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Health services at breaking point. ...don’t panic?

The Waikato District Health Board (DHB) is ‘stretched to breaking point’ with 90 unfilled vacancies (including eighteen doctors), routine patients unlikely to be seen in the foreseeable future, intensive care and renal medicine staff levels too low and emergency patients not being treated within deadlines.

Radiation therapists in Auckland, Waikato and Mid Central DHBs have been considering strike action while cancer patients express alarm. Young doctors look to overseas posts to help repay their loans raising concern at possible worsening of the doctor shortage. Rural general practitioners report they are unable to cope and some are walking away from their practices. This is a sample of concerns reported in the daily newspapers over a two week period. Not included are concerns regarding patient safety during the strike of nurses and allied health professionals at Canterbury DHB, or the worries of senior medical staff at Green Lane Hospital when they are shoe-horned into the central Auckland campus, or the long-standing crisis in psychiatric services in Southland.

Here is the issue. What should we, as professionals, do when, for reasons beyond our control, we are no longer able to serve our patients at a standard we find acceptable? This scenario has confronted many doctors in New Zealand, especially during the last decade of restructuring/reforms, exclusion of health professional input from planning, inadequate and misplaced funding, loss of continuity of patient care, etc. Should we ‘just’ work 10% harder than last year to cover the gaps (at the expense of CME, audit, research, reflection and family life), then 10% harder again next year?

Returning to problems at the Waikato DHB, Chief Operating Officer John Mollett is reported to have warned senior medical staff at Green Lane Hospital when they are shoe-horned into the central Auckland campus, or the long-standing crisis in psychiatric services in Southland. Is it time to panic? Perhaps not but it is long overdue that we spoke out for the patients.

Do we, under circumstances such as those at Waikato where patients are bound to suffer, remain silent, keep our heads down and do our individual best? Or do we, through our elected professional representatives, make it clear, in writing, to management and DHBs that standards of patient care are unacceptable? Of course, some would see it as unprofessional to show emotion or to panic. “The control of the emotions and the prevention of panic is always presented by technocrats as a sign of their professionalism” says JR Saul.

Perhaps it is time, not to panic, but to express our concerns, in a collective fashion, about what has happened to standards of patient care in this country. Indeed, we argue that it is our duty, as one group of patient advocates (not the only one), to raise our voices, with emotion if necessary, so that managers, DHB boards, CCMAU, Treasury and especially our elected representatives hear and understand these concerns. If we hold our tongue when standards of patient care have fallen to unacceptable levels, we must be prepared to explain this silence to our patients when they suffer the consequences. Post hoc explanations that resources were inadequate, the workload was too great cannot be expected to convince grieving, angry relatives.

Why is it that the medical profession is currently viewed with such uncertainty, suspicion and distrust? No doubt there are several reasons. One major factor, we contend, is that the profession has failed to speak out clearly and collectively when it has been sidelined and when standards of patient care have been compromised by forces beyond our control. Is it our job to explain to patients why resources are diverted from direct health care into an ever-expanding bureaucracy, and why the student loan scheme means our hospitals will be understaffed? Our clear role is to care for patients to the best of our ability and to speak with (and for) them when standards slip for whatever reason.

Health services in New Zealand are near breaking point in many areas despite the very best efforts of health professionals. Is it time to panic? Perhaps not but it is long overdue that we spoke out for the patients.

The Editors

The recent history of prevention and acute management of stroke has often been more in expectation rather than certainty of effectiveness. Over the last decade or so, however, critical evaluation has redefined the role of many treatment modalities.

In the primary and secondary prevention of stroke, the long recognised importance of treating hypertension has been confirmed repeatedly.1 The more modern era has also seen controlled randomised trials confirming the stroke-protective effect of anti-platelet drugs,2 of anticoagulants for chronic atrial fibrillation,3 of carotid endarterectomy4 and of statins, especially in patients with coexisting coronary heart disease.5 The translation from proven efficacy in a trial environment to adoption in routine clinical practice, however, has been far from uniform. Even warfarin prophylaxis in chronic atrial fibrillation, for example, has not been as widely adopted as it should.6

In acute stroke, the last decade has seen a resurgence of interest in treatments that might limit damage and improve the outcome. The early hope that neuroprotective therapies effective in the laboratory would, when used in the clinical setting, limit infarct size has not yet been realised despite extensive endeavour.7 What has been recognised, however, is that vigilance for the development of complications following stroke and treating them promptly can impact significantly upon outcome. Thus, there are varying levels of evidence supporting benefits from prompt attention to dysphagia, dehydration, pyrexia, hyperglycaemia, deep venous thrombosis prevention and the early use of aspirin.8 But the most important development in the last decade has been recognition of the outcome benefits from applying traditional medical and rehabilitational therapies early, in an organised pattern of care, by experienced health practitioners committed to stroke management.

Although there is no consistent definition of either a stroke unit or of organised stroke care, there is consensus that each involves multi-disciplinary management in a planned, coordinated and integrated way from the time of admission. This concept embraces various models of acute stroke care. Those based on geographically defined areas include models as diverse as intensive stroke care units treating selected patients, stand-alone stroke units admitting unselected patients or stroke areas treating patients within a general medical ward setting. An alternative to the fixed-location approach has been the mobile specialist practitioner model, in which a stroke team advises on treatment, usually across multiple general medical wards. Until relatively recently, the benefits of any of these approaches was presumed rather than proved. A systematic review undertaken by the Stroke Unit Trialists’ Collaboration, however, changed all that.9 In an analysis of all randomised controlled trials comparing conventional hospital care in a general medical ward with that in a stroke unit, the benefits of treatment in the specialised environment were conclusively confirmed.

Stroke unit care was associated with a significant reduction in mortality, dependency or need for institutional care. The outcome benefits are sufficiently substantial that the number of patients needed to treat in order to achieve one extra ‘good’ outcome is as few as 32 for survival, eighteen for regaining independence and sixteen for returning home.10 Importantly, the improved survival is achieved without an increase in the number who survive with greater disability. Further confirmation of the value of stroke unit care are the finding that the benefits are seen across the whole age- and stroke-severity spectrum, and that the benefits are durable.11

Because the design and configuration of stroke units are not uniform, it has not been possible to determine whether there is a single ingredient that makes the outcome difference. It is, however, widely assumed that the benefits result from the bringing together of multi-disciplinary practitioners who interact closely, share their skills and adhere to an organised plan of care. The evidence suggests that benefits may be greater when a stroke service is undertaken in a geographically defined stroke unit area, than it is when the service consists of a mobile team of consulting practitioners.12 As the benefits from coordinated stroke care have been widely known for five years or more, it is surprising that metropolitan hospitals, in particular, have been slow to adopt this approach. A recent review of stroke care in the UK concluded that it was patchy and sub-optimal,13 and there is evidence that stroke unit care in the USA is under-utilised.14

The survey of acute stroke services in New Zealand by Barber et al published in this issue of the Journal is therefore timely. On the basis of responses from a questionnaire targeted to key clinicians in all New Zealand hospitals, they present a snap shot of the provision of stroke services in this country. Of 41 hospitals that routinely admit acute stroke patients to general medical wards, only four had areas designated for acute stroke care and only ten had developed critical pathways for stroke care. Approximately half of all patients with acute stroke were admitted to hospitals without guidelines covering the management of swallowing dysfunction, blood pressure management, DVT prevention and secondary stroke prevention.

The reason for the slow acceptance of the stroke unit approach to care can only be speculated upon. While it is customary to assume that the introduction of any new service will cost money, this argument has only limited substance when applied to establishing a stroke unit. The benefits most likely derive from improved organisation of management in a designated area rather than being a novel strategy requiring additional staffing and expensive facilities. It has been estimated in the UK that well over 90% of the costs associated with inpatient management of stroke are accounted for by hospital overheads and staff salaries.15 As the patients are admitted to hospital anyway, these costs are effectively the same regardless of whether the patient is managed in a stroke unit or a general medical ward. Although evidence from the randomised trials is somewhat variable, stroke unit care is generally not associated with increased length of stay; it may in fact reduce it and thus lead to cost savings. The improved functional outcome from treatment in a stroke unit would be expected to provide further cost savings through reduction in domiciliary support services needed when the patient is returned to the community.

Practitioners in this country appear reluctant to accept and adopt the new acute stroke management philosophy. A survey published as recently as two years ago revealed that of 171 New Zealand physicians active in the acute management of stroke, little more than half were convinced that stroke units were beneficial and several common deficiencies in their management of acute ischaemic stroke were identified.16 The findings from the current survey by Barber et al17 reveal the continuing failure to implement best practice guidelines.

Philip Parkin, Neurologist, Department of Neurology, Christchurch Hospital, Christchurch.

Doing our best for patients with stroke

The recent history of prevention and acute management of stroke has often been more in expectation rather than certainty of effectiveness. Over the last decade or so, however, critical evaluation has redefined the role of many treatment modalities.

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in New Zealand, despite the overwhelming evidence of the benefits. We would do well to heed the conclusion of Barber et al that the standard of stroke care across hospitals in this country will change only when physicians, geriatricians and neurologists with an interest and expertise in stroke display a commitment to the concept of organised stroke care and ensure that it happens.

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**ORIGINAL ARTICLES**

**Acute stroke services in New Zealand**

P Alan Barber, Director of the Auckland Hospital Stroke Service; Neil E Anderson, Associate Professor of Neurology, Department of Medicine, University of Auckland; Patricia Bennett, Clinical Research Coordinator, Auckland Hospital; John Grommans, General Physician and Geriatrician, Hawke’s Bay Hospital, Hastings.

**Abstract**

**Aims.** To obtain an overall picture of the organisation of acute stroke management in hospitals throughout New Zealand.

**Methods.** A questionnaire was sent to all New Zealand hospitals. The survey included questions about access to organised stroke care, the presence of designated areas for stroke patient management, guidelines for stroke management and audit.

**Results.** Responses were received from all hospitals surveyed, with 41 admitting stroke patients acutely. Five hospitals (four regional and one large urban) had organised inpatient care. Five hospitals (three regional and two large urban) had stroke physicians. Only 40-60% of the New Zealand population had access to hospitals with guidelines for the management of complications following stroke or secondary prevention. Only fifteen of 41 hospitals had audited local stroke care. There were few differences in the management of stroke patients between urban and regional centres, but care in some regional hospitals was ‘better’ than that in most large urban hospitals.

**Conclusions.** The development of an organised approach to inpatient stroke care in New Zealand and the training of general physicians, geriatricians and neurologists in stroke medicine must be seen as a priority.

**Stroke** is the third most common cause of death and the most common cause of long-term disability in the developed world. In New Zealand, there are over 6000 people with new strokes every year. 20% of these people die within the first month and 55% are either dead or dependent at one year. The survivors add to the 20 000 people living in our community with disability as the result of stroke.

After years of therapeutic nihilism, there are now three interventions that have been shown to improve outcome following an ischaemic stroke in randomised controlled trials. These are organised stroke care with a ward or team that exclusively manages patients with stroke, aspirin 160-300 mg within 48 hours of symptom onset, and intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) within three hours of symptom onset.

However, there is little information on the utilisation of these therapies or the provision of stroke services in New Zealand. Most available information has come from large urban hospitals with little known about stroke services in smaller urban and regional hospitals. The aim of this study was to obtain an overall picture of stroke services throughout New Zealand. Of particular interest was the organisation of stroke management in the first few days after admission to hospital and whether or not intravenous rt-PA is used or capable of being used within the current stroke care framework.

**Methods.** A questionnaire was sent to the medical director or a physician known to have an interest in stroke at each of 49 hospitals thought to admit patients acutely. These hospitals were identified from a New Zealand hospital directory and covered the whole of the country, including the Chatham Islands. The hospitals were divided into three groups according to the population served: large (urban hospitals serving populations > 180 000), medium (urban or regional hospitals serving populations of 40 000-180 000) and small (regional hospitals serving populations <40 000).
The questionnaire was designed by the authors to identify different aspects of stroke patient management and took approximately ten minutes to complete. Questions were asked about access to specialist or organised stroke care, the presence of designated areas for stroke patient management, the availability of brain and vascular imaging, guidelines for stroke care and audit. The questionnaire was sent out in April 2001 with a second one sent to those hospitals not responding at four weeks. Centres that had still not responded at six weeks were contacted by telephone.

