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EDITORIALS

Endarterectomy for symptomatic carotid stenosis: some of the lessons learned so far

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In response to mounting concerns in the 1980's that the efficacy of carotid endarterectomy had not been conclusively proved,[1] two multinational trials of endarterectomy in symptomatic patients were mounted – the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and the European Carotid Surgery Trial (ECST). These landmark studies were the largest controlled trials ever undertaken of a surgical procedure and, collectively, involved over 200 participating centres from 16 countries. The initial reports from each trial were published within months of each other in 1991[2,3] and had an immediate influence on clinical practice.[4-6] Final reports from each trial have been published recently.[7,8] In the seven years between, however, there have appeared a series of supplementary publications with additional data analyses from each trial which has allowed us to answer many of the questions that have previously surrounded the optimal treatment of symptomatic carotid stenosis.

Does carotid endarterectomy reduce the risk of stroke in symptomatic patients?

Both trials emphatically confirm that in skilled surgical hands the procedure reduces the risk of stroke in symptomatic patients with high grade carotid stenosis. In patients with stenosis levels equivalent to 70% or more (as measured by the NASCET method vide infra) endarterectomy results in an approximately 65% relative risk reduction for stroke in the next two years.[3,7-9] For patients with low grade stenosis the incidence of stroke in patients treated medically is sufficiently low that the risks of surgery are not justified. In those with moderate stenosis the benefits from surgery are marginal. It is hoped that more detailed risk factor analysis might allow the development of a predictive tool capable of determining, in an individual patient, what the risk:benefit balance is likely to be, and which would permit more precise targeting of surgery to those high grade stenosis patients who most need it.[7,10]

How long does the benefit last?

The stroke reduction that results from surgery is limited to the first two to three years following treatment. This is because the risk of stroke in patients treated medically in both trials fell significantly two to three years after the initial ischaemic event and, thereafter, was comparable to that of the surgically treated patients.[7,8]

What are the risks of surgery?

The combined risk of stroke (in any vessel territory) or death within 30 days of surgery was approximately seven percent.[7,8] Extrapolation from these trials to practice in a non-trial environment is subject to the operative mortality/morbidity experience of an individual surgeon being at around this same level. Indeed, if a surgeon's morbidity/mortality figure exceeds that in the trials, any benefits may be nullified and the patient might more appropriately be treated medically. This has led to a call for individual surgeons' operative experience to be audited and known to those who refer patients for treatment.[11,12] Without this knowledge, it may not be appropriate to extrapolate this trial experience into local clinical practice.

Is the method of measuring carotid stenosis important?

The major methodological difference between the two trials was in the technique used to quantify the degree of stenosis present on carotid angiography. The European trial determined the point of maximum stenosis on angiography relative to the estimated normal diameter. The North American method compared stenosis relative to the diameter of the distal vessel. This difference in measurement method results in absolute differences that are quite significant. For example, stenoses measured by the NASCET method of 70% and 50% are equivalent to ECST stenoses of 85% and 75% respectively.[13] It is essential, therefore, when determining best therapy for an individual patient based on one or other of these trials, that the clinician ensures the stenosis measurement method used is the same as that used in the reference trial.

Is the morphology of the plaque on angiography of prognostic significance?

A study of all 659 angiograms showing >70% stenosis from the NASCET suggested that the presence of plaque ulceration, as defined angiographically, indicated a higher risk of stroke, and the greater the severity of stenosis, the more significant a risk factor plaque ulceration became.[14] The ability of angiography to accurately define ulceration, however, has been questioned. When 500 endarterectomy specimens from the NASCET were compared with the pre-operative angiograms, the specificity and sensitivity of angiography in predicting ulceration was only 74% and 46% respectively.[15] Notwithstanding this, it appears that there are characteristics of plaque morphology seen on angiography – in addition to the severity of stenosis - that predict a patient who is at higher risk of stroke.

Are the risks of stroke and of surgery altered when the symptomatic carotid stenosis is so severe that the vessel is almost occluded?

The risk of stroke in medically treated patients in the NASCET showed close correlation with the severity of stenosis, and was maximal at levels of around 90-95% stenosis. With even greater narrowing resulting in near-occlusion of

the vessel, the stroke risks did not significantly increase further.[16] Nor was the risk of surgery greater in these patients. The evidence suggests therefore, that it is worth considering surgery for patients with near occlusion of the carotid artery.

In patients who have sustained a non-disabling carotid territory stroke, should surgery be delayed?

The optimal timing of surgery following a non-disabling stroke has been a matter of uncertainty, with concern that early endarterectomy, before a recent infarct has had the opportunity to mature, might risk haemorrhagic conversion of the infarct.[17] In the North American study, however, the surgical risks for those patients operated on within 30 days of their infarct (median 16 days)[18] were no different to those whose surgery was deferred for a month.[19] This suggests that patients who have sustained a non-disabling stoke can be treated in the same way as those presenting with transient ischaemic symptoms, rather than delaying surgery and exposing them to the risk of further stroke.

Is the risk of stroke influenced by whether the presenting symptoms are of retinal, rather than hemispheric, ischaemia?

The stroke risk in patients with high grade stenosis who present with amaurosis fugax or retinal infarction is less than half that of patients presenting with hemispheric ischaemic symptoms.[20] This difference in the natural history is therefore important when planning optimal treatment for patients whose presenting ischaemic symptoms are ocular rather than hemispheric.

Conclusions

I have presented but a glimpse of the wealth of clinically relevant information provided by these two trials. Additional issues that each has addressed include: the diagnostic value of the carotid bruit;[21] the relationship of stenosis severity to radiological findings of white matter change[22] or of borderzone infarction;[23] and the risks from contralateral carotid disease,[24,25] from additional distal disease[26] or from finding infarction on CT.[27] The data derived from these landmark trials will undoubtedly continue to have a significant impact in the coming years on the management of symptomatic carotid stenosis.

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Type 2 diabetes and tight blood pressure control: how low to go?

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Several recent publications, most notably the United Kingdom Prospective Diabetes Study (UKPDS) and Hypertension Optimal Treatment (HOT) study, have re-focused clinical attention on the management of hypertension in patients with type 2 diabetes.[1,2] Hypertension is common in type 2 diabetes occurring in up to 60% of patients.[1,3] The UKPDS, HOT and other randomised controlled trials show a conclusive reduction in the risk of stroke, cardiovascular events and diabetic retinopathy, with tight blood pressure control.[1,2,4] The angiotensin converting enzyme inhibitor (ACE-I) ramipril had a vasculoprotective and renoprotective effect in diabetic subjects, with a baseline (pre-treatment) blood pressure of 141/80 mmHg.[5] Other studies show a reduction in the rate of progression of nephropathy, following treatment of hypertension in patients with established microalbuminuria.[4] Tight control of hypertension also reduces the risk of heart failure in diabetes.[1] Both the UKPDS and HOT studies failed to demonstrate a blood pressure level below which a further reduction does not confer additional benefit, thus a threshold blood pressure target has yet to be defined. The UKPDS also demonstrates that it is easier to achieve good blood pressure control than it is to achieve good glycaemic control, and that the benefits of tight blood pressure control are manifest sooner than those of tight glycaemic control. This is especially important for older diabetic patients, as

their limited life expectancy means that they may not have sufficient exposure to hyperglycaemia to place them at risk of developing clinically significant microvascular complications, but they nevertheless have a 2 to 3 fold increased risk of myocardial infarction and stroke.[3,4] Polypharmacy, with three or more antihypertensive agents, may be needed to achieve adequate control of hypertension.[1]

In unselected patients with type 2 diabetes, blood pressure reduction *per se* appears to be more important than the type of antihypertensive drug treatment used.[3] There is no agreed "best" anti-hypertensive agent or combination of agents.[4] The ACE-Is are often recommended as the first line agent, with some, but not all studies showing a reduction in the number of cardiac events, compared to other agents with a similar blood pressure lowering effect.[3,4,6] Angiotensin II antagonists have recently become more readily available in New Zealand and preliminary studies are demonstrating favourable results.[4] The UKPDS showed that the beta blocker atenolol was as efficacious as the ACE-I captopril with regard to reducing cardiovascular endpoints.[7] Beta-blockers also confer an advantage in survival post myocardial infarction, in both the diabetic as well as non-diabetic populations.[8] Calcium channel blockers and alpha blockers also have a role, either as monotherapy or in combination with other treatment.[4] High dose thiazide diuretics should be avoided because of their adverse metabolic effects. Some, but not all studies suggest that low dose thiazide therapy also has detrimental metabolic effects.[4,6,9]

Tight control of hypertension in type 2 diabetes confers benefits which are both clinically and economically worthwhile. For example, the tight blood pressure control arm of the UKPDS achieved a mean blood pressure of 144/82 mmHg compared to a mean blood pressure of 154/87 mmHg in the group allocated to less tight control. Comparing the tight with the less tight arm of the study, the number needed to treat, to prevent a death from a diabetes related cause, was fifteen.[1] The pharmaco-economic analysis of the UKPDS shows that the overall costs to a health provider, required to achieve a mean blood pressure 144/82 mmHg is minimal, because of the associated reduction in costs of managing both the macrovascular and microvascular complications.[10] In the HOT study, diabetic patients achieving a diastolic pressure of less than or equal to 80 mmHg had a 51% reduction in major cardiovascular events, in comparison with patients who achieved a diastolic blood pressure of 90 mmHg.[2]

Clearly, the message here is that we should be striving to achieve tight blood pressure control in diabetic patients. Traditionally, hypertension in diabetes has been defined as a blood pressure of 140/90 mmHg or above.[11] Revised definitions of hypertension in type 2 diabetes which incorporate findings from more recent studies have yet to be made by most diabetes organisations and definitions are likely to require further modification following publication of results from studies in progress. With regards to the treatment targets, the World Health Organisation, the International Society of Hypertension and the American Diabetes Association have recommended a target blood pressure below 130/85 mmHg in diabetes.[11,12] Lower blood pressure treatment targets are recommended for patients who have established end-organ damage or who are at especially high risk of end-organ failure. There will, however, be a blood pressure threshold below which any additional risk reduction benefits become minimal and are outweighed by drug side effects and economic costs. This threshold level has yet to be defined.

How well are we doing in New Zealand?

A Christchurch primary care diabetes audit documented a mean blood pressure of 140/81 mmHg in a group of patients with predominantly type 2 diabetes. Forty seven percent of these patients were prescribed medications for hypertension.[13] More recently, the Otago Diabetes Team Project have documented a mean blood pressure of 141/79 mmHg in a similar group of patients recruited from 90 general practices in Otago during 1998 (K Coppell, personal communication). Although this looks superficially reassuring, it implies that around half the hypertensive patients have a blood pressure above the conservative target of 130/85 mmHg. Whilst clinicians await results from ongoing trials in type 2 diabetes which may help define exact blood pressure treatment targets, there is clearly much work that can be done in the interim with the aim of achieving the current recommended blood pressure treatment target of 130/85 mmHg or below.

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Third sector primary health care in New Zealand

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Abstract

Aims. To describe key organisational characteristics of selected third sector (non-profit and non-government) primary health care organisations.

Methods. Data were collected, in 1997 and 1998, from 15 third sector primary care organisations that were members of a network of third sector primary care providers, Health Care Aotearoa (HCA). Data were collected by face-to-face interviews of managers and key informants using a semi-structured interview schedule, and from practice computer information systems.

Results. Overall the populations served were young: only 4% of patients were aged 65 years or older, and the ethnicity profile was highly atypical, with 21.8% European, 36% Maori, 22.7% Pacific Island, 12% other, and 7.5% not stated. Community services card holding rates were higher than recorded in other studies, and registered patients tended to live in highly deprived areas. HCA organisations had high patient to doctor ratios, in general over 2000:1, and there were significant differences in management structures between HCA practices and more traditional general practice.

Conclusions. Third sector organisations provide services for populations that are disadvantaged in many respects. It is likely that New Zealand will continue to develop a diverse range of primary care organisational arrangements. Effort is now required to measure quality and effectiveness of services provided by different primary care organisations serving comparable populations.

