
Background:	Formoterol (Oxis® and Foradil®) inhaled from dry powder device 12-24 mcg bd.
	Salmeterol (Serevent®) inhaled from MDI or dry powder device, 25–50 mcg twice a day.
	Tiotropium (Spiriva®) inhaled from dry powder handihaler device, 18 mcg once a day
Regulations:	Both LABAs are only allowed for asthma**,** not for COPD. For Oxis 6 mcg, the prescriber has to vouch that the drug is for a "certified condition"; and for the other LABAs, a special authority is required.
	Tiotropium requires a special authority application giving a diagnosis of COPD, a 100-metre walking limit, and an FEV_1 below 40% predicted (i.e. severe disease).
Clinical usefulness:	These two classes of inhaled drug are very effective bronchodilators in COPD, being superior to short-acting agents in improving lung function, breathlessness, exercise tolerance, and quality of life.
	Their use also leads to a reduction in severe exacerbations, resulting in a favourable pharmacoeconomic profile.

Background: COPD is a major cause of death and disability in New Zealand. Our country's burden is probably similar to the United Kingdom where 7.5% of adults have chronic cough and phlegm[1] and 1% carry a doctor's diagnosis of COPD.[2] Indeed, in the United Kingdom there are 2–4 times as many GP consultations for COPD as for angina; and in 65–74 year old men, 7.3% of hospital admissions are due to COPD.[3] Although the burden of COPD disease is increasing (it is projected to be the fourth leading cause of disability worldwide by 2020), it comes bottom of a list of 21 common diseases in attracting monetary resources.[4]

COPD causes breathlessness, exercise limitation, social isolation, and dependence. Exacerbations disrupt normal life, and result in major community cost when hospital admissions result. Because the COPD population tends to be elderly and stoically uncomplaining, we do not hear a loud clamour demanding their share of the health dollar. Many of them feel self-blame about this largely tobacco-related problem, and perhaps some in the corridors of power take this view too.

The modern management of COPD puts aside nihilism. It involves a multidiscipline approach of layered interventions depending on severity, with the aims being to relieve symptoms, improve quality of life, reduce exacerbations, and (if possible) extend useful life. The approach is codified in several guideline documents, with GOLD[5] (Global Initiative in Obstructive Lung Disease) being the most generally accepted.

Common to all guidelines is the central role of inhaled bronchodilators, because—contrary to the old idea of COPD being "irreversible"—these agents do have a useful effect in reducing airflow limitation in the vast majority of such patients. It is worth stating here that although a bronchodilator delivered to the airways of a COPD patient may produce just a small increase in FEV1, dilatation of smaller airways produces beneficial effects not reflected in this standard measure. The residual volume falls, improving diaphragm function, and freer airflow through small airways delays the dynamic hyperinflation that occurs with exercise and is probably the main cause of exercise limitation.[6,7]

A summary of the management recommended by GOLD is reproduced below (see Table 1). Attention is drawn to the recommendation that patients classed as moderate or severe (FEV_1 less than 80% predicted) should use "regular treatment with one or more long-acting bronchodilators".[8]

LABAs were introduced to the UK market in 1990, and have been used commonly for COPD as well as asthma since 1997. Tiotropium has been available in the UK since 2003, with no restriction in its prescription by any medical practitioner for treatment in COPD.

Table 1. GOLD recommendations for treatment at each severity level of COPD.

