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Twice monthly except December & January
Preventing neonatal HIV infection

There have been significant successes in control of spread of the human immunodeficiency virus (HIV) in New Zealand as the rate of spread has been reduced within the homosexual male population, eliminated from therapeutic blood products and controlled within the injecting drug-using community. With these successes there has come a change in the epidemiology of HIV infection. More cases in New Zealand are now being reported in heterosexual males and females. In the 12 months to 21.12.98, 43.7% (51 cases, 15 female) of new cases were within this patient group. This raises the risk that infants will become infected by this route. This tragedy can be prevented in most cases if the infection is detected during pregnancy and known preventative measures implemented.

Control strategies
The ACTG trial 076 showed that administration of oral zidovudine during the second and third trimesters of pregnancy, intravenously during labour and orally to the infant during the first six weeks of life reduced transmission from mother to child from 25.5% to 8.3%. Thus for every 5.9 patients treated, one infection will be prevented. Adverse effects on mother and fetus were minimal and follow-up studies of the treated infants up to the age of five years have shown no late adverse effects. Several subsequent studies have also reported significant reduction of neonatal infection, with either the maternal or infant part of the protocol only, and have also failed to demonstrate significant ongoing adverse effects to either mother or infant.

The continued but reduced rate of infection suggested that zidovudine alone was not sufficiently potent to control HIV replication adequately and there are ongoing studies with more potent regimens combined with viral load monitoring in pregnancy. At present there is no consensus as to which drugs are most effective but recent guidelines include zidovudine where possible. Monotherapy remains an option and may be considered early in the course of HIV infection in patients with a low viral load.

The other reason for continuing infections was increased exposure to maternal blood at the time of delivery. Initial studies of twins and the effects of Caesarean section on transmission yielded conflicting results, but a new European cohort study has demonstrated that elective Caesarean section can prevent transmission. In this study of 2834 singleton pregnancies in HIV-infected women, 902 received zidovudine during pregnancy. Of these treated pregnancies, transmission occurred in 6.4% delivered vaginally, compared with 0.8% in those delivered by elective Caesarean section at 37 weeks gestation. In comparison, transmission was 11.4% (p=0.002) in those delivered by emergency Caesarean section. Multivariate analysis showed that HIV transmission was five-fold lower with elective Caesarean section than vaginal delivery, and also that, in other cases the transmission rate was significantly lower with Caesarean section than other forms of delivery, provided there was membrane rupture of less than four hours duration. While this was not a randomised study, the results are sufficiently compelling to establish Caesarean section as an effective means of further reducing transmission from mother to child. It is also prudent to avoid invasive monitoring and augmented labour by rupture of membranes.

Breast-feeding has been convincingly shown to be an important route of HIV transmission and may double the rate of infant infection in undiagnosed maternal HIV infection. Studies in the United Kingdom show that most women chose not to breast-feed if diagnosed before delivery and thus education at this time was an effective preventative measure.

Not only does the identification of HIV-infected pregnant women offer the mother the chance to prevent transmission to her baby, but it also offers her the possibility of treatment with the highly effective antiviral therapy that undoubtedly prolongs life of good quality in many patients, as well as the possibility of diagnosis and treatment of other members of her family.

Diagnostic considerations
The techniques for diagnosis of HIV are now well established and have been well evaluated. The new generation tests are extremely reliable. The system of using a screening test with a confirmatory Western blot has a very high sensitivity (>99%) and specificity (>99.98%) so that the false positive rate is extremely low (0-6.0 per 100 000 people tested) even in low prevalence populations. Despite this, positive tests should always be
The present policy in New Zealand has been to offer HIV testing if risk factors for transmission are detected or a woman requests a test. This means that consideration of HIV testing depends heavily on the practitioner’s interest and knowledge of HIV and belief in the value of early diagnosis. The diagnosis may also be missed if the sexual partner’s risk factors, such as previous drug use or bisexuality, are not known to the patient. There is no information as to how often tests are offered in New Zealand but it is likely to be highly variable and the overall rates low. Studies in the United Kingdom, where there had been a policy of offering testing to all women in areas of known or suspected high prevalence and elsewhere to women with recognized risk factors, reported widely differing rates of testing (3-51%) despite broadly similar stated policies. Furthermore, results of unlinked anonymous surveys compared with information contributed by obstetricians showed only 16% of HIV-positive women were known to their obstetricians prior to delivery and, of these, only 5% were diagnosed during pregnancy.

There have been calls within New Zealand to make the diagnosis in pregnancy a higher priority but the resulting action is unclear. More recently, attention has been given to improving the rate of antenatal testing in the United Kingdom. Other countries such as France and Sweden have been performing much better than the United Kingdom. New Zealand should follow these initiatives. A key element of the revised programme includes the “normalisation” of the HIV test by being offered and recommended to all women at their initial antenatal visit, alongside other blood tests. There will be debate as to whether such a policy should be implemented in all geographic locations as most cases are likely to occur in major centres however, this should not delay addressing the issue in these centres. An inclusive policy need not cause anxiety or harm to the system had they failed them dismally if they were denied the chance to consider and implement preventative measures.

ST Chambers, D Tcele, DR Aickin, Christchurch,
K Grimwood, Wellington.

4. Mercey D. Antenatal HIV testing has been done badly in Britain and needs to improve. BMJ 1998; 316: 151-2.
17. Mercey D. Antenatal HIV testing has been done badly in Britain and needs to improve. BMJ 1998; 316: 241-2.
Cardiovascular risk factors levels of Pacific people in a New Zealand multicultural workforce

David Schaaf, HRC Training Research Fellow; Robert Scragg, Senior Lecturer in Epidemiology; Patricia Metcalf, Biostatistician; Department of Community Health, University of Auckland, Auckland.

Abstract

Aims. To compare cardiovascular risk factors among the major Pacific Island communities participating in a New Zealand multicultural workforce survey.

Method. There were 650 employed Pacific Island participants (Samoan 357, Cook Islands 177, Tongan 71, Niuean 45), aged 40-65 years, who were interviewed in a work-based, cross-sectional survey. During an oral glucose tolerance test, blood samples were collected for determination of blood glucose and serum lipids. Participants provided information on smoking and leisure time physical activity. Blood pressure, weight and height were measured and body mass index calculated. Ten-year risk of cardiovascular disease was calculated using equations from the Framingham study.

Results. Among men, their ten-year risk of a cardiovascular event was similar for the four communities compared (range 11.5% to 13.2%). However, individual risk factors did vary between the ethnic groups with Cook Island men having significantly higher total cholesterol, blood pressure and urinary microalbumin than other Pacific Island ethnic groups, while Tongan men were more likely to smoke and had lower HDL levels than other groups. Among women, Samoan and Cook Island participants had significantly higher ten-year cardiovascular risk scores (5.7%) than Niuean (4.4%) and Tongan (3.7%), due primarily to elevated total cholesterol levels.

Conclusion. Cardiovascular risk factor levels vary between Pacific Islands communities in New Zealand. Targeted interventions to specific Pacific communities may be more beneficial than the current homogeneous prevention strategy applied to all communities.

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short of breath (i.e. aerobic), as moderate if it was non-aerobic, while participants who did neither were classified as inactive. This definition of activity, which is commonly used in epidemiological studies, is associated with serum levels of the major lipids. Blood pressure was measured twice, after a five-minute rest in the sitting position, with a Hawksley random zero sphygmomanometer and averaged. Weight (to the nearest 0.2 kg) and height (nearest 0.5 cm) were measured with shoes and heavy clothes removed. Blood lipids were measured on fasting serum samples. Commercial methods were used to measure total cholesterol and triglycerides (Technicon, Tarrytown, NY) and HDL cholesterol (Boehringer, Mannheim, Germany). LDL-cholesterol was calculated by the Friedwald formula for participants with fasting triglycerides ≤4.50 mmol/L. Blood glucose was measured by using a Cobas Fara centrifugal analyser (F Hoffman-La Roche, Basle, Switzerland). The WHO criterion for epidemiological studies (two-hour glucose >11.1 mmol/L) was used to determine diabetes status.11

Body mass index (BMI) was calculated by dividing weight (kg) by the square of height (m). A summary ten-year risk score of cardiovascular disease was calculated for each participant using equations from the Framingham Heart Study. This was based on age, sex, systolic blood pressure, cigarette smoking status (current smoker or quit within last year), diabetes status (currently treated or undiagnosed) and serum total cholesterol.

Means or proportions, for all variables, were calculated using, respectively, PROC GLM and PROC FREQ in SAS. Variables which were not normally distributed (triglyceride and microalbuminuria) were logged and tolerance factors (1.96 x standard error) calculated.

Results

Data for men and women, separately, are shown in Tables 1 and 2. Ethnic-specific mean ages did not differ between the four Pacific groups within each sex (p>0.05). Therefore, there was no need to adjust for age when making ethnic comparisons.

Cigarette smoking varied between Island groups among men, being highest for Tongan (56%) and lowest for Cook Island (33%) and Niuean (34%) (Table 1); while among women, who had smoking prevalences varying from 6% to 24%, there were no significant ethnic differences (Table 2). Leisure time physical inactivity levels were similar for the four communities within each sex, although in contrast with smoking, women were more likely to be inactive than men. Ethnic-specific diabetes prevalences were also similar (p>0.05).

There were also ethnic differences in biological variables. BMI was significantly higher in Samoan women compared to other women (Table 2), while there were no ethnic BMI differences in men. Systolic and diastolic blood pressures were significantly higher in Cook Island men compared with each of the three other ethnic groups (Table 1). Mean total cholesterol and LDL levels were highest in Cook Island men and women, while HDL-cholesterol was lowest in Tongan men. There were no significant ethnic differences in mean levels of fasting and two-hour plasma glucose, of fasting triglyceride, and of the ratio of total cholesterol to HDL cholesterol, among both men and women. Mean level of microalbuminuria was significantly higher in Samoan and Cook Island men compared to Tongan and Niuean (Table 1). The summary ten year risk score of cardiovascular disease (i.e. the probability of having a cardiovascular disease event within the next ten years) was higher in Samoan and Cook Island women than in Tongan and Niuean (Table 2). By contrast, there were no significant ethnic differences in risk score among men; while their ten year risk (range 11.5% to 13.2%) was more than double that of women (range 3.7% to 5.7%).

Discussion

These results show that there are differences in the levels of some cardiovascular risk factors between the Pacific Island communities in New Zealand. Among men, although their ten-year risk of a cardiovascular event was similar for the four communities compared, individual risk factors did vary between the ethnic groups. Cook Island men had significantly higher total cholesterol, blood pressure and urinary microalbumin than other Pacific Island ethnic groups; while Tongan men were more likely to smoke and had lower HDL levels than other groups. Among women, Samoan and Cook Island participants had higher ten year cardiovascular risk scores, due primarily to elevated total cholesterol levels, compared with Tongan.

Possible limitations in our sample include: its work-based sampling frame which may not be representative of the

Table 1. Comparison of cardiovascular risk factors among Pacific Island men aged 40-65 years.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Samoan</th>
<th>Cook Island</th>
<th>Tongan</th>
<th>Niuean</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>205</td>
<td>105</td>
<td>55</td>
<td>12</td>
</tr>
<tr>
<td>Age in years (mean SE)</td>
<td>46.9 (0.4)</td>
<td>48.0 (0.5)</td>
<td>47.3 (0.7)</td>
<td>47.9 (1.0)</td>
</tr>
<tr>
<td>Smokers (%) *</td>
<td>46%</td>
<td>33%</td>
<td>56%</td>
<td>34%</td>
</tr>
<tr>
<td>Inactive leisure time (%)</td>
<td>47%</td>
<td>38%</td>
<td>47%</td>
<td>34%</td>
</tr>
<tr>
<td>Diabetes (%) #</td>
<td>8%</td>
<td>8%</td>
<td>5%</td>
<td>13%</td>
</tr>
</tbody>
</table>

Means (SE)

| BMI (kg/m²)                | 31.1 (0.3) | 31.3 (0.4) | 30.9 (0.6) | 29.6 (0.8) |
| Blood pressure (mmHg)     | 126 (1.0)  | 113 (1.3)   | 126 (1.8)  | 126 (2.4)  |
| Systolic                  | 81 (0.8)   | 86 (1.1)    | 79 (1.5)   | 78 (1.9)   |
| Diastolic                 |            |             |           |           |
| Plasma glucose (mmol/L)   | 6.08 (0.16) | 6.24 (0.22) | 5.77 (0.30) | 6.64 (0.40) |
| Fasting 2 hour            | 5.76 (0.27) | 6.13 (0.37) | 5.19 (0.51) | 6.47 (0.67) |
| Serum lipids (mmol/L)     | 6.02 (0.08) | 6.53 (0.11) | 6.05 (0.15) | 5.71 (0.20) |
| HDL-cholesterol           | 1.15 (0.02) | 1.25 (0.03) | 1.10 (0.04) | 1.15 (0.05) |
| Triglycerides             | 1.53 (1.08) | 1.62 (1.12) | 1.46 (1.16) | 1.16 (1.22) |
| LDL-cholesterol           | 4.09 (0.08) | 4.45 (0.13) | 4.25 (0.15) | 3.86 (0.20) |
| Ratio Total/HDL           | 5.50 (0.11) | 5.20 (0.16) | 5.84 (0.22) | 5.25 (0.28) |
| Microalbuminuria (mg/L)   | 11.8 (1.19) | 15.2 (1.27) | 6.7 (1.40)  | 6.3 (1.55)  |
| Ten-year risk score of CVD| 11.5%     | 12.9%       | 13.2%     | 11.6%     |

CVD: cardiovascular disease; *Current smokers plus those who stopped in last 12 months; p-value for c² test <0.05; #Treated plus undiagnosed diabetes; Geometric mean (tolerance factor).
Cardiovascular disease in Polynesians. Evidence that serum cholesterol is not related positively to risk score can be applied to Pacific Islands people given ethnic differences; and the assumption that the Framingham small sample sizes for Tongan and Niuean participants includes those receiving government benefits); the relatively Pacific Islands people communities in New Zealand. Each Zealand. If we are to reduce the high rates of cardiovascular disease in Pacific Islands people in New Zealand. may have important policy implications for preventing may be due to cultural differences in lifestyle. This finding the four major Pacific Island groups in this survey, which ethnic differences of cardiovascular risk factor levels between higher prevalences are higher than those in our study (Table 1).

