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NEWSLETTER

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EDITORIAL

Poverty – Still a Health Hazard

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Poverty makes people sick. A reminder, if we need one, is provided by a recent account of child health in South Auckland.[1] One in ten New Zealand children live in this area, and many live in economic and social hardship. One third of the population of South Auckland over the age of 15 is receiving income support, 20% of households have no car and 11% have no telephone. Rates of most diseases are higher here than elsewhere in New Zealand. Some of the increases are spectacular: for example, 1% of Pacific Island infants in South Auckland developed a meningococcal infection in 1997.[1] Many of the problems are well known to be poverty-related, such as respiratory and skin infections sufficiently severe to require hospital admission. "Modern" markers of socioeconomic deprivation include vaccine-preventable diseases and injury from motor vehicle accidents. (Infant deaths in road crashes are three times more common in this population than in the remainder of New Zealand.) Comparisons within South Auckland make the same point: children in the most deprived areas are twice as likely to be admitted to hospital as children in the most affluent areas.[1]

What applies in South Auckland holds for the rest of the country as well: the National Health Committee has documented the strong and consistent links between social and economic disadvantage and poor health.[2] The National Health Committee concluded that the social and economic factors that have the greatest influence on health are income and poverty, employment and occupation, education, housing and culture and ethnicity. The Committee recommended that the Minister of Health take a leading role in promoting cooperation between agencies to reduce the effects of factors such as poverty. There were 37 specific recommendations made by the Committee[2] and these included interventions in the health sector to reduce social inequalities (such as improved immunisation and tobacco control), a review of key macroeconomic and social policies (such as housing and income support), population-based services and environmental measures (fluoridation being a good example) and community development and intersectoral initiatives.

In general we know more about the association between social and economic disadvantage and poor health than the mechanisms that might explain cause and effect, but a recent report from the Family Centre Social Policy Unit sheds light on how poverty in modern New Zealand affects the ways in which people live, and as a result influences their chances of disease and injury.[3] Charles Waldegrave and his colleagues interviewed 401 people from a random sample of New Zealand households with dependent children and a combined annual income of under $25 000. Half the participants reported that they had been unable to provide meals for the household at some time in the last three months, due to lack of money. Forty percent of households were over-crowded, using criteria developed by the Ministry of Housing (and 25% were paying half or more of their income on rent or mortgage payments). More than half the households had members who did not visit a doctor when they thought they needed to in the previous year because they could not afford it.

How big is the problem of ill-health due to poverty? This depends to some extent on definitions. Poverty and socio-economic disadvantage are overlapping concepts used to describe disadvantage. Commonly used measures include the proportion of people living on less than 60% of median household income (this was the basis of Waldegrave's selection of low income households), the gap between those on the top and bottom deciles of income, and area-based indices of socio-economic deprivation. All these measures point in the same direction: the number of people in New Zealand who experience hardship as a result of low income is substantial. For example, roughly 20% of New Zealand households with children live on less than $25 000 a year. However, poverty is not a dichotomous variable, and in many ways it makes more sense to assess degrees of poverty than its presence or absence. The relation between life expectancy, for instance, and socio-economic deprivation is continuous: there is no evidence of a threshold beyond which further change in social and economic circumstances has no impact on health status. Moreover, the effects of poverty are far-reaching and are not adequately captured by considering only the health of "the poor". A good example is tuberculosis: for the first time in many years local transmission of this disease is occurring in New Zealand on a significant scale, most likely due to both environmental factors promoting infection (such as crowding) and treatment failures. The incidence is concentrated in disadvantaged sub-groups, but to some extent everyone in the population is at risk as a result of the re-emergence of TB.

The government that will lead New Zealand into the 21st century faces a challenge. New Zealand's slide down the international health status rankings is matched by a drift in economic indicators. Over the last decade and a half average incomes have changed little, but income inequalities have increased. Statistics New Zealand reports that between 1982 and 1996 average household disposable income was static or declined slightly for low and middle income households.[4] However, the disposable income of the wealthiest 10% of households increased by almost a third. The most vulnerable groups in the community, including Maori, were hardest hit by the recession of the 1980s and the subsequent policy reforms. It is important to note that economic disadvantage explains part of the health gap between Maori and non-Maori, but not all. Whatever measures are used to adjust for poverty, disease rates are higher amongst Maori. (And we

should not lose sight of the question of why Maori suffer disproportionate levels of poverty in the first place.)

How should we respond? Encouraging "health lifestyles" in poor areas is, on its own, a futile exercise. We should do more than try to turn poverty into a healthy experience. Better health services for those most in need reduce the harm done by poverty, but do not touch the fundamental causes of the problem. In this vein, the latest World Bank annual report is significant, as it signals an important change in direction.[5] The Bank acknowledges that the policies it has adopted in the past have had limited success and poverty is increasing globally. The way forward, according to the Bank, must be guided by human needs and not narrow economic theory, must build and sustain public institutions, and must be socially inclusive. Income and wealth do not "trickle down", the Bank reports (whoever believed it would?), and economic and social development require stability that can only be provided by a society in which all can participate.

What should we expect from a government in New Zealand? A good start would be to respond to the recommendations in the NHC report, although this considers only actions that can be taken within the health sector.[2] Broader social and economic policies are required in relation to housing, income and education. Also required are strong, well-resourced institutions to make sure that these policies are effective. To paraphrase the World Bank, it is not healthy to have society widely divided along class, ethnicity and income lines. We need a radical response to poverty and this means turning around the policies that presently marginalize and exclude many New Zealanders.

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Tuberculosis in New Zealand: why do we have twice as much as Australia?

Tuberculosis (TB) is an ancient disease and probably emerged when human populations increased and aggregated because of the domestication of cattle.[1] It has had a profound influence on man's thinking and is described frequently in literature; Bunyan called it "the captain of all the men of death." There is a convincing operatic portrayal in La Bohéme (Puccini) and, in the 19th century, some authors speculated that it might bring about the end of European civilisation.[2]

It is estimated that in the 1990s a third of the world's population was infected with TB (1.7 billion) and it was the leading cause of death from an infectious agent.[3] In 1993, the World Health Organisation declared a "global emergency" and in 1995, TB caused more deaths than in any other year in history. Since then the situation has deteriorated and it is estimated that in the year 2000, there will be 10.1 million new cases and 3.5 million deaths.[4] In New Zealand we cannot take comfort in the belief that most of these will occur far from home since 60% of the cases in 2000 will be in our near neighbours in South-East Asia and the Western Pacific, in countries from which we receive migrants. Calder et al in this issue of the Journal discuss a recent outbreak in Auckland. This is a timely reminder that New Zealand cannot afford to ignore the worsening global problem.

TB notifications in New Zealand, which had been declining since the beginning of the century, increased during the 1980s, and have remained high. In the first quarter of 1999, notifications were 11.1 / 100 000. There are big differences between districts, with rates in March 1999 varying between 23.7 in Central Auckland and 17.3 in Wellington to 0 in the Manawatu. Rates are low in the South Island. Australia recently reported rates of 5.47 for the whole population and 1.7 in Australian-born people.[4] Evidence from other countries has shown increased rates of tuberculosis to be influenced by immigration from high-incidence countries and by low socio-economic conditions.