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Historically, primary medical services in New Zealand have been provided predominantly by general practitioners (GPs), with private business approaches to funding for GP and staff reimbursement, and fee-for-service charging and subsidy arrangements.[1] Over the last ten years an increasing number of primary care providers have developed a different approach to both organisation and funding. The term third sector has been adopted to describe organisations which regard themselves as being non-government and non-profit, and usually employ salaried GPs. This third organisational form for New Zealand general practice thus differs from the private business general practice model and the publicly owned special medical area model.[2]

Third sector primary care organisations started having a significant presence in the late 1980s. The first union health centres were set up in 1987 in South Auckland and in Newtown, Wellington. Subsequently six more union health centres were established in Otara, West Auckland, Hamilton, Christchurch, Porirua and Lower Hutt. These centres were supported by successive Ministers of Health, and a diverse range of third sector primary health care providers emerged during the early and mid 1990s, most notably iwi based initiatives, for example Raukura Hauora O Tainui Trust.[3] Although concern was expressed regarding the cost and justification for the South Auckland and Newtown centres,[4] the Ministry of Health actively promoted these initiatives especially in terms of funding mechanisms, perceiving them to be a way of providing health care to vulnerable communities.[5] Community trusts and iwi owned health services were also seen by government, Maori groups and communities as worthwhile alternatives to traditional primary care arrangements.[6]

In 1993 the union health centres formed a national office, consisting of a GP based in Wellington and a financial advisor based in Auckland. The function of the national office was to provide strategic management and co-ordination for the eight union health centres. In 1994 the union health centres national office reconstituted itself as a national network of non-profit primary care providers called Health Care Aotearoa (HCA). HCA aimed to provide a support network for its member organisations. HCA members are community managed organisations placing strong emphasis on providing services for low-income populations, and on working bi-culturally. They are referred to as community managed because they are governed, in principle at least, by patients and consumers.[7] In 1997 HCA members included 15 comprehensive primary health care organisations, and several specialist organisations such as mental health and alcohol and drug services. By 1999 there were 33 member and associate member organisations in the HCA network, 19 of which provided comprehensive primary health care services (personal communication, P Glensor, National Co-ordinator, HCA).

Third sector primary care organisations have developed in New Zealand for a variety of reasons, some of which can be identified from the scant literature on this subject. Matheson[8] identified two motivations: first as a response to financial barriers to access for primary health care services, especially amongst low income populations, and second in response to a desire of iwi and consumer groups to exercise more control over primary health care services. HCA organisations have also developed in areas where particular populations have found it hard to obtain primary care services. These include, for example, refugee populations and populations containing large numbers of patients with severe and enduring mental illness.

To date there have been few studies of third sector primary health care organisations in New Zealand. In addition, most evaluations of primary care organisational structures have been carried out on one or two organisations (for example McGrath[9] and Flight).[10] This paper reports selected results from a study of 15 third sector primary health care organisations, and aims to describe their key organisational characteristics.

Methods

Data collection. Data were collected from 15 third sector primary care organisations that were members of the HCA network. There were two primary means of data collection. First face-to-face interviews of managers and other key informants were carried out using a semi-structured interview schedule (available from the authors on request). Second, information from practice computer information systems was down-loaded. All data were gathered in the second half of 1997, and the first half of 1998. Information was collected on a range of organisational features, including history, legal structure, management structure, community participation in governance, services provided, mix of personnel, appointments systems, information systems, patient recruitment, after hours arrangements, geographic location,

quality management, user charges, salaries and financial data. Information down-loaded from computer information systems included age-sex registers (registered patients only), utilisation data, and disease register data. Multi-centre ethics committee approval was obtained for the study (North Health, Tairawhiti, Taranaki, Manawatu-Whanganui, Wellington, Christchurch).

Organisations included in the study. Primary health care organisations included in the study were: Hokianga Health Enterprise Trust, Northland; Community Medical Centre Trust, Auckland; Waitakere Union Health Centre, Auckland; Otara Union Medical Centre, Auckland; Otahuhu Union Health Centre, Auckland; Trust Health Care Manurewa, Auckland; Ngati Whatua O Orakei Health Clinic, Auckland; Ngati Porou Hauora, Te Puia Springs; Te Waipuna Health Centre, Wanganui; Ruanui Health Centre, Hawera; Ora Toa Medical Centre, Porirua; Porirua Union and Community Health Service, Porirua; Hutt Union and Community Health Service, Lower Hutt; Newtown Union Health Service, Wellington; Christchurch Union and Community Health Service, Christchurch.

Measurement of ethnicity. Ethnicity codes were recoded to the following five groups: European, Maori, Pacific Island, other, not-stated. The Statistics New Zealand hierarchical ethnicity coding method was used as the basis for classifying Maori and Pacific Island ethnicity.[11]

Measurement of community services card and high use health card holding rates. Community services card (CSC) and high use health card (HUHC) holding was measured by counting all patients who had a CSC or HUHC number recorded. Expiry dates were not taken into account.

Measuring deprivation of registered patient populations. Deprivation is strongly associated with health status[12,13] *NZDep96* is an area-based measure of deprivation,[14] combining nine variables from the 1996 census that reflect eight dimensions of deprivation.[15] These are the proportion of people with no access to a telephone; aged 18-59 receiving a means tested benefit; aged 18-59 unemployed; living in households with equivalised income below an income threshold; with no access to a car; aged <60 living in a single parent family; aged 18-59 without any qualifications; not living in own home; living in households below equivalised bedroom occupancy threshold. Equivalisation refers to methods used to control for family composition. *NZDep96* provides a deprivation score for each meshblock in New Zealand. Meshblocks are geographical units defined by Statistics New Zealand, containing a median of 90 people. The *NZDep96* scale runs from 1 to 10, so that a value of 10 indicates that the meshblock is in the most deprived 10 percent of areas in New Zealand. For measuring deprivation of area of residence of registered patients the following process was carried out: 1) patient age-sex registers were obtained; 2) registers were "cleaned" (addresses were organised in a standard format); 3) addresses were geocoded (assigned a meshblock number); 4) the *NZDep96* deprivation scale was assigned to each geocoded address; 5) the percentage of patients in each deprivation level was calculated.

Results

Table 1 summarises the age and ethnicity profile of 14 registered practice populations (register data were not available from Ora Toa Medical Centre). Overall the populations served were young, only 4.03% of patients being aged 65 years or older compared with 11.68% of the New Zealand population.[16] Ethnicity records were generally of high quality and complete (92.4% of patients had ethnicity recorded). The ethnicity profile of the registered practice populations was highly atypical for New Zealand, with 21.8% European, 36% Maori, 22.7% Pacific Island and 12% other. The young and largely non-European population is characteristic of the lowest income groups in New Zealand.[17,18]

Table 1. Age and ethnicity* profiles of combined registered practice populations.

Age and ethnicity	Percent of registered patients
Age not stated	0.2
0-5 years	12.2
6-17 years	22.6
18-34 years	31.9
35-64 years	29.0
65+ years	4.03
With ethnicity recorded	92.4
Maori	36.0
European	21.8
Pacific Island	22.7
Other	12.0

*Ethnicity defined according to hierarchical classification method.[11] Hokianga Health Enterprise Trust coded Europeans as "Other".

Figure 1 shows the deprivation scores for 14 organisations combined. A total of 59 299 patient addresses were geocoded (90% of the total not including Ngati Porou Hauora and Hokianga). Due to difficulties geocoding rural addresses, and the geographically well demarcated areas served by Ngati Porou Hauora and Hokianga Health Enterprise Trust, deprivation scores for these two organisations were calculated using census tract data rather than practice registers. Of the 1312 addresses for Ruanui Health Centre, only 1131 (86.2%) lay within the town area and were therefore potentially geocodable. Of these potentially geocodable addresses 81.6% were geocoded. Overall, registered patients tended to live in highly deprived areas (compared with an "average" New Zealand population that has roughly equal numbers of people living in areas at each level of deprivation).

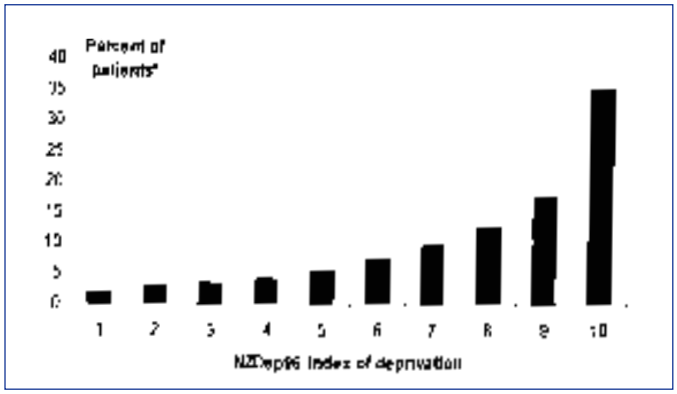

Figure 1. Deprivation profile of 14 Health Care Aotearoa practice populations. *Percentage of patients with geocoded addresses.

Figure 2 shows the percent of patients holding a CSC at each level of deprivation. Several possible "natural" cut-off points exist for *NZDep96* when comparing the scale with CSC holding. Specifically, cut-off points were identified between *NZDep96* categories 2 and 3, 6 and 7, 7 and 8 and 9 and 10. The complexity of this relationship is not surprising considering that deprivation cannot be expected to conform to the dichotomous pattern implied by CSCs.

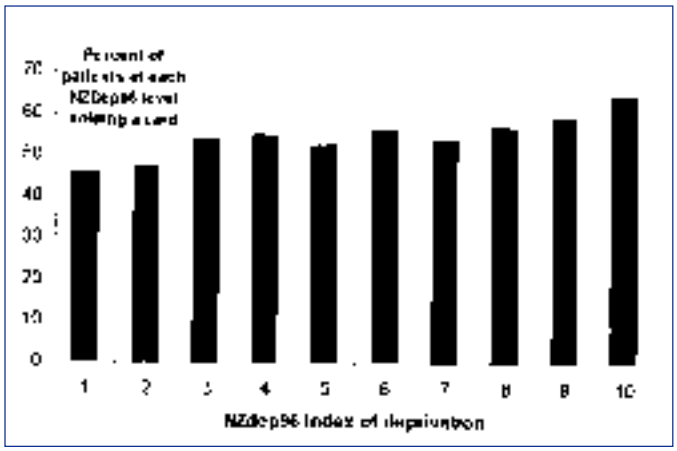

Figure 2. Percent of patients holding a CSC at each NZDep96 level. CSC = community services card.

Table 2 shows CSC and HUHC holding rates.

Legal and management structures. Legal structures varied, and included incorporated societies (8), charitable trusts (3), and limited liability companies (4). In most cases patients and community members were represented on the board of management. The total number of committee members ranged from 5 to 30 people, with average 10. The number of community representatives on the committee

ranged from 0 to 30 people, with average 8. The number of staff representatives on the committee ranged from 0 to 5 people, with average 2. Community representatives represented trade unions, iwi or hapu groups, community organisations or other primary care providers. Patients were represented on 12 of the 15 management committees by committee members who were also patients. In three organisations all members of the management committee were registered patients.

Table 2. Recorded CSC* and HUHC* holding rates of registered practice populations.

Organisation	Percent of registered patients CSC	HUHC
Hutt Union and Community Health Service	75.0	4.3
Ruanui Health Centre	74.3	0.0
Trust Health Care Manurewa	71.4	0.4
Te Waipuna Health Centre	67.0	0.9
Community Medical Centre Trust	66.6	1.1
Newtown Union Health Service	65.2	3.1
Porirua Union and Community Health Service	63.4	2.0
Ngati Porou Hauora	56.9	2.4
Orakei Health Clinic	49.1	0.3
Otahuhu Union Health Centre	49.0	3.1
Christchurch Union and Community Health Centre	46.6	1.4
Otara Union Medical Centre	45.4	1.7
Waitakere Union Health Centre	43.6	1.8
Hokianga Health Enterprise Trust†		

*Holding rates as claimed for capitation funding purposes. Data not available for Ora Toa Medical Centre †Hokianga Health Enterprise Trust did not record community services card (CSC) and high user health card (HUHC).

Management committee election processes were formal in nine health centres, and evenly divided between mixed formal/informal (3) or informal (3) in the remainder. Formality means elections for the management committee were held on a regular basis (usually annually) and committee representatives were nominated rather than appointed.

All staff were accountable to the governing body, mostly via the manager, with one exception where lines of accountability were not clear. Each organisation had a manager (from a non-medical background) who made day-to-day decisions, and was responsible to the management committee. Most managers had spending authority within agreed budgets.

Staff were represented on the management committee in two thirds of the organisations. The majority of organisations had staff meetings where both clinical and financial management issues were discussed, and decisions were either made at this level or taken to the management committee. In one organisation professional staff managed their own budget for their part of the service. In four organisations clinical staff had little or no input into financial management.