Despite these possible concerns, previous cross-sectional studies carried out between 1978 and 1987 in 15 Pacific population groups from nine countries, excluding Tonga, have identified ethnic differences in cardiovascular risk factors similar to our findings.14 These studies observed higher levels of total cholesterol, blood pressure and BMI in Cook Island men compared with Samoan and Niuean. The findings in women, while not as consistent with our results, showed higher levels of total cholesterol, diastolic blood pressure and BMI in Cook Island and Samoan women than Niuean. In terms of lifestyle risk factors, Samoan men (75%) had a higher percentage of smokers compared with Cook Island (40%) and Niuean (63%), although these smoking prevalences are higher than those in our study (Table 1).

Overall, our results suggest that there are significant ethnic differences of cardiovascular risk factor levels between the four major Pacific Island groups in this survey, which may be due to cultural differences in lifestyle. This finding may have important policy implications for preventing cardiovascular disease in Pacific Islands people in New Zealand. If we are to reduce the high rates of cardiovascular disease events in Pacific Islands people in New Zealand we need to acknowledge that there are ethnic differences among Pacific Islands people communities in New Zealand. Each community has a unique culture and lifestyle, just as for the various immigrant European communities. Targeting interventions to specific Pacific communities may be more beneficial than the current homogeneous strategy for prevention applied to all communities.

Acknowledgements. Major funding for the Workforce Diabetes Study was provided by the New Zealand Health Research Council. Supplementary funds were received from Lotteries Medical Research and the National Kidney Foundation.

Correspondence. David Schaaf, Department of Community Health, University of Auckland, Private Bag 92019, Auckland.

Table 2. Comparison of cardiovascular risk factors among Pacific Island women aged 40-65 years.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Samoan</th>
<th>Cook Island</th>
<th>Tongan</th>
<th>Niuean</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>152</td>
<td>72</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>Age in years - mean (SE)</td>
<td>46.7 (0.4)</td>
<td>47.6 (0.6)</td>
<td>45.4 (1.3)</td>
<td>47.2 (1.4)</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>24%</td>
<td>21%</td>
<td>6%</td>
<td>15%</td>
</tr>
<tr>
<td>Inactive leisure time (%)</td>
<td>55%</td>
<td>51%</td>
<td>69%</td>
<td>69%</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>9%</td>
<td>10%</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Means (SE)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Samoan</th>
<th>Cook Island</th>
<th>Tongan</th>
<th>Niuean</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI (kg/m²)</td>
<td>35.0 (0.5)</td>
<td>32.6 (0.7)</td>
<td>31.9 (1.5)</td>
<td>32.3 (1.7)</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>123 (1.1)</td>
<td>124 (1.6)</td>
<td>122 (3.6)</td>
<td>123 (3.8)</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>77 (0.7)</td>
<td>78 (1.1)</td>
<td>76 (2.4)</td>
<td>73 (2.6)</td>
</tr>
<tr>
<td>Fasting</td>
<td>6.07 (0.19)</td>
<td>6.02 (0.27)</td>
<td>5.53 (0.58)</td>
<td>5.46 (0.65)</td>
</tr>
<tr>
<td>2 hour</td>
<td>6.62 (0.33)</td>
<td>6.91 (0.48)</td>
<td>5.71 (1.02)</td>
<td>4.48 (1.13)</td>
</tr>
<tr>
<td>Total cholestrol</td>
<td>5.84 (0.10)</td>
<td>6.04 (0.14)</td>
<td>5.14 (0.29)</td>
<td>5.28 (0.13)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>1.29 (0.02)</td>
<td>1.29 (0.03)</td>
<td>1.18 (0.07)</td>
<td>1.33 (0.08)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>1.10 (1.08)</td>
<td>1.24 (1.12)</td>
<td>1.17 (1.25)</td>
<td>1.18 (1.38)</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>3.98 (0.08)</td>
<td>4.10 (0.12)</td>
<td>3.66 (0.25)</td>
<td>1.51 (0.29)</td>
</tr>
<tr>
<td>Ratio Total/HDL</td>
<td>4.66 (0.10)</td>
<td>4.83 (0.15)</td>
<td>4.76 (0.31)</td>
<td>4.15 (0.15)</td>
</tr>
<tr>
<td>Microalbuminuria (mg/L)²</td>
<td>8.4 (1.19)</td>
<td>8.8 (1.28)</td>
<td>6.8 (1.70)</td>
<td>5.8 (1.80)</td>
</tr>
<tr>
<td>Ten-year risk score of CVD</td>
<td>5.7%</td>
<td>3.7%</td>
<td>3.7%</td>
<td>4.4%</td>
</tr>
</tbody>
</table>

CVD: cardiovascular disease; *Current smokers plus those who stopped smoking in last 12 months; †Treated plus undiagnosed diabetes; ¶Geometric mean (tolerance factor); ¶p<0.05 vs Tongan ‡p<0.05 vs Niuean ¶p<0.05 vs Cook Island.

wider Pacific population living in New Zealand (which also includes those receiving government benefits); the relatively small sample sizes for Tongan and Niuean participants which may have prevented the detection of other significant ethnic differences; and the assumption that the Framingham risk score can be applied to Pacific Islands people given evidence that serum cholesterol is not related positively to cardiovascular disease in Polynesians.15

Cystic fibrosis diagnosed in adult patients

Tanya J McWilliams, Registrar; Margaret L Wilsher, Respiratory Physician; John Kolbe, Respiratory Physician, Respiratory Services, Green Lane Hospital and Department of Medicine, University of Auckland, Auckland.

Abstract

Aim. To review the presentation, diagnosis and long-term, clinical follow-up of cystic fibrosis in adult patients diagnosed in adulthood at Green Lane Hospital.

Methods. A retrospective review of the case notes of patients with cystic fibrosis diagnosed in adulthood at Green Lane Hospital or referred there for management. Information was collected on diagnostic tests, including sweat tests and genotyping. Relevant family history was documented as were spirometry results and microbial colonisation.

Results. Six patients conclusively fulfilled the diagnostic criteria for cystic fibrosis. There was a wide range of ages at diagnosis (18-68) and half of the patients had a positive family history. A single mutation was identified in all, but in only one of the cases was the second mutation identified. All patients had evidence of bronchopulmonary suppuration and all had retained pancreatic function. Colonisation with P aeruginosa was associated with marked impairment in lung function.

Conclusion. The patients at Green Lane Hospital represent part of the broad-spectrum disease in adult patients diagnosed with cystic fibrosis and highlight the differences between this group and those patients diagnosed in childhood with the more classical phenotype. Patients generally have less severe lung disease and retain pancreatic function. Sweat testing is useful diagnostically but gene testing is of limited value in making the diagnosis.

IN PRACTICE

Cystic fibrosis (CF) is the most common, lethal genetic disease amongst Caucasians in New Zealand. It has an incidence of about 1 in 2500 live births (in New Zealand 1 in 3179 Caucasian births) and 1 person in 25 is an asymptomatic carrier. The gene responsible for CF was cloned in 1989 and codes for the cystic fibrosis transmembrane regulator (CFTR), a phosphorylation-regulated, chloride channel located in the apical membrane of epithelial cells.

Although the disease is caused by mutations in this single gene coding for CFTR, the disease has a variable clinical phenotype. The most common mutation is ∆F508, due to a deletion of phenylalanine in the 508 position on the CFTR gene and comprises approximately 80% of the mutations in New Zealand. The classic picture of cystic fibrosis is that associated with the ∆F508 homozygote genotype and includes pancreatic insufficiency, male infertility and severe sino-pulmonary disease.

In such patients, colonisation with P aeruginosa and S aureus may be associated with deterioration in lung function. The typical electrophysiological picture is of abnormal transport of sodium and chloride ions across the apical membranes of affected cells. There are more than 600 mutations in the CFTR gene now identified. While clinical variation exists within a single genotype due possibly to modifying genes or environmental and management factors, it is well recognised that certain genetic types are associated with a milder phenotype, with retention of normal pancreatic function and milder lung disease.

The spectrum of disease associated with the CF gene is much wider than previously thought. Some patients who have milder disease and lack the typical features are only diagnosed in adulthood. It is important that general practitioners and physicians are aware of these atypical presentations of CF so that the correct diagnosis can be made and appropriate management (including genetic counselling) and long-term follow-up instituted. To illustrate this point we reviewed patients with CF presenting in adult life in Auckland.

Methods

We retrospectively reviewed the case notes of patients with CF diagnosed in adulthood and currently under the care of Green Lane Hospital. We included patients diagnosed at Green Lane Hospital and those referred there for management. Information was collected on demographics, clinical presentation, lung function, diagnostic tests (specifically sweat tests and genotyping) and relevant family history. Exocrine pancreatic function at the time of presentation to Green Lane Hospital was assessed on history and confirmed if necessary with a three-day, faecal fat test or a faecal elastase test. Patient progress from diagnosis was followed with spirometry and microbial colonisation.

Results

The diagnosis of CF depends on presentation with an appropriate phenotype (or sibling with CF) and evidence of abnormal ion and water transport (elevated sweat electrolytes and/or abnormal nasal potential differences). Six patients fulfilling this definition have been diagnosed as adults at Green Lane Hospital.

All patients were European (one patient was born in South Africa, the others in New Zealand) and five out of six were female. None were related. The age at diagnosis ranged from 18 to 68 years. Three patients had no family history of CF. One patient was diagnosed at the age of 68 after her grandmother who had died of chronic suppurative respiratory disease. Only two of our patients were smokers.

All patients presented with respiratory symptoms. Two had a history compatible with bronchiectasis. One had a long history in adulthood and was finally diagnosed with CF at the age of 31 in 1996. Two further patients had chronic cough as a presenting complaint. A further patient, diagnosed at the age of 68, primarily had sinusitis. In our group of patients computed tomography (CT) scans of the chest had been performed as part of the original diagnostic work-up some time before the diagnosis of CF was made. Three patients had CT scans showing upper lobe bronchiectasis and one had a lower lobe pattern. At least two of these patients were initially diagnosed with bronchiectasis.
and a typical pattern for CF on CT was one clue to the correct diagnosis.

Diagnosis was confirmed in all by a positive sweat test (the sweat sodium results ranged from 64 to 127 mmol/L). Five out of six of these patients had an identifiable mutation in the CF gene (Table 1). All cases had normal pancreatic function. Immunoglobulin levels were uniformly normal.

Table 1. Adult patients with cystic fibrosis at Green Lane Hospital.

<table>
<thead>
<tr>
<th>Age (at diagnosis)</th>
<th>Gender</th>
<th>FEV1 (% predicted) at diagnosis</th>
<th>Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37 F</td>
<td>42</td>
<td>F508/R117H</td>
</tr>
<tr>
<td>2</td>
<td>19 F</td>
<td>82</td>
<td>F508/</td>
</tr>
<tr>
<td>3</td>
<td>31 F</td>
<td>78</td>
<td>not known</td>
</tr>
<tr>
<td>4</td>
<td>18 F</td>
<td>76</td>
<td>F508/</td>
</tr>
<tr>
<td>5</td>
<td>68 F</td>
<td>115</td>
<td>R117H/</td>
</tr>
<tr>
<td>6</td>
<td>27 M</td>
<td>42</td>
<td>F508/</td>
</tr>
</tbody>
</table>

There was a range of severity of respiratory disease. In our patients one was colonised with Aspergillus species and Mycobacterium avium intracellulare. However, this man and another patient had \( P \) aeruginosa in their sputum. Both of these patients had FEV\(_1\)'s below 50% predicted at diagnosis in contrast to the other four. Two patients have had a decline in lung function tests during follow-up and were the only two smokers in this group.

Discussion

Patients with cystic fibrosis diagnosed later in life rather than in childhood have variable and atypical presentations, and often have milder disease\(^1\) and a better, long-term prognosis.\(^1\) Correct diagnosis allows institution of correct therapy, more informed discussion of prognosis and appropriate genetic counselling. The following discussion addresses issues of the diagnosis, genetic basis and varied clinical picture of CF in these patients.

Diagnosis. All of our patients satisfied the current criteria for the diagnosis of CF, namely a compatible phenotype plus abnormal sweat electrolytes. In one patient a CF mutation could not be identified amongst the limited number of mutations tested for in New Zealand. In three patients there was also a family history of CF but in only one was this in a sibling. The range of normal values for sweat sodium and chloride, in children younger than 15 years is less than 60 mmol/L and 95% of results are less than 40 mmol/L. However, levels of sweat sodium and chloride normally rise slowly throughout childhood and early adulthood. Values of greater than 60 mmol/L are considered abnormal in adults and the causes of false positives are readily excluded on clinical grounds. In CF, levels of sweat sodium and chloride are usually in the range of 80-190 mmol/L.\(^1\) A minimum of 0.04 g of sweat is required for a reliable result.