In 1990, Ormerod reported inadequate screening of new immigrants into the United Kingdom (UK).[5] He described a system of sending information about new arrivals to Public Health units and, among the arrivals screened as part of this study, high rates of active disease were found. In the United States (US), the percentage of cases who were foreign-born increased from 21.6% in 1986 to 29.6% in 1993.[6] These authors were concerned about the adequacy of screening of new arrivals and problems with the interpretation of x-rays and sputum examinations in the countries of origin; they noted variation in the quality of these procedures and in local policies and practices; there were cases of deliberate fraud.[7] The Centres for Disease control in the US have recommended screening of foreign-born groups thought to be at highest risk and have asked TB-control programmes to identify barriers to diagnosis and treatment.[8] The province of Milan in Italy has high rates of immigration from developing countries and these immigrants provide 22.8% of all TB cases; 77.2% are diagnosed within five years of arrival.[9] This report shows that most of these cases are due to reactivation of infection which was acquired in the country of origin. It advises measures for early diagnosis of TB disease and also for the detection of dormant TB infection so that chemoprophylaxis can be offered to those at high risk. The success in Milan is attributed to the provision of services that diminish psychological, linguistic and financial barriers which hinder compliance.[10] Australia has immigration regulations which include a "health undertaking for tuberculosis" whereby immigrants who are thought on screening to be at risk of developing TB agree to report to public health authorities within a month of arrival. Compliance in one study was only 58% prompting suggestions that these regulations need to be strengthened.[11]

In New Zealand data from ESR Information Services show that between 1985 and 1998, annual notifications of TB in Europeans fell from 154 to 49 and in Maori from 95 to 55. In the same period notifications of Pacific Island Polynesians rose from 61 to 77 and in those of other ethnicity there was a rise from 49 to 144; in people whose ethnicity was not known, there was a rise from 0 to 15 in this period (Brett M, personal communication). In 1998, there were 340 people notified with TB, of whom 186 were foreign-born, 109 were born in New Zealand and the birthplace of 45 is not known. Of those born abroad the greater number were from Africa (48), South East Asia (35), or the Pacific Islands (35). Eleven were notified within a

month of arrival, 20 between one and three months, 30 between three months and a year, whilst 91 were notified more than a year after arrival. The time since arrival is not recorded for 34 notifications (Brett M, personal communication). The large number of cases being diagnosed in immigrants within one year of arrival has led to concerns about the adequacy of the screening procedures for immigrants and visitors to New Zealand.[12] In 1999, in addition to increasing rates among foreign-born people, there are increases amongst those born in New Zealand, which is an additional and worrying development.

In 1993 Spence in the UK compared notification of TB by Council Ward in Liverpool, over a six-year period, and correlated this with four indices of poverty.[13] He found that notification rates were correlated with all measures of poverty but most strongly with the Jarman index, a composite score designed to include factors such as unemployment, single parent families and people of high mobility. This link was independent of ethnicity. Another study in the UK used the Jarman index to identify relatively underprivileged localities.[14] Between 1988 and 1992, there was a 12% increase in TB notifications in England and Wales. The increase was 35% in the poorest 10% of the population, 13% in the next 20% and in the remaining 70% there was no increase. This study found a major role for socio-economic factors and only a minor role for immigration. Commenting on these findings, Darbyshire referred to 19th century descriptions of TB being caused by "confined and overcrowded dwellings" and a "low state of general health and the resisting power of the body".[15] She considered that medical interventions may be less important than measures aimed at improving the conditions of the poorest section of the population.

In New Zealand we do not have good information about the prevalence of poverty and no studies comparable to those in the UK to look at the relationship between socio-economic disadvantage and health indicators such as TB disease and infection. Data from Statistics New Zealand suggest that, although the gap between rich and poor is widening, this is mainly due to improvements among the better off.[16] However, this report noted that there has been an increase in the proportion of households with incomes of less than 80 per cent of the median. Many working in the health field have subjective impressions that the ranks of the poorly off are growing.

It is disturbing that New Zealand TB rates are more than twice those of Australia, the country with which we most often compare ourselves; high rates in our two largest urban centres need to be explained. One cannot escape the conclusion that immigrants are developing active disease within a year of arrival; the burden of this falls most heavily on vulnerable migrant communities. Whilst those reported in the first month are likely to represent cases detected by arrival screening, the cases notified after the first month and before the end of the first year are probably patients detected by medical services. There is an urgent need for a co-operative study by Public Health agencies and the Immigration Service to determine if current processes for screening immigrants and visitors are adequate; there is concern that they are not.

Historically, TB has been strongly linked to poverty. Studies from the UK show that it still is. In New Zealand we lack information about the relationship between poor living conditions and many diseases of disadvantage. It is sobering to remember that effective application of medical interventions in TB is less than fifty years old and yet incidence had been falling for at least fifty years before that. This was almost certainly due to improvements in the living conditions of the poorest sections of the community. Measures aimed at improving living standards may yet be one of the most pressing issues as we enter a new millennium.

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Prioritisation and cardiac events while waiting for coronary bypass surgery in New Zealand

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Generating numerical ranking systems for public hospital waiting lists has become a boom industry, but application of scoring systems to several specialties in New Zealand has met with considerable reservation. Priority criteria for coronary artery bypass grafting (CABG) were developed in Ontario in 1989[1] and subsequently modified and expanded by cardiologists and cardiac surgeons at Green Lane Hospital.[2] Further modifications were carried out by cardiologists and surgeons throughout New Zealand with coordinators contracted by the National Health Committee and Regional Health Authorities.[3]

At the outset clinicians emphasised that "ranking systems are no more than aids to establishing priorities". If the resource is inadequate "there is no equitable or clinically acceptable way to modify priorities to reduce waiting times".[2] Despite these concerns Minister of Health Mrs Shipley decreed in May 1996 that public funding for CABG would be available only for those patients with a prioritisation score of 35 or greater, and all patients with a lower score were removed forthwith from public hospital waiting lists. This was judged by government to be the

financially sustainable level. On the basis of extensive data clinicians insisted that a base score of not more than 25 with flexibility for patients on the boundary was clinically realistic, but the level of 35 was enforced with the undertaking to review the situation at some unspecified later date - not yet reached.

Three recent papers have appeared in Heart assessing management of waiting lists for CABG, one from Holland (which also included valve surgery),[4] and the others from Christchurch[5] and Green Lane Hospitals.[6] Outcomes in the New Zealand studies were deaths and acute ischaemic events leading to hospital readmission. In the periods covered by the New Zealand studies referral for CABG was based on agreed clinical criteria, not quantified by a scoring system. In each study both the Ontario Urgency Score[1] and a further modification of the New Zealand score[3] (including the important factor of left ventricular function) were calculated retrospectively for each patient and related to outcomes.

For comparison the death rate for patients with stable angina is around 1.1 per 100 patient years.[7] Deaths on the waiting list for coronary surgery in Ontario were 0.4 per 100 patient years which the Canadians thought unacceptable. In Holland Plomp reported a death rate of 7 per 100 patient years in weeks 2-4, falling to 3.5 beyond 12 weeks. Comparable Christchurch and Green Lane figures are shown in the table. During the study periods Green Lane patients averaged higher scores, but Christchurch scores have since risen to the mid 50's. There is one major difference in management, the remarkably high percentage of Green Lane patients who remained in hospital from referral until operation. Compared with the Christchurch study this was associated with a highly significant reduction in death rate, unstable angina and myocardial infarction after referral. Despite this the mortality of the generally lower risk Green Lane patients assigned to the waiting list approached that of the Christchurch cohort. The Green Lane policy appears unique in recorded literature but it carries the significant cost of an average five pre-operative days in hospital for those treated acutely. This waste of money is imposed by inadequacy of the clinical resource.

Table 1. Waiting list characteristics: Christchurch and Green Lane Hospitals.

Years	Christchurch 1994-5	Green Lane 1993-6	p value† differences
Number of patients	324	1422	
Coronary score*			
: all patients	46	55	
: operation on index referral	78	64	
: operation from waiting list	41	45	
Percentage of patients			
: operation on index referral	21%	51%	
: unstable angina on waiting list	34%	14%	0.001
: infarct on waiting list	6%	2%	0.003
: died after listing	4%	1.5%	0.004

*Approximate Green Lane Hospital scores were calculated from Tables 5 and 6.[6]
†Fisher's exact test.