Table 3 shows staff numbers for each organisation (full time equivalent (FTE) corresponds to a 37.5-40.0 hour week). All organisations employed their key management and clinical staff, except Ngati Ruanui Health Centre where the doctors were self-employed. A number of additional staff, such as rongoa and dental staff, are not included in Table 3. Table 4 shows the range of services provided. A number of additional services, such as mirimiri and rongoa services, are not included in Table 4.

Discussion

This paper describes key organisational features of selected third sector primary care organisations. The populations served were highly atypical compared to the New Zealand average, being young and largely non-European - factors characteristic of low income groups. Overall CSC holding rates were higher than recorded in other general practice studies,[19] and deprivation distributions were highly skewed. There is thus a consistent picture of third sector organisations serving vulnerable patient groups. Support for this finding is provided by the identification of "specialist" services offered by some of the organisations to vulnerable groups such as refugees and substance dependent patients.

The pattern of care offered by third sector organisations may differ from other general practice services in a number of ways. In New Zealand the geographical distribution of GPs varies considerably from 1070 to 1916 patients per GP.[20] By comparison, HCA organisations had high patient to doctor ratios, in general over 2000 (Table 3). There are several possible reasons that may account for these high ratios, including the expanded role of nurses, service patterns and incentive structures inherent in capitation funded practices (which tend towards lower overall utilisation[21,22]), patient registration with multiple providers, underestimation of actual full time equivalent doctors, and possible underservicing of low income populations. There is a lack of New Zealand research regarding the impact of different patient: doctor ratios on clinical care. Further research with HCA providers to assess service quality in relation to patient doctor ratios would be helpful.

This study highlights two other issues regarding general practice and health services research in New Zealand. The first concerns the usefulness of CSC holding rates as a measure of need, and the second the difficulty in comparing the structure and functioning of New Zealand primary care services given the sparse information available.

Registered patients living in the least deprived areas had a CSC holding rate of 46%. Conversely, registered patients living in the most deprived areas had a CSC holding rate of 64%. These results suggest that CSC holding rates (as recorded in patient registers) in low-income populations do not strongly differentiate between widely different deprivation levels. This lack of differentiation may be accounted for in at least three ways. First, many needy families may live in relatively non-deprived areas, and many non-deprived families may live in deprived areas. Second, the lack of differentiation between areas may indicate that CSCs fail to differentiate between differing levels of deprivation (and by extension, need). Third, the failure of CSCs to differentiate between levels of deprivation may be due to the previously documented non-uptake of cards or failure of organisations to record current CSC status. The Ministry of Health estimates that about 75% of those eligible to hold a CSC actually hold a card (personal communication, Grant Johnston, Ministry of Health). Similarly, in a retrospective study of GP records carried out in 1993/94, Gribben estimated the CSC uptake rate was 77%.[19] Parks carried out a survey of 508 patients (or their accompanying relative) presenting at their doctor in 1995.[23] She found that 28% of non-CSC holders interviewed were eligible for a CSC but did not have one. Pakeha were significantly more likely to have CSCs if eligible than other ethnic groups. Pacific Island people were significantly less likely to have CSCs if eligible than other ethnic groups. One means of exploring the capacity of CSCs to differentiate between levels of need, would be to calculate CSC eligibility rates by *NZDep96* level by carrying out a formal survey.

The *NZDep96* analyses were carried out on the subset of records where *NZDep96* values were assigned (ie had been geocoded). There is possible bias in the results if the distributions of CSC and *NZDep96* values for the records

Table 3. Staff numbers, patient: doctor ratios and patient: nurse ratios.*

Organisation	Numbers of full time equivalent staff							Patient: doctor and patient: nurse ratios	
	Manager	Reception	Administration	Doctors	Nurses	Community Workers	Midwives	Patient:doctor ratio	Patient:nurse ratio
Hokianga Health Enterprise Trust	5.0	0	10.6	4.6	25.6	3.5	0.5	2220	340
Orakei Health Clinic	0.5	1	0	1	2.5	1.2	0	1596	638
Otahuhu Union Health Centre	0.6	2.9	0	3.2	2.9	0	0	2657	2983
Otara Union Medical Centre	0.8	1	0	2.5	2	0	0	2761	3452
Waitakere Union Health Centre	0.6	1	1	3	2.6	0	0	2271	2620
Community Medical Centre Trust	0.8	1	0.4	2.3	2	0	0	2443	2809
Trust Health Care Manurewa†	0.9	2	0	4	2.6	0	0		
Ngati Porou Hauora	3	3	1.7	1.5	1.9	7	2	2387	1885
Ruanui Health Centre	1.3	0.2	0	0.5	1	0.3	0	2624	1312
Te Waipuna Health Centre	1	1	1	2.5	2	0	3	2787	3484
Newtown Union Health Service	1	2.8	0.5	4.2	5.4	1.2	2.8	1859	1446
Porirua Union and Community Health Service	0.8	2.5	1	2.6	2.4	0	1	2352	2600
Hutt Union and Community Health Service	1	2	0.5	2.6	2	0.5	3	2214	2912
Christchurch Union and Community Health Centre	0.8	1	0.5	2.7	3	1	0	1887	1699

*Data not available for Ora Toa Medical Centre. †It was not possible to calculate patient doctor/nurse ratios for Trust Health Care Manurewa as they care for two separate practice populations.

Table 4. Range of services provided.

Organisation	GP	Nurse practitioner*	Maternity†	Mental Health‡	Health promotion§	Counselling‖	Community worker	Suit case clinics¶	Own after-hours**
Hokianga Health Enterprises Trust	Yes	Yes	Yes	Yes	Yes	No	Yes	No	Yes
Orakei Health Clinic	Yes	Yes	No	Yes	Yes	Yes	Yes	No	No
Otahuhu Union Health Centre	Yes	Yes	Yes	No	No	No	No	No	No
Otara Union Medical Centre	Yes	Yes	No	No	Yes	Yes	No	No	No
Waitakere Union Health Centre	Yes	Yes	No	No	No	No	No	No	No
Community Medical Centre Trust	Yes	No	No	No	No	No	No	No	No
Trust Health Care Manurewa	Yes	Yes	Yes	No	No	No	No	No	No
Ngati Porou Hauora	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Ruanui Health Centre	Yes	Yes	No	Yes	Yes	Yes	Yes	No	Yes
Te Waipuna Health Centre	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No
Newtown Union Health Service	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes
Porirua Union and Community Health Service	Yes	Yes	Yes	No	Yes	Yes	No	No	No
Hutt Union and Community Health Service	Yes	Yes	Yes	No	Yes	No	Yes	Yes	No
Christchurch Union and Community Health Centre	Yes	Yes	No	No	Yes	Yes	Yes	Yes	No

Data not available for Ora Toa Medical Centre; *Nurse practitioner refers to nurses who control nursing services and are responsible for independent case loads; †Maternity services refers to antenatal, intrapartum and postpartum care; ‡Mental health services refers to some form of specialised mental health service provision; §Health Promotion refers to services provided largely outside normal clinical consultations, including group programmes (for example, diabetes education) and individual programmes (for example smoking cessation counselling);‖Counselling refers to specialist counselling services; ¶Suit-case clinics refer to clinical services offered regularly off-site (for example, in a community hall); **Own after-hours refers to organisations that provided their own on-site after hours service.

which could not be geocoded were substantially different from the distributions for records which could be geocoded. There is no means of judging the magnitude or direction of these possible biases, but we see no reason to suppose that the deprivation or CSC status of the addresses that could not be geocoded differed overall from those that could.

General practice services in New Zealand have traditionally been delivered by doctors working in small and often isolated groups. It has been difficult to obtain accurate information from general practice, with data generated largely from cross sectional studies such as the WaiMedCa study[24] or from networks of practices such as that of the Royal New Zealand College of General Practitioners network co-ordinated from Otago.[25] The findings from this study would be placed in a more complete context if there were comparable data from other general practice sources. There are significant differences in management structures between HCA practices and more traditional general practice, with non-clinical staff having a greater say in health care policy. There is also a greater dependence on non-medical clinical staff with, for example, extended roles for nursing staff. More detailed comparison between different types of primary care providers will hopefully be possible in the future with the co-ordination of general practice infrastructure provided by the advent of independent practitioner associations (IPAs).[26]

Third sector organisations have as their main aim the provision of care to vulnerable populations. It is not clear to what degree, and in what ways New Zealand general practice as a whole provides services to vulnerable populations. It is clear GPs in non-HCA settings exercise discretion in charging policies and that these policies have changed over time. In 1989 Dovey found that no fee was charged to the patient in 22.5% of cases.[27] By 1993, although 19.4% of consultations generated no fee to the patient, the proportion of cases in which a less than normal fee was charged had risen 7.9 times from 3.5% in 1989 to 27.5%.[28] Malcolm highlighted the gross underutilisation of and expenditure on primary medical care and related services to Maori and other New Zealanders in poor circumstances.[29] These issues are clearly important to HCA organisations which have developed funding streams and contracting opportunities which take account of the populations they serve.

It seems likely that, for a number of reasons, New Zealand will continue to develop a diverse range of primary care organisational arrangements. There is the need to deliver culturally appropriate services for Maori and other ethnic groups. Recent developments such as the emergence of IPAs and urgent medical centres have encouraged plurality in primary care organisation. There has also perhaps been a lack of clarity as to a government vision for primary care and general practice. This study shows that third sector organisations are providing services for vulnerable populations. Considerable effort is now required to measure quality and effectiveness of services provided by primary care organisations serving comparable populations.

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Nosocomial blood stream infection in Auckland Healthcare hospitals

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Abstract

Aim. To report the epidemiology of nosocomial bloodstream infections in Auckland Healthcare Hospitals.

Methods. From January 1995 to December 1997 every positive blood culture result was followed up by an infection control nurse who recorded relevant clinical, laboratory and treatment information on a data collection sheet. The clinical significance of each isolate was determined and the most likely source recorded.

Results. During the three year study period, there were 1046 nosocomial blood stream infections yielding 1147 isolates. The most common isolates/groups were: coagulase negative staphylococci 19%, *S. aureus* 18%, *E. coli* 12%, streptococci 10%, other *Enterobacteriaceae* 10%, *Enterobacter* spp. 7%, *Pseudomonas* spp. 5%, anaerobes 2%, and yeasts 4%. The most common sources were: intravascular lines 40%, urinary tract 8%, skin/soft tissue 8%, gastrointestinal 7%, and unknown 25%. The overall results were strongly influenced by the neonatal intensive care unit at National Women's Hospital where 58% of blood stream infections had intravascular-lines as the source and 53% of the isolates were coagulase negative staphylococci. The overall blood stream infection rate was approximately 6/1000 admissions. Rates per 1000 inpatient days for haematology, intensive care, oncology, neonatal and all other patients were 13, 11, 3, 3 and 1 respectively.

Conclusions. Surveillance data that are clinically relevant are useful in identifying areas where infection prevention strategies can be implemented. Because of the importance of lines as a source of nosocomial blood stream infections all aspects of line care are being reviewed with the aim of reducing these devices as a source of blood stream infection.

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Blood stream infections (BSI) comprise approximately 20% of nosocomial infections.[1] It is estimated that up to 35% of nosocomial BSI can be prevented if an effective surveillance programme is in place.[2] Surveillance of BSI is recommended because of the associated morbidity and attributable mortality.[3] BSIs prolong patient hospitalisation by 7-10 days[3] and are therefore costly both to the healthcare organisation and the patient.

The Crown Company Monitoring Advisory Unit (CCMAU) monitor, for the New Zealand Government, public hospitals to ensure they provide quality health care. BSIs were chosen by CCMAU as a performance indicator for a number of reasons, one of which was to ensure that some infection control surveillance was being undertaken. Hospital and Health Services (HHS) are now required to report each month the BSI rate per 1000 inpatient admissions to CCMAU. The first two years of

data have recently been published.[4] The average rate of BSI for the six tertiary HHS's was 3.7 per 1000 inpatient admissions.[4] While this gives an indication of how frequent BSI's are, it does not provide information on the local epidemiology of nosocomial BSI, ie. organisms encountered, their sources and the different rates for different clinical services. We, therefore, have collected other relevant information in order to guide initiatives to reduce nosocomial BSI's.

