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THE NEW ZEALAND MEDICAL JOURNAL

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EDITORIALS

Medical professionalism – a Charter for all doctors

In February this year, The Lancet[1] and the Annals of Internal Medicine[2] published simultaneously an article that has the potential to restore the relationship between patients and doctors and to renew a sense of medical professionalism. A starting point is recognition that "…medicine's commitment to the patient is being challenged by external forces of change within our societies." This view, from the European Federation of Internal Medicine, the American College of Physicians, the American Society of Internal Medicine and the American Board of Internal Medicine, reflects concerns expressed repeatedly in New Zealand since the ill-advised health reforms starting in the late 1980s. An editorial article in this Journal in mid-2001 addressed the same issue.[3] We now have a chance to put things right.

The Lancet/Annals paper is a potential landmark and a way forward if we are prepared to be bold. The Charter, by physicians, is relevant to all doctors. It "supports physicians' efforts to ensure that the health-care systems and the physicians working within them remain committed both to patients' welfare and to the basic tenets of social justice".[1,2] The authors note the "increasing disparities among the legitimate needs of patients, the available resources to meet those needs, the increasing dependence on market forces to transform health-care systems, and the temptation for physicians to forsake their traditional commitment to the primacy of patients' interests."

Three fundamental principles and are listed: the primacy of patients' welfare, the principle of patients' autonomy and the principle of social justice. Then follows a set of professional commitments to:

- professional competence
- honesty with patients
- patients'confidentiality
- maintaining appropriate relationships with patients
- improving quality of care
- improving access to care
- a just distribution of finite resources
- scientific knowledge
- maintaining trust by managing conflicts of interest
- professional responsibilities.[1,2]

The relevance of this Charter to New Zealand and its potential importance for the future of medical care in this country is so clear that we publish the paper (the introduction section is abbreviated) in this issue of the Journal along with a commentary by Richard Horton, Editor of The Lancet, who challenges us all: "Doctors can no longer be silent about their work, leaving advocacy to a small group of medical politicians."

The Medical Council of New Zealand published ethical guidelines for doctors' duties in an environment of competition or resource limitation[5] and guidelines on responsibilties of doctors in management and governance.[6] The Charter incorporates many aspects of the Medical Council guidelines and the Revised Code of Ethics published recently by the New Zealand Medical Association. Action is needed now if the continuing erosion to our professionalism is to be halted and reversed. What should we do?

In our view, the specialty medical colleges in New Zealand together with the New Zealand Medical Association and the Medical Council need to consider the Charter as a matter of urgency. A Charter similar to this could be adopted and implemented in New Zealand. In their dealings with the Minister of Health, Treasury and the Ministry of Health, they would need to ensure that all planning proposals were consistent with the Charter. A watchdog role by the Medical Council (and perhaps the specialty colleges) may be needed.

Had such a Charter been a guiding force in New Zealand over the last fifteen years, we could have avoided a long list of unnecessary, sad episodes that stain the history of medicine in this country.

The Charter gives a lead. Are we prepared to act?

The Editors

1. Medical Professionalism Project. Medical professionalism in the new millenium: a physicians' charter. Lancet 2002; 359: 520-2.
2. Medical Professionalism Project. Medical professionalism in the new millenium: a physicians'charter. Ann Intern Med 2002; 136: 243-6.
3. Bagshaw P, Begg E, Moller P et al. Reaffirming professionalism in medicine. NZ Med J 2001; 114: 347-8.
4. Horton R. The doctor's role in advocacy. Lancet 2002; 359: 458.
5. Medical Council of New Zealand. Ethical guidelines for doctors' duties in an environment of competition or resource limitation. Wellington: Medical Council of New Zealand; 1999.
6. Medical Council of New Zealand. Guidelines on responsibilities of doctors in management and governance. NZ Med J 2000; 113: 386-7.

The doctor's role in advocacy

Richard Horton, Editor, The Lancet

In his Rock Carling lecture last year, entitled "Fads in medical care policy and politics: the rhetoric and reality of managerialism", Theodore Marmor spoke of the "linguistic muddle" and "conceptual confusion" that marked the organisation of medicine during the past decade.[1] Business ideology infiltrated health care when costs spiralled and governments reconsidered their long- standing commitment to the welfare state. As professors were deposed by chief executive officers, and as the fog of human-resources jargon obscured an emptiness of serious thought, so mediocrity became the benchmark for running a health service. Priorities shifted. Quality was eroded by a concern for quantity, effectiveness gave way to efficiency, and notions of professionalism were subsumed by mission statements. Morale collapsed, cynicism became commonplace.

But medicine is governed by an ethos, not a balance sheet, and doctors are now reasserting the values that politicians, managers, accountants, and lawyers have either dismissed or manipulated, according to their own partisan interests. The results of a European-American project to produce a charter of fundamental principles and professional responsibilities for physicians follows. This unusual collaboration between the European Federation of Internal Medicine, American College of Physicians, American Society of Internal Medicine, and American Board of Internal Medicine sets clear standards for practice, which can be used by doctors to defend the interests of patients at the bedside and in the community.

Why does this charter matter? Doctors can no longer remain silent about their work, leaving advocacy to a small group of medical politicians. Doctors cannot assume that they have either the trust of the public or the support of governments unless they are willing to take part in the public debate about what kind of society they want for the sick and impoverished. More doctors, irrespective of their specialty, responsibility, or seniority, need to enter the public arena of dispute. This idea is embodied in the charter's principle of social justice, together with its parallel commitments to improving quality of care, enhancing access to health services, redistributing resources to those in most need, and strengthening the research base of medicine.

Some doctors will argue that advocacy should be left to the few rather than be encouraged among the many. A doctor's task, they might say, is to care, not to coerce. But advocacy can mean as little as writing a letter to a newspaper, posting a comment on a website, or asking a question at a meeting. Advocacy only means taking the problems that one faces day to day and pursuing their resolution outside their usual place of presentation. If every doctor who reads the physicians' charter wrote a letter, posted a comment, or asked a question, the voice of medicine would be less easily dismissed or manipulated.

Reprinted from Lancet 2002; 359: 458, with permission.

1. Marmor T. Fads in medical care policy and politics: the rhetoric and reality of managerialism. Rock Carling Fellowship, 2001. Norwich: The Stationery Office, on behalf of Nuffield Trust, 2001.

CHARTER ON MEDICAL PROFESSIONALISM

Medical professionalism in the new millennium: a physicians' charter

Medical Professionalism Project

NZ Med J 2002; 115: 203-4

Physicians today are experiencing frustration as changes in the health-care delivery systems in virtually all industrialised countries threaten the very nature and values of medical professionalism. Recently, voices from many countries have begun calling for a renewed sense of professionalism, one that is activist in reforming health-care systems. Responding to this challenge, the European Federation of Internal Medicine, the ACP-ASIM Foundation, and the ABIM Foundation combined efforts to launch the Medical Professionalism Project (www.professionalism.org) in late 1999. These three organisations designated members to develop a "charter" to encompass a set of principles to which all medical professionals can and should aspire. The charter supports physicians' efforts to ensure that the health-care systems and the physicians working within them remain committed both to patients' welfare and to the basic tenets of social justice. Moreover, the charter is intended to be applicable to different cultures and political systems.

Preamble

Professionalism is the basis of medicine's contract with society. It demands placing the interests of patients above those of the physician, setting and maintaining standards of competence and integrity, and providing expert advice to society on matters of health. The principles and responsibilities of medical professionalism must be clearly understood by both the profession and society. Essential to this contract is public trust in physicians, which depends on the integrity of both individual physicians and the whole profession.

At present, the medical profession is confronted by an explosion of technology, changing market forces, problems in health-care delivery, bioterrorism, and globalisation. As a result, physicians find it increasingly difficult to meet their responsibilities to patients and society. In these circumstances, reaffirming the fundamental and universal principles and values of medical professionalism, which remain ideals to be pursued by all physicians, becomes all the more important.

The medical profession everywhere is embedded in diverse cultures and national traditions, but its members share the role of healer, which has roots extending back to Hippocrates. Indeed, the medical profession must contend with complicated political, legal, and market forces. Moreover, there are wide variations in medical delivery and practice through which any general principles may be expressed in both complex and subtle ways. Despite these differences, common themes emerge and form the basis of this charter in the form of three fundamental principles and as a set of definitive professional responsibilities.

Fundamental principles

Principle of primacy of patients' welfare. This principle is based on a dedication to serving the interest of the patient. Altruism contributes to the trust that is central to the physician-patient relationship. Market forces, societal pressures, and administrative exigencies must not compromise this principle.

Principle of patients' autonomy. Physicians must have respect for patients' autonomy. Physicians must be honest with their patients and empower them to make informed decisions about their treatment. Patients' decisions about their care must be paramount, as long as those decisions are in keeping with ethical practice and do not lead to demands for inappropriate care.

Principle of social justice. The medical profession must promote justice in the health-care system, including the fair distribution of health-care resources. Physicians should work actively to eliminate discrimination in health care, whether based on race, gender, socioeconomic status, ethnicity, religion, or any other social category.

A set of professional responsibilities

Commitment to professional competence. Physicians must be committed to lifelong learning and be responsible for maintaining the medical knowledge and clinical and team skills necessary for the provision of quality care. More broadly, the profession as a whole must strive to see that all of its members are competent and must ensure that appropriate mechanisms are available for physicians to accomplish this goal.

Commitment to honesty with patients. Physicians must ensure that patients are completely and honestly informed before the patient has consented to treatment and after treatment has occurred. This expectation does not mean that patients should be involved in every minute decision about medical care; rather, they must be empowered to decide on the course of therapy. Physicians should also acknowledge that, in health care, medical errors that injure patients do sometimes occur. Whenever patients are injured as a consequence of medical care, patients should be informed promptly because failure to do so seriously compromises patients' and societal trust. Reporting and analysing medical mistakes provides the basis for appropriate prevention and improvement strategies and for appropriate compensation to injured parties.

Commitment to patients' confidentiality. Earning the trust and confidence of patients requires that appropriate confidentiality safeguards be applied to disclosure of patients' information. This commitment extends to discussions with people acting on a patient's behalf when obtaining the patient's own consent is not feasible. Fulfilling the commitment to confidentiality is more pressing now than ever before, given the widespread use of electronic information systems for compiling data on patients and an increasing availability of genetic information. Physicians recognise, however, that their commitment to confidentiality must occasionally yield to over-riding considerations in the public interest (for example, when patients endanger others).

Commitment to maintaining appropriate relationships with patients. Given the inherent vulnerability and dependency of patients, certain relationships between physicians and patients must be avoided. In particular, physicians should never exploit patients for any sexual advantage, personal financial gain, or other private purpose.

Commitment to improving quality of care. Physicians must be dedicated to continuous improvement in the quality of health care. This commitment entails not only maintaining clinical competence but also working collaboratively with other professionals to reduce medical error, increase patients' safety, minimise overuse of health-care resources, and optimise the outcomes of care. Physicians must actively participate in the development of better measures of quality of care and the application of quality measures to assess routinely the performance of all individuals, institutions, and systems responsible for health-care delivery. Physicians, both individually and through their professional associations, must take responsibility for assisting in the creation and implementation of mechanisms designed to encourage continuous improvement in the quality of care.

Commitment to improving access to care. Medical professionalism demands that the objective of all health-care systems be the availability of a uniform and adequate standard of care. Physicians must individually and collectively strive to reduce barriers to equitable health care. Within each system, the physicians should work to eliminate barriers to access based on education, laws, finances, geography, and social discrimination. A commitment to equality entails the promotion of public health and preventive medicine, as well as public advocacy on the part of each physicians, without concern for the self-interest of the physician or the profession.

Commitment to just distribution of finite resources. While meeting the needs of individual patients, physicians are required to provide health care that is based on the wise and cost-effective management of limited clinical resources. They should be committed to working with other physicians, hospitals, and payers to develop guidelines for cost-effective care. The physician's professional responsibility for appropriate allocation of resources requires scrupulous avoidance of superfluous tests and procedures. The provision of unnecessary services not only exposes patients to avoidable harm and expense but also diminishes the resources available for others.

Commitment to scientific knowledge. Much of medicine's contract with society is based on the integrity and appropriate use of scientific knowledge and technology. Physicians have a duty to uphold scientific standards, to promote research, and to create new knowledge and ensure its appropriate use. The profession is responsible for the integrity of this knowledge, which is based on scientific evidence and physicians' experience.

Commitment to maintaining trust by managing conflicts of interest. Medical professionals and their organisations have many opportunities to compromise their professional responsibilities by pursuing private gain or personal advantage. Such compromises are especially threatening in the pursuit of personal or organisational interactions with for-profit industries, including medical equipment manufacturers, insurance companies, and pharmaceutical firms. Physicians have an obligation to recognise, disclose to the general public, and deal with conflicts of interest that arise in the course of their professional duties and activities. Relationships between industry and opinion leaders should be disclosed, especially when the latter determine the criteria for conducting and reporting clinical trials, writing editorials or therapeutic guidelines, or serving as editors of scientific journals.

Commitment to professional responsibilities. As members of a profession, physicians are expected to work collaboratively to maximise patients' care, be respectful of one another, and participate in the process of self-regulation, including remediation and discipline of members who have failed to meet professional standards. The profession should also define and organise the educational and standard-setting process for current and future members. Physicians have both individual and collective obligations to participate in these processes. These obligations include engaging in internal assessment and accepting external scrutiny of all aspects of their professional performance.

Summary

The practice of medicine in the modern era is beset with unprecedented challenges in virtually all cultures and societies. These challenges centre on increasing disparities among the legitimate needs of patients, the available resources to meet those needs, the increasing dependence on market forces to transform health-care systems, and the temptation for physicians to forsake their traditional commitment to the primacy of patients' interests. To maintain the fidelity of medicine's social contract during this turbulent time, we believe that physicians must reaffirm their active dedication to the principles of professionalism, which entails not only their personal commitment to the welfare of their patients but also collective efforts to improve the health-care system for the welfare of society. This Charter on Medical Professionalism is intended to encourage such dedication and to promote an action agenda for the profession of medicine that is universal in scope and purpose.

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Detection of *APC* Mutations in Faecal DNA

An early genetic change in the pathway to colorectal cancer is a mutation in the adenomatous polyposis coli (*APC*) gene. On the basis of the premise that cells with mutant *APC* genes are shed into the faeces, these investigators devised a powerful molecular method to find such genes in faeces from patients with colorectal cancer. Whereas faeces from normal subjects had no detectable mutant *APC* genes, stools from over half the patients with colorectal cancer or colonic polyps contained such genes.

This study tested the feasibility of finding mutant APC genes in stools from patients with early colorectal cancer. The method developed for this purpose is a technical tour de force, akin to finding the proverbial needle in a haystack. Refinements of this method may lead to a specific and sensitive screening test for colorectal cancer.

N Engl J Med 2002; 346: 311-20.