What were the effects of setting a high threshold for CABG in the public sector? At Green Lane 130 patients with a score below 35 were removed from the waiting list and 125 have been followed. Twenty months later about one third remained unlisted, one third were returned to the waiting list usually because of deteriorating clinical status, and one third were admitted with acute coronary syndromes. Five have died (one after operation). Fifty-nine have undergone surgery in the public sector (24 as emergencies) and nine in the private sector. Hospital costs of assessment, treatment and emergency operation were around 40% higher than those of an operation following a controlled admission. Even though some increased funding has subsequently been made available to boost surgical numbers, the arbitrary cut off entry level has manifestly cost both lives and money. The additional unassessed social and economic costs in the community, including those for treatment of acute ischaemic episodes, are considerable. A recent study from Christchurch reports that "patients are frequently distressed in many ways while waiting" and "many patients lost paid employment due to worsening angina while waiting".[8] Arbitrary restraints on public health funding, ignoring clinical imperatives, produce classic false economy.

Funding available for health care will always be limited. Two myths, assiduously promulgated from political sources, could however be usefully put to rest. First, in most countries the demand for expensive health care is not unlimited. The exception, manifested in the USA, is the society where a high proportion of health services have a profit-making prepaid insurance basis which has the perverse influence of encouraging over-consumption. A number of Western countries, including Australia, approach optimal medical care with manageable funding. Most New Zealanders are prepared to accept tolerable disabilities but become angry when denied effective treatments which can have a big impact on lifestyle or greatly reduce the likelihood of premature death. The second myth is that we have been faced with an explosion of public health expenditure. In the years 1980 to June 1998 total health funding from government sources and ACC rose 1% per year per capita in standarised dollars.[9] Similar public funding for hospital services (whether provided in public or private) rose 0.4% per year.

These figures define an exceptionally feeble explosion. The increase required to provide a balanced and cost effective service throughout all sub-specialties is not enormous, and our public deserve an honest presentation of current expenditure and the consequences of extreme financial restraint. As has been shown already in a number of specialties, a scoring system for waiting lists has a role but it is a blunt instrument. Common sense is required to set the appropriate level and allow flexibility. To date the innovations have been driven too much by emphasis on short-term cost saving, and unrealistic cut off points perpetuate inequity and inefficiency.

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ORIGINAL ARTICLE

A school and community outbreak of tuberculosis in Auckland

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Abstract

Aim. To describe a school and community outbreak of tuberculosis in South Auckland in 1997/8.

Methods. Cases were diagnosed according to national guidelines at Middlemore, Green Lane and Starship Hospitals. Public health follow-up was conducted by Auckland Healthcare.

Results. Twelve cases were diagnosed during the outbreak. Nine cases were from the same South Auckland secondary school; six reported no association outside school. Three cases were in younger children who had close household contact with two of the school cases. Nine cases (including eight from the school) had identical *Mycobacterium tuberculosis* isolates on restriction fragment length polymorphism testing. No microbiological culture was obtained from the three remaining cases. Contact investigation detected five of the cases. Chemoprophylaxis was prescribed for twenty-six school students, two adult staff, and nine household contacts.

Conclusion. This is the first published account of a tuberculosis outbreak in a New Zealand school setting for decades. Recognition of the outbreak was delayed. DNA fingerprinting played a valuable role in the investigation. The source case may have been a school student. The social impact of the outbreak and preventability with routine adolescent BCG vaccination are discussed.

NZ Med J 2000: 113; 71-4

A series of tuberculosis (TB) cases were diagnosed in late 1997 and early 1998 in a South Auckland secondary school. This report describes the course of the outbreak, the clinical cases, the contact investigation, the role played by molecular typing and the control measures adopted.

Methods

Cases were diagnosed and treated at Middlemore Hospital, Green Lane Hospital and Starship Hospital in Auckland on the basis of standard guidelines.[1]

DNA fingerprinting was performed according to the standard method proposed by van Embden et al.[2] Briefly, the technique entails the growth of *M tuberculosis* and extraction of genomic DNA, which is digested with the restriction enzyme Pvu II. A Southern blot is performed and probed with a 245 base pair fragment of insertion sequence IS6110. The reference strain of *M tuberculosis*, Mt 14323, was included as an internal control and known band size markers. Isolates from two unrelated cases and one isolate of *M bovis* were used as additional controls.

Public health investigation of household and school contacts by the Public Health Protection Service of Auckland Healthcare involved 5 tuberculin unit Mantoux testing and, if indicated, a chest x-ray.

Students were asked to take home explanatory letters and consent forms for informed parental consent for Mantoux testing and radiologic follow-up. Letters were sent and home visits made by a public health nurse to non-responding parents. If parents still failed to respond they were visited by specially trained Pacific Islands or Maori members of a community group who provided education about TB and sought consent for testing. Parents who still did not respond were visited by an out-of-hours public health nurse.

Cutting points adopted for a positive Mantoux test followed national guidelines for school teachers and household contacts.[1] For the school students, however, we departed from the national guidelines by adopting a single cutting point, whereby a Mantoux reaction ≥ 10mm was considered positive. Past BCG vaccination status was considered positive if there was a scar overlying the insertion of the left deltoid muscle.

Results

Cases. Features of the cases are summarised in Table 1. The relationships between the outbreak cases are shown in Figure 1.

Cases 1-6 were diagnosed between December 1997 and February 1998. Case 1 was an adolescent at the secondary school and had no contact with case 2, a two year old child with infectious disseminated TB. Cases 3-6 were household contacts of case 2. Cases 3 and 4 presented at Middlemore Hospital with symptomatic disease before their contact screening procedures could be completed. Cases 5 and 6 were asymptomatic and were detected by contact screening. Five of the first six cases were members of the same household. In May 1998 there were a further two notifications (cases 7 and 8) in students attending the school and one of these was sputum smear positive. Contact investigation detected three more asymptomatic cases (9, 10 and 11). All had active pulmonary TB. Case 12, who had been a casual contact of one case in school and one case outside school, had left the school in March 1998 and presented to Middlemore Hospital with severe pulmonary TB almost one year later.

The cases were all of Pacific Islands ethnicity but only two were born overseas. All nine *M tuberculosis* isolates obtained were fully susceptible to first-line antituberculous agents and shared an identical restriction fragment length polymorphism (RFLP) pattern (Figure 2), including eight of the school students (of whom only four had any association outside school). In two of the other three cases no attempt was made to isolate an organism as epidemiological and x-ray findings were thought to be adequate for a diagnosis. Only one of the 12 notified cases (case 8) was an obvious infectious risk to others, having strongly smear positive sputum for the first four weeks of antituberculous treatment and symptoms which lasted for eight weeks after treatment started. This case had extensive shadowing in both lung fields on chest radiograph. None of the cases identified during the first phase of the outbreak (cases 1 - 6) appeared to be a major infectious risk, judging by sputum smear negativity (for acid-fast bacilli), lack of cough and sputum, and minimal or no chest radiograph abnormalities. Seven of the nine school cases carried scars consistent with past BCG vaccination. All cases were treated by directly observed therapy.

Table 1. Outbreak cases.

Case	Age*	Born in NZ	Past BCG vaccination	Date of notification	Approx date of symptom onset	Site of disease	Smear positive specimen	Culture positive specimen	Susceptible to HREZS	Outbreak RFLP pattern
1	14	Y	N	29/12/97	18/12/98	Pulmonary and pleural effusion	N	Pleural aspirate	Y	Y
2	2	Y	N	20/01/98	06/01/98	Miliary	Gastric aspirate	Gastric aspirate	Y	Y
3	15	N	Y	13/02/98	06/02/98	Pulmonary	N	Sputm, gastric washings	Y	Y
4	16	N	Y	18/02/98	01/02/98	Pleural effusion	N	Pleural aspirate	Y	Y
5	5	Y	N	18/02/98	Nil	Pulmonary	Not done	Not done	NA	NA
6	9	Y	Y	18/02/98	Nil	Pulmonary	Not done	Not done	NA	NA
7	15	Y	Y	08/05/98	07/04/98	Pulmonary	N	N	NA	NA
8	15	Y	N	16/05/98	01/03/98	Pulmonary	Sputum	Sputum	Y	Y
9	16	Y	Y	22/09/98	Nil	Pulmonary	N	Sputum	Y	Y
10	16	Y	Y	04/09/98	Nil	Pulmonary	N	Sputum	Y	Y
11	16	Y	Y	18/05/99	Nil	Pulmonary	N	Induced sputum	Y	Y
12	17	Y	Y	16/02/99	01/01/99	Pulmonary, pleural effusion, nodal	Pleural biopsy	Sputum	Y	Y

Y= Yes; N= No; NA= Not applicable; HREZS= isoniazid, rifampicin, ethambutol, pyrazinamide, streptomycin; * on 29/12/97, the date of notification of the first case.