Methods

All positive blood cultures at Auckland Healthcare hospitals between January 1995 and December 1997 were reviewed. Auckland Healthcare is New Zealand's largest HHS. It undertakes almost 40% of tertiary care services in the country and provides hospital services for 40% of the country's population. Three of the four hospitals in the Auckland Healthcare HHS were included in this study. Auckland Hospital with 576 beds is a tertiary teaching hospital affiliated with Auckland University Medical School which provides acute and elective general medical and surgical services, as well as major haematology, oncology, trauma and neurosurgical services. Green Lane Hospital with 219 beds provides tertiary cardiology, cardiothoracic surgery, respiratory medicine and otorhinolaryngology services. National Women's Hospital with 256 beds provides obstetric and gynaecology services including a neonatal intensive care unit with 16 beds. Data were collected and recorded by infection control nurse practitioners for all patients with positive blood cultures during the three year period. The significance and probable source for each isolate were determined after reviewing all relevant clinical and microbiological data with the clinical microbiologist.

A nosocomial BSI was defined as a positive blood culture obtained 48 hours or more after admission to hospital, provided there was no evidence that the BSI was due to an infection present or incubating at the time of admission, unless related to a previous admission. Common skin contaminants (eg. *Corynebacterium* spp., *Bacillus* spp. and coagulase-negative staphylococci [CNS]) were included only if isolated from two independently collected blood culture sets unless, on review, single positive cultures were thought to be clinically relevant.[4] BSI's detected by blood cultures obtained within 48 hours of admission were counted as nosocomial if they were clearly related to a previous admission, for example in a cancer patient with a long-term central line, or *S. aureus* bacteraemia due to a surgical site infection.

Results

From January 1995 to December 1997 there were 1046 episodes of nosocomial BSIs. The most frequent source of infection was intravascular lines (40%), (Table 1). Central venous catheters predominated over peripheral lines. The proportion of BSI with intravascular lines as a source was significantly higher at National Women's Hospital than the other two hospitals (Table 2). The source of 25% of the BSIs could not be determined.

Table 1. Sources of nosocomial blood stream infection, 1995-7.

Source	Number (%)
Intravascular lines	415 (40%)
- central lines	366 (35%)
- peripheral lines	49 (5%)
Urinary tract	81 (8%)
Gastrointestinal tract	74 (7%)
Skin/soft tissue	85 (8%)
Respiratory tract	50 (5%)
Other	83 (8%)
Unknown	258 (25%)
Total	1046 (100%)

The 1046 BSIs yielded 1147 isolates with 95 (9%) being polymicrobial (89 with two isolates, 6 with 3 isolates). The predominant pathogens were CNS (19%), *Staphylococcus aureus* (18%) and *Escherichia coli* (12%) (Table 3). The overall number of CNS infections was strongly influenced by BSIs in the neonatal intensive care unit at National Women's Hospital where 58% had intravascular lines as the source and 53% of the isolates were CNS (Table 4). *S. aureus* caused 22% of intravascular line related BSIs, 79% of peripheral line BSIs, but only 9% of central line BSIs.

Table 2. Proportion of blood stream infections intravascular lines as the source, 1995-7.

	Source		
Hospital	Intravascular lines	Others	Total
1. Auckland	221 (33%)	451	672
2. Green Lane	77 (45%)	95	172
3. National Women's	117 (58%)	85	202
Total	415 (40%)	631	1046 (100%)

1 vs. 2 p = 0.005
1 vs. 3 p = <0.001
2 vs. 3 p = 0.01

Table 3. Organisms causing nosocomial blood stream infections, 1995-7.

Organism/group	n	(%)
Coagulase-negative Staphylococci	210	(19)
Staphylococcus aureus	205	(18)
Escherichia coli	144	(12)
Streptococcus spp.	118	(10)
Other *Enterobacteriaceae*	117	(10)
Enterobacter spp.	80	(7)
Pseudomonas spp.	62	(5)
Enterococcus spp.	51	(4)
Candida spp.	45	(4)
Anaerobes	19	(2)
Other gram-positives	40	(3)
Other gram-negatives	56	(5)
Total	1147	(100)

Table 4. Proportion of blood stream infections due to coagulase-negative staphylococci, 1995-7.

	Causative organism		
Hospital	CNS	Others	Total
1. Auckland	71 (10%)	661	732
2. Green Lane	25 (13%)	174	198
3. National Women's	114 (53%)	103	217
Total	210 (19%)	938	1147

1. vs. 2. p = 0.3,
1. vs. 3. p = <0.001,
2. vs. 3. p = <0.001.
CNS = coagulase negative *Staphylococci*.

Over three years the BSI rate for Auckland Healthcare Services increased from 4 to 7 per 1000 inpatient admissions. The overall rate for the three year period for each hospital was Auckland 8, Green Lane 6 and National Women's 5 per 1000 inpatient admissions. Sixty percent of the BSI's occurred at Auckland Hospital. Higher rates per 1000 inpatient days occurred in haematology 13, and intensive care unit patients 11, compared with 3 in oncology and neonatal and 1 for all other patients. Thirty two percent of the BSI's at Auckland Hospital occurred in haematology patients although they occupied only 4% of the bed days. BSI's in neonates made up 80% of all episodes at National Women's Hospital.

Discussion

This study provides clinically useful and epidemiologically relevant information beyond simply knowing the overall BSI rate. Varying definitions and populations studied make direct comparisons with other studies difficult. Intravascular lines are the most frequent source of BSI in, for example, Spanish ICU's (37%)[5] and in the USA (19%).[6] Studies in both the USA and Europe show that the risk of BSI is increased significantly (odds ratio approximately 5) in patients with central venous lines.[7,8]

Our surveillance results have been used to educate staff on the importance of intravascular line management. They were also instrumental in the establishment of a HHS wide group to review and develop a multidiscipline intravascular line policy.

The recently published Centres for Disease Control and Prevention (CDC) guidelines for the prevention of intravascular device- related infections provides a framework for the prevention of these infections.[9] The CDC emphasise the importance of device associated rates when comparing infection rates between units or hospitals.[10] Although we have identified intravascular lines as the most common source of BSI we have only recently begun to record central intravascular line days as a measure of patient exposure to these devices. A prospective central line associated BSI study for high risk patients has commenced. This will permit comparisons with other studies, including those from New Zealand.[11]

The predominant pathogens encountered in our hospitals are similar to those reported in other studies. In Spain BSI isolates in ICU's were CNS (24%) and *S. aureus* (19%).[5] In the USA the most common isolates are *S. aureus* (18%), CNS (12%), *E. coli* (11%), *Pseudomonas* (6%), and *Candida* spp. (10%).[6] The National Nosocomial Infections Surveillance system (NNIS) in the USA which studies isolates from ICU's found CNS (34%), *S. aureus* (13%), enterococci (13%), and *Candida* (6%) to be common organisms.[12] The NNIS system data also show that gram-positive organisms are the predominant, and increasing, cause of nosocomial BSI.[13] The increasing use of central venous lines has influenced the prevalence of different pathogens dramatically[7] especially the predominance of CNS intravascular line related BSI in neonates.

The BSI rate increased early in this three year observational period, but since mid-1996 has remained steady at approximately 6/1000 admissions. Auckland Hospital (~8/1000) and the HHS as a whole (~6/1000) have BSI rates higher than the Australian Council on Healthcare Standards threshold level of ~3/1000 used by Jones[4] as a baseline. The NNIS system in the USA has shown that BSI rates increase with the size of the hospital and are highest in the largest teaching institutions.[8]

Increasingly, healthcare providers are being asked to benchmark and compare rates of key events. This is a complex and difficult undertaking when comparing nosocomial infections between institutions because they are affected by a variety of factors, some of which, such as underlying health status of the population served by the hospital, are outside the hospital's control.[14] CDC emphasise the importance of surveillance data that adjust for specific infection risks in order to provide better interhospital comparison.[9] As Jones identified,[4] progress should continue with the development of strategies for national hospital infection surveillance and control. Appropriate data need to be collected before comparisons between tertiary services can be made.

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Cumulative incidence of hepatitis C seroconversion in a cohort of seronegative injecting drug users

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Abstract

Aim. To measure the cumulative incidence of hepatitis C virus seroconversion over a two year period in a group of seronegative injecting drug users.

Methods. The study involved follow-up, in 1996, of a cohort (n=85) of injecting drug users identified as hepatitis C virus seronegative in 1994. Participants were interviewed about risk factors for hepatitis C. A blood sample was also taken for anti-hepatitis C virus antibody and hepatitis C virus RNA testing.

Results. Forty-four participants were interviewed and 39 gave blood for testing. Most (80%) were aged 29 years or under and two thirds (n=26) were male. Around half reported borrowing (49%) or lending (57%) needles and syringes since 1994 and both of these behaviours were associated with seroconversion. The majority (88%) also reported sharing other injecting equipment. Nine were anti-hepatitis C virus positive giving a seroconversion rate over two years of 23% (13 per 100 person years). Four out of the nine seropositive specimens tested were also hepatitis C virus RNA positive.

Conclusions. This study demonstrates a high rate of recent hepatitis C virus seroconversion amongst a group of New Zealand injecting drug users. Transmission of hepatitis C virus appears to be unabated by current control measures. These findings confirm the need to develop more effective policy and practices to prevent further spread, not just of hepatitis C, but of other blood-borne viruses in injecting drug user populations.

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Since the identification of hepatitis C virus (HCV) in New Zealand, as in other countries, seroprevalence studies have found high rates of infection in injecting drug users. The first of these studies, by Miller and colleagues based their estimate of 72% on samples from blood donors who reported injecting drug use.[1] Woodfield and colleagues found a seroprevalence rate in injecting drug user clients of drug treatment services in Auckland of 73% (using a first generation antibody assay), in contrast to a rate of 4% in clients reporting only oral drug use.[2] Robinson's study of Wellington methadone clinic clients found a seroprevalence rate of 78%[3] and Chetwynd and colleagues found a rate of 84% in clients of the Christchurch methadone programme.[4] Kemp and his colleagues in their national multi-centre study found an overall seroprevalence of 64% (range 42-100%) though rates amongst injecting drug users in treatment were higher and consistent with the other local studies.[5]

Some of these studies have provided data on specific risk factors for HCV infection among New Zealand injecting drug users, most of which are consistent with published information from other countries.[6-10] Age of injecting drug users has been found to be associated with their risk of acquiring HCV.[1-4] Duration of injecting drug use has also been confirmed as a risk factor by Robinson[3] and the Christchurch methadone programme study.[4] Age and duration of injecting use are not independent risk factors. The Christchurch study also found frequency of lending and borrowing needles and syringes to be a risk factor for infection. The greater the number of people sharing injecting equipment the greater the risk.[4] One factor identified as protective in the Christchurch study was reported ease of access to new injecting equipment.[4]

Studies elsewhere, including some in Australia, suggest that both the prevalence and incidence of HCV infection is high in injecting drug user populations.[7,8,10] No data on the recent or current incidence of HCV infection in New Zealand injecting drug users were available, though anecdotal reports suggested that the situation could be similar here. This study aimed to obtain an estimate of the cumulative incidence of HCV seroconversion in a group of injecting drug users identified as seronegative in a previous national HCV prevalence study.[5]

Methods

The sample population consisted of 85 injecting drug users who had participated in a national seroprevalence study conducted by Kemp and colleagues in 1994. This cohort represents all those who were found to be seronegative at the time of participation in that study and who indicated a willingness to be recontacted in future (85 of 241 drug users). These potential participants were resident in Auckland, New Plymouth, Wellington, Christchurch and Dunedin at the time of the original study. The original sample was recruited using a snowball technique including approaches to clients of needle exchange programme outlets, injecting drug users in the community and injecting drug users receiving drug treatment.[5]

Data collection for the study was carried out between May and December 1996 by one of us (RK) assisted by trained injecting drug user fieldworkers. Attempts were made to locate and recontact potential participants using contact information supplied at the time of the previous study. Once contact was made, information was provided about the study and they were invited to take part. If they consented, the fieldworker administered a questionnaire that sought basic demographic details, injecting history, current injecting practices and other possible risks for hepatitis C in the interval between studies and the results of any interval testing for hepatitis C. Questionnaire data were analysed using Epi-Info, version 6.[11]

A blood sample was taken by the fieldworker at the time of interview and sent to ESR's Communicable Disease Group for HCV antibody testing using a third generation assay (anti-HCV EIA, Abbott Laboratories). All reactive samples were then retested with an alternative third generation assay (Murex anti-HCV EIA Version III). Any samples giving discrepant results between the two assays were also tested with the Chiron RIBA 3.0 immunoblot assay. Both seropositive and seronegative samples were tested for HCV RNA by RT-PCR (Roche Amplicor HCV test). The results of testing were personally returned by fieldworkers to all participants who requested them.