(From: GOLD Executive Summary updated 2004 [as at reference 5], page 22)

Table 8 - Therapy at Each Stage of COPD					
Old	**0: At Risk**	**I: Mild**	**II: Moderate** IIA / IIB		**III: Severe**
New	**0: At Risk**	**I: Mild**	**II: Moderate**	**III: Severe**	**IV: Very Severe**
Characteristics	• Chronic symptoms • Exposure to risk factors • Normal spirometry	• $FEV_1/FVC < 70\%$ • $FEV_1 \geq 80\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $50\% \leq FEV_1 < 80\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $30\% \leq FEV_1 < 50\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $FEV_1 < 30\%$ or $FEV_1 < 50\%$ predicted plus chronic respiratory failure
	Avoidance of risk factor(s); influenza vaccination				
		Add short-acting bronchodilator when needed			
			Add regular treatment with one or more long-acting bronchodilators *Add* rehabilitation		
				Add inhaled glucocorticosteroids if repeated exacerbations	
					Add long-term oxygen if chronic respiratory failure *Consider* surgical treatments

Evidence from the literature

Bronchodilator studies have usually used *lung function* measurements as the outcome demonstrating effectiveness—and there are several confirming that LABAs[9,10] and tiotropium[14,18] compare favourably against placebo and the short-acting agents in COPD. A recent example showed that once-daily tiotropium and twice-daily formoterol alone (and in combination) improved morning and evening spirometry.[11]

Of more relevance to the sufferers of COPD are those studies showing improvement in *symptoms and in quality of life*. In a randomised parallel group study of 674 COPD patients,[9] salmeterol improved day and night symptoms and the need for salbutamol as well as the FEV_1.

Salmeterol improved symptoms and quality of life more effectively than qid ipratropium in 408 COPD patients[12] and, in another study, was better than the fenoterol/ipratropium combined inhaler.[13]

Tiotropium once daily was shown to improve day to day breathlessness and increase respiratory health status in 921 COPD patients in a 12 month study.[14]

Impaired exercise capability is the universal limiting symptom in severe COPD. In a recent study of COPD patients undergoing a rehabilitation programme, tiotropium convincingly improved exercise capacity and reduced dyspnoea.[15] The effectiveness of salmeterol was shown last year.[16]

If a pharmaceutical agent is able to *reduce exacerbations* of COPD, especially those resulting in hospital admission, it will be welcomed by the COPD sufferer and by health service funders. Tiotropium has been demonstrated to do this in placebo-comparison studies.[17,18] Both salmeterol and tiotropium reduced exacerbations in a 6-month study comparing each drug and placebo.[19] Both drugs—tiotropium moreso—had a beneficial effect on clinical outcomes and reduced health service use, with consequent economic advantage.

Economic savings were estimated in a Dutch/Canadian pharmacoeconomic study and found especially favourable for tiotropium.[20,21] A similar finding was described in the US context in two 12-month studies.[22]

Opinion

Long-acting bronchodilators are a logical treatment in COPD, because studies have shown them to be better than short-acting agents in relieving symptoms, improving exercise capacity and quality of life, and reducing rate of exacerbations. The newest agent —tiotropium—is probably better than long-acting beta-agonists in COPD patients, and a combination of the two drugs may be additionally effective.

At this point, PHARMAC still does not allow prescription of LABAs for COPD, despite ample evidence that they provide substantial benefit and GOLD guidelines recommending their use. Also, for tiotropium, the Special Authority PHARMAC has set the qualifying severity level well below what is supported by evidence and what is recommended by GOLD.

The time has now come for PHARMAC to recognise the body of literature and expert opinion, and to extend the availability of long-acting bronchodilators for this vulnerable group of people.

For COPD patients with an FEV_1 below 80% predicted, a long-acting beta-agonist or long-acting anticholinergic should be prescribable without bureaucratic hurdles. Tiotropium is probably the better first-line choice. A combination of both groups may help the more severe patients with FEV_1 below 50%, but more secure evidence for this recommendation is still needed.

Conflict of interest statement: I do not receive funding from any pharmaceutical or health product company. Over my career I have assisted various companies by contributing to educational activities, and have received support to enable attendance at meetings.

I have previously held positions on the medical advisory panel and the Council of the Asthma Foundation. In 1997, I chaired the organising committee of the annual scientific meeting of the Thoracic Society of Australia & NZ, which received considerable support from the pharmaceutical and equipment industry.