Genetics. Recent advances in genetic testing have identified a large number of CFTR mutations; some are becoming associated with particular phenotypes. The range of mutations includes other amino acid deletions, missense (point) mutations, e.g. \( R \) 117H, \( R \) 334W and \( R \) 347P,\(^4\) nonsense (stop code) mutations and frame-shift mutations. The most common sites for clinically significant mutations are the two-nucleotide binding domains. Patients who are \( \Delta F508 \) homozygotes have no CFTR in the apical membranes of their sweat glands, (although some remains in the cytoplasmic perinuclear granules) and generally have severe sino-pulmonary disease and pancreatic insufficiency.

Cystic fibrosis mutations are usually described as mild or severe depending on whether they are associated with pancreatic sufficiency\(^3\) (\( R \) 117H, \( R \) 334W, \( R \) 347P, \( A \) 445E and \( P \) 547H) or insufficiency (\( A \)F508 and \( G \)551D). In some patients one “mild” mutation is enough to preserve pancreatic function even when paired with a “severe” allele, which can also be differences in severity of disease within the IVST-8 genotype when the quantity of functioning CFTR is affected by alternative splicing of the mRNA.\(^7\)

The relationship between genotype and phenotype is not so clear in lung as in pancreatic disease. One study\(^1\) in the Netherlands found that patients with the \( A \)445E mutation had less severe lung disease. They had better lung function, were less likely to be colonised with \( P \) aeruginosa, and had less pancreatic insufficiency. This occurred even when the allele paired with \( A \)445E was one usually associated with severe disease (e.g. \( A \)F508, 1717-1G-A, E60X, G542X and R553X).

In our study the patients were all pancreatic sufficient as has been the case in previous studies.\(^1,3\) Similarly, all of our patients presented late. Although four of our patients were \( \Delta F508 \) heterozygotes, in only one case was the other allele able to be identified (\( \Delta F508/R117H \)). The \( R117H \) allele is known to be associated with milder disease.\(^3\)

The patients included in this review all had their genotype analysed in New Zealand. Four out of six were \( \Delta F508 \) heterozygotes with the other allele being identified in only one case. All of these \( \Delta F508 \) heterozygotes must have a second allele with a CFTR mutation and it is this allele that usually determines the severity of disease. Since all four of the patients who are \( \Delta F508 \) heterozygotes were pancreatic sufficient we must assume that their other allele is one which has a mutation that codes for pancreatic sufficiency. One patient was a \( R117H \) heterozygote, the other did not have a genotype that can currently be identified in New Zealand. As only limited genotyping is undertaken in New Zealand, the failure to detect two mutations does in no way exclude the diagnosis of CF in this group.

Clinical features. Respiratory symptoms predominated in this group of patients, with all having retained exocrine pancreatic function. This is consistent with organ-specific variable dependence on CFTR within the sweat glands, pancreas and lungs, the lungs needing a higher functioning level of CFTR than the pancreas. The vas deferens is generally considered to require the highest functioning of CFTR of any organ and, therefore, the fertility of one of our male patients is extremely unusual.

Two of our patients were colonised with \( S \) aureus and \( P \) aeruginosa and these two both had FEV\(_1\)'s below 50% predicted, at presentation. In patients who are homozygotes for \( \Delta F508 \), colonisation with \( P \) aeruginosa and \( S \) aureus is usually associated with deterioration in lung function.\(^3\)

In our patients who presented with “bronchiectasis”, radiological features were helpful in making the diagnosis of CF, particularly when other potential causes such as allergic bronchopulmonary aspergillosis and immunoglobulin deficiency had been excluded. A typical CT scan in cystic fibrosis may demonstrate cylindrical bronchiectasis with peribronchial thickening, peripheral nodular opacities and ring shadows. Bronchial cysts (56%) and interstitial cysts (32%) may also be present. A distribution of apical rather than basal bronchiectasis is often a clue that cystic fibrosis, rather than idiopathic bronchiectasis, is the underlying diagnosis.

Conclusion

CF is no longer thought of as a homogeneous disease due to a single genetic abnormality as is reflected in our group of patients. The spectrum of clinical presentations is increasing, as is the identification of new genetic abnormalities underlying them. People are presenting with mild forms of
Changes to infant sleep practices in Canterbury

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Abstract

Aim. “Reducing the risk” is a public health primary initiative to minimise the incidence of Sudden Infant Death Syndrome (SIDS) in New Zealand. A number of SIDS risks relate to infant sleep practices. We describe current prevalences of these practices.

Methods. A cohort of Canterbury mothers delivering live infants during May 1997 (n=411) were mailed a questionnaire in July surveying their infant’s sleep practices. Survey results were compared to results derived from the Canterbury control infant component of the 1987-90 New Zealand Cot Death Study (NZCDS) (n=174). Those mothers using either plastic or rubber mattress covers (n=63) were issued a subsequent questionnaire pertaining to this mattress-wrapping practice.

Results. Completed questionnaires were returned by 274 (66.7%) mothers. Room sharing with mother was usual for 133 (48.5%) infants, no different from the 94 (54.0%) recorded in the NZCDS ($\chi^2$=5.6, df=2, p=0.06). However, of those infants sharing a room with their mother, 101 (75.9%) slept in their own bed compared to 46 (48.9%) in the NZCDS ($\chi^2$=57.0, df=2, p<0.01). Only 8 (2.9%) infants were regularly placed prone to sleep, considerably fewer than the 69 (39.7%) reported in the NZCDS ($\chi^2$=101.1, df=1, p<0.01). Mattress-wrapping with plastic (14.6% vs. 4.0%; $\chi^2$=12.8, df=1, p<0.01) and rubber (8.4% vs. 3.4%; $\chi^2$=12.6, df=1, p<0.01) has significantly increased since the NZCDS. Results from the subsequent questionnaire, completed by 42 (66.7%) respondents, indicated that most, 25 (59.5%), wrapped their infant’s mattress to stop soiling. Less than half, 18 (42.9%), wrapped the mattress for the “safety of their baby”.

Conclusion. The “non-prone sleeping” campaign has been successful in Canterbury. Most infants are now routinely placed non-prone for sleep. Of those infants sharing a room with their mothers, an increased proportion is sleeping in separate beds. The use of “drycot” under-blankets and sheepskins has diminished. While impermeable mattress-wrapping usage has significantly increased, over three-quarters of Canterbury mothers did not use plastic or rubber mattress-covers on their infant’s beds.

Sudden Infant Death Syndrome (SIDS) risk has been demonstratively linked to the sleeping environment of the infant, both nationally and internationally.1–4 In New Zealand, researchers have identified that infant prone sleeping, bed sharing with smoking mothers and solitary room sleeping have been associated with increased SIDS risk.3,6,7 Since the promotion of these and other epidemiologically identified risk factors, the New Zealand SIDS rate has decreased by more than one third.8 Similar programmes promoting these factors have been equally successful in reducing SIDS incidence in many other countries.9,10

In New Zealand, it has also been claimed that mattress-wrapping with plastic or rubber sheeting prevents SIDS.11, 12 This mattress-wrapping theory has been given much public attention.13,14 incited debate15,16 and impelled the Ministry of Health to prepare a discussion document on the matter.17

This study surveys a cohort of mothers within the Canterbury region to understand the current infant sleeping practices. The results were then compared to control infants from the Canterbury component of the 1987-90 New Zealand Cot Death Study (NZCDS) to ascertain the direction and magnitude of any change in infant sleep practices.3,18

Methods

All live births within the Canterbury region during the month of May 1997 (n=411) were identified from the Department of Justice register of birth notification forms (RC9). In July, when the infants were eight weeks old, mothers of this birth cohort were each posted a short questionnaire for self-administration. Mothers were asked to tick the box(es) that best described the situation regarding “at night my baby usually sleeps”: in a room alone, in a room with mother or in a room with other(s); in a bed alone, in a bed with mother or in a bed with other(s); with over-bedding including sheet(s), blanket(s), duvet(s) and quilt(s); with under-bedding including sheet(s), blanket(s), sheepskin, plastic mattress cover, rubber mattress cover, mattress or other; on the back, side or tummy. Each mother was then asked to return the questionnaire in an enclosed stamped addressed envelope.

All respondents declaring that they used either plastic or rubber mattress covers (n=63) were mailed a second short questionnaire surveying their rationale for using such
mattress covers, how they covered their infants' mattresses and the type of mattress covers used. Again this questionnaire was self-administered and returned in an enclosed stamped addressed envelope.

Where possible, comparisons in sleeping practice frequencies were made with the control figures derived from the Canterbury component of the New Zealand Cot Death case-control Study (NZCDS). The methodological details of the NZCDS have been extensively described elsewhere. Within the Canterbury region, there were 174 eligible control infants. Questions pertaining to room sleeping arrangements, bed sleeping arrangements and infant sleep position were based on the usual practice over the last two weeks; while under-bedding such as sheepskin, plastic mattress cover, rubber mattress cover, "drycot" (a woollen, water-resistant, under-blanket) and other mattress coverings usage was based on the last sleep.

The standard Pearson's $\chi^2$-test was employed to analyse frequency tables. An a-level of 5% was deemed statistically significant.

Approval from the Southern Regional Health Authority Ethics Committee was given for this ongoing project to survey infant health practices in Canterbury.

## Results

### Infant sleep practices

From the cohort of 411 mothers, 274 (66.7%) questionnaires were completed and returned, 28 (6.8%) were returned undelivered as "returned-to-sender" (indicating that the mother had moved), one (0.2%) advised that the baby had been adopted and 108 (26.3%) were simply not returned. Infant sleep practice frequencies from this survey (1997) and the Canterbury component of the NZCDS (1987-90) are given in Table 1.

### Bed sharing

Of those infants usually room sharing with their mothers, significantly more infants slept in their own bed compared to those documented in the NZCDS (75.9% vs. 48.9%). Correspondingly, significantly fewer infants (20.3% vs. 48.9%) were sharing a bed with their mother ($\chi^2=57.0$, df=2, p<0.01).

For infants usually sharing a room with person(s) other than their mother, no significant change has occurred in bed sleeping practices between studies ($\chi^2=0.4$, df=1, p=0.54). Approximately 73% usually slept in their own bed while 27% shared their bed with someone other than their mother.

### Prone sleeping

The proportion of infants placed prone to sleep has reduced from more than ten-fold since that recorded in the NZCDS. Only eight (2.9%) mothers declared that their infants were usually placed on their front for sleep compared to 69 (39.7%) in the NZCDS ($\chi^2=100.1$, df=1, p<0.01).

### Mattress covers

There were 40 (14.6%) infants who usually slept upon a plastic mattress cover in 1997. This is more than a three-fold increase from the 7 (4.0%) recorded in the NZCDS ($\chi^2=12.8$, df=1, p=0.01). Similarly, the use of rubber mattress covering has increased, with 23 (8.4%) infants usually sleeping upon this type of cover in 1997 compared to the six (3.4%) recorded in the NZCDS ($\chi^2=4.4$, df=1, p=0.04). Sheepskin usage has diminished, with 84 (30.7%) infants placed on a sheepskin (most covered with a sheet) in 1997 compared to 80 (46.2%) recorded in Canterbury under-sheets were also used less frequently than that reported in the NZCDS ($\chi^2=18.9$, df=1, p<0.01) while there was no evidence of change in other waterproof cover usage ($\chi^2=0.8$, df=1, p=0.38).

### Plastic or rubber mattress-wrapping practices

Of the 63 mothers issued with a subsequent questionnaire, responses were received from 42 (66.7%) mothers, two (3.2%) were returned undelivered as "return-to-sender", and the remaining 19 (30.2%) were not returned. Results from this survey are presented in Table 2.

The majority of mothers, 25 (59.5%), covered their infant's mattress to stop soiling, while safety was among the reasons for mattress-wrapping for 18 (42.9%). In Table 2, respondents were separated into "Safety" and "Other" groupings. One mother answering that both safety and soiling were her reasons for mattress-wrapping was allocated to the "Safety" group.

In the "Safety" group, one (5.6%) had a cover that lay directly on top of the mattress and 15 (33.3%) had a cover that enveloped the entire mattress. By contrast, in the "Other" group, 20 (83.3%) had a mattress cover that lay on the mattress and three (12.5%) had a cover that encased the mattress ($\chi^2=25.2$, df=2, p<0.01). Of the 21 respondents reporting that they covered the top of their infant's mattress, ten stated that the top was partially covered. The remaining 11 respondents did not state the extent of their mattress top covering.

Method of attachment to the mattress was significantly different between "Safety" and "Other" groups: a sheet or blanket used in four (22.2%) versus nine (37.5%) cases; ties or elastic used in one (5.6%) versus 14 (58.3%) cases; and adhesive tape used in 13 (72.2%) versus one (4.2%) cases, respectively, ($\chi^2=23.1$, df=2, p<0.01).

Mattress-wrapping was encouraged by family or friends for five (11.9%) mothers and advised by a midwife in one (2.4%) instance. No respondents reported that their doctor or nurse advised them to wrap their infant’s mattress.