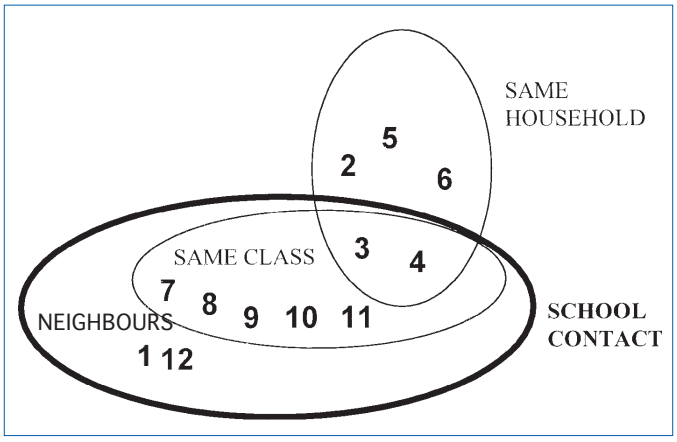

Figure 1. Relationships between the outbreak cases.

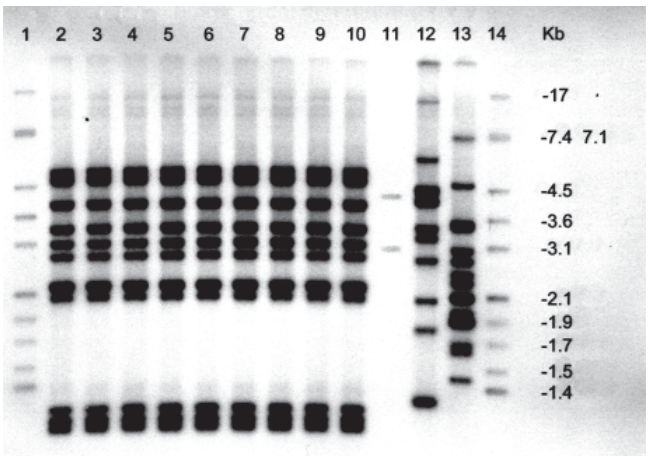

Figure 2. DNA fingerprints of *Mycobacterium tuberculosis* strains. Lanes 1 and 14 Reference strain Mt 14323; Lanes 2-10 Cases 1,2,3,4,8,9,10,11,12; Lanes 11-13 Unrelated *M. tuberculosis* isolates; Kb = kilobase.

Contact investigation. Since no family contacts of case 1 were infected, school contacts were not screened. A number of household contacts of case 2 were found to be infected. After notification of cases 3 and 4 limited screening was conducted in the school. Testing of four students and 15 adult staff yielded no cases and only one Mantoux-positive person. Cases 5 and 6 were children and screening was confined to household contacts. After the notification of cases 7 and 8, we extended the contact investigation in the school to cover all students who had been exposed to any school case. We also requested RFLP typing, which had just become available in Auckland Healthcare, of the *M tuberculosis* isolates. The extended contact investigation in the school revealed three further cases (9, 10 and 11). The contact investigation was extended further to encompass all unscreened students in the school and the remainder of exposed staff. The community group which assisted with the investigation obtained 123 of 148 outstanding consents (83%) within two weeks. The results of the contact investigation are shown in Table 2. Twenty-one household contacts and 40 school contacts were found to be Mantoux positive. No adult source case of lung disease was discovered despite screening of 42 adult household contacts and 45 adults among school staff. Ten school staff were not screened (one refused and nine were lost to follow-up).

Chemoprophylaxis of contacts. Thirty-seven people were placed on antituberculous chemoprophylaxis. For Mantoux-positive students this was carried out by directly observed preventive therapy (DOPT). Duration of chemoprophylaxis varied depending on the public health staffing available and the proximity of the start of treatment to the summer vacation. Three students received six months' uninterrupted isoniazid (H); 19 students received four months' uninterrupted rifampicin and isoniazid (RH); four students received five months' RH interrupted by the summer vacation (eight weeks). Standard doses of R and H were prescribed for twice-weekly treatment.[1]

Discussion

We have described a school- and community-based outbreak in which twelve cases of TB and 61 Mantoux-positive people were identified.

In retrospect, recognition of the school outbreak was delayed. Initially only close household and very few school contacts were tested. No household contacts of case 1 were infected, indicating low infectivity of this case. Therefore no school contacts were examined. Cases 2, 3 and 4 shared close household or social relationships outside the school and only case 2, a preschool child, was smear positive. Limited screening of staff and student contacts of cases 3 and 4 yielded only one Mantoux-positive person, while there was extensive tuberculosis infection and disease in the household. At this stage we were unconvinced of transmission within

Table 2. Outcome of the contact investigation of twelve cases of tuberculosis.

		Number of contacts identified	Number (%) tested†	Number (%) refusing testing	Number (%) lost to follow-up	Number (%) not tested as already known to have clinical disease	Number (%) not tested owing to past disease
School contacts	Students	491	445 (91)	3 (1)	33 (7)	5 (1)	5 (1)
	Adults	55	45 (82)	1 (2)	9(16)	0	0
Household contacts	Children	34	34(100)	0	0	0	0
	Adults	43	42 (98)‡	0	0	0	0
		Number (%)§ Mantoux negative	Number (%) Mantoux Positive	Number (%) identified with clinical TB disease	Number (%) on chemoprophylaxis	Number (%) followed up with serial chest X-rays	
School contacts	Students	415 (93)	30 (7)	3 (0.7)	26 (6)	1(0.2)	
	Adults	35 (78)	10 (22)	0	2 (4)	8 (18)	
Household contacts	Children	27 (79)	7 (21)	2 (6)	4 (12)	0	
	Adults	28 (67)	14 (33)	0	5 (12)	8 (19)	

† Percentages do not add up to 100% due to rounding; ‡ One person not tested due to concurrent illness; § % = percentage of those tested.

the school, even though school staff were expressing concern about the unusual number of cases. This initial cluster of cases was in a community with a high proportion of Pacific Islands students, an ethnic group with known high rates of TB infection. It was only with the appearance of two further school cases, one of whom was sputum smear positive (case 8), and the availability of RFLP testing that an outbreak could be confirmed.

RFLP testing played a valuable role in confirming the findings of the epidemiologic investigation. Experience to date with RFLP testing of approximately sixty *M tuberculosis* isolates in Auckland indicates a high degree of strain heterogeneity (personal communication, Arthur Morris, 4/3/99). Therefore the identical DNA pattern found in the isolates in this outbreak provides powerful evidence of person-to-person transmission within the school, particularly since five of the nine school cases reported no contact with each other outside school. RFLP testing could not be conducted on three of the cases (5,6,7) from whom no *M tuberculosis* was isolated. We are however, highly suspicious on epidemiological grounds that they were part of the outbreak.

Accounts have been published of outbreaks in primary[3-7] and secondary[8-13] schools in several countries. In these reports an adult source case was often implicated. We did not discover an unequivocal source case for this school outbreak. Case 8 is the most likely source. It is puzzling that the reported onset of symptoms in case 8 was ten weeks after the onset in case 1. However, case 8 had extensive chest radiographic shadowing on presentation, slow radiographic improvement and prolonged sputum smear positivity despite aggressive antituberculous treatment. We believe that active tuberculosis must have been present for many weeks before presentation. Alternative sources include one of the other school cases who may have spontaneously remitted to a less infectious stage by the time of diagnosis, an undetected adult source case in one of the students' households, or a source among staff or students at the school whom we were unable to screen. No such case, however, has presented spontaneously since the end of the outbreak. Case 12, who presented in February 1999, had no symptoms until ten months after leaving school and was an unlikely source.