I am a member of the organising committee of the annual Respiratory Workshop, which is supported by Boehringer Ingelheim. The workshop subject themes in the last three years have been airway immunology, mucociliary clearance, and pleural disease.

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Vol 118 No 1222 ISSN 1175 8716

Human instincts, normal and pathological: the moral sense

This extract comes from a speech read at the Annual Meeting in Auckland by Herbert Barraclough, M.B., Auckland, and published in the New Zealand Medical Journal 1905, Volume 4 (16), p212–213.

The moral sense should be very briefly considered in this connection. This sense is not an instinct, but rather an acquired quality, as ideas of morality vary considerably from age to age. The right of yesterday is the wrong of tomorrow. Many moral lapses, however, occur which are often ascribed to lack of religious training, but which I cannot but attribute to far other causes. I have already referred to some, but here I will only mention those that more especially affect young people, and are mostly of a sexual nature.

Offences against the Criminal Law Amendment Act are terribly frequent in this colony. One of the reasons for this is undoubtedly lack of parental control and guidance; but I have often thought that one of the causes of precocious sexual desire is the excessive use of meat by young people. Nitrogenous food is a well-known sexual excitant, and still it is administered to children to an altogether undesirable extent. It is my firm opinion that children should be almost, if not entirely, vegetarians until the age of puberty is passed.

Another cause of permanent immorality is the lack of charity towards the one who has fallen. “Judge not, that ye be not judged,” does not enter into the modern Christian’s scriptural quotations. This applies more especially to the woman, if not entirely. A man may rise after many falls, but once a woman has fallen sexually many hands are raised to thrust her down, but scarcely one voice is heard to bid her rise. Only women of rarely strong characters and with an innate purity can hope to win back their way to the respect of their fellows. I can only plead for more charity for the sins of the young, and then we shall indeed have less opportunities for exercising such charity.

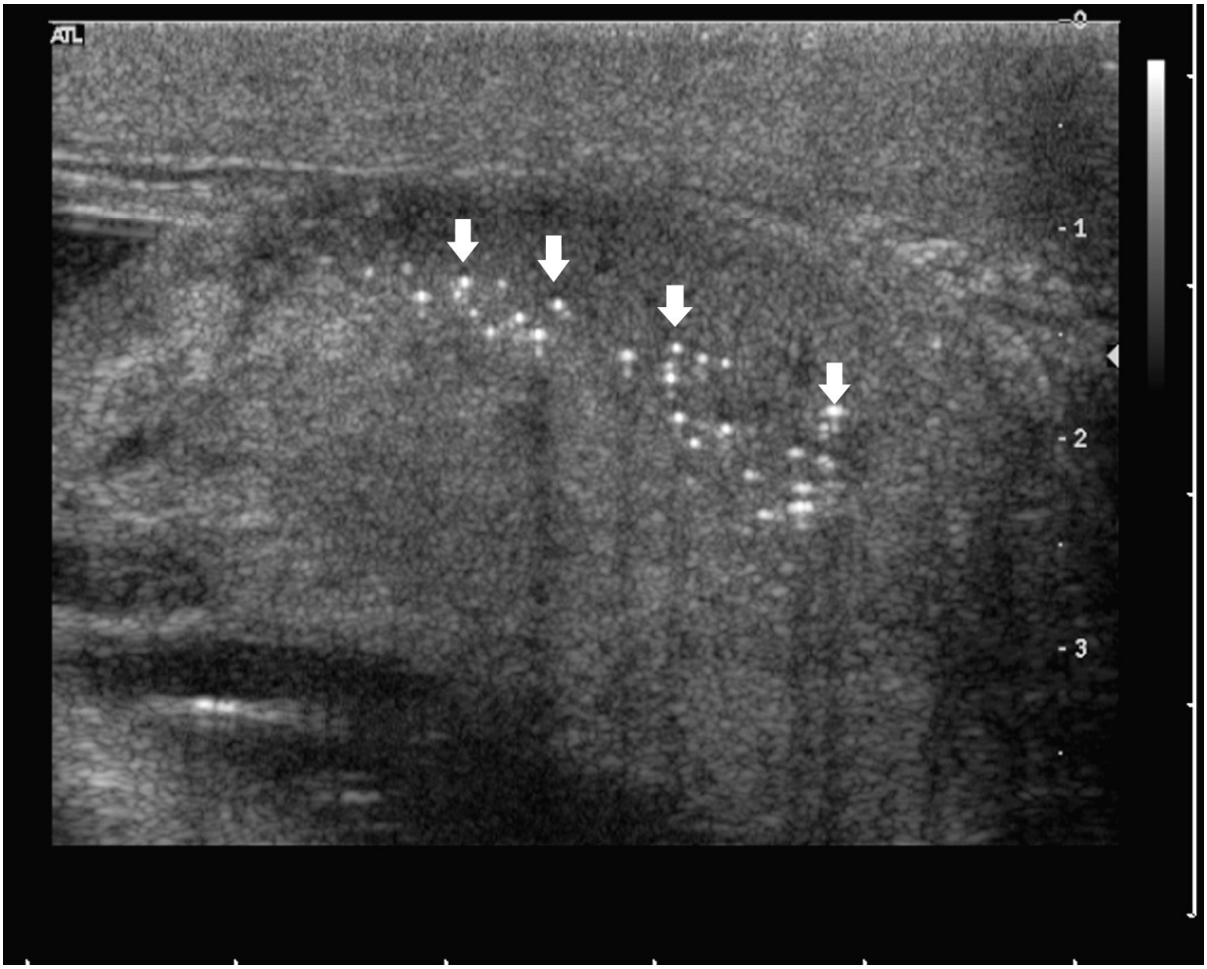
THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1222 ISSN 1175 8716