The study was approved by the Canterbury, Otago, Wellington, Taranaki and Auckland Ethics Committees.

Results

Interviews were completed with 44 of the original 85 seronegative injecting drug users and blood samples were obtained from 39. Five of those who completed interviews did so by telephone from Australia. Of the remaining 41 injecting drug users who did not participate in the follow-up study, 26 were unable to be located, nine were known to be overseas but without a contact location and one was known to have died. Only five refused to participate when contacted.

The mean age of those completing interviews was 27 years (range 18-42), though almost 80% were aged 29 or under. Twenty six participants were male and 18 female. All but three identified themselves as European. All participants supplied information about their injecting histories and practices. Their mean age at initiation of injecting drug use was 19 years (range l2-24) and the mean reported duration of injecting use was five years. A third (34%) were currently in drug treatment. Non responders had a similar age distribution and a similar mean age at initiation of injecting drug use to responders (see Table 1). A higher proportion of non-responders was male (73% vs 59%), though this difference was not statistically significant. Both responders and non-responders were equally likely to have a history of drug treatment. However, non-responders were much more likely to have a history of imprisonment (35% vs 7%) and this difference was significant (see Table 1).

Table 1. Comparison of characteristics of questionnaire responders and non-responders.

	Responders (n=44)	Non Responders (n=41)	p value*
Age in years (mean)	24.7	26.0	0.31
(range)	(18-42)	(17-43)	
Sex			
Males	26 (59%)	30 (73%)	0.25
Females	18 (41%)	11 (27%)	
Age at first injecting (mean)	18.9	19.9	0.99
(range)	(13-23)	(15-42)	
Duration of injecting drug use (years)	5.9	6.0	0.82
History of drug treatment at time of previous study	43	41	1.00
History of imprisonment at time of previous study	3 (7%)	14/40 (35%)	0.001

* P values for all comparisons are derived from Fishers exact text (two-tailed), except for those referring to age, age at first injecting and total duration of use for which the Kruskal Wallis test was used.

At follow-up, 49% reported borrowing needles and syringes regularly since the previous study in 1994 and 57% reported lending their needles and syringes over the same period. A much larger proportion (87%) reported sharing of other items of injecting equipment, such as spoons, filters and dregs in the interval between the studies.

Of the 39 participants who gave blood samples, nine were found to have antibodies to HCV. Four out of nine of those who were seropositive had detectable HCV RNA in their serum. None of those who were seronegative had detectable HCV RNA. These results represent a cumulative seroconversion rate over two years of 23%(95% CI 11-39). This is equivalent to an incidence of 13 per 100 person years (95% CI 6-25), assuming seroconversions took place at the midpoint between the original study and follow up. As far as could be ascertained from questionnaire responses about previous HCV testing and medical records of participants who seroconverted, some seroconversions occurred in each

year since 1994. Although participants were not specifically asked about their reasons for being tested for HCV in the interval between the two studies many volunteered this information. Some said they had sought testing because a partner had been found to be seropositive, others had been tested after an at-risk incident, such as a needlestick injury and one had testing done during an acute episode of jaundice.

Most of those who were found to be seropositive (8/9) already knew their status having been tested in the interval between the studies. Characteristics of seronegative and seropositive participants are compared in Table 2. The mean age of seropositive participants was slightly younger than those found to be seronegative though this difference was not significant. All were European and two thirds were male. Their mean age at initiation of injecting use was 18.7 years and they had been injecting on average for 4.9 years. Significantly more reported borrowing or lending needles and syringes than those who were seronegative, though the reported frequency of sharing other injecting equipment was high in both groups. Most (7/9) were currently in drug treatment. There were no significant differences between the two groups in the frequency of other potential risk exposures such as tattooing and skin piercing. Only one from each group had a history of recent imprisonment and none from either group had a history of blood transfusion. Several of the seropositive participants attributed their seroconversion to a particular event, including needlestick injury and sharing injecting equipment with someone known to be seropositive. Two couples were amongst the seroconverters and in both cases, the male partner had been known to be seropositive first. Two further participants volunteered the information that their partners (with whom they had shared injecting equipment and had sexual contact) had also seroconverted during the followup period, though these people were not part of the study cohort and it was thus not possible to obtain laboratory confirmation.

Table 2. Comparison of seronegative and seropositive participants' demographic and drug use characteristics.

	anti-HCV -ve (n=30)	anti-HCV +ve (n=9)	p value*
Age in years (mean)	27.4	25.4	0.2
(range)	(18-42)	(22-33)	
Sex			
Males	17	6	
Females	13	3	0.7
Age at first injecting (mean)	18.7	19.1	0.7
(range)	(12-24)	(16-24)	
Duration of injecting drug use in years (mean)	5.2	4.9	0.9
(range)	(0.5-17)	(2.5-9.0)	
Borrowing needles/syringes	12 (40%)	9 (100%)	0.002
Lending needles/syringes	10 (33%)	8 (89%)	0.008
Sharing other injecting equipment	24 (80%)	9 (100%)	0.3
Currently in drug treatment	7 (23%)	7 (78%)	0.005

* P values for all comparisons are derived from Fishers exact test (two-tailed), except for those referring to age, age at first injecting and total duration of use for which the Kruskal Wallis test was used.

Discussion

This study represents the first estimate of HCV seroconversion rate in a New Zealand injecting drug user population. The measured rate is both high and consistent with rates found in HCV incidence studies on injecting drug users elsewhere of between 10-20 per 100 person years.[7,8,10] The number involved in our study, however, is small and the confidence interval around the point estimate is correspondingly wide. The data we obtained on demographic and behavioural risks for HCV are also consistent with previous New Zealand studies of drug user injecting histories and practices.[3-5,12,13] Most injecting drug users previously identified as seronegative were young, reflecting the known association of HCV infection with age in this population. Similarly, as has been observed in other studies, most had short injecting histories. More recent studies elsewhere suggest that the rate of seroconversion is highest in the first year after initiation of injecting drug use.[14]

Behaviours which increase the risk of HCV transmission such as borrowing or lending needles and syringes are still common and in this group were associated with a higher risk of seroconversion. The sharing of other injecting equipment is still almost universal. The retrospective exposure assessment in this study cannot, however, allow firm inferences to be drawn about the timing and relative importance of these particular risk practices.

Because our study used a previously identified group of injecting drug users for whom no formal arrangements were made at the time of the previous study to permit recontact, we experienced difficulty in locating members of the group. Thirty percent of the original sample could not be relocated. Identifying data provided to the original study for most of these individuals were insufficient to permit a search of death records to establish if any had died. Individuals in our sample were mobile, Australia being a common temporary location, though others were known to be in Europe and South East Asia at the time of the study. Our response rate was low, but the characteristics of non-responders, as measured by their responses to the 1994 prevalence survey (see Table 1) were similar to those of responders with respect to known associations with seroconversion such as age, duration of injecting and history of drug treatment. It is possible, however, that those we were unable to recontact represent a more itinerant group with potentially higher rates of exposure to other risk factors for seroconversion such as less easy access to clean injecting equipment. The higher reported rate of prior imprisonment in non-responders might be a marker for higher risk exposures in that group. The effect of these biases is likely to have led to under-estimation of the actual rate of seroconversion amongst previously seronegative injecting drug users. It was encouraging that so few of those we contacted refused to participate and their comments indicated that New Zealand injecting drug users are interested in research and information about hepatitis C.

The high rate of new infections with HCV found in this study, while not unexpected, is of serious concern. Although the current prevalence of HIV infection in New Zealand injecting drug users is low at 0.5%[15] both the prevalence and incidence of HCV infection provides a forceful reminder of just how far and how quickly other blood borne viruses can spread amongst injecting drug users.[16] Current prevention strategies, particularly the needle exchange programme, appear to have been effective in controlling HIV, a disease of low prevalence in New Zealand injecting drug users, but our findings suggest they have been largely ineffective in containing the spread of HCV which has a much higher prevalence.

Behaviour change towards safer injecting has occurred in New Zealand injecting drug users, but this study and previous ones in New Zealand show that their practices have not changed enough to prevent the spread of HCV.[3,4]

The implications of an unabated epidemic of hepatitis C in New Zealand injecting drug users are considerable. Injecting drug users account for the majority of the incident cases of hepatitis C in New Zealand since screening of donated blood for HCV was introduced in mid 1992.[17] The fact that chronic health consequences of hepatitis C infection are common[18,19] and that currently available treatments are both costly and of limited effectiveness[20,21] means that HCV infected injecting drug users are likely to continue to require significant treatment resources. There are thought to be between 13 500-26 500 current injecting drug users in New Zealand,[22] most of whom will have hepatitis C already. The population of injecting drug users is, however, a dynamic one, with individuals commencing and ceasing to inject over time. Our study confirms that those injecting drug users who do not yet have the disease are at high risk of infection, as are new initiates. Many of those who have ceased injecting may still have to face the chronic consequences of HCV infection. Development and implementation of more effective prevention strategies is urgently needed. Continued neglect of this situation cannot be justified in the face of the available evidence.

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VIEWPOINT

Intellectual origins and principles of the internal market in New Zealand

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In its July 1991 Budget, the New Zealand government officially adopted an "internal market' in health through its policy statement frequently referred to as the 'Green and White Paper'.[1] While much has been said about that document considerably less has been said about the ideological framework that helped formulate its introduction.

Despite a confused interlude under the short-lived Coalition (National-NZ First) Health Agreement which had a contrary emphasis on cooperation and public service values, the framework continues to shape the current direction of New Zealand's health system. The description has changed, however, from "competition" to "managed care" and then to "integrated care".

The intellectual basis of the internal market originated with the analysis by Alain Enthoven who advocated an experimental internal market in which providers in the National Health Service (NHS) would compete thereby compelling the development of proper costing systems and creating more cost efficiency and cost sensitivity.[2]

The British Government under Margaret Thatcher readily adopted Enthoven's internal market making structural changes with the introduction of NHS Trusts. This was contrary to his advice that it should first be tested through a small trial. New Zealand, however, was initially more cautious than Britain. The Labour Government (1984-90) established a major review of public hospitals, known as the Gibbs Taskforce[3] that recommended an internal market. Despite support from the Minister of Finance, Roger Douglas, the Government rejected this aspect of the Taskforce's report. Across the Tasman Australia had also considered the internal market as part of a national health strategy but only as an issue for discussion and debate rather than a policy proposal.[4] Election of the National Government in October 1990, however, provided a political environment conducive to the Internal market. New Minister of Health Simon Upton was keen to implement it quickly.

Business round-table report

Perhaps the most influential pressure group in New Zealand, the Business Round-Table, had already commissioned a report by American Professor Danzon and

the former Treasury official Susan Begg that provided a detailed ideological and intellectual framework for the development of the internal market.[5] The report emphasises competition as the most effective means of improving efficiency and choice. It claimed that the management of area health boards faced divided loyalties between the Minister of Health, taxpayers, electors (consumers), patients and employees. Without explanation it asserted inevitable and implicitly unresolvable conflict between these multiple accountabilities. The report argued that area health boards failed to develop effective performance evaluation. It felt that patients should be able to make "trade-offs" between different aspects of quality and between quality and costs. Quality, according to the report, was "multi-dimensional" including cost, technical competence, waiting times, amenities, privacy and the attitudes of professional staff. In order to achieve good outcomes it was concluded, "... ultimately the best measure of performance is market survival."

As area health boards were not required to earn a normal return on the effective equity provided by government, they were given an unfair advantage relative to private organisations operating on a commercial basis. Further, there was a lack of incentive for enhancing performance because of the lack of competition between boards. Consequently the public had little influence on boards with regard to the quality and quantity of health care they desired.

Health providers had 'captured' the public health service ensuring that their interests were predominant. The writers observed that, until providers are allowed to compete in a competitively neutral environment, potential pressures for cost minimisation would not be exerted on providers. All this resulted in an alleged lack of patient choice over the site of treatment and services offered, where they could be treated and what services would be offered. The report argued that there was a need to put the public and private systems on the same footing. It signalled as remedies patient fees in the public sector and contracting out public services.

The report's overall conclusion was to advocate corporatisation, and separation, of funding and providing health services. Previously, under the 1984-90 Labour Government, state commercial activities had been corporatised into state owned enterprises. Some subsequently, such as telecommunications and printing, were privatised. Corporatisation therefore was a logical extension into the health service.

In arguing the case for corporatisation the report offered two options. First, the funding and providing functions of area health boards should be separated and corporatised. Both public and private hospitals would become competing providers. Second, individuals should be allowed to opt out of the public system.