National guidelines[1] would have required us to adopt different cutting points for a positive Mantoux test for different students depending on age, closeness of contact and BCG status. In a departure from the national guidelines, we adopted ≥ 10mm as the cutting point for a positive Mantoux test for all students in the school investigation. Our reasons were twofold: to increase the sensitivity of the test for detecting infection, thus increasing our chances of controlling the outbreak; and because of the administrative complexity of determining and explaining different cutting points for each student during a large testing programme.

A second Mantoux test was not carried out to test for conversion because more than twelve weeks had elapsed between the departure of the last infectious case and the commencement of testing.[1] Some of the positive Mantoux results would therefore have been due to remote infection or vaccination, rather than exposure during this outbreak.

We wanted to ensure compliance with chemoprophylaxis by Mantoux-positive students. We did not consider that self-medication was suitable and instituted DOPT at school. The usual regimen used in New Zealand is six months uninterrupted isoniazid taken daily.[1] Since community-based DOPT for all students was beyond our staffing resources during the summer vacation, we adopted alternative prophylactic regimens for some students: five months' RH, with interruption of the regimen during the vacation; or four months' uninterrupted RH. Evidence for the effectiveness of shorter regimens has been published.[14]

The school's routines were heavily disrupted by the public health investigation and follow-up. Intense local and national media attention stretched the resources of the school and the public health service during the outbreak. The publicity had an adverse affect on the morale of the school, generating shame among the students and alarm amongst staff. This caused considerable distress in the school community, which may in turn have led to the difficulties we experienced in conducting contact screening. Obtaining consent was time-consuming. The loss-to-follow-up rate of 7% among students was achieved only by a substantial input of resources. Our employment of a community group to obtain outstanding consents for screening demonstrated that it is possible to train community members rapidly even if they have no health background. Stigmatisation of cases and their families is a continuing obstacle to effective TB control. The difficulties encountered in this investigation suggest that in-depth education about TB should be planned with affected communities.

This outbreak should be viewed with concern. In the experience of public health offices, tuberculosis transmission in New Zealand usually occurs in family groups as a result of household contact. We could find no reports of school outbreaks, (i.e. transmission within the school setting), in New Zealand since 1966, in either Medline or the records of

the New Zealand Communicable Disease Centre (personal communication, Yvonne Galloway). Only two of the cases were born outside New Zealand, which raises the possibility that declining social conditions in New Zealand were a factor.

Seven of the nine school cases carried scars consistent with past BCG vaccinations. These must have been received earlier in life since routine vaccination in secondary school was discontinued in Auckland in the 1980s. Further outbreaks of this magnitude will raise the question of whether routine tuberculin screening and BCG vaccination and/or chemoprophylaxis of adolescents should be reintroduced in schools which have high numbers of Pacific Island, Asian and African students, given that in New Zealand these ethnic groups have high rates of TB.

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Problem drinking profiles of patients presenting to general practitioners: analysis of Alcohol Use Disorders Identification Test (AUDIT) scores for the Auckland area

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Abstract

Aim. To quantify the prevalence and demography of at-risk and problematic drinkers in the population attending a random selection of general practices and to compare this with similar studies.

Method. A study examining the uptake and utilisation of the "DRINKLESS" package to 369 New Zealand general practitioners was conducted during 1995/6. The "DRINKLESS" package was developed with the World Health Organisation collaborative study for brief intervention for at-risk alcohol consumption. The package uses the Alcohol Use Disorders Identification Test (AUDIT). There were 15 670 completed AUDIT questionnaires collected during the study. These were analysed to ascertain the prevalence and demography of at-risk and problematic drinkers attending general practitioners.

Results. There were 16% of patients identified as having either "risky drinking" or "problematic or dependent drinking". This pattern varied according to the occupation, age and gender of patients.

Conclusions. The data confirm that large numbers of patients presenting to general practitioners experience alcohol problems of varying degrees. This study also suggests that the AUDIT will have satisfactory detection rates in a primary care setting.

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New Zealanders experience a substantial number of alcohol-related problems. Wyllie et al[1] found that 26% of men and 16% of women in a 1990-2 Auckland survey of 4662 respondents reported some level of harm from their own drinking in the previous twelve months. In 1986, Wells et al[2] used the Diagnostic Interview Schedule to study 1498 Christchurch residents aged 18-64 years and found that men were more likely than women to experience DSM III classified alcohol abuse/dependence at some stage in their life; (lifetime prevalence of 32.0% for males and 6.1% for females; 14.1% and 2.6%, respectively, looking at the previous six months). Recently commissioned research by the Alcohol Advisory Council of New Zealand suggested approximately one in four teenagers to be at-risk or problematic drinkers.[3]

A large number of people experiencing problems with alcohol present themselves to general practitioners, possibly at a rate twice the average.[4] Chilvers[5] recently surveyed 500 consecutive consultations with patients over the age of 17 years in Wairoa, a small rural New Zealand town. Of the 500 patients surveyed 195 were male and 305 female; 44 males (22.6%) and 36 females (11.8%) were drinking above the Alcohol Advisory Council of New Zealand's recommended maximum weekly levels, which is 16.0% of those questioned. McMenamin[6] surveyed 202 patients aged 18-29 years who had previously participated in a screening health check in his general practice using the AUDIT. Sixteen (11 men and 5 women) were classified as at-risk drinkers and a further 11 (8 men and 3 women) met the criteria for alcohol use disorder according to DSM III R.

These data calculated 13.4% of the screened group as either at-risk (7.9%) or dependent drinkers (5.4%) with men outnumbering women by over 2:1.

The general practice setting represents an important opportunity to deliver health education/promotion and early intervention for problematic and risky alcohol use - especially given that most people with alcohol problems do not present to specialist treatment services.[7] A general practice-based strategy has the following advantages. Firstly, general practitioners are highly accessible to the population requiring interventions. Secondly, general practitioners have role legitimacy with respect to the delivery of advice about alcohol consumption.[8,9] Thirdly, there is reason to believe that brief interventions delivered by general practitioners have a positive impact on the alcohol consumption levels of their patients. A comprehensive review of early intervention studies documented seven out of eight randomised controlled trials in health settings demonstrating significant reductions in alcohol use after early intervention, five of which involved delivery of advice by general practitioners.[10] Putting all of these factors together, it might be expected that a concerted effort by general practitioners would have a substantial impact on the level of alcohol-related harm in New Zealand society.

Unfortunately, the uptake and utilisation of brief intervention strategies by general practitioners remains very limited. Gomel et al[11] commented that the potential of general practitioners to reduce the prevalence of alcohol-related problems contrasts sharply with current practices. General practitioners fail to identify a large proportion of patients who are drinking at harmful or at-risk levels. This statement is supported by studies such as those by Rydon et al,[12] which show between 65.0% and 82.3% of patients with alcohol-related problems as identified by consumption levels or by screening tests such as the CAGE, are not identified in general practice. Similarly, Reid et al[13] found that general practitioners were able to identify only 27.5% of patients classified by Australian Medical Association criteria as "high risk" drinkers and 45.2% of patients classified as "moderate to heavy" drinkers.

One strategy for encouraging general practitioners to take a more active role with respect to alcohol problems is to provide support around screening and brief intervention. As part of a World Health Organisation (WHO) collaborative project the authors have previously explored the incentives and disincentives for general practitioners providing interventions.[14] One barrier identified was "Doctors do not have a suitable screening device to identify problem drinkers who have no obvious symptoms of excess consumption".[15] This has been addressed within the WHO collaborative study by the development of the DRINKLESS package.[16]

A suitable screen is necessary because alcohol problems are not usually immediately apparent. Clients may also be unwilling to volunteer information about their situation for fear of being labelled an "alcoholic" or otherwise stigmatised. Fortunately, screens for alcohol problems can perform significantly better at detecting problems than physicians operating without the use of such tools.[17,18] A cross-national dataset rated the sensitivity of the AUDIT, for example, at 92% for hazardous and harmful alcohol use combined and its specificity at 94%.[19]

This paper is based on the analysis of 15 670 AUDIT questionnaires completed as part of a research project studying the distribution of the DRINKLESS screening and early intervention package.[20] The main aims of the project reported in this paper are to: 1. Quantify the prevalence of patients with either "risky drinking" or "problematic or dependent drinking" (according to AUDIT criteria) attending the selection of general practices surveyed in our earlier study.[20] The purpose was to affirm the value of conducting alcohol problem screening within the general practice setting. 2. Compare our findings with other surveys to ensure, or otherwise, that our findings were generalisable to general practice as a whole. This was because our sample was drawn from a study not expressly designed to survey general practice consultations.