ORIGINAL ARTICLES

Comparison of cancer mortality and incidence in New Zealand and Australia

David CG Skegg, ***Professor;*** **Margaret RE McCredie,** ***Professorial Research Fellow, Department of Preventive and Social Medicine, University of Otago, Dunedin.***

Abstract

Aims. To compare cancer mortality and incidence data from New Zealand and Australia, in order to gauge the potential for reducing deaths from cancer in New Zealand.
Methods. For 1996 and 1997, numbers of deaths from cancer, numbers of new cases, and population data were stratified in 5-year age-groups. Numbers observed in New Zealand were compared with numbers expected from Australian rates. Age-standardized mortality and incidence rates for each sex were analysed.
Results. New Zealanders of both sexes experienced more deaths from cancer than expected in every age group. If Australian rates had applied, there would have been 215 fewer cancer deaths per year in New Zealand males, and 616 fewer in females. The largest differences related to breast cancer and lung cancer in women, and colorectal cancer in both sexes. The overall incidence of cancer was higher in New Zealand, but mortality/incidence ratios were also higher for many sites – suggesting that survival after treatment has been poorer in New Zealand than in Australia.
Conclusions. Considerable scope exists for reducing cancer mortality in New Zealand. For a national cancer control strategy, it will be essential to clarify reasons for the high incidence of cancer and to study survival following treatment.

NZ Med J 2002; 115: 205-8

New Zealand women are reported to have the sixth highest cancer mortality rate among 175 countries, while New Zealand men are ranked 33rd.[1] Since 1960 cancer mortality in both men and women in New Zealand has increased, while our position has deteriorated in comparison with people living in Australia, Canada, the United Kingdom and the USA.[2] In order to clarify the potential for reducing deaths from cancer in New Zealand, we compared mortality and incidence data with those from Australia – a country with similar lifestyles and close affinities in medical practice.

Methods

Cancer mortality and incidence data, together with population statistics, were extracted from New Zealand and Australian reports for 1996 and 1997.[3-8] These were the most recent years for which all the required information was available; two years were used because the small population of New Zealand makes some rates unstable.

Numbers of deaths from cancer, numbers of new cases, and population data were stratified in 5-year age-groups (up to 85+ years). The numbers of deaths observed in New Zealand were compared with those that would have been expected if the Australian age-specific mortality rates (for each sex) for the same years had prevailed. While the calculations were done in 5-year age-groups, results are presented in broader age-groups. Cancer mortality and incidence rates for the two countries were also directly age-standardized, using the Australian 1991 population as a standard. This standard was chosen because it resembles the age structure of the two countries more closely than Segi's world population (which is often used).

Common sites of cancer were grouped using the International Classification of Diseases (ICD), 9th revision. The ICD categories were those shown in the Australian reports.[7,8] Non-melanoma skin cancers (ICD 173) were excluded from this study, because the incidence of these is not generally registered.

Separate analyses were also conducted for Maori and non-Maori New Zealanders. In the years studied, ethnic data were based on self-identified ethnicity ("mixed ethnicity").[6] Under this system individuals (or their representatives) are expected to select the ethnic groups they consider they belong to; the data are then "prioritised" so that any person who selects Maori as one of their ethnicities is counted in the Maori group. Statistics for non-Maori were obtained by subtracting data for Maori from those published for the total population.

Results

During 1996 and 1997, 7706 males and 7037 females died from cancer in New Zealand. Table 1 shows the observed numbers of deaths and the numbers that would have been expected if Australian cancer mortality rates had applied in each sex and age-group. In the right-hand column of the Table, the differences between observed and expected numbers in the two years have been halved (and rounded to whole numbers) to give the "annual excess" of deaths from cancer in New Zealand. Both sexes experienced more deaths than expected (on the basis of Australian rates) in every age group. If Australian rates had applied, there would have been 215 fewer cancer deaths per year in New Zealand males, and 616 fewer in females. The majority of excess deaths (78% in males and 59%in females) were in people under 70 years of age.

Table 1. Numbers of deaths from cancer in New Zealand in 1996 and 1997 (combined) according to sex and age, compared with numbers expected from Australian mortality rates.

Age-group (years)	Observed	Expected	Annual excess*
Males			
All ages	7706	7275.3	215
0-14	48	33.2	7
15-29	82	54.7	14
30-39	114	91.7	11
40-49	309	296.5	6
50-59	887	788.6	49
60-69	2009	1847.4	81
70-79	2546	2523.7	11
80 or older	1711	1639.5	36
Females			
All ages	7037	5804.1	616
0-14	33	25.2	4
15-29	62	42.9	10
30-39	185	138.3	23
40-49	482	373.3	54
50-59	956	708.5	124
60-69	1459	1158.7	150
70-79	2052	1728.7	162
80 or older	1808	1628.6	90

*(Obs – Exp)/2

Table 2 shows the numbers of deaths from ten leading sites of cancer in each sex, together with the numbers expected from Australian mortality rates. The rank order of sites was similar in the two countries. For nearly every site, there was

a relative excess of deaths in New Zealand. By far the most important contributors were colorectal cancer in males, and three sites – breast, colorectal, and lung – in females. The latter three sites accounted for 377 excess deaths per year in females (61% of the total).

Table 2. Numbers of deaths from specific cancers in New Zealand males and females in 1996 and 1997 (combined), compared with numbers expected from Australian mortality rates.

Cancer site	Observed	Expected	Annual excess*
Males			
All sites	7706	7275.3	215
Lung	1786	1778.9	4
Colorectal	1162	953.6	104
Prostate	1027	967.1	30
Stomach	332	288.8	22
Non-Hodgkin's lymphoma	289	289.6	0
Leukaemia	276	277.8	-1
Brain	232	226.7	3
Melanoma	228	220.6	4
Bladder	221	208.4	6
Kidney	167	176.2	-5
Females			
All sites	7037	5804.1	616
Breast	1301	1014.5	143
Colorectal	1060	830.1	115
Lung	1032	794.3	119
Ovary	345	299.1	23
Pancreas	312	322.0	-5
Non-Hodgkin's lymphoma	287	273.9	7
Leukaemia	211	208.1	1
Melanoma	167	127.4	20
Cervix	155	115.7	20
Uterus†	127	105.7	11

*(Obs – Exp)/2
†Body of uterus, or part unspecified (ie ICD 179 + 182)

To investigate the relative importance of cancer occurrence and survival following treatment, incidence and mortality rates were compared in the two countries (Table 3). Mortality/incidence ratios are also shown. For all sites combined, incidence and mortality rates were both higher in New Zealand males. The mortality/incidence ratios for all sites combined in males were similar in the two countries. The recorded incidence of prostate cancer was very high in New Zealand; if this disease is excluded, the mortality/incidence ratios for males were 0.56 in New Zealand and 0.51 in Australia.

The overall incidence of cancer among females was also higher in New Zealand, but there was a more marked difference in mortality. The mortality/incidence ratios for females were 0.45 in New Zealand, and 0.40 in Australia. The corresponding difference was particularly striking for breast cancer. The incidence rate of this disease was only 1.2% higher, whereas the mortality rate was 28% higher, in New Zealand. The only malignancies for which the mortality/incidence ratios were lower in New Zealand females were cancers of the ovary and pancreas, and leukaemia.

Table 4 shows a similar analysis for Maori and non-Maori New Zealanders. The problems inherent in the assignment of ethnicity in health statistics need to be borne in mind. In comparison with non-Maori, Maori males were reported to have a lower incidence but a higher mortality from all cancers combined. Maori females had both higher incidence and mortality rates than non-Maori. Because of the small size of the Maori population, only a few leading sites are shown in the Table. In each case except for stomach cancer in females, the mortality/incidence ratio was higher for Maori than for non-Maori New Zealanders. Maori men and women had very high rates of lung cancer. Stomach cancer was also much more common among Maori, whereas the incidence of colorectal cancer was higher in non-Maori. For cervical cancer in the years studied, the incidence rate was 2.2 times higher in Maori women – but the mortality rate was 3.9 times higher.

The incidence and mortality rates for Maori and non-Maori can be compared with those shown for Australians in the right-hand part of Table 3. The overall cancer incidence and mortality rates were higher in non-Maori New Zealanders than in Australia. The mortality/incidence ratio was actually slightly lower in non-Maori males. Apart from the influence of prostate cancer mentioned above, this reflects the fact that non-Maori men (but not women) had lower rates of lung cancer than Australians. If prostate cancer and lung cancer are excluded, the mortality/incidence ratios were 0.47 in the non-Maori New Zealand males and 0.44 in Australia.

For comparison with Table 2, the excess numbers of deaths among Maori (compared with expected numbers based on Australian rates) were calculated (data not shown). There were estimated to be 138 excess cancer deaths per year in Maori males, including 74 from lung cancer, and 22 from stomach cancer. There were 160 excess cancer deaths per year in Maori females, including 68 deaths from lung cancer, 28 from breast cancer, and 17 from cervical cancer. From Table 2 it will be seen that, if attention were confined to non-Maori, there would be an annual excess of 77 cancer deaths in New Zealand males and 456 cancer deaths in females.

Discussion

Both males and females in New Zealand have higher cancer mortality rates than their counterparts in Australia. The differences were estimated to account for 215 excess deaths annually in New Zealand males, and 616 in females. These extra deaths result partly from a higher incidence of cancer in New Zealand, and some of the differences in cancer incidence rates may be long-standing. In addition, mortality/incidence ratios suggest that survival after the treatment of some cancers may be poorer in New Zealand than in Australia.

In comparison with Australia and other countries, New Zealand has lost ground in the control of cancer over recent decades.[2] New Zealand women have one of the highest mortality rates from cancer in the world.[1] For comparisons between countries or time periods, mortality rates are most reliable because the recording and coding of deaths tends to be consistent and virtually complete in developed countries. Differences in mortality rates may be due to differences in the incidence of cancer or in the effectiveness of treatment. Incidence rates need to be interpreted more cautiously than mortality rates, because they are affected by variation in the completeness of cancer registration. Both New Zealand and Australia have population-based cancer registries, and it is likely that standards of registration in the two countries have become similar since New Zealand introduced statutory notification of cancer in 1994. A further consideration is that screening practices can have an important effect on the recorded incidence of some malignancies, such as cancers of the breast and prostate.

Regrettably no comprehensive analysis of survival from cancer in New Zealand has been carried out since 1984,[9] although a study is now in progress. Mortality/incidence ratios provide a crude index of survival, but their limitations need to be borne in mind. As with more formal survival analyses, they depend on the completeness of registration and will be affected by any tendency to include in situ cancers with invasive cancers. In addition, it needs to be remembered that people dying from cancer in a particular year usually have been diagnosed in earlier years. Hence

Table 3. Cancer incidence and mortality rates* in New Zealand and Australia, 1996-1997.

Cancer site	New Zealand			Australia		
	Incidence	Mortality	Mortality/ incidence ratio	Incidence	Mortality	Mortality/ incidence ratio
Males						
All sites	506.9	234.0	0.46	485.8	221.3	0.46
Lung	57.2	53.9	0.94	59.9	53.6	0.89
Colorectal	72.4	35.0	0.48	68.8	28.8	0.42
Prostate	146.6	33.2	0.23	114.1	31.2	0.27
Stomach	14.7	10.0	0.68	13.6	8.8	0.65
Non-Hodgkin's lymphoma	17.5	8.7	0.49	19.0	8.7	0.46
Leukaemia	16.1	8.2	0.51	13.3	8.4	0.63
Brain	8.5	6.7	0.78	7.9	6.5	0.83
Melanoma	44.2	6.7	0.15	49.1	6.5	0.13
Bladder	23.5	6.9	0.29	22.5	6.6	0.29
Kidney	12.7	5.0	0.40	13.6	5.3	0.39
Females						
All sites	371.0	165.9	0.45	341.5	135.3	0.40
Breast	97.8	31.4	0.32	96.7	24.5	0.25
Colorectal	55.6	24.5	0.44	46.3	18.8	0.41
Lung	29.3	25.4	0.87	23.3	19.1	0.82
Ovary	13.9	8.4	0.61	11.2	7.2	0.64
Pancreas	7.7	7.2	0.93	7.3	7.2	0.99
Non-Hodgkin's lymphoma	13.5	6.7	0.50	13.4	6.3	0.47
Leukaemia	10.6	4.9	0.46	8.3	4.8	0.57
Melanoma	38.7	4.0	0.10	36.0	3.1	0.08
Cervix	11.3	3.9	0.34	8.7	2.8	0.32
Uterus†	13.8	3.0	0.22	13.2	2.4	0.18

* Rates per 100 000 population, age-standardized to the Australian 1991 population. †Body of uterus, or part unspecified (ie ICD 179 + 182).

Table 4. Cancer incidence and mortality rates* in Maori and non-Maori New Zealanders, 1996-1997.

Cancer site	Maori			Non-Maori		
	Incidence	Mortality	Mortality/ incidence ratio	Incidence	Mortality	Mortality/ incidence ratio
Males						
All sites	450.0	338.6	0.75	509.6	226.7	0.44
Lung	104.0	131.7	1.27	54.1	49.3	0.91
Colorectal	39.2	27.5	0.70	74.4	35.3	0.47
Prostate	106.2	34.3	0.32	148.5	33.0	0.22
Stomach	26.5	27.8	1.05	13.7	8.8	0.64
Females						
All sites	392.5	258.1	0.66	369.1	159.1	0.43
Breast	91.2	41.4	0.45	97.9	30.4	0.31
Colorectal	31.9	27.9	0.87	57.5	24.7	0.43
Lung	85.9	78.0	0.91	25.6	22.0	0.86
Stomach	24.0	16.5	0.69	6.1	4.6	0.76
Cervix	21.9	11.6	0.53	10.0	3.0	0.30

* Rates per 100 000 population, age-standardized to the Australian 1991 population.

mortality/incidence ratios will be confounded by any rapid changes in the incidence of particular cancers.

The chief aim of the present study was to identify priorities for research and public health action. The outstanding issues that emerge are the high mortality from breast cancer and lung cancer in New Zealand women, and the high mortality from colorectal cancer in both women and men.

Whereas the incidence rates of breast cancer are similar in the two countries, New Zealand women have a mortality rate that is 28 per cent higher than in Australia. This implies that the survival of women diagnosed with breast cancer is poorer in New Zealand. Breast cancer mortality has been higher in New Zealand for several decades, and further investigation is required to analyse possible contributing factors. Australia introduced mammographic screening for breast cancer earlier than in New Zealand, but this is unlikely to be the main explanation because the difference in mortality is found in all age-groups – including women younger than 40 years (data not shown). It is possible that New Zealand has lagged behind Australia in implementing advances in the management of breast cancer, such as the use of adjuvant radiotherapy, chemotherapy and endocrine therapy. In Australia many women with this disease are now treated in multidisciplinary settings, especially in metropolitan areas. Women with breast cancer are treated by a very large number of specialists in New Zealand, but there has been no attempt to survey practice in a systematic way. In contrast, population-based surveys of the management of breast cancer have been occurring in Australian states for over a decade; a national study has also been reported.[10]

The higher mortality from lung cancer in New Zealand women reflects a higher incidence than in Australia, especially among Maori (Table 4). The great majority of lung cancer cases are due to cigarette smoking, which is exceptionally common among Maori women (and men).[11] The mortality rate from lung cancer among New Zealand women has increased since 1980, while that among men has declined.[11]

The incidence of colorectal cancer was also higher in New Zealand than in Australia, with the difference being especially notable (20% higher) in females. Both men and women in New Zealand have among the highest incidence and mortality rates for colorectal cancer in the world.[12] Although dietary factors are often assumed to be responsible,

there has been little epidemiological research on colorectal cancer in New Zealand. Analysis of trends in mortality[13] and research on migrants between New Zealand and England and Wales[14] suggest that exposures or behaviours early in life are critical to the high risk of colorectal cancer in New Zealand. The incidence of colorectal cancer appears to be lower in Maori and in Pacific Islands people living in New Zealand.[12]

For many sites in both sexes, mortality/incidence ratios were higher in New Zealand. The overall ratio for men was probably distorted by the fact that prostate cancer diagnoses had been inflated by the recent acceptance of screening by some New Zealand men.[6] In the years studied here, the recorded incidence of prostate cancer was 28% higher in New Zealand than in Australia, whereas mortality was only 6% higher. When this disease was excluded, the overall mortality/incidence ratio was higher in New Zealand males as well as females. The limitations of mortality/incidence ratios have been mentioned, but the findings of this study suggest that survival following treatment of some cancers has been poorer in New Zealand. This could reflect delays in presentation, less optimal treatment, or a combination of these factors. Further information could be provided by comparisons, for each site of cancer, between New Zealand and individual Australian states. Of course the Australian rates are dominated by the experience of New South Wales and Victoria, and the small populations of several states make reliable comparisons difficult.