The objective of a corporatised hospital would be to operate as a "successful commercial business" contracting to provide services. It should pay tax and earn a return on capital as well as pay dividends to the shareholding government. It would only be paid for the services it provides.

Some potential problems with corporatisation, however, were identified. It was still monopolistic and a residual difficulty of continuing political interference remained. Consequently a second option of individuals opting out with a "premium" was advocated allowing individuals to choose between private and public insurers. This would also encourage full medical facilities to become available in the private sector. The introduction of user charges for patients and the allocation of "premiums" would facilitate the privatisation of hospitals and encourage full private insurance.

Supportive role of treasury

The Treasury provided a decisive report[6] to the newly elected National Government that slightly pre-dated the Danzon-Begg report. The ideological direction was identical with Susan Begg involved in this report also. It stated that 7.5% of Gross Domestic Product was spent on health in 1989, 80% from central government. It claimed widespread dissatisfaction with waiting lists, inferior performance, and was concerned about future pressure on the health service from an ageing population in the face of a worsening fiscal situation. Substantial inefficiency of public hospital services was alleged. Reference was made to the Arthur Anderson report, commissioned by the Gibbs Taskforce, which claimed that there could be up to a 32% reduction in hospital costs without loss of outputs. Treasury estimated that a more realistic figure was around 10-15%. But there had already been a 2.6% real reduction (inflation adjusted) in aggregate area health board funding since 1987-88.

Some improvements, such as increased surgical output through day surgery, in area health boards were acknowledged. To maintain gains several issues were raised: further institutional reform was necessary to ensure increased efficiency rather than service cuts; boards had inefficient information services to assess performance and non-performance; and boards did not have full discretion over the management of their resources (for example, leasing out surplus assets, capacity for fee paying private patients).

Like Danzon-Begg, Treasury argued that "consumers" had little choice because area health boards as funders faced conflicting local and national political pressures that provided confusing signals to managers. These helped impede the accountability of boards to "consumers". Further, interest groups such as health professionals were too influential.

This Treasury report presented two options for change identical to but less detailed than those proposed by Danzon-Begg. First, funding or purchasing should be separated from provision in order to lead to more competition, including an ability to compete on wage costs. The development of the private insurance industry was encouraged to avoid a funding monopoly. Second, an increase of private funding through the introduction of user charges for public hospital services and, similar to the Danzon-Begg "premiums", individual entitlements for consumers.

Official government policy

The National Government's policy was announced in the 'Green and White Paper', by Minister of Health Simon Upton. Despite claiming to be "... expressly, not ideologically driven"[1] the policy statement closely followed the ideological principles contained in the Danzon-Begg and Treasury papers and it also focused on constraining government expenditure. It faithfully followed the separation of purchaser and provider by replacing 14 area health boards with four purchasers (regional health authorities) and 23 public providers (state-owned companies called crown health enterprises run by boards of directors). Some area health board facilities would also be available to be run as "community trusts".

Regional health authorities (RHAs) would contract with crown health enterprises (CHEs), private hospitals and other providers through purchase agreements. "Service providers who performed well would be rewarded by

winning more RHA funds, with which they can expand their services". Each RHA would seek out, through competitive tendering, appropriate providers of services – public, private, and voluntary – and contract with those which offered the best value for money. To avoid funding monopolies alternative health care plans (HCPs) were advocated in which people could take their entitlement with them from an RHA to an HCP. This was effectively a voucher system similar to the "premium" advocated by Danzon-Begg. HCPs could specialise in certain types of health or could be insurance companies. Ironically, given the strong advocacy for separating funding and provision, an HCP could also be a provider.

A further major platform was the establishment of a review of core health services which were "... health services to which we believe everyone should have access on affordable terms and without unreasonable waiting times." Non-core services would be those that people paid for themselves or through insurance. This would limit the growth of public expenditure.

Consultancy advice on the internal market

The 'Green and White Paper' established a National Interim Provider Board (NIPB) to advise Government on the formation of CHEs. The NIPB, in turn, contracted a major consultancy agency, CS First Boston NZ Ltd, to provide advice. This advice was consistent with the Danzon-Begg and Treasury reports, and significantly CS First Boston now employed Susan Begg.

The principle task of the NIPB was to ensure an "... unleashing [of] market incentives for efficient reorganisation rather than in any senses planning what an efficient industry should look like".[7] To operate as a successful business with maximum flexibility it was necessary for CHEs to be established along commercial lines similar to the corporatisation of state-owned enterprises.

Funding of both primary and secondary core health services should be integrated in order to overcome fragmentation. Funding and provision of health care should be separated in order to strengthen competition. User charges would be implemented and competition established among funders through the ability to opt out into health care plans. The Government's health restructuring was likened to transforming a major industry in a 'Soviet-style' planned system.

The report outlined an unashamedly commercial approach including the selling of assets and functions whose market value exceeded the return the crown health enterprise expected to achieve. Services and inputs that could be provided at lower cost by outside parties would be contracted out. CHEs should therefore be initially established as "commercial profit maximising businesses". Once established they should be given as much flexibility as possible to enable resources to be transferred into other forms of organisation where this would provide efficiency benefits. Further, the Government should not support CHEs if they developed financial difficulties.

The report was concerned at the lack of competition in particular services and locations in the public health service. Nevertheless, it noted the growth of the private sector, especially Southern Cross (the largest private insurance company in New Zealand owning nine private hospitals) which could compete for core health services from regional health authorities. Also, the voluntary sector, including agencies such as Plunket (pre-school child health), the Intellectual Handicapped Society, and the Salvation Army were potential competitors. Significant in terms of economy of scale, international insurers operating in New Zealand had major providers operating overseas.

The report then proceeded to stress the importance of actively promoting new entrants to health care provision to achieve competition and avoid monopoly. Examples of how this could be achieved were provided. First, it was necessary for RHAs to treat public, private and voluntary providers equally without any preference. Second, there should be no unnecessary regulatory impediments to new entrants. Third, the capacity to opt out of RHAs into health care plans should be encouraged. Fourth, the management of public sector health infrastructure should be contracted out to the private sector.

The report also stressed that the government had two criteria in its 'health reforms' – efficiency and fairness. As fairness was to be met through funding (by RHAs) the principle objective therefore of provider reform should be economic efficiency.

National interim provider report

The key NIPB report was presented to Government in May 1992. It noted that the 14 area health boards covered about 155 hospitals and 21 300 beds (70% of the total number of hospital beds in New Zealand). The bank value of board assets was estimated at $3.2 billion and there were about 60 000 employees.

Consistent with the ideological perspective of previous reports it boldly concluded that: "Competition is the only way of ensuring on a continuing basis, constant innovation and best value at optimum quality for every health dollar ... recommendations are based on proven principles that work".[8]

The Minister of CHEs, the Hon Paul East, said that the report provided an excellent foundation for new health service provision.

The report outlined the goals of the 'health reforms' as liberating the innovation and initiative of health care personnel, performance based accountability, linking the management of staff to the success of their organisations, and a patient focus. It concluded that: "Competition among providers of health care is the mechanism which has been selected as most likely to achieve those goals."

The report recommended a profit-making business model for crown health enterprises that included paying dividends from 1 July 1993. The underlying key principles were commercially oriented providing for managerial autonomy and mechanisms for accountability of managers.

Initially CHEs would provide the existing range of services. However, this would be subsequently re-assessed by RHAs through purchasing arrangements arising out of competitive bidding. RHAs would fund irrespective of provider ownership. State-owned providers would have no competitive advantage or disadvantage over non-state providers, and operate on commercial balance sheets with debt/equity ratios comparable to those of the private sector. CHEs should be subject to the Commerce Act in order to prevent anti-competitive abuses.

Twenty four acute care, accident and emergency, and trauma services were one area there was no strong private competition because of the requirement for continuous provision of staffing, spare beds and equipment to cope. Consequently to ensure competition, where area health boards had two or more 24-hour acute care hospitals these should be broken down into competing CHEs.

CHEs were not the only organisational form for providers. Different models include diversifying into separate more specialised competitive units, or community trusts without government guarantee. Examples of community trusts were care for the elderly, general practitioner clinics, maternity services, bases for visiting specialists, and care for the terminally ill. Trusts could

compete with both themselves and CHEs for RHA funds or alternatively compete by sub-contracting to provide services to CHEs.

Reinforcing the true business directions of the 'health reforms' the report acknowledged the prospect of business failure of state-owned providers and concluded that this should be permitted in the health service.

Health and Disability Services Act 1993

This act provided the legislative framework for facilitating the internal health market and faithfully followed the preceeding discussion papers and reports.

Four regional funders (RHAs) and 23 state-owned provider companies (CHEs) replaced the 14 area health boards. All the CHEs were centred on a hospital providing some form of acute 24-hour care except the second CHE in Christchurch, Healthlink South, which was based on community, elderly, psychiatric and maternity services. This separation into two CHEs in Christchurch was contrary to the overwhelming opposition of senior medical staff and other expert advice. The decision, which was pre-determined, disregarded the contrary advice obtained through the consultation process.[9]

Although not specified the opportunity for private sector alternative health care plans was allowed by the liberal definition of "purchaser". Section 20 of the Act states that a "purchaser" may also incorporate any "... person that has agreed, and that has been declared by the Minister ... to be a purchaser for the purposes of this Act".

The mechanism for the negotiation between purchasers and providers was the "purchase agreement". Such was the extent of the new health market that purchasers were not confined to RHAs and providers were not confined to the state-owned companies (CHEs were to be registered as companies under the Companies Act). A purchaser was permitted, by Section 22 of the Act, to provide money to any "... person in return for the person providing, or arranging the provision of, health services ...".

The original Bill aroused considerable controversy, in part, because the definition of the principle objective of CHEs stated they should operate "as a successful business that provides health services..." and be as "... profitable and efficient as comparable businesses that are not owned by the Crown...".

In an attempt to soften opposition, this provision was amended in the final Act (Section 11) by removing the reference to "profitable", a presentational rather than substantive amendment. The objectives of CHEs were stated in Section 11 as follows: "(1) The principle objective of every CHE shall be to: (a) Provide health services or disability services, or both; and (b) Assist, in meeting the Crown's objectives under section 8 of this Act by providing such services.... while operating as a successful and efficient business. (2) Without limiting subsection (1) of this section, every CHE shall have the following objectives: (a) To exhibit a sense of social responsibility by having regard to the interests of the community in which it operates; (b) To uphold the ethical standards generally expected of providers of health services or disability services, or both, as the case may be; (c) To be a good employer; (d) To be as successful and efficient as comparable businesses that are not owned by the Crown."

Whilst the term "profit" was deleted from the final Act, there was no need for a direct reference to a profitability requirement as CHEs were now companies registered under the Companies Act. Further, they were required to compete with each other and other competitors for funding, and to be "... as successful and efficient" as comparable private businesses which were themselves expected to generate profits and would be regarded as failures and susceptible to bankruptcy if they failed to do so.

Conclusion

The internal market, although based on minimal international precedence and experience, did not arrive in an intellectual vacuum despite arguably being conceived in an intellectual straitjacket. Further, New Zealand ignored Enthoven's advice of first trialling the internal market on a limited basis. It marked a qualitative change in direction that can't credibly be legitimised as a continuation of earlier more gradual reforms such as the move from hospital to area health boards. Rather it represented an unprecedented endeavour to utilise market forces as the driver of health service organisation and delivery.

As no market existed in publicly provided health care one had to be created. It is in this context that the Business Round-Table and Treasury reports as formulators, and the CS First Boston and NIPB reports as shapers of policy, should be evaluated. Both the 'Green and White Paper' and the Health and Disability Services Act were largely loyal to this ideological framework. The process was undemocratic to the extent that outcomes were pre-determined by a small closely-knit group of people without sufficient concern for the impact on the sick and vulnerable.

Although the 1993 Act was amended on 1 July 1998 to allow implementation of the Coalition Health Agreement, the framework still prevails. These 1998 legislative changes were more of form than substance with minimal impact on the post-1991 drive to convert the health system from a social service into a commodity producer based on a commercial transaction process. Also it may be indicative of continuing cognitive consistency that the head of Treasury during 1990-1 was the first head of the Health Funding Authority.