Methods

General practitioners (all from separate practices within greater Auckland) were selected by computer random number generation from a database of just over 900 individual general practitioners maintained by the Department of General Practice, University of Auckland. The first general practitioner chosen from each separate practice was entered into the study; subsequent general practitioners from the same practice were not enrolled. Random selection ceased once 369 general practitioners were identified, each belonging to a separate general practice.

The intervention procedure delivered to each doctor was the "DRINKLESS" package developed with the WHO collaborative study for early intervention for at-risk alcohol consumption.[16] The DRINKLESS package included information for the doctor, receptionist and patient, the AUDIT alcohol screening questionnaire and a scoring template. A standard process was prepared to ensure waiting room screening of every patient 16 years of age and over.

Of the 369 Auckland doctors sampled, 237 agreed to receive the DRINKLESS package and 96 agreed to utilise the package. Of these, 83 started and 65 provided a complete data set. Among the surveys and other data collected during this second study was a database of 15 670 completed AUDIT questionnaires.

The AUDIT alcohol screening questionnaire was developed from a six-country WHO collaborative project for use in primary health care settings.[19] The AUDIT has ten items which cover the domains of alcohol consumption, drinking behaviour and alcohol-related problems. Each question has a minimum score of 0 and a maximum score of 4 thus giving a range for the AUDIT of 0 to 40. The questionnaire classifies scores of 8 to 12 as indicating "risky drinking" and scores of 13 or more as indicating "problematic or dependent drinking". Unlike most other screening instruments for alcohol disorders, the AUDIT is concerned with identifying those with risky or problem drinking, not exclusively those with alcoholism.

The raw AUDIT total and item scores were entered into a computer database and then analysed using SPSS v8.0. The AUDIT scores were recorded into the main divisions - "responsible drinking", "risky drinking", and "problematic or dependent drinking". Analyses of variation in all of these categories were conducted for the total population and for different subgroups identified by gender, age and occupation. Separate analyses of AUDIT scores for male and female patients were conducted according to age.

Analyses of the relationship between occupational grouping and grouped AUDIT score were conducted using the chi-squared test. Yates' continuity correction was applied. The relationship between AUDIT scores and gender was analysed using the Mann-Whitney test. The distributions were insufficiently parametric to use the t-test.

Results

Approximately one in every six people (16.0%) who completed one of the 15 670 AUDITs was identified as having either "risky drinking" (10.2%) or "problematic or dependent drinking" (5.8%). In some general practices there were substantially higher proportions in these two categories. The maximum recorded was 38.8% of patients having either "risky drinking" or "problematic or dependent drinking" (N=468 patients).

The pattern varied considerably according to the occupation of the patient. People in the "income support", "elementary", "trades", or "machinery" categories had over four times the chance of scoring with "problematic or dependent drinking" compared with people in other occupations (14.5% vs 3.6%) ($\chi^2 = 551.2$, df = 1, $p < 0.001$). The percentage for "income support" was the highest at 18.9%. Looking at the other end of the continuum, people in the "retired", "home responsibilities", "professional", or "clerk" categories were less than a fifth as likely to receive a "problematic or dependent drinking" AUDIT score

compared with people in other occupations (1.8% vs 9.4%) ($\chi^2 = 409.2$, df = 1, $p < 0.001$).

As can be seen from Figure 1, there is a clear relationship between age and overall AUDIT score; as we look at increasingly older populations there is a substantial decrease in the percentage identified with either "risky drinking" or "problematic or dependent drinking". Patients aged 18-29 are the most likely to receive high overall scores.

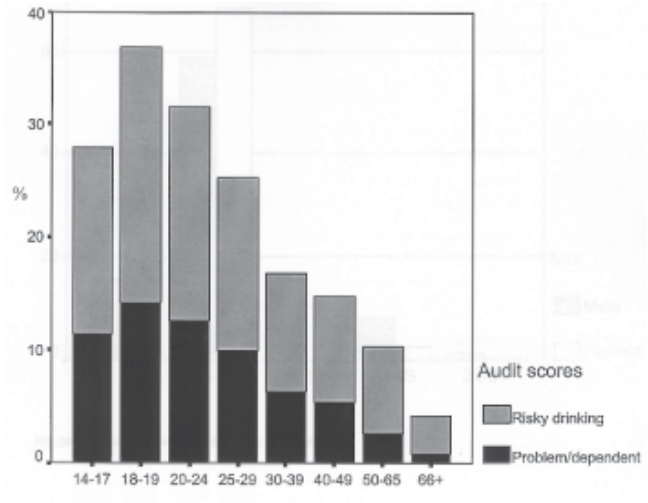

Figure 1. Alcohol problems by age.

Our results show that approximately one third (32.0%) of young people under the age of 25 years attending general practitioners are drinking at either "risky drinking" levels (19.2%), or at "problematic or dependent drinking" levels (12.7%). This means they are either at risk of problems with their health, the law, relationships or their work, or they are already experiencing problems. Patients aged 18-24 had the highest scores and patients over 66 the lowest scores. To some extent the lower scores in the older age groups represents the effect of ageing on levels of consumption. Another factor is that patients with heavy drinking practices are more likely to die earlier.

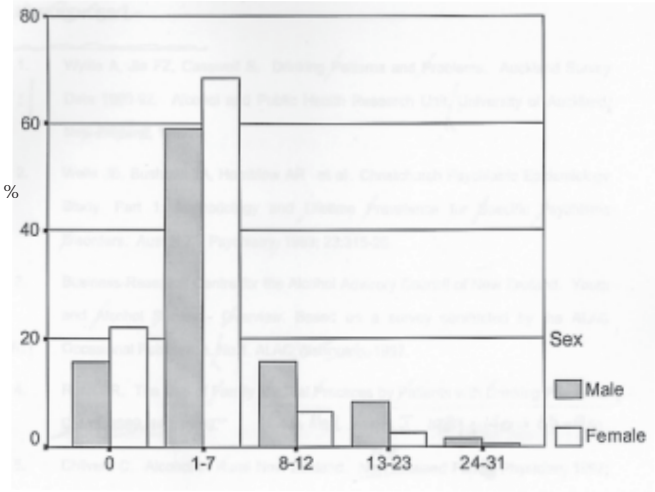

Figure 2. Audit scores grouped by gender.

Male patients gained significantly higher AUDIT scores than female patients (Figure 2). The average AUDIT score for men was 5.5 while the average score for women was 3.2 ($t = 29.1$, df = 10396, $p < 0.001$). The gap between males and females was smallest for fourteen to seventeen-year-olds but was pronounced thereafter.

Focusing on the interaction of gender and age, males and females both begin their drinking careers with almost identical AUDIT scores according to our data. However, females quickly move to an average score more than three points lower by age group 20-24 (8.4 for males and 5.0 for females). This gap is maintained through all age groups until the sixties when it begins to noticeably narrow.

Looking at male scores in isolation, the two age ranges with the highest scores were 18-19 and 20-24, with nearly half (47.8% and 47.3%, respectively) being identified as either "risky drinking" or "problematic or dependent drinking".

Discussion

Before discussing the results of the data analysis, two sources of potential bias should be mentioned. The first is self-selection bias on the part of the general practitioners. It is conceivable that the general practitioners who declined to utilise the DRINKLESS package may have differed in some significant way from those who agreed. It is possible, for example, that willingness to participate fully was influenced by the perceived level of problems in the patient population.