From the data in Table 4, the New Zealand disadvantage in survival would appear to be particularly striking for Maori men and women. Recent work has shown that Maori mortality rates have been substantially underestimated from routine data.[15] The mortality/incidence ratios for Maori would also be misleading if Maori ethnicity was more likely to be recorded on death certificates than at the time of cancer registration. The existence of such an artefact is suggested by the fact that, for lung cancer and stomach cancer in Maori males, the mortality/incidence ratios estimated were greater than unity. Further evidence of an artefact is provided by observations of the changes in mortality and incidence rates that occurred when the system of self-identified ethnicity was introduced for all health statistics: whereas the (all-cause) mortality rate for Maori increased by about 25%,[2] cancer incidence rates for Maori declined.[6] Clearly formal survival analyses are essential, and there is a need to investigate why Maori men and women appear to have a high mortality from cancer in relation to incidence. The high incidence of lung cancer in Maori men and women is also a major challenge for cancer prevention.

Unfortunately the gap between New Zealand and Australia in controlling cancer is likely to widen during the next few years. Australia has already implemented a national cancer control initiative, but repeated calls for similar action in New Zealand (as recommended by the World Health Organization) went unheeded during the 1990s.[16,17] A new body called the New Zealand Cancer Control Trust is now working with the Ministry of Health to promote the development of a national cancer control strategy. Whether this welcome initiative will lead to constructive action remains to be seen. Public attention has been focused on inadequacies in provision of treatment services (such as radiotherapy), but priority also needs to be given to efforts to discover reasons for the high incidence of cancer in New Zealand, and to enhance prevention, appropriate screening, and early diagnosis.

Both epidemiological research and audits of cancer management have become very difficult in New Zealand, because of an increasing emphasis on privacy as an overriding principle. Recent events in relation to cervical screening would suggest that the population-based surveys of cancer management, which have such potential to improve cancer treatment in Australia, could not be conducted adequately in New Zealand under present conditions. It is for society to decide on the appropriate balance between privacy and autonomy, on the one hand, and the welfare of citizens, on the other. Perhaps awareness of the higher risk of dying from cancer in New Zealand will lead to some reassessment of this balance.

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Curious connection

A retrospective review of hospital patients with bacteraemia has found reduced mortality among those taking statins. The study, from the United States, involved 368 male veterans and two women with bacteraemic infections caused by aerobic gram-negative bacilli or *Staphylococcus aureus*. Although the 35 patients (9%) who were taking statins were more likely than those not taking statins to have diabetes, hypertension and coronary artery disease, they had significantly lower rates of death from any cause (6% v 28%; p=0.002), and attributable to the infection (3% v 20%; p=0.01). The authors suggest that this may result from the effects of statins on the inflammatory process. This interesting finding will need to be confirmed in a prospective study.

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Return to work outcomes following Accident Compensation Corporation Work Capacity Assessment

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Abstract

Aims. To determine the proportion of Accident Compensation Corporation (ACC) claimants who have returned to fulltime work after ceasing to receive ACC weekly compensation following Work Capacity Assessment (WCAP). To assess what factors impact on return to work. To assess whether ACC's research findings into return to work outcomes WCAP are valid.

Methods. A structured questionnaire telephone follow-up survey was conducted with ACC claimants seen for WCAP.

Results. 43% of those exited from ACC weekly compensation after WCAP were currently working fulltime. Claimants who had exited ACC after WCAP were significantly more likely to be working than those remaining on ACC. Claimants over 40 years of age were significantly less likely to be working. Gender, race, length of time since injury, and retraining made no difference to return to work. 80% of claimants felt that the WCAP process was unfair.

Conclusion. Nearly half of those claimants certified as being unfit for work but now exited from ACC via WCAP were working fulltime. This may indicate that ACC's rehabilitation is successful, or that claimants tend to remain on ACC for economic rather than injury reasons, or that WCAP results in claimants returning to physically unsuitable work putting them at risk of further injury. ACC's research finding that 79% of claimants were working after WCAP does not appear to be valid.

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Accident Compensation Corporation (ACC) is New Zealand's national injury insurer. ACC administers New Zealand's no fault compensation scheme, is responsible for rehabilitation, and pays weekly compensation (set at 80% of pre-injury income) if claimants are unable to return to their pre-injury work. ACC covers injuries in and outside the workplace.

In legislation a claimant receives weekly compensation until they have a capacity to work 30 hours per week in a job they are physically and vocationally suited for. (New legislation has changed this to 35 hours per week). The Work Capacity Assessment (WCAP) under new legislation now called Vocational Assessment, was the test used by ACC when they felt a claimant had finished rehabilitation and had a capacity to work for 30 hours per week, and was therefore eligible to cease receiving ACC weekly compensation. Claimants were seen by an occupation assessor, who produced a list of job options the claimant is vocationally suited for, and a list of physical tasks involved for each job option. The claimant was then seen by a Medical Assessor, who determines whether the claimant had a capacity to work for 30 hours per week in each job option listed. If the claimant was assessed as having a capacity to work in any job option, ACC could cease their weekly compensation three months after the medical assessment. Note that for ease of reading, in this article 'exited or exiting from ACC' should be taken as meaning 'ceased or ceasing to receive ACC weekly compensation'.

ACC has undertaken it's own research into the numbers of ACC claimants who have returned to work after exiting ACC via WCAP. Claimants exited from ACC between 1/7/98 and September 1999 were matched with Work and Income New Zealand (WINZ) data to see if they were receiving a WINZ benefit. ACC reported that "79% did not go onto a benefit within three months of leaving the WCAP".[1] From this finding ACC reported that "we can infer that the 79% who did not receive a benefit re-entered the workforce."

It is disappointing that such a major socio-political program as WCAP, where ACC claimants can have their weekly income ceased, and face a return to work or going onto a WINZ benefit such as the Unemployment or Sickness Benefit, has such little published information about its effects on claimants.

The aim of this research was to determine the proportion of those ACC claimants who I had seen for WCAP and who were assessed as having a capacity to work, that had actually returned to work (using the then legislative requirement of working at least 30 hours per week). Also, an assessment was made as to whether factors such as age, years since injury, ethnicity, gender, retraining, and injury site impacted on the return to work. An assessment was made as to whether ACC's research method and findings into the percentage of ACC claimants who have returned to fulltime work following WCAP are valid and reasonable.

Methods

ACC claimants I had seen for WCAP, and who were assessed as having a capacity to work in one or more job options, were administered a structured telephone questionnaire. This was administered after receipt of verbal consent, and included the following questions: Did you feel that the WCAP process was fair? Are you still receiving ACC weekly compensation? Are you currently working for 30 hours per week or more? Are you male or female? What is your ethnic group, race, or culture? Are you over 40? Where on your body was your injury? (*If back:* Have you had a back operation?). Was your work at the time of your injury heavy or light work? Had ACC provided retraining (*If yes:* Are you now working in that area?).

Inclusion criteria were any ACC claimants seen by me for WCAP from 1/7/98 to 30/6/99 inclusive and who were assessed by me as having a capacity to work in one or more job options (and therefore were eligible to exit ACC in three months time). There were no exclusion criteria.

The survey was conducted in November 2000. The average length of time since WCAP assessment was 20 months (range 17-28 months). A total of 184 claimants met the inclusion criteria. 37 claimants were unable to be contacted, and a further six declined to take part. 141 participants took part in the survey, giving an overall response rate of 76%, and a 96% response rate from those who were contacted.

The data were analysed using chi-squared tests and multiple regression.

Results

Of all 141 respondents who completed the survey, 54% were male, 9% Maori, 63% aged 40 years or more, 80% felt the WCAP process was unfair, 59% had been injured five or more years before the WCAP assessment, 67% worked in heavy manual work at time of injury, 82% reported that no retraining of any form had been provided by ACC, and 15% were still receiving ACC weekly compensation (21 claimants out of 141). There are a number of reasons why a claimant

may remain on ACC weekly compensation even after being assessed as having a capacity to work, including injury worsening, sustaining another injury, and/or succeeding in a Review against the decision to cease weekly compensation. Data from those respondents who had been exited from ACC (120 out of the 141) are summarized in Table 1.

Table 1. Data for respondents who had exited from ACC (120 out of 141).

51% male.
8% Maori
61% age 40 or more,
78% felt WCAP was unfair.
56% were injured more than 5 years before the WCAP assessment.
66% worked in heavy manual work at time of injury.
85% reported no retraining of any form.
37% of those over 40 working.
45% of those whose injury occrred more than 5 years ago now working
44% of those working in heavy work at time of injury now working.
44% of those working in light work at time of injury now working.
51% of males now working.
36% of females now working.
33% of Maori now working.
44% non-Maori now working.
43% of the group were currently working 30hrs per week or more.

'Working' indicates working for 30 or more hours/week.

There were many sites of injury, and some claimants reported multiple injury sites. The four main injury sites were: 79 backs, 55 arms, 36 necks, and 22 other. Of those with back injuries 62% were men. 77% of back injuries occurred to workers in heavy physical work. Of those exited from ACC with back injuries, 52% currently were working 30 hours or more per week. Of those exited from ACC with back injuries who had an operation, 45% were currently working 30 hours or more per week. Of those with neck injuries 58% were female. 52% of those exited from ACC with neck injuries were currently working 30 hours or more per week. Of those with arm injuries, 58% were women, and 38% of those exited from ACC with arm injuries were currently working 30 hours or more per week.

Significant differences between those claimants still on ACC and those exited from ACC are summarized in Table 2.

Table 2. Statistically significant differences between claimants still on ACC and those exited from ACC.

1. Those not on ACC were more likely to be working (52 out of 120) than those still on ACC (1 out of 21), (χ^2 p=0.0008).
2. Claimants who have had retraining were more likely to still be on ACC (8 out of 26) than those who did not have retraining (13 out of 115), (χ^2 p=0.01).

Significant findings noted using logistic regression (p<0.05):
1. Claimants seen earlier in sample period were more likely to be working.
2. Claimants seen later in sample period were more likely to be still on ACC.
3. Claimants over 40 years were less likely to be working.

'Working' indicates working for 30 or more hours/week.

No significant relationship (χ^2 or logistic regression) was found between those working and not working and: thinking WCAP was fair or unfair; being injured more or less than five years ago; male or female; Maori or non-Maori; doing heavy or light manual work at time of injury; being retrained by ACC; having had a back operation for back injury; and site of injury. One finding approached statistical significance; claimants with back injuries exited from ACC were more likely to be working (34 out of 66) than those with non-back injuries exited from ACC (19 out of 54), (χ^2 p=0.07).

Comparing my data to ACC's data is difficult as different questions were used to gather each data set. The ACC data comes from an inference that those not on a WINZ benefit have returned to work, whereas my data come from the question; if you have ceased receiving ACC weekly compensation, are you currently working 30 hours or more per week? Also, the time periods of the studies were slightly different. Nevertheless, as a comparison, in the ACC group of 3887 claimants, 79%, or 3071 were "working" vs 816 "not working". In my group 52 claimants were working 30 hours or more per week vs 68 not working 30 hours per week. The difference in the percentage of those working in my data and ACC's data has a p value of <0.0001(χ^2)

Discussion

There are potentially serious problems with ACC's inference that 79% of claimants exited from ACC weekly compensation have returned to work. This is because there may be many other reasons (besides a return to work) that ACC claimants are not receiving a WINZ benefit. For instance claimants who are not working but who have a partner or spouse at work may not be eligible for a WINZ benefit. Also, claimants, who are not working but who have a partner or spouse on a WINZ benefit may not be eligible for a WINZ benefit. Other claimants may have a separate income source and may not have requested a WINZ benefit, even if they have not returned to work. Some claimants may have left New Zealand. Therefore the inference made by ACC that the 79% of claimants exited from ACC who were not on a WINZ benefit had all re-entered the workforce would seem to be an invalid inference.

The substantial difference between my data and that from ACC supports the view that ACC's use of WINZ benefit data is not a valid tool for estimating the number of claimants that have returned to work after exiting from ACC via WCAP. The practice of using WINZ benefit data as a proxy measure of whether a claimant has returned to work seems flawed, as there are many other reasons besides a return to work that a claimant may not be receiving a WINZ benefit.

Further, the WCAP criteria, set in ACC legislation, was that claimants must have a capacity to work for 30 hours per week. Therefore any research looking at the results of WCAP should be looking specifically at whether claimants have returned to work for 30 hours or more per week. ACC's research does not report this, and the 79% of those claimants not on a WINZ benefit, whom ACC "infer" are working, may include many claimants who are working less than 30 hours per week.

There are a number of limitation in my study. There is a potential for bias as 24% of those claimants who met the inclusion criteria were not contactable. Nevertheless all claimants who met the inclusion criteria would have been included if contacted and consented. That they were not contactable by phone could mean they were more likely to have returned to work (ie moved to another area for work, or gained work and moved house), or they were less likely to have returned to work (ie now receiving less income so have moved house, or receiving less income meaning the phone had been cut off). Another limitations is that this study depended almost totally on claimant recall. This is difficult to counter in such a telephone survey. No attempt was made to verify the claimant's report of whether they had received retraining, their injury site etc. It could be that what ACC considers retraining may differ from what claimants consider retraining.

There were some unexpected findings. In particular, having been retrained by ACC was associated with being more likely to still be on ACC, but not more likely to be working. This raises a number of questions: does ACC tend to retrain those claimants with more severe injuries, and are those claimants more likely to remain on ACC? Do those

part-way through retraining tend to stay on ACC until their training is finished? Is the training realistic for current job market and the claimant's injury?

The finding that 43% of claimants having exited ACC after WCAP were currently working 30 hours or more per week is perhaps surprisingly high, given that many claimants were reported by their GPs to be either unable to work at all, or able to work only limited hours (and thus were receiving weekly compensation). I have been unable to find other studies assessing return to work outcomes following cessation of compensation after mandated work capacity assessment process. However, the 43% of claimants returning to work corresponds well to the 47% of musculoskeletal pain claimants who had returned to work fulltime at a median of five months post rehabilitation program at WorkWell.[2]

The fact that 43% of claimants exited from ACC had returned to work fulltime could be seen as an indication that the rehabilitation provided by ACC was entirely appropriated and adequate. Alternatively it could be seen as showing that the reason some claimants remained on ACC was an economic one rather than an injury one; ie they did have a capacity to work but it was financially advantageous for them to remain on ACC weekly compensation. Finally it could be that some claimants, due to financial necessity after ceasing to receive weekly compensation, have now returned to a work situation that is physically unsuitable for them, and may be at increased risk of further injury. From the data published by ACC and the data in this study it is impossible to distinguish between these possibilities.