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Room for more women in clinical specialties

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NZ Med J 2000; 113: 105-7

It is over ten years since Professor Barbara Heslop of the University of Otago reflected, in this Journal, on the dearth of women in the higher echelons of medicine, particularly in the clinical specialties and in academia.[1] At that time, she concluded that the training system effectively culled many women with children before they had the chance to acquire a postgraduate qualification, thus severely limiting their career options.[1] This was followed by a comprehensive review, commissioned by the Department of Health, of the experiences of women and men in medicine in New Zealand.[2] This review in 1989 made strong recommendations in order to improve opportunities for women in medicine. The situation for women in the late 1990s, however, may be worse than it was ten years ago. For example, this decade has seen the demise of the Medical Reserve Scheme that had formerly provided a government-funded career maintenance option for medical women under training, and the introduction of the health reforms, the Employment Contracts Act and higher student fees. Each of these may, in its own way, have an adverse effect on career opportunities for women.

This article examines the distribution of women within the medical workforce and discusses why women are not entering and completing clinical specialty training in greater numbers. Strategies for women who do wish to specialise in these areas and for institutions and society as a whole are presented. While the recommendations in this paper are primarily for women, they may benefit the medical workforce as a whole.

Women in the medical workforce

Over the last 15 years, there have been nearly equal numbers of male and female medical students in New Zealand medical schools. Yet, in 1997, only 29% of doctors in all roles (excluding house officers) were female and only 26% of specialists[3] (see Figure 1). By far the largest group of medical women is in general practice. General practitioners make up 40% of the medical workforce and 33% are female, although not all of these women are on the vocational register. Within the other clinical specialties the percentage of women varies but is generally far lower than in general practice. Women comprise only 13% of adult physicians[3] and 3% of surgeons (RACS data, personal communication). There are some clinical specialties, however, with relatively higher numbers of women specialists such as paediatrics (23%), obstetrics and gynaecology (26%), and psychiatry (27%).[3]

Part of the explanation for the disparity between the percentage of women in medical school and the percentage in the clinical specialties is that there have been significant numbers of women medical students only in recent years. Female doctors have a median age of 36 compared with 42 years for males.[3] However, despite there being equal numbers of men and women at medical school, women do not enter clinical specialty training programmes at the same rate as men. For example, women make up around 25% of adult physician trainees (RACP data) and about 10% of surgical trainees (RACS data). It should be noted that medicine is a specialty in which advanced training is open to almost all who pass the primary examination whereas surgery restricts the numbers embarking on its training schemes.

In contrast, more than 40% (but still less than half) of the trainees in paediatrics are women (RACP data), yet this training is overseen by the same college and is similar in type and duration to adult physician training. Some possible reasons for this are outlined later in this paper but a detailed study into the reasons why women choose paediatrics and not adult medicine would be timely.

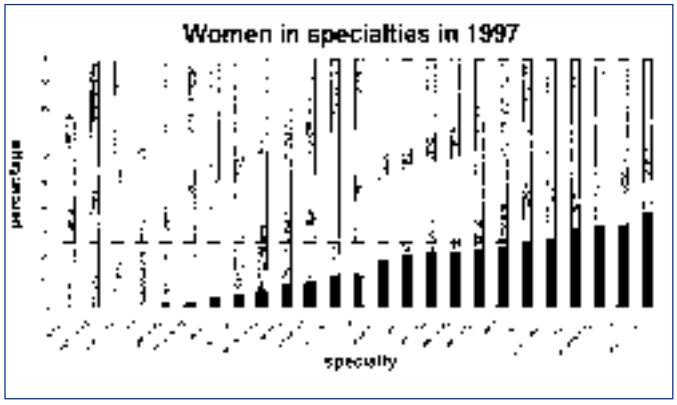

Figure 1. Percentage of women in each specialty in 1997 (as reported to the New Zealand Medical Council). Women comprise 26% of specialists overall, compared with 50% of medical students. The horizontal line has been drawn at 26% to highlight the difference across specialties in the percentage of women.

The relative lack of women entering and completing clinical specialty training is of concern for the workforce as a whole. Heslop has calculated that New Zealand's medical schools can supply only about half of the specialists that the country requires, given the lower rates of specialisation for women.[4] Already, in Auckland hospitals, medical graduates from outside New Zealand are required to fill some of the clinical registrar positions (Auckland Healthcare, personal communication).

Care-giving roles

The majority of medical women will enter long-term relationships and have children, and for those women undertaking postgraduate training, this will overlap with years of childbearing and raising. In a 1992 survey of FRACP women, 76% were married and 79% of these had children (mean number 1.5 (range 1-4)).[5] Their average age at the birth of their first child was 31 years. Of RACP medical trainees, 22% had children under the age of 16.

Women doctors continue to be the main care-givers for their children. In a study of all Melbourne female medical graduates, matched by year of graduation with an equal number of males, 25% of female doctors reported that their partner took primary responsibility for child care compared with 97% of males.[6] In New Zealand, 37% of medical women compared with 7% of men stated that their choice of a specialty was influenced by whether it fitted in with family commitments.[2] Different cultural experiences may also affect how women doctors balance their commitments to children, partner, extended family and career.

Approximately a fifth of women specialists completed at least some of their training part-time.[2] Despite the sanctioning of part-time or interrupted training by most colleges, in reality, it remains very difficult to arrange a part-time job in the clinical specialties. To do so requires either a job-sharing partner with a similar need at the same stage of training or else a reconfigured full-time job. Few colleges reduce the total training requirements for part-time trainees. Heslop argues that the triple workload of service, family and study commitments is far too great a burden, even for women working part-time,[4] and that part-time training just prolongs the agony. If training is interrupted for parental leave, re-entry into an acute care specialty can be daunting, particularly if still breast-feeding and experiencing sleep deprivation. Options, therefore, for women who wish to perform at a high level in a clinical specialty and have children, are to delay childbirth, to limit the size of their families, or to abrogate the primary care-giving role.

Single medical women

Medical women are, on the other hand, less likely than men to be in long-term relationships. In New Zealand, 22% of female doctors reported they were single compared with 14% of male doctors;[2] in Melbourne graduates, 40% of females and 25% of males were single;[6] and in US academics, 25% of females and 7% of males were single.[7] This disparity may result from medical women choosing to avoid the threat to career posed by family commitments, a choice most men never have to make. This strategy to remain single may allow women to achieve more in their careers but could result in a lack of emotional support for that demanding career. These single medical women are likely to be worse off financially than their single male counterparts for reasons outlined below.

Financial considerations

Data from the survey of Melbourne medical graduates shows that even corrected for hours worked, women doctors earn on average about $A20 000 less per annum than men.[6] The major reason for this discrepancy is that women work in lower paying specialties or types of practice. More recent data from USA suggest that when corrected for type of practice, for younger physicians at least, there is little difference between men and women in terms of their remuneration.[8] In New Zealand, women doctors are far more likely to enter primary care specialties than the better remunerated surgical specialties (Figure 1).

Because of lower rates of pay and the likelihood of interrupted practice, women will have more difficulty in paying back student loans. A 1998 survey estimated the mean debt halfway through the sixth (final) year of medicine at the University of Auckland to be $26 000 (95% CI $22 400-$28 800 personal communication, Auckland University Medical Students Association). There is no significant gender difference in debt levels. This level of debt will doubtless increase now that fees for medical students have increased from $2884 to $7980 per year. A medical student has estimated recently that it would take 60 years on a salary of $60 000 to pay back a $45 000 loan that had accrued interest at 9%.[9]

Another particular problem for women trainees is that junior medical staff and research fellows are usually on annual contracts, which may preclude the maternity leave provisions available to those on longer term appointments.

Sexual harassment

Sexual harassment is defined under Section 62 of the Human Rights Act 1993 as "use of behaviour of a sexual nature, that is unwelcome or offensive, is repeated, or of such a significant nature, that it has a detrimental effect on a person". About three-quarters of medical women have been sexually harassed at some time in their careers, by superiors, other staff or patients.[10,11] Very few formally report the harassment, although 50% will tell a female colleague later.[11] A smaller proportion (22%) of male residents also reported that they had been harassed.[10] The effects of sexual harassment are insidious, result in loss of self-esteem and productivity, and may influence choice of specialty or cause drop-out of talented doctors from training. Good employers will have implemented anti-harassment policies, although the presence of these may not be widely known to junior staff on short-term appointments.

Competence

Women perform just as well as their male counterparts at medical school and, in medical practice, women are as competent. In fact, they are less likely to be sued for medical malpractice than men.[12] At present, there is no universally accepted definition of minimum time required to maintain medical competence. Specialist women who work part-time generally need to fulfil all the requirements of the continuing medical education programme in which they are enrolled. Part-timers should be considered at least as dedicated as their full-time colleagues because of their commitment in the face of extra demands. Time spent away from work to care for family is as valid a reason as any other not to work full-time. The skills required to be an effective parent and to run a household are invaluable to medical practice.

Academic clinicians

In US academics, women are promoted more slowly than their male counterparts and this is not explained by differences in productivity, attrition, specialty or work schedule.[7] Other explanations are, therefore, that women are not putting themselves forward for promotion or that the promotion process disadvantages them. There are encouraging trends in some clinical departments at the University of Auckland. In the Department of Paediatrics, of sixteen clinical academics, six are women (37%) and three of these are professors. It is widely accepted that in the early days of this department, young women doctors were actively encouraged to embark on a career in academic paediatrics by the head of department. This strategy has resulted in the provision of a number of important role models in a department that is regarded as being flexible to women's needs. This may also explain why there are higher numbers of women trainees and specialists in this clinical specialty. In the Department of Obstetrics and Gynaecology, there are seven clinical academic women, (two professors, two associate professors and three senior lecturers) out of a total of seventeen (41%). In Psychiatry, there are nearly equal numbers of female and male clinical academics, but no women above Senior Lecturer level.

Where to from here?

Despite reaching equality in terms of number and performance at medical school, women continue to be underrepresented in the higher levels of clinical specialist practice. The reasons for this are multiple and complex as has been outlined in this paper and elsewhere.[1,2,4,6] To improve representation will require individual, institutional and societal commitment at many levels.

In 1993, the Head of Department of Medicine at the University of Auckland established a working party that addressed issues facing women in academic medicine. A

report was presented to the Department and the Faculty in 1994. The recommendations from the original report have been reviewed by a second working party in 1998. The recommendations of the working parties that have particular relevance to women in clinical specialties are listed below.

I. Strategies for individuals contemplating a clinical specialty

- train with ambition and conviction in an enjoyable specialty
- develop a supportive network of family, friends and colleagues
- seek out a trusted mentor for support and career guidance
- state needs clearly and negotiate reasonable conditions and pay
- get involved in the organisations that make key decisions
- recognise that parenting skills are valuable to everyday medical practice

II. Strategies for institutions and society

("Institutions" are public and private employers, universities, government, and professional bodies).

Recruitment

- foster career paths of student doctors, especially over the critical time between medical school and specialty training
- recognise the challenges for students and trainees who are also caregivers
- encourage and provide mentors and role models
- eliminate harassment and discriminatory practices
- address these issues in teaching programmes at several levels

Work and training environment

- encourage flexible work practices to meet employee needs
- ensure part-timers have equal access to the information, facilities and opportunities that are available to full-timers
- provide adequate maternity leave and cover
- facilitate re-entry into the workforce when required
- agree on suitable job or training expectations prior to employment
- base performance review and promotion on previously-agreed goals
- encourage women to apply for promotion

Future directions

- determine the critical features of those specialties that are attractive to women
- assess the long-term impact of medical student loans
- consider reintroduction of career maintenance training options
- monitor regularly the status of women in medicine
- continue to lobby for accessible and tax-free child care.

The challenge for institutions and society is "to adjust to the reality of women's lives instead of denying it, so that they can be better mothers and better doctors."[12] If this does not occur we will continue to be faced with a group of talented women who fail to achieve their full potential in the field of their choice. In addition to the disenchantment for the women themselves, this is wasteful in terms of training resources and may contribute to a shortage of New Zealand-trained specialists in the future.

Members of Department of Medicine Working Party on Women In Academic Medicine, 1994: Drs Anna Fenton (Convenor), Robyn North, Stella Milsom, Philippa Wiggins and Phillippa Poole. 1998: Drs Phillippa Poole (Convenor), Jill Cornish, Stella Milsom, Robyn North, Ruth Bonita and Ms Gillian Whalley.

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PROCEEDINGS

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Suitability of maternal plasma CRH measurement for predicting timing of delivery. J Ellis, T Prickett, A Howe, J Livesey, R Reid, L Hull, P Benny, W Inder, R Donald. Departments of Endocrinology, Christchurch Hospital and Obstetrics and Gynaecology, Christchurch Women's Hospital, Christchurch.