A second potential threat to the generalisability of our results to general practice consultations as a whole, could be that doctors selectively chose to screen those they expected to have alcohol problems. The overall demography of those completing our AUDIT screens was compared with that obtained in the Waimedca study.[21] This 1992 study surveyed general practitioner consultations around Hamilton, a medium-sized New Zealand city. This enabled comparison of their data with the age and gender distribution of those completing our AUDIT screen. There was no indication that doctors had selectively screened those who they suspected may have an alcohol problem as the two populations appeared almost identical on the axes available for comparison.

The average finding that 16.0% of the population were either "risky drinking" or "problematic or dependent drinking" is the same as the percentage found by Chilvers[5] and only slightly higher than McMenamin's figure of 13.4%.[6] Taking into account the age range of patients included in McMenamin's sample (18-29), however, his figures are much lower than for a comparable selection of patients from our sample (29.4%). His lower figure may have been produced by some of his heavy drinkers being unwilling to participate or, given the sample size of 202, may just represent random variation. Wyllie[1] and Wells[2] both measured different indicators of problem drinking in the community but their results are not inconsistent with our own.

Results from other studies supported the finding that higher alcohol problem levels were associated with youth. In the research of Wyllie et al,[1] respondents aged 18-24 were most over-represented in the top 10% of drinkers by alcohol consumption and half of heavier drinking women were in this age group. Data on alcohol consumption and the frequency of heavier drinking showed a peak at ages 18-19 and a steady decrease thereafter. Age was also a strong predictor of alcohol-related problems. Grant's United States data[22] show 18-24-year-olds as having the highest rates of alcohol dependence according to DSM-IV criteria.

Male patients in this study had higher AUDIT scores than female patients. Once again, other studies tended to support this analysis. This finding was entirely consistent with the McMenamin,[6] Chilvers[5] and Wells[2] findings discussed in the introduction. Chilvers and McMenamin both report male to

female problem drinking ratios of about 2:1. The study by Wyllie et al[1] reported that males were substantially over-represented in the top 10% of drinkers and that more males than females reported feeling drunk as frequently as once a week (14% and 4%, respectively). In contrast, however, gender was found to have only a weak relationship with most measures of alcohol-related problems. In Grant's study,[22] male dependence rates as measured against DSM-IV criteria were higher than female (6.3% and 2.6%, respectively). The gap reported between the AUDIT scores for male and female patients is indirectly supported by Wyllie et al,[1] who showed similar male/female rates of alcohol consumption and heavier drinking in the 14-17 age group, with males rapidly gaining higher rates thereafter.

In general terms, this study has confirmed that large numbers of patients presenting to general practitioners experience alcohol problems of varying degrees. The therapeutic task for the doctor is to shift patients with alcohol-related problems along the continuum to a safer point. General practitioners can realistically achieve a considerable amount without having to "cure" all their problem drinking patients. It is perhaps in recognition of this fact that there has been increasing interest in the role of general practitioners in alcohol treatment in recent years.[23] Another general finding of this study is that screening using the AUDIT or a comparable tool is likely to have a satisfactory detection rate. There is no need for concern that screening will entail a large amount of work with little tangible benefit for patients.

Screening strategies can be customised in a number of ways to suit a particular need. Options include the incorporation of alcohol screens into broader lifestyle surveys, the use of waiting-room time to administer earlier steps in the screening process, the use of self-administered computerised surveys and an expansion of the practice nurse role in this area.[6,24]

None of this is likely to remove all potential concerns about screening. Anderson,[23] for example, quotes one general practitioner expressing what might be a common source of trepidation: "One of the things I don't do is ask too many questions because I don't want to uncover a whole lot of things I can't deal with ... I am not going hunting out problems I don't know how to treat". But it does suggest that New Zealand has a lot to gain if it can find a way of harnessing the potential of general practitioners in the delivery of alcohol interventions.

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Blood donation by healthy individuals with haemochromatosis

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Abstract

Aim. To determine how many individuals with haemochromatosis undergoing therapeutic venesections in our department might be eligible for blood donation.

Methods. Patients with genetic haemochromatosis were assessed with respect to complications of their disorder, the presence of other medical conditions and their suitability to be blood donors.

Results. Of 74 patients, 53 have been tested and shown to be homozygous for the Cys282Tyr mutation, and 30 of these (40%) fulfilled criteria for blood donation. This group is having 409 units of blood removed annually or 13-units per individual that is currently discarded. Of these 30 patients, all (100%) were keen to be blood donors.

Conclusion. It is timely to review the policy regarding use of this wasted blood for transfusion.

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Genetically defined haemochromatosis, an autosomal recessive disorder of iron metabolism, affects 1 in 200 of those in populations of northern European origin, with more than one in ten being heterozygous asymptomatic carriers.[1] Advanced tissue iron accumulation can cause cirrhosis, cardiac damage, arthritis, diabetes, hypogonadism, hepatocellular carcinoma and reduced life expectancy.[2] Early identification and treatment that reduces and maintains iron levels at a low level prevents the development of these complications and such individuals have a normal life span.[3] Diagnosis is now easier with the availability of a genetic test to identify homozygosity for a G to A mutation which results in a cysteine to tyrosine substitution at position 282 (Cys282Tyr) of the HFE gene which is present in approximately 90% of patients.[4] This test was used by us to investigate those with high iron saturation and/or ferritin and/or liver disease and/or other organ dysfunction, and to screen individuals with a family history of haemochromatosis.

Venesection on a regular basis effectively removes the excess iron.[2] Currently in New Zealand the blood removed from these patients is often discarded. If affected individuals were otherwise eligible to be blood donors, such disposal could be considered a waste of a valuable resource. Since the NZ Blood Service (NZBS) is developing policies to ensure standard national practice (personal communication Dr Peter Flannagan, Medical Director, NZBS), it seemed appropriate to review patients with haemochromatosis to determine what proportion is potentially eligible to be blood donors.

Patients and methods

Patients with haemochromatosis attending the Haematology Day Ward at Middlemore hospital for regular venesections, clinical surveillance and laboratory monitoring, were assessed for complications of their genetic disorder. Those without clinical consequences were assessed further with respect to their suitability as blood donors.[5] They were asked if they were keen to donate blood. Testing for infectious diseases e.g. hepatitis B and C was not part of this assessment.

Results

Of 74 patients, 53 were homozygous for the HFE mutation, and 21 were diagnosed on other criteria before gene testing was available. Of the 53 homozygous patients, 23 were ineligible for blood donation – reasons for exclusion are listed in Table 1. The remaining 30 (40%) who met criteria for blood donation[5] are currently being venesected between 2 and 52 times per annum. If the blood removed from these patients with genetic haemochromatosis was added to the donor pool, it would provide 409 units a year, or an average of 13-units per patient venesected. Each patient in this group (30 of 30) was willing to attend the blood service as a donor.

Table 1. Reasons for ineligibility to be blood donors.

Liver disease including cirrhosis and chornic hepatitis	17
Heart disease and/or hypertension	9
Psychiatric conditions, on medication	3
Bowel disorders	3
Alcohol abuse	3
Obstructive sleep apnoea	2
Epilepsy	2
Lost to follow up or non compliant	3
Thrombo-embolism on anticoagulants	
idiopathic angioedema, Down syndrome, IDDM, asthma, Jehovah's witness	1 each

Note: more than one reason for exclusion was present in several patients.
IDDM: insulin dependent diabetes mellitus.

Discussion

The European population in South Auckland exceeds 180 775.[6] There are likely to be many undiagnosed individuals in this community since the number with genetic haemochromatosis should be closer to 900 than the 74 currently receiving treatment at Middlemore Hospital. Since the majority of blood donors in New Zealand are European, it is reasonable to assume that approximately one in 200 donors have undiagnosed uncomplicated haemochromatosis and that their blood is currently being used safely.[7] This is supported by the fact that several of our patients were previously blood donors, some at the time of their diagnosis, which lead to them being retired as donors.

In New Zealand there has not been a uniform policy with respect to the use of blood removed from those with haemochromatosis. The blood from suitable individuals with this genetic disorder has been used for transfusion in Christchurch, but not in Auckland for example. The recent formation of the NZBS should allow a national policy to be established.