This study did not attempt to look at current work roles, compare current work with the WCAP job options, compare current and pre-injury income, or look at whether those claimants who have returned to work fulltime have an increased injury rate compared to other workers. These are all valid questions and I believe that further research into the effects that the WCAP program may have on claimants after they have exited from ACC needs to be undertaken.

If a study, with greater numbers, was to be performed, there are many other areas that could also usefully be looked at: do WCAP doctors all have the same threshold for determining capacity to work? If not is there a need for more training? Is there a difference in return to work rates in different regions? If so, is this an economic effect or a medical effect? Do some groups (eg, those over 40) need more intensive rehabilitation than other groups? Do back operations change the return to work rates for claimants with back injuries? Is an increased return to work rate a useful measure of success for a back operation? Should ACC's retraining program be re-evaluated? (given that the only thing that retraining was related to in my group was remaining on ACC!)

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Co-morbidity and health outcomes in three Auckland hospitals

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Abstract

Aims. To establish the burden of co-morbid disease using the Charlson Index among hospital inpatients and its relationship to key health outcomes.

Methods. An initial screen was carried out on 1 575 medical records selected by systematic list sample from admissions for 1995 in three public hospitals in the Auckland region. In the course of the administration of the instrument, screeners were required to record the occurrence of co-morbid disease using the Charlson Index.

Results. A third of patients had co-morbid disease, of which chronic pulmonary disease and congestive heart failure were the most frequently recorded. While the Charlson Index was associated with age of patient, length of stay, inpatient mortality, and adverse event status, the simple presence or absence of co-morbidity was as an effective predictor as the extended index. Co-morbidity was more likely to be recorded for Maori, for patients from deprived areas, and for circulatory or respiratory diagnoses. Specific co-morbid conditions were predictive of health outcomes.

Conclusions. Levels of co-morbid disease established for patients using the Charlson Index in three Auckland public hospitals are similar to those recorded internationally. Co-morbidity is predictive of key health outcomes that are of clinical and managerial significance. Controlling for co-morbidity will be important in making comparisons of the quality of care.

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There has been growing pressure on the acute public hospital sector in New Zealand over the last decade. While the number of hospital beds has declined, inpatient discharges have increased markedly, and the average length of stay has virtually halved.[1] At the same time, public expenditure on health as a proportion of the total has declined.[2] Furthermore, a number of hospitals over this period were under severe financial and managerial strain.[3,4] It should be noted, however, that the pressure of hospital restructuring is an international phenomenon.[5]

Under these circumstances it becomes increasingly important to develop effective measures of performance and outcome in the hospital sector. In particular, evaluations of such criteria using administrative data need to control for co-morbidity in any comparisons across interventions, providers, or hospitals.[6] One of the earliest and most widely applied measures of co-morbidity has been the Charlson Index, originally developed as a prognostic indicator of mortality risk in longitudinal studies. The index is based on a prognostic taxonomy of co-morbid conditions, with weights assigned according to the number and seriousness of such conditions.[7] The application of the index has since been extended to risk adjustment in claims-based research.[8]

The object of the present study is to evaluate this extension of the index by assessing the pattern of co-morbidity identified using the Charlson protocol in an acute hospital population and to estimate its predictive potential for key health outcomes - namely, length of stay, inpatient mortality, and the occurrence of adverse events.

Methods

The three hospitals in the study are the major, non-specialist public hospitals in the Auckland region. The sampling frame for each hospital was a list of all eligible admissions in that hospital. New Zealand Health Information Service (NZHIS) selected a cross-sectional systematic list sample of 525 admissions from each of these hospitals for the year 1995, ordered by admission date. The number of records was divided by 525 to give a sampling interval, and then a random starting point between 1 and 100 was used to determine the first record sampled. Thus, sampled records were evenly spread over the entire year.

Standard hospital inpatient information for each sampled admission was provided by NZHIS. This included admissions information (dates of admission and discharge, admission type and source), socio-demographic data (age, gender, ethnicity, domicile code), and clinical data (diagnostic classification). Fuller methodological details of the study have been published eslewhere.[9]

Co-morbidity data were collected as part of a screening step in a two-stage retrospective review of sampled medical records.[9] This stage was undertaken by registered nurses working from a standardised protocol. The purpose of this stage was to ascertain if the hospitalisation in question met any of 18 screening criteria that might indicate any untoward event. As part of the assessment screeners were required also to answer questions on the presence of co-morbidity, defined as a pre-existing medical condition unrelated to the principal diagnosis (Appendix). These 19 items were selected for their closeness to the construction of the Charlson Index. Trained screeners identified co-morbid conditions according to detailed definitions.[7]

Co-morbidity data were subsequently checked for potential overlap between principal diagnoses and recorded co-morbidity items. Only those items additional to the principal diagnosis were finally classified as co-morbidities. Miscellaneous 'other' conditions were either reclassified or excluded according to the specific co-morbidity types.

A total Charlson Index (ranging from 0 to 12) was calculated for each admission from the number of co-morbid conditions and their weights according to seriousness using the published protocol.[7]

Key health outcomes for this study were:

(1) Adverse Event (AE) - An AE was operationally defined as (a) an unintended injury, (b) resulting in temporary or permanent disability, including increased length of stay and/or financial loss to the patient, (c) that was caused by health care management rather than the underlying disease process.
(2) Length of stay, the number of days from admission to discharge; the mean was calculated for sampled admissions.
(3) In-patient mortality/death.

Key hospital inpatient factors were: age; gender; ethnicity (European, Maori, Pacific, other); area deprivation score (NZDep96 deciles were derived from patient domicile codes);[10] admission type (planned or acute) and admission source (routine or transfer from another hospital); and principal diagnosis or reason for admission (25 Major Diagnostic Categories derived from AN-DRG 3.1).[11]

Logistic regression was used to determine the contribution of continuous and binary versions of the Charlson Index, and also co-morbidity types, to predicting key binary health outcomes; that is, adverse event occurrence, an above average length of stay, and inpatient mortality. Initial models generated crude univariate odds ratios for each outcome. Further models were run that adjusted for age (in years), gender (male, reference = female), and case mix according to principal diagnosis (nervous system, respiratory system, circulatory system, digestive system, musculoskeletal system, skin/tissue/breast, pregnancy/childbirth, newborn/neonates, reference = remaining Major Diagnostic Categories each comprising less than 5% of all admissions). The adjusted odds ratios show the effect of the Charlson Index on the health outcomes controlling for the effects of all other variables in the model.

Results

The full list of types of co-morbidity itemised in the Charlson Index are outlined in Table 1 with index weight. They are ranked according to the frequency of their occurrence in patient records. The most frequent types of co-morbidity noted form the medical records were chronic pulmonary disease, congestive heart failure, myocardial infarction and moderate-to-severe renal disease; these were each present in over 5% of admissions.

Co-morbid disease was recorded for a third of admissions. In aggregate these admissions were associated with significantly higher than average patient age, length of stay, likelihood of inpatient mortality, and probability of an adverse event occurring.

In Table 2 values of the Charlson Index have been grouped and are outlined against various indicators. Average age of patient, length of stay, likelihood of adverse event occurrence all showed significant linear increases with Charlson Index, though the association was not monotonic for inpatient mortality.

In Table 3 odds ratios are reported from a series of logistic regression models for continuous and binary versions of the Charlson Index. The odds ratio for continuous index measures the effect on outcome of increasing the Charlson Index by one point. From the pattern of odds ratios it appears that information on the presence or absence of any co-morbidity is just as predictive of key health outcomes as the full index. This is confirmed by the respective R-square statistics, a measure of goodness of fit. After adjustment, the Charlson Index is significantly predictive of adverse event occurrence, while the binary measure is related to length of stay.

Given the predictive power of the simple absence or presence of co-morbidity, this measure is applied in Table 4. The presence of co-morbidity is tabulated in the first column against a number of key socio-demographic, administrative and clinical variables. It is apparent that the elderly, European patients, acute admissions and transfers, adverse events, and certain diagnostic groupings (circulatory, respiratory, kidney and urinary) are significantly associated with co-morbid disease.

In the second column these same data are adjusted for age. While the first column quantifies the likely burden of co-morbid disease associated with various patient categories, the second column identifies the risk factors for co-morbidity after adjusting for age. Thus, after adjustment, the relationship of ethnic group and area deprivation score to co-morbidity changes, with Maori and patients from disadvantaged areas emerging as being significantly at risk of co-morbid disease.

The extent to which specific co-morbidity types are predictive of health outcomes is considered in Table 5. While no co-morbid types predict inpatient mortality, congestive heart failure, moderate-severe renal disease and dementia

Table 1. Relationship between co-morbidity type and outcomes.

Co-morbidity type	Charlson Index weight*	% of Admission† (n=1326)	Age (mean years)	Length of stay (mean days)	Outcome % Inpatient Deaths	% Adverse Events
Chronic pulmonary disease	1	9.0% (119)	58.0	7.4	5.0%	18.5%
Congestive heart failure	1	8.1% (107)	75.6	9.5	5.6%	21.5%
Myocardial infarction	1	6.2% (82)	72.6	7.6	1.2%	17.1%
Moderate-severe renal disease	2	5.6% (74)	71.0	9.9	5.4%	20.3%
Cerebrovascular disease	1	4.2% (56)	73.8	9.7	3.6%	17.9%
Diabetes	1	3.9% (51)	67.5	8.9	3.9%	9.8%
Connective tissue disease	1	3.7% (49)	68.0	9.7	8.2%	22.5%
Tumour	2	3.5% (47)	69.5	7.6	4.3%	21.3%
Peripheral vascular disease	1	3.4% (45)	70.5	8.1	8.9%	15.6%
Ulcer disease	1	2.9% (38)	72.2	9.8	7.9%	23.7%
Dementia	1	2.0% (27)	81.5	12.9	3.7%	18.5%
Metastatic tumour	6	1.7% (22)	68.6	5.9	9.1%	31.8%
Diabetes with sequelae	2	1.4% (18)	65.1	7.2	0	16.7%
Moderate-severe liver disease	3	0.8% (11)	53.3	12.1	0	27.3%
Mild liver disease	1	0.5% (7)	70.7	11.6	14.3%	42.9%
Hemiplegia	2	0.5% (6)	68.5	15.7	0	33.3%
AIDS/ARC	6	0.3% (4)	36.5	2.0	0	0
Leukaemia	2	0.2% (3)	48.0	13.3	0	33.3%
Lymphoma	2	0.1% (1)	45.0	3.0	0	0
Patients with any co-morbidity		33.3% (441)	66.0 p=0.00001‡	8.1 p=0.00001‡	4.8% p=0.001§	15.2% p=0.001§
Total (n=1326)		-	45.0	5.7	2.5%	10.7%

*see Charlson ME et al, 1987.[7] †multiple responses are possible. ‡t-test. §chi-squared test.

approximately double the likelihood of a longer hospital stay. Chronic pulmonary disease, congestive heart failure, metastatic tumour, and mild liver disease are co-morbid conditions significantly predictive of adverse events.

Table 2. Relationship between Charlson Index and outcomes.

Charlson Index	% of Admission (n=1326)	Age (mean years)	Outcome Length of stay (mean days)	Outcome % Inpatient Deaths	Outcome % Adverse Events
0	66.7% (885)	34.5	4.4	1.4%	8.5%
1-2	22.4% (297)	64.2	7.6	4.0%	11.1%
3-4	6.5% (86)	71.3	8.7	8.1%	20.9%
5+	4.4% (58)	67.6	10.0	3.5%	27.6%
		p=0.0001*	p=0.0001*	p=0.001†	p=0.001†
Total	100% (1326)	45.0	5.7	2.5% (33)	10.7% (142)

*Pearson correlation. †Mantel-Haenzsel chi-squared test for linear association.

Discussion

Using detailed chart review, and applying the relatively widely used Charlson Index of co-morbidity, this study has confirmed that approximately two-thirds of patients admitted to three broadly representative acute public hospitals exhibit no defined pre-existing co-morbid conditions at admission. This figure is not notably discrepant from the level of co-morbidity recorded in an international benchmark study using a closely comparable protocol and chart review in the United States. In that study the proportion of admissions without co-morbidity in major public teaching hospitals - also defined according to the Charlson Index - was just over 55%.[12] In contrast, however, another study relying on a computer-based algorithm and expanding the range of co-morbidities reported a much lower figure of 40.3%.[6]

Some of the variability in the proportion of admissions with co-morbid conditions may relate to differences in the application of the Charlson Index. Thus it is likely that this study's exclusion of all co-morbidities directly related to principal diagnoses resulted in the finding of a relatively smaller number of admissions with co-morbid conditions compared to other research. Some complexity and subjectivity exists in which conditions can be regarded either as an exacerbation of a pre-existing disease or a new disorder.

Although the results of this study show that co-morbidity was associated with all three key outcome variables, it was also striking to note that the simple presence or absence of co-morbidity was as predictive of the outcome variables as the more elaborate and fully estimated Charlson Index. It should be borne in mind, however, that co-morbidity was strongly associated with other variables like age and diagnosis, such that controlling for these confounders greatly weakened any predictive power. Nevertheless, applying the simplified, binary measure identified patient groups likely to present a great burden of co-morbid disease, while a more refined analysis adjusting for age clarified the influence of underlying risk factors such as ethnic group, area deprivation score, and principal diagnosis.

Table 3. Crude and adjusted odds ratios for Charlson Index as predictor of outcomes.

		Outcome* Adverse Event	Outcome* Inpatient Death	Length of stay >mean†
Charlson Index 0-12	Crude OR‡ (95% CI)	1.28 (1.17-1.39)¶	1.24 (1.08-1.44)¶	1.29 (1.19-1.38)¶
	Adjusted OR§ (95% CI)	1.23 (1.12-1.36)¶	1.10 (0.92-1.32)	1.07 (0.98-1.16)
	R^2‖	0.0406	0.0346	0.1393
Charlson Index ≥1 vs 0 (ie co-morbidity present/absent)	Crude OR (95% CI)	1.94 (1.36-2.75)¶	3.64 (1.77-7.46)¶	3.03 (2.37-3.88)¶
	Adjusted OR§ (95% CI)	1.38 (0.91-2.11)	1.77 (0.78-4.04)	1.41 (1.05-1.91)¶
	R^2	0.0304	0.0352	0.1411

*logistic regression models were run for each outcome. †mean length of stay=5.7 days. ‡effect on the outcome of increasing Charlson Index by one point. §adjusted for age (years), gender, case mix (Major Diagnostic Category). ‖R-square is a measure of goodness of fit of the model. ¶p<0.05.

Table 4. Presence of co-morbidity by hospital inpatient factors and adverse event status.