Results

Questionnaires were returned by all 49 hospitals. 41 of the 49 hospitals routinely admitted acute stroke patients and are the subject of this report. There were seven large, seventeen medium, and seventeen small hospitals (Table 1). The total population served by each of these three groups of hospitals varied and the results are presented as both the numbers of hospitals responding to a particular question and the percentage of the population served by these hospitals. The seven large hospitals served 53% of the New Zealand population, the seventeen medium hospitals served 39% of the population and the seventeen small hospitals served 8% of the population. Of the eight hospitals excluded from this analysis, one was a large rehabilitation and psychiatric hospital, six were small rehabilitation hospitals or community health centres and one had been decommissioned.

### Table 1. Summary of responses by hospital group.

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<th>Hospital group</th>
<th>Number of hospitals</th>
<th>Large urban</th>
<th>Medical staff</th>
<th>Stroke physicians</th>
<th>Neurologists</th>
<th>Visiting neurologists</th>
<th>Designated areas for acute care</th>
<th>Rehabilitation</th>
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**Table 1. Summary of responses by hospital group.**

Medical staff: 30 of 41 hospitals (serving 94% of the population) had general physicians on the staff. The other eleven hospitals (6% of the population), all small regional hospitals, were staffed by general practitioners or medical officers, special scale. Nine of 41 hospitals (47% of the population) had neurologists on staff. A further twelve hospitals (37% of the population) had visiting neurologists. However, the frequency of visits by neurologists varied from three times per week to once every six months. Only five hospitals, two large and three medium hospitals (26% of the population), had stroke physicians. These physicians had trained as neurologists or geriatricians but spent a significant amount of time specifically managing stroke patients and provided leadership for local stroke services.

The initial medical assessment of stroke patients was made by house physicians (sixteen hospitals), registrars (seven hospitals), medical officers special scale (nine hospitals), general practitioners (six hospitals) or emergency medicine specialists (three hospitals). Patients were never routinely first seen by general physicians, stroke physicians or neurologists.

**Usual location of stroke care.** 37 of 41 hospitals routinely admitted stroke patients to general medical wards. Only four hospitals, all medium sized, had areas designated for acute stroke care. This was in a general medical ward (three hospitals) or a designated area for both acute and rehabilitation stroke care (one hospital). One large hospital had a mobile stroke team consisting of stroke physicians and a stroke nurse specialist, and a second has advanced plans for a geographical stroke unit.

Rehabilitation was carried out in designated areas in general medical wards in three of 41 hospitals and in designated areas in general rehabilitation wards in seven hospitals. Stroke rehabilitation in one large hospital was routinely carried out in a designated area of a general ward at a nearby hospital. Post-acute care in the other 30 hospitals (59% of the population) was carried out in general rehabilitation wards. This included six small hospitals that routinely sent patients to general rehabilitation wards at another hospital. Thus, the majority of New Zealanders did not have access to organised acute stroke care or specialised rehabilitation units.

**Use of clinical pathways and guidelines for stroke care.** Clinical pathways had been developed in 10 hospitals (serving 38% of the population). Guidelines for the management of various aspects of stroke care had been developed in 23 of 41 hospitals (serving 78% of the population). These guidelines were mainly concerned with the acute investigation of stroke patients. However, 40-60% of the New Zealand population were admitted to hospitals without guidelines covering the management of swallowing, the management of blood pressure, prevention of venous thromboembolism and secondary prevention of stroke. Only sixteen of 41 hospitals (serving 49% of the population) had audited local inpatient stroke care.

**Availability of brain and vascular imaging.** 23 hospitals (serving 88% of the population) had CT on site. Patients usually had CT within 24 hours of admission in eighteen of these 23 hospitals (65% of the population). This included two medium sized hospitals where the usual wait for CT was less than three hours. The usual wait for CT was 24-48 hours in four hospitals (two large and two medium) and 48 hours to one week in one medium sized hospital. CT was not available on site in eighteen hospitals (two medium and sixteen small hospitals). However, all eighteen of these hospitals had access to CT at another hospital, with a usual wait of 24-48 hours. Overall, one third of the population were served by hospitals in which the usual wait for CT was greater than 24 hours after admission.

Ten of 41 hospitals (49% of the population) had MRI on site and nine (49%) had cerebral angiography. More common were on site carotid duplex ultrasound scanning (26 hospitals, 90% of the population), transthoracic echocardiography (23 hospitals, 89% of the population) and transoesophageal echocardiography (fourteen hospitals, 75% of the population). However, the availability of carotid ultrasound varied between the 26 hospitals with on site imaging with a usual wait of less than one week in seventeen hospitals (64% of the population), between one week and one month in four hospitals (11% of the population) and more than one month in five hospitals (15% of the population).

**Treatment with tissue plasminogen activator.** Only sixteen patients have been treated with rt-PA in nine hospitals (five large and four medium). All but one of these
Discussion

Questionnaires offer a convenient means of surveying clinical practice in a large number of hospitals. However, questionnaires can also be problematic. The most appropriate individual within an institution may not be targeted and responses to a survey may not reflect actual practice. Attempts were made to contact physicians with a known interest in stroke at each institution, but this was usually not possible and most questionnaires were addressed to the ‘medical director’. We did not attempt to verify responses but it was made clear that no hospital would be identified. It is reasonable to assume that the responses reflect the state of acute stroke management in New Zealand.

The major finding of this study is that most New Zealanders do not have access to organised inpatient stroke care. This is despite a meta-analysis of all randomised and quasi-randomised studies, which demonstrated a reduction in the odds of death or institutionalisation care for patients receiving some form of specialised inpatient stroke care compared with conventional care. Only eighteen patients need to receive organised inpatient stroke care to prevent one from dying or being dependent at one year. Organised inpatient stroke care does not increase (and possibly decreases) length of hospital stay and is not more expensive than care in a general ward. The Melbourne declaration of the Asia Pacific consensus forum on stroke management recommended the establishment of stroke units or teams to provide acute stroke care. The development of an organised approach to inpatient stroke care in New Zealand should be addressed urgently.

New Zealand guidelines recommend that all stroke patients should be assessed by neurologists, geriatricians or general physicians with special interest in stroke. Despite this recommendation, few New Zealanders have access to hospitals with stroke physicians. Furthermore, the development of organised inpatient stroke care may be hampered by this lack of stroke physicians. Five of the six hospitals with a stroke unit, advanced plans for a stroke unit or a stroke team, have stroke physicians on their staff. These physicians have probably been the driving force behind the development of organised stroke care in their hospitals. Conversely, it is unlikely that organised stroke care will develop in other hospitals until physicians with a special interest in stroke join the staff. The training of general physicians, geriatricians and neurologists in stroke medicine should be seen as a priority.

Only one in four hospitals used clinical pathways for the management of stroke and only sixteen of 41 hospitals have audited local stroke care. The use of guidelines was more widespread, but these primarily covered the use of investigations. 40-60% of the population was served by hospitals where there were no guidelines for the management of complications following stroke or for the secondary prevention of a further stroke. This suggests that there is an ad hoc approach to the care of many stroke patients, and that the opportunity to identify and address local deficiencies in stroke care is missed.

The rational management of stroke patients requires confirmation of the diagnosis, identification of the site, extent and age of an infarct or cerebral haemorrhage, and clarification of the underlying pathophysiology. This cannot be achieved on the basis of clinical findings alone with physicians unable to accurately differentiate cerebral infarction and intracerebral haemorrhage. Most New Zealanders had access to hospitals with 24 hour CT scanning, carotid ultrasound and transthoracic echocardiography. However, the lack of CT on site in two medium sized regional hospitals, serving a combined population of 200 000 people, is of concern.

Despite access to on site CT, one third of the population were admitted to hospitals where the usual wait for CT after stroke was more than 24 hours. This included two large urban hospitals. The earlier the distinction can be made between ischaemic stroke and intracerebral haemorrhage, the sooner appropriate investigations and secondary prevention can be started. The delay in obtaining carotid ultrasound studies is also of concern as the risk of stroke complicating carotid stenosis is greatest in the first weeks after a stroke or transient ischaemic attack.

Only sixteen New Zealand patients have been treated with intravenous rt-PA in the six years since the National Institute of Neurological Disorders and Stroke (NINDS) trial was reported. The NINDS trial found that patients treated with rt-PA within three hours of symptom onset were approximately one third more likely to have complete or near complete recovery compared to those receiving placebo. Only sixteen patients need to be treated with rt-PA to prevent one from dying or becoming dependent. Treatment with rt-PA is cost effective. Subsequent case series of open label therapy have confirmed that rt-PA can be used safely and effectively outside a trial setting. However, deviation from the NINDS protocol can be harmful. Inappropriate use of thrombolysis in New Zealand is possible, as seven of nine hospitals in which patients have been treated with rt-PA did not have protocols for this therapy.

There are several impediments to the use of rt-PA in New Zealand. Recombinant rt-PA is not licensed for use in acute stroke but can be given after informed consent has been obtained. Most stroke patients have CT after the three-hour time window for treatment with rt-PA. A major change in the speed with which patients are imaged is required to increase the number of potential candidates for thrombolytic therapy. Finally, there are few stroke physicians to supervise therapy. In particular there is no hospital where a stroke physician or neurologist routinely sees patients on hospital arrival.

In general, the care of stroke patients in medium sized urban and regional hospitals was similar to large urban hospitals. Indeed, care in some regional hospitals was ‘better’ than most of the large hospitals. Three of the five hospitals with stroke physicians, and the only hospitals with designated areas for the acute management of stroke patients, were medium sized hospitals. Small regional hospitals were frequently staffed by general practitioners or medical officers special scale and patients were usually transferred to larger hospitals for further investigation and rehabilitation. Short of more rapid transport to these larger hospitals, it is unlikely that stroke care can be changed for the small segment of the population served by small regional hospitals.

There has been a failure to implement best practice guidelines in New Zealand for the acute care of stroke patients. The evidence in favour of organised inpatient care is overwhelming, and achieving this goal should be the highest priority. The situation in New Zealand is unlikely to change without training more general physicians, geriatricians and neurologists as stroke physicians. In the interim, there is a need to identify physicians in each hospital who will be responsible for stroke services.

Acknowledgements. Supported by the Julius Brendel Trust (PAB and PB).

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The widespread use of the inhaled beta-agonists, isoprenaline, is the most likely explanations were the introduction and management of asthma mortality epidemics, during the 1960s, and 1970s. Gradual rise in mortality, New Zealand experienced two increases in asthma severity with higher hospital admission rates provide information on morbidity, and are indicators of health care access and individual patterns of medical practice as well as true asthma severity. The available evidence indicates, however, that these are real increases which cannot be attributed to changes in prevalence or diagnostic fashion. The available evidence indicates, however, that these are real increases which cannot be attributed to changes in prevalence or diagnostic fashion. In addition to this gradual rise in mortality, New Zealand experienced two asthma mortality epidemics, during the 1960s, and 1970s. The most likely explanations were the introduction and widespread use of the inhaled beta-agonists, isoprenaline forte and fenoterol. Studies undertaken in the 1990s indicate a slight decline in asthma mortality in some western countries.

Increases in asthma severity with higher hospital admission rates have also been reported in the last few decades. Trends in admission rates provide information on morbidity, and are indicators of health care access and individual patterns of medical practice as well as true asthma severity. Reviews of admission data indicate that the severity of asthma attacks increased throughout the 1980s and that these could not be explained by changes in medical practice alone. Following the restriction of fenoterol, admission rates in New Zealand also declined.

A few studies have examined the seasonal patterns of asthma hospitalisation and mortality. Access to medical care or other factors associated with changes in routine asthma management have been suggested as contributing to the differing seasonal patterns found.

The Maori Asthma Review analysed data on trends in asthma mortality in Maori and non-Maori during the period 1960-1988 and in hospitalisations during 1980-1988. It concluded that asthma was more severe and that admission and mortality rates for Maori exceeded those of non-Maori

due primarily to inadequate access to appropriate health care and asthma education. In particular, asthma prevalence was similar in Maori and non-Maori children, but Maori children experience excess morbidity and higher hospital admission rates. Furthermore, the two asthma mortality epidemics disproportionately affected Maori.

We have updated and extended the analyses presented in the Maori Asthma Review by examining data on asthma mortality for Maori and non-Maori between 1962-1998 and asthma hospitalisations during 1976-1998. An examination of seasonal patterns in asthma hospitalisations and death rates between 1978-1998 is also presented. Analyses are confined to the 5-34 and 35-74 year age groups because of the well established problems with the accuracy of the classification of asthma deaths outside of these age groups.