### Discussion

Information originating from various sources has been disseminated to the public promoting infant care practices

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**Table 1. Infant sleep practice frequencies for the 1997 survey (n=274) and the Canterbury component of the NZCDS (n=174).**

<table>
<thead>
<tr>
<th>Sleep practice variables</th>
<th>Survey (1997)</th>
<th>NZCDS (1987-90)</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baby sleeps in a room:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>alone</td>
<td>127 (46.4)</td>
<td>64 (16.8)</td>
<td>5.6</td>
<td>2</td>
<td>0.06</td>
</tr>
<tr>
<td>with mother</td>
<td>133 (48.5)</td>
<td>94 (54.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>other</td>
<td>14 (5.1)</td>
<td>16 (9.2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baby sleeps in mother's room:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>in own bed</td>
<td>101 (73.9)</td>
<td>46 (48.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>in mother’s bed</td>
<td>27 (20.3)</td>
<td>46 (48.9)</td>
<td>20.7</td>
<td>2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>in other's bed</td>
<td>5 (3.8)</td>
<td>2 (2.1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baby sleeps in other's room:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>in own bed</td>
<td>11 (78.6)</td>
<td>11 (68.8)</td>
<td>0.4</td>
<td>1</td>
<td>0.54</td>
</tr>
<tr>
<td>in other's bed</td>
<td>3 (21.4)</td>
<td>5 (31.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed covers under the baby:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>sheet</td>
<td>260 (94.9)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>blanket</td>
<td>97 (35.4)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>sheepskin</td>
<td>84 (30.7)</td>
<td>80 (46.0)</td>
<td>10.4</td>
<td>1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>plastic cover</td>
<td>40 (14.6)</td>
<td>7 (4.0)</td>
<td>12.8</td>
<td>1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>rubber cover</td>
<td>23 (8.4)</td>
<td>6 (3.4)</td>
<td>4.4</td>
<td>1</td>
<td>0.04</td>
</tr>
<tr>
<td>drycot</td>
<td>66 (24.1)</td>
<td>17 (18.9)</td>
<td>1.01</td>
<td>1</td>
<td>0.38</td>
</tr>
<tr>
<td>waterproof cover</td>
<td>7 (2.6)</td>
<td>7 (4.0)</td>
<td>0.8</td>
<td>1</td>
<td>0.38</td>
</tr>
<tr>
<td>other cover</td>
<td>27 (9.9)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep position:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>non-prone</td>
<td>264 (97.1)</td>
<td>105 (60.3)</td>
<td>100.1</td>
<td>1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>prone</td>
<td>8 (2.9)</td>
<td>69 (39.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Room sharing**: No statistically significant changes in the usual room sharing arrangements of the infant have occurred between the two studies ($\chi^2=5.6$, df=2, p=0.06). Approximately 51% of infants room shared with their mother, 43% of infants slept in a room alone and the remainder slept with others.
that purport to reduce the risk for SIDS.\textsuperscript{11,19} We examined the effect this information has had on such practices in Canterbury.

Infant prone sleeping has been associated with increased SIDS risk (odds ratio, OR: 3.53).\textsuperscript{1} The prone sleeping message continues to be heeded by most (97.1\%) Canterbury mothers, as it has since 1992.\textsuperscript{16,21}

Infant bed-sharing has been associated with increased SIDS risk, particularly amongst those with smoking mothers.\textsuperscript{6,7,22} In response to the public health message, the frequency of infant bed-sharing has substantially reduced. Infant room-sharing with one or more adults has been identified to reduce the risk of SIDS (OR: 0.27).\textsuperscript{6} This risk factor has received little promotion within the Canterbury region and was not significantly different from that measured in the NZCDS.

Bedding has also been related to SIDS risk in the NZCDS.\textsuperscript{2} Using “sheet only” under-bedding as the reference category, it was reported that “sheet and drycot” usage was associated with a statistically significant decrease in SIDS risk (adjusted OR: 0.42; 95\% confidence interval, CI: 0.25-0.70), while “sheet and waterproof (plastic, rubber and other)” usage had no such association (adjusted OR: 0.86; 95\% CI: 0.52-1.41).\textsuperscript{2} Moreover, “sheet and sheepskin usage (adjusted OR: 0.95; 95\% CI: 0.66-1.41) and “uncovered sheepskin” usage (adjusted OR: 1.28; 95\% CI: 0.72-2.29) were not significantly associated with SIDS.\textsuperscript{2}

Despite these results, in our current study, an increased number of Canterbury mothers used either plastic or rubber mattress covers. Conversely, ‘drycot’ and sheepskin under-bedding usage has declined.

Paragraph withdrawn – See editorial page 2

There have been substantial changes in infant sleep care practices following various promotional campaigns. The large fall in the Canterbury SIDS rate has been attributed to the avoidance of the prone sleep position.\textsuperscript{1} Statistically significant changes in bed sharing practices have occurred and may also have contributed to the decline in SIDS numbers. Although the use of plastic and rubber mattress-covers has increased, over three-quarters of Canterbury mothers did not use these on their infants’ beds.

Correspondence. Associate Professor Rodney PK Ford, Community Paediatrician, Community Paediatric Unit, Private Bag 4710, Christchurch.

Table 2. Overall and sub-grouped (by rational for wrapping) frequencies for region of mattress-wrapping and method of attachment (n=42).

<table>
<thead>
<tr>
<th>Region of mattress covered:</th>
<th>Overall n (%)</th>
<th>“Safety” n (%)</th>
<th>“Other” n (%)</th>
<th>χ² df p</th>
</tr>
</thead>
<tbody>
<tr>
<td>top</td>
<td>21 (50.0)</td>
<td>1 (5.6)</td>
<td>20 (83.3)</td>
<td>25.2   2 &lt;0.01</td>
</tr>
<tr>
<td>top and sides</td>
<td>3 (7.1)</td>
<td>2 (11.1)</td>
<td>1 (4.2)</td>
<td>3 (12.5)</td>
</tr>
<tr>
<td>all the mattress</td>
<td>18 (42.9)</td>
<td>15 (83.3)</td>
<td>3 (12.5)</td>
<td></td>
</tr>
</tbody>
</table>

Method of attachment:
- sheet or blanket
- ties or elastic
- adhesive tape

<table>
<thead>
<tr>
<th></th>
<th>Overall n (%)</th>
<th>“Safety” n (%)</th>
<th>“Other” n (%)</th>
<th>χ² df p</th>
</tr>
</thead>
<tbody>
<tr>
<td>sheet or blanket</td>
<td>13 (31.0)</td>
<td>4 (22.2)</td>
<td>9 (37.5)</td>
<td>23.1   2 &lt;0.01</td>
</tr>
<tr>
<td>ties or elastic</td>
<td>15 (35.7)</td>
<td>1 (5.6)</td>
<td>14 (38.3)</td>
<td></td>
</tr>
<tr>
<td>adhesive tape</td>
<td>14 (33.3)</td>
<td>13 (72.2)</td>
<td>1 (4.2)</td>
<td></td>
</tr>
</tbody>
</table>

*One mother answered that both safety and mattress soiling were her reasons for mattress wrapping. She was classified with those respondents mattress-wrapping for “Safety”.

14. Anonymous. While they were sleeping... Consumer 1997; October: 4-8.
Methanol poisoning

Roland J Meyer, Physician; Michael EJ Beard, Clinical Director General Medicine; Michael W Ardagh, Emergency Physician; Seton Henderson, Intensive Care Physician; Christchurch Hospital, Christchurch.

Abstract

Aims. This study examines clinical experience with methanol poisoning during a one-year period.

Methods. All admissions with the diagnosis of suspected methanol toxicity were analysed and the current guidelines for the management of this problem were reviewed.

Results. Twenty-four subjects were identified. Most had a history of chronic use of methylated spirits. Four died before admission to hospital and the other 20 patients had 26 admissions to hospital and form the basis for this report. Four patients died in the Intensive Care Unit. In total 11 patients were admitted to the Intensive Care Unit. Seven patients received haemodialysis. There was no correlation between the methanol level and the outcome. The strongest predictor of death or a poor outcome was a blood pH < 7.0. Some patients, in spite of potentially lethal methanol levels of up to 160 mmol/L, did not develop signs of toxicity.

Conclusions. The overall mortality was high and ethanol was given to most of the patients for up to several days. Some patients did not show any toxicity and some of those were not given ethanol. It is recommended that chronic meths drinkers, who are not acidaemic and are generally well, do not require ethanol treatment. Only the complete removal of methanol from methylated spirits will reduce the morbidity of this condition.

Methanol poisoning is not common but there have been regular reports of outbreaks.1,3 Methanol is used as a cleansing solution and to denature industrial alcohol. Methylated spirits, which contains 5% methanol, is used as a cheap alcohol substitute by individuals or groups who are alcohol-dependent. The presentation of classical methanol toxicity includes visual symptoms and a high anion-gap, metabolic acidaemia. Clinical guidelines for the management of toxicity recommend the use of alkali, haemodialysis, ethanol and folic acid but clinical trials have not been carried out to validate these recommendations.2,4-6 We report the experience in Christchurch over a one-year period during which there were two episodes and several sporadic cases.

Methods

Between 14 June 1995 and 4 June 1996, all cases with the principal diagnosis of toxic effect of methyl alcohol (ICD 9 Code No. 9801) were reviewed. The Christchurch Hospital admission records, the Emergency Department records and the database of the Intensive Care Unit (ICU) were screened and all case notes reviewed.

Results

During a one-year period, 20 individuals had a total of 26 admissions to Christchurch Hospital following ingestion of methanol containing beverages. The age range was 25 to 65 years, and all but two were men (Table 1).

There were two discrete episodes and, during one of these, there were ten further presentations to the Emergency Department with suspected toxic effects of methyl alcohol, not resulting in hospital admission. Eleven patients were admitted to the ICU seven of whom received haemodialysis. Four patients died after admission. Survivors' accounts, police information and ambulance records revealed four further patients who died out of hospital. All patients admitted to hospital were given folic acid and all but three were treated with ethanol. Those patients who had an acidaemia received intravenous sodium bicarbonate and haemodialysis.

Selected case reports

Several members of the crew of a fishing boat presented to a small rural hospital one and a half days after a party during which they had consumed methylated spirits. One seaman died before receiving medical attention and another died shortly after arrival. Four others (Patients 1-4) were flown to Christchurch Hospital.

Patient 1 initially presented with loss of vision and confusion. Shortly after arrival, he developed convulsions and later circulatory shock. His blood pH was 6.66 and his methanol level 64 mmol/L. Despite aggressive therapy he died nine hours later.

Patients 2 and 3 complained of blurred vision, with one also complaining of nausea. Both received a single course of haemodialysis using subclavian access, with good recovery. However, one patient developed subclavian vein thrombosis, secondary to the dialysis catheter, which necessitated treatment with thrombolysis and anticoagulation.

Eight months later there was a further episode (Patients 5 - 19), but this time originating in the city of Christchurch. Many of these patients were known to have a history of alcohol abuse and to consume methylated spirits regularly. During a drinking binge, unknown quantities of methylated spirits were consumed.

Patient 5 came by taxi to the Emergency Department with severe respiratory distress. Within minutes of arrival, she became comatose and developed profound shock. A severe metabolic acidemia (pH=6.82, one hour later pH=6.59), and also a high anion gap were noted. Despite treatment the patient died in the ICU 14 hours later.

Patient 6 presented to the Emergency Department within minutes of patient 5’s death. He had spent one day of voluntary abstinence and detoxification at a Christchurch city charity centre before developing respiratory distress, nausea, vomiting and severe abdominal pain. Within one hour of presentation he deteriorated and, despite treatment, he died 32 hours later.

News of the death of these patients quickly spread throughout the community and during the next three weeks there were a further 27 presentations to the Emergency Department with three admissions to the ICU (patients 6 - 8) and 14 admissions to a general medical ward (patients 8, 10 -18). Patients 8, 11, 12, 13 had to be hospitalised twice. Patients 7 and 13 had already presented several months earlier as sporadic cases. These patients had only minor symptoms of alcohol-related toxicity and none had an acidaemia.
Fatal outcome after ingestion of as little as 15 mL of concentrated methanol has been reported, but also there have been several reports of ingestion of more than 400 mL without any obvious sequelae of toxicity. In individuals who chronically abuse methanol-containing beverages a high tolerance has been reported in spite of blood methanol concentrations considered to be potentially lethal, but others without such a history have also been noted to remain unharmed. Methanol is cheaper than ethyl alcohol, it is readily available and often it is substituted for ethyl alcohol as an inebriating beverage. In New Zealand, the abuse of methylated spirits ("purple lady"), which contains 5% methanol and between 70 - 90% ethyl alcohol, is commonplace. Some countries, including Australia, have abolished the use of methanol to denature alcohol, limiting the availability of this substance for abuse, with a subsequent significant reduction in cases of toxicity.

Symptoms of classical methanol toxicity are visual disturbance, nausea and vomiting. Clinical signs on presentation are hyperaemia of the optic disc, mydriasis and abdominal tenderness possibly due to gastritis or pancreatitis. Kussmaul respirations or tachypnoea are signs of metabolic acidaemia. Convulsions and coma are signs of severe toxicity. The toxicity does not correlate with the availability of this substance for abuse, with a subsequent significant reduction in cases of toxicity.