Hospital Inpatient Factors	%Co-morbidity Present (n=1326)		% Co-morbidity Present Adjusted for Age-group (n=1326)	
Age group:				
0-14	2.2%	(4)	-	
15-29	7.7%	(19)	-	
30-44	16.4%	(37)	-	
45-64	36.6%	(85)	-	
65+	68.1%	(296)	-	
		p=0.001†*		
Gender:				
Male	35.1%	(218)	34.7%	
Female	31.6%	(223)	32.1%	
		p=0.180‡		p=0.302§
Ethnicity:				
European	38.7%	(342)	32.7%	
Maori	22.5%	(31)	42.9%	
Pacific	20.1%	(28)	33.5%	
Other	24.1%	(40)	27.8%	
		p=0.001‡*		p=0.045§*
Area deprivation score:				
1&2	33.7%	(92)	30.0%	
3&4	30.8%	(92)	30.2%	
5&6	39.2%	(102)	35.3%	
7&8	33.8%	(76)	35.0%	
9&10 (high deprivation)	30.0%	(76)	39.5%	
		p=0.682†		p=0.009§*
Admission type:				
Acute	41.9%	(340)	36.4%	
Planned	19.7%	(101)	27.5%	
		p=0.001‡*		p=0.001§*
Admission source:				
Transfer	41.7%	(20)	40.2%	
Routine	32.9%	(421)	33.0%	
		p=0.208‡		p=0.297§
Principal diagnosis (Major Diagnostic Category):				
Circulatory system	64.2%	(113)	48.4%	
Respiratory system	48.6%	(51)	43.5%	
Digestive system	37.1%	(49)	32.4%	
Musculoskeletal system	31.3%	(42)	30.7%	
Nervous system	40.7%	(37)	31.4%	
Kidney and urinary tract	47.2%	(25)	41.6%	
Skin, tissue & breast	29.1%	(23)	25.2%	
Injuries, poisonings & drugs	17.4%	(8)	25.1%	
Pregnancy, childbirth	5.4%	(8)	5.0%	
Other (remaining 15 MDCs, excluding newborns/neonates)	36.6%	(85)	34.0%	
		p=0.001‡*		p=0.001§*
Adverse Event Status				
AE	47.2%	(67)	38.6%	
Non-AE	31.6%	(374)	32.4%	
		p=0.001‡*		p=0.139§

*p<0.05. †Mantel-Haenzsel chi-squared test for linear association. ‡chi-squared test. §age-adjusted.

There are a number of problems with the Charlson Index. It was developed for a highly specific research purpose that may not make it generalisable to other data applications. This means that it cannot be assumed that the co-morbidities included in the index are comprehensive.[6] Aside from any shortcomings of the Charlson Index, it is not always straightforward distinguishing co-morbid - that is, pre-existing - conditions from complications arising after admission, even when using chart review rather than administrative data. Furthermore, there may be a certain arbitrariness in distinguishing between the principal diagnosis and other secondary - assumedly co-morbid - conditions. For all its shortcomings, however, it should be noted that a Charlson Index based on chart review has shown superior predictive power to the same index adapted for administrative data.[13]

Nevertheless, this study has shown that the simple presence of any one of 19 co-morbidity types associated with approximately one third of hospital admissions is significantly predictive of an adverse event, an inpatient death, or a greater than average length of stay. Further research needs to be carried out to determine whether this simple measure of illness complexity is of assistance in other clinical and managerial tasks.

While the Charlson Index was originally developed with a limited research purpose in view - that is, risk adjustment in longitudinal studies - it has been widely applied for a range of predictive and risk-adjustment purposes. Furthermore, with increasing availability of routine hospital administrative data it has been possible to translate the original chart method into a computer-based algorithm applying standard coding systems to secondary diagnoses recorded in administrative data bases.[14,15] Given the broadly similar admissions mix in this regional pilot to the results reported for a subsequent, nationally-representative study,[16] there is

Table 5. Adjusted odds ratios (95% CI) for co-morbidity type as predictor of outcomes.

Co-morbidity type	Outcome* Adverse Event	Inpatient Death	Length of stay >mean†
Chronic pulmonary disease	1.77 (1.05-2.97)‡	1.83 (0.70-4.76)	0.89 (0.58-1.36)
Congestive heart failure	1.98 (1.13-3.46)‡	1.18 (0.44-3.19)	2.08 (1.33-3.26)‡
Myocardial infarction	1.21 (0.64-2.28)	0.18 (0.02-1.38)	1.16 (0.72-1.87)
Moderate-severe renal disease	1.72 (0.92-3.24)	1.48 (0.47-4.68)	2.12 (1.27-3.54)‡
Cerebrovascular disease	1.35 (0.64-2.83)	1.11 (0.24-5.12)	0.83 (0.47-1.46)
Diabetes	0.74 (0.28-1.93)	0.94 (0.21-4.30)	1.47 (0.82-2.65)
Connective tissue disease	2.05 (1.00-4.21)	2.13 (0.65-6.96)	1.39 (0.76-2.53)
Tumour	1.56 (0.74-3.29)	1.00 (0.22-4.51)	0.55 (0.29-1.06)
Peripheral vascular disease	1.31 (0.56-3.07)	2.56 (0.81-8.09)	0.92 (0.50-1.72)
Ulcer disease	1.95 (0.88-4.31)	2.88 (0.78-10.81)	1.35 (0.68-2.67)
Dementia	1.27 (0.46-3.55)	0.46 (0.06-3.71)	2.64 (1.08-6.48)‡
Metastatic tumour	3.15 (1.23-8.11)‡	2.60 (0.53-12.81)	0.70 (0.28-1.74)
Diabetes with sequelae	1.33 (0.37-4.78)	-§	1.14 (0.43-3.02)
Moderate-sever liver disease	2.88 (0.72-11.48)	-	2.79 (0.79-9.82)
Mild liver disease	5.82 (1.11-30.39)‡	6.98 (0.69-71.10)	0.68 (0.12-3.84)
Hemiplegia	4.01 (0.69-23.24)	-	2.39 (0.41-13.80)
AIDS/ARC	-	-	-
Leukaemia	3.05 (0.27-34.27)	-	-
Lymphoma	-	-	-

*logistic regression models were run for each outcome, adjusted for age (years), gender, case mix (Major Diagnostic Category). †mean length of stay = 5.7 days. ‡p<0.05. §no cases in one of the outcome categories.

potential for this wider application to be assessed in the New Zealand context to complement chart-based studies of specific conditions.[17]

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Appendix. Co-Morbidities

Question: Does the medical record indicate that the patient has any of the following co-morbidities at the time of the index admission: (Please provide an answer for all the conditions listed below)

Yes	No	
❑	❑	Myocardial infarction
❑	❑	Congestive heart failure
❑	❑	Peripheral vascular disease
❑	❑	Cerebrovascular disease
❑	❑	Dementia
❑	❑	Chronic Pulmonary disease
❑	❑	Connective tissue disease
❑	❑	Ulcer disease
❑	❑	Mild liver disease
❑	❑	Moderate to severe liver disease
❑	❑	Diabetes
❑	❑	Diabetes w/ sequelae
❑	❑	Hemiplegia
❑	❑	Moderate to severe renal disease
❑	❑	Leukaemia
❑	❑	Lymphoma
❑	❑	AIDS/ARC
❑	❑	Tumour
❑	❑	Metastatic tumour
❑	❑	Other _____
❑	❑	Other _____
❑	❑	Other _____

Home intravenous antimicrobial service - twelve months experience in Christchurch

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Abstract

Aim. To review the clinical practice and complications of the home intravenous antimicrobial service at Christchurch Hospital after twelve months of full operation.

Methods. Clinical and microbiological diagnoses, antimicrobial therapy, and complications of home intravenous antimicrobial therapy were entered prospectively on an Excel data base.

Results. Of the 153 patients, 113 (74%) suffered from skin, soft tissue or bone and joint disease. A bacteriological diagnosis was made in 108 patients (71%). 119 patients were treated with the narrow spectrum agents -penicillin 20 (13%), flucloxacillin 55 (36%) and cephazolin 44 (29%). Ceftriaxone was used for treatment in fifteen (10%) patients. Peripherally inserted central catheters (PICC's) were used in 129 patients, midlines fifteen, peripheral angiocaths in eight, and a Portacath in one. An elastomeric infusion device was used in 80 patients and an infusion pump in 34. Complications developed in 31 (20%) patients including three infections and one jugular vein thrombosis. Fifteen patients (10%) were readmitted within one month of discharge.

Conclusions. The home intravenous therapy programme successfully used first line narrow spectrum agents initiated in hospital with avoidance of unnecessary broad spectrum agents. Complication rates were acceptable and likely to improve with experience in patient selection and provision of support services.

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Home delivery of intravenous antimicrobial therapy has become an established form of treatment in the USA and UK that is being applied to an increasing range of conditions.[1-4] Improvements in maintenance of venous access and delivery of antimicrobial agents have meant that the choice of therapy need not be driven by the logistics of delivery of the antimicrobial agent but can be based on spectrum, efficacy and safety. However, most recent published reports are of programmes that relied on long acting agents chosen because they offer convenient dosing regimens irrespective of spectrum of activity and potential to cause resistance.[5-8]

In early 1999 the Department of Infectious Diseases initiated the development of a home intravenous antimicrobial service at Christchurch Hospital primarily to manage patients requiring intravenous antimicrobials for seven days or longer. A review of the literature and local costing indicated that several strategies would be cost saving compared with continued hospitalisation.[1-4] We elected to use the same narrow spectrum agents for outpatient as for hospital therapy to ensure safety and a high quality of service rather than selecting cost savings as the guiding principles. To achieve this we have made extensive use of continuous infusion devices for administration of agents with short half-lives, peripherally inserted central catheters (PICC), and a community nursing service to supervise therapy.

We report our experience of the first twelve months of our home intravenous antimicrobial service, after an initial set up period of six months.

Methods

All patients accepted onto the home antimicrobial programme at Christchurch Hospital were included in the study. Patients with cystic fibrosis were excluded. Those with urinary tract infections were included only if oral therapy was unavailable and cellulitis only if longer term parenteral therapy (>7 days) was likely.

The study began six months after the service was set up and was accepting patients regularly. The decision to accept a patient onto the service was made by an infectious diseases physician and nurse specialist. An infectious diseases physician made the decision as to choice and dose of the antimicrobial agent, and the method and duration of therapy. A dedicated nursing team placed PICC and midline catheters. Weekly complete blood counts, serum creatinine and liver function test measurements were performed on all patients. Follow-up was through the infectious diseases outpatients clinic. In the first instance patients were seen once daily by a community nurse to administer antimicrobial therapy and monitor the patient's condition. Some patients were able to self-administer without daily supervision. A nurse was available 24 hours a day by phone to trouble shoot and a consultant infectious diseases physician was available as back up. All results were recorded prospectively and entered into an Excel database.

Results

153 patients were treated over a twelve month period. Of these 65 were female (42%) and the median age was 55 years (range 16-91). Median duration of treatment was 15 days (range 2-49). The majority of patients (113, 74%) treated suffered from skin, soft tissue or bone and joint disease (Table 1).

Table 1. Number of days antimicrobial therapy administered at home.

	Number	Median	Range
Cellulitis/bursitis	43	11	2-41
Osteomyelitis	29	21	5-49
Infected prosthesis	29	22	6-48
Septic arthritis	12	20	2-41
Endocarditis	9	20	7-34
Abscess	7	17	4-24
Septicaemia	7	10	2-26
Infected vascular graft	3	14	9-35
Meningitis	3	8	6-9
Other	11	9	2-36
Total	153	15	2-49

A microbiological diagnosis was made in 108 patients (71%) (Table 2). Most of those treated without a microbiological diagnosis suffered from cellulitis or bursitis. Ten isolates of *S. aureus* were susceptible to penicillin and there were no isolates of MRSA. Four isolates of coagulase-negative staphylococci were resistant to methicillin, but all were susceptible to vancomycin and teicoplanin.

Table 2. Organisms isolated from patients treated with home intravenous therapy.

Staphylococcus aureus	65	(54%)
Alpha haemolytic streptococci	12	(10%)
Coagulase-negative staphylococci	10	(8%)
Group B streptococci	7	(6%)
Group G streptococci	5	(4%)
Streptococcus pneumoniae	3	(3%)
Anaerobic streptococci	3	(3%)
Group A streptococcus	1	(1%)
Gram negative bacilli*	10	(8%)
Other organisms†	5	(4%)
Total	121	

15 patients had multiple isolates and in 45 no organism was found. **Escherichia coli* 3, *Enterobacter cloacae* 1, *Klebsiella pneumoniae* 1, *Proteus mirabilis* 1, *Pseudomonas aeruginosa* 3, *Serratia marcescens* 1. †*Cytomegalovirus*, *Enterococcus faecalis*, *Kingella sp*, *Propionobacterium sp*, *Mycobacterium cheloneae*.

119 patients were treated with narrow spectrum agents - penicillin 20 (13%), flucloxacillin 55 (36%) and cephazolin 44 (29%). Ceftriaxone was used for treatment in fifteen (10%) patients. These were five patients with endocarditis (alpha haemolytic streptococci), cellulitis four (unknown pathogen), osteomyelitis two (*Enterobacter cloacae*), meningitis one (unknown pathogen), septicaemia one (Gp B streptococcus), urinary tract infection one, Lyme disease one. Teicoplainin was used to treat both beta lactam resistant coagulase-negative staphylococcal infections of prosthetic devices (4) and as empiric therapy in patients with a painful joint, raised inflammatory markers, and pus in a joint aspirate but no bacterial isolate (6). Eight patients received two agents; gentamicin with a beta lactam in five of these (Table 3). Intravenous gancyclovir was given to a liver transplant patient with cytomegalovirus infection.

Table 3. Antimicrobial agents used in home therapy.

Penicillin	20	(12%)
Flucloxacillin	55	(34%)
Cephazolin	44	(27%)
Ceftriaxone	15	(9%)
Cefuroxime	4	(2%)
Teicoplanin	10	(6%)
Gentamicin	5	(3%)
Other*	8	(5%)
Total	161†	

*Other: amoxycillin(4), piperacillin(2), amikacin(1), ganciclovir(1), †8 patients received two agents.

Venous access was maintained using 134 PICC's in 129 patients, seventeen midlines in fifteen patients, peripheral angiocaths in eight, and a portacath in one. An elastomeric infusion device (Homepump®) was used to deliver antimicrobial agents in 80 patients, spring driven device (Paragon®) in three, infusion pump (Microjet®) in 34 and self administration in eight (Microjet® four, intermittent administration four).

Complications of antimicrobial treatment developed in 31 (20%) patients (Table 4). Septicaemia with *Klebsiella pneumoniae* occurred 28 days post PICC line insertion in one patient. Exit site inflammation associated with a heavy growth of coagulase-negative staphylococcus from the PICC line occurred in another patient. A third patient developed pain, swelling and redness around the Portacath access site two days after insertion. *Staphylococcus aureus* was isolated from pus taken from the area. All catheter related infections resolved with removal of the devices. Severe phlebitis was managed by catheter removal; five of the nine were cultured and all of these were sterile. Phlebitis occurred more than once in three patients.

Table 4. Complications of home intravenous therapy

Phlebitis	9
Occlusion	12
Leakage/Breakage	6
Infection	3
Clinical Thrombosis	1
Mechanical pump failure	4
Total*	35

*Occurred in 31 patients.

Catheters were reinserted for partial or complete catheter occlusion in four cases. One occluded catheter was managed successfully with urokinase, six partially occluded catheters remained viable after flushing, and two patients were changed to oral therapy.