Lothario's Scrotum

Mustafa Secil

A 39-year-old man presented after a gunshot injury of the genital region traversing the left hemiscrotum. Scrotal sonography images demonstrate numerous air bubbles inside the left testis (Figure 1, arrows) indicating the track of the bullet fragment.

Figure 1



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Vol 118 No 1222 ISSN 1175 8716

How to manage new-onset epilepsy

When an individual presents for the first time with a single epileptic seizure or new-onset epilepsy they are usually fully investigated. More often than not brain imaging is negative. Bearing in mind that a single seizure is associated with an approximately 50% risk of recurrence the question arises—should this patient start antiepileptic medication now or should treatment be deferred until a second seizure occurs? Usually in New Zealand the watch and wait policy is adopted. Is there any real evidence to justify either position? Apparently not—hence a trial report (and commentary) in a recent Lancet are important. Over 1400 patients with a first seizure were randomised to immediate or deferred antiepileptic drug treatment. And the outcome—immediate antiepileptic drug treatment reduces the occurrence of seizures in the next 1–2 years, but does not affect long-term remission in individuals with single or infrequent seizures.

Will this trial change our tactics?

Lancet 2005;365:1985–6 & 2007–13

Clostridium difficile-associated diarrhea and probiotic therapy?

Clostridium difficile-associated diarrhea (CDAD) is a major problem in hospitals and indeed the community generally. Probiotics, or naturally occurring "good bacteria" have been suggested as a means of both preventing and treating the disease. The "good bacteria" include several lactobacilli, enterococci and also yeasts. But do they work? A meta-analysis of eight relevant trials revealed insufficient evidence for the routine clinical use of probiotics to prevent or treat CDAD. The researchers recommended that better designed and larger studies are needed. Methuselah recommends less inappropriate antibiotic usage.

CMAJ 2005;173:167–70

C reactive protein and infections of the lower respiratory tract

Clinicians in and out of hospital often have difficulty in differentiating viral and bacterial lower respiratory tract infections. This has a bearing on whether or not the patient is treated with antibiotics. It is widely believed that the serum C reactive protein, an acute phase reactant, can elucidate this matter. A group of Dutch sceptics have meta-analysed 17 studies which refute the hypothesis. And they conclude that C reactive protein is neither sufficiently sensitive to rule out nor sufficiently specific to rule in a bacterial aetiology of lower respiratory tract infection and the "evidence does not consistently and sufficiently support a wide introduction of C reactive protein as a rapid test to guide antibiotics prescription".

BMJ 2005;331:26–9

Clinical trials in the sub-continent

Indian life sciences featured in a recent edition of Nature. One article pointed out that multinational companies are flocking to India to launch their trials. Why? Well for a start "it's a lot cheaper to do things in India", says Sameer Deb, general manager of government affairs at GlaxoSmithKline's office in Mumbai. Furthermore, India has several advantages as a host for such trials. Its biggest asset is probably the size of its population at more than 1 billion. In addition, Indians are increasingly suffering from the same illnesses as Americans and Europeans—diseases for which companies are desperate to find cures. For instance, at least 70 million Indians suffer from heart disease and 35 million have diabetes. It also has the edge over most developing countries because of its sophisticated hospitals and because many of its medical personnel speak English.

On the other hand there are thorny ethical problems. For example, in 2003 private clinics across India used a generic version of the anticancer drug letrozole to treat more than 435 women with fertility problems. This trial did not have clearance from the health ministry and the women involved did not know that the drug was not approved for this use. The Indian Council of Medical Research is addressing these issues.

Nature 2005;436:485

"Standard" stroke management

A recent study from Queensland compares the management of stroke patients in four regional hospitals and a teaching hospital in Brisbane. It is no surprise that they found that patients treated at regional hospitals were significantly less likely to have a CT head scan within 24 hours of admission, carotid duplex or echocardiography, estimations of lipids and glucose, a swallow assessment, assessment by allied health professionals, or be prescribed DVT prevention strategies, compared to patients treated at metropolitan or larger hospitals. Not surprising because the regional hospital patients were in general medical wards in hospitals which were relatively under-resourced, whereas those in Brisbane were managed in a well resourced stroke unit with all the trimmings.

Turning to secondary prevention strategies showed a different story and there were no significant differences between hospitals in the use of antithrombotic therapies in ischaemic stroke/TIA patients surviving to discharge. Furthermore, the proportions of all stroke survivors receiving blood pressure lowering therapy, and ischaemic stroke/TIA survivors receiving ACE inhibitor or a statin therapy, were not different between the groups. And finally, the outcomes were no different.

Intern Med J 2005;35:447–50

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Proposed article for the New Zealand Medical Journal

I have been assisting a client in respect of her concerns arising out of the unauthorised use of her research material and the inclusion of that material in an article written by McKenna and MacLeod which is proposed for publication in NZMJ. You will have also noted that the questionnaire used by McKenna and MacLeod was adapted, without consent, from that designed and used by my client in 2001.

I note that you have passed on some of my client's concerns to the authors of the proposed article and as a result the authors have deleted Figure 4 and any mention of its data in the text.

You have advised that after studying the amended manuscript and associated correspondence, the editor of NZMJ considers the proposed article is now suitable for publication in either the August or September issue.

My client has not been given the opportunity to review the amended article so cannot make any specific comment. However she wishes to emphasise that she does not wish to be associated in any way with the article and assumes that references to her have been deleted.

My client is committed to the improvement of palliative care in New Zealand. She does not see the publication of a flawed article in the NZMJ as being in the best interests of palliative care generally. As noted above, my client has not seen the amended article so she does not know whether her concerns are well founded or not.

She suggests that the amended article be peer reviewed by an independent reviewer with recognised standing and expertise in palliative care and management of motor neurone disease.

My client is raising her concerns with NZMJ in a hope that whatever is published is accurate and a true reflection of palliative care in New Zealand. I would be happy to discuss this further with you.

Matthew F McClelland
Barrister
Wellington

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Mosquito-borne diseases in New Zealand: has there ever been an indigenously acquired infection?

Imported cases of a number of mosquito-borne diseases are regularly reported in New Zealand. The list of imported diseases includes malaria, yellow fever, dengue fever, Ross River virus, Barmah Forest virus, and Japanese encephalitis. There is, however, controversy regarding the previous occurrence or not of an indigenously acquired mosquito-borne infection in man in New Zealand. Claims seem to be made more frequently regarding human infection caused by Whataroa virus (*Togaviridae*: *Alphavirus*).[1]

Whataroa virus has been isolated from the endemic mosquitoes *Culiseta tonnoiri* (Edwards) and *Culex pervigilans* Bergroth in South Westland, and detailed descriptions of the ecology of this virus were published in the 1960s and 1970s,[2–5] in which the evidence suggested a bird-mosquito cycle.[3] Native and exotic bird species have been infected by Whataroa virus, but there is no evidence of illness or death amongst the birds studied indicating that the infection is clinically unapparent.[3]

Although Maguire et al suggested at the time that there was indication that Whataroa virus infected man,[2] Hogg et al seems to be the only publication ever to provide evidence of human infection with an arbovirus in New Zealand.[6] However, the evidence provided cannot be considered conclusive. Haemagglutination inhibition (HI) antibodies are only significant if a considerable rise (8–16 fold or greater) in titre from the acute to the convalescent phase of an illness a few weeks later can be shown.

This was not the case in Westland, and the titers identified by Hogg et al were relatively low.[6] In addition, the antibodies reacted with Group B antigens, but 10 years of subsequent work only led to the isolation of Whataroa virus in the area, which is Group A.

The low levels of antibody detected by HI methods in Westland are not sufficient to make the claim that any specific virus has been confirmed as being present in New Zealand, especially since HI tests are not specific for any one agent, but are broadly group-specific. The conclusive evidence would be the isolation of the agent, accompanied, if possible, by positive antibody results. Therefore, one cannot say with confidence that human infection with any arbovirus has ever occurred in Westland.

The only reliable evidence of a mosquito-borne infection acquired in New Zealand seems to be the case of a man infected with malaria in 1927.[7] The diagnosis was established beyond doubt, and since the patient had never been to a malarious country, had not left New Zealand for 13 years, and had not been ill before, the only possible conclusion was that infection occurred in the country, probably in Auckland.[7]

There are no records of a population of anopheline mosquitoes in New Zealand, and the known established species are very unlikely to be competent vectors of malaria (even though no studies have been done to test this assumption). The described infection appears to have been an incident of ‘seaport malaria’. Anophelines could have arrived in this country from Australia or Panama by ocean transport,[7] and since

numerous people regularly returned to New Zealand after contracting malaria abroad, transmission by a hitch-hiking mosquito was possible.

Nonetheless, we are yet to see a confirmed indigenously acquired infection in humans in New Zealand, in which a pathogen was transmitted by a female mosquito from a local vector population. Over many years, the Virus Research Unit at the University of Otago (Dunedin) carried out serological studies on some thousands of New Zealand human sera searching for evidence of local arbovirus infection, but the only positive results were obtained from people who had travelled overseas.

New Zealand can, therefore, still be considered a 'virgin soil' when it comes to mosquito-borne diseases.

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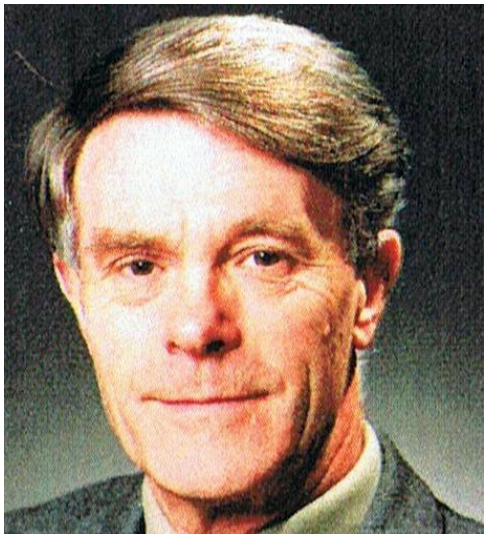
Vol 118 No 1222 ISSN 1175 8716

Alfred Philip Poole

Alf Poole, who will be remembered for his immense contribution to medicine, the community and the historical, cultural and sporting areas, died recently in Dunedin aged 83.



Dr Poole was born in Invercargill on January 15, 1922, to Philip and Ivy Poole. His father was the youngest son in the family timber merchants, George Poole and Sons. Alf was followed by a brother, Elman, and a sister, Ivy.

Alf's mother died shortly after Ivy's birth. Dr Poole was educated at Southland Boys High School where he took a keen interest in sport, particularly surf life-saving. He was a member of the school team that won the Otago and Southland high schools life-saving competition for three years in succession from 1938.

After graduating from the University of Otago Medical School in 1945, Dr Poole returned to Invercargill and Southland Hospital. There he met a nurse, Nan Brown, of Oamaru, who became his wife and who worked alongside him as his practice nurse throughout his career until his retirement in 1991. In 1954 they and their 3 year old daughter, Elizabeth, travelled to Edinburgh, where Dr Poole gained specialist medical qualifications and diplomas in anaesthetics, obstetrics and gynaecology. This made him a versatile and valuable asset to Southland Hospital on his return in 1957.

He has been described as a man before his time. As a physician with a special interest in cardiovascular diseases, he pioneered many techniques and treatments for his patients in Southland, including introducing a change in the rehabilitation of coronary care patients from full rest to exercise within their limits. He saw one of his major achievements as establishing the Southland Cardiac Club for former patients and their families, with monthly meetings that included medical rehabilitation, craft activities (he taught wool spinning) and lectures from visiting specialists. His skill in enlisting others to the cause saw the establishment of the Southland Hospital coronary-care unit, the Southland Medical Foundation, and a mobile cardiac ambulance service.

Fiercely defensive of specialist medical services in Southland and Otago, he spoke out passionately in 1990 in support of his Dunedin colleagues for the retention of the Dunedin Hospital cardiology unit, at a time when there was considerable political pressure for it to close. For the University of Otago Medical School, he was an enthusiastic honorary lecturer in clinical medicine, keen to pass on his ideas and new techniques to senior medical students visiting Southland Hospital.

In 1971, his considerable professional achievements saw him made a Fellow of the Royal College of Physicians in Edinburgh, and in 1973 of the Royal Australasian College of Physicians.

He served for nearly 40 years as honorary medical officer for the Southland Swimming Association, the Royal Life Saving Society, and the Oreti Surf Life-Saving Society. He was also the honorary medical officer to the New Zealand swimming team at the Commonwealth Games in Christchurch in 1974.

Another long association with young people with through the YMCA, where he was a trust board member for 24 years, steering the board and several committees as chairman.

Dr Poole also found time to turn his energies to pressing community issues. He was incensed by property developments in the early 1970s when buildings of historical value and importance were torn down.

He was the founding chairman of the Southland branch of the New Zealand Historic Places Trust in 1973, remaining chairman until retirement. Many of the Invercargill buildings regarded today as historically significant owe their survival to his efforts. He took the Southland Times Hindsight Award, Southlander of the Year, in 1979 for these achievements.

Dr Poole did not confine his interest to buildings. In 1975, he joined the Southland Museum and Art Gallery and was immediately elected chairman at a time when the museum's viability was uncertain. He, along with the then director of the museum, Russell Beck, initiated and implemented much needed changes, turning the museum into a lively, thriving hub. He would not be beaten by a challenge. At the museum, the tuatara were not reproducing. He introduced the technique of artificial induction of eggs, which significantly boosted the population.

Dr Poole also took an avid interest in establishing an ongoing acquisition policy for the gallery's art collection. The Trustbank Southland Art Foundation owes its existence to his initiative, as does the redevelopment of the museum gallery pyramid complex completed in 1990. A generous donation from Dr and Mrs Poole provided for the completion of the museum's library and administration facilities, as well as a much needed café which opened a few years after his retirement from the chair.

In 1993, Dr Poole received a CBE for services to medicine and the community. Though his honesty and sharp logic led him to not suffer fools lightly, his sense of humour was appreciated by all who knew him.

He is survived by his wife and daughters.

We are grateful to Mrs Poole for allowing publication of this shortened version of the obituary which appeared in the Otago Daily Times on 20 August 2005. We also thank Dr David Pottinger (Invercargill) for the photograph and coordination.

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Andrew James Ogilvy

Dr Andy Ogilvy fell tragically to his death while descending Mt Barth in the Ahuriri Valley on the 17th of April 2005, devastating friends and family both here and in his native England. He was 41.

Andy was employed as a Consultant Anaesthetist and Intensive Care Specialist for the Otago District Health Board at Dunedin Hospital since October 2002.

Andy grew up on Merseyside and later studied Medicine in Leeds, UK, then pursued a career in Anaesthesia working as a Senior Registrar and Lecturer in the University of Leicester Department of Anaesthesia, and then Consultant in Anaesthesia and Intensive Care Medicine at Leicester General Hospital.

Andy was hugely popular as an academically gifted and caring doctor with a particular flair for teaching. His talent for explaining difficult concepts in novel ways and with tremendous enthusiasm was genuinely inspirational for many medical students and postgraduate trainees who had the good fortune to be taught by him. He was a keen faculty member of the CCrISP (Care of the Critically Ill Surgical Patient) Course both in Leicester and here in New Zealand and became a key teacher in the Aeromedical Evacuation course run in Otago each year.

Andy will be most remembered though for his boundless appetite for life and his infectious smile. At work, Andy always pretended that things were dreadful, but his actions showed otherwise. He became an integral member of the Emergency Retrieval Service. He was a hugely respected clinician, astute and very well read, yet quite down-to earth and humble. He made decisions calmly and with authority when they were needed and he led by example. Above all, he was an extremely funny man, and could lighten even the most dire situation in the Intensive Care Unit with a dry quip or a good dose of black humour.

His many interests included golf, playing the guitar badly, and the great outdoors.

As a climber, he was renowned for his concern for others and his meticulous safety standards. For him to slip and fall as he did came as an unbelievable shock.

In just two years here in Otago, Andy Ogilvy became so much a part of this Intensive Care Service, our hospital, and our community that he seemed to have been here for much much longer. He is survived by his wife Jenny, his mother, and two sisters.

Mr Mike Hunter (Dunedin) and Dr Andrew Fox (Leicester, UK) wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 118 No 1223 ISSN 1175 8716

The 17th Hospice NZ Palliative Care and NZ Pain Society Conference: Making a Difference

The 17th Hospice NZ Palliative Care and NZ Pain Society Conference: Making a Difference will be held in October next year in Dunedin, New Zealand. The conference is scheduled for October 26–28, 2006.

For more details, contact event project manager Barry Woodland at Conference Innovators, phone +64 (0)3 379 0390 or email barry@conference.co.nz

To register your interest in presenting, please contact:

- David Jones, New Zealand Pain Society, at davidjones@healthotago.co.nz, or
- Simon Allan, Hospice NZ clinical medical adviser, Arohanui Hospice medical director and Palmerston North Hospital Regional Cancer Treatment Service clinical director, at Simon.Allan@midcentral.co.nz