Levels of corticotrophin releasing hormone (CRH), derived from a placental source, rise exponentially during human pregnancy. Our objectives were to demonstrate an association between plasma CRH concentration and timing of delivery in a group of Christchurch women and to investigate a possible CRH cut-off value which might predict pre-term labour (<37 weeks).

Blood samples were taken every 4 weeks (wk) from 16-20 wk gestation. Plasma CRH was measured by radioimmunoassay. From individual regressions of logCRH on gestation time (≥24 wk) the CRH concentration at 26 wk was estimated and compared to the difference between observed and expected delivery dates. Data were also examined for sensitivity and specificity.

Maternal plasma CRH showed a strong correlation with number of weeks gestation at time of sampling (r^2=0.74, for all 616 samples from 107 women), rising from early pregnancy levels of ≤5pmol/L. Regressions within individuals were similar (r^2 range: 0.92-1.00). Estimates of 26 wk-CRH concentrations correlated with timing of delivery (r^2=0.40, p<0.001). At a cut-off of 59 pmol/L CRH the sensitivity for detection of prematurity was 90% with a false positive rate of 13.7%. At 65 pmol/L the values were 80% and 9.5% respectively. By comparison, a cut-off value of 110 pmol/L gave a false positive rate of 0% but sensitivity of only 60%.

We conclude that 26 wk-CRH levels show an association with timing of delivery. While final definition of a pre-term cut-off value

awaits completion of the study, a value between 59 and 110 pmol/L CRH is likely.

Predicting renal survival in primary focal glomerulosclerosis (FGS) from the time of presentation. VC Chitalia, JE Wells, RA Robson, M Searle, KL Lynn. Department of Nephrology, Christchurch Hospital, Christchurch.

A retrospective analysis was performed on 111 patients, diagnosed at Christchurch Hospital from 1965-1998 to predict the risk of developing chronic renal failure in patients with primary focal glomerulosclerosis (FGS) using predictors available at the time of presentation. The predictors studied were age, gender, systolic and diastolic blood pressure, serum albumin, plasma creatinine, presence of haematuria and amount of proteinuria. An injury score (combination of percentage of sclerosed glomeruli and proportion of tubulointerstitial fibrosis) was derived from a review of the initial kidney biopsy. Log-logistic accelerated failure time parametric models were used. The median renal survival was 16.4 years (Kaplan-Meier estimate). The best single variable model was that using the proportion of tubulointerstitial fibrosis (global chi-square 55.99, $p<0.0001$). However, inclusion of plasma creatinine significantly improved the fit of the model (global chi-square 65.04, $p<0.0001$). Both the models were validated using the statistical technique of jackknifing. These models can be used to predict the median renal survival and the risk of developing chronic renal failure at any time in a patient with primary FGS if the proportion of tubulointerstitial fibrosis and plasma creatinine are known at the time of presentation.

Throwing light upon saccadic suppression of displacement in the dark. MR MacAskill,[1] RD Jones,[1,2] TJ Anderson.[1,3]
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[2]Department of Medical Physics and Bioengineering, Christchurch Hospital.
[3]Department of Neurology, Christchurch Hospital, Christchurch.

During saccades (fast eye movements) vision is impaired. For example, the detection threshold for stimuli is raised during saccades. This and other related forms of "saccadic suppression" are caused by visual masking effects due to a structured, well illuminated environment. A small part of the effect is due to an extraretinal (probably efferent) signal however. In contrast to other forms of suppression, saccadic suppression of displacement (SSD, a decrease in sensitivity to visual displacements during saccades) has often been considered to be due to this efferent component rather than to visual factors.

The aim of this experiment was to explicitly assess the importance of visual conditions in SSD for the first time. A small computer-generated stimulus made random horizontal jumps (of 8-24 deg). An infrared limbus tracker was used to detect the saccade toward the new position, triggering a small (0.75-7.2 deg) centripetal displacement. Subjects' awareness of these intrasaccadic displacements was reported by keypress. The task was performed by 22 subjects in both a well lit environment and in complete darkness. Awareness of target displacements was similar in each condition.

These results argue against a visual approach, which predicts that subjects should be much more sensitive to target displacements in the dark, when masking could not operate to cause suppression. Rather, our results are consistent with the theory that SSD is neither a simple afferent nor efferent process, but is due to a higher level cortical comparison of the fixations preceding and following a saccade.

Interleukin 1 increases basal and AVP stimulated ACTH in equine anterior pituitary *in vitro*. T Prickett, M Evans, W Inder, R Donald. Department of Endocrinology, Christchurch Hospital, Christchurch.

The acute-phase cytokine interleukin 1 (IL-1) is known to stimulate the hypothalamic pituitary adrenal axis, primarily by stimulating corticotrophin releasing hormone (CRH) release from the hypothalamus. Whether IL-1 has a direct effect on adrenocorticotrophic hormone (ACTH) release from pituitary corticotroph cells is controversial. The aim of this study was to determine if IL-1β could directly stimulate ACTH release from perifused equine anterior pituitary cells, and whether CRH pretreatment, known to up-regulate IL-1 receptors on corticotroph cells, affected corticotroph responsivity.

Isolated equine anterior pituitary cells were pre-incubated with media containing 10nM CRH or vehicle for 20 hours, before being loaded onto columns and perifused with a medium containing 0.02nM CRH and 100nM cortisol to mimic *in vivo* levels in the horse. Columns were given a 5 minute pulse of arginine vasopressin (AVP, 10nM), perifused for 4 hours with 0 or lnM IL-1β, then given a further 5 minute pulse of AVP (10nM). ACTH was measured by radioimmunoassay in 5 minute fractions.

Cells pre-incubated with 10nM CRH and perifused with lnM IL-lβ for 4 hours showed increased basal ACTH release compared to control (114 ± 6 pM vs 86 ± 4 pM (means ± SEM), $p<0.001$). In addition cells perifused with IL-1 demonstrated a significantly greater ACTH response to the final AVP pulse compared to cells perIfused with media alone (240 ± 32% vs 96 ± 30%, $p=0.009$, expressed as % of ACTH integrated response to the initial AVP pulse). The ability of IL-lβ to increase AVP responsiveness was dependent on pre-incubation with CRH, since this was not observed in cells pre-incubated with vehicle alone ($p<0.001$, CRH vs vehicle pre-incubation).

In conclusion IL-1 increases basal ACTH release from equine corticotroph cells pre-incubated with CRH, and also potentiates responsivity to the hypothalamic secretagogue AVP. We speculate that upregulation of the protein kinase C pathway by IL-1 may account for these observations.

High voltage water purification. PT Johnstone, PS Bodger. Department of Electrical Engineering, University of Canterbury, Christchurch.

A new type of water purification device that uses a high magnitude electric field to kill micro-organisms has recently been developed.[1] This device uses a 6000V, 50 Hz high voltage supply applied directly across a flow of deionised water. The area of high magnitude electric field causes disruption of cellular membranes and leads to a loss of cell viability and infectiveness. This disinfection process required some technical changes to enable its commercial development. A new prototype model of a domestic drinking water device has been constructed with these changes in mind. The initial electrode system and power supply were redesigned to enable them to be easily manufactured. The redesigned system uses a different electrode shape, material and dimensions, although the critical parameters of electric field strength and flow rate remain unchanged. To verify that the electrical and biological performances were still valid, tests were undertaken on the new design. It was shown that the new electrode system is more electrically robust, is easily and cheaply manufactured, and provides a similar disinfection performance. The device was tested on a common bacteria, *Serratia marcescens*, and proved a close to three log reduction in viability. The device was also tested on a parasitic protozoan, *Giardia lamblia*. No viable *Giardia* cysts were detected following treatment.

[1] PT Johnstone and BS Bodger. 1997 "High voltage disinfections of liquids", IPENZ Trans., Vol 24, No. 1, EMCh, pp30-35.

Involuntary movements and stuttering: is there a causal link with basal ganglia dysfunction? HF Mulligan,[1,2] TJ Anderson,[2,3] RD Jones,[2,4] MJ Williams,[5] IM Donaldson.[2,3] School of Physiotherapy, University of Otago, Dunedin. Department of Medical Physics and Bioengineering, Christchurch Hospital, Department of Speech and Language Therapy, University of Canterbury, Christchurch.

Developmental stuttering affects 1% of the population. Its cause is unknown. The aim of this study was to characterise involuntary movements (e.g. excessive blinking, grimacing) in developmental stuttering. Sixteen adult stutterers, aged 15 to 70 years, and 16 matched controls were audio-videotaped while speaking freely and reading. Dysfluencies (stuttering) were classified from audio data. Involuntary movements (IMs) were identified then classified from video data as tics, chorea or dystonia.

Most IMs were simple or complex motor tics similar to those in patients with other movement disorders. Stutterers had a higher proportion of classic (within-word) dysfluencies accompanied by IMs than controls during speech (24.4% vs 4.5%, $p=0.054$) and reading (28.6% vs 4.9%, $p=0.033$). There was, however, no difference between the two groups for IMs accompanying normal (between-word) dysfluencies. IMs occurred infrequently during fluent words in free speech (3.9% vs 3%, NS) and reading (2.4% vs 0.8%, $p=0.03$).

This study demonstrated that stuttering is associated with simple and complex tics. Interestingly, patients with tic disorders (e.g. Tourette's syndrome) have a higher incidence of stuttering. These results support the notion that developmental stuttering has a causal link with dysfunction of the basal ganglia or its cortical connections.

Antibacterial viruses and antibacterial agents: a one-two punch? B Adams, JA Heinemann. Department of Plant and Microbial Sciences, University of Canterbury, Christchurch.

The rise in bacterial resistance to conventional antibiotics has re-inspired the search for alternative therapeutics. Some investigators are attempting to develop phage therapies, that is, the use of these bacteria-specific viruses to kill pathogenic bacteria *in vivo*. The idea is to use virulent, rather than temperate, phages because lysogens would be immune to further infection. We investigated the combined effects of virulent phage and antibiotics on bacteria.

Phage kill their hosts only when the host can express genes and produce enough energy to support the phage life cycle. Antibiotics and other environmental stresses could prevent phage from establishing a sustainable infection in bacterial populations because those stresses block phage reproduction. However, others have found that phage genomes can persist in starving bacteria, a condition referred to as pseudolysogeny. If the phage genome were similarly stable in the host bacterium during periods of antibiotic-induced dormancy, then the phage might persist and kill any bacteria that survive initial antibiotic treatment.

T7 and λvir phage were allowed to infect *Escherichia coli* fully inhibited by chloramphenicol. The antibiotic and remaining "free" phage were then washed from the culture. Like starvation, chloramphenicol induced pseudolysogency because infected bacteria produced progeny phage upon removal of chloramphenicol. Up to 90% of the T7, and 95% of the λvir, plaque forming units enumerated on fresh lawns of E coli arose from bacteria previously infected in the presence of chloramphenicol. Therefore, an irreversible phage infection can be established during antibiotic treatment *in vitro* followed by phage-induced host death after treatment.

Stealth antibiotic resistance. A Gunn, JA Heinemann. Department of Plant and Microbial Sciences, University of Canterbury, Christchurch.

When antibiotic resistance arises from acquiring new genes or mutations, it is stable and demonstrable after laboratory culture. However, microbes found susceptible to certain drugs during laboratory culture can acquire temporary phenotypic resistance. If the assay for resistance were to mislead the clinician about the *in vivo* phenotype, then infections might persist longer even during appropriate chemotherapy.

Gentamicin purportedly induces temporary resistance in *Pseudomonas aeruginosa*. Is this resistance due to a change in bacterial physiology caused by the drug or a selection of resistant mutants that are uncompetitive in the absence of the drug? Our "fluctuation analysis" suggests that the apparent effect of gentamicin on the bacteria could be explained by the selection of a subpopulation resistant by mutation.

Like others, we found frequencies of gentamicin resistance much higher in cultures previously exposed to gentamicin, even at sub-lethal doses. Near uniform sensitivity returned approximately 10 generations later. If the transient nature of the resistance phenotype were due to switching to a physiological state maintained by gentamicin, then the number of resistant bacteria in a series of cultures should cluster around a mean. Instead, the variance in the number of resistant bacteria in 8 different series of up to 20 individual culture was significantly ($p<0.05$) different from a Poisson distribution. Thus, it appears that this phenotype is due instead to a mutation which is deleterious in the absence of the drug. Diagnosing gentamicin resistance will, unfortunately, remain a difficult problem for the clinical microbiologist.