### Methods

**Calculation of Maori and non-Maori rates.** There are considerable problems in the calculation of Maori and non-Maori mortality and hospitalisation rates during 1962-1998, particularly when examining time trends because of changes in both the numerator and denominator information. These issues have been reviewed in depth elsewhere but will be considered briefly here. Prior to 1986, both deaths and census data were based on a biological definition of Maori; from the 1986 census the question became one of self-identification, and for the 1986 and 1991 censuses the ‘sole Maori’ definition is the most appropriate in calculating mortality and hospitalisation rates because this provides reasonable consistency over time. Changes to ethnicity recording for death certificates in 1995, and further modification of the ethnicity question in the 1996 census means that for numerator and denominator data, the ‘Maori ethnic group’ definition is most appropriate from 1996 onwards.

**Mortality and hospitalisation data.** Mortality data for all asthma deaths during 1962-1998 were obtained from the New Zealand Health Information Service (NZHIS) national death registry. Mortality data were based on deaths classified using the International Classification of Diseases (ICD) for asthma as the underlying cause of death. Mortality statistics are considered unreliable for the transition period between the old and new death certification process in 1995 thus, no mortality data have been included for that year for either Maori or non-Maori. Mortality data for 1998 were provisional only and may not represent the final numbers (NHIS, personal communication). Asthma hospitalisation data were also obtained from NZHIS and includes all public hospital discharges (due to recovery or death) by year and/or month (for seasonal trends) during 1976-1998. These comprised all discharges with ICD code 493 as the principle diagnosis.

**Statistical analysis.** Because the numbers of asthma deaths were small, and the age-specific rates for individual years were unstable, we calculated five-year moving averages for presentation of the mortality and hospitalisation data. Each year’s estimate represents a weighted average of five years data (the year was given full weight, and the years ±1 year either side were given 2/3 weight and the years at ±2 years either side were given 1/3 weight). Weighted estimates were therefore not obtained for the first and last years in each series, and the estimates for the second and second-to-last year’s data points are a weighted average of three years data.

In the seasonality analyses, the annual populations at risk for Maori and non-Maori were summed separately for the 1978-1998 period, and multiplied by the fraction of days per year contributed by each month. This figure provided monthly totals of person-years at risk. The total numbers of deaths and hospitalisations for the whole period, for each month, was divided by the monthly person-years at risk to obtain monthly age-specific rates.

### Results

**Mortality.** Figure 1 shows time trends in asthma deaths in Maori and non-Maori in the 5-34 and 35-74 year age-groups during 1962-1998. In the 5-34 year age group Maori death rates increased markedly through the mid-1970s and in the peak year of 1979, were twice that of non-Maori (7.4 vs 3.7 per 100 000). The Maori Asthma Report reviewed these rates to be the highest in the world at that time. A significant reduction in mortality rates was seen for both Maori and non-Maori from the late 1980s onwards with similar rates for both groups between 1992-1994. The most recent data for 1996/1997 indicates a rise in Maori death rates while non-Maori rates continue to decline. In the older age group, the rates were disproportionately higher for Maori (17.22 per 100 000 person-years at risk) compared to non-Maori (7.9 per 100 000). After 1990 the rates declined significantly for Maori and remained relatively stable between 1994-1997 for both Maori and non-Maori.

### Hospitalisations

Figure 2 shows time trends in asthma hospitalisations for Maori and non-Maori between 1976-1998. For the 5-34 year age group, the peak hospitalisation year was 1987 with the Maori rate being 878 per 100 000, more than double the rate for non-Maori. In contrast with the fall in mortality rates for Maori after 1979, hospitalisation rates continued to increase up until 1987. The most recent Maori hospitalisation figures for 1998 show 341 per 100 000, a drop of 61.2% from the 1987 rate. An increased hospitalisation rate was also seen in the 35-74 year age group for Maori, although these were less marked than rates in the younger age group. Comparatively, non-Maori hospitalisation rates, for both age groups, were less variable throughout the same period.

**Seasonal patterns.** Figure 3 shows seasonal variation of mortality and hospitalisations in Maori and non-Maori 5-34 and 35-74 year age groups between 1978-1998. In the younger age group, different seasonal patterns of mortality and hospitalisation were seen for both Maori and non-Maori. For Maori, deaths were highest in the spring months of September and October and lowest in winter (July) with May being the peak month for hospitalisations. For non-Maori, deaths were highest in summer (January) while hospitalisations peaked in the winter month of June. In the 35-74 year age group for both Maori and non-Maori, the seasonal pattern was less discernible than in the younger age groups.

**Discussion**

There are some limitations to our data. Firstly, as mentioned earlier, the lack of standardized ethnicity data means that the monitoring of Maori health trends is not straightforward.
Secondly, the hospitalisation data represent episodes of care and may include people who have been hospitalised on more than one occasion. However, this is unlikely to have significantly affected the reported time trends or seasonal patterns for hospitalisations. Finally, the mortality data involve small numbers and age-specific rates for individual years are highly unstable, making analysis difficult. This is minimised to some degree through the presentation of the data as five year moving averages.

Despite these limitations, several conclusions can be drawn from the data. Mortality rates, while significantly reduced from the 1970s-80s asthma epidemic years, continue to be higher for Maori than non-Maori. Mortality rates for Maori were similar to non-Maori between 1992-95 but diverged again in 1996 and 1997. It is unclear whether this represents a true increase or if it could be attributable to changes in the recording of ethnicity in 1995 which has led to an apparent overall increase in reported Maori mortality.18

Hospitalisation rates remain disproportionately higher for Maori than for non-Maori. Using hospitalisation data as a measure of asthma severity these findings support previous reports that Maori experience excess asthma morbidity and higher admission rates.12,13,20 This is in contrast with evidence that asthma prevalence rates are similar in Maori and non-Maori children.21,22 Passive exposure to tobacco smoke may explain some, but not all, of the higher admission rates in Maori children.23 Asthma symptoms are more common amongst adult Maori and increase with age, in contrast to what occurs in non-Maori adults.23 Again, this may be due to increased tobacco exposure, both active and passive, in Maori.24 Additionally, several studies have concluded that differential management of asthma and inadequate access to appropriate health care are significant contributing factors to the high asthma morbidity rate amongst Maori.3,12,15,20,22

A different seasonal pattern in mortality and hospitalisation is seen for both Maori and non-Maori in the 5-34 year age-group. The results for non-Maori are similar to those presented in a previous New Zealand study23 and others undertaken overseas showing asthma mortality peaks during the summer months while the greater number of asthma hospitalisations occur during the winter.10,11,26 The seasonal pattern suggests that for both Maori and non-Maori, there are different underlying circumstances which result in death in some asthmatics and hospitalisation in others. A number of reasons for these different patterns have been proposed including seasonal changes precipitating the ‘triggering’ of an asthma attack, or an altered routine, perhaps associated with holidays when there may be reduced access to health services.10,11,26 Findings in the 35-74 year age group for both Maori and non-Maori show a seasonal pattern which is more consistent with the general increase in hospitalisations and mortality rates seen among older age groups during the winter months.26

In summary, our findings show that while there has been a reduction in asthma mortality and hospitalisation rates for Maori over the last 38 years, Maori continue to have a higher rate of hospital admissions than non-Maori. Further research would clearly be of value, particularly with regards to the seasonality patterns. Nevertheless, the findings reported here are consistent with the conclusions of the Maori Asthma Review that asthma severity is greater in Maori, despite the lack of difference in prevalence in children, and that this may in part be due to differential access to asthma health services.

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References
12. Garrett JE, Mulder J, Wong Y. Is true increase or if it could be attributable to changes in the recording of ethnicity in 1995 which has led to an apparent overall increase in reported Maori mortality.18

Figure 3. Seasonal Variation 1978-1998.
Changing the minimum legal drinking age - its effect on a central city Emergency Department

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Abstract

Aims. To quantify the effect of a recent national law change on the presentation of ethanol intoxicated patients to a central city Emergency Department (ED).

Methods. All records of ethanol intoxicated patients presenting to the ED for twelve months before and after the change to the minimum legal drinking age were studied. Each patient was classified as having laboratory confirmed intoxication, clinical suspicion only, or no record of intoxication. Three age groups were identified, 15-17 year olds, eighteen and nineteen year olds and over twenty year olds. Within each age group the proportion of presentations with ethanol intoxication was compared across the two time periods.

Results. The number of intoxicated 18 and 19 year olds increased in the twelve months after the national law change from 66 to 107 (52 to 80 for laboratory confirmed intoxication and fourteen to 27 for clinical suspicion only). This represented an increase in the proportion of presentations in this age group with intoxication (p=0.009) from 2.9% to 4.4%, a 50% increase (RR=1.51, 95%CI 1.11-2.03). There was no evidence of an increase in the proportion intoxicated for those over nineteen years (3.4% vs 3.3%, p=0.48, RR=0.97, 95%CI=0.89-1.06) although the numbers increased slightly (963 to 992). However there was a worrying trend for an increase in the 15-17 year olds, with numbers increasing from 72 to 95 and the proportion increasing from 5.0% to 6.7% (p=0.07, RR=1.35, 95%CI=0.98-1.88).

Conclusion. The recent lowering of the minimum legal drinking age from 20 to eighteen years has resulted in increased presentations to the ED of intoxicated eighteen and nineteen year olds. A similar trend was seen in the 15-17 year olds.

Auckland Hospital is a 500-bed facility that caters for most tertiary referrals. The Emergency Department (ED) is an adult-only unit (over fifteen years) receiving approximately 42 000 attendances per annum. The Auckland Public Health quarterly report consistently confirms that ethanol intoxication is the commonest overdose presentation to the ED.1

On 1 December 1999, legislation came into effect changing the minimum legal drinking age from 20 to eighteen years. Eighteen and nineteen year olds could now buy ethanol in bars, restaurants, wine shops and supermarkets. This study assesses the effect that this legislation has had and continues to have on the ED.

Methods

This was a retrospective observational study.

Inclusion criteria. All patients presenting to the ED were eligible for inclusion in the study.

Exclusion criteria. The only exclusion was for incomplete data. 624 study patients were excluded, as their records were incomplete for age, date of presentation or date of birth. This represents less than 1% of the 66 280 patients seen over the study period and 12% of those 5132 patients with laboratory ethanol levels or clinical suspicion of intoxication.

Data collection. There were three data sources used. Firstly, the presenting complaints, as accepted by the triage nurse, and discharge diagnosis given by the attending ED doctor were recorded in the electronic case management system utilised in the ED Computerised Hospital Integrated Patient System (CHiPS®A+). This record was searched using the terms ‘ethanol’, ‘alcohol’ or ‘intoxication’ and a file of 1308 (Table 1) records was created. The second data collection method relied on the attending ED doctor to collect demographic data regarding each overdose on a standardised collection form. The forms were collected each day and checked to ensure completion. Missing data were later entered retrospectively from the written medical record, giving a higher detection rate than for the CHiPS®A+ system alone. Thirdly, the laboratory database was searched for all ethanol levels for the corresponding period. This provided 4075 records (Table 1) that had a complete data set, of which 1862 showed an ethanol level of ≤17 mmol/L.

875 patients were captured by both the clinical and laboratory data collection methods and were included only once for analysis. This allowed some assessment of the accuracy of clinical opinion regarding the patient’s level of ethanol intoxication (Table 1). Of the 875 records, 154 had an ethanol level ≤17 mmol/L and were regarded as not intoxicated. Chi squared tests (with Yates’s correction) were used to determine whether there was an association between the ratio of intoxicated patients presenting before and after the law change. This was performed for three separate age groups: <18, 18-19 and >19 years.

The number of intoxicated eighteen and nineteen year olds increased in the twelve months after the national law change from 66 to 107 (52 to 80 for laboratory confirmed intoxication and 14 to 27 for clinical suspicion only). This was a significant increase in the proportion of presentations

<table>
<thead>
<tr>
<th>Total</th>
<th>Ethanol ≤17 mmol/L</th>
<th>Ethanol ≥17 mmol/L</th>
<th>Clinical suspicion</th>
<th>Not Recorded</th>
<th>Not Requested</th>
</tr>
</thead>
<tbody>
<tr>
<td>4075</td>
<td>2139</td>
<td>1862</td>
<td>154</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>Emergency Data</td>
<td>1308</td>
<td>2139</td>
<td>1154</td>
<td>154</td>
<td>74</td>
</tr>
<tr>
<td>Total for analysis</td>
<td>4508</td>
<td>2139</td>
<td>2295</td>
<td>74</td>
<td>74</td>
</tr>
</tbody>
</table>
in this age group with intoxication (p=0.009) from 2.9% to 4.4%, a 50% increase (RR=1.51, 95% CI 1.11-2.03). There was no evidence of an increase in the proportion intoxicated for those >nineteen years (3.4% vs 3.3%, p=0.48, RR=0.97, 95% CI=0.89-1.06) although the numbers increased slightly (963 to 992). However, there was a worrying trend for an increase in the 15-17 year olds, with numbers increasing from 72 to 95 and the proportion increasing from 5.0 to 6.7% (p=0.07, RR=1.35, 95% CI=0.98-1.88), Table 2.

When intoxicated presentations were stratified by laboratory data and clinical suspicion data, there was still a significant difference in the ratios of presentations in the key eighteen and nineteen year age groups (p=0.023 and 0.009, Table 2).

**Discussion**

The opportunity to perform this type of study is rare. To our knowledge this is the first New Zealand study to statistically corroborate anecdotal evidence supporting a significant increase in the presentation rate of ethanol intoxicated eighteen and nineteen year old patients. There are limitations to the information. We believe that not all patients presenting with intoxication were captured by the existing data collection systems. Importantly, however, there were no differences in data collection techniques and no difference in training given to staff during the study period so it is envisaged that this would minimise the introduction of bias. There is evidence to support this assumption. In the year prior to the law change 123 of 2271 patients aged eighteen and nineteen were tested for level of ethanol intoxication. In the year following, 157 of 2446 patients were tested. This difference did not reach statistical significance (p=0.145). Furthermore 42% of those 123 tested prior to the law change had a level consistent with significant intoxication compared to 51% of the 157 tested afterwards. This also supports the assumption that staff were not testing more patients after the law change. Of the 875 records where a laboratory level was obtained to correlate with clinical suspicion of intoxication, only 154 records were found to be under the legal driving limit. This shows that laboratory levels confirmed significant intoxication 82.4% of the time when a laboratory level was requested.

The low total number of intoxicated patients may reflect the attitude of ED clinical staff to a significant public health problem. Cherpital et al² showed that 91 of 988 representative patients were found to have an ethanol-related ED visit but only ten of these were identified as having an ethanol problem by ED staff with only one referred for ethanol treatment. It may well be that ethanol as a factor in presentation is often overlooked or not considered for further action. The apparent trend toward newer classes of drug intoxication may be reducing the emphasis placed on ethanol abuse by EDs and the public in general.

The importance of research, in particular quality local research, to form part of the policy debate regarding ethanol legislation cannot be overestimated. It has been identified by Wagenaar⁵ that good research in this area can, and does, influence policy debate. In a review, Casswell et al⁶ stated “there is a lack of directly relevant local research to inform the policy debate”. There is no doubt that ongoing local research will add validity to argument in this important area of public debate.

There is little doubt that ethanol consumption and the legislation affecting it has an effect on morbidity and mortality. Douglass and Millar⁷ published compelling evidence linking ethanol and road traffic crashes in Michigan. After reduction in the drinking age to eighteen years in 1972 there were “at least 4600 additional road traffic crashes, in the 18-20 year age group, associated with ethanol from 1972-1975”. MacKinnon and Woodward⁸ published results comparing states that had raised the minimum legal drinking age and those that had not. In those states with a new higher minimum legal drinking age there were “significant immediate reductions in fatalities among younger drivers”.

Muller⁹ produced work suggesting that an increase in per capita ethanol consumption can be linked contemporaneously to an increase in hospital admission rates. This study looked at 42 community hospitals across five decades. Whereas our study does not show an increase in ethanol consumption in the 18-19 year age group there is no doubt that ongoing local research will add validity to argument in this important area of public debate.

**Table 2. Presentations before and after the law change.**

<table>
<thead>
<tr>
<th>Age</th>
<th>Intoxication</th>
<th>Before</th>
<th>After</th>
<th>p-value*</th>
<th>p-value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-17 yrs</td>
<td>Lab confirmed</td>
<td>49</td>
<td>70</td>
<td>0.125</td>
<td>0.072</td>
</tr>
<tr>
<td></td>
<td>Clinical suspicion only</td>
<td>72 (5.0%)</td>
<td>95 (6.7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not intoxicated</td>
<td>1362</td>
<td>1329</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-19 yrs</td>
<td>Lab confirmed</td>
<td>52</td>
<td>80</td>
<td>0.023</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>Clinical suspicion only</td>
<td>14</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total intoxicated</td>
<td>66 (2.9%)</td>
<td>107 (4.4%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not intoxicated</td>
<td>2205</td>
<td>2339</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;19 yrs</td>
<td>Lab confirmed</td>
<td>800</td>
<td>811</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clinical suspicion only</td>
<td>163</td>
<td>181</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total intoxicated</td>
<td>963 (4.4%)</td>
<td>992 (3.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Not intoxicated</td>
<td>27478</td>
<td>29272</td>
<td>0.571</td>
<td>0.48</td>
</tr>
</tbody>
</table>

*p-value calculated with three intoxication categories (df=2). †p-value calculated for lab and clinically suspected intoxication combined.

**Figure 1. Ethanol intoxicated 18 and 19 yr old admissions before and after 1 December 1999.**
is local work to support this proposal. Casswell and Zhang published a longitudinal study showing that an adolescent’s access to ethanol was a powerful determinant of the quantities of ethanol drunk, more powerful than peer or parental influences. These data support our finding that increasing the minimum legal drinking age may well have increased the rate of ED attendance.

Not all international work supports our findings. Two studies from the UK suggest otherwise. Graham et al published data from a six week study from an ED in Edinburgh showing no change in ethanol or assault related presentations following some restrictions placed on extensions to permitted licensing hours. Rhodes et al found no overall change in presentation rates to an ED in Newcastle after liberalisation of the drinking laws allowed “all-day drinking”. Neither of these studies, however, looked at age-related presentations and the Newcastle study did show a trend towards increased frequency of nighttime attendees.

Our study lends statistical weight to the body of opinion that suggests increasing the legal availability of ethanol to young people may well increase morbidity in that same group. By relying on measured blood ethanol concentration to identify cases, our study almost certainly underestimates the extent of hazardous drinking in the <20 year age group. Maio et al demonstrated that up to 33% of patients <20 presenting to one ED admitted to ethanol use/misuse yet salivary ethanol levels identified only 5% of these patients. The blood ethanol level cut-off was set at the legal driving limit as a widely recognised level of impairment. However, this may well mean the findings in our study present a conservative view.

Acknowledgements. Thanks to Dr Peter Freeman, director of the Auckland Hospital Emergency Department, and Teena West, statistician.

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Cancer institute director’s exit leaves NIH in the lurch
Richard Klausner resigned on 11 September as director of the National Cancer Institute (NCI) to become president of the new Case Institute of Health, Science and Technology. The institute is bankrolled by Steve Case, the founder of America Online (AOL).

Klausner’s departure after six years at the NCI accentuates the leadership void at the US National Institutes of Health (NIH). The biomedical research agency now lacks permanent directors both at its headquarters and at its largest institute – the NCI accounts for $3.7 billion of the NIH’s $20.4 billion budget this year.

The positions are vacant at a time when the agency is facing several vexing issues, including the lack of a long-term strategy for managing its recent unprecedented expansion. There is also controversy over the government’s role in funding human embryonic stem-cell research and growing concern about patient safety in NIH-supported clinical trials.

Sources inside and outside the NIH had been predicting Klausner’s imminent departure for more than a month. They said Klausner and officials at the Department of Health and Human Services, the NCI’s parent agency, were at odds over salary increases Klausner had given senior administrative staff and over travel by NCI staff to scientific meetings.

Klausner says that such issues “were not even a component” of his decision to resign. He attributes speculation about tensions between himself and health-department officials to “the internal rumour mill”.

“Whenever an administration changes, these things come and go,” he says, “But they were never in my mind as I was determining whether I should stay or go. There is a tendency for people to put forth their own issues when they’re trying to explain events.”

“I love this institution, but the reality is that the NIH director does not get closer to the science – he gets further from the science,” Klausner says. “And what I was missing more and more was being close to the content”.


A quick sniff could do wonders for your sex life
An aphrodisiac nasal spray that is more potent than an oyster and faster-acting than Viagra has been developed by researchers in the US. If clinical trials are successful, this “desire aerosol” could provide the first effective treatment for women who suffer from a low libido.

Tests on animals and people have shown that the experimental drug PT-141 made by Palatin Technologies in Edison, New Jersey, can stimulate desire and sexually arouse both sexes. But unlike Viagra, the target of PT-141 is the brain rather than the sexual organs.

PT-141 is a synthetic copy of a naturally occurring neuropeptide called α-melanocyte-stimulating hormone. MSH plays a role in stimulating sexual function and appetite. Within 10 to 15 minutes of squirting PT-141 up the nose, the drug activates melanocortin receptors in the hypothalamus region of the brain. This prompts the release of other sex hormones in a domino-like effect, says neuro-scientist Annette Shadiack, who directs the biological research function and appetite. Within 10 to 15 minutes of squirting PT-141 up the nose, the drug activates melanocortin receptors in the hypothalamus region of the brain. This prompts the release of other sex hormones in a domino-like effect, says neuro-scientist Annette Shadiack, who directs the biological research

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Antibiotic resistance in uncomplicated urinary tract infection: problems with interpreting cumulative resistance rates from local community laboratories

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Abstract

Aims. To determine the resistance rates and patterns in bacteria causing uncomplicated urinary tract infections (UTIs) presenting to general practitioners (GPs) in Christchurch.

Methods. 82 randomly selected GPs in Christchurch participated in the study. Midstream urine (MSU) samples were prospectively collected for standard microbiological analysis on all women between the ages of 16 and 50 years presenting with symptoms of dysuria and frequency and who had positive dipstick testing to either (or both) nitrites or leucocytes. MSUs were submitted for bacterial colony counts and resistance testing of isolates present in adequate numbers.

Results. 374 specimens were collected. 299 filled the inclusion criteria, of which 94 fulfilled criteria for significant infection. Trimethoprim resistance was found in 8, (8.5%) (95%CI 2.8, 14.2) overall with a resistance rate for Escherichia coli (E. coli) to trimethoprim of 11.5%. This compared with cumulative resistance rates from local community laboratories for E. coli to trimethoprim of 19%. For a woman in this age group presenting with symptoms of UTI we estimated that her probability of having a trimethoprim resistant organism was 2.7%.

Conclusion. Trimethoprim remains a reasonable first line treatment for uncomplicated UTI in Christchurch. Actual resistance rates are significantly less than those derived from routine pooled laboratory specimens, and when used in an intention to treat calculation to inform empiric prescribing, become even less significant. While collection of these routine data is essential to provide early warning of emergent resistance, a truly representative rate should be determined to inform prescribing decisions if resistance appears to be increasing.

Antibiotic resistance is a growing worldwide problem. Inappropriate and excessive use of antibiotics contributes significantly to this growth. It is desirable to prescribe antibiotics only when necessary, and with the most appropriate antibiotic for the known or likely organism(s) causing the infection. The majority of human antibiotics are prescribed in general practice, mainly in short courses for acute infections. Urinary tract infections (UTIs) represent a common presentation in general practice. For uncomplicated UTI (cystitis) in women, there is an increasing trend toward empiric therapy.

Ideally the empiric prescribing choice of first line antibiotic should be guided by knowledge (if available) of local sensitivity patterns of likely pathogens. The most popular choice reflects the published recommendations in New Zealand for single dose or short courses of trimethoprim. There has been debate as to whether trimethoprim should remain first choice for empiric therapy as resistance rates appear to be increasing (sensitivity testing by Medlab South, Jan-June 2000, and by Diagnostic Medlab, Auckland, 1999). It is unclear what level of resistance represents an unacceptable rate. It has been suggested that an antibiotic should no longer be considered as a suitable first line empiric treatment if the antibiotic resistance rate exceeds 20%. Systematic surveillance that would allow careful appraisal of resistance rates is not carried out in New Zealand. Available resistance data are generally derived from routine clinical specimens being processed by laboratories. Recent cumulative resistance rates from local community laboratories in Canterbury show trimethoprim resistance in E. coli (the predominant infecting organism) at 19% (sensitivity testing Medlab South and Southern Community Laboratories 2000). Equally high rates (22%) have been found in Auckland (susceptibility testing Diagnostic Medlab, Auckland, 1999). The source of specimens processed influences the cumulative resistance rates from local community laboratories for a number of reasons. Clinical behaviour is changing; the move toward empiric therapy results in a greater proportion of specimens sent being for complicated infections and treatment failures, creating potential for a bias towards higher reported rates of antibiotic resistance. Added to this, community laboratory data are usually presented by organism, cultured from multiple sites.

Determining meaningful resistance data to inform empiric prescribing decisions in general practice requires systematic collection of representative samples from a general practice network. A randomised and representative (sentinel) network of 87 GPs in the Christchurch area has recently been set up as detailed below. This group of clinicians prospectively supplied specimens for microbiological analysis to obtain this information. In this paper, we report the first study from this network.

Methods

We took a stratified random sample using the RandSamp program[15] of 87 GPs from the Pegasus Health Independent Practitioners Association (IPA). This IPA includes 80% of all Christchurch GPs. All the IPA GPs were stratified into three groups according to the proportion of consultations with community service card holders to balance the tendency for higher socioeconomic areas to have greater density of GPs. The RandSamp program was used to randomly select 29 from each group, 87 in total. Eighty two (94%) agreed to participate.

Inclusion criteria were: women aged between 16 and 50 years who presented with symptoms of cystitis supported by the presence of white cells or nitrites in the urine by dipstick in whom empiric antibiotic
treatment was to be commenced (‘intention to treat’). Exclusion criteria were: clinical evidence of pyelonephritis (temperature ≥38°C, renal angle tenderness or rigors), known abnormal urinary anatomy, recent instrumentation of the urinary tract and pregnancy. MSUs were collected and processed in the usual way, laboratory forms were identified with a sticker, and any recent antibiotic therapy noted. Both Christchurch community laboratories were involved and they performed standard analysis (standard dipstick, microscopy, culture and disc sensitivities). Results were sent back to the referring GP in the normal way. The initial results were collated by the laboratories and identifying details removed before further analysis.

A UTI was defined as ≥ 20 leucocytes per mL of urine and pure growth of ≥10⁵ organisms per mL of a uropathogen. Antibiotic susceptibility was initially determined by disc testing according to National Committee for Clinical Laboratory standards (NCCLS). The minimum inhibitory concentration (MIC) of isolates identified as resistant was determined by agar dilution except for Proteus spp which were tested by the broth dilution method. Both techniques were performed according to NCCLS guidelines by a central laboratory. Simple descriptive statistics were obtained in EXCEL and SAS and proportions, normal 95% confidence intervals and tests for linear trend were performed using the CIA software programme.

Results

The average consultation rate for the GP group was 100/week. International studies have found around 1% of consultations are for urinary tract infection (WaiMedCa data reports 1.6% for ‘urological conditions’). Pegasus IPA data report 29% of MSU laboratory samples were from the 16-50 year age group. 87 GPs x100 consultations x 12 weeks x 0.01 x 0.29 gave an estimated potential sample size of 303. 374 samples were collected in the twelve-week period, indicating the sample was representative. 75 samples did not meet inclusion criteria for age, sex or uncomplicated UTI and were excluded from subsequent analysis. Of the remaining 299 samples, 223 had WBC >20wbc/mL, and 94 (31%) of these 10 or more organisms/mL pure growth of a uropathogen. A further 36 samples had pure growth of 10⁴ -10⁵ cfu/mL. One sample grew both E. coli and Group B Streptococcus in significant numbers. The NCCLS guidelines do not include resistance levels for Group B Streptococcus so the MICs are reported.

The disc sensitivity testing showed the following total in vitro resistance rates for significant isolates: trimethoprim 9/94 (9.6%) (95% CI 3.6, 15.3), co-trimoxazole 9/94 (9.6%) (95% CI 3.6, 15.3), amoxicillin 27/94 (28.7%) (95% CI 19.6, 37.9), amoxicillin/clavulanate 3/94 (3.2%) (95% CI 7, 9.0), nitrofurantoin 1/94, 95% (1.1%) (CI 0.0, 5.8) and norfloxacin 0%. Two organisms were unable to be revived for determination of the MIC (one E. coli and one Corynebacterium) and these were classified as resistant, using the original disc test result.

Resistance rates determined by MIC are shown in Table 1. By MIC one of the resistant organisms on disc testing was found to be sensitive so the MIC resistance rate to trimethoprim was 8/94 (8.5%) (95% C.I. 2.8, 14.2). In a small number of specimens there was a discrepancy in classification resistance to amoxycillin/clavulanate. The difference was within one dilution on MIC testing which is within the acceptable range except in one case where it appears there was an error in specimen labelling. Where there was a difference in classification, the MIC result was used. On MIC testing all organisms that were resistant to trimethoprim were also resistant to amoxycillin/clavulanate.

The group B streptococcus MICs were as follows: amoxycillin/clavulanate <0.5/0.5 trimethoprim 16/16 nitrofurantoin 32/32 sulphamethoxazole ≥256/≥256 ciprofloxacin 1.0/1.0 norfloxacin 8/4.

Resistance varied according to the infecting organism. As can be seen in Table 1 S. saprophyticus showed a lower resistance rate than E. coli.

Table 2 and 3 show the proportions of infecting organism and resistance rates by age bands. E. coli infections increased significantly with age from 43% of infections in the 16-21 year age band rising steadily to 77% in the 41-50 year age band (χ² statistic 5.0, p=0.02). There was also a significant difference in in vitro trimethoprim resistance in different age bands, with more organisms in women aged 31-40 resistant to trimethoprim (χ² 10.4, 3 d of f, p=0.02). There was no evidence of a significant linear trend with increasing age using the χ² test for trend. The difference between age bands was non significant for amoxycillin/clavulanate (Chi squared statistic 3.69, 3 d of f, p=0.297).

Table 7. Infecting organism by age.

<table>
<thead>
<tr>
<th>Age band (years)</th>
<th>E. coli</th>
<th>S. saprophyticus</th>
<th>% other</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-21</td>
<td>42.9</td>
<td>28.6</td>
<td></td>
</tr>
<tr>
<td>22-30</td>
<td>61.9</td>
<td>19.1</td>
<td></td>
</tr>
<tr>
<td>31-40</td>
<td>66.7</td>
<td>22.2</td>
<td>12.0</td>
</tr>
<tr>
<td>41-50</td>
<td>76.8</td>
<td>12.0</td>
<td>12.0</td>
</tr>
</tbody>
</table>

There was a significant linear trend for increasing proportion of E. Coli with age (Chi sq test 5.187 p=0.02).

Table 3. Resistance by age.

<table>
<thead>
<tr>
<th>Age band (years)</th>
<th>Nitrofurantoin % resistant</th>
<th>Amoxycillin/ clavulanate % resistant</th>
<th>Trimethoprim resistant</th>
<th>Sulphamethoxazole resistant</th>
<th>% resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>16-21 (n=25)</td>
<td>0</td>
<td>4.76</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>22-30 (n=21)</td>
<td>9.52</td>
<td>4.76</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>31-40 (n=27)</td>
<td>0</td>
<td>18.52</td>
<td>22.22</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>41-50 (n=25)</td>
<td>4</td>
<td>8</td>
<td>8</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

There was a statistically significant difference in trimethoprim resistance in different age bands (Chi sq statistic 10.41, 3 d of f, p=0.015). The difference between age bands was non significant for amoxycillin/clavulanate (Chi sq statistic 3.69, 3 d of f p=0.297). There was no evidence of a significant linear trend using Chi sq test for trend for either.

Table 1. Resistance rates to antibiotics by organism.

<table>
<thead>
<tr>
<th>All organisms</th>
<th>N=94</th>
<th>E. coli</th>
<th>S. saprophyticus</th>
<th>N=59</th>
<th>N=39</th>
<th>Other</th>
<th>N=16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxycillin</td>
<td>27</td>
<td>20</td>
<td>21</td>
<td>4</td>
<td>3</td>
<td>26</td>
<td>9</td>
</tr>
<tr>
<td>Amox/clavulante</td>
<td>7</td>
<td>6</td>
<td>21.1</td>
<td>4</td>
<td>1</td>
<td>3.6</td>
<td>1</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>8</td>
<td>3</td>
<td>7</td>
<td>9</td>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Sulphamethoxazole</td>
<td>10</td>
<td>7</td>
<td>7</td>
<td>9</td>
<td>8</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>16.6</td>
<td>10.2</td>
<td>0</td>
<td>15.7</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Norfloxacin</td>
<td>8/4</td>
<td>8</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Figures in brackets are 95% confidence intervals. All results represent MIC resistance rates except amoxycillin where disc results are reported. Included in ‘other’ were: Streptococcus agalactiae, Serretia sp, Proteus mirabilis, Klebsiella pneumoniae, Enterobacter, Corynebacterium, Citrobacter freundii, Gausdalea Negativus Staphylococcus and Lactobacillus sp. Percentages are not calculated individually as there was only one of each organism type where resistance was found. These are noted below: *1 Serretia 1 Klebsiella pneumoniae 1 Citrobacter freundii 1 Enterobacter cloacae 1 Corynebacterium 1 Proteus mirabilis 1 Proteus mirabilis, Enterobacter cloacae.

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Only fourteen women had taken antibiotics in the last month. The proportions of infecting organism type in this group were very similar to those in women who had not taken antibiotics in the previous month (n=80): *E. coli* 64% vs 63% *S. saprophyticus* 21% vs 20% other 14% vs 17%. The number of resistant organisms was too small to give enough statistical power to determine whether recent antibiotic use affected *in vitro* resistance.

**Discussion**

We are aware there is debate about the criteria for defining infection. However, we did not wish to enter the discussion around definition of infection, but rather to report *in vitro* resistance rates in specimens where it could be agreed there is definite infection. We therefore chose ≥10⁵ organisms/mL as this is a conservative and agreed definition of infection. It is noteworthy however that the *in vitro* resistance rates to trimethoprim were similar in the 36 samples with ≥10⁵ - <10⁶ cfu/mL of a uropathogen. The resistance rate to trimethoprim is the key finding. If the original sample of 299 were used for an intention to treat analysis, for a GP about to initiate antibiotic treatment for a woman aged 16–50 years with symptoms of an uncomplicated UTI the probability of that woman having a proven infection with a trimethoprim resistant organism is around 2.7%. We note the importance of considering the likelihood of infection with chlamydia where there are leucocytes but no infecting organism.

It is interesting to note that there appeared to be a significantly higher *in vitro* resistance rate in the 31–40 age band. This is not explained by the proportion of *E. coli* infections in this band (which are more likely to be resistant), as the 41–50 age band had an even higher proportion of *E. coli* but a lower *in vitro* resistance rate. The reasons for this apparent difference are unclear and worthy of further study if this finding is confirmed in a different sample. International papers have noted an association between resistance and increasing age. The resistance rate for trimethoprim found in *E. coli* (11.9%) is lower than that reported from the regional laboratory where it was found to be 19%. (Medlab South, cumulative susceptibility tests, 2000). There may be several reasons for this – firstly the laboratory data are opportunistically collected and with increased empiric treatment of urinary tract infection based on symptoms and dipstick results, there is an increasing tendency for MSUs to be sent only when infection is complicated or treatment has failed. The reported results also represent a population that is not confined to women with an uncomplicated UTI, but includes males, children, those with urological conditions and abnormal urinary anatomy. Finally the reported results represent all *E. coli* infections from all sites not just urinary pathogens. For these reasons the laboratory results do not provide a representative sample for indicating community levels of antibiotic resistance in uncomplicated UTIs. It is also important to note that, while *E. coli* is the most common organism responsible for UTIs, it has a higher resistance rate than other causative organisms such as *S. saprophyticus*, which is known to be inherently sensitive to most antibiotics *in vivo* and this needs to be considered when ‘intention to treat’ is empirically based on rates which are presented by organism rather than by site. It is imperative that accurate data are obtained both as an early warning of the emergence of significant levels of resistance in the community, but more importantly to guide clinical practice. The cumulative resistance rate data from local community laboratories is an essential first step in informing this process.

This study shows it is essential that areas of apparent increased resistance highlighted by these data are more rigorously explored before changes in clinical practice are contemplated. General practice involves much empiric treatment and there is often sparse local evidence to inform prescribing decisions. In this instance laboratory reported *E. coli* resistance rates to trimethoprim were approaching the level (20%) where an alternative first line antibiotic would be considered. *In vitro* resistance rates to amoxycillin/clavulpanate are also low however, it has a high rate of gastrointestinal side effects. Concerns have also been raised about *in vivo* resistance to trimethoprim due to differential rates of amoxycillin and clavulanate excretion in the renal tract raising questions about its use in the treatment of amoxycillin resistant *E. coli*.

We note a resistance rate of 29% to amoxycillin in our study so for the reasons above would not recommend this as a first choice for empiric treatment of urinary tract infections. It is intended to use this methodology to monitor changes in antibiotic resistance for pathogens from urinary, respiratory tract and other sites.

**Acknowledgements.** We thank the 87 GPs and practice nurses of the Sentinel Network who did all the work collecting and identifying the specimens. Pegasus Health funded and supported this study and Chris Leathart and Harsed Chima were on the original steering group which developed the concept. Medlab South and Southern Community Laboratories in Christchurch identified and processed results from all specimens. Elisabeth Wells provided biostatistical advice and support and Alison Parsons provided invaluable office support.

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Heart failure is a major public health problem in New Zealand with hospitalisations due to heart failure estimated to consume about 1% of the total health budget.1 Admissions for heart failure increased by more than 50% between 1988 to 1997.2 The New Zealand approach to improving heart failure outcomes has been through the development of treatment guidelines, promotion of best practice in continuing education programmes for general practitioners and investment in ‘integrated care’ for patients.

This paper compares heart failure hospitalisation and mortality rates for Mäori aged 45 years and over with those for non-Mäori New Zealanders. There are good reasons to focus on differential outcomes for Mäori with heart failure. Firstly, despite a decline in mortality rates from heart disease in New Zealand, cardiovascular diseases still account for the greatest number of deaths and there remains a marked difference in cardiovascular mortality between Mäori and non-Mäori.3,4 Secondly, heart failure is a condition with good evidence on which to base action. Relatively simple pharmacological and health service interventions can reduce hospitalisation rates and decrease mortality.5,6 Ethnic differences in age of onset, aetiology, admission rates, quality of care and mortality from heart failure are apparent in other countries.7-11 This highlights the need to recognise sub-populations with different risk profiles and consider the influence of ethnicity on health system responses.

Methods
Data on deaths (1988-97), and public hospital admissions (1989-98) with heart failure listed as a primary or secondary cause, were obtained from the New Zealand Health Information Service.

Mortality data included deaths with the following causes (ICD-9 codes): heart failure - cause unspecified: 428, 429.1, 429.3; hypertensive heart disease: 402; primary cardiomyopathy 425.4; alcoholic cardiomyopathy 425.5; myocarditis: 422, 429.0 and other causes of heart failure 429.4, 425 (except 425.4 and 425.5). Heart failure during or resulting from a procedure (997.1) was excluded.

Two types of hospitalisation data were obtained from discharge diagnoses from public hospitals (discharge alive or death in hospital): firstly, those with a primary diagnosis of heart failure as defined by the ICD-9 codes listed above; secondly, admissions with a primary diagnosis of chronic rheumatic heart disease (391-398), ischaemic heart disease (410-414), non-rheumatic valvular heart disease (424), hypertensive heart disease (401-405), diabetes (250), cardiomyopathy (425), which also had a secondary diagnosis of heart failure in any other diagnostic field. Age-sex specific rates of all hospitalisations (including re-admissions) and deaths were calculated for Mäori and non-Mäori in the age groups 45-64 years and 65 years and over. Hospitalisations were examined yearly for the period 1989-98. Mortality data were aggregated for the decade 1988-97 due to the smaller number of events.

Within each age group, ratios of Mäori to non-Mäori rates of death and hospitalisation were calculated using rates directly standardised to Segi's world population. 95% confidence intervals were calculated for standardised rates and ratios.14 Yearly proportions of readmissions were estimated by calculating the number of people who had more than one admission for heart failure during the calendar year, and dividing the sum of their readmissions over the total number of admissions during the period.

Ethnicity classification - numerator. In New Zealand until September 1995, deaths were officially registered as Mäori if the decedent was recorded (usually by funeral directors) as half or more Mäori descent. Thereafter the ethnic group(s) of the deceased was nominated from a predetermined list. Before the 1995 change in data collection, there was known under-reporting of Mäori deaths.15 The National Health Index (NHI) reports the ethnicity of individuals, based on data from hospital admissions (supposedly self-defined although Mäori are likely to be under-reported),16 cancer registrations, or death registrations. Since July 1996, multiple ethnic groups can be recorded. However, during 1997-1998 less than 2% with ethnicity specified were reported with more than one ethnic group.15 Among the heart failure admissions reported here, from 1996 to 1998 only 5% out of 4570 Mäori admissions (1%) were recorded with more than one ethnic group.

We classified deaths as Mäori if the decedent was coded as Mäori on either the death registration or the NHI. Otherwise deaths were classified as ‘non-Mäori’. This resulted in 139 (24%) more Mäori deaths at ages 45 and over than in the official mortality data using death registrations. From 1988 to 1995, 16% more deaths from heart failure were recorded as Mäori on the NHI than on the death registrations. After 1995 however, the direction reversed with 37 (24%) more deaths recorded as Mäori on the death registrations than on the NHI. This suggests under-reporting of Mäori deaths in both registrations and NHI prior to 1993, and our data are likely to underestimate Mäori mortality from heart failure.

Denominator. Changes in the classification of ethnicity in mortality, hospital and census data during the period studied, and the effect of these changes on health trend data, have been described elsewhere.16 Mortality and hospitalisation rates were calculated using yearly mean population estimates of Mäori (single ethnicity) and non-Mäori based on the 1986 Census for the years 1988-90 and on the 1991 Census for the years 1991-1998. Acknowledging several potential denominator populations may be used to define ‘Mäori’ rates leading to different mortality rates,17 we also calculated rates and ratios using an alternative denominator population (Mäori Ethnic Group based on 1996 Census, for the years 1996-98).

Results
Hospitalisations. Mäori rates of hospitalisations with a primary diagnosis of heart failure were on average 8.2 and 9.5 times higher than non-Mäori among males and females respectively aged 45-64 years, and 3.0 and 4.1 times higher among those aged 65 years and over. Disparities were also

Heart failure: ethnic disparities in morbidity and mortality in New Zealand
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Abstract
Aims. To compare heart failure outcomes for Mäori and non-Mäori New Zealanders.


Results. Mortality from heart failure was more than 8.8 times higher among Mäori males aged 45-64 years than non-Mäori (95% confidence interval 7.6 to 10.1), and 3.5 (3.1 to 4.1) times higher among Mäori aged 65 years and over. Mortality ratios for females were similar. Hospital admissions with a primary diagnosis of heart failure averaged eight times higher among Mäori males aged 45-64 and nine times higher among Mäori females compared to non-Mäori. Mäori males and females aged 65 years and over had three and four times the non-Mäori rate of admission.

Conclusions. Disparities between Mäori and non-Mäori in outcomes for heart failure are high in New Zealand. Effective, evidence-based interventions have not yet impacted on populations most ‘at risk’.
observed in the rates of admission for Māori with a secondary diagnosis of heart failure compared to non-Māori, with ratios averaging 3.5 and 6.2 in males and females aged 45-64 years, and 1.4 and 2.0 respectively in the older age group. Table 1 presents Māori and non-Māori hospitalisation rates and ratios with 95% confidence intervals for the beginning and end of the period.

Readmission. Of the admissions with a primary diagnosis of heart failure, approximately 40% of the Māori admissions in each year were readmissions, compared to 33% of the non-Māori admissions. Of the Māori patients admitted to hospital in any calendar year, 21% on average had more than one admission during that year, while 17% of the non-Māori patients had more than one admission.

Mortality. Between 1988 and 1997 there were 614 deaths (all ages) with a primary cause of heart failure among Māori males and 355 among Māori females, 3232 non-Māori males and 4320 non-Māori females died from heart failure during this period. 66% of the deaths among Māori men occurred before the age of 65 years, compared to 23% of the deaths among non-Māori men. Among Māori women 41% occurred before the age of 65 years and 7% among non-Māori women.

There was marked disparity in mortality between Māori and non-Māori aged 45-64 years, with ratios of more than eight for both men and women. Among those aged 65 years and over, Māori still had over three times the rate of non-Māori deaths (Table 2). Disparities remained marked when recalculated using Māori as defined on death registrations only and the larger Māori Ethnic Group denominator for 1996-97 based on the 1996 census, with Māori to non-Māori ratios for males of 6.0 (45-64 years) and 2.2 (65 years and over) and for females of 6.0 (45-64 years) and 2.2 (65 years and over).

Discussion
This retrospective analysis of hospitalisations and deaths due to heart failure shows significant disparities between Māori and non-Māori. Mortality rates from heart failure are more than eight times higher for Māori than for non-Māori aged 45-64 years, and three times higher among those aged 65 and non-Māori. Mortality rates from heart failure shows significant disparities between Māori and non-Māori. This retrospective analysis of hospitalisations and deaths due to heart failure shows significant disparities between Māori and non-Māori. While significant ethnic differences have been found in other international studies, the disparities were less marked. The age-adjusted death rate from heart failure for the black population <65 years in the United States, for instance, is approximately 2.5 times that of whites and hospitalisation rates 33-50% higher for blacks than whites are reported.

While the limitations of available data present potential weaknesses for this study, there is no evidence to suggest that these factors selectively altered data related to Māori more than non-Māori. Ethnicity classification problems may lead to under-estimation of Māori admissions and deaths, as discussed above. Despite these uncertainties, no matter which populations we use, the magnitude of the ratios is sufficiently high to support a conclusion that a major disparity exists. These large disparities in outcomes may reflect a higher prevalence of heart failure in Māori compared to non-Māori, differential access to effective health care interventions or a combination of both.

Māori are known to have a higher prevalence than non-Māori of diseases that commonly cause heart failure, particularly hypertension, coronary artery disease, valvular disease and diabetes. However, a study of Māori and European who died after myocardial infarction found no evidence of a higher prevalence of cardiomyopathy in Māori.

The aetiological profile of heart failure in Māori is unknown. A South Auckland study of admissions with heart failure over one year found that 34% of Māori had known diabetes, rising to 50% in those aged 40-59 years. Ischaemic heart disease was a relatively uncommon cause of heart failure in this Māori population (13% of Māori, 38.6% of Europeans). It is likely that valvular disease contributes to a higher proportion of heart failure in Māori than non-Māori, given the higher prevalence of rheumatic heart disease in Māori.

Māori may have poorer access to early detection and treatment of conditions that precipitate heart failure than non-Māori in New Zealand. After controlling for income and self-reported health status, Māori have a lower primary care consultation rate than non-Māori (Scott K, Marwick J,

| Table 2. Mortality from heart failure, 1988-97 age-specific rates per 100 000. |
|-----------------------------|--------------------|---------------------|---------------------|
| Age group | Māori males | Non-Māori Māori: non-Māori* ratio | 95% CI | Māori females | Non-Māori Māori: non-Māori* ratio | 95% CI |
| 45-64 years | 139 | 17 | 8.8 | 7.6, 10.1 | 32 | 6 | 8.6 | 6.8, 10.9 |
| 65+ years | 446 | 133 | 3.5 | 3.1, 4.1 | 361 | 183 | 3.4 | 3.0, 3.9 |

*ratio of age-standardised rates.

| Table 1. Hospital admissions for heart failure, Māori and non-Māori age-specific rates per 100 000, and Māori/non-Māori ratios. |
|-----------------------------|--------------------|---------------------|---------------------|-----------------------------|--------------------|---------------------|---------------------|
| Age group | Year | Māori males | Non-Māori males | Māori:non-Māori* ratio | 95% CI's | Māori females | Non-Māori females | Māori:non-Māori* ratio | 95% CI's |
| Primary diagnosis of heart failure. | | | | | | | | | |
| 45-64 years | 1989 | 1174 | 149 | 7.9 | 6.7, 9.2 | 772 | 77 | 10.0 | 8.1, 12.3 |
| | 1998 | 1427 | 160 | 8.9 | 7.8, 10.2 | 900 | 89 | 10.1 | 8.5, 12.0 |
| 65+ years | 1989 | 3525 | 1146 | 3.1 | 2.6, 3.7 | 2977 | 754 | 4.0 | 3.3, 4.7 |
| | 1998 | 4401 | 1237 | 3.6 | 3.1, 4.1 | 3638 | 840 | 4.3 | 3.8, 4.9 |
| Secondary diagnosis of heart failure. | | | | | | | | | |
| 45-64 years | 1989 | 295 | 99 | 3.0 | 2.2, 4.0 | 277 | 38 | 7.2 | 5.2, 10.0 |
| | 1998 | 694 | 147 | 4.7 | 4.0, 5.6 | 440 | 69 | 6.4 | 5.1, 8.0 |
| 65+ years | 1989 | 775 | 557 | 1.4 | 1.0, 2.0 | 676 | 379 | 1.8 | 1.3, 2.5 |
| | 1998 | 1608 | 927 | 1.7 | 1.4, 2.1 | 1496 | 628 | 2.4 | 2.0, 2.9 |

*ratio of age-standardised rates.
Crampton P. Unpublished observations. The 1997 national nutrition survey found that only one-third of Māori with hypertension were on medication compared to almost half of non-Māori. In those receiving treatment, the medication did not produce normal blood pressure on the day of the survey for three in four Māori men, and just under half of the medicated Māori women compared to 25% of non-Māori men and women. Access to coronary revascularisation and other relevant surgical procedures that can prevent or delay the onset of heart failure is poor for Māori, relative to need and to non-Māori.

Barriers to early detection and treatment of left ventricular dysfunction are likely to be greater for Māori than non-Māori. Restricted access to echocardiography in New Zealand will impact differentially on Māori, given that they are less likely than non-Māori to be referred to specialists and less likely to have health insurance (allowing access to private sector echocardiography and cardiology). Inequities in funding between primary care organisations with high proportions of Māori patients and those with proportionally few Māori patients and incentives under pharmaceutical budget-holding arrangements may have exacerbated under-treatment of Māori. In patients with established heart failure, a comprehensive approach that includes partnership with patients has been shown to decrease admission rates while improving quality of life.

High hospitalisation rates for Māori reflect a failure by health professionals to implement this evidence.

Previous responses to disparities by ethnicity have sought answers solely in terms of biological parameters and risk factors. Our evidence suggests that Māori are disproportionately affected by the failure of health services to implement interventions of known efficacy. The high prevalence of conditions that can precipitate heart failure such as hypertension, diabetes and rheumatic heart disease in Māori is, in itself, a reflection of poorer access to effective health service delivery. This is compounded by relatively poor access to early detection and optimal treatment of heart failure for Māori, leading to substantially poorer outcomes.

Populations most at risk need to be prioritised for ‘gold standard’ interventions. A broad approach is required, including interventions at social policy level to address determinants of ill health in Māori; strategies to support early detection and treatment of predisposing conditions; assertive, evidence-based treatment by Māori-focused services for individuals with heart failure; and an active attempt to audit and address differential access to effective health care. There is a long history of resistance to discussion about the role of racism in differential access to health care in New Zealand. However, this research suggests that some people get more access to effective intervention than others.

To make progress, we have to actively question how health services (and health professionals as an integral part of these) contribute to disparities; firstly to their existence and secondly to their elimination.

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Metaiodobenzylguanidine (MIBG) scintigraphy has become an important tool in the assessment of phaeochromocytoma, both in location of tumours in patients with negative CT/MRI scans, and in the detection of metastatic disease. Although the sensitivity of MIBG scans is not high, the specificity has been reported as being up to 95-98%. Boersma et al recently published a case report describing increased uptake in the contralateral adrenal region on 123I-MIBG scintigraphy following resection of an adrenal phaeochromocytoma. Their patient, with no evidence of ongoing catecholamine secretion, had increased uptake of 123I-MIBG in the contralateral adrenal gland three months following unilateral adrenalectomy. Twelve months postoperatively the uptake in the contralateral adrenal gland was back to normal. The increased uptake at three months was postulated to be secondary to compensatory hyperplasia of previously less active adrenal medullary tissue. We describe a patient with a malignant phaeochromocytoma and ongoing catecholamine secretion in whom a false positive postoperative 131I-MIBG scan led to removal of a normal adrenal gland. This case raises further questions about the role of MIBG scanning in the early postoperative period in assessing the presence of recurrent phaeochromocytoma.

Case Report

A nineteen year old man presented with hypertension, headache, sweating and palpitations. Measurements of 24 hour urinary catecholamines strongly suggested a phaeochromocytoma with elevation in excretion of noradrenaline, adrenaline and dopamine. A CT scan revealed a 3.5cm low density mass in the right adrenal gland with no evidence of ongoing excessive catecholamine secretion. A laparotomy was undertaken and a metastatic phaeochromocytoma was excised from the right peritoneum. This was confirmed on histology. The patient remains hypertensive and we are considering further treatment with 131I metaiodobenzylguanidine, pending further catecholamine estimations.

This case raises several important issues. It provides histological confirmation that compensatory adrenal medullary hyperplasia occurs in the contralateral adrenal gland in humans as previously postulated. This information is supported by an animal model as compensatory adrenal medullary hyperplasia has been reported in rats after unilateral adrenalectomy. It also raises important clinical concerns regarding the role of MIBG scanning in the postoperative period following unilateral adrenalectomy. In this case a normal adrenal gland was removed from a patient with a malignant phaeochromocytoma because of a false positive 131I-MIBG scan. We advise caution in interpreting unilateral positive MIBG scans in the first few months after contralateral adrenalectomy, even in patients with clear evidence of ongoing excessive catecholamine secretion.

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Chronic infection with hepatitis B virus (HBV) is endemic in New Zealand, particularly in Maori, Pacific and Asian populations. Globally some 800 000 deaths annually are secondary to chronic infection with HBV, mostly from cirrhosis and primary liver cancer, which may affect 40–50% of male carriers. While risks to carriers may be less than in South East Asia, liver cancer alone is estimated to occur in around 10–15% of male carriers in New Zealand, and the human and economic burden of liver transplants from HBV related liver disease is considerable. Around 35% of New Zealand liver transplants are secondary to hepatitis B compared to 5% in USA and 3% in UK (E Game, personal communication). Despite recent advances in treatment the morbidity of hepatitis B infection is significant and prevention of chronic infection is a health priority.

Infants of mothers who are carriers of HBV are at particularly high risk of becoming chronic carriers from infection during labour. Without intervention, mothers who have a high load of HBV (HBeAg positive) transmit the chronic carrier state to their offspring in around 85–95% of cases. Infection at birth also predisposes to a more prolonged period of high infectivity and to a markedly increased risk of later liver cancer. Fortunately it is possible to block the effects of infection at birth by the administration of hepatitis B immune globulin (HBIg) within the first few hours following delivery, and when combined with a course of hepatitis B vaccine commencing in the first days of life, this prevents the carrier state in 91–94% of cases. Some failures do occur, probably because infection has already been transmitted in-utero by a transplacental bleed. Once immunity has been developed there is no need for later boosters as protection against chronic infection is sustained for many years even if antibody is lost. Poorer countries unable to afford HBIg rely on vaccine alone to prevent chronic infection and most reports estimate this to be effective in around 70–85% of infants of HBeAg positive mothers. Lamivudine prophylaxis to reduce the concentration of virus in maternal blood is also being trialled as an alternative to HBIg. When HBIg is given it must be close to birth, ie within a few hours, to be effective.

New Zealand was one of the first countries to adopt vaccination against hepatitis B for high-risk infants from 1985 and for all in 1988. There are now more than 100 countries that have a policy of universal vaccination of infants, which has been remarkably effective in the reduction of HBV carriers and is already showing an effect in preventing mortality from liver cancer. Regrettably, the effective delivery of prophylaxis to the high-risk group of children born to HBV carrier mothers is deficient. A report from the Auckland public health team on babies born at Middlemore hospital found that 4% of the mothers were carriers, but of the 98 identified only 83 had received both HBIg and vaccine in the perinatal period. This failure is likely to be compounded by poor vaccine uptake in infancy, reported as 45% for Maori and 53% for Pacific children in the North Health survey. Indeed the South Auckland workers were able to identify a general practitioner in only 75% of instances, and to trace only half the families. Carrier rates are relatively low in Europeans but much higher in many other ethnic groups including Maori, Pacific peoples, and South-East Asians. Maori and Pacific births alone total almost 20 000 annually. Any failure to protect infants of HBeAg mothers will lead to infants becoming high-risk carriers, at least 25% of whom could be expected to require costly treatment and significant morbidity in later life.

In 1997 a South Island working group looked at the outcome for babies born to hepatitis B carrier mothers in the Canterbury area. It found that many infants had not received the correct vaccinations either at birth or in the subsequent months. Many providers were unaware of the protocol for these infants or of the implications of perinatal infection. Poor flow of information between the lead maternity carer and the well child provider meant that babies were not being actively followed up for further vaccinations, and in many cases the primary health care services were unaware of the ‘high risk’ status of the infant. The opportunity to offer testing and free vaccination to members of the family and sexual partners was missed, and the recommended surveillance of the mother’s liver function was not initiated. These omissions were all health provider errors.

A working group was formed to develop a strategy for improving the outcome for these infants. Two hepatitis B information packs were developed, one for the mother and one for the lead maternity care provider. Laboratories throughout the South Island agreed to send out these packs when they had obtained an HBsAg positive test in an antenatal screen, ensuring that the information and resources needed for the mother and baby were brought to the attention of the provider. The information pack for the lead maternity care provider also contained a notification form to the local Medical Officer of Health to be signed by the mother allowing for follow-up of the infant for vaccination and a blood test following completion of the course. An immunisation coordinator working on behalf of the Medical Officer of Health then ensured delivery of these services. After commencement of this program in Canterbury, notifications of hepatitis B positive antenatal cases rose from an average of thirteen per year to 28 (1998) and 35 (1999) with 39 doses of HBIG given, though this was still short of the laboratory identification of 55 cases.

This South Island initiative underlines the need for some national consistency in approach to ensuring effective prophylaxis to these infants. Providers need to be aware of their responsibilities and potential liability if recommendations are not followed and infants become carriers of hepatitis B. Education sessions for key providers have been undertaken by a national coordinator to improve knowledge and awareness of the importance of these issues.
SPECIAL ARTICLE

“But it’s mine – isn’t it?”

Judge Anand Satyanand, Ombudsman, Wellington.


Judge Anand Satyanand, one of the country’s two Parliamentary Ombudsmen, spoke to the Wellington Medico-Legal Society in August 2001, describing the range of complaint mechanisms and government accountability measures that New Zealand has developed during the past 50 years. This edited paper focuses on matters encountered by the medico-legal community.

“But it’s mine isn’t it?” can be the question of a patient venturing an opinion about access to their file. “But it’s mine isn’t it?” can be the medical practitioner speaking about the same file. “But it’s mine isn’t it?” can also be the hospital manager’s position, or a ministerial official asserting ownership, not only of the file but of the territory.

Changes in fifty years 1951 - 2001

In 1951, New Zealand had a population of 1.9 million with 2463 medical practitioners and 1854 legal practitioners. The Official Secrets Act that year conveyed a sense of the ownership, not only of the file but of the territory.

The South Island initiative is an example for other regions with much higher infection rates. The conclusions from this are both specific and general. To improve the delivery of prophylaxis to this small but high-risk group of children we recommend:

1. Lead maternity carers and hospital maternity services need to recognise the particular needs of babies of carrier mothers and to have effective policies in place to ensure prompt delivery of both HBcAg and vaccine in the neonatal period.

2. High-risk babies should be tracked and followed to ensure that they receive follow-up doses of vaccines in infancy. Follow up should be the responsibility of an identified individual in the public health sector and subject to audit.

3. All babies of carrier mothers should have a blood test for HBsAg and antiHBs between eight and fifteen months of age. This will identify infants who have failed to respond to prophylaxis and give the opportunity to offer further vaccination if both markers are negative.

4. All mothers identified as carriers of hepatitis B in pregnancy should be offered long-term surveillance, and their families and household contacts should be offered testing to detect chronic infection or immunity to hepatitis B. Free vaccine should be offered to susceptible individuals.

In addition to these specific measures we must give greater urgency to developing co-ordinated and audited delivery of well-child health (such as the KIDZNET initiatives). The provision of effective well-child health care to minority groups and mobile families is a major challenge that we must tackle if we are to improve our poor record in child health.

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international ombudsman community of 200 in 100 jurisdictions. A definition of ‘ombudsman’ by the International Bar Association states it is “an office provided for by the constitution or by action of the legislature or parliament and headed by an independent, high-level public official, who is responsible to the legislature or parliament, who receives complaints from aggrieved persons against government agencies, officials and employees, or who acts on [his] own motion, and who has the power to investigate, recommend corrective action, and issue reports.”

New Zealand ombudsmen are officers of parliament. By convention all members of parliament from all parties resolve unanimously on an appointment. Independence is underpinned by funding for the office coming from parliament instead of through government action. At first, ombudsman jurisdiction was limited to investigation of complaints about central government. In 1975 the jurisdiction was extended to local government, education establishments, and many statutory entities.

Professor Philip Joseph pictures the ombudsman’s supervision of the bureaucracy as one exercised by a ‘generalist’ disinclined to intervene in specialist matters involving professional judgement. When a policy is found wanting, an ombudsman may recommend a reconsideration or an alternate approach. Ombudsmen, he notes, have been reluctant to second-guess decisions involving application of a judgement. In other words, ombudsmen have ordinarily restricted themselves to ensuring fair processes.

In \textit{Botany Council v The Ombudsman}, Justice Michael Kirby traversing the difference between judicial and ombudsman functions said “[The] ombudsman lacks the powers to make orders as a court may do. But the sanction of the provision of a report to the responsible minister and to parliament and the requirement upon the minister to respond promptly to any such report also affords significant sanctions. These have proved effective in all jurisdictions in which the office of the ombudsman has been created, to obtain reconsideration of administrative action found by the ombudsman to be unlawful, unreasonable, mistaken or wrong...”. In New Zealand, nearly 40 years after installation of the office, some 6000 people bring cases to the ombudsmen annually.

\section*{Privacy and its development}

Changes to ensure the preservation of individual privacy and its support by the state have been provided by several statutes. These are necessities in a modern democratic state and involve two principles. First the separation of powers, whereby parliament, the courts and the government administration are separate and not in ascendency over the other. Secondly, adherence to the Rule of Law whereby a community consents to law governing it in return for power being exerted over it in an orderly and accountable way.

Professor Colin Munro recently addressed the difficulty in defining Privacy. “…the wealth and diversity of literature devoted to the concept could suggest that there are difficulties in formulating it as a legal right. …Public opinion is probably supportive of such a right… However, in legal and practical terms definition of the ambit of privacy is likely to be highly problematic.”

Acknowledgement and protection of privacy has progressively become part of our legal landscape. The United Nations was formed after World War II with establishment of a charter in favour of peace and justice. The United Nations’ Universal Declaration of Human Rights Article 12 stated: “No-one shall be subjected to arbitrary interference with his privacy, family, home or correspondence nor to attacks upon his honour and reputation. Everyone has the right to the protection of the law against such interference or attacks.” This did not oblige member states to enact domestic legislation to uphold it. However, in 1966 attention was focused on human rights not specifically spelled out in the Universal Declaration in two International Covenants: that on Economic Social and Cultural Rights (ICESCR) and that on Civil and Political Rights (ICCPR). These created an obligation for countries to legislate, in particular Article 17 of the ICCPR stated: “No-one shall be subjected to arbitrary or unlawful interference with his privacy, family, home or correspondence, nor to unlawful attacks on his honour and reputation”.

There has also followed an avalanche of development in the electronic and surveillance worlds calling for rules covering data collection, use and transfer, surveillance, entry and search of premises. In New Zealand we have increasingly recognised the issue of privacy through the privacy commissioner and the ombudsmen. The 1993 Privacy Act is pivotal and exists “to promote and protect individual privacy and in particular to establish principles on the collection, use and disclosure of information relating to individuals and on access by individuals to information held about them. The Act covers both the public and private sectors.” The Act delivered compliance with international norms concerning the collection, security, use and disclosure of personal information.

Ombudsmen connect with privacy in two ways. First, in upholding the protection of personal privacy when a request for release of official information is made. Ombudsmen are required to confer with the privacy commissioner when release of information might endanger privacy. Secondly, when a government organisation is less mindful than it ought to be, of a person's privacy. A citizen can complain to the ombudsman if he feels that there has been an act of maladministration which requires redress.

\section*{Freedom of information}

New Zealand is one of many countries where there is freedom of access to information enabling people to play an informed role in the democracy. Information is to be made available unless some withholding ground is able to be invoked. Ombudsmen determine whether the withholding tests have been met. Prior to the Official Information Act, the Official Secrets Act 1951 was modelled on United Kingdom legislation under which it was said, notionally, that governmental information was “the property of the Queen and her advisers”.

In 1980, a committee of senior civil servants produced a well-regarded report favouring a more open approach which concluded: “The case for more openness in government is compelling. It rests on the democratic principles of ensuring participation in public affairs and ensuring the accountability of those in office; it also derives from concern for the interest of individuals. A no less important consideration is that the Government requires public understanding and support to get its policies carried out. This can come only from an informed public.” Legislation followed quickly and provides for access to official and personal information held by ministers of the crown and government organisations. If a particular request for information is declined, the requester has the right to ask an ombudsman to investigate and review the decision.

The Official Information Act sets out withholding provisions, some of which are conclusive - for example, prejudice to the security or defence of New Zealand, or to its economy, or to maintenance of the law. Other withholding grounds are defeasible on the grounds of public interest and
include protection of privacy, trade secrets, or protection of legal professional privilege. An ombudsman sifts the information and undertakes a balancing test, expressing what the public interest may be in the particular situation and makes a recommendation. Those recommendations are then considered and acted upon by the withholding organisation. There is a potential right of veto of an ombudsman's recommendation by cabinet. There has been no occasion of use of that veto to date, which gives a measure of the efficacy of the ombudsman process. If the recommendation is for release of the information at issue, and no veto has occurred, this may impose a public duty upon the organisation concerned in that regard.

The public sector

The public sector serving the community, with which both the ombudsmen and the privacy commissioner deal, has itself been the subject of change. In the last fifteen years, the core public service has been reduced in numbers to about 35 000 people from a figure nearly three times that many in the mid 1980s. Key governmental powers tend today to be retained centrally with only limited devolution. There is an emphasis upon use of incentives to enhance performance by both departments and individuals. Much use of short-term contracts and performance-based remuneration occurs.

The health and disability commissioner

Patient rights have become a familiar catch-cry in many countries in recent years. The situation has been described by the health and disability commissioner - “In New Zealand in 1987-88 the Cervical Cancer Inquiry marked a watershed in public attitudes to the medical profession. The media spotlight on the events uncovered at National Women's Hospital and [the] call for statutory recognition of patients' rights turned the tide against prevailing currents of paternalism and beneficence. The Cartwright Report led to the appointment of patient advocates at some hospitals... ethics committees, and the enactment in 1994 of the Health and Disability Commissioner Act 'to promote and protect the rights of health consumers and disability services consumers, and to that end to facilitate the fair, simple, speedy and efficient resolution of complaints relating to infringement of those rights'.“

Despite all the change and improvements referred to, a number of problems call for ongoing consideration and review. A pressing example is the unevenness in the way that information can be collated and used without the knowledge and consent of the medical patient. Collation, sharing and use of information in the public sector, inclusive of medical information will need attention. Misapprehension by officials of the duty to deal with official information, and misuse of privacy legislation to avoid undertaking responsibility for the basis of release of official information, are additional difficulties.

Conclusion

What kind of prospect does this changed world offer for the contemporary participant in medico-legal issues? For lawyers, some sense of what may be needed, emerges from a sign at the Stanford University Law School: “Lawyers are the most active participants in the process of working out accommodations and solutions to human problems; for the first class lawyer is an unusually productive mix of technician, analyst, gladiator, counsellor, tactician, constitutional architect, politician and scholar...”

For medical practitioners, an element of what may be needed is offered by Justice Michael Kirby in his address to the World Congress in Dermatology (June 1997): “It is that special dependence – going to the essence of human existence or well-being – that elevates the health care professions to a particular nobility. In the age of gene therapy, of computer aided imaging, of countless pathology tests, there still stands beside the bed ... the concerned human being with the will to provide relief. No computer and no technology that we have yet devised or can yet imagine can exhibit that human quality.”

For perhaps both professions, Kirby concluded: “In the age of cutbacks, of economic rationalisation and of technology, we should be constantly reminded and constantly remind ourselves – of the elements that set the professions apart. They include the skill and knowledge that come from training. But more importantly they require an ultimately selfless caring spirit, an insatiable curiosity, a concern for fellow human beings and a capacity to communicate with them.”

For the members of the public whom we serve, the community's emphasis on rights and the growth in its sense of consumerism will ensure that they will continue to ask: “But it's mine isn't it?”

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1. New Zealand Yearbook (1951)
5. Munro C. Public Law 2001; Spring Ed 1.

Doctors object to companies offering to store cord blood

Doctors in Britain have criticised companies for persuading expectant parents to part with up to £700 ($980) to have blood collected from their newborn baby's umbilical cord to protect against future illnesses when little evidence exists to support the practice.

“Stem cells collected from umbilical cords have been successfully used to treat some illnesses such as leukaemia, but it is speculation to suggest that they may be used in future years to cure a wider range of illnesses such as Parkinson's disease, diabetes, and heart disease,” he said.

Even in childhood leukaemia, where stem cells have been frequently used in the past, there is a move away from using cord blood transplants because other types of treatment are just as effective, he added.

Patient requests for unwarranted treatment

Jonathan Coates, Senior Solicitor, Buddle Findlay, Wellington.

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Practitioners are likely to be familiar with the situation where a patient requests that he be provided with a certain type of treatment or medicine which the practitioner does not think is appropriate. This scenario is likely to become increasingly common with television advertising of prescription medicines and increased access to medical information, most notably via the internet where it is impossible to guarantee integrity of the information. When confronted with such a request, should the practitioner refuse to treat or prescribe medicines as requested, or does a patient have the right to decide what treatment is best?

The short answer to this dilemma is that a medical practitioner cannot be required to adopt a course of treatment which, in his clinical judgment is contra-indicated as not being in the best interests of the patient. A patient cannot demand a particular treatment and expect to receive it automatically.

However, there are a couple of additional matters to be aware of. The practitioner's refusal to comply with the request must be reasonable. Whilst the decision as to what is in the patient's best interests will be a decision for the practitioner, then the request must be reasonable. Whilst acceptable, is not the practitioner's preferred method. From the practitioner's point of view, it is essential to ensure that the treatment, whilst not the preferred option, is still clinically appropriate. What about the situation where the demand for treatment is made by parents of a child? Suppose a child is terminally ill, and the clinical view is that the child should receive only palliative care, but the parents demand more aggressive or invasive treatment? This is the reverse situation of the highly publicised Liam Williams-Holloway case where the parents refused treatment that medical staff believed was indicated. Once again, the practitioner cannot be required to provide treatment that is not in the child's best interests. Assuming the practitioner's opinion is reasonable, the parent's request should not be followed. In refusing to treat the child, the practitioner will not be in breach of his duty to the child, nor will the failure to treat amount to the crime of failing to provide the necessary care. In such a case, however, the practitioner would be wise to discuss the case with colleagues before making a decision.

With the increase in awareness of medical issues, and increasing access to information on the internet, patient requests for treatment are likely to become more common. Practitioners will need to consider the request that is made, and assess whether there is any clinical justification in acceding to the request. If not, the demand should not be met.

Reforming complaints systems: UK and New Zealand

The long-awaited report into the UK’s National Health Service complaints system published early this month describes high levels of dissatisfaction among users. An inquiry into New Zealand’s (NZ) procedures, the Cull report, described a similar picture. Despite this similarity, two different policy responses emerged.

Both reports follow high-profile public scandals about errant doctors. The reports come in a climate of increasing public concern and concomitant demands for more effective and transparent systems of scrutiny and assurance. The attempts from both countries to address these expectations take different forms. The UK report follows a trend towards insular but detailed consideration of one aspect of the overall system of public protection. By contrast, the NZ report looks at complaints procedures as part of an examination of the bigger picture.

The regulatory landscapes in the UK and NZ are populated with multiple agencies set up to act on concerns about standards of care. The Cull report notes: “Currently, fourteen organisations potentially can each undertake an investigation into the same adverse medical event, contemporaneously or cumulatively without reference to the other”. The regulatory landscape in the UK looks similar with new organisations such as the National Clinical Assessment Authority, to address concerns about doctors’ performance; the Commission for Health Improvement, to address issues of organisations’ competence; and the National Patient Safety Agency, to act as a watchdog for adverse events.

In view of this crowded landscape the Cull report was given a radical remit, which is reflected in the proposal for one investigative process for all health-service-related complaints. This system would incorporate a “one-stop shop” for complaints, adverse events, and concerns about health professionals. One body would be the repository of complaints with responsibility for assessing cases, onward referral, and coordinating information-sharing, as well as making recommendations for improvement and potential compensatory awards. The model also promotes one disciplinary tribunal for judging charges against any health professional.