Table 1. Clinical and laboratory findings.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Methanol (mmol/L)</th>
<th>Ethanol (mmol/L)</th>
<th>pH</th>
<th>Initial Status</th>
<th>Inpatient Stay (days)</th>
<th>Minimal therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30/M</td>
<td>64</td>
<td>21</td>
<td>6.66</td>
<td>Coma</td>
<td>1</td>
<td>CAVHD</td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>28/M</td>
<td>36</td>
<td>0</td>
<td>7.12</td>
<td>Nausea</td>
<td>4</td>
<td>HD</td>
<td>Recovery</td>
</tr>
<tr>
<td>3</td>
<td>25/M</td>
<td>31</td>
<td>0</td>
<td>7.26</td>
<td>Visual</td>
<td>11</td>
<td>HD</td>
<td>Recovery</td>
</tr>
<tr>
<td>4</td>
<td>41/F</td>
<td>6</td>
<td>0</td>
<td>7.29</td>
<td>No Sx</td>
<td>2</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>5</td>
<td>45/F</td>
<td>10</td>
<td>0</td>
<td>6.59</td>
<td>Coma</td>
<td>1</td>
<td>CAVHD</td>
<td>Died</td>
</tr>
<tr>
<td>6</td>
<td>38/M</td>
<td>48</td>
<td>0</td>
<td>6.65</td>
<td>Coma</td>
<td>2</td>
<td>CAVHD</td>
<td>Died</td>
</tr>
<tr>
<td>7</td>
<td>30/M</td>
<td>20</td>
<td>2</td>
<td>7.39</td>
<td>Nausea</td>
<td>4</td>
<td>HD</td>
<td>Recovery</td>
</tr>
<tr>
<td>8</td>
<td>61/M</td>
<td>30</td>
<td>58</td>
<td>7.42</td>
<td>No Sx</td>
<td>9</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>9</td>
<td>63/M</td>
<td>13</td>
<td>10</td>
<td>7.43</td>
<td>No Sx</td>
<td>2</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>10</td>
<td>65/M</td>
<td>7</td>
<td>0</td>
<td>7.40</td>
<td>Nausea</td>
<td>3</td>
<td>Support</td>
<td>Recovery</td>
</tr>
<tr>
<td>11</td>
<td>44/M</td>
<td>25</td>
<td>59</td>
<td>7.42</td>
<td>Nausea</td>
<td>5</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>12</td>
<td>39/M</td>
<td>27</td>
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<td>7.40</td>
<td>Nausea</td>
<td>8</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>13</td>
<td>29/M</td>
<td>140</td>
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<td>7.44</td>
<td>Nausea</td>
<td>13</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
<td>14</td>
<td>32/M</td>
<td>130</td>
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<td>7.44</td>
<td>Nausea</td>
<td>3</td>
<td>Support</td>
<td>Recovery</td>
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<tr>
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<tr>
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<td>15</td>
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<td>Recovery</td>
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<tr>
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<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
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<td>49</td>
<td>7.37</td>
<td>No Sx</td>
<td>4</td>
<td>EOH</td>
<td>Recovery</td>
</tr>
<tr>
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<td>CAVHD</td>
<td>Disabled</td>
</tr>
<tr>
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<td>35/M</td>
<td>22</td>
<td>29</td>
<td>6.65</td>
<td>Coma</td>
<td>2</td>
<td>CAVHD</td>
<td>Died</td>
</tr>
</tbody>
</table>

Patients 1-9, 19 and 20 were admitted to the ICU; *=Readmission; Methanol= Methanol blood level on admission; Ethanol= blood alcohol level on admission; A pain=abdominal pain; CNS=CNS symptoms; Coma=Glasgow Coma Score 1; all these patients also were in a state of circulatory shock; No Sx=asymptomatic; EOH=per oral ethanol treatment; CAVHD=Continuous arterio venous haemodiafiltration; HD=Haemodialysis; Support=Supportive/systomatic therapy only.

Patient 13’s admission was his sixteenth, with an alcohol-related, medical problem in seven years. On this occasion he complained of nausea and vomiting. He was treated with oral ethanol given 1 - 2 hourly, receiving a total of 214 doses during his admission. His management on the ward was complicated by aggression, abuse and a tendency to wander in and out of the ward. After 13 days his methanol levels had decreased to near zero, he had shown no signs of toxicity and he was discharged.

Patient 16, who had had ten previous alcohol-related admissions since 1982, presented in a similar manner. He was brought in by a city charity for assessment because, he like patient 6, had been abstinent for nearly two days after a significant binge. His methanol level was 160 mmol/L, but there were no symptoms nor signs of toxicity. He was treated with oral ethanol alone and it took 15 days and a total of 298 doses of ethanol before the methanol was eliminated.

Patient 19 was found in a stuporous state at his home next to his dead partner and four empty, one-litre bottles of methylated spirits. After 11 days in the ICU he stayed a further 87 days in hospital and was left with a severe neurological deficit.

Patient 20 was a sporadic case who presented with fatal toxicity.

Discussion

Epidemic, as well as isolated cases of methanol poisoning have been reported for more than 100 years. Mortality rates of 10 - 20% are recorded with no evidence of a better outcome in more recent series. There is evidence of a striking individual variability in the response to methanol. Rates of 10 - 20% are recorded with no evidence of a better outcome in more recent series. There is evidence of a striking individual variability in the response to methanol. Accumulation of lactate is seen as a marker of secondary cellular dysfunction (Table 2: Patients 5 and, 11,12 ADH has a 20-times higher affinity for ethanol, therefore if methylated spirits are ingested, the ethanol component will be metabolised before the methanol component. If, after the ingestion of methanol, further ethanol is administered the elimination of methanol will be delayed until the ethanol level has decreased below 20-30 mmol/L and only then will formate begin to accumulate. Formate interferes with oxidative metabolism. Accumulation of lactate is seen as a marker of secondary cellular dysfunction (Table 2: Patients 5 and, 6). In some of our patients an almost complete inhibition of the elimination of methanol could be observed at blood ethanol concentrations of greater than 36 mmol/L. Below a blood level of 22 mmol/L, i.e.
the generally recommended therapeutic level, there is, however, a significant variability of the methanol elimination between different subjects; e.g. between 0.9 mmol/L/h in patients 7 and 19, and up to 2.4 mmol/L/h in patient 11.

In humans, there is some elimination of methanol via the lungs and kidneys, estimated to be about 2.5% and 1%, respectively. Rodents are able to catabolise formate using a catalase reaction and a folate-dependent pathway. As yet, it remains unknown whether in humans the enormous variability depends on these alternative pathways.

In this study, at least 19 cases would have been candidates for haemodialysis on the basis of their methanol levels on presentation. Only seven cases were given that therapy. Five patients with haemodynamic instability received continuous arteriovenous haemodiafiltration but all but one died. Two others had a single course of haemodialysis. The elimination of methanol using continuous arteriovenous haemodiafiltration was increased several fold: elimination rates between 1.3 mmol/L/h and 9.5 mmol/L/h could be achieved (Table 2). This compares with previously reported figures.

In summary, we describe a one-year experience of patients admitted after ingestion of methylated spirits. Most were chronic alcoholics with a previous history of methanol ingestion. There was significant morbidity and a greater than 20% mortality. There were no specific patient characteristics which could predict the individual response to the ingestion. Forced abstinence may have led to a fatal outcome in some but not in others.

Chronic alcoholics and/or habitual methanol drinkers who present to hospital after ingestion of methanol may not need hospitalisation or ethanol treatment. The acid-base status of these patients, however, needs to be monitored carefully. The absence of an acidemia while methanol levels are declining is good evidence against an accumulation of toxic formate. After exclusion of toxicity in the initial assessment of the patient we propose a period of clinical monitoring, without specific treatment, with repeat determination of the blood pH and base excess after 6 and 12 hours. Our internal guidelines for the management of these patients have been modified accordingly.

Methanol poisoning causes considerable morbidity and can lead to a high utilisation of hospital resources. In people with alcohol dependency, readmissions with the same presentation are common. As long as methylated spirits are available to be used as a cheaper alcohol substitute, further presentations of this kind are inevitable. A complete removal of methanol from methylated spirits would be the most significant measure to reduce the morbidity and the mortality of this problem. In New Zealand other denaturing agents such as Bitterex are available and there is no need for the use of methanol in methylated spirits.

Acknowledgements. Special thanks to all the physicians and the medical and nursing staff who cared for the patients in this series, to Canon David Morrell and the Christchurch City Mission, to Mrs Paddy Brocherie and the Social Work Department at Christchurch Hospital.

Table 2. Methanol elimination and additional findings.
Variability within general practitioner prescribing over time

Nadine Bishop, Junior Research Fellow; Tim Maling, Associate Professor and Director, Wellington Drug Utilisation Research Unit, Wellington School of Medicine, Wellington.

Abstract

Aims. We have described intra-general practitioner (GP) prescribing variability over time in terms of volume, cost and average item cost of prescription items, within New Zealand general practice.

Methods. Longitudinal data over the financial years 1992-94 were studied for two GP samples. Prescription data for a regional sample of 305 GPs were obtained for the first six months (January to June) from the New Zealand pharmaceutical pricing office, Health Benefits Limited. Prescription data from a second national sample of 74 GPs were obtained from the PreMeC prescription analysis (PAS) database of GPs who had participated in three consecutive September to December prescription analyses. The coefficient of variation was used to measure the intra-GP variability over time in total prescription cost, volume of prescription items and average prescription item cost.

Results. The median intra-GP variability over time for the regional GP sample, based on reimbursement data, was 9% in total cost, 9% in total volume and 5% in average item cost. The median intra-GP variability in the national sample was very similar to the regional sample when based on reimbursement data, but when PAS data were used the variability was 16% in total cost, 17% in total volume and 8% in average item cost.

Conclusions. The year-on-year, intra-GP variability for cost was 9%, for volume 9-10% and for average item cost 5-6%. Pharmaceutical budget estimates should reflect year-to-year intra-GP prescribing variability of the order of 9%.

Appropriate or rational prescribing can be defined as the prescription of medicines which are effective and economic.1 Prescribing efficiency, which can be defined as the ability of the individual prescriber to maintain rational prescribing across therapeutic conditions, is becoming increasingly difficult to maintain in the face of a plethora of therapeutic alternatives.2

Prescribing variability is a fundamental phenomenon reflecting the interplay of multiple factors “within-doctor” and “between-doctor”, which contribute significantly to overall expenditure and health status.3 Definition of the extent of prescribing variability can indicate strategies for improving efficient prescribing, since irrational prescribing will manifest concurrently with variations in prescribing behaviour.4 The relationship of prescribing variability to prescribing efficiency is unclear.5

There have been two previous attempts to describe variability in prescribing of individual general practitioners (GPs) in New Zealand.6,7 In both studies prescribing variability was not analysed at a national level and the lack of longitudinal data limited description of variability. These studies were also confined to the same region and were cross-sectional. We describe intra-GP prescribing variability over time, in terms of the volume, cost and average item cost of prescription items, within New Zealand general practice.

Methods

The analysis of longitudinal data was limited to the financial years 1992-4 (July 1991-June 1995) due to national policy changes occurring within the 1991 and 1995 financial years. These policies affected the volume and expenditure of prescription items in New Zealand. Two GP samples have been studied to take account of the different data currently used by different organisations for analysis.

(i) A regional sample of 497 GPs within a division of the Health Funding Authority (HFA) were identified from PreMeC records. Prescription data for the total number of items prescribed (volume), the total cost of these items and overall average item cost for each GP obtained for the first six months (January to June) of 1992, 1993 and 1994 from the New Zealand pharmaceutical pricing office, Health Benefits Limited (HBL). HBL reimburses pharmacies the cost of the prescription not covered by the patient co-payment. The data based on reimbursement cost which includes a wholesale mark-up (10%), a variable pharmacy mark-up, GST (12.5%) and dispensing and container fees ($2.93) and is exclusive of the patient co-payment (e.g. an A3 pays $15 whereas an Al pays $3). As a consequence, HBL data are exclusive of prescription items that fall below the patient co-payment and the reimbursement is dependent upon variables external to the prescription.

(ii) A national sample of GPs, who participated in three consecutive September to December PreMeC prescription analyses (PAS) between 1992 and 1994 was identified from the PreMeC PAS database.8 PAS data exclude the confounders of patient co-payments and pharmacy mark-ups by reporting the ex-manufacture cost of prescription items.9 Up until 1995 the PAS data attempted to capture prescription items that fall below the patient co-payment via a system described in detail previously.10 For each of the GPs included in this sample, reimbursement data were so captured for comparison with the regional sample.

GPs within either sample whose prescribing volumes were less than the lower 10th percentile of the sample in any year were excluded from the analysis of both the regional and national samples.

Information on the total cost and volume of pharmaceuticals in New Zealand for the study period was obtained from the Ministry of Health.10 GPs common to both the national and regional samples were excluded from the analysis. The measure of variability used in both samples was the intra-GP coefficient of variation (CV) over time for total expenditure, volume of items prescribed and average item cost. A chi-squared test was used to examine the validity of the geographical dispersion of the national GP sample.

Results

In the regional sample, 192 GPs were excluded because they either failed to have prescription data extending over the study period or had prescription volumes in the lower 10th percentile of the sample. Three hundred and five GPs were included in the analysis. In the national sample, 74 GPs were identified. No GPs were removed from this sample. There
was no difference in the proportion of GPs represented per HFA division (chi-squared, p=0.9).

The median annual growth over the study period in reimbursed cost, volume and average item cost within each sample, and for New Zealand, is summarised in Table 1. During this period the annual growth in cost and volume of pharmaceuticals decreased by at least half. Although the average item cost in New Zealand over this period was stable, an 8% increase was observed between 1992 and 1993 in the national sample and 3% in the regional sample. In both samples, growth between 1993 and 1994 decreased to 3% and 0%, respectively.

The magnitude of intra-GP prescribing variation over the study period based on reimbursement data is very similar between both groups (Tables 2 and 3). The year-on-year, intra-GP variability for cost was 9%, volume 9-10% and average item cost 5-6%. Both samples demonstrated a skew towards higher variability in total cost and volume by a few GPs.

When PAS data were used to calculate the intra-GP variability in the national sample (Table 3), the variability in cost per GP increased from 9% based on reimbursement data to 16% and for volume, from 10% to 17%. There was only a small difference in the average item cost based upon calculations using reimbursement data (6%) and PAS data (8%).

Discussion

We have estimated crude intra-GP variability in the total cost, volume and average item cost of pharmaceuticals between the financial years 1992 and 1994. Despite the limitations of the analysis, our findings indicate that budget estimates should reflect year-to-year, intra-GP prescribing variability of the order of 9% and also indicate the need to review the current practice of using single-point, historical prescribing variability data as a basis for setting pharmaceutical budgets in general practice. The use of the coefficient of variation as a measure of variability assumes that all GPs practising in New Zealand will have been exposed to the same policy and environmental pressures at any given time.

Data collection in New Zealand is not sufficiently advanced at present to define prescribing variability in relation to fundamental variables such as consultation rate, age and gender. The reimbursement system operated by HBL is currently the only national source of prescription data. We have used the PreMeC PAS data to observe prescribing variability because confounders such as the patient co-payment and the variable nature of pharmacy mark-ups have been removed from prescription costs and prescription items that fall under the patient co-payment have been captured to a significant extent.

It is possible that those GPs who elected to participate in PreMeC prescription analyses may not be representative of New Zealand GPs. It is therefore notable that the estimates of variability based on reimbursement data in both the national and regional samples are almost identical in magnitude despite their independence. These estimates are also based on data obtained during a period when the growth in subsidised pharmaceutical expenditure and utilisation had halved.

### Table 2. Coefficient of variation within GP reimbursement prescribing (Jan-June) 1993-95; - regional sample.

<table>
<thead>
<tr>
<th>Total cost per GP</th>
<th>Total volume per GP</th>
<th>Average item cost per GP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Median</td>
<td>9%</td>
<td>9%</td>
</tr>
<tr>
<td>Minimum</td>
<td>1%</td>
<td>0%</td>
</tr>
<tr>
<td>Maximum</td>
<td>80%</td>
<td>89%</td>
</tr>
</tbody>
</table>

IQR: interquartile range.

In the national sample, intra-GP variability based on PAS data was much higher for the total cost and volume of prescription items than that observed when reimbursement data are used. Variability in the data capture of the under co-payment prescriptions using the PAS system could have contributed. However, the similarity in the variability in average item cost in the national sample (Table 3), between reimbursement and PAS data sources, would suggest that any variation is small because variations in the volumes of low-cost items captured will impact on the average item cost. Although not directly related to budget holding, many of the prescription items that are not captured by HBL because they fall under the patient co-payment, such as hypnotics and antibiotics, have important implications for rational prescribing. Our findings indicate a considerable increase in estimated variability when attempts are made to account for these prescriptions and also highlight the limitations of the HBL reimbursement data in any analysis of prescribing variability.

This study does not define whether the variability in the cost, volume and average item cost is desirable or whether it is at an appropriate level. This would require investigation of the numerous components and processes comprising the variability. Current opinion for reducing prescribing variability embodies two strategies. Berwick’s theory of ‘bad apples’ represents one extreme of a range of strategies for managing outliers. It relies on finding and excluding physicians by considering inappropriate variability, perhaps defined as ‘high cost’, and removing the cause. Alternatively, the theory of ‘continuous improvement’ seems more appropriate as it encourages all physicians to analyse and improve their performance by removing the cause of inappropriate variability themselves. Prescribing variability is not consistent within and between conditions, undermining reliable identification of substandard or consistently inefficient prescribers, even if consensus were to be achieved as to appropriate criteria.

Our findings indicate a degree of prescribing variability in the basic measures currently used in budgeting and pharmaceutical management in New Zealand. The results are similar in two independent samples.
understanding of “pre-existing conditions” and their contribution to lumbar spine development had occurred either secondary to accident or in the course of the patient’s occupation (AI Act 1998, Section 29, clauses 2 and 4). Degenerative diseases would only be covered if their cover on the basis of “pre-existing degenerative conditions”, who have sustained significant injuries have been denied cover only for injury secondary to accident and not chronic injury to the lumbar spine. The intention was to provide cover for people sustaining personal injury by accident, and delivered in different ways”.

The ACC legislation enacted in 1974 provided universal coverage for people sustaining personal injury by accident, both within and outside the workplace. This cover included injury to the lumbar spine. The intention was to provide cover only for injury secondary to accident and not chronic disease processes. Problems arose when patients with chronic disease, chronically symptomatic pre-existing disorders or symptomatic degenerative disorders of the musculoskeletal system, developed symptom exacerbation following minor injury. It is clear that the original intention, quite reasonably, was not to provide comprehensive coverage for these degenerative disorders. The ACC legislation enacted in 1974 provided universal coverage for people sustaining personal injury by accident, both within and outside the workplace. This cover included injury to the lumbar spine. The intention was to provide cover only for injury secondary to accident and not chronic disease processes.

Accident Insurance Act 1998

“An Act to maintain a no fault, comprehensive, insurance-based scheme to rehabilitate and compensate in an equitable and financially affordable manner those persons who suffer personal injury and to provide opportunities for the scheme to be managed and delivered in different ways”. The ACC legislation enacted in 1974 provided universal coverage for people sustaining personal injury by accident, both within and outside the workplace. This cover included injury to the lumbar spine. The intention was to provide cover only for injury secondary to accident and not chronic disease processes. Problems arose when patients with chronic disease, chronically symptomatic pre-existing disorders or symptomatic degenerative disorders of the musculoskeletal system, developed symptom exacerbation following minor injury. It is clear that the original intention, quite reasonably, was not to provide comprehensive coverage for these degenerative disorders. The ACC legislation enacted in 1974 provided universal coverage for people sustaining personal injury by accident, both within and outside the workplace. This cover included injury to the lumbar spine. The intention was to provide cover only for injury secondary to accident and not chronic disease processes.

In recent years ACC has focused much attention on the contribution of “pre-existing conditions” to lumbar spine disability. There has been close observation of the role of any accident and possible pre-existing disorders or conditions in the genesis of symptom development, symptom exacerbation or symptom magnification. Often the understanding of “pre-existing conditions” and their significance is poor. Radiological findings of questionable importance may be considered as “pre-existing disorders”. Increasingly sophisticated imaging techniques have heightened the sensitivity to possible “pre-existing disorders” and hence potentially influence cover. Patients who have sustained significant injuries have been denied cover on the basis of “pre-existing degenerative conditions”, even when a meticulous history indicates that the “pre-existing condition” had been completely asymptomatic. To those who practise in this area, it is very clear that the application of cover by ACC is inconsistent. There is confusion regarding these issues from ACC administrators, medical referees and in the judgements at review and district court level. It is pertinent to review the literature on potential “pre-existing disorders” in the lumbar spine. Understanding of the natural history of normal ageing is important, as is the interpretation of other imaging findings.

Ageing and degeneration of the lumbar spine

“Degenerative changes in discs are ubiquitous among humans. The slow, unrelenting changes that are associated with disc degeneration begin in persons during their 20s. In fact, for the lower lumbar discs, the alterations concomitant with degeneration are usually present for a greater part of one’s life than is the so-called normal state”. Garfin and Herkowitz agreed that ageing and degeneration are closely linked and concluded it inappropriate to consider the degenerative disc as a disease state. With age the disc nucleus becomes dehydrated and the proteoglycan components change. Reducible collagen cross-links increase and the collagen type changes. This corresponds to macroscopic changes to the disc and intervertebral joints. The degenerate nucleus develops clefts. Annular fissures occur. The intervertebral disc loses height and the annulus

<table>
<thead>
<tr>
<th>Total cost per GP</th>
<th>Total volume per GP</th>
<th>Average item cost per GP</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Reimbursement</td>
</tr>
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<td>21%</td>
</tr>
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<td>16%</td>
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<td>2%</td>
</tr>
<tr>
<td>Maximum</td>
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</tr>
<tr>
<td>IQR</td>
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<td>21%</td>
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<tr>
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<td>16%</td>
</tr>
<tr>
<td>Minimum</td>
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</tr>
<tr>
<td>IQR</td>
<td>10%</td>
<td>21%</td>
</tr>
</tbody>
</table>

IQR: interquartile range.

Acknowledgement. We are grateful for the assistance of the staff at the National Preferred Medicines Centre, Wellington.

bulges circumferentially. Reactive bone changes in adjacent vertebrae are manifest by end-plate sclerosis and the development of spondylophytes, osteophytes and/or traction spurs. Increased load born by the facet joints, secondary to disc height loss, may result in a picture of osteoarthritis within the synovial joints. This process of degenerative change or ageing is also referred to as lumbar spondylosis.

The plain x-ray of the aged or spondylotic spine will demonstrate significant disc narrowing, end plate sclerosis, spur or osteophyte development, facet joint arthrosis and any spinal malalignment. CT scans will demonstrate circumferential disc bulging, any disc herniation and give a better picture of facet joint changes and/or bone changes adjacent to the disc. Magnetic resonance imaging (MRI) is the most sensitive investigation currently available. It examines hydration of the nucleus. Annular tears may be imaged and detailed narrow changes in the adjacent vertebral discs are seen. As the incidence of these degenerative changes increase with the passage of time, it is important to understand the incidence of these abnormal findings and the relationship between imaging abnormalities, advancing age and symptoms.

Degenerative changes within the intervertebral disc become universal with advancing years. Symptomatic patients will frequently have correlation with x-ray changes in the spine. The reverse implication - that radiological changes predict symptoms and disability - is unfounded. With increasing years radiological changes of ageing universally increase yet symptoms become less prevalent after middle age. X-ray abnormalities may be equally prevalent in both low back pain sufferers and asymptomatic individuals, have variable correlation with prevalence and magnitude of back pain and are of little use for screening workers who may develop low back pain. The ability of MRI to demonstrate normal ageing changes risks gross over-apportionment of significance to these abnormalities. MRI will also detect significant, yet irrelevant, findings when performed in asymptomatic patients. In 35% of asymptomatic subjects, aged 20-39, Boden et al found evidence of disc bulging and degeneration.

Because of the known incidence of irrelevant findings on CT and MR scanning, treating physicians must be encouraged to correlate pathological lesions with clinical findings before instituting management. Indeed because of the excessively sensitive and unselective nature of MRI, it is inappropriate and dangerous to use MRI to “see if there is anything wrong with the spine”. In the absence of symptoms it is inappropriate to consider ageing changes to be either a disease state or a significant “pre-existing disorder”.

Spondylosis and isthmic spondylolisthesis

Spondylosis is an acquired stress or fatigue fracture of the pars interarticularis, most frequently found in the fifth lumbar vertebra. Its presence may lead to a slippage of one vertebra in relation to another (usually at L5/S1) - an isthmic spondylolisthesis. It is accepted that spondylosis is exceptionally rare at birth but has an incidence of 4 per cent at age six and 6 per cent in adulthood. This later increase in incidence is related to athletic activity. Higher incidences of spondylosis occur in specific sporting groups, employment and racial groups. Isthmic spondylolisthesis (developing from spondylosis) occurs with variable and lesser frequency.

The listhesis (slippage) occurs between ages 10 and 14, and is rarely progressive in adulthood. It is not known whether or not spondyloysis or low-grade isthmic spondylolisthesis predisposes to an increased risk of low back pain or injury. Medical literature exists to support both views. Some have suggested that these abnormalities predispose to an increased risk of low back pain, yet others have argued there is no increased risk of back pain in these individuals, or that any increased risk is limited to specific sections of the population. It is probable that these different findings relate to differing definitions of the process and methodological variations of study techniques. Because of the ethical difficulties associated with radiography in asymptomatic individuals, it is unlikely that better information will ever be available to clarify the risk of back pain in these individuals with spondylosis or low-grade isthmic spondylolisthesis. It is likely that when spondylolisthesis becomes of an increasing grade (>25%) the risk of back pain increases. What is abundantly clear is that there are a large number of patients with spondylolysis or low-grade isthmic spondylolisthesis who are not troubled by low back complaints and live active lives of normal quality. It has been demonstrated that the finding of a spondylolysis at L5 in an unselected middle-aged patient is not associated with an increase in back symptoms or decrease in function. Many authors advise that no limitations be applied to people with spondylosis or low-grade (less than ten per cent displacement) isthmic spondylolisthesis. In the absence of higher grades of spondylolisthesis this spectrum of disorders is not a clear predisposing factor for an increased prevalence of low back pain.

ACC

The 1992 Act covered personal injury occurring in New Zealand or when a person normally resident in New Zealand suffered injury outside the country. The Act also covered “gradual process, disease or infection arising out of and in the course of employment”. Gradual process, disease or infection were not covered unless this occurred in the course of employment (ARCI Act 1992, Section 8, 9). These issues were further clarified by the statement “(2) for the avoidance of doubt it is hereby declared that (a) personal injury caused wholly or substantially by the ageing process... is not covered by this Act” (ARCI Act 1992, Section 10). This has been repeated in the 1998 Act (Al Act 1998, Section 29). The relevant sections of the legislation makes no reference to the terms “pre-existing disorder”, “pre-existing condition”, or “degenerative” disorders.

The trend to limit cover of people suffering personal injury has often referred to these definitions. Patients whose symptom onset related to injury but who had spondylolysis / degenerative / ageing changes that predated the accident, have had cover declined because of gradual process or disease. Investigation findings referred to as “disc disease”, “degenerative disc disease” or “degenerative spondylitis” have been considered to represent the ageing process or a gradual process. Similar reasoning has been used to exclude patients with spondylosis on the basis that it represented a “pre-existing disorder”. This exclusion has occurred despite the absence of symptoms prior to an accident and a clear history of the accident precipitating the development of pain and disability. Such a trend away from coverage of these low back disorders is clearly a departure from the original direction of ACC legislation, aiming to provide coverage for accident victims ((ARCI Act 1992 (Title), Al Act 1998 (Title)). As noted, these patients’ only “predisposing disorder” was to have normal ageing changes or a previously undiagnosed radiological abnormality (spondylosis / spondylolisthesis), the latter of which places the individual at no significantly different risk for the development of
symptoms over the normal population! Section 10/2/(a) (1992) offers further clarification for those having suffered personal injury by accident. This section states “.... For the avoidance of doubt.... (a) personal injury caused wholly or substantially by the ageing process is not covered.” This sentiment is repeated in the 1998 Act - .... (4) “Personal Injury” does not include (a) Personal injury caused wholly or substantially by the ageing process;....

The definition of “substantially” includes “to all intents and purposes; in the main” (Oxford English Dictionary). It is the adverb of “substantial” - “being largely but not wholly that which is specified” (Webster's Dictionary). Therefore “wholly or substantially by the ageing process” means that the total or main component of cause of personal injury (symptoms/disability) is the ageing process. In a hypothetical patient who is symptom-free prior to an accident, yet is found to have imaging evidence of pre-existing degenerative/spondylotic/ageing change in the spine, the main or substantial cause of the personal injury (symptoms/disability), or indeed the whole cause, is the accident. Had the accident not occurred the symptoms would probably (most likely / in all probability) not have developed. The substantial cause of symptom onset or personal injury is the accident - not the ageing process.

The radiographic finding of spondylolisthesis or low-grade isthmic spondylolysis in a previously asymptomatic individual presents further difficulties. Spondylolisthesis is not a gradual process - it is a stress or fatigue fracture. Isthmic spondylolisthesis revealed in adulthood is not a gradual process - but one that has been present from adolescence or early adult life. Development or progression of spondylolisthesis in adulthood is exceptionally rare. Spondylolysis or isthmic spondylolisthesis is not considered a disease and is clearly not an infection. Under these circumstances it is unreasonable to exclude previously asymptomatic individuals who develop symptoms after injury even if there is an incidental finding of spondylolysis or isthmic spondylolisthesis.

In further reference to spondylolisthesis and isthmic spondylolisthesis, officers of the ACC have further questioned the time-frame of “soft tissue injury healing” as a guide to the length of cover that patients with these disorders may receive (Dawson AG, Corporate Medical Advisor, ACC, Head Office, personal communication, 1 October 1998). It has been observed that the development of symptoms in spondylolisthesis may occur after relatively minor trauma.51-55 It seems that if the “soft tissue damage” from the accident, regardless of the magnitude of trauma, can be assumed to last for a limited time, then ongoing symptoms may be related to any pre-existing or underlying condition that can be identified and hence relieve the ACC of responsibility of cover. Unfortunately this seems inappropriate and the authors can find no evidence that such interpretations are intended by the respective sections of the current and past legislation. Neither is there any attempt to quantify the magnitude of “application of force” within the definition of an “accident” (Al Act 1998). It is well accepted that a wide variation in symptom magnitude or duration occurs after low back injury56 and chronic pain can occur from a number of complex mechanisms.17 In any musculoskeletal injury, symptoms and disability frequently persist well beyond time-frames of histological tissue repair. Again it seems unlikely that either the ARCI (1992) or AI (1998) Acts are designed to limit cover for those suffering personal injury to any time-frame of histological tissue repair, or for those suffering personal injury by accident - but only if the severity of the accident is considered adequate by an employee of the corporation!

Summary

The purpose of the ARCI (1992) and Al (1998) Acts is to cover those who suffered from personal injury by accident. This purpose should not be distorted. This review does not aim to suggest that cover be extended to victims of disease. Equally it is inappropriate that imaging be used to detect asymptomatic, age-related change - or reveal asymptomatic abnormalities that do not place the patient at increased risk of symptoms over the general population - so as to unfairly deny coverage to the victims of accidents. This is particularly important to those who suffer personal injury to the lumbar spine where the cause is wholly or substantially an accident, and in whom, without the specified accident having occurred, personal injury (symptoms and disability) would have not been likely to occur. Physicians managing spinal disorders must correlate clinical findings with imaging studies when planning treatment. Those considering entitlement for cover under third party / ACC provisions must pay close attention to the history and clinical evaluation, correlating these with the investigation findings, and not assume that the abnormalities found on sensitive investigations are the cause of the symptoms.

Correspondence
Mr PA Robertson, The Orthopaedic Clinic, Mercy Specialist Centre, 100 Mountain Road, Epsom, Auckland 1003.

Dr Bob Tennent and Leptospirosis

The obituary for Bob Tennent (NZ Med J 1999; 112: 477) was excellent. As a novel general practitioner in Paeroa in 1960, it was not long before I discovered the low lying flood problems of the Hauraki Plains which created a serious risk for Leptospirosis. Flood water was readily contaminated with bloody urine of infected cows, pigs, rats and dogs.

I soon discovered the sheer energy of Bob of Ngatae in relation to spreading the word about the little recognised fever amongst dairy farmers on the Hauraki Plains. If Bob wasn’t on the phone, he was on your surgery doorstep or quoted in rural newspapers talking about the spirochaete and its various serotypes causing differing symptoms. Thanks to Bob, we soon had known that this notifiable infection responded to a one off high dose injection of penicillin given by deep intramuscular injection. Many a dairy farmer was soon back in the cowshed even if nursing a tender injection site!!

As an elderly semi retired general practitioner I can only pray that general practitioner’s of Bob’s calibre are incubating amongst our ranks today.

Graham Hall
Milford,
Auckland.

Impact of changes in the death registration process upon Maori mortality statistics

Spore and Pearce’s recent letter raised important issues regarding Maori mortality statistics: namely the change in ethnicity data collection on registration. In September 1995, and the mismatch between the classification of ethnicity in mortality and census data.1 We wish to raise further issues.

With regard census data, the population described as “50% or more Maori” prior to 1986 was approximately equivalent to the sole Maori ethnic group in the 1986 and 1991 censuses, and thus could be used for calculating mortality time trends. With the new ethnicity question in the 1996 census, the sole Maori ethnic group nor the sole Maori population were congruent with previous census data (Figure 1).

Prior to September 1995, Maori mortality was long known to be undercounted, mainly due to missing data on death registrations.2 A study of Wellington funeral directors found that 30% never filled in the Maori descent question on the death registration form.3 Now all funeral directors report they fill in the ethnicity question,4 giving more complete Maori mortality data, particularly in the 15-24 year age group (Figure 2).

Figure 1. Maori populations in successive censuses.

Prior to September 1995, Maori mortality was long known to be undercounted, mainly due to missing data on death registrations.2 A study of Wellington funeral directors found that 30% never filled in the Maori descent question on the death registration form.3 Now all funeral directors report they fill in the ethnicity question,4 giving more complete Maori mortality data, particularly in the 15-24 year age group (Figure 2).

Despite the ethnicity question on the new death registration form being consistent with the 1995 census, the high proportion of Maori deaths recorded as sole Maori (%1)5 implies that the ethnicity question on the death registration form is, in the main, still being applied as a single option question. Maori ethnic group deaths may still be under-reported. Therefore, neither the sole Maori denominator nor the Maori ethnic group denominator from the 1996 census match the mortality numerator data.

There is evidence that the 1996 ethnicity question is more likely to elicit responses based on ancestry than on ethnicity, compared with the 1991 census.6 It has not been implemented by hospitals, despite policies directing hospitals to collect data consistent with the census.2 Currently, therefore, there are moves to return to the 1991 question for the 2001 and 2006 censuses.7 We promote the use of population projections based on the 1991 census until the 2001 census is available. It is also necessary to work with those who collect mortality data to ensure that wherever possible, families fill in the actual ethnicity question themselves as best proxy for the deceased.

The Maori ethnic group and the sole Maori group have different sociodemographic profiles and therefore are likely to have different health experiences. It is important to have the capability to monitor the health outcomes of both groups.

Ricci Harris, Vera Keefe, Papaarangi Reid, Bridget Robson.
Te Ropu Ragahau Hauora e Ur Pomo Wellington School of Medicine
Wellington.


Case report: ‘Necrotic Araneism’

A 10 year old girl came into my surgery recently with a sore right leg, because “her hot water bottle had burnt her”. She was in pain with a temperature of 37.5°C and pulse of 100/minute. She had a 2-3 cm red ring on the right leg, with small central pustular areas. A diagnosis of cellulitis secondary
to spider bite was entertained. Swabs were taken, the wounds cleaned and dressed and fluoxacillin commenced. Over the ensuing week two 3-4 cm rings of necrosis developed, to full skin thickness, despite antibiotics and dressings. *Staphylococcus aureus* sensitive to fluoxacillin was cultured from both wounds. Skin grafting was performed at Middlemore Hospital and these have healed well.

An infected insect bite is a common problem in general practice, and it usually follows a benign course, though occasionally antibiotics are prescribed. Several spider stings are easily identified, and typically produce a local venom effect, with pain and swelling around a wheal, usually subsiding in 24 hours. A delayed cutaneous reaction with induration and swelling may ensue. This is usually a local cellulitis, but it is also usually an immune complex reaction. Infection, and even cutaneous necrosis may occur, more commonly after wasp stings.1 Not uncommonly, the identity of the culprit will be a mystery.

Less benign are spider bites1 especially those of the *Lampona* species the indigenous *Katipo* (L. *katipo*), and its imported Australian cousin, the *Red-back spider* (L. *basilea*). Another transvaalism, the white-tailed spider (Lampona *cylendrata*), has gained a reputation for severe bites, sometimes resulting in ulceration and necrosis of skin and underlying tissue (necrotic araneism or arachnidism).2 The evidence associating the white-tailed spider to date has been circumstantial, as in this case. *Lampona* species are not known to produce necrotic wounds, and the culprits in typical cases of necrotic araneism in the Americas proved to be recluse or fiddleback spiders, both *Loxosceles* species, though notable other species were suspected.3,4 *Loxosceles* is not native to Australasia. Nevertheless, in 1976, *Loxodes* was first reported in Adelaide and Sydney, and it may have spread to New Zealand. Recently, white-tailed spiders and black house cellars, (Badumna *species*) have been identified as responsible for skin necrosis, though positive identification was made in only three cases.5

An infectious agent following necrotic spider bites has been suspected, and in this case *Staphylococcus* was identified. In addition there may be necrosis-inducing factors in venoms, particularly *Loxodes*, where a pyoderma gangrenosum-like process has reportedly been documented. Treatment includes attention to wound care, antibiotics for suspected bacterial infection and tetanus prophylaxis.6 Where hynomenota are the culprits, antihistamines and steroids may be helpful. Conservative treatment for *Loxodes* is encouraged- surgical debridement seems to worsen the prognosis and delay healing.5,6 Hyperbaric oxygen treatment is gaining in popularity, and appears to have clinical benefit for ulcers from white-tailed spider bites.8 As yet, there is no evidence that this treatment has value in *Loxodes*-induced necrosis. Specific antivenoms exist for *Ladrodectus* species, and are efficacious against the neurototoxic venom. There are antivenoms against *Loxodes* available in Brazil, Peru and Argentina. The efficacy against necrosis is limited. We await with interest the first New Zealand siting of the recluse spider.

Medicolegal Conduct unbefitting and professional misconduct

Dr Julian Meredith Clive White, general practitioner of Cambridge admitted before the Medical Practitioners Disciplinary Tribunal (the Tribunal) each of a charge amounting to either conduct unbefitting a medical practitioner so as to reflect adversely on the practitioner’s fitness to practise medicine, or professional misconduct. The charge was brought by a Complaints Assessment Committee of the Medical Council of New Zealand.

**Charge A.** Conduct unbefitting a medical practitioner and that conduct reflects adversely on that practitioner’s fitness to practise medicine.

Particulars of charge A. Failed to exercise and show due care when prescribing or ordering drug treatments in that he:

1. Treated xx at various places in Cambridge during the months of November and December 1997 by changing his heart medication without proper clinical examinations.

2. During the years of 1993 to 1997 treated xx on a regular basis during which time he caused to be administered the prescription drug Kenacort in excess of acceptable clinical practice without appropriate warnings as to the possibility of adverse side effects.

3. On one occasion between the years of 1993 and 1997 at Cambridge prescribed to xx sixty tablets of the prescription drug Halcion without warning her as to its addictive potential.

4. Between the years 1996 to 1998 at Duke Street Medical Centre, Cambridge, failed to maintain or adequately maintain a record of narcotic use in accordance with accepted clinical practice.

5. During 1997 at Duke Street Medical Centre, Cambridge, altered the existing treatment regime of patients of the Centre attended to by Dr xx without consultation or proper clinical examination.

**Charge B. Professional misconduct. Failed to observe the acceptable standards required of a medical practitioner in clinical procedures in that he:**

1. During the year of 1997 at Duke Street Medical Centre, Cambridge inadequately or inaccurately labelled pathological specimens.

2. During the years of 1993 to 1998 at Cambridge failed to evaluate and follow-up cervical smear results in respect of a patient xx.

3. During the years of 1996 and 1997 in Cambridge failed to adequately monitor and respond to abnormal INR results.

4. During 1987 at Cambridge re-used hypodermic needles on different patients.

5. During the year of 1997 at Cambridge in the course of his clinical practice used unsterile instruments and procedures.

**Charge C.** Conduct unbefitting a medical practitioner, and that conduct reflects adversely on that practitioners fitness to practice medicine.

Particulars of charge C. Failed to observe patient privacy and confidentiality in breach of the Code of Ethics:

1. During the year 1997 allowed an unqualified person xx to be present during consultations and surgical procedures.

2. During the years 1996 to 1998 in waiting/ reception area in Duke Street Medical Centre, Cambridge made statements about personal and medical matters concerning his patients in hearing of other patients and members of the public.

**Charge D.** Conduct unbefitting a medical practitioner, and that conduct reflects adversely on that practitioner’s fitness to practise medicine.

Particulars of Charge D. Failed to conduct himself in a professional and ethical manner in dealings with other medical practitioners, staff and patients.

1. During the years 1993 to 1997 at Cambridge used offensive language in consultation with patients and in public areas of the centre.

2. During the years of 1997 and 1998 impugned the reputations of other health professionals, namely Dr xx, Mr xx, Mxx and Mxxx.

3. During the months of August and September during 1997 at Cambridge applied undue pressure to his former patient Mr xx and wife to return as patients to his practice.

4. During the months of April and May 1988 at Cambridge in his capacity as a designated doctor for Income Support solicited patients seen pursuant to that scheme to return to him for ongoing care.

**Charge E. Professional misconduct**

Particulars of charge E. Failed to ensure that medical care was available to patients.

1. That on the 19th and 22nd days of December 1997 at Cambridge he intentionally interfered with the after hours emergency medical service of the Duke Street Medical Centre in such a manner it prevented patients from accessing emergency medical care.

During the hearing the Tribunal determined that the seriousness of Charge B (4) and (5) and Charge E warranted elevation of these particulars from professional misconduct to disgraceful conduct in a professional respect. After being so advised Dr

John P Bradley
Kaitaia


TRAMAL AD
White elected that the hearing continue. In response to the charges as admitted, it was explained by counsel that Dr White would have been eligible to seek his practising certificate to the Medical Council and submitted himself to the Medical Council’s Health Committee for assessment. A medical certificate was produced to the Tribunal indicating Dr White had been suffering from depression for the previous two years. At the conclusion of the hearing the Tribunal ordered that Dr White be suspended from practice pending assessments by a psychiatrist, physician and psychologist to assist the Tribunal in completing its determination. On receipt of the medical reports the Tribunal in a Supplementary Decision on 26th August 1999 ordered that Dr White’s name be removed from the medical register, that he be censured, that he contribute $23,000.00 towards the costs and expenses of the inquiry and hearing and that publication of the Tribunal’s orders be made in the New Zealand Medical Journal.

Failure to attend

The Medical Practitioners Disciplinary Tribunal (“the Tribunal”) – found Dr Jacobus Petrus de la Porte general practitioner formerly of Hokitika, guilty of conduct unbecoming a medical practitioner which reflects adversely on fitness to practise medicine. The Tribunal ordered that Dr White be suspended from practice pending assessments by a psychiatrist, physician and psychologist to assist the Tribunal in completing its determination. On receipt of the medical reports the Tribunal in a Supplementary Decision on 26th August 1999 ordered that Dr White’s name be removed from the medical register, that he be censured, that he contribute $23,000.00 towards the costs and expenses of the inquiry and hearing and that publication of the Tribunal’s orders be made in the New Zealand Medical Journal.

Obituary

Richard Thomas Aldridge

Dick was born in Auckland on 18 June 1930. He received his early secondary education at Greythorn Technical School and then moved to Palmerston North Boys High School where he was Doctor of the School. He entered Victoria University College for his medical intermediate and qualified MBChB from Otago Medical School in 1951. His House Surgeon years were in Wellington and he then spent six months at Stratford Hospital before returning to Wellington as Orthopaedic Registrar and then Junior Surgical Registrar in 1957. He was Surgical Registrar at Hastings Memorial Hospital in early 1958, then travelled to Britain with a young family and was admitted as an Edinburgh Fellow later that year. He was Surgical Registrar at Barnt General Hospital, London in 1959 and admitted as a Fellow of the Royal College of Surgeons of England. His overseas experience was completed with a year as Surgical Registrar at University College Hospital and he returned home with his wife, Margaret and two daughters, Victoria and Jane and one son, Richard.

He became a Fellow of the Royal Australasian College of Surgeons in 1961 while serving as Casualty Surgeon and Admitting Officer at Wellington Hospital. From 1962 Dick had two years as Surgical Tutor in the Wellington Clinical School and then was appointed fulltime Surgeon in general and paediatric surgery.

He was a gentle, quiet, and skilled surgeon, earning high regard from his Wellington colleagues. From 1970 to 1989 he was on the visiting staff of Wellington Hospital, combined with a busy private practice - he then took early retirement.

Dick was a keen territorial soldier, joining the Otago University Medical Company as a student, receiving his commission in 1954 and was then Medical Officer to the Divisional Signals Regiment. He joined the 2nd General Hospital and from 1968 to 1970 was its commanding officer. In 1971 he was promoted to Colonel as President of the Central Medical Board at Defence Headquarters. He was awarded the Efficiency Decoration for long and distinguished service.

He married Joan Curle, Theatre Supervisor at Wellington Hospital, in 1974 and their only daughter, Robyn, is a Trainee Intern in the Wellington School of Medicine.

Ischaemic heart disease did not deter Dick from an active retirement. Bowls was a special hobby and he was President of the Karori Bowling Club in recent years. Pottery was a new found pleasure in his retirement and friends commented on his artistic skills. He was a member of the Ruapehu Ski Club and for 14 years was a member of the ski Patrol. He was a member of the Karori Golf Club.

He took his share of administrative duties, being Registrar of the Court of Examiners of the RACS from 1968-70. He gave of his great depth of experience during the ten years that he served on the Wellington Divisional Disciplinary Committee.

He was Chief Examiner for the Wellington Free Ambulance, on various staff committees of the hospital, the Medical Advisory Committee of the New Zealand Red Cross Society and Nursing Education Committee.

He died suddenly on 27 July 1999 while happily involved in his potting class.

A large congregation of friends and medical colleagues, at St Ninians in Karori, paid their tributes to the life of a well-rounded man. The Church shared the tributes of life long friends, colleagues, at St Ninians in Karori, paid their tributes to the life of a well-rounded man. The Church shared the tributes of life long friends, colleagues, and relatives, to the life of a well-rounded man. Andrew Cameron Howitt Wilkinson, b China; d 12.8.1999; m Marilyn. One daughter and one son.

We are indebted to Dr MH Watson for this obituary.

James Kelly

Jim Kelly came to New Zealand from Northern Ireland with his wife Evelyn in 1953. He went into practice in Timaru, which at that time was a settlement north of Wellington, where he guided the development of the general practitioner service, working long hours and delivering many babies, including, with specialist help, a set of quadruplets in 1961.

Jim and Evelyn acquired a large property behind Tawa, where birds and animals freely wandered. Large dogs greeted one at the front door and it was not uncommon for a baby to escape to run up to the 100 selected trees and gardener. His passion for farming also led him to acquire a property behind Waverley. On social occasions, people frequently mistook him for a farmer. They moved to Wanganui in 1976, purchasing a 50-acre farmlet at Papaiti. Sheding the onerous responsibilities of a large group practice, Jim remained in practice in the city of Wanganui until 1987. He then became Medical Officer at Jubilee Hospital until he retired in 1992. He had a large range of interests, with a special concern for conservation and for keeping New Zealand “nuclear-free.” He was active in the “Save Manapouri” campaign and, as a member of the Medical faculty, a proponent for liberalisation of the abortion laws. In 1972, Jim joined with David Minnitt,
Bill Newman, Geoff Stokes and Roger Ridley-Smith in establishing the General Practitioner Society. He edited the Journal of the Society for a number of years.

In September 1974, Jim, on behalf of the GP Society, arranged a poll of the readers of metropolitan newspapers on the matter of the liberalisation of the abortion law. The second of two questions asked was, “Do you believe that termination of pregnancy should be a matter between a woman and her doctor.” Twelve thousand replies came in from all over New Zealand, of whom well over ninety per cent said “yes.” This ground-breaking poll got a lot of publicity and made a profound impact.

Jim Kelly had a most engaging personality, a dry sense of humour and a soft Irish accent that he never lost.

James Kelly, born Belfast, 1926, died Wanganui 1999; MBChB, BAO (Queen’s University, Belfast) 1952; m Evelyn, two sons, one daughter, who all survive him.

We are indebted to Dr Roger Ridley-Smith for this obituary.

Notices


In June 2000, The Royal New Zealand College of General Practitioners will be hosting the WONCA Asia Pacific Regional Conference. This combined conference is anticipated to attract up to 1800 delegates. Past conferences in this series have attracted primary care practitioners from around the world. The Royal New Zealand College of General Practitioners is proud to be hosting this conference which will provide a unique opportunity for delegates to meet and interact with colleagues from across the Asian Pacific Region. The scientific programme will concentrate on innovations in teaching, clinical practice and the delivery of care. Registration information as well as the Call for Abstracts may be obtained from the Conference website www.rnzcp.org.nz or from Conference Innovations Ltd, PO Box 1370, Christchurch. Ph: +64 3 379 0390, Fax: +64 3 379 0460. E-mail: WONCA@conference.co.nz

The Heart Foundation 2000 Grant Applications

Project Grants
Project grant applications will be considered at the July 2000 meeting of the Scientific Committee. The closing date for applications is 1 March 2000.

Fellowships and Scholarships
Applications for Senior Fellowship, Overseas Training Fellowships, Research Fellowships and Postgraduate Scholarships will be considered in July 2000. The closing date for applications is 1 June 2000.

Limited Budget Grants
Small Project Grants
Applications for small project grants will be considered three times a year with the closing dates being: 1 February, 1 June and 1 October 2000.

Grants-in-Aid
Applications for grants-in-aid will be considered three times a year with the closing dates being: 1 February, 1 June and 1 October 2000.

Travel Grants
Applications for travel grants will be considered three times a year with the closing dates being: 1 February, 1 June and 1 October 2000.

Priorities for Research
Applications must fit within the aims of The Heart Foundation which are “to promote good health and to reduce suffering and premature death from disease of the heart and circulation”.

Format for Applications
Applications should follow the format outlined in the revised “A Guide to Applicants for Research and Other Grants”.

Applications should be sent to: Dr Boyd Swinburn, Medical Director, The Heart Foundation, PO Box 17 160, Greenlane, AUCKLAND.

Email: boyds@akl.nhf.org.nz

Practice Note no 1

1. THE TRIBUNAL wishes to confirm its existing procedures, and to GIVE NOTICE of the procedures which it intends to continue to follow in regard to the rights of affected persons provided pursuant to Section 106 of the Medical Practitioners Act 1995. The following procedures will remain in place until further notice of the Tribunal.

2. WHEN a Charge is received by the Tribunal, no information regarding the Charge or Charges, including the names of the parties, the complainant and any other person or persons affected by the Charge, will be disclosed by the Tribunal to any person other than members of the Tribunal and the Tribunal’s administrative staff pending the receipt and determination of any applications made pursuant to section 106 of the Medical Practitioners Act 1995 (the Act).

3. ACCORDINGLY, pursuant to section 106 of the Act, no information as to the identity of any person named in the Charge, or otherwise affected by the Charge, or any identifying details or other information may be disclosed by the Tribunal members or its staff to any member of the public or the news media pending the receipt and determination of any such application, without the prior written approval of the Chair of the Tribunal.

4. ALL applications made under section 106 of the Act are to be submitted to the Tribunal strictly in accordance with any timetable set by the Tribunal and advised to the parties. Notice of the date by which such application must be received by the Tribunal will be given to the parties together with Notice of the Charge, and a Directions Checklist will also be issued for completion by the parties. The Directions Checklist will also be issued for completion by the parties. The Directions Checklist is to be returned to the Tribunal by the stipulated date and must contain notice to the Tribunal as to whether or not the parties intend to seek any orders under section 106.

5. ANY such timetable orders and any other pre-hearing Directions which are of a merely procedural nature may be made by any of the Chair, Senior Deputy Chair or Deputy Chair for and on behalf of the Tribunal.

6. THE Tribunal will hear and determine applications made under section 106 of the Act as soon as reasonably practicable after an application is received.

7. IN the event that NO application pursuant to section 106 is made by the date given to the parties by the Tribunal, and the Tribunal is not advised of any intention to seek such orders by the date advised for same, the names of the respondent and any other party, and/or the complainant/s may be disclosed, together with any other information relating to the Charge as the Tribunal shall consider appropriate, subject to the requirements of section 107 of the Act and Clause 5 of the First Schedule to the Act.

8. IN the event an application for any of the orders specified in section 106 (2) is declined the Tribunal hearing the application shall, on the application of any person made at the hearing of the application, make an interim order under section 106(2)(d) pending further order of a District Court Judge under section 120(2), or such other orders as may be made by a District Court Judge under section 120(2), or such other orders as may be made by a District Court in relation to any appeal.

9. THIS Practice Note is made pursuant to sections 106 and 117 of the Act and clause 5 of the First Schedule thereto.

Dated at Auckland 7th December 1999.

W N Brandon
Chair
Medical Practitioners Disciplinary Tribunal.