Fifteen patients (10%) were readmitted within one month of discharge from hospital. Two required further washout of an infected prosthesis, three had slow resolution of cellulitis, one developed jugular vein thrombosis and another line sepsis. Three patients were readmitted for uncontrolled pain, two from spinal osteomyelitis and one osteomyelitis of the foot. One patient was discharged to an inappropriate level of care. Four were readmitted for unrelated conditions - depression, GI bleed, heart failure and chronic renal failure.

Discussion

The Christchurch Hospital outpatient intravenous antimicrobial service was set up with the stated aim of administering first choice therapy chosen for efficacy and minimising the impact of resistance on the microbial environment. The majority of patients had skin, soft tissue, or bone and joint infections. Narrow spectrum therapy that would have been used in hospital was administered in the majority of patients (78%). Cephazolin was substituted for flucloxacillin in some patients because it is stable and some infusions were manufactured off site. It is difficult to make direct comparisons with other centres because of differences in both patient mix and organisms treated. Some studies reported in the older literature used similar agents to those used by us.[9,10] In recent studies, ceftriaxone has been used extensively (31%-70% of cases)[11-13] and some groups have used teicoplainin as a preferred agent.[13] This runs counter to international calls to limit vancomycin and teicoplainin use because of spread of resistance in both hospitals and the community.[14,15]

We have not performed a cost benefit analysis as similar programmes elsewhere have been shown to be cost effective.[1-5,7] The duration of home intravenous therapy does not represent hospital days saved for some conditions, such as cellulitis, since the decision to stop was determined at weekly outpatient visits. There is thus potential for patients to be treated for longer periods than in hospital. In other cases such as bacterial endocarditis, treatment periods are well defined and this is a measure of hospital days saved.

The value of narrow spectrum agents compared with broad spectrum therapy is not included in most cost benefit analyses as the costs are difficult to measure and may occur over the longer term. Third generation cephalosporins have been associated with higher rates of super-infection by organisms such as *Clostridium difficile* than penicillins, which can be expensive if hospitalisation is required[16,17] and broad spectrum antimicrobials promote colonisation with resistant organisms in hospital.[18] It is highly likely that resistant organisms will spread in settings such as nursing homes and rest homes if broad spectrum agents are used in these settings and antimicrobial resistance patterns will become more like those in hospitals.[19] These risks are important and warrant a

prudent policy of using narrow spectrum agents when possible.

Overall some complication of therapy occurred in 31 patients (20%). Most of these were relatively minor, such as "bland phlebitis", and were managed as outpatients by the nursing team. Complete or partial occlusion of lines was relatively common (8.5% of all lines, 8.2% of PICC lines) which is also consistent with rates reported elsewhere (1-13% in PICC lines).[20-23] The more severe complications were a cause of concern and often resulted in hospital admission. Infection was recognised in 2% of PICC lines which is consistent with previous studies.[20-22] However, we did not culture all lines to detect subclinical infection. The infection of the portacath occurred at the time of implantation and was unrelated to home therapy. A worrisome clinical thrombosis occurred in one patient with a PICC line; this is similar to the rate (0.8%) reported elsewhere.[23] All of these complications were recognised early and treated effectively. We do not think such complications should prohibit the use of these devices but it is essential patients are warned of possible complications, nurses are well trained and patients are reviewed regularly at clinic visits by medical staff.

It is of concern that fifteen patients (10%) were readmitted to hospital. However, if the four cases unrelated to home intravenous therapy are omitted, this rate is similar to that in other series (1-8%).[24,25] In retrospect some of these complications could have been avoided with better selection of patients and nursing services.

As with any new service, outcomes are likely to improve with the benefit of experience. It is difficult to overemphasise the importance of an efficient team approach with all support services for the successful management of home therapy.

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Nursing today

Your correspondent returns nostalgically to the happy days when an amiable triumvirate ran our hospitals, matron and superintendent directing patient care, and board secretaries handling the nuts and bolts.

Nurses in training learnt by the apprentice system and supplied a hierarchy of skill and experience. Ward sisters shared nursing clinical observations with the medical staff in ward rounds.

Today's training system produces technocrats with, initially, little clinical experience, reading and writing case notes which largely reduplicate the medical record. Machines take our temperatures, blood pressure, and pulse.

Surely there are now too many chiefs and not enough Indians when both are needed? Is present nursing staffing appropriate and economical? Are communications between medical and nursing staff optimal for patient care?

Where now are the cool little hands that once took our pulses and soothed our fevered brows?

Alistair Burry. The Press 8/2/02. p14.

Public knowledge and attitudes regarding smoking and smoking cessation treatments

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Abstract

Aims. To investigate current public knowledge and attitudes to tobacco smoking and smoking cessation treatments.

Method. A telephone survey of 250 individuals randomly selected from the Christchurch Electoral Roll and assigned into one of three groups: current, ex and life-time never smokers.

Results. Significantly more current than ex-smokers cited habit as a major reason for continuing to smoke and a greater number reported using nicotine transdermal patches during a cessation attempt. Fewer ever smokers than never smokers stated health as a likely major motivation for cessation by smokers and believed doctors' advice and illness of a significant other highly influenced quit attempts. 55.7% of respondents believed nicotine patches to be the most effective smoking cessation method followed by 'cold turkey' (49.4%) and hypnotherapy (33.9%). While the majority of participants supported banning tobacco advertising (69.6%), banning tobacco sponsorship (59.6%), lower insurance rates for non-smokers (89.1%) and fully subsidised smoking cessation programmes (71.9%), significant differences were detected between groups regarding attitudes to tobacco control initiatives.

Conclusions. This sample were relatively ill informed regarding smoking practices in New Zealand and unaware of useful information to aid cessation. While evidence emerged to support current smokers being slightly better informed regarding proven strategies for cessation than ex-smokers, few current smokers were aware of efficacious interventions for smoking cessation.

NZ Med J 2001; 115: 219-22

For more than 30 years, there has been a gradual decline in smoking prevalence rates in New Zealand.[1] As in the USA and UK, however, this decline appears to have come to an end over the past decade.[2] Like Australia, Britain, Scotland, Japan and the United States, New Zealand has taken steps and at times led the way in countering this global trend.[1] However, despite a wide array of methods being available to the New Zealand smoker for some time to assist them to 'kick' their habit, few smokers have quit. Rather smokers have either reduced the amount smoked or switched to less costly hand rolled cigarettes.[3] Despite compelling evidence to support smoking cessation as being extremely cost-effective,[4-6] efficacious interventions including brief advice, behaviour therapy and nicotine replacement therapy (NRT)[6-8] may not have been easily accessed by the New Zealand nicotine addict, due to a lack of information. During the 1990s little emphasis was placed on marketing treatment strategies with little government expenditure directed into media campaigns advertising effective ways to quit smoking, or into the development of cessation products or programmes (personal communication with M Linton, Ministry of Health 1999). Furthermore, research conducted both overseas and in New Zealand on what continuing smokers perceive would be helpful in quitting has been inadequate, as have investigations relating to reasons given by ex-smokers for long-term abstinence.

In order to ascertain current knowledge and attitudes to tobacco smoking and smoking cessation interventions, this study aimed to investigate two key questions. First, how well informed are the general public regarding tobacco smoking and treatment methods for nicotine addiction? Second, do differences exist between current, ex and never smokers in their personal experiences, knowledge of and attitudes towards tobacco use and cessation interventions?

Methods

A telephone survey was conducted during November 1998 and March 1999 on 250 individuals randomly selected from the Christchurch Electoral Roll (n=165 933). Of the 478 people assembled, 144 could not be contacted. Of the 344 remaining, seventeen did not fit the criteria and 67 refused to participate, leaving a surveyed sample of 250. The response rate was 79.9% and the refusal rate was 16.7%. Each subject was interviewed once using a structured questionnaire, taking approximately ten minutes to administer. Responses were sought on questions regarding general attitudes to smoking and cessation practises, current tobacco legislation and the perceived role of government in assisting smoking cessation. Standard sociodemographic data were gathered including age, ethnicity, education level, employment status, source of income and living arrangements. Current smokers were administered a Fagerstrom Test for Nicotine Dependence and asked about their general smoking habits and past cessation attempts. Ex-smokers were asked questions relating to past smoking practices and their history of quit attempts. Never smokers were used as a comparison group and were asked questions to gauge their opinion on the reasons people give for quitting and their perceived effectiveness of existing cessation interventions.

Statistical Analysis. All data were entered into SPSS (Statistical Package for the Social Sciences, Version 8.0 for Windows) from which summary descriptive statistics were generated. Two tailed tests were performed and any statistical difference between individuals was detected based on p values, with the significance level set at $p<0.05$. Normally distributed data were compared using independent sample t-tests and analysis of variance. Non-parametric data were analysed using either Kruskal-Wallis or Mann Whitney-U tests. Categorical variables were investigated using Chi squared tests.

Results

Associations of smoking with sociodemographic variables. Of those sampled, 54% reported being never smokers, 28.8% considered themselves ex-smokers and 17.2% were current smokers. 85.5% reported being New Zealand European/Pakeha and 3.6% New Zealand Maori. The mean age was 40.8 years with ex-smokers being significantly older (43.9 years) than current (38.4 years) and never smokers (40.0 years) ($p=0.01$). 70% of participants had some tertiary education or had completed a tertiary degree (39.2% and 30.8% respectively) with significantly more never smokers (40%) than current (14%) or ex-smokers (23.6%) gaining a tertiary degree ($p=0.01$). Over two-thirds of participants were in either full-time, part-time or self-employment and 10% reported being unemployed, with a government benefit being their main source of income.

Cessation practices. Of the 43 current smokers, 37 (86%) had attempted to quit at least once, with the mean number of quit attempts being 5.8. The mean number of quit attempts made

by ex-smokers was 5.7 and the mean number of years smoke free was 11.5. While the mean number of previous quit attempts by ex-smoking men was 9.4 compared to 3.1 for their female counterparts (p=0.04), no statistical differences were detected based on gender for current smokers. Significant differences were detected between groups when asked their opinions about stopping smoking with many responses given by never smokers differing markedly from ever smokers. Significantly more never smokers stated health as the primary motivation for smokers quitting with advice from a doctor influencing smokers to quit, and encouragement from others and illness of a significant other providing motivation for cessation attempts (p<0.001). 'Cold turkey' was the most common method used during a cessation attempt with 102 (95.3%) ever smokers using this method during a quit attempt. Significantly more current (30.6%) than ex-smokers (13.1%) used nicotine patches at least once (p=0.04).

When asked to rate their opinions on the effectiveness of seven cessation methods the proportion of respondents considering various smoking cessation methods helpful was: 55.7% for nicotine patches, 49.4% for 'cold turkey', 33.9% for hypnotherapy, 30.8% for acupuncture, 27.8% for Nicobrevin, 27.7% for nicotine gum and 12% for nicotine spray (Table 1). Despite the majority of current and ex-smokers using 'cold turkey' during at least one cessation attempt, this method was rated second to nicotine patches in effectiveness. While significantly more current and ex-smokers than never smokers suggested 'cold turkey' was very helpful (p=0.007), this trend reversed regarding the usefulness of transdermal patches, with 68.1% of never smokers compared with current (45.2%) and ex-smokers (38.9%) rating patches as being helpful (p<0.001). Statistically significant results were also detected between groups in respect to nicotine nasal spray (p=0.003), nicotine gum (p=0.003), Nicobrevin capsules (p=0.005), acupuncture (p<0.001) and hypnotherapy (0.001), with fewer current and ex-smokers than never smokers reporting these methods as helpful.

Attitudes regarding smoking control practices. Table 2 outlines responses regarding initiatives aimed at reducing smoking. While less than 40% of those surveyed agreed that higher taxes would help people to quit smoking and prevent children from becoming smokers, 26.2% of smokers supported taxation as an aid to quitting and 30% agreed that higher taxes would prevent smoking uptake. Significantly fewer current smokers than non-smokers agreed that sponsorships should be banned (19.4%, 61.3% and 71.3%) (p<0.001) and tobacco advertising should be prohibited (52.8%, 69.8% and 71.3%) (p=0.04). While 90% of those polled either agreed to insurance companies reducing premiums for non-smokers, significantly more current smokers (26.2%) disagreed with the proposition compared with ex (9.4%) and never smokers (6.8%) (p=0.002). Just over 70% of the sample supported the proposition that the government should provide free, stop smoking programmes and of those who disagreed, significantly fewer were current smokers (11.9%) (p=0.02).

Table 1. Perceived effectiveness of methods available to stop smoking as reported by current smokers, ex-smokers and never smokers in a random community sample (n=250) %.

Methods and effectiveness	All participants (n=250)	Current smokers (n=43)	Ex-smokers (n=72)	Never smokers (n=135)	χ^2 (df=6)	p
Nicotine Gum						
Helpful	27.7	21.4	16.7	35.6		
Neither helpful/unhelpful	10.0	7.1	4.2	14.1		
Unhelpful	17.3	19.0	18.1	16.3		
Don't know	45.0	52.4	61.1	34.1	20.20	0.003
Nicotine Patches						
Helpful	55.8	45.2	38.9	68.1		
Neither helpful/unhelpful	8.8	14.3	6.9	8.1		
Unhelpful	11.6	21.4	12.5	8.1		
Don't know	23.7	19.0	41.7	15.6	28.88	<0.001
Nicotine Spray						
Helpful	12.0	4.8	2.8	19.3		
Neither helpful/unhelpful	7.2	7.1	5.6	8.1		
Unhelpful	8.8	7.1	5.6	11.1		
Don't know	71.9	81.0	86.1	61.5	19.59	0.003
'Cold Turkey'						
Helpful	49.4	62.8	63.9	37.3		
Neither helpful/unhelpful	8.4	7.0	5.6	10.4		
Unhelpful	36.9	27.9	25.0	46.3		
Don't know	5.2	2.3	5.6	6.0	17.77	0.007
Hypnotherapy						
Helpful	33.9	29.0	19.4	43.3		
Neither helpful/unhelpful	10.9	7.1	8.3	13.4		
Unhelpful	17.3	26.2	17.0	15.0		
Don't know	37.9	38.1	56.0	28.4	21.55	0.001
Acupuncture						
Helpful	30.8	14.3	19.7	42.0		
Neither helpful/unhelpful	10.9	9.5	11.3	11.2		
Unhelpful	16.6	26.2	11.3	16.4		
Don't know	41.7	50.0	57.7	31.0	24.49	<0.001
***Nicobrevin Capsules**						
Helpful	27.8	19.5	12.5	38.5		
Neither helpful/unhelpful	3.6	4.9	4.2	3.0		
Unhelpful	8.5	7.3	8.3	8.9		
Don't know	60.1	68.3	75.0	49.6	18.71	0.005

*Nicobrevin is a non-nicotine herbal product.

Table 2. Attitudes towards smoking control practises by smoking status in a random community sample (n=244) %.