International practices vary. In Sweden, Canada and Wales, healthy people with haemochromatosis are termed 'super blood donors' because they are allowed to donate more frequently than normal volunteers.[8,9] In USA and England, the issue has been debated because of concerns that such highly motivated people may misrepresent donor information so as not to have their blood wasted.[8,10] The fact that patients in the USA with haemochromatosis incur medical costs for therapeutic phlebotomy has raised the question of whether their desire to donate is entirely altruistic.

To be acceptable as blood donors, patients would need to meet criteria as set out in the NZBS Donor Standards document and sign the donor declaration.[5] If eligible for blood donation, they could be handled as per usual with respect to being a donor, except that the volume of blood removed would need to be increased above the usual maximum allowed of four donations per annum, if clinically indicated.

Also, there are issues to be resolved relating to the monitoring of ferritin levels, clinical decision making re frequency of venesections, and the responsibility for extended family testing. The aim of therapeutic venesection is to rapidly reduce the serum ferritin to a low plasma level to ensure adequate removal of organ iron, and then to maintain the level below 100ug/L[2]. An individual with haemochromatosis who is unable to donate blood for whatever reason (gastrointestinal infection or hepatitis C for example) would need to be retired as a donor temporarily or permanently.

In conclusion, people with haemochromatosis express ongoing concern that their blood is being wasted and have a strong desire to donate blood. Accordingly, and in view of the potential advantages to the blood transfusion service, there is every reason to develop a national protocol to enable suitable individuals with uncomplicated heaemochromatosis to become blood donors.

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Laboratory expenditure in Pegasus Medical Group: a comparison of high and low users of laboratory tests with academics

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Abstract

Aims. To determine, through the use of clinical vignettes, whether low and high cost users of laboratory tests in Pegasus Medical Group (Pegasus) differed in their choice of laboratory tests from academics as a means of further investigating issues relating to quality and cost in laboratory testing.

Methods. Seven clinical vignettes were drawn up and sent to 30 selected members in Pegasus whose actual laboratory expenditure per consultation ranged from a mean of $2.3 in a low cost group (15 members) to $12.2 in a high cost group (15 members). The vignettes were also sent to 15 general practitioner academics. Respondents were requested to complete a laboratory form as to which tests they would use for each individual scenario. The answers were analysed for overall cost as well as numbers of laboratory tests requested.

Results. There were 14 academic responses and 13 each from the bottom and top laboratory users. Overall results for the seven vignette cases showed that low cost laboratory users would spend a total of $176.3, the academics $188.8, and the high cost users $219.5 on the cases. The mean per case costs were $25.2, $27.0 and $31.4 respectively. There was a clear tendency for high volume users of tests in each vignette to be high in others suggesting that doctor rather than patient factors were the main explanation of the variation.

Conclusions. Clinical vignettes do not appear to be a useful strategy in clarifying issues related to quality and cost in laboratory utilisation. Test ordering behaviour appears, from the international literature and this study, to be determined more by personal doctor factors than by objective evidence and clinical need. Further work is needed to clarify the relationship between quality and the wide variation observed in utilisation and expenditure.

NZ Med J 2000; 113: 79-81

A recurrent theme in the analysis of medical decision making is the wide variation in utilisation and expenditure between doctors even when patient factors are taken into account.[1-3] An evaluation of laboratory budget holding by Pegasus in 1995-96 showed a significant reduction in total laboratory expenditure as compared with the previous year and with national and regional trends as well as a reduction in variation between general practitioners in expenditure per consultation.[4] However, considerable variation between high and low cost users remained.

The question arose as to whether those whose laboratory expenditure was low were under-investigating, i.e. was there a quality of care issue to be addressed? On the other hand it is possible that those whose expenditure per consultation was high were over-investigating. A reduction in their expenditure per consultation could lead to significant gains in efficiency and savings overall.

It was suggested that further work should be undertaken using clinical vignettes to compare the high and low cost per consultation users of laboratory tests with a group of academics who might be considered to be models of good quality utilisation of laboratory tests. This paper reports the results of this study.

Methods

Consultation data and laboratory expenditure for Pegasus members for 1995/96 was analysed to identify 15 low and 15 high expenditure-per-consultation members. A set of clinical vignettes was put together by one of the authors from actual consultations involving laboratory tests. For example the following was vignette number 1:

"A 20 year old woman comes into your office. She complains of fatigue and lassitude for the last six months. There is nothing to find on examination of her upper respiratory tract, neck, nor her chest. Your impression is that she might be a little pale? Do you request any laboratory tests? If so, please detail your choice on the laboratory requisition form below".

Other vignettes were: a young man with a sore throat and malaise with cervical lympadenopathy; a woman of 35 having recent dysuria and frequency, a slight vaginal discharge and vague abdominal aches; a 65 year old retired man, with symptoms suggestive of transient cerebral ischaemia and a tendency to bruise easily; a 50 year old woman with a cyst on her back removed for cosmetic reasons; a 70 year old widow with congestive heart failure on frusemide and enalapril; and a young man with recent diarrhoea and an overseas trip a year previously.

These vignettes were sent to each of the low and high expenditure-per-consultation members in Pegasus and a selected sample of 15 general practitioner academics throughout New Zealand. The academics were selected on the basis that they had close links to academic departments of general practice and were involved in teaching and were thought to be a possible benchmark for good quality laboratory use.

Those approached were asked to read the vignettes and to complete the laboratory forms attached indicating which laboratory tests they would use. The responses were analysed to compare the three groups for the laboratory tests, which they would have used in each vignette.

In order to put this study into context, a review of the international and New Zealand literature was undertaken of studies relating to attempts to influence laboratory utilisation and to improve laboratory practice. The review provided a basis for recommendations for further action regarding management of laboratory services.

Results

Of the 15 possible respondents selected from each group there were 14 responses from the academics and 13 each from the low and high cost members of Pegasus.

Table 1 compares the low and high groups for their laboratory expenditure in 1995-96. Although there was very little difference in the mean expenditure per item requested, the mean expenditure-per-consultation was nearly six times greater in the high as compared with the low group. In other words expenditure variation is explained almost entirely by volume, not price, of test ordered.

Table 2 compares the mean expenditure, which the three groups indicated they would request for each vignette. Overall the low expenditure group indicated they would request tests costing $176.3, the academics $188.8 and the high group $219.5. Per case the expenditure indicated was $25.2, $27.0 and $31.4 respectively. The differences were

relatively small and not statistically different as variability within groups was high. As in Table 1 there was very little variation in the expenditure per laboratory item between the three groups.

Table 1. Comparison of low and high expenditure-per-consultation users of laboratory tests of Pegasus in 1995/96 for their mean expenditure per item, per consultation and range for the group.

Group (no)	Mean expenditure per item (NZ$)	Mean expenditure /consultation (NZ$)	Range in expenditure/ consultation (NZ$)
Low group (13)	10.9	2.3	1.3-3.0
High group (13)	11.8	12.2	10.0-15.8

Table 2. Comparison of mean expenditure which the three groups, low, academic and high users, would request for each vignette and the overall mean and expenditure per case for all vignettes.

Vignette Group	Mean expenditure (NZ$) Low (n=13)	Academic (n=14)	High (n=13)
1	26.5	28.3	32.7
2	36.5	33.7	42.3
3	34.0	40.8	39.3
4	15.8	8.1	16.3
5	32.7	43.3	55.6
6	14.5	16.9	25.3
7	17.0	14.9	8.4
Overall mean	176.3	188.8	219.5
Mean per case	25.2	27.0	31.4

Discussion

The response rate for the three groups was good (87% for Pegasus and 93% for the academics) and can be regarded as adequately representative of the groups. There are reservations about the selection of the groups on the basis of low and high expenditure per consultation. A better denominator would have been total patients registered rather than number of consultations but this information was not available. To assess this a follow up analysis of total patients for all members with data, as well as the selected low and high groups, was undertaken from recent electronