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Statesmanship in the health service

The Editors

“It is imagination which allows us to drag our intellect out of its self-referential tendencies, just as it is ethics which helps us to stay away from logical truths which are profoundly destructive. And it is the shared knowledge of common sense which protects us against intellectual nonsense. And the context and shape of memory which can help to steer us away from the ideological certainty which convinces us we can cut free from all that exists and do something else. These qualities drag our reason onto fertile ground and keep it away from the isolating delusions of purity and instrumentalism.”

JR Saul.1

Over the last three years many of our editorials have addressed problems that have developed in New Zealand’s health service. The unnecessary deaths in Christchurch and from cervical cancer, tragic though they were, have not been the dominant problem. It has been the subjection of the service to centralised financial ideology and autocratic corporate management. These have subjugated the imagination of staff, ethics, common-sense and professional and institutional memory, leading to the outcomes which characterise their absence - as Saul (above) points out. Take for example the financial decision to withdraw funding for several statins forcing thousands of patients to change medication. Lack of prospective measurement means that the morbidity and mortality and the cost of hospital admissions consequent on that one administrative sleight-of-hand will never be known. To change medication of well controlled patients without medical reason was contrary to good medical practice and was therefore unethical.

A recent editorial from Auckland2 along with earlier editorials serves to emphasise the systemic issues that have damaged the morale and effectiveness of services throughout the country. This has been an inevitable result of the breakdown of communication and cooperation between clinical professionals and corporate management. Serious difficulties began with the change to general management which imposed pyramidal authoritarian control and a funder-provider split that resulted in management decisions being driven, directed and controlled by the financial imperative. As Charlton 3 pointed out in the UK: “Of course, all organisations must be run on lines of proper financial probity, and it is trivially obvious that financial considerations cannot be allowed to dictate the fundamental nature of health services - especially not the specifics of clinical care.” In New Zealand, although contracts were negotiated with the Health Funding Authority, direct interference in the provision of services by Treasury, initially via CCMAU, led to aggressive business plans which caused a decline in standards of service.4

It is now important that health professionals are more effectively involved in planning and the development of policy. The real issue is how that is best achieved. We have argued that the medical input should be provided by doctors elected to management
positions by their peers for a limited period. The formation of a clinical board would facilitate their effectiveness. This approach would ensure that ideas of health professionals are heard and bad news is presented early -not dismissed or delayed. A limited tenure will foster fresh approaches, reflect a wider body of opinion and minimise the risk of divided allegiance or self-seeking.

Some consider it would be useful for some permanent managers to have a medical background. Restructuring plans to incorporate a few health professionals more firmly into the administration have appeared. Actually, this will strengthen linear command and control and is a structural answer to a situation that really requires an attitudinal shift.

Better democratic input and locally tailored, more experimental approaches are needed, using test situations to determine feasibility and effectiveness. Pilot studies of genuine peripheral budgeting in some units would be a useful start.

The relationships between the manager, doctor and nurse require a spirit of co-operation which seeks the best for patients. Whilst the duty of providing fiscal advice and maintaining financial records might rest with the manager, and clinical planning and consultation with the doctor and nurse, all decisions of policy should be reached by consensus. We need to change to a more cooperative environment and reverse the trend of clinicians, nurses, allied health professionals, technicians and medical scientists retiring early, going overseas or leaving the profession.

The responsibility lies firmly and directly with the Minister of Health, who is accountable for the appointment of some members of the Boards and for the way in which our Health Boards function. Statesmanship is required. The Minister needs to ensure that the attitudes of administrative personnel and the systems in which they work promote a cooperative environment.

Financially driven ideology and authoritarian corporate management have damaged the trust between individuals and the effectiveness of clinical teams (including service managers). They have also severely damaged effective planning. Democratic input, along with common-sense and empathy for the common good are needed. It is vital that management within our hospitals and District Health Services embraces these broader characteristics. A genuine partnership with staff would be a good beginning.

References:

A re-appraisal of the burden of infectious disease in New Zealand: aggregate estimates of morbidity and mortality

Clair F Mills, Martin Tobias, Michael Baker.

Abstract

Aim To assess the aggregate burden of infectious disease in New Zealand in terms of mortality and hospital admissions.

Methods New Zealand mortality records for the years 1980-1998, and hospital discharges for the period 1988-2000, were re-analysed using a recoding of ICD-9 codes to estimate the aggregate burden of infectious disease. The recoding scheme was modified, as in an earlier analysis, from that developed by Centers for Disease Control and Prevention.

Results Following recoding, the proportion of deaths attributable to infectious disease increased from 0.7% of deaths to 6.6% of deaths. Likewise recoding of hospital discharges showed an increase in the proportion due to infectious disease from 2.2% to 12.6%, second only to “complications of pregnancy, childbirth and the puerperium”. Over the study period infectious disease mortality rates have showed little decline, and there has been a nearly 60% increase in infectious disease hospital discharge rates.

Conclusions The findings confirm and extend those of an earlier study, indicating the substantial burden of disease that is still attributable to infectious disease in New Zealand. The burden remains inequitable.

The belief that the infectious disease burden in industrialised countries could be eliminated has been challenged in the last two decades by the emergence of new diseases, and the re-emergence of diseases thought to be controlled. However, there have been few assessments of the total burden of infectious disease in developed countries and attention has focused on individual diseases rather than infectious disease as a whole.

Following the initial analysis of mortality data carried out by Christie and Tobias covering the period 1980-1993,1 we updated the estimate of the aggregate burden of disease in New Zealand in terms of mortality over the period 1980-1998, and also calculated it in terms of hospital discharges for the period 1988-2000.

In New Zealand, rates of some infectious diseases continue to remain high for a developed country, and there are also large inequities in the distribution of this burden.2 An analysis of the aggregate burden can assist in increasing the focus on infectious disease as a major public health problem, and identify the priorities, the resources allocated and the prevention and control measures necessary to reduce this burden. This analysis contributed to the development of the “Integrated Approach to Infectious Disease: Priorities for Action 2002-6”, a national five-year plan that identifies priority actions for infectious disease control in New Zealand.3
Methods

Mortality data. Mortality records were obtained from the New Zealand Health Information Service (NZHIS) for the period 1980-1998. These records are derived from death certificates registered each year, supplemented by post mortem reports, coronial reports, hospital separations data and other death investigations when available. Coders assign an “underlying cause of death” using standard algorithms set out in the International Classification of Diseases (ICD-9-CM) coding manual. We have restricted our analysis to the single underlying cause of death, as did the previous study. This is likely to underestimate the contribution of infectious diseases to fatal outcomes.

Hospital discharges data. Hospital discharge data were obtained from NZHIS for years 1988-2000. The data on hospitalisations are based on inpatient discharges from public hospitals and publicly funded discharges from private hospitals. No distinction is made between discharges from a single admission or a readmission, and no account is taken of length of stay.

ICD recoding scheme. The conventional ICD coding for “Infectious and parasitic diseases” excludes many infections that are coded under organ systems. For example, bacterial meningitis is coded under Chapter Six (Diseases of the Nervous System and Sense Organs), while pneumonias and respiratory infections are coded under Chapter 8 (Diseases of the Respiratory system). An analysis of the impact of infectious disease requires recoding the ICD codes to account for this factor.

To recode the mortality and hospital discharge data we used the ICD-9 recoding scheme initially developed by the US Centers for Disease Control and Prevention (US-CDC) with minor modifications. The US-CDC scheme involved examination of each of the ICD-9 codes by a panel of experts, comprising infectious disease physicians, internists, public health physicians and disease coding experts. Codes were reclassified into the following categories:

A: an infectious disease
B: possibly an infectious disease
C: late effect of an infectious disease
D: possibly a late effect of an infectious disease
E: not an infectious disease
F: unknown aetiology
G: consequence of treatment or prophylaxis for an infectious disease

Each code reclassified into A, B, C, D and G was further subcategorised depending on whether (a) all or (b) only a proportion of the code was considered to have an infectious aetiology. For example, meningococcal meningitis is coded as an Aa, while rheumatic heart disease is coded as Ca.

In this analysis, only the deaths or discharges recoded to Aa, Ca and Ga were included, that is, those that were an infectious disease, the late effect of an infectious disease or the consequence of treatment or prophylaxis for an infectious disease, in all cases.

New Zealand modifications. Some modifications were made, as in the earlier analysis, to the US-CDC recoding scheme to reflect coding practices and disease epidemiology in New Zealand:

- over 80% of primary hepatocellular carcinoma in New Zealand is thought to result from hepatitis B infection. All these deaths/discharges were recoded to category Ca.
- the evidence for the causal role of HPV in cervical dysplasia and cervical cancer is sufficient to recode cervical cancer deaths and discharges related to cervical dysplasia and cancer as Ca.
- deaths and discharges due to “meningitis (unspecified)”, “acute myocarditis (unspecified)” and “encephalitis (unspecified)” are considered to represent infectious aetiologies in greater than 80% of cases in New Zealand. These deaths and discharges were recoded to category Aa.

Evidence for assigning an infectious aetiology to gastritis (Helicobacter pylori) and coronary artery disease (chlamydia pneumoniae) was judged insufficient to include these diseases in an infectious disease category.

A further sub-categorisation was made to those coded Aa, Ca, and Ga, based on the major route of transmission or control of that disease or intervention. This was based on the disease groupings identified for development of the Integrated Approach to Infectious Disease. Those with multiple transmission routes were assigned the route considered to be dominant in the New Zealand setting. Local estimates of the proportion of disease attributable to particular modes of transmission have been made for some categories, such as food-borne illness. However, many hospital discharges could not be assigned a specific code due to inadequate information from ICD-9 coding data eg non-specific abscesses and infections.

Data analysis. Data were extracted from the mortality and hospital discharge databases and analysed using SAS software. Age standardisation of rates was carried out by the direct method (using Segi’s
world population\textsuperscript{12} as the standard). Age specific rates were calculated for some specific ‘indicator’
diseases.

\section*{Results}

\textbf{Aggregate analysis of mortality.} Over the period 1980-1998, 510 994 deaths were
recorded on the mortality database. ICD-9 Chapter One ("Infectious and parasitic
diseases") accounted for 0.68\% of this total, ranking 13\textsuperscript{th} by single cause of death
analysis.

Following recoding with the modified US-CDC scheme, infectious disease was
estimated to account for 6.6\% of total deaths, thus increasing by nearly tenfold the
proportion of deaths attributed to infectious aetiology over the study period. Infectious
disease deaths then become the 4\textsuperscript{th} leading single cause of death behind diseases of
the circulatory system, neoplasms and deaths due to injury and poisoning (Figure 1).

\textbf{Figure 1. Deaths by cause, recoded by modified US CDC scheme:}
1980-98.

\textbf{Aggregate analysis of hospital discharges.} Over the period 1988-2000, 5 984 086
discharges were recorded on the hospital discharge database. ICD-9 Chapter One
("Infectious and parasitic diseases") accounted for 2.2\% of discharges in this period.
Recoding increased that proportion to 12.6\%, thus increasing by nearly six-fold the
proportion of hospitalisations attributable to infectious aetiology over the study period
(Figure 2).

\textbf{Age standardised death rates.} Death rates from infectious disease showed little
decline over the period, with a more marked fall seen from 1997 (Figure 3). Given
greater accuracy of ethnicity data for the 1996-1998 period only, Maori and ‘all other’
ethnic group rates showed similar trends- ie there was no reduction in inequalities
between the two.

\textbf{Age standardised hospital discharge rates.} Hospital discharge age standardised
rates showed a very different picture from mortality rates, with hospital rates for
infectious disease rising from 1537/100,000 to 2417/100,000 (an increase of nearly 60%) in the period 1988-2000. Hospital discharges overall increased dramatically in this period rising from 412,039 in 1988-9 to 603,940 in 1999-2000 - a factor of 1.46; infectious disease discharges rose even more steeply (from 45,081 in 1988-9 to 84,115 in 1999-2000 - a factor of 1.86).

Figure 2. Hospital discharges by cause, recorded by CDC ID analysis, 1988-2000.
Sub group analysis

**Age.** Overall, the greatest proportion of deaths, as would be expected (over 70%) was in elderly Pakeha (aged 65 years and over). Hospital discharges showed an inverse relationship - with 29.3% of infectious disease-related discharges in children aged less than five years, and the bulk in people aged less than 40 years. Overall, under-5 year infectious disease hospital discharges accounted for 3.7% of all hospital discharges.

**Sex.** There was a slightly higher rate of infectious disease mortality and hospital discharges in males for all ethnic groups in all years. A marked sex difference was seen in hospitalisations for sexually transmitted infections (STIs) and their latent effects (ectopic pregnancy, pelvic inflammatory disease (PID), cervical dysplasia and neoplasia) where rates in women were over ten times those for men.

**Ethnicity.** Data quality relating to ethnicity in New Zealand has been problematic, especially for mortality data. Coding practice changed in 1995 for death certificates, so the mortality data were analysed here with respect to ethnicity only since that time. Hospital discharge ethnicity data has been based on self-reported ethnicity for a longer period and is likely to be a more accurate reflection of trends in infectious diseases between ethnic groups. However there are still limitations in the data quality. Despite those constraints, it is evident that there remain unacceptably large gaps between rates of infectious disease in Maori and Pacific people compared with other New Zealanders (Figure 4).
Young Maori under 5 years accounted for 8.1% of all infectious disease hospital discharges; Pacific children accounted for 3.7% and Pakeha, 17.5%.

**Route of transmission.** Only 41% of hospital discharges and 84% of deaths could be classified by transmission mode according to our schema from their ICD-9 coding, due to inadequate detail in coding information. Respiratory infections accounted for the largest group of infectious diseases by defined transmission route, both in terms of morbidity (31.3% of hospital discharges) and mortality (67.8% of all deaths). However, there were differences by age group, with elderly Pakeha contributing the largest proportion in respiratory infectious disease mortality, while the under-five age group were a significant proportion in terms of hospital discharges.

Sexually transmitted infections and their sequelae (predominantly in women) and diseases transmitted by close physical contact were the other dominant groupings in the defined groupings of hospital discharges. Blood-borne infections and vaccine-preventable diseases in adults accounted for 3.6% and 2.9% respectively of infectious disease deaths.

**Acute versus latent burden of disease.** The long-term impact of infectious disease is reflected by the 10% of infectious disease deaths that are due to the late effects of infectious disease. For mortality, this effect was greatest in those aged 45 to 80 years, and for females, largely due to the impact of cervical cancer. The late effects of infectious disease morbidity were seen in young and mid-life women (aged 20-50 years), related to pelvic inflammatory disease and cervical dysplasia as noted above.

**Discussion**

Re-analysis of data based on the modified US-CDC recoding scheme clearly demonstrates the continuing burden of infectious disease in terms of morbidity and mortality in New Zealand. The re-coding system used is conservative, thus underestimating the contribution of infectious disease to the total. It is notable that while the burden of non-infectious disease is now decreasing in some cases (eg coronary
heart disease), there is no similar fall in infectious disease rates and the burden remains inequitably distributed (affecting the young and old, and Maori and Pacific peoples disproportionately). Given the problems associated with quality of coding of ethnicity, this analysis cannot give a reliable time trend for inequalities between Maori, Pacific peoples and other New Zealanders, but only a snapshot of 1996-98. However the coding improvements in place since 1996 will form the basis for future time trend analysis.

The disaggregation attempted here by transmission mode, given the information available from ICD codes, is limited, although it is clear that respiratory infections and sequelae of STIs contribute a significant proportion to the total burden of infectious disease in terms of hospital morbidity and mortality. Rates of admission for respiratory disease in children in New Zealand are known to be higher than in Australia, for example.\textsuperscript{13} This difference may reflect both higher risk factor exposure (over-crowding and poor housing,\textsuperscript{14} poverty, smoking exposure) but also other factors such as inadequate access to primary health care (research suggests, for example, that Maori and Pacific peoples are not accessing primary care services at rates consistent with the levels of increased health need seen in the community\textsuperscript{15,16}). The high rates of cellulitis and other causes of ‘avoidable’ hospitalisations may reflect similar factors. Hospitalisation rates are known to increase with increasing deprivation at all ages, for both sexes. New Zealand data shows this gradient is particularly steep for infectious disease admissions.\textsuperscript{17}

The rising trend for hospitalisations, with a near doubling between 1988 and 1999, is a national trend. This increase can be explained in part by changing demographics, changes in admission criteria and length of stay, and possible access problems in primary care, but a true rise in ill-health can not be excluded. The rise in infectious disease hospitalisations is even more pronounced in the recoded data. This is in comparison to mortality data, with deaths attributable to infectious disease stable or declining slightly between 1980-1996, with a further fall from 1996. This last decline is almost all due to a fall in mortality from infectious respiratory diseases and is likely to be largely an artefact of changes in coding practice (there was a small fall in overall mortality in 1998). In 1996, NZHIS rewrote the “Guide to Writing Death Certificates” which requested re-classification of primary or underlying cause of death (personal communication, NZHIS, June 2001). This resulted in an increase in classifying chronic diseases including dementia and Parkinson’s disease as the primary cause of death, and a reduction in pneumonias and ‘ill-defined’ causes. As mortality data are not yet available for 1999-2000, it is not certain whether this pattern has continued.

This analysis supports the need for development and implementation of a cohesive national policy to address the infectious disease burden in New Zealand, signalling as it does that there are still substantial improvements to be made. This analysis could be usefully repeated at regular intervals as a monitoring tool for measurement of strategic plans addressing infectious disease control in New Zealand. Further work could also be carried out to refine the system used for assigning diseases to their predominant mode of transmission, possibly to allow for the multiple modes of transmission that apply to many infectious diseases.

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Appendiceal diverticulitis and actinomycosis

Richard Flint.

Diverticular disease of the appendix and actinomycosis were first described over 100 years ago but were long disregarded as a cause of disease. Appendiceal diverticulosis was seen as a variant of true appendicitis whereas actinomycosis was considered a contaminant of other infections. Here we describe a case of actinomyces causing appendiceal diverticulitis.

Case Report

A 21 year old Caucasian male presented to hospital with a two day history of right iliac fossa pain. This pain was peritonitic in nature, without associated gastrointestinal symptoms. He admitted to having similar episodes over the last six months. He was afebrile but had guarding in the right iliac fossa. Laboratory investigations included a white cell count of 14.9 x 10⁹/L.

Surgery was performed to remove an inflamed appendix. Histology revealed a small, acutely inflamed diverticulum at the tip. Within the lumen of the diverticulum there was actinomyces within an inflammatory exudate. Peritoneal swab culture taken at the time of operation did not grow any organisms.

The patient was discharged the next day without antibiotics. Five days later he presented again with a fever of 38.6 °C, low abdominal tenderness, and a white cell count of 21 x 10⁹/L. A CT scan revealed an inflammatory mass in the pelvis. The patient was treated with intravenous cefuroxime and metronidazole and discharged two days later to complete a course of oral antibiotics. There were no sequelae.

Discussion

Acquired appendiceal diverticula lack a complete layer of muscularis in their walls. They are discovered usually at surgery, either during appendicectomy or at an unrelated laparotomy. Occasionally they are found at barium enema. Up to 2.1% of appendicectomy specimens have diverticula, and two thirds of these show signs of inflammation. Appendiceal diverticulitis has four times the risk of perforating than true appendicitis, and perforation occurs early in the course of the illness.

Abdominal actinomycosis is caused by gut commensal gram positive, anaerobic, filamentous bacteria of the genus actinomyces. It can occur at any level of the gastrointestinal tract but 65% of infections involve the ileocaecal valve and appendix. Disease may be spontaneous but most commonly follows appendicectomy. Multiple sites can be affected and intra-abdominal fistulae often result.

As in this case, both abdominal actinomycosis and appendiceal diverticulitis are commonly overlooked at initial presentation. Less than 10% of abdominal actinomycosis and even less of appendiceal diverticulitis are diagnosed preoperatively. This is because they mimic other more common conditions. They can simulate appendicitis, or produce a right iliac fossa mass that may be mistaken for an
appendiceal abscess, neoplasm, Crohn's disease, tuberculosis, helminthoma or amoebiosis. Clues at presentation are symptoms lasting weeks to months, as these are typical of both appendiceal diverticulitis and actinomycosis. Other features include right iliac fossa pain that has not migrated from the periumbilical region, and the absence of anorexia and nausea.

The diagnosis is also difficult to make postoperatively. Appendiceal diverticulitis is often overlooked unless the pathologist specifically searches for it in the appendix specimen. Likewise, actinomycosis can easily be missed as the characteristic sulphur granules are not present in 50% of cases. Fortunately the preoperative diagnosis of appendiceal diverticulitis is rarely necessary as suspicion of true appendicitis leads to early appendicectomy. Difficulty arises when the non-inflamed diverticulum is found incidentally because the risk of diverticulitis is unknown. It would be reasonable to avoid appendicectomy (even though inflammation will lead to a high rate of rapid perforation) because those asymptomatic diverticula discovered radiologically have rarely led to complications. Actinomycosis causing appendicitis can be treated by appendicectomy alone. As in this case, residual infection is effectively treated by antibiotics.

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References:

Capitation funding of primary care services: principles and prospects

Peter Crampton, Frances Sutton, Jon Foley.

Aims

This paper has three aims. First, it aims to inform readers of the forthcoming implementation of capitation funding for primary health organisations (PHOs). Second, the paper describes the factors that are likely to be included in the capitation funding formula, as well as their rationale for inclusion. Third, the paper raises for discussion some key unresolved issues related to capitation funding of PHOs.

Background

The history of capitation funding of primary care services in New Zealand dates back to the first Labour Government. In 1941 the government implemented a patient medical benefits scheme (with its Social Security Amendment Act 1941), that equated to a subsidy scheme for general practitioner (GP) services. Only about 51 GPs ever opted for capitation funding under the original scheme, the vast majority opting for fee-for-service payments. The number of patients on the combined capitation lists did not exceed 80 000. By 1948 there were only about 23 GPs operating under capitation funding, and by 1949 this had dropped to eighteen.

A group of enthusiasts at Otumoetai Health Centre generated renewed interest in capitation funding when it was introduced there, and subsequently evaluated, in 1979. Moves to capitation funding of individual general practices accelerated following the health reforms introduced in 1993. As a result of these policies by 1997 about 15.1% of GPs were on capitation funding (whereby they received capitiated government subsidies for eligible patients, but retained the right to charge fee-for-service co-payments), with regional rates varying from 4.8% to 45% of GPs using capitation funding. By the end of 2001, it is estimated that nationally 22% of GPs were on capitation funding.

The 1999 Labour Government’s Primary Health Care Strategy has added new impetus to the trend towards capitation funding by signalling ‘needs-based funding for population care’ to replace fee-for-service funding approaches. The intention of the new strategy is to encourage population-based approaches (as well as a focus on individual care), provision of coordinated and comprehensive services for enrolled populations, multidisciplinary team-based approaches, and increased community involvement in governance. While the emphasis of this paper is on capitation funding of PHOs, similar principles can be applied to capitation funding of individual general practices (although the Primary Health Care Strategy is not prescriptive on this point).
The formula

Objectives of the formula. The development of a nationally-consistent capitation funding formula was initiated by the Health Funding Authority (HFA) in 1999. One of the primary objectives was to develop a formula that could replace the plethora of capitation formulae in use at the time. These formulae included needs-based formulae that applied to the general medical services benefit (GMS) and practice nurse subsidies, and which included community services card (CSC) and high use health card (HUHC). These needs-based formulae were in use predominantly in the Midland and Central regions, while historical or block funding approaches based on an organisation’s historical fee-for-service (GMS) expenditures were in use mainly in the Northern and Southern regions. The HFA and others in the primary care sector were concerned about the inequitable distribution of primary care funding across the country through use of varying formulae.

A second objective was to better target resources to communities with high health need. The CSC, eligibility for which is based on family income and family size, is a crude barometer of socio-economic status. (Also, the inconsistent uptake of the CSC — as low as 72% of eligible people in some high need communities — further hampers its use as an indicator of need.) The New Zealand deprivation index (NZDep), which is a small area measure based on nine aspects of socioeconomic deprivation, is thought to be a more refined indicator of health need for populations than CSC status. In addition, ethnicity, which is closely associated with health need, is another measure proposed for targeting health resources. A third objective was to promote enhanced roles for nurses and other members of the primary care team. Under fee-for-service, a practice is only eligible for the GMS subsidy if a GP provides direct services to the patient. In many circumstances (eg, health education) appropriately trained nurses are able to provide the needed services, thereby freeing the GP to handle more complex cases and improve the efficiency of service delivery.

A fourth objective was to promote the management of populations over a period of time (as opposed to episodic care for individuals). Payment based on enrolled members requires that PHOs gain a better understanding of those whom they are treating. This ‘denominator management’ facilitates measurement of service coverage rates for enrolled persons with high health need (eg, diabetics) and uptake of age-related preventive services for all enrolled persons (eg, childhood immunisations).

These four objectives — equitable funding, targeting high health need, primary care team approach, and denominator management — are key underpinnings of the Primary Health Care Strategy, which was approved by Cabinet in December, 2000. Table 1 shows the PHO services that are presently planned to be funded through capitation.

Main variables in the formula. Several families of formulae were modelled. They differ in how socioeconomic status is incorporated, with the CSC family using CSC status, the Dep family using NZDep96 (the deprivation index), and the CSC/Dep family using both.

Each formula is defined by a set of per-head dollar amounts. The CSC model, for example, consists of 48 per head amounts for each of the four services. The 48 cells arise from all combinations of the categorical variables: age, sex, CSC and HUHC status.
Table 1. Services that may be funded through capitation.*

<table>
<thead>
<tr>
<th>Services</th>
<th>Key variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Medical Services (GMS)</td>
<td>Age, gender, CSC, HUHC, NZDep and ethnicity</td>
</tr>
<tr>
<td>Practice Nurse Services</td>
<td></td>
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<tr>
<td>Population-based health promotion</td>
<td></td>
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<tr>
<td>Services to improve access for high need</td>
<td></td>
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<tr>
<td>PHO management support</td>
<td></td>
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<tr>
<td>Referred services management (laboratory and pharmaceuticals)</td>
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*The services in italics are new services which are required of any organisation designated as a primary health organisation (PHO). PHO management support includes funds to: implement enrolment; meet reporting requirements; manage patient registers; and undertake referred services management. Though PHOs are required to undertake some form of referred services management, use of a need-based population formula for budget setting will not be required in the initial stages of PHO development.

CSC = community services card; HUHC = high use health card; NZ Dep = New Zealand deprivation index.

All the formulae include age and sex among their categorical variables. Age is of fundamental importance in a capitation formula because the per head annual cost of services varies so much across age groups. For example, in all the formulae, the average pharmaceutical per head cost for age 65+ years is 16 times the value for age 5-14 years. Omitting age from the formulae would therefore tend to disadvantage PHOs with older populations. The case for including sex in the formulae is weaker, as the age-specific cost-per-head differentials are lower. For example, the female-to-male ratios of per-head pharmaceutical cost vary from 0.9 for age 0-4 years to 3.5 for age 15-24 years. Nevertheless, the size of these ratios, together with the fact that the sex structures of PHO populations differ somewhat, makes the case for including sex in the capitation formulae.

All the formulae also include HUHC. Including this variable reduces the risk of creating perverse incentives against PHOs taking on patients with expensive chronic conditions. The additional amount for an HUHC holder is substantial. For pharmaceuticals, for example, the formula awards six times more per head, on average, for an HUHC holder than a non-holder. (This average is the population-weighted average of ratios of the HUHC per head cost to the non-HUHC per head cost; the cost rates are age-, sex- and CSC-specific).

Socioeconomic status is a variable in all the formulae. It is already a factor in subsidy arrangements, through the CSC. The CSC family of formulae, which comprises a single formula, attempts to reflect current national subsidy arrangements and utilisation rates. It thus reflects the higher levels of age-and sex-specific per head costs incurred by CSC-holders. Bringing in NZDep, either in addition to CSC or alone, allows for greater targeting towards lower socioeconomic status. Within the Dep and CSC/Dep families of formulae, the individual formulae differ in the steepness of targeting. Targeting tends to be steeper in the Dep family than in the CSC/Dep family, as in the latter CSC handles some of the targeting.

Although Maori and Pacific Island populations experience relatively poor health status even after controlling for socioeconomic position, the empirical evidence suggests the primary care utilisation and expenditures for Maori and Pacific Island populations are similar to or below those for other New Zealanders. To address this disparity, a Maori/Pacific Island weighting is proposed for the formulae. The extra
funds derived from this weighting are to be used for services that improve access for these populations.

Key unresolved issues

While there is an increasing trend towards capitation funding in New Zealand, there is attendant controversy and debate. It should be noted that in New Zealand capitation funded practices currently retain the right to charge fee-for-service co-payments. Even when co-payments are held constant, fee-for-service and capitation subsidy arrangements are likely to be associated with different patterns of utilisation. For example, fee-for-service funding mechanisms may encourage increased utilisation, and capitation mechanisms may encourage reduced utilisation. However, it is not possible to predict what changes may occur to utilisation rates given the complex mix of incentives associated with New Zealand’s hybrid mix of capitated and fee-for-service state benefits (for eligible patients), and fee-for-service patient co-payments.

Doctors are forced to charge some level of co-payment because the amount of the government subsidy (with the possible exception of the under six subsidy of $32.50) does not cover their costs. For example, a doctor serving a CSC-holding adult is able to claim only $15 in government subsidy per consultation, whereas the true cost of the consultation is probably $35 -$40. Capitation rates based on current subsidy levels will not cover this gap (assuming that the patients seek care consistent with the national average). As a result of the Community Services Card Review, conducted from August through October 2001, the government is considering raising subsidy levels such that, where a PHO is serving a very high need population, the level of co-payment will be minimal because the government subsidy will support near universal care.

Capitation systems are appealing to some providers because, in theory, they should be less administratively intense: instead of generating a claim for each unit of service on a daily or weekly basis, the practice must keep track of enrolled persons and submit information quarterly, semi-annually, or annually. However, from the funder’s monitoring and accountability standpoint, there may be a need to capture transaction level information. For example, if the funder is concerned about primary care follow-up care for a child discharged from hospital for an asthma episode, there is a need to know that a particular patient had a consultation within a particular time period with a certain provider. In the mid 1990s, the need to monitor quality led purchasers of managed health care services in the US to form the National Committee for Quality Assurance (www.ncqa.org), which establishes quality standards and data reporting requirements.

Potential problems of fairness and perverse incentives arise when capitation funding is targeted by area deprivation, but patient co-payments are individually targeted using CSC status. These potential problems arise as much for pharmaceutical benefits as they do for the GMS benefit. It is not possible for practices to target co-payments using area deprivation due to socioeconomic heterogeneity at an individual level even within small areas... Low income practices will typically include a minority of higher income patients; they will enjoy more generous subsidies than their individual circumstances dictate. This may induce higher income people, especially those with health problems, to enrol in low income practices, thereby reducing targeting...
effectiveness. These potential problems will need to be monitored carefully; however if co-payments are reduced substantially over the next ten years as a result of increased funding to PHOs then the size of the potential problem will be much diminished.

Capitation funding may be easier to apply to general medical and practice nurse services and laboratory tests than to pharmaceuticals, for two reasons. First, capitation involves prospective budget-setting, which presents a problem because it is not possible accurately to predict future drug prices or the impact of accepting new drugs into the Pharmaceutical Schedule or licensing existing drugs for new conditions. Second, inter-patient variability in drug cost is very high, making it difficult for PHOs to monitor and control expenditure. The effectiveness of capitation funding for pharmaceuticals will need to be monitored carefully, and problems with drug pricing and inter-patient variability should be actively managed.

Participation in the Primary Health Care Strategy is voluntary for providers and consumers. The needs-based approach to funding should attract most providers serving high concentrations of low socioeconomic groups. PHOs serving more well-off patients may develop more slowly. The Government’s strategy of funding higher subsidy levels universally through PHOs from 2003/04 should provide incentives for more broad-based PHO development.

In conclusion, the information presented in this paper refers to policy developments in progress — it is possible that further changes will occur prior to their full implementation. Capitation funding of PHOs is a key element of the Government’s Primary Health Care Strategy, and is aimed at achieving more equitable funding of primary care services, better targeting of resources to communities with high health need, enhancing the primary care team approach, and encouraging a population focus by PHOs. The widespread implementation of capitation funding of PHOs is likely to have a direct impact on most primary care providers and will exert an important influence on the future development of primary care in New Zealand.

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“Let’s talk of graves, of worms and epitaphs”

Scrutator

Not everyone with an interest in graves and epitaphs is as concerned with mortality and the loss of regal power as Richard II.¹ Some years ago, a local festival included a series of graveside lectures. The gravemaster – lecturer dressed in black cloak and black broad brimmed hat, addressed through a loudhailer a crowd of about two hundred assembled at the graveside. The crowd was led from grave to grave by the gravemaster’s apprentice – a lad with shoulder high ghetto-blaster playing the Dead March and other funereal music. The gravemaster told the history and not infrequent scandals of the interred notables. In life, some slept elsewhere than the marriage-bed, but in death, all rest in the family grave.

The tranquillity of the graveyard is deceptive. Personal and social histories, triumphs and tragedies are rendered in stone by the quiet skill of the monument mason and the poetry of the Authorized Version.² “Greater love hath no man than this, that a man lay down his life for his friends”² recalls a drowning in Napier. “Man found in Rangitata” – unnamed, unknown and unclaimed – tells of the pioneering history of high country Canterbury. A headstone in a northern English town comments on eighteenth century public health in recording the deaths from fever of five children of one family within ten days followed many years later by their parents.

Genealogists are interested in death certificates which in some ways are medical epitaphs. The local genealogical society invited a pathologist, a surgeon, a physician and a general practitioner to interpret the historical, sometimes archaic entries of their forebears’ certificates. There was the expected toll from tuberculosis, streptococcal disease and infantile diarrhoea in all their many forms. We guessed at some such as “marasmus”, “atrophy”, and “lightening”, and had to do homework on some such as “the English cholera” – a usually milder form than the deadlier “Asiatic or Indian cholera”. “General paralysis in conjunction with insane episodes” was an embarrassment, softened by a little relief laughter.

We were perplexed by the certificate of one who had died in a nineteenth century Canterbury mental asylum of “Visitation of God”. John Clare wrote of the interpretation of mental disease in supernatural terms during his long incarceration in Northampton County Asylum – “I am! Yet what I am who cares, or knows?”³ Clare concludes his poem with a wish for his own “Visitation of God” and the peace of a mind in equilibrium.

“I long for scenes where man has never trod –
For scenes where women never smiled or wept –
There to abide with my Creator, God,
And sleep as I in childhood sweetly slept.
Full of high thoughts unborn, So let me lie,–
The grass below; above, the vaulted sky ”.³

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¹ NZMJ 07 June 2002, Vol 115 No 1155
² http://www.nzma.org.nz/journal/ © NZMA


PHARMAC and availability of pharmaceuticals - and response

In his editorial,1 Professor Menkes makes a number of points to which we would like to respond. We support the principle of obtaining pharmaceuticals at the best possible price and of prescribing in a way to get the best health outcome for the money available. We agree that it is in nobody's interest for expensive but ineffective treatments to be sanctioned. However, we question whether PHARMAC adopts a systematic unbiased approach to reckoning cost effectiveness in pharmaceuticals. There is evidence that a number of pharmacological agents, discussed in the article,2 which are available in most other countries, are useful in depression when treatment resistance or side effects occur and it is the fact that these data seem to be selectively noted or ignored by PHARMAC that we have difficulty with. There are also some specific points to which we would like to respond.

Firstly, on the basis of other studies of drug treatment data which apparently show that drug sponsorship has affected the interpretation and also in some cases the data, Professor Menkes casts doubt on company sponsored trial data and presumably, by implication, the meta-analysis of Thase.3 However, he finds no fault in this particular meta-analysis and does not criticise interpretation of the data. Since there are no other data available on this subject, for reasons which we discussed in our article, we find this approach unhelpful.

Secondly, Professor Menkes specifically criticises the study by Poirier and Boyer4 of venlafaxine versus paroxetine in treatment resistant depression stating that it "unfortunately does not specify what proportion of patients failed to respond to SSRIs during the index episode". In fact, the paper quite clearly specifies that in the venlafaxine group 66% of patients previously failed to respond to an SSRI and in the paroxetine group 65% had failed to respond to an SSRI. This is comparable with the number of patients that have previously failed to respond to a tricyclic antidepressant in each group. His assertion that it would be unsurprising that SSRI non-responders tend not to respond when challenged with another SSRI is not supported by the data or our clinical experience. Open trials quite clearly show that the response rate to a second SSRI in the presence of non-response to a first SSRI is approximately 50%,3 and the strategy of switching from one SSRI to another is commonly clinically used.

Furthermore the pharmacological profile of venlafaxine is similar to that of some tricyclic antidepressants, making this, in our opinion, a very fair comparison. Indeed, this is a trial of a novel strategy (venlafaxine) versus a commonly used strategy (switching to a second SSRI). The suggestion that venlafaxine be compared with the novel strategy of augmentation of SSRI with reboxetine (which is not funded in New Zealand) seems to us to be far more open to methodological criticism.

Finally we would like to draw the attention of readers to a forthcoming meta-analysis of venlafaxine5 which adds to the data suggesting that this may be more effective than SSRIs in the treatment of depression. Once again this is drug company sponsored.

Readers, PHARMAC and Professor Menkes can decide whether to evaluate the data
critically or simply to dismiss it on the grounds that it may be affected by this sponsorship.

Dr Richard Porter
Senior Lecturer in Psychological Medicine.

Roger T Mulder
Associate Professor in Psychological Medicine, Christchurch.


Response

I am pleased that Porter and Mulder agree that pharmaceuticals be obtained at the best possible price and used in a way which maximizes health outcomes -- this is entirely in line with PHARMAC's operating principles. However, they question whether PHARMAC adopts a systematic unbiased approach in reckoning cost effectiveness, based on the agency's decision not to purchase certain drugs available in other countries. Of course in themselves such decisions can hardly be taken to indicate bias. The real issue is whether these decisions are reasonable given the evident cost-effectiveness of venlafaxine and the other drugs in question. Since a drug's cost-effectiveness is calculated as a ratio of benefit to cost, any treatment can become 'not worth it' if the supplier demands too high a price.

The argument boils down to a simple question: are new drugs such as venlafaxine worth what the companies want to charge, given the existing drugs budget and the competing priorities both within and without psychiatry? Thus far, it would seem that PHARMAC's calculations have yet to justify purchase of venlafaxine, but the potential usefulness of this and other agents in treatment of resistant mental disorders has left them on an 'investment list' prompting continuing negotiations with suppliers. Looking back over PHARMAC's history, the same arguments previously unfolded with regard to purchase of branded SSRIs and atypical antipsychotics. In both cases, good evidence has become available vindicating PHARMAC's hard-nosed purchasing policy. In both cases, enormous price differentials (one to two orders of magnitude) relative to conventional treatments effectively prohibited the unrestricted availability of the branded drugs -- the additional benefits simply were not worth it. In both cases, targetting the new drugs' availability to those most likely to benefit was both clinically and fiscally sensible. Unfortunately, awareness of this simple point was obscured,
inter alia, the very effective lobbying and marketing strategies of the pharmaceutical companies concerned, and by the rhetoric of a number of indignant prescribers.

Because PHARMAC has been given responsibility for judiciously allocating scarce resourcing it is inevitable that hard decisions will need to be made, and that some prescribers and their patients will be frustrated. If Porter and Mulder have constructive suggestions for affordable improvements to PHARMAC’s methods of economic analysis,2 or ways to make ends meet such as how to increase revenue or to reduce the prices demanded by suppliers, then their suggestions will be welcome. Meanwhile they should not be surprised if PHARMAC’s efforts to avoid bias lead to an apparent, often frustrating, resistance to focused lobbying. Such lobbying reflects both dedication and the individualism which is part of our clinical medical culture,3 and which inevitably collides with the utilitarian ethos of government agencies.

Just because a study is funded by a drug company does not make it worthless but is cause for healthy scepticism. Thus I seem to be somewhat more sceptical than Porter and Mulder regarding the RCT of venlafaxine versus paroxetine in refractory depression.4 I am grateful to my Canterbury colleagues for bringing to my attention the proportion of patients who failed to respond to SSRIs during the index episode in that study. However, concern remains when a single sponsored RCT is relied upon to make important funding decisions. This is particularly true given very recent evidence from a large (n=168) RCT confirming that more than 50% of chronically depressed SSRI or tricyclic non-responders improve significantly when crossed over to the alternate antidepressant class,5 suggesting a cheap alternative for many patients who might otherwise receive vanlafaxine.

Porter and Mulder also take issue with my ‘implied’ scepticism about sponsored meta-analysis, such as the one by Thase et al,6 and find my approach unhelpful since "there are no other data available on this subject." I am indeed sceptical about that meta-analysis, specifically because other data apparently do exist and have been excluded from the analysis, probably on the basis of publication bias.7 As Porter and Mulder note in their original article8 "...Reputable journals are extremely careful about the review and publication of studies with possible conflicts of interest and we have no reason to suppose that the British Journal of Psychiatry is any different in this regard." Exactly so, and it is fascinating to note recent concerns about the links between Wyeth (the manufacturer of venlafaxine) and the editor of the British Journal of Psychiatry, in which the aforementioned meta-analysis appeared.9

Venlafaxine will, I predict, shortly become available in New Zealand, but since the price demanded by Wyeth is many times that of the generic SSRI and tricyclic alternatives, its availability may sensibly be restricted to use in a specific population of patients refractory to both conventional antidepressant classes. The net economic impact of venlafaxine is difficult to anticipate, partly due to externalized costs of depression and its treatment, but also because the majority of pharmaco economic analyses may suffer from sponsorship bias, sometimes with embarrassing transparency.10

David Menkes
Professor of Psychological Medicine, University of Wales.


Supervision of junior doctors

Jonathan Coates raised the important issue of the supervision of junior doctors in his Medicolegal Diary.1 He referred to a case where a house surgeon failed to note whether a patient was oriented, and the consultant's failure to ensure that the patient's records were comprehensive breached the Code of Rights.

Each complaint received by the Commissioner is decided on its particular facts. We thought readers might be interested to know the facts behind the Commissioner's opinion on this complaint:

A 44-year-old man was admitted to a rural hospital with a history of headache, confusion and incontinence following a fall. The admitting house surgeon recorded only a cursory examination of the patient's nervous system and there was no record of higher mental function. According to the notes, the patient was next seen by a doctor three days later at the time of the consultant ward round. If an examination took place, it was not recorded. Despite ongoing confusion and further falls, a thorough nervous system examination was first documented six days after admission. An independent expert advised that, with this history, the nervous system should have been examined in detail on admission. The standard of clinical documentation by medical staff was unacceptably poor and contributed to a seven-day delay in diagnosing a subdural haematoma. The Commissioner's opinion, guided by the comments of the expert advisor, was that the consultant breached Right 4(2) of the Code by not ensuring that his patient's medical record complied with professional standards.

The full opinion can be found on the Commissioner's website www.hdc.org.nz. The correct opinion number is 98HDC16860.

The basic principle in New Zealand is that a specialist has responsibility for the overall clinical care and management of the patients under his or her care. However, he or she may delegate aspects of care, and is entitled to rely on information and assessment from junior doctors, so long as he or she has good reason to believe that they are competent to carry out such tasks. Where aspects of clinical care are delegated to junior doctors, specialists have a duty to provide supervision with reasonable care and skill and in accordance with professional standards.

Mr Ron Paterson,
Health and Disability Commissioner.

Dr Marie van Wyk,
Legal Advisor to the Health and Disability Commissioner.

Bernard Shieff

Bernard Shieff (Bill) who died last year (2001) had been a successful and popular general practitioner in the Eastern Suburbs of Auckland prior to becoming a specialist obstetrician and gynaecologist and a member of the staff of the National Women's Hospital.

Bill was born in Glasgow in 1916. His family had come from Eastern Europe and they moved to New Zealand in 1926, settling in Takapuna. At the time he was old enough to appreciate the difference and was forever grateful for being able to live in this part of the world. He attended Takapuna Grammar School and the Otago Medical School, graduating in 1942. After being a house surgeon at Auckland Hospital he joined the NZ Army Medical Corps and with the rank of captain served in the Middle East, Italy and Japan.

While serving in the Middle East his unit was on one occasion moved to Palestine. During that time he visited relatives living in a Zionist settlement. His arrival caused considerable consternation because he was wearing the uniform of an army officer, apparently British, and he could not speak Hebrew. Fortunately he could remember enough Yiddish from his Glasgow childhood to avoid a dangerous situation.

On his return from Japan in 1946 he married and commenced general practice in Mission Bay. In a short time he had a large obstetric component and was encouraged by Professor Harvey Carey to specialise in O & G. In May 1955 he took a considerable drop in income to become first a house surgeon then registrar at National Women's Hospital.

In 1958 he went to London, obtained the MRCOG and spent the next 20 months there doing locums. On his return to Auckland in 1961 he joined the staff of the National Women's Hospital. He was made FRCOG in 1975. He had an early interest in the treatment of infertility, particularly tubal surgery, introducing microsurgical techniques to Auckland. He also developed an interest in the surgical treatment of vulval carcinoma. For many years he assisted the late Alistair Macfarlane with radical vulvectomy procedures.

Bill had a busy private practice and was one of a generation who had his rooms in Remuera Road, delivered babies at the Mater and operated at the Lavington Private Hospital in Epsom. In 1978 he retired from the National Women's Hospital and continued in private practice for a short time.

As a devout member of the Jewish faith he was highly respected in the Auckland Hebrew Congregation. He had an active retirement and appreciated the opportunity of pursuing his many interests. Soon after its inception in 1989 he joined the Remuera University of the Third Age and entered into its activities with characteristic enthusiasm.

We are grateful to Dr John Stewart for this obituary notice.
Depression in patients in general practice – and response

I have concerns about the article by Arroll et al (NZ Med J 2002; 115: 176-9). The title of this article is misleading. It suggests the study had the goal of looking at the prevalence of depression in an Auckland general practice. It turned out the article had different goals. The authors' prime goal was to study the abilities of general practitioners (GPs) to diagnose depression as compared to the gold standard of the Beck's Diagnostic Inventory scale. The second goal was to measure the prevalence of depression in Maori and non-Maori patient groups. The final goal was to compare the prescribing of antidepressants in these two groups of patients.

The authors addressed the measurement biases inherent in the use of BDI. However, they did not address the selection bias inherent in their method. The GPs obtained consent from the patient before the gold standard (the BDI) was administered to patients. (There was no information provided on the 29% of patients in whom consent was not obtained - so an estimate of the effect of the bias could not be made). Furthermore, the GP could signal to the interviewer that a patient was considered to be suicidal. This means the interviewers administering the BDI questionnaire were not blind to the diagnostic performance of the GPs.

The threshold used in the study was >16 for BDI, which represented borderline clinical depression. Using this cut off, there were 17 patients in whom the GPs missed depression and the BDI was positive. There were 20 patients in whom the GPs diagnosed depression and the BDI was negative. There was agreement with the BDI for 18 patients who were depressed and 198 patients who were not depressed. One needs to have a firm belief in the reliability of BDI as a 'gold standard' measure of patient suffering to claim that GPs have missed a worrying amount of depression. Furthermore, given that this was a study of diagnostic abilities of five GPs, there was no information about the individual GPs or their individual diagnostic performance.

Table 2 reports comparisons between Maori and non-Maori patients. Although this is an important comparison, the figures rely on a cut-off of >10 for BDI which was not the level at which the GP performance was measured. If the original cut-off level of >16 was used, then no difference was found between the two groups in the use of anti-depressants medication. The small numbers in this study and the above mentioned biases make one wary of believing the conclusions about Maori and non-Maori differences.

Dr Marjan Kljakovic,
Senior Lecturer in General Practice,
Wellington School of Medicine.

Response

We are puzzled with Dr Kljakovic's concern over the title. His concern was that it suggested that the study had the goal of looking at the prevalence of depression in an Auckland general practice. While this was one of the aims of the study there is nothing in the title to suggest that. The main aim was to look at the "rate of detected
and undetected depression in general practice patients." In the paragraph previous to the one above we made the point that the prevalence of depression in New Zealand general practice has not been clearly established. By measuring detected and undetected depression we obtained an estimate of the prevalence. The secondary aim (not stated) was to look at the rates of depression among Maori. When we found no difference we decided to look at the usage of antidepressants between Maori and non-Maori. As this was not a primary outcome we suggested that further work be done on this. Given that there are disparities in use of health care, under-use seems likely.\textsuperscript{1,2} Informed consent had to be obtained before administering the test as directed by the ethics committee. We asked that patients if they would consent to completing a questionnaire on their health and mood to conceal the fact that it was a study focussing on depression.

Our research assistant was instructed to not look at the form from the GP (thereby remaining blind to the diagnostic performance of the GP) stating that the GP thought the patient was depressed and that the patient was safe or unsafe. This piece of information was needed to ensure that a potentially suicidal patient was screened and not treated. The Beck Depression Inventory (BDI) is self administered and hence we feel confident that any bias due to unblinding would be minimal.

The number of patients who declined was 19\% not 29\%. We asked the GPs to make a note of those who declined but this was rarely done. It is difficult to ask GPs to perform extra tasks in the research situation as Dr Kljakovic will be well aware.

We agree that the BDI is not considered a gold standard for depression. In retrospect we would have used at least one other tool but we thought there would be patient resistance to this. In our current study we are using three instruments. The BDI has advantages in that it is self administered and has been validated against other gold standards. We presented our data to show the multiple cut points so that readers could make their own comparison. We know from other work that the cases of depression missed by GPs tend to be the less serious cases yet there may be advantages in knowing the mental state of a patient other than providing therapy. As only 35 cases of depression were found with the cut point at seventeen it was meaningless to report the sensitivity and specificity of the six doctors individually as the confidence intervals are enormous.

We have reassessed the cut point for antidepressant use and with a cut point of 17 there were nine Maori patients and only one was on antidepressants while in the non-Maori there were 34 and 17 of them were on or had been on antidepressants. This was significant $p=0.03$ (Fishers exact test). Thus we are still concerned about this matter and intend to pursue it.

Bruce Arroll,
Associate Professor.

Felicity Goodyear-Smith,
Senior Lecturer,
Department of General Practice and Primary Health Care,
University of Auckland.

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Review of developments in colorectal surgery in New Zealand


Colorectal surgery as a subspeciality has evolved considerably in New Zealand in the last ten years. This evolution has been a result of both changes in the structure of general surgery with increasing subspecialisation in New Zealand and overseas, and the importation of newer treatments from overseas. Subspecialisation has developed as a result of the recognition of the depth of knowledge, experience and skills required to treat patients with specific diseases. Many overseas studies have shown better results with subspecialisation in colorectal surgery, especially with rectal cancer. To facilitate this, post-fellowship training programs in colorectal surgery have developed (there are two recognised training posts for the Australasian training program in colorectal surgery available in New Zealand hospitals), some surgeons restrict their practice to colorectal surgery, and colorectal units have developed in our larger public hospitals.

These organisational changes relate to how health care is delivered. There are also many changes in what is delivered. This article reviews some of the newer operative and nonoperative means of managing colorectal disease. The results of these changes are reflected in the way we see colorectal disease managed in our community.

Colorectal cancer

Colorectal cancer is a major cause of death and morbidity, with 2299 new colorectal cancer registrations in New Zealand in 1997. About half of all patients diagnosed with colorectal cancer will die of the disease. In the last ten years there have been major developments in the technical aspects of surgery for rectal cancer and the use of radiotherapy.

Total mesorectal excision (TME) versus transanal endoscopic microsurgery (TEM). Most rectal cancers should be removed by major surgery using a technique that follows the fascial plane investing both the rectum and the mesorectum (usually referred to as total mesorectal excision (TME)). This technique has reduced local recurrence rates to 3-8%, compared to traditional techniques, which cone down into the mesorectum and do not respect its surrounding fascia, producing local recurrence rates of 20-40%.

Major abdominal surgery for rectal cancer is associated with significant morbidity and mortality. Frequency and urgency of evacuation and minor soiling are not uncommon afterwards, and if damage to pelvic nerves occurs, impotence and bladder dysfunction may result. Accurate local staging with endorectal ultrasound or MRI can identify early cancers that do not invade into the muscle wall of the rectum and are suitable for local excision, avoiding this morbidity. The recent development of TEM involves using a system of binocular optics built into a specialised operating sigmoidoscope.
with laparoscopic type instrumentation. This enables accurate and complete local excision of early cancers higher in the rectum without compromising the oncological outcome.\(^6\) Patients are able to go home 1-2 days after a TEM procedure with minimal or no discomfort.

In patients with significant co-morbidity, and/or a limited life expectancy, the indications for local excision (to avoid major surgery) are reasonably extended to include cancers invading into or even through the muscle wall of the rectum. TEM has a role here when local recurrence rates of to 10-40% at two years for local excision\(^6\) may be acceptable. Radiotherapy can be added to improve local control.

**Radiation.** Radiation to improve the results of surgery has been standard practice for the last decade where postoperative radiation has been used for node-positive rectal cancer (Dukes C). More recently, with increased understanding of the importance of pathological analysis of rectal specimens, patients with positive or close radial margins have been offered postoperative radiation for about six weeks.\(^7\) Preoperative radiation has generally been thought to be more effective, but as many patients do not require any radiation, it was used only in cases where there was a large or bulky tumour.\(^8\) The delay in surgery (usually 3-4 months) with long course preoperative radiation may be associated with some psychological stress. Swedish studies have suggested that a short course (one week) of radiation immediately preoperative is as effective as the postoperative six weeks of radiation where resectability is not the indication for preoperative radiation.\(^8\) Increased recognition of the complications of radiation postoperatively on bowel, bladder and sexual function\(^9\) means surgeons and oncologists are increasingly trying to avoid radiation except where proof of benefit exists. The data showing reduction in local recurrence following radiation is also complicated as it was collected when conventional coning down surgery, not TME, was used. With lower rates of local recurrence following TME, radiation may not be required. There is at present a large, ongoing international trial of short course preoperative and long course post-operative radiation with at least one New Zealand centre taking part. This study is also looking at the effect of TME and the need for radiation (MRC-CR07).\(^10\) The results from a Dutch study looking at TME plus or minus radiation have recently been published, showing a local recurrence rate of 8% with TME alone and 2.7% with TME and radiation.\(^11\)

**Laparoscopic Surgery.** Laparoscopic surgery has changed many aspects of intra-abdominal surgery with more rapid post-operative recovery, early post operative hospital discharge and return to work, and improved wound cosmetics. The place of laparoscopic surgery in the management of colorectal cancer, however, remains controversial. At present there are three large international trials (Mayo, UK/MRC CLASSIC trial and an Australasian trial ALCAS) underway to establish the place of laparoscopic surgery in patients with colon cancer. All three trials specifically exclude patients with rectal cancer. The American Society of Colon and Rectal Surgeons and the UK National Institute for Clinical Excellence (NICE) recommend that laparoscopic resection for colorectal cancer be undertaken only in the context of formal prospective trials. The initial concern about possible poorer oncological results following laparoscopic surgery, particularly due to tumour implants into port sites, has considerably lessened as data become available from small, non-randomised studies and laboratory based research.\(^12\) These trials are ongoing and it will be some years before results are available.
Colorectal Stents. For many patients who present late with colorectal cancer, cure is no longer possible and what is needed is palliation and relief from obstruction. Self-expanding metallic stents have been used in New Zealand since 1997 to relieve malignant large bowel obstruction. This has resulted in shorter hospital stays and the avoidance of major surgery or a stoma, appealing options to the patient who is not fit or has incurable obstructing colorectal disease. Stents have also been used in patients with acute left sided obstructions where cure is still possible to allow decompression of the obstructed colon and subsequent elective resection after appropriate bowel preparation. Technically, the obstructing lesion must be above the mid rectum and able to be intubated with a guide wire, allowing the stent to be inserted using the Seldinger technique. The cost of the stent (about NZ$2500), together with radiology and endoscopy time must be balanced against the costs of theatre and hospital stay, as well as the physical and psychosocial costs to the patient of resectional surgery, particularly for those whose surgery is palliative. Failure to intubate the stricture, perforation with guide wire or stent, failure to relieve the obstruction despite adequate position, and stent migration are all potential problems with the procedure. This is, however, a useful technique for some patients for relief of malignant colorectal obstruction and avoidance of major surgery and/or a stoma.

Population Screening for Colorectal Cancer. Given the incidence of colorectal cancer and its high mortality rate, much research has been conducted into the place of population screening. Faecal occult blood testing (FOBT) followed by investigations of those with positive results is the most studied screening tool. FOBT has been extensively investigated in three randomised-controlled trials from the United States, Denmark, and the UK, of which the latter two were purely population-based. Meta-analysis of the two European trials showed a 16% reduction in mortality in a population screened over an 8-10 year period. However, to achieve this reduction, 2% of the screened population required a colonoscopy after the initial FOBT, and 4% needed colonoscopy at some stage during the screening program. The implications for New Zealand were examined by a National Working Party. This group looked not only at the mortality data, but also at the risks of screening and at the resource implications of a FOBT program. The recommendation of the Working Party was that, based on available data, general population screening with FOBT was not recommended. However, the Working Party suggested that the issue needs reviewing again in the future.

Separate from the general population, there are individuals at increased risk of developing colorectal cancer. Examples are those with inherited disorders such as familial adenomatous polyposis, inflammatory bowel disease, a past history of adenomatous polyps or a family or personal history of colorectal cancer. A working party has been formed to examine the role of screening in this group in New Zealand and will report, hopefully, during 2002.

Anal intraepithelial neoplasia (AIN)

AIN is a premalignant condition of the anus. It is part of the group of intraepithelial neoplasms and often may be considered a field change. The group includes vulval (VIN), vaginal (VaIN), and cervical intraepithelial neoplasia (CIN). The risk factors for AIN include the presence of VIN, VaIN or CIN, previous renal transplantation and HIV infection. In many patients the disease is found almost incidentally when a
haemorrhoid or anal skin tag is excised. Often the anus can look normal and the patient may have minimal symptoms. Screening may become appropriate in high-risk groups using a smear technique as in cervical screening. However, this has been shown to have a very high false negative rate. There is a much higher pick up rate when the anus is examined in patients having colposcopy for cervical, vulval or vaginal abnormalities. AIN is graded like CIN into three categories: AIN I, II and III. Grade three is high-grade dysplasia, and micro-invasive carcinoma may be found in resected specimens. The ideal treatment for patients with AIN is unknown. It is uncertain how many patients with lower grade AIN go on to frankly malignant disease. The treatment is excision, which can cause significant morbidity with anal pain, discomfort, discharge and faecal incontinence. Many patients have minimal symptoms when the disease is discovered, so radical excision is not indicated. The recommended treatment at present is observation for AIN I and II with follow up by colposcopy and biopsy, and excision only in AIN III.20

**Incontinence**

Faecal incontinence can be a devastating problem to the individual and is personally and socially incapacitating. In a recent study it was shown to significantly affect 8.1% of New Zealanders.21 It has such a social stigma that patients are often unwilling to seek help and doctors are reluctant to inquire about it. The aetiology of faecal incontinence in women was redefined in the 1990’s. Previously, most faecal incontinence was thought to be due to degeneration of the pudendal nerves that supply the anal sphincter mechanism. Endoanal ultrasound has enabled the anatomy of the external anal sphincter to be visualised. It is now clear that during childbirth, disruption (due to stretching) of the anterior portion of the external anal sphincter occurs in 10-30% of primiparous women.22 This causes problems after delivery in only 1% or less of women. However, as the decades pass, striated muscle mass decreases and the external sphincter muscle weakens. With increasing age the sphincter may no longer be able to compensate for this earlier disruption and later weakness, and faecal incontinence develops.

If conservative treatments of diet, constipating and bulking drugs fail, an overlapping sphincter repair is successful at restoring continence in 80% of cases with sphincter defects. The use of biofeedback has become popular but the results of this are uncertain. The use of antegrade colonic enema (ACE) procedures where the appendix is used as a conduit to flush the colon has had limited application but has produced excellent results in some patients especially those with low spinal injuries or spina-bifida.23 Other treatments such as dynamic anal graciloplasty, and the artificial sphincter are presently only available in a very limited way in New Zealand. A dynamic graciloplasty involves taking the patient’s own gracilus muscle (from the inside of the leg) and detaching the distal end. This is then wrapped around the anal canal and a stimulator inserted into the nerve supply. When turned on, this causes contraction of the muscle and continence and the patient turns it off to defecate. Overseas studies suggest it is effective in 65-75% of selected cases and is cost effective.24 For these patients, the only alternative is usually a colostomy. An artificial sphincter is also now available, and is similar to the urinary artificial sphincter. It has not been used in New Zealand to date. Overseas evidence was initially favourable but there is now concern about the sphincter eroding through the anal canal, and many have been removed because of this.
Haemorrhoids

The majority of patients with first and second degree haemorrhoids are easily and simply treated in the clinic by injection sclerotherapy using 5% phenol in almond oil, rubber band ligation or infra red coagulation. For patients with prolapsing (third and fourth degree) haemorrhoids there have been a number of new developments. Diathermy dissection of haemorrhoidal tissue and anal tags from the internal anal sphincter without the need for ligation of the pedicle is now commonly practised.25

The well-documented problem of post operative pain following open haemorrhoidectomy is due to the dense innervation of the anoderm and perianal skin with sensory fibres. A week of oral metronidazole following open haemorrhoidectomy has been shown to reduce pain especially towards the end of the first week presumably by a reduction in secondary anaerobic infection of the operative site.26

A novel surgical technique of stapling and resecting the distal rectal mucosa just above the dentate line has recently been introduced for prolapsing haemorrhoids.27 The technique is currently termed stapled haemorrhoidectomy but would more accurately be termed stapled anopexy. At least nine randomised studies have documented that the procedure is effective and results in less pain than traditional open haemorrhoidectomy. It does not treat directly the external anal tags that can accompany large prolapsing haemorrhoids but experience suggests these can reduce size following stapling, but may be better excised at the time of stapling. Ten-year follow up data have been reported recently from Italy where the technique was developed and favourable.28 Further long term follow up is required before this procedure can be widely adopted.

A note of caution for all practitioners treating patients with haemorrhoids is required. All procedures are associated with occasional complications related to the procedure employed. Septic complications can develop in immunocompetent as well as immunosupressed patients.29 HIV-positive patients can develop overwhelming and sometimes fatal infection following outpatient procedures and such patients are best advised to continue with non-surgical options.30

Conclusions

There have been considerable developments in colon and rectal surgery in the last ten years. There are developments also in the management of inflammatory bowel disease that might have been touched on, but due to space limitations this paper is brief. The knowledge base has widened and increased in depth, which has in part lead to the development of colon and rectal surgery as a more defined subspeciality. As in many areas of surgery it has been shown that the best results are obtained by adequate training and case numbers.

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References:

Assessment of snorers in primary care: straight path to treatment

Bryn Sparks, Alex Bartle, Lutz Beckert

This viewpoint article describes a model for primary care management of snorers. Our model is designed to provide a guide for general practitioners (GPs) to group snorers based on the two dimensions of sleepiness and nocturnal hypoxaemia. We have carefully avoided the topic of whether one could use pulse oximetry in place of polysomnography as an initial investigation for Obstructive Sleep Apnoea (OSA) within a specialist facility. Rather, we are offering a way to think about snoring patients in primary care. Some of those snorers will include people with OSA, but the model does not purport to diagnose those patients specifically.

Why is snoring something that medical practitioners need to take seriously?

Heavy snoring is an important health issue for both those who snore, and those who live with snorers. Epidemiological studies suggest the prevalence of snoring may be 20%-60% of the adult population. The prevalence increases with age, particularly among men.\(^1\)\(^-\)\(^3\) Snoring is characterised by recurrent inspiratory noise arising from oropharyngeal vibration. Habitual snoring is the most common symptom of OSA,\(^4\) a condition which has been shown to be associated with disturbed sleep, excessive daytime sleepiness and hypertension. Snoring itself is independently associated with hypertension in both men and women even when frank OSA is absent.\(^5\)

There are social consequences to snoring. Habitual snoring is a cause of familial disruption particularly when it leads to use of separate beds or even bedrooms. Not surprisingly, sleeping partners of heavy snorers are more frequently affected by symptoms of fragmented sleep and chronic fatigue than are partners of non-snorers.\(^6\)

What are the current guidelines for management of snorers?

Current practice guidelines urge GPs to refer patients who present with snoring and daytime sleepiness to specialist sleep services for comprehensive investigation using polysomnography.\(^7\) Polysomnographic investigation is a robust and well established method for detecting sleep disorders that are often associated with snoring (OSA and Upper Airway Resistance Syndrome (UARS)), and particularly other less prevalent sleep disorders. Unfortunately patient access to polysomnographic investigation can be limited through lack of local facilities. A recent position paper prepared by the New Zealand branch of the Thoracic Society of Australia and New Zealand described long waiting times for polysomnographic investigation in most centres due to limited health care delivery resources.

Those patients who are referred for management of snoring to non-sleep specialists without initial polysomnographic investigation risk having a potentially debilitating sleep disorder remain undiagnosed.\(^8\) Those who are referred to sleep specialists in the first instance often face considerable delays before receiving appropriate treatment for snoring or for OSA. The dilemma for the GP is whether to refer a patient suffering...
from disruptive snoring to a specialist (and how to decide which specialist) or to advise conservative treatment.

**There are alternative investigations that could guide primary practice management of snoring – a two-dimensional approach.**

Partner-reporting has been validated against polysomnography and found to be very reliable for establishing the presence of habitual snoring.\(^4,6\) Polysomnography is not needed to confirm a diagnosis of snoring.

We believe it would be useful for GPs to assess two further symptomatic dimensions before deciding on the next appropriate management step for snorers - excessive (or problematic) daytime sleepiness, and nocturnal hypoxaemia. Together, those two dimensions reflect the increased morbidity associated with snoring. The Epworth Sleepiness Scale (ESS)\(^9\) can be used to assess excessive daytime sleepiness, and recording pulse oximetry can be used to assess nocturnal hypoxaemia. Both these tools could easily be used in general practice.

The ESS has been used extensively in both clinical and research settings. It is an eight-item questionnaire that asks patient to self-rate their propensity for dozing in a range of everyday situations. The rating (0-3) for all items are added to give an ESS score of 0 (insignificant daytime sleepiness) to 24 (excessive daytime sleepiness). While it does not correlate particularly well with polysomnography-based tests designed to measure sleepiness (Maintenance of Wakefulness Test, and Multiple Sleep Latency Test) it has been argued that the ESS contains a psychosocial element that relates better than those tests to the lifestyle impact of sleepiness.\(^10\) The ESS has been found to be a good predictor of long-term adherence to nasal continuous positive airway pressure therapy (nCPAP), the treatment of choice for OSA.\(^11\) Most importantly, the severity of OSA rated according to the frequency of obstructive events is not predictive of long term adherence to therapy in those patients who are not also suffering excessive daytime sleepiness (ESS = 10).\(^12\)

Pulse oximetry has been used increasingly over the last two decades to measure absolute nocturnal hypoxaemia and, more recently, patterns of desaturation.\(^13\) It has been used as a case-selection tool in the first published mechanical-placebo controlled randomised trial of nasal continuous positive airway pressure treatment for obstructive sleep apnoea.\(^14\) There is a close correlation between oxygen saturation dips and obstructive respiration,\(^15-29\) particularly among patients who have normal daytime lung function.\(^30\) It is important for GPs to have some indication of the frequency of desaturation before choosing the next step in managing problematic snoring. An independent relationship between frequency of obstructive respiration and systemic hypertension has been reported from the initial phase of the Sleep Health Heart Study, a large cohort study designed to investigate the relationship between sleep-disordered breathing and cardiovascular disease.\(^31\)

**Putting the dimensions together – the Sparks Chart.**

Most studies reported in the literature have focused on using ESS and/or pulse oximetry to assist in the diagnosis of sleep-disordered breathing in a secondary healthcare setting. We suggest that combining the two dimensions of sleepiness (ESS) and hypoxaemia (oxygen desaturation index (ODI)) would provide primary care...
providers with a system for grouping their otherwise healthy snoring patients, and that management decisions could be made based on those groupings (Figure 1).

Figure 1. The Sparks Chart – grouping snorers according to combined Epworth Sleepiness Score (ESS) and 4% oxygen desaturation index (ODI).

Where do the axes cross?
The distribution of ESS scores for 72 normal subjects have been published elsewhere showing a reference range of 0-10 for ostensibly healthy male and female workers, aged 22-59 years, in Australia. Evidence from a recent randomised controlled trial suggested that an initial ESS score below 10 predicts a patient is unlikely to tolerate nCPAP therapy even if they have a high frequency of obstructive respiratory events. When considering how best to manage snoring, we suggest applying a group demarcation at ESS = 10. The ESS contributes to the assessment by identifying those snorers in whom non-respiratory sleep disorders might need to be considered, and also by identifying those who are unlikely to tolerate nCPAP even if OSA is the primary diagnosis.

The correlation between the frequency of obstructive respiratory events and ODI strengthens as the frequency increases. When considering how best to manage snoring, we suggest applying a group demarcation at ODI = 20. This could be adjusted according to availability of local resources. The ODI contributes to the assessment by identifying those snorers who are thought to be more likely to develop cardiovascular morbidity if left untreated.

The groups in detail.

**Group A – Sleepy snorers.** This group will mostly contain those snorers who have mild OSA or UARS, but may also contain snorers with co-existing non-respiratory
sleep disorders such as periodic limb movement syndrome, or narcolepsy. As such, they should be referred to a specialist sleep service for further management that may include polysomnographic investigation, although the specific approach will vary between centres. Treatment outcomes should include elimination of snoring and normalisation of ESS.

**Group B – Sleepy hypoxic snorers.** This group will mostly contain snorers who have moderate or severe OSA, but may also contain snorers with co-existing cardiac dysfunction. As such they should be referred to a specialist sleep service for urgent consideration of nCPAP therapy. Treatment outcomes should include elimination of snoring and normalisation of both ESS and ODI.

**Group C – Hypoxic snorers.** This group will mostly contain snorers who have moderate or severe OSA, but may also contain snorers with co-existing cardiac dysfunction. However, nCPAP is unlikely to be tolerated as a long-term therapeutic option without considerable specialist intervention. They should be referred to a specialist cardiorespiratory service on an urgent basis. Intensive support may improve nCPAP response in this group. However, these patients require additional investigative procedures before commencing therapy. Treatment outcomes should include elimination of snoring and normalisation of ODI.

**Group D – Snorers.** This group will mostly contain those who have snoring alone, but may also contain snorers with co-existing mild OSA. However, nCPAP is unlikely to be tolerated as a long-term therapeutic option without considerable intervention. Therefore other treatment options should be discussed with the patient, and consideration given to conservative management, or referral to orthodontic/ENT specialist services. Treatment outcomes need only include elimination of snoring.

**How does this look in practice?**

A retrospective analysis of 100 consecutive patients with problematic snoring who presented over twelve months to a general practice located in the city of Christchurch (population 350,000) was performed, and the patients were grouped according to the criteria outlined earlier (ESS = 10 and ODI = 20, Table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>A (ESS≥10, ODI&lt;20)</th>
<th>B (ESS≥10, ODI≥20)</th>
<th>C (ESS&lt;10, ODI≥20)</th>
<th>D (ESS&lt;10, ODI&lt;20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>48</td>
<td></td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>35</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

35% of the study sample (Group D) were identified as uncomplicated snorers who would likely benefit from specialist ENT or specialist dental assessment, or who could try conservative treatment co-ordinated through their GP. 14% (Group B) were identified as needing urgent consideration for nCPAP. Three percent (Group C) were hypoxic snorers who did not report daytime sleepiness and hence would likely benefit from further assessment from a specialist respiratory physician. 48% of the sample (Group A) were found to be sleepy snorers in need of further assessment by a specialist sleep service, but not on an urgent basis.
Summary

Habitual snoring needs to be taken seriously, both as a symptom of other sleep disorders and as a condition in its own right. GPs approached by patients with problematic snoring face a dilemma regarding whether (and to which service) those patients should be referred for a specialist opinion. Using the Sparks Chart, snoring patients can be grouped according to the two symptomatic dimensions of excessive daytime sleepiness and nocturnal hypoxaemia. We believe that the approach outlined in this article offers GPs a coherent and pragmatic guideline for referring and/or managing problematic snoring by using a simple questionnaire and pulse oximetry. The method has the potential to improve primary and secondary liaison. Most importantly, it offers patients a straight path to treatment.

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Gastrointestinal decontamination in paediatric exploratory ingestions

Claire Dillon, Paul Gee.

Abstract
Aims To review the effect of treatment changes in paediatric exploratory ingestion at Christchurch Hospital.

Methods We carried out a retrospective review of paediatric patients presenting with potentially toxic ingestion during six month periods of 1994, 1996 and 1999.

Results All three groups were comparable in respect to age and gender. There were minor changes in the range and proportion of substances ingested - with those in the 1999 group more likely to have taken paracetamol. In 1994, 36% of children were treated with syrup of ipecac. By 1996, only 9% were given ipecac, with 49% treated with activated charcoal. By 1999, 12% were treated with activated charcoal, while 88% received no decontamination. There was a lower admission rate in the 1999 group with no overall change in outcome.

Conclusions It is rare for paediatric exploratory ingestions to result in significant toxicity. Gastrointestinal decontamination should not be routinely used in these patients as the risk of the procedure may outweigh the risk of the poison exposure.

Paediatric exploratory ingestion is a common reason for emergency department presentation but toxicity from poison exposures is rare in this group. In the past, children were exposed to various gastrointestinal decontamination (GID) methods with the implicit assumption this would benefit them. However, these procedures themselves have measurable morbidity and mortality, and observation alone may be appropriate for the majority of exploratory ingestions. No previous studies have compared GID with no GID. This study examines three historical cohorts reflecting differing preferences for decontamination and compares the outcomes.

Methods
Christchurch Hospital is a 640 bed metropolitan hospital serving a city population of 330 000 and rural surrounds. The Emergency Department sees all referrals and emergency presentations. The details of paediatric poisoning are prospectively collected manually in a schedule that records patient particulars, substance ingested, decontamination method (if any) and disposal. All poison exposures in patients under six years of age for the six month periods January 1 to July 1 1994; December 1 1995 to May 31 1996; and January 1 to July 1 1999 were included. Both Emergency Department and hospital records were retrospectively reviewed. Data collected included age, gender, substance ingested and dose, weight (where recorded), time to presentation, method of GID (if any), and disposal. Any adverse effects from the ingested substance were also recorded. The 1999 group had phone call follow-up attempted for all patients, and Emergency Department computer records were checked for the three months following treatment to see if any patients had represented. The coroner’s office was also contacted to ensure there were no missed deaths in the study group. Patients were excluded if the Emergency Department record was unable to be retrieved. The measurements of admission, mode of decontamination, and substance taken were analysed using EpiInfo 6 for Chi-squared testing.
Results

There were a total of 435 cases of paediatric exploratory ingestion over the three study periods. Thirteen records were unable to be retrieved (two in 1994, six in 1996, and five in 1999). This left 422 (144 in 1994, 158 in 1996, and 120 in 1999). Average ages were 27 months, 35 months, and 35.7 months respectively. The gender mix was 213 female and 209 male (p=0.18) with no significant difference between the three time periods. Substances ingested represented what was available, and didn’t change significantly over the three periods, except for paracetamol and plant substances (Table 1). Of note, the proportion of paracetamol ingestions increased significantly over the study period (p=0.004) while plant ingestions were significantly reduced (p=0.03).

GID in the form of ipecac was undertaken in 36.1% of all cases in 1994 and 20.1% were offered activated charcoal, while 6.3% received both (total 56.2% receiving some form of GID). In 1996, 9% of children received ipecac and 37% were offered activated charcoal while 6.9% were given both (total 48% receiving GID). In 1999, only 12% of children received activated charcoal whilst 88% had no GID. The changes among study years in gastrointestinal decontamination were statistically significant (p<0.0001, Table 2, Figure 1).

There was a significant decline in admission rates: 41% were admitted in 1994, 36.8% in 1996 and 28% in 1999 (p=0.03). While most of those admitted stayed less than six hours, they were nevertheless registered as admissions. A large proportion of children were admitted whilst awaiting blood testing for paracetamol levels at four hours. Our current practice is to allow these children to stay with a caregiver, until a level is drawn, as they are no longer required to present for GID.

<table>
<thead>
<tr>
<th>Substance taken (%)</th>
<th>1994</th>
<th>1996</th>
<th>1999</th>
<th>*</th>
<th>†</th>
<th>‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>paracetamol</td>
<td>25 (17.4)</td>
<td>39 (24.7)</td>
<td>39 (32.5)</td>
<td>0.15</td>
<td>0.02</td>
<td>0.004</td>
</tr>
<tr>
<td>plant</td>
<td>30 (20.8)</td>
<td>28 (17.7)</td>
<td>13 (11.0)</td>
<td>0.03</td>
<td>0.09</td>
<td>0.03</td>
</tr>
<tr>
<td>benzodiazepines</td>
<td>6 (4.2)</td>
<td>5 (3.2)</td>
<td>5 (4.1)</td>
<td>0.94</td>
<td>0.87</td>
<td>0.98</td>
</tr>
<tr>
<td>distillates</td>
<td>8 (5.5)</td>
<td>3 (1.9)</td>
<td>6 (5.0)</td>
<td>0.33</td>
<td>0.22</td>
<td>0.75</td>
</tr>
<tr>
<td>household</td>
<td>16 (11.1)</td>
<td>8 (5.1)</td>
<td>6 (5.0)</td>
<td>0.06</td>
<td>0.07</td>
<td>0.05</td>
</tr>
<tr>
<td>antihistamines</td>
<td>3 (2.0)</td>
<td>0 (0)</td>
<td>4 (3.3)</td>
<td>0.16</td>
<td>0.09</td>
<td>0.50</td>
</tr>
<tr>
<td>psychiatric meds</td>
<td>2 (1.4)</td>
<td>4 (2.5)</td>
<td>2 (1.7)</td>
<td>0.60</td>
<td>0.75</td>
<td>0.84</td>
</tr>
<tr>
<td>Ventolin syrup</td>
<td>4 (2.8)</td>
<td>7 (4.4)</td>
<td>2 (1.7)</td>
<td>0.23</td>
<td>0.40</td>
<td>0.65</td>
</tr>
<tr>
<td>NSAIDS</td>
<td>5 (3.5)</td>
<td>4 (2.5)</td>
<td>4 (3.3)</td>
<td>0.90</td>
<td>0.88</td>
<td>0.93</td>
</tr>
<tr>
<td>cardiac</td>
<td>4 (2.8)</td>
<td>3 (1.9)</td>
<td>4 (3.3)</td>
<td>0.90</td>
<td>0.75</td>
<td>0.81</td>
</tr>
<tr>
<td>oils</td>
<td>6 (4.2)</td>
<td>5 (3.2)</td>
<td>7 (5.8)</td>
<td>0.86</td>
<td>0.55</td>
<td>0.54</td>
</tr>
<tr>
<td>others</td>
<td>25 (17.4)</td>
<td>52 (32.9)</td>
<td>28 (23.3)</td>
<td>0.002</td>
<td>0.007</td>
<td>0.20</td>
</tr>
<tr>
<td>Admission</td>
<td>59 (41.0)</td>
<td>63 (36.8)</td>
<td>34 (28.5)</td>
<td>0.009</td>
<td>0.10</td>
<td>0.03</td>
</tr>
</tbody>
</table>

*χ² goodness of fit with equal number expected over all years; (χ² test for change in numbers). †χ² test for contingency table (χ² test for change in percents). ‡χ² test for linear trend.

Follow-up by phone call was achieved in 76% of the 1999 group. The other 24% were not contactable. There were no adverse effects noted by the caregivers following discharge from the paediatric ward or Emergency Department. One child who initially presented having ingested 20-30 mL fragrant oil had two further Emergency Department presentations the following day with wheeze which was thought, by the attending emergency physician, to be due to asthma. There were no other representations in the three months following initial assessment. During the study there was only one major adverse outcome. In the 1994 group a two year old girl...
Table 2. Mode of gastrointestinal decontamination (GID) – totals (%).

<table>
<thead>
<tr>
<th>Decontamination</th>
<th>1994</th>
<th>1996</th>
<th>1999</th>
<th>χ²</th>
<th>χ²</th>
<th>χ²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>144</td>
<td>158</td>
<td>120</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No GID</td>
<td>63 (43.8)</td>
<td>63 (40.0)</td>
<td>106 (88.3)</td>
<td>0.0003</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Ipecac</td>
<td>52 (36.1)</td>
<td>15 (9.4)</td>
<td>0 (0)</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Charcoal</td>
<td>29 (20.1)</td>
<td>80 (50.6)</td>
<td>14 (11.7)</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Both ipecac and charcoal</td>
<td>9 (6.3)</td>
<td>11 (6.9)</td>
<td>0 (0)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* χ² goodness of fit with equal number expected over all years; (χ² test for change in numbers). † χ² test for contingency table (χ² test for change in percents). ‡ χ² test for linear trend.

Discussion

In Western countries there has been a dramatic decline in childhood poison morbidity and mortality.1-3 This decline has been attributed to law changes regarding medicines and the introduction of child resistant packaging. Poison centres, introduced 50 years ago, have also reduced emergency department attendance.4,5

During the last decade in the South Island (population 800 000) there have been three poisoning deaths in children (personal communication with the coroner). One of these was directly attributable to GID when a three-year-old child suffered charcoal aspiration following nasogastric administration for unwitnessed paracetamol ingestion. Another child died from lead poisoning following chronic ingestion of paint on a cot, and one from infanticide due to sodium chloride overload. In addition, two children suffered long term disability. One suffered severe hypoxic brain injury following ingestion of Lomotil, and the other developed oesophageal stricture from caustic soda burns. Of note, it is unlikely that any of these children would have benefited from gastrointestinal decontamination when they presented and none would fit our current criteria for its use.

Protocols and practice in Christchurch Hospital Emergency Department have changed significantly during the study periods. In 1994 many children presenting following ingestions received syrup of ipecac (36.1% overall). Although there were no major adverse outcomes from the decontamination itself, at least one child required...
admission because of ongoing vomiting. Ipecac may delay the administration and/or effectiveness of activated charcoal, oral antidotes and whole bowel irrigation. It should not be administered to a patient who has a decreased level, or impending loss of consciousness or who has ingested a corrosive substance or hydrocarbon with high aspiration potential. In fact, ipecac is no longer available in New Zealand.

In 1996 activated charcoal had almost replaced ipecac as the favoured method of GID. With the ease of giving activated charcoal the total number of children undergoing GID had increased to 60% (9.4% given ipecac, 50.6% given activated charcoal and 6.9% receiving both). Activated charcoal has been shown to be at least as effective as ipecac or gastric lavage and is better tolerated. One study suggested that placebo and decontamination have comparable outcomes in adult poisoning. In pharmacokinetic models the effectiveness of activated charcoal in reducing absorption falls off sharply after 60 minutes, and most children present after this time. The average time to presentation was 94 minutes in the 1999 group with only 39% of those presenting in less than one hour. Although charcoal GID is considered a low risk treatment there have been documented cases of adverse outcome and death from aspiration. The risk increases with insertion of a nasogastric tube, which is often required in a non-complaint child. The risk of insertion into the tracheobronchial tree is documented at 0.3-15%.

Gastric lavage is not recommended in children because the calibre of tube able to be passed is usually too small to enable significant gastric emptying.

Whole bowel irrigation was not used during the periods studied. It can cause nausea, vomiting, bloating and abdominal cramp and needs a nasogastric tube for administration. It potentially has a role in ingestions of sustained release or enteric coated drugs, substantial ingestions of iron, and for other poisons not well adsorbed by activated charcoal.

In 1999, 88% of children received no GID and there were no major adverse outcomes in either group (treated or nontreated) in hospital or at follow-up.

Australasian and international evidence has shown that the risk of poisoning achieving toxicity in this group of patients is very low as they are exploring their environment rather than attempting to poison themselves, and tend to ingest a small amount. In these children, therefore at low risk of toxicity, the use of decontamination procedures can be more dangerous than the exposure itself, and most present too late for activated charcoal to be of any benefit. Therefore the routine use of gastrointestinal decontamination in children with exploratory ingestion cannot be recommended.

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Acknowledgements: We thank Chris Curry for 1994 and 1996 data, and Dug Yeo Han for statistical analysis.

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References:


Venous thromboembolism in cancer patients in Christchurch, 1995-1999

Steven Joung, Bridget Robinson

Abstract

Aims To establish the incidence of venous thromboembolism (VTE) in oncology patients, describe risk factors, and assess outcome.

Methods The clinical records of Christchurch Hospital were searched for all patients with a history of deep venous thrombosis (DVT) and/or pulmonary embolism (PE) between January 1995 and December 1999, who were registered with the Oncology Service. Follow up closed in December 2000.

Results Of 7987 patients referred to the Oncology Service, 106 patients had 122 episodes of DVT and/or PE. The overall incidence rates per 1000 for VTE, PE and DVT were 13.1, 5.5 and 7.6 respectively. The recurrence rate was 226 per 1000, 50% occurring within four months. 70% of patients with VTE had one or more risk factors in addition to their malignancy: previous VTE (8%), tumour compression (7%), hospitalisation at the time of VTE diagnosis (23%), chemotherapy (25%), radiotherapy (21%), hormonal therapy (10%), surgery (8%). 26% were smokers. 36 patients were not anticoagulated after their initial VTE because of contraindications, including brain metastasis, terminal illness or recent bleeding. 34 of these 36 patients died, 23 within three months of initial VTE, including ten of PE. The median survival time was 5.2 months for DVT and three months for PE. Survival plateaued at 22% for DVT and 16% for PE.

Conclusions VTE is a relatively common problem in cancer patients. The high recurrence rate and mortality within one year emphasise the need for better understanding of the role of predisposing factors and better guidelines for prophylaxis.

Venous thromboembolism (VTE) is a common clinical problem associated with significant morbidity and mortality. While cancer patients appear to be at a higher risk of having VTE, estimates of incidence range from 1% for women on tamoxifen for breast cancer up to 17% for women on chemotherapy for advanced breast cancer.1,2 There are few guidelines for prophylaxis in cancer patients other than in the post operative period. The aim of this study was to establish the incidence of clinically significant DVT and PE in a nonselected series of cancer patients and determine whether known risk factors could be identified as a background to developing a guideline for prophylaxis.

The Christchurch Hospital Oncology Service receives referrals from Christchurch, North Canterbury, and many parts of the Northern half of the South Island of New Zealand. Christchurch Hospital is the only acute hospital in Christchurch to which patients with VTE are referred. The Oncology Service maintains a comprehensive prospective database on all referred patients, whether inpatients or outpatients.
Patients with complications from their cancer or its treatment are referred back to the Oncology Service for further treatment, since there is no other service provider. These data were used to investigate the epidemiology of cancer patients who have had VTE between 1995 and 1999.

Methods

**Study population.** Clinically significant DVT or PE was defined as that requiring hospital therapy or admission or that which was diagnosed during an inpatient stay. In the last year of the study, some patients who would have been admitted for anticoagulation were instead managed by the new outpatient haemostasis service and these have been included. The hospital records were searched for patients who were registered with the Oncology Service, Christchurch Hospital and had a diagnosis of pulmonary embolism (PE) and/or deep venous thrombosis (DVT) between January 1995 and December 1999. The International Classification of Diseases, Tenth Revision (ICD-10-AM) codes were used; for PE, ICD-10-AM diagnosis code 415.1, 415.11, 415.19, and for DVT, ICD-10-AM diagnosis code 451.1, 451.11, 451.19, 451.2, and 451.8 (includes venous thrombosis of both lower and upper extremities). The following data were recorded; gender, birth date, diagnosis, symptoms and signs from VTE, previous VTE, method of diagnosis, treatment, any complications, survival from diagnosis of VTE, status of disease at diagnosis of VTE, anticoagulant use and any subsequent episodes of VTE. The study period predated CT pulmonary angiography for diagnosis of PE. Possible predisposing factors for PE and DVT were recorded including hospitalisation at the time of PE and DVT diagnosis; chemotherapy (CT), radiotherapy (RT), hormonal therapy (HT), or surgery within the previous three months; history of smoking; family history of VTE; and documentation of compression by tumour on the limb(s) with venous thrombosis.

To estimate the number of patients at risk for incidence calculation, the number of new patients referred to the Oncology service between 1995 and 1999 was obtained from the Oncology database. This comprehensive database documents birth date, date of referral, date of cancer diagnosis, type of cancer, date of radiotherapy, chemotherapy, hormonal therapy and date of death. The incidence of VTE was calculated as the number of patients presenting with VTE occurring in an age-sex group during the five year period from January 1 1995 through December 31 1999, divided by the number of patients referred to the Oncology Service in the same age-sex group in the same time period. The patients were divided into two groups, depending on whether the initial episode in the five year time period was DVT or PE with/without DVT. The cumulative survival rate was determined using Kaplan-Meier survival analysis.

Results

Over the five year study period, January 1995 to December 1999, a total of 7987 new cancer patients were registered, of whom 2147 received at least one course of chemotherapy and 4809 at least one course of radiation treatment. 51.9% were female. During this same five years, 106 patients presented with 122 episodes of VTE. DVT occurred in 68 (56%) cases and PE with/without DVT in 55 (44%). Patient details at the initial episode of VTE are shown in Table 1. Diagnosis of DVT was ascertained by duplex ultrasonography in 38 cases (56%), ultrasound guided venogram in four cases (6%) and by the association of clinical symptoms and signs in 26 cases (38%). Diagnosis of PE was ascertained by moderate to high probability lung V/Q scan with high clinical suspicion in 27 cases (50%), and by the association of clinical symptoms and signs in the remaining 27 cases (50%).

Figure 1 shows the age- and gender-adjusted incidence rates. The overall incidence rate for the first episode of VTE during the study period was 1.3% (95%CI, 1.1-1.5). The incidence of DVT was 0.75% (0.56-0.94) and of PE (with/without DVT) was 0.55% (0.39-0.71). No patients below the age of 20 years had an episode of VTE. However, they made up less than 1.2% of the referrals to the Oncology Service.
Table 1. Demographic features in cancer patients presenting with DVT or PE, January 1995 – December 1999.

<table>
<thead>
<tr>
<th></th>
<th>DVT</th>
<th>PE</th>
<th>Total VTE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number</strong></td>
<td>61</td>
<td>45</td>
<td>106</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>35 (57%)</td>
<td>21 (47%)</td>
<td>56 (53%)</td>
</tr>
<tr>
<td><strong>Age (years), mean ± SD</strong></td>
<td>63.2 +/-13.3</td>
<td>60.5 +/-13.6</td>
<td>61.9+/-.13.3</td>
</tr>
<tr>
<td><strong>Treatment in last 3 months</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>14 (23%)</td>
<td>12 (27%)</td>
<td>26 (25%)</td>
</tr>
<tr>
<td>Radiation</td>
<td>9 (15%)</td>
<td>13 (29%)</td>
<td>22 (21%)</td>
</tr>
<tr>
<td>Hormonal therapy</td>
<td>8 (13%)</td>
<td>3 (7%)</td>
<td>11 (10%)</td>
</tr>
<tr>
<td>Surgery</td>
<td>6 (10%)</td>
<td>2 (4%)</td>
<td>8 (8%)</td>
</tr>
<tr>
<td>Hospitalisation, current</td>
<td>11 (18%)</td>
<td>13 (29%)</td>
<td>24 (23%)</td>
</tr>
<tr>
<td>Tumour compression</td>
<td>7 (11%)</td>
<td>0 (0%)</td>
<td>7 (7%)</td>
</tr>
<tr>
<td>Previous VTE</td>
<td>4 (7%)</td>
<td>4 (9%)</td>
<td>8 (8%)</td>
</tr>
<tr>
<td>Family history VTE</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

Figure 1. Age and gender adjusted incidence rates of venous thromboembolism

Table 1 summarizes the prevalence of risk factors for VTE presenting during the study time period. The three commonest potential risk factors (excluding ‘having cancer’) were CT within three months, hospitalization and RT within three months. Each of these risk factors was found in more than 20% of cancer patients with VTE. 71% of patients with VTE had one or more risk factors other than having cancer. Thus, of the 106 patients with VTE, 39% had one risk factors, 27% two risk factors and 5% three or more risk factors. Fourteen patients with DVT (23%) and fourteen with PE (31%), 28 total (26%) were smokers, similar to the population frequency. The incidence of DVT in the 2147 patients receiving chemotherapy was calculated as 0.8% (seventeen cases), and the incidence of PE was 0.7% (15 cases) giving a total incidence of VTE of 1.5%. For the 4809 patients receiving radiation, the incidence of DVT was 0.2% (9 cases), and of PE was 0.33% (16 cases) giving a total incidence of
VTE of 0.52%. Recalculation excluding patients from outside North Canterbury gave a similar incidence.

Out of the total 106 patients, nineteen had two or more episodes of VTE. The total number of recurrences in these nineteen patients was 24. The overall recurrence rate was 22.6%. The male cancer patients had a recurrence rate of 32%, compared with 14% for females. Six of the nine males having their first episode of VTE below the age of 50 years had a recurrent episode. Eleven of the 24 (46%) recurrent VTE episodes occurred within four months of the previous episode, but three (12%) occurred more than three years later. At the time of the recurrent VTE episode, twelve of the 24 patients were on no anticoagulant prophylaxis, seven (29%) were on warfarin, three (13%) were receiving low molecular weight heparin and two were taking aspirin. No reason was documented for not using anticoagulation in six patients, but three had recent bleeding, and three had last experienced VTE more than three years ago.

After diagnosis of the first VTE event during the study period, 70 patients were started on oral anticoagulation (OAC). The remaining 36 were not anticoagulated; seven because they were terminal, seven because of cerebral tumour (primary or secondary), six had recent bleeding, three died at presentation with PE, and no reason was given for ten. The outcome was compared with respect to whether OAC was used or not (Table 2). Among the patients who were not given OAC, 63.8% died within three months of their first VTE episode, including 27.8% from PE. In the patients who were given OAC, the three months mortality was 28.1% and only 2.8% from PE. Among the patients who were given OAC, four had brain metastases and one had recent haematuria. These five patients died within three months of their initial episode. Complications of OAC were relatively rare. Out of 70 patients who were given OAC, there was only one episode of major intracranial haemorrhage and three episodes of minor bleeding, none being fatal.

Table 2. Outcome after VTE (1995-1999) whether or not oral anticoagulation (OAC) was used.

<table>
<thead>
<tr>
<th></th>
<th>Anticoagulation</th>
<th>No anticoagulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>70</td>
<td>36</td>
</tr>
<tr>
<td>Mortality within 3 months</td>
<td>20 (28.6%)</td>
<td>23 (63.8%)</td>
</tr>
<tr>
<td>Mortality due to PE</td>
<td>2 (2.9%)</td>
<td>10 (27.8%)</td>
</tr>
</tbody>
</table>

Of the twelve patients recorded as dying of PE, this was judged as significantly shortening expected survival in two patients (low grade lymphoma, partial remission after chemotherapy; recurrent cerebral lymphoma, first cycle of chemotherapy) and probably shortening survival in three (carcinoid lung with brain metastases, mesothelioma and thyroid cancer with lung metastases).

Survival curves after the diagnosis of PE or DVT are shown in Figure 2. One year survival was 22% for PE and 39% for DVT. The median survival from the time of diagnosis of DVT was 5.2 months (CI 0.9-9.5 months), and from the time of diagnosis of PE was 3 months (CI 1.4-4.6 months). The survival curves reached a plateau at 22% for DVT and 16% for PE after about three years.
Discussion

This study, carried out in a well defined region of New Zealand, found an overall incidence of VTE of 13.1 per thousand (1.3%). The incidence in this nonselected cancer population was higher than the VTE incidence in the general population found in other studies, where the rates varied between 0.2-2.7 per thousand.3-9 Our denominator is well established from the oncology database, and the most likely inaccuracy is that patients were not referred in to the oncology service for treatment of VTE. Thus the incidence is at least 1.3%. Previous studies have shown an increasing incidence with age.3-6 However, in our cancer population women showed a constant incidence with age while men showed the highest incidence in the 40-49 year age group. In general populations,3,6 PE accounted for an increasing proportion of events of venous thromboembolism with increasing age, from 22% for the age groups 20-39 years to 40% above 75 years. However, in our study, the proportion of PE was 50% for the age group 20-39 and 40% above 80 years. This difference may be due to relatively small numbers and less intensive ascertainment in elderly unwell patients with cancer. Some oncology patients were referred from outside the North Canterbury area, and VTE episodes could have been treated locally without referral to Christchurch Hospital, but recalculation of incidence using data from only the North Canterbury area yielded the same incidence.

Hospitalisation, chemotherapy and radiotherapy were the three commonest potential predisposing factors in both the DVT and PE populations. 70% of VTE patients had one or more risk factors, in addition to having cancer. Smoking was recorded since it has been shown to be a risk factor in some studies,10 and was present in 26% of cases.
However, without a non-PE, non-DVT control group, the relative risk conferred by these factors cannot be calculated. The incidence of VTE in the total population of patients who were receiving chemotherapy was similar to the overall VTE incidence, at 1.5%, in contrast to reports of rates as high as 17% associated with breast cancer chemotherapy.\(^2\) Underdiagnosis is unlikely while on chemotherapy because of close and regular clinical review.

The recurrence rate of VTE in these cancer patients was higher than the rates reported in the general population.\(^7,11\) The overall recurrence rate in our cancer population was 22.4% compared with general population rates of 2-9% and 17.2% in two older studies.\(^7,11\) Medicare Claims Data risk analysis has shown a similar 22% chance of recurrent VTE within six months in patients with malignancy compared with 6.5% if there is no malignancy.\(^12\)

The case fatality for both PE and DVT was high over short term among the cancer patients, and the diagnosis of PE and DVT appeared to be a marker for early mortality. A Danish registry study\(^13\) and a study in elderly patients\(^3\) both found an increased mortality rate for patients with VTE who had cancer. An autopsy study has shown that PE is more likely to be fatal if it occurs in cancer patients.\(^14\) However, it remains unclear whether the poorer prognosis for cancer patients with VTE is due to their malignancy or VTE complications. The outcome of the patients in our study who were given OAC was better than that of those not given OAC which may reflect the decision not to give OAC in patients with poor prognosis from their cancer. Furthermore, five patients appeared to have significantly poorer survival because of a fatal PE. There is some support for a lower mortality from cancer if anticoagulation for VTE is used for six months rather than six weeks\(^15\) but a randomised trial found no survival benefit from prophylactic warfarin used in small cell lung cancer.\(^16\) According to Bona et al\(^17\) there did not appear to be a clinically significant increased risk of bleeding complications in cancer patients treated with oral anticoagulation.

Our study has some limitations. Death due to pulmonary embolism may go unrecognised without autopsy, while symptomatic patients may not come to the attention of physicians. In addition, some cases might not have been reported after being diagnosed outside the area, or some deep venous thromboses may have occurred without exhibiting symptoms, especially among surgical patients and immobilised medical patients. Only DVT/PE episodes that warranted hospital treatment were thought to be clinically important in this population, and hence the classification of ‘clinically significant’ DVT or PE. Data for this region, however, are likely to be more complete than from centres with more than one service provider. Therefore, the calculated incidence is an estimate of the true VTE incidence in the cancer population. Nevertheless, since under-recognition is most likely, the rate of VTE is at least 1.3% in this unselected series of oncology patients.

Our data support the claim that the incidence of VTE is increased in cancer patients, but we have not found an excessive risk in our chemotherapy population. Recurrence rates appear to be higher than in noncancer populations and there is a significant mortality within the first few months, suggesting the need for anticoagulation unless very strong contraindications are present. The relatively high frequency and high mortality in cancer patients emphasises the need for better risk assessment and guidelines for prophylaxis.
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Acknowledgements: Steven Joung undertook this work as a Christchurch School of Medicine summer studentship. We are grateful to Chris Frampton for statistical advice, Rowena Fisher for secretarial assistance, and all our colleagues in the Oncology Service who treated the patients.

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References:


Pat Fox has died (14/4/02), and for this we are the poorer. For all who knew him he was a luminary, a raconteur, and it is a matter of great sadness that Alzheimer's robbed these gifts from him in his final years. Throughout his career a consultation with Pat was an opportunity for an anecdote, and the only complaint of his patients would be that the surgery would be finished before the story was complete.

Pat was born 29/7/18 in Masterton, the second son of five children. He grew up in Hastings and received his secondary school education as a boarder at St Patrick's, Silverstream. He excelled at sport and with two fellow students won an international shooting competition using .303 Lee Enfield rifles held amongst all the secondary schools of the British Empire. From school he entered the seminary at Greenmeadows in Hawkes Bay but after two years the discovery of more earthly pleasures persuaded him to abandon a calling to the priesthood and convert to medicine. Denied overseas service in the Armed Forces during World War II by the government policy of insisting medical students complete their studies, he graduated MB ChB (Otago) in 1944. He remained a committed, practising Catholic throughout his life.

While in Dunedin Pat represented Otago University, the province of Otago and New Zealand Universities at rugby playing hooker with a vigour that might be described as highly competitive. He had New Zealand University blues in rugby, shooting and water polo. His early postgraduate years were spent in Auckland, where his family now lived, and where he met Rosemary Garland before travelling to the United Kingdom. He obtained dual Membership of the RCPs (Edinburgh and London) in 1948 and pursued his studies in dermatology, particularly in Edinburgh. Rosemary had followed him to the UK and in 1949 they married. They returned to New Zealand in 1951.

Pat commenced private practice and took up a visiting consultant's post at Auckland Hospital where he gave 30 years service to the public health system. He continued in his private practice until retirement in 1992. Pat's dermatologic skills were recognised by the wider medical community and his faithful patients with a flourishing practice as well as his dermatology colleagues who always appreciated his educated, common sense approach to the specialty. He was a foundation Fellow of the Australasian College of Dermatologists (1967) and became FRCP (Edinburgh) in 1972 and FRACP (1977). His attitude and sayings will survive him in the practice of those who learned from him and those who they in turn have taught. Of a suspect pigmented lesion: "No one ever died from a surgical scar". His compassionate approach to the elderly: "They are never too old to do the right thing".

Pat was, dermatologically, a Renaissance man with skills in dermatologic medicine, surgery and radiotherapy. His practice commenced before the introduction of topical steroids when inflammatory dermatoses that are now relieved in days of outpatient therapy required weeks of inpatient therapy and Auckland Hospital had two dermatology wards. His large hands and large stitches cured many, and the occasional white cat with a squamous carcinoma on its nose that sought refuge after treatment
behind the radiotherapy unit in his rooms simply added colour to his already rich vocabulary.

Pat always wore well-tailored suits with a white shirt to work. He suffered from recurrent polyneuritis and always maintained his physical fitness to combat this. He enjoyed swimming and was President and Patron of the Parnell swimming club. His interest in rugby was lifelong and he coached the Auckland University rugby team to win the premier club rugby competition. He lived the outdoor life, with duck shooting and fly-fishing among his leisure pursuits; his golf swing never quite came right, which remained a great consternation to him. His long partnership with Rosemary ended with her sudden death in 1999; their five children and their families survive him. His coffin left St. Michael’s church to the tune of "Bye Bye Blackbird".

We are grateful to Dr Leicester Hodge for this obituary notice.
Southern Trauma Symposium, 8-9 November 2002.

Hotel Sofitel Melbourne, VIC, Australia.

Please contact Emma Waygood, STS Manager, Conference Action Pty Ltd, PO Box 576, Crows Nest, NSW, 1585 Australia.
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19th Annual Scientific Meeting of the Australasian College for Emergency Medicine, 17-21 November 2002.

Sheraton on the Park, Sydney, Australia.

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Anticoagulation and non-traumatic splenic rupture

Although anticoagulant-associated bleeding fatalities following valve replacement for bacterial endocarditis are not common (NZ Med J 2002; 115: 124-6), clinicians should have a high degree of suspicion for such events because delayed recognition tends to increase mortality. One diagnosis which may be initially confusing is nontraumatic splenic rupture. While splenic infarction is not uncommon in patients with bacterial endocarditis, nontraumatic splenic rupture after bacterial endocarditis is rare but can be fatal if not recognised and treated early. Reports have documented nontraumatic splenic rupture in patients medically managed for bacterial endocarditis who survived after splenectomy,1-4 whereas other clinicians have reported endocarditis patients treated with a combination of medical management, vegetectomy or valve replacement who died following splenic rupture.5-7 The routine postoperative anticoagulation of patients undergoing valve replacement may act in conjunction with endocarditis-related pathology of the spleen (eg infarcts, varices or abscesses) to increase the likelihood of rupture,6-7 although the rarity of the latter condition given the number of patients on anticoagulation therapy and the prevalence of splenic infarction makes one question such an association. Nontraumatic rupture has also been reported in patients undergoing thrombolytic therapy for conditions such as acute myocardial infarction, pulmonary embolism, atrial fibrillation and mitral stenosis.8-10

The diagnosis of nontraumatic splenic rupture is sometimes difficult. It has been mistaken for myocardial infarction, bacterial peritonitis, pulmonary embolism, acute pancreatitis and appendicitis. Delay in diagnosis and treatment can be devastating. Some common signs and symptoms are left-sided pleuritic pain which may radiate to the left shoulder, hypotension, tachycardia, peritoneal signs, abdominal distension, left upper quadrant mass and a rapid, unexplained drop in haematocrit. Computed tomography, ultrasonography and peritoneal lavage are useful diagnostic tools. Surgical intervention should be considered early if splenic rupture is suspected.

Recently, I lost a patient to this complication of bacterial endocarditis, weeks after valve replacement and the subsequent initiation of anticoagulation therapy with coumadin. Clinicians need to remain vigilant in the postoperative monitoring of patients with bacterial endocarditis. Splenic rupture in endocarditis patients is not common, but one has to think of this diagnosis in order to make it.

Dr James Bradley Summers,
Department of Internal Medicine, University of South Alabama, USA.

Retention of body parts: reflections from anatomy

D Gareth Jones, Kerry A Galvin.

In February 2002, it was revealed publicly that up until as late as 1996, Green Lane Hospital in Auckland had been retaining children’s hearts following postmortem examinations without parental consent, or indeed knowledge. Similar practices have been uncovered in the United Kingdom and Australia, leading to public inquiries. At both the Bristol Royal Infirmary and the Alder Hey Children’s Hospital in Liverpool children’s organs were removed at postmortem and retained without parental consent.1,2 At the Institute of Forensic Medicine in New South Wales, the situation was similar but in this case, long bones and joints were removed from adult cadavers, once again with lack of consent from next-of-kin.3 In all of the above cases, the rationale behind the retention of organs and tissues was research and teaching.

A variety of scandals have also come to light in the United States, involving anatomy departments, tissue banks, nonprofit and for-profit private biotechnology companies4,5 and more recently, crematoria.6 Medical schools have become embroiled in some of these situations.7 While commercial pressures skew some of these American cases, the underlying drive remains the centrality of human tissue for research, therapy and teaching. Even more broadly, there is a global traffic in human organs, which is now being widely acknowledged.8

Discussion of these and a growing number of similar incidents is dominated by the adequacy or otherwise of the legal situation, and the role of informed consent in obtaining human material of potential value in research and in teaching. This material is generally obtained at postmortem, although some may emanate from bodies bequeathed to anatomy departments. However, further reflection reveals that incidents such as these raise a host of far broader issues concerning the availability of human material for research and teaching. They also point to noteworthy inconsistencies in the manner in which use of this material is regulated. This problem is not peculiar to New Zealand.

Human Tissue Act 1964

This Act controls many facets of the use of human tissue in New Zealand, although it is far from comprehensive. Its major thrust appears to be oversight of the bequest of human bodies to medical schools, and in particular anatomy departments, for dissection. This is referred to as an ‘anatomical examination’ which means “examination of a body or any part of a body for the purpose of the study of the science of anatomy”. It also controls the retention of material from such bodies. Also included in the Act are stipulations regarding postmortems and the removal of human tissue for therapeutic purposes or for use in medical education or research. Of particular significance is the establishment of Schools of Anatomy, where anatomical examinations will be carried out, the appointment of licensed anatomists within these Schools, and of inspectors (senior police personnel) to oversee all aspects of cadaver usage and retention in Schools of Anatomy. Underlying the Act is the stipulation that postmortems and anatomical examinations are to be carried out in an ‘orderly, quiet
and decent manner’, undoubtedly reflecting the dignity and respect to be accorded the dead human body.9,10

From this it is evident that Schools of Anatomy are strictly controlled in their activities, and in the procedures they employ to obtain and subsequently use human bodies. Informed consent is integral to the bequest process, thereby separating it ethically from the use of unclaimed bodies.9,10 The latter occurred in New Zealand up to the 1950s and is still found in a wide range of other countries today.11

Similar control over anatomy departments and the use of cadaver bequests takes place in the United Kingdom, with its Anatomy Act 1984, which covers the acquisition of bodies for anatomical examination. In addition, there is the Human Tissue Act 1961, covering the use of bodies for research and teaching following postmortems. The Anatomy Act is highly regulated with a government appointed inspector who visits anatomy departments regularly, whereas less attention has been given to oversight of the Human Tissue Act.12

The recent incidents involving the retention of body parts at postmortem in New Zealand and other countries raise a number of issues, quite apart from those widely discussed in the media. These additional issues emerge when the focus of discussion is shifted to the nature of the requirements expected of anatomy departments when they are in receipt of cadaveric donations.

**Model of bequests**

Whenever there is no consent for the use of body parts following a postmortem, the potential exists for a double tragedy – the tragedy of the death itself, plus the tragedy of the (unknown) retention of body parts. In the types of situation which have arisen recently, the two tragedies may be separated by many years. The grief of the initial loss is compounded by the reawakened grief when it is revealed that organs have been retained unbeknown to the relatives. This is a stark reminder of the double penalty experienced in early British anatomy history, where the penalty of execution was exacerbated by the penalty of dissection, thereby preventing burial in sacred ground.13

While this is far from the intention when body parts are used for research, the end-result is unnervingly similar.

In contrast, when consent is obtained, the death may to some extent be redeemed for the relatives by giving them the opportunity to bequeath body parts of the deceased to be used for good ends.14 This is akin to organ transplantation following a tragic death, on condition that the body parts are freely willed by next-of-kin. The driving force in these instances is altruism, which is far preferable ethically to the double tragedy alternative.

Another parallel is between bequeathing bodies for anatomy teaching and research, on the one hand, and using unclaimed bodies on the other.9 Reliance upon unclaimed bodies becomes problematic on the premises that cadavers have intrinsic and instrumental value, that the manner in which they are treated is of moral interest, and that giving one’s body for dissection or donation is preferable to coercion. The lack of consent inherent within it points to the inability of the person concerned to defend their own bodily integrity, and the lack of opportunity to offer their bodies for the good of others.
Model of anonymity

There is a need to distinguish very clearly between a situation where the organs available for study or research are anonymous, and where they are identifiable. Anonymous specimens are those for which identifying details were not originally collected, or if collected, have since been lost or destroyed. In contrast, identifiable samples will have personal information such as a name or patient number attached, and can thus be readily identified. The identifiability, or lack thereof, of organs and tissues has wide-ranging implications for the way in which they should be treated.

A common theme emerging in recent recommendations is that anonymous tissue or organs may be used for teaching and researching purposes without consent. For example the 1999 report by the National Bioethics Advisory Council in the United States makes a clear distinction between stored tissue which is fully anonymous and that which has identifiable links to a patient. This report concluded that already existing anonymous specimens stored in repositories and museums do not fall under the regulatory definition of research with human subjects. As such, informed consent is not required for any subsequent use of this tissue. Further, any restrictions imposed on the use of stored anonymous tissue could potentially seriously hinder valuable and worthy research projects. A similar recommendation has been made by Jones and Harris in relation to prehistoric human skeletal material where there are no known descendants.

In contrast, research on identifiable specimens can be considered human subjects research, so that fully informed consent and review should be mandatory for any further uses of the tissue. Whenever material is identifiable, it fits alongside bequeathed bodies, and it is this model that should be used for analysing specimens of this nature. It should come under some jurisdiction, possibly an ethics committee or the Inspector of Anatomy model.

A question which arises is whether material that is originally identifiable should remain identifiable. The value of being identifiable stems from the availability of a clinical history, rather than the availability of personal details. One imagines the two could be kept separate, and could be dealt with separately. This does not dispense with a need for informed consent; rather, it points to the necessity for a mechanism for tracking and auditing the material. It also suggests that mere storage of such material for years on end does not constitute adequate justification for retaining it. There must be a convincing scientific or educational rationale.

Unlike anonymous tissue, identifiable material can never be regarded as archival. Organs in this category have known associations, which have to be taken into account. This is akin to human skeletal material where there are known descendants. This places identifiable material in a separate category from that of old museum specimens of unknown origin.

Model of ongoing control

What emerges from the above is that the control exercised over bodies bequeathed to anatomy departments is far more stringent and over-arching than that exercised in any of the other areas discussed. There are a number of differences.

First, informed consent is central to the bequest of bodies (the use of unclaimed bodies is allowed for under the Human Tissue Act 1964, although this has not been exercised for many years in New Zealand). However, there is still lack of clarity over
the demands for informed consent in the use of body parts following postmortems, although we have argued that this should never be by-passed.

Second, the Inspector of Anatomy has responsibility for overseeing all body parts in anatomy departments, including those retained indefinitely. In contrast, there appears to be no formal mechanism for tracking body parts obtained at postmortem, even when there has been informed consent. There is no parallel to the Inspector of Anatomy. The anachronism is that, even in anatomy departments, body parts obtained from postmortems do not fall under the jurisdiction of the Inspector of Anatomy. Hence, even in these departments, there is a two tier regulation of body parts.

Third, body parts obtained at operation and subsequently used with consent for research or teaching purposes, are subject to a human ethics committee. This regulates the research or teaching conducted on the material, although even in this instance one has to ask how stringently the process oversees any subsequent use of this material.

Fourth, if one were to use unclaimed bodies in an anatomy department, they would still be regulated by the Inspector of Anatomy. As theoretical as this may be in New Zealand, it provides a model for oversight of body parts (whether identifiable or anonymous) obtained without consent at postmortem. A means of auditing these latter body parts would seem to be essential.

Fifth, organs collected for research and obtained without consent at postmortem, will not have been approved by any ethics committee. Consequently, no one has jurisdiction over the collection. As a result, this human material is subject to far less constraints than material obtained from experimental laboratory animals. The unsatisfactory nature of this situation appears to be self-evident.

Model of ethical awareness

Regardless of the precise legal requirements that may emerge to regulate the use of human cadavers and body parts from a variety of sources, medical schools and allied hospitals need to be characterized by an ethical awareness of these issues. It is not sufficient for health professionals in the current ethical climate to resort to perceived legal ambiguity or lack of clarity as justification for using human material without adequate informed consent. It would be sad if the medical profession gave the impression that it is incapable of acting ethically without legislative oversight. After all, we expect scientists and even students to do this, since we do not accept the legitimacy of scientific fraud or plagiarism.

It has been evident for very many years that the uses to which any society will allow human material to be put is relative to that society’s moral values. Every use of such material requires justification, and the bounds are set by society, since there is no automatic right to use human material for research or teaching purposes. Future uses of body parts depend on social acceptance, and on convincing the public that the uses to which material will be put justify the public’s support. The perceived benefits of the research have to be set alongside the ethical environment in which the research is to be conducted. This should encourage the medical profession to be extra vigilant about its performance in this area.

The problem with the Green Lane situation, and indeed the other overseas examples appears to be a lack of ethical awareness. What is required is an understanding of why the human body or human tissue should be treated with dignity and respect. Once this is appreciated, the role of informed consent follows automatically, as does a shift in
emphasis from ‘taking’ and ‘retaining’ to ‘donation’. Serious teaching of ethical principles to medical students and all those associated with the medical profession is obligatory.

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John Hutchison Saunders

John Saunders, born in 1920, spent his childhood in Lower Hutt, attended Scots College, where he showed ability as an athlete, then went to Dunedin where he graduated MB.ChB in 1943. He was soon serving overseas as a medical officer, in a General Hospital in Italy, then later in Japan.

He travelled to London, as aspiring surgeons did in those days, and obtained both his English Fellowship and a comprehensive grounding in orthopaedics, which included two years at the Royal National Orthopaedic Hospital. While in London he married Margaret Salmond, a 1948 Otago graduate; they were to have two daughters and a son. They returned to Wellington in 1952, where John had a year as senior orthopaedic surgeon at Wellington Hospital. He remained in this post until his retirement in 1985, which was itself an achievement in a period when orthopaedic surgeons in Wellington were not noted for their longevity. At various times he also provided an orthopaedic service in Marlborough and the Wairarapa.

He was a careful but dextrous surgeon (always a gifted craftsman, he had built an additional room on to his first home in Lower Hutt) and his opinion, though given with some reticence, was always valued by his colleagues. He spoke little at meetings and published less, but this was a measure of his modesty and he was a wise and thoughtful contributor when he did speak. As a medicolegal witness he was impartial, precise and convincing; as an adviser to governments and their agencies, scrupulously fair.

He served on the Court of Examiners of the Royal Australasian College of Surgeons for a decade from 1962. He was secretary of the New Zealand Orthopaedic Association for a term in the 1960s, and its vice-president 1977-79. His OBE, totally unsought, touched him profoundly as he became aware of the regard in which he was held.

Always a good sportsman, he played rugby as a student, was a keen skier and a competent tennis player; but his abiding challenge was golf, at which he achieved a single-figure handicap. Indeed he celebrated his first total hip replacement by winning the New Zealand Orthopaedic Association's golf cup.

Unfortunately golf of that quality takes a toll of joint replacements, and John's later years were tarnished by ill-health; but he could still achieve a flash of pleasure when old friends called on him, and Margaret's loyalty was outstanding. They had, after all, been more than a mere husband-and-wife team, for she might at one point be his anaesthetist and at another might scrub with him.

At his funeral I was prompted to observe that in over forty years as colleagues, he and I had never exchanged a cross word. There are plenty of people in respect of whom I would not make such a claim.

We are grateful to Mr AW Beasley for the obituary notice.
Roger Neale Wallis

Immediately following Roger's birth in Christchurch on 16/10/19, the Wallis family shifted to Rotorua when his Father was appointed Medical Superintendent to Rotorua Hospital. Roger was educated at Southwell Primary School and Christ's College. As a youth he showed great sporting ability, representing his school at Rugby and at age 18 was a member of the Bay of Plenty cricket team. At a young age he was taught, by professionals, the rudiments of golf and fly fishing which was obvious in later life by the skill shown at both sports. At the outbreak of World War II he joined the Fleet Air Arm as a pilot, served in all the theatres of war and during 1945, until the war against Japan ended, he was engaged in aerial mapping of Japanese occupied South-East Asia. This task was carried out in a plane equipped with only a camera.

After demobilisation Roger studied medicine at Dunedin graduating in 1952. He spent a year as a house surgeon at New Plymouth hospital and then joined the practice of the late Dr James Church. When Dr Church retired Roger carried on a solo general practice, becoming a member of the RNZCGP until his own retirement ten years ago. Roger had all the attributes of a caring practitioner - always available to his patients, and his consultations and visits were never hurried; many of his patients became lifelong friends. He was appointed a Medical Officer of Special Scale and worked at both public and private hospitals as an operating assistant for the orthopaedic surgeons. His orthopaedic colleagues regarded his work for twenty years as of the highest order.

Roger chaired the Taranaki Division of the NZMA at the time when the annual meeting of the New Zealand Association was held in New Plymouth in the early 60’s. He was an honorary medical officer to both the Taranaki Rugby and Cricket Associations, Port Health Officer, Past President of the Officers Club and a member of the Brevet Club. He was awarded a silver medal by the Stratford Mountain Club in recognition of his service to skiing and played a big part in getting FIS races in New Zealand. He was also a deeply knowledgeable jazz enthusiast.

Roger had suffered a stroke just after his retirement which limited his activity but not his enjoyment of life. His final illness was swift and met with characteristic calm. He died in New Plymouth 23/2/02. He was predeceased by his wife Pat, whom he met and married in Colombo when she was a Wren and is survived by his children Caroline and Colin and their families, whose loss is shared by a host of friends. We are grateful to Dr RM Davie for this obituary notice.