Question	All participants (n=244)	Current smokers (n=42)	Ex-smokers (n=69)	Never smokers (n=133)	χ^2 (df=2)	p
Higher taxes on tobacco products would help people to quit smoking:						
Strongly Disagree/Disagree	60.5	73.8	60.3	56.1		
Strongly Agree/Agree	39.5	26.2	39.7	43.9	4.11	0.13
Higher taxes on tobacco products will prevent children from becoming smokers:						
Strongly Disagree/Disagree	62.3	70.0	56.0	63.1		
Strongly Agree/Agree	37.7	30.0	44.0	36.9	2.14	0.34
The law prohibiting the sale of tobacco products to minors should be strictly enforced:						
Strongly Disagree/Disagree	11.0	19.0	10.1	8.7		
Strongly Agree/Agree	89.0	81.0	89.9	91.3	3.50	0.17
All advertising about tobacco products should be forbidden by law:						
Strongly Disagree/Disagree	30.4	47.2	30.2	25.6		
Strongly Agree/Agree	69.6	52.8	69.8	74.4	6.18	0.04
Tobacco sponsorship should be forbidden by law:						
Strongly Disagree/Disagree	40.4	80.6	38.7	28.7		
Strongly Agree/Agree	59.6	19.4	61.3	71.3	30.73	<0.001
Life and medical insurance companies should charge lower insurance rates to non-smokers:						
Strongly Disagree/Disagree	10.9	26.2	9.4	6.8		
Strongly Agree/Agree	89.1	73.8	90.6	93.2	12.63	0.002
The government should provide free, stop smoking programmes:						
Strongly Disagree/Disagree	28.1	11.9	35.3	29.8		
Strongly Agree/Agree	71.9	88.1	64.7	70.2	7.35	0.02

Discussion

Hughes argues that the population of smokers is changing and that future tobacco consumers will be comprised of the uneducated and poor.[9] To some degree our study supports this prediction with both current and ex-smokers inclined to be less educated, unemployed and receiving a government benefit than never smokers. Furthermore, while ever smokers were more closely aligned in their educational, employment and income status, more current than ex-smokers reported being unemployed, having less education and receiving government assistance.

How well informed are the general public regarding tobacco smoking and treatment methods for nicotine addiction? Generally, our sample was relatively ill informed regarding smoking practices in New Zealand and unaware of useful information that may assist cessation. Despite the large body of literature reporting high relapse rates associated with 'cold turkey',[10-11] this was the technique of choice for quitting by most ever smokers. Conversely, while NRT has been available in New Zealand for a number of years and many studies support its effectiveness over most other traditional aids for cessation,[12-14] few ever smokers had used any form of NRT or were aware of their effectiveness.

Do differences exist between current, ex and life-time never smokers in their personal experiences, knowledge of and attitudes towards tobacco use and cessation interventions? Not only did significantly more current than ex-smokers in our sample state they had used nicotine patches, a greater number regarded NRT as being more effective and fewer were unaware if these methods were effective or not. While this result could attest to the ineffectiveness of patches (with almost a third of current smokers in this sample employing the method but subsequently relapsing), it is important to consider how long nicotine patches have been available in New Zealand. When transdermal nicotine was introduced into New Zealand ten years ago its value was questionable. Not only were they considered expensive but it was thought that because patches contained levels of nicotine they would sustain physical dependence.[15] Therefore it is likely that because ex-smokers in this sample quit smoking on average 11.5 years ago, for many of them nicotine patches would not have been an option. They may not have been available at the time of quitting, the negative publicity surrounding their application may have discouraged their use, or the cost may have proved prohibitive. However, with the recent implementation of cessation programmes incorporating subsidised patches, availability is less of a barrier.

Significant differences were detected between groups in regard to attitudes towards tobacco use and cessation interventions. More never smokers than current or ex-smokers stated health concerns, doctor's advice, encouragement from others and illness of a significant other as major reasons for quitting. It is possible that many people who have never smoked overestimate the level of cessation intervention performed by health professionals. While even brief intervention from a healthcare worker is effective in aiding cessation, less than half of all smokers report being asked about their smoking status or advised to quit by a medical practitioner, and fewer still receive specific advice on how to stop successfully.[16,17] With the exception of 'cold turkey', significantly more never smokers compared to ever smokers regarded all other cessation methods listed as being a helpful aid to quitting. A number of explanations could be offered to

account for these findings including the possibility that never smokers may be better informed about the usefulness of cessation methods than ever smokers. If this is the case then the slowing down of quit rates may be attributable in part to smokers being disadvantaged, as they may be limited by their ability to access necessary information regarding effective cessation methods such as NRT. The efficacy of NRT is well established with the use of transdermal nicotine doubling abstinence rates at six and twelve months when compared to unassisted methods of cessation.[7] However, while this therapy may be viewed in the literature as being an effective treatment strategy, it is possible that such information has not been adequately relayed to the smoking public. Conversely it could be argued that these never smokers were no more informed than their smoking peers, rather their responses may have been based upon perceived assumptions rather than acquired knowledge or personal experience. Perhaps those never smokers who regarded 'cold turkey' as unhelpful had observed smokers attempting to quit a number of times without success. Despite 'cold turkey' being the most common method employed to quit smoking for many years,[7,10,11] relapse rates are high and often multiple attempts are made before long-term abstinence is achieved.[18] As over half of all ever-smokers have used this method at least once,[11] many never smokers may have come in contact with cyclical abstaining/relapsing smokers. Therefore it is not unreasonable to expect someone who has never smoked to form a negative view about a procedure that is used repeatedly without apparent success. The perceived effectiveness of hypnotherapy, acupuncture and Nicobrevin further supports the assumption that this sample were unaware of effective smoking cessation treatments. Schwartz states that hypnosis as a cessation aid produces only modest results and the few studies investigating the effect of acupuncture on smoking quit rates report poor outcomes.[18] Furthermore, Nicobrevin has never been properly evaluated as a cessation treatment according to the criteria that would normally be employed (written communication with K Richardson, Action on Smoking and Health UK, 2001).

Despite current smokers being less supportive of legislative measures to curtail smoking than non-users, some degree of restriction on smoking was widely supported by all three groups. While fewer participants agreed that higher taxation on tobacco products would encourage smoking cessation or prevent smoking uptake by children, surprisingly almost 30% of those supporting these forms of tobacco control were smokers. Clearly the reasons for supporting government interventions differ based on smoking status. Comments made by a number of participants may provide some insight. In regard to legislative measures banning tobacco sponsorship and advertising, many smokers believed that since tobacco companies make considerable profit from their tobacco sales, these companies should contribute to sporting and recreation activities. Despite many smokers acknowledging the detrimental health effects of smoking to themselves and others, they believed displaying a tobacco company's logo on a sporting jersey would not encourage smoking uptake. Regarding the proposition that government should fully fund cessation programmes, current smokers are inclined to belong to less affluent lower socio-economic groups and subsequently are less able to afford cessation aids, hence free quit programmes should be beneficial. Why a high proportion of non-smokers were in favour of this proposition is not clear.

There were limitations in this study. While selection bias was mitigated through random selection, an imbalance occurred whereby fewer Maori (despite being over-represented in smoking statistics) or current smokers took part. In addition, although data extracted from telephone interviewing are of acceptable quality and commonly used in epidemiological studies to obtain information about public health issues, this technique does have shortcomings particularly in relation to coverage and response bias.[19] Limitations include the potential to exclude possible participants who do not own a telephone or have unlisted numbers.[19] Furthermore, evidence from other studies suggests that while a greater proportion of younger people consent to taking part in telephone interviews, fewer elderly, particularly those 65 years and over are willing to participate.[19] Finally, because responses provided by ex-smokers were taken at face value, determining the true rate of cessation was not possible. Ideally biochemical verification using expired air carbon monoxide or cotinine levels in biological fluids would have readily corroborated cessation by providing an objective quantitative marker of smoke intake.[20]

What is needed in New Zealand is further development of a multifaceted approach to smoking cessation that encompasses the ongoing evaluation of traditional smoking control measures with the development of interventions that encourage and facilitate cessation. Based on data from this study that smokers may be disadvantaged against quitting because information on effectiveness of some cessation programmes may not be filtering through, research is needed to determine the effectiveness of smoking cessation education and support. A follow-up investigation needs to be performed within the next few years so a detailed assessment can be made on whether the smoking public are receiving adequate information regarding the efficacy of the newest NRT product, nicotine nasal spray, and the more widely marketed and distributed nicotine patches. This research could also evaluate consumer satisfaction with the initiatives introduced by the Health Funding Authority during 1999 and 2000, namely, The Quitline, subsidised NRT and the Guidelines for Smoking Cessation distributed to all general practitioners.[21]

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CASE REPORT

Two cases of adrenal suppression following a Chinese herbal remedy: a cause for concern?

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Previous reports have suggested that herbal remedies may contain steroids.[1-4] We report two cases with adrenal suppression following ingestion of the Chinese herbal remedy, *Shen Loon* (Shen Loon She Enterprise SDN BHD, Penang, Malaysia). Further investigation was unable to demonstrate the presence of any known steroid in extracts of the remedy.

Case One

A 29 year old female with a three year history of seropositive rheumatoid arthritis, commenced *Shen Loon* as treatment for the arthritis. At the time, she was eleven months post partum with severe joint symptoms to the extent she could barely lift her infant. She had previously been treated with hydrochloroquine, salazopyrin and intermittent steroid injections. After taking two *Shen Loon* capsules daily for two days her joint symptoms resolved, allowing her to stop current non-steroid anti-inflammatory drug treatment. After one week, the dose of *Shen Loon* was reduced to one daily with continued control of symptoms. Overall wellbeing improved and she regained 20 kg in weight. Inflammatory markers also improved - April 2001: Hb 99 g/L, ESR 46 mm/hr, rheumatoid factor 332 IU/mL; August 2001: Hb 126 g/L, ESR 34 mm/hr, rheumatoid factor 234 IU/mL.

After four months on *Shen Loon*, and in view of her marked symptomatic response, her general practitioner (PLM) became concerned that this preparation may contain a glucocorticoid. Her morning plasma cortisol level was found to be abnormal at 8 nmol/L (normal >250 nmol/L) and a short synacthen test (250µg of ACTH) several days later, confirmed adrenal suppression with the 0800hr plasma cortisol 30 nmol/L, 60 minutes post synacthen 180 nmol/L (normal >550). Ambulant plasma aldosterone was 105 pmol/L (normal 100-800), plasma renin activity 1.0 nmol/L per hour (normal 0.4-2.3) and plasma ACTH at 1000 hours <0.2 pmol/L (normal 1.2-15.6).

Further enquiry (PJH) elicited facial redness, a tendency to bruise easily, longstanding mild acne and facial hirsutism, with regular menses. Examination revealed plethoric facies, thin skin, old bruises over the legs, mild perioral acne and hirsutism but no other features of steroid excess. Musculoskeletal examination showed a symmetrical increase in synovial tissue over her hand joints and a right knee effusion.

Shen Loon was stopped and she was commenced on prednisone 10 mg daily for one week before reducing to 5mg daily. Presently, joint symptoms are controlled on prednisone 5mg daily and further rheumatological review is planned, as well as reassessment of the hypothalamo-pituitary axis.

Case Two

A ten year old girl presented with weight gain and was noted by her general practitioner (DB) to have a cushingoid appearance. She had incidentally noted an improvement in her chronic severe eczema and asthma without the use of her regular inhaler (fluticasone) or topical steroid. The eczema and asthma dated from the age of two years and many treatments had been tried, both orthodox and complementary. She had taken *Cheng Kum* capsules for six months until they became unavailable in New Zealand. Subsequently, she took *Shen Loon* capsules one daily for five months until cushingoid features were noted. Weight at this time was 53.7kg (above the 97th centile, having been previously on the 90th centile), and height 140cm (50th centile). Her BP was 122/82 mmHg. An 0800hr plasma cortisol was 8 nmol/L with an ACTH level of 0.3pmol/L. *Shen Loon* was stopped and physiological replacement of prednisone instituted. Four months after stopping *Shen Loon*, cushingoid features were less noticeable and prednisone was stopped after a normal synacthen test.

Further investigations. In order to investigate possible adrenal suppression, two of the authors (PAE, JGL) had blood samples taken for plasma cortisol and ACTH before and after ingestion of *Shen Loon* as shown in Table 1. The cortisol and ACTH levels after *Shen Loon* were comparable to those seen following a 1 mg dexamethasone test, a cortisol of less than 100 nmol/L being a normal response

Table 1. Plasma cortisols (nmol/L) on the two volunteers. Days 1 and 2 are basal and day 3 following *Shen Loon* ingestion. Basal cortisol samples were within the method specific normal range (250-800 nmol/L).

Sample	Subject 1 cortisol (nmol/L)	Subject 1 ACTH (pmol/L)	Subject 2 cortisol (nmol/L)	Subject 2 ACTH (pmol/L)
Day 1 (0900) Pre *shen loon*	334	3.2	342	5.6
Day 2 (0900) Pre *shen loon*	303	3.1	332	3.7
Day 3 (0900) Post *Shen Loon*	87	1.2	72	0.7

Each capsule of *Shen Loon* contained approximately 480 mg of a yellowish powder. The packaging ingredient list included snake and plant extracts. We analysed the contents by thin layer chromatography (TLC), immunoassay and high performance liquid chromatography (HPLC) for steriods.

TLC and HPLC studies. Shen Loon capsules were extracted with 10 mL hexane and the dried extract reconstituted with either methanol for TLC, or 5 mL buffer for immunoassay. This was designated the organic extract. Alternatively the capsule was directly extracted with 5 mL buffer, the aqueous extract. Both organic and aqueous extracts were run under the same solvent system on aluminum backed silica gel plates containing 60 F_{254} silica gel 0.2 mm thickness. The solvent system was composed of dichloromethane: diethyl ether: methanol: water in ratio of 77:15:7:1. Capsule solutions were compared with solutions of

various steroids (1mg/mL). For the organic extract the markers were prednisone, cortisone, cortisol, fludrocortisone, prednisolone, 11-deoxycortisol and dexamethasone. There was no sign of any glucocorticoid in the organic extract and no spot was observed on the TLC plate. For the aqueous extract the marker steroids were prednisolone, cortisone, cortisol, 6β hydroxycortisol, 21-deoxycortisol and dexamethasone. Similarly, compared to the markers no spots were observed in the aqueous extract although a large amount of extract remained at the origin. Dexamethasone was undetectable by HPLC analysis. In addition *Shen Loon* was extracted with 10 mL of methanol and this extract also subjected to TLC. There were traces of three spots within the glucocorticoid region, which were none of the above steroids. In addition two further spots were observed with relative mobilities well outside the glucocorticoid region.

Immunoassay. Both organic and aqueous extracts and dilutions were analysed for cortisol, prednisone, 11-deoxycortisol, corticosterone, progesterone, 17α-hydroxyprogesterone, dehydroepiandrosterone sulphate, androsterone and epiandrosterone sulphates, testosterone, androstenedione, estrone-3-glucuronide and pregnanediol-3-glucuronide by established immunoassays. Essentially there were undetectable levels of immunoreactive material in the organic extract. In the aqueous extract there were minimal levels of immunoreactive material for most substances (<25 ng /capsule), including 11-deoxycortisol and prednisone (150 ng and 250 ng of immunoreactive material/capsule respectively). These apparent levels are at least 1000 fold lower than the recognised levels of steroid required for overnight adrenal suppression. Based on the known cross-reactivity profiles of the antibodies used for the immunoassays and the high concentrations of extract analysed (up to 100 mg/mL) we would expect significant inhibition of antibody binding, in at least some assays, even if less common steroids were present at significant levels. This was not the case and hence we concluded that the capsules did not contain significant levels of known steroids.

Discussion

We have described two cases with adrenal suppression following ingestion of the Chinese herbal remedy, *Shen Loon*. The overnight suppression of cortisol and ACTH seen in two healthy male subjects following ingestion of *Shen Loon* is comparable to that seen in a 1mg overnight dexamethasone suppression test. Potent steroid-like activity is therefore present in this preparation. Despite this, we were unable to identify any recognisable steroid in an extract of *Shen Loon* by TLC, HPLC or immunoassay. However, TLC of the methanol extract showed the presence of minor amounts of unknown compounds, some that chromatographed with apparent glucocorticoid like mobility. Since the time of submission of this article, the New Zealand Medicines and Medical Devices Safety Authority (www.medsafe.govt.nz) issued a communication that *Shen Loon* has been found to contain betamethasone in a dose range of 0.1 to 0.3mg per capsule. It is debatable, in our opinion whether this would be sufficient to account for the observed degree of adrenal suppression and this does not preclude the presence of other non-steroidal substances capable of adrenal suppression. Some Chinese herbal remedies, however have been shown to contain the potent topical steroid fluocinolone acetonide,[2] which would have been detected in *Shen Loon*, if present by TLC and immunoassay. Other herbal remedies of the Maritime Indians have been shown to contain sterols and triterpenes.[5] We consider it unlikely that isoflavones are the candidate substances as we have recently demonstrated that the ingestion of isoflavone extracts do not affect plasma cortisol levels.[6] In our opinion, *Shen Loon* capsules contain non-steroidal substances, which are capable of inducing marked adrenal suppression. The identification of the adrenal suppressant(s) in *Shen Loon* capsules would be a complex task with a requirement for both analytical and biological studies.

The occurrence of adrenal suppression following ingestion of products available over the counter is a matter of major concern. Abrupt withdrawal of such products causing adrenal suppression could precipitate acute adrenal failure with the potential for fatality. Medical practitioners need to be aware of such consequences. On a more positive note, identification of the active compound(s) may result in a substance with new medicinal value.

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Testing Time

Last century, the renowned US physician and scientist, Lewis Thomas, observed, "Today, with the advances in medicine's various and complicated new technologies, the ward round at the foot of the bed, the drawing of blood samples for automated assessment of every known biochemical abnormality, the rolling of wheelchairs . . . down to the x-ray department, there is less time for thinking." Lewis further noted that medicine "no longer involves the laying on of the hands", but rather "the reading of machines".

Medicine's reliance on testing continues unabated. The annual expenditure by Medicare for both pathology and radiology tests exceeds $2 billion and the annual growth for these services is a robust 5%.

Why so many tests? Apart from being definitive diagnostic tools in patient management, tests have other roles. There are "pressure tests" ordered to placate pressure from patients, family and peers; "routine tests" required by hospital protocols; "reassurance tests" to reduce the anxiety attending the uncertainty of practice; "fishing tests" to angle for remote diagnostic possibilities; "gamesmanship tests" to prevent upstaging by other doctors' and, finally, "lawyer's tests" as defence props in possible future medicolegal tussels. Ultimately, tests are easy to order, are readily available and, importantly, are paid by someone else.

But the compelling reason remains a lack of time. Time constraints, overwork and the pressure for immediate answers rule modern medicine. In this environment, it is easier to order tests than to conduct a rigorous history of physical examination, or, indeed, allow time to be the diagnostician.

The time for talking and the time for thinking is currently curtailed in consultations. Testing is now the surrogate for time.

Martin B Van Der Weyden. Med J Aust 2002; 176: 141.

Research Seminar for the Waikato Clinical School, April 18, 2001

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Management of unstable angina and non ST-elevation myocardial infarction in New Zealand: Do Cardiologists do it better? PJ Conaglen, C Jayaraman, C Nunn and G Devlin, Departments of Cardiology and General Medicine, Waikato Hospital, Hamilton and C Sebastian and A Abraham, Taranaki Hospital.

In New Zealand, a large proportion of patients with acute coronary syndromes (ACS) are managed by general physicians. Evidence suggests that patient care is improved by subspecialisation.

A retrospective study of the management of ACS at Waikato Hospital (W) and Taranaki Base Hospital (T). At W, the management of all ACS is specialist based with ready access to invasive cardiac procedures. General physicians manage the majority of patients at T.

A Total of 301 consecutive patients, with a diagnosis of unstable angina or myocardial infarction, without ST elevation, were admitted to W (n=144), and T (n=157). Mean age 68 years, 54% male. No significant difference was noted in risk factors or medications at discharge. Patients in W were more likely to undergo inpatient risk stratification, intervention and subsequent revascularisation with a shorter stay noted (median 4 v 5 days). At 6 months readmission rates were similar with a trend noted to reduced mortality in W.

Table

	Taranaki Base Hospital (n=157)	Waikato Hospital (n=144)	P
Catheterisation	20 (12.7%)	44 (30.6%)	0.0002
PTCA	4 (2.6%)	14 (9.7%)	0.01
CABG	6 (3.8%)	11 (7.6%)	
Pts undergoing Cath, ETT Echo or Nuclear Scan	72 (45.9%)	94 (65.3%)	0.0008
Readmission	35 (22.3%)	36 (25.0%)	
- Revascularisation	1 (0.6%)	10 (7%)	
Mortality	21 (13.4%)	14 (9.8%)	Ns

Direct admission with an ACS to cardiology services results in more frequent revascularisation, shorter hospital stay and a trend to reduced mortality at 6 months but similar readmission rates.

The use of respiratory muscle exercise equipment to improve exercise tolerance and breathlessness in people with COPD. R Vickery, K Evison and J Morrow, Physiotherapy Department, Waikato Hospital, Hamilton.

Chronic obstructive pulmonary disease (COPD) is the most common and debilitating condition in people over 45 years of age. Air trapped in lungs alters the shape of the rib cage, decreasing the strength, endurance and efficiency of the muscles used for breathing, leading to increased breathlessness and a decrease in the ability to carry out simple activities of daily living.

A pilot study was conducted to determine the benefit of re-training the breathing muscles based on the established principles of training normal skeletal muscles, using the Test of Incremental Respiratory Endurance (TIRE) protocol, in patients with COPD. The sample population was taken from people who had previously completed the Pulmonary Rehabilitation program at Waikato Hospital. 20 subjects were randomly assigned to either a control group (n=10) completing three times weekly general body exercise training, or the experimental group (n=10) completing three times weekly general body exercises and TIRE training. The trial was completed over a seven-week period.

Inspiratory pressures, breathlessness during activity and exercise tolerance were measured. The groups showed significant differences ($p<0.005$) in maximum inspiratory pressure (MIP) and sustained maximal inspiratory pressure (SMIP) post training. There were also improvements ($p<0.10$) in the incremental shuttle walk distance and associated perceived breathlessness using the BORG scale.

The outcome of this trial suggests that completing a seven week period of TIRE training three times a week, in addition to general body exercises, increases exercise tolerance in people with COPD.

Expression of *Igf-I* in response to growth hormone is mediated by the JAK-STAT5b intracellular signalling pathway. T Xie, MJ McLachlan, DJ Waxman, DR Grattan, RJ Wilkins and HW Davey, Biotechnology Group, AgResearch Ruakura, Hamilton.

The postnatal growth-promoting effects of growth hormone (GH) until recently were thought to be mediated by IGF-I produced in the liver. However, while *Igf-I*$^{-/-}$ knockout mice have severely retarded body growth, mice with a liver specific knockout of *Igf*-I have normal growth, indicating that liver-derived IGF-I is dispensable for normal body growth and that IGF-I produced locally in peripheral tissues mediates the major effects of GH on postnatal growth. In this work we have focussed on the expression of *Igf*-I and the stimulation of *Igf*-I expression in response to GH. Growth hormone is known to activate a number of intracellular signalling molecules including those involved in the JAK2-STAT5b pathway that have previously shown to be required for sexually dimorphic body growth and gender-specific liver gene expression patterns. The relative contribution of these pathways to the expression of *Igf*-I has been difficult to define. However the availability of mice that are deficient in STAT5b has provided a *in vivo* model to investigate the role of STAT5b. We found that in liver and skeletal muscle, *Igf*-I mRNA was lower in *Stat*5b$^{-/-}$ than wild type male mice. In females, while the *Igf*-I mRNA in the liver of *Stat*5b$^{-/-}$ mice was similar to that in wild type mice, in skeletal muscle *Igf*-I mRNA levels were higher in *Stat*5b$^{-/-}$ than in wild type mice. We further evaluated the role of STAT5b in mediating GH activation of *Igf*-I expression by measuring *Igf*-I mRNA in hypophysectomised wild type and *Stat*5b$^{-/-}$ male mice stimulated with pulses of GH. GH induced *Igf*-I mRNA expression in hypophysectomised wild type, but not in hypophysectomised *Stat*5$^{b-/-}$ mouse. These finding demonstrate that Igf-I expression levels are tissue and gender specific and that STAT5b plays an important role in the increased expression of *Igf*-I in response to GH.

A comparison of the frequency of Simian Virus 40 sequence in British and New Zealand mesotheliomas. F Mayall and K Barratt, Department of Pathology, Waikato Hospital, Hamilton, R Cursons, Department of Biological Science, University of Waikato, Hamilton and J Shanks, Christie Hospital, Manchester, UK.

Simian Virus 40 (SV40) DNA has been detected in human mesotheliomas in at least 30 different laboratories. However, there have been marked geographic differences. For example no evidence of SV40 has been found in Finnish or Turkish mesotheliomas but SV40 sequence was found in approximately half of mesotheliomas in the USA. SV40 is known to have contaminated most early batches of polio vaccine used in New Zealand but contaminated only a minority of the batches used in Britain.

We compared the frequency SV40 virus sequence in mesotheliomas from Britain and New Zealand.

Sixteen mesotheliomas from New Zealand and seventeen mesotheliomas from Britain were collected. DNA was extracted from formalin fixed paraffin embedded blocks of these tumours. DNA was extracted. PCR was used to amplify a 105 base pair region corresponding to the large T antigen region of SV40. PCR was performed using a Light Cycler for 40 cycles using a "touch down" program. A similar sized target on the beta-2 microglobulin gene was used as a positive control.

None of the seventeen British mesotheliomas showed SV40 sequence but two of the sixteen New Zealand mesotheliomas were positive for SV40 sequence and a further two were possibly positive. We conclude that SV40 sequence may be more common in New Zealand mesotheliomas than in British mesotheliomas.

Evaluation of a Rapid Immunochromatographic Test for the detection of *Streptococcus pneumoniae* antigen in urine samples from adults with community-acquired pneumonia. GD Mills and NC Karalus, Waikato Hospital, Hamilton, DR Murdoch, Clinical Microbiology Laboratory, Duke University Medical Center, Durham, NC 27710, USA and Microbiology Unit, Canterbury Health Laboratories, Christchurch, S Mirrett and LB Reller, Clinical Microbiology Laboratory, Duke University Medical Center, Durham, NC 27710, USA and RTR Laing and GI Town, Christchurch School of Medicine, University of Otago, Christchurch.

Streptococcus pneumoniae is the most common cause of community-acquired pneumonia, but is underdiagnosed. Isolation of *S. pneumoniae* from blood is specific, but lacks sensitivity, while isolation of *S. pneumoniae* from sputum may represent colonisation. We evaluated a new immunochromatographic test (NOW® *S. pneumoniae* Urinary Antigen Test, Binax, Portland, ME) that is simple to perform, and can detect *S. pneumoniae* antigen in urine within 15 minutes. Urine samples from 420 adults with community-acquired pneumonia and 169 control patients who did not have pneumonia were tested. Urine from 315 (75%) of the pneumonia patients and all controls was tested both before and after 25-fold concentration, while the remaining 105

samples were only tested without concentration. *S. pneumoniae* urinary antigen tests were positive in 120 (29%) patients with pneumonia and none of the controls. Of the urine samples tested with and without concentration, 96 were positive, of which 6 were positive only after concentration. *S. pneumoniae* antigen was detected in the urine from 16 of the 20 (80%) patients with positive blood cultures for *S. pneumoniae*, and 28 of the 54 (52%) patients with positive sputum cultures for *S. pneumoniae*. The absence of *S. pneumoniae* antigen in the urine from controls suggests that the specificity is high. Concentration of urine prior to testing resulted in a small increase in yield. The NOW® *S. pneumoniae* Urinary Antigen Test should be a useful adjunct to culture for determining the etiology of community-acquired pneumonia in adults.

The impact of self-concept enhancement on adolescent mood: Preliminary results. John M Fitzgerald, The Psychology Centre, Hamilton.

We examined the role of self-esteem and self-concept in the treatment of depression in adolescents and to develop and evaluate self-concept enhancement techniques in a clinical setting.

Six adolescents consecutively referred to a provincial community Child and Adolescent Mental Health Service participated in a study using a multiple single case study design. The self-concept enhancement component (three focussed sessions) was introduced in a staggered fashion to provide contact control. All participants completed a pre- and post-intervention assessment battery, and a somewhat shorter assessment battery at each session.

All participants reported significant improvement in mood over the course of the intervention. Measure of self-esteem and self-concept produced positive results. Quantitative data were generally supported by qualitative results, although the latter clearly demonstrates the complexity of adolescent self-esteem and self-conceptions.

This preliminary study provides some support for conceptualising intervention pathways with depressed youth in terms of the evaluation and enhancement of self-concept.

Magical ideation in schizophrenia: how do we distinguish it from everyday experience? AJ Tamatea and IM Evans, Clinical Psychology Research Laboratory, Psychology Department, University of Waikato, Hamilton.

We investigated clinical interpretations by mental health professionals of "Magical Ideation" (i.e., statements and beliefs thought to be odd, bizarre, or to have little validity in our predominantly Western culture), and to examine how typical individuals judge the significance of these beliefs.

A version of the Magical Ideation Scale (Eckblad & Chapman, 1983) was used. Instead of the conventional administration, in which respondents endorse items as applying to themselves, undergraduate students in psychology (N=60) were asked to judge whether the statements, if expressed by someone else, might be indicative of having a mental illness. Subsequently, new items were added to the scale. These represented either spiritual beliefs that are especially common in Maori culture, or common "urban myths" that appear to be held by significant numbers of ordinary people. The revised scale was analysed qualitatively by 3 focus groups: clinical psychology trainees (N=7), community-based mental health support staff (N=3), and clinical-level mental health practitioners (N=5). Participants in the latter 2 groups were predominantly Maori. Transcribed data from the focus groups were analysed qualitatively using a general inductive approach, derived from grounded theory.

The presence of the "lie" items in the original scale complicated the judgement task, nevertheless typical judges tended to rate many items from the Magical Ideation Scale as unlikely to be indicative of mental illness, with Maori participants tending to be more accepting of some items in the scale. The focus groups expanded on the importance of spiritual and cultural beliefs needing to be taken seriously, however identified certain criteria whereby a belief could be judged as typical within a religious or cultural context as opposed to reflecting some degree of thought disorder.

The Magical Ideation Scale, in its present form, is unsuitable as an assessment tool for thought disorder or proneness to schizophrenia in contemporary New Zealand. Non-scientifically based beliefs are common in the general population. Spiritual beliefs are probably not often confused with schizophrenic thought disorder when assessed by clinicians. However, their frequency within contemporary Maori and New Zealand culture overall, means that diagnostic judgements in psychiatry are viewed with some suspicion even by experienced mental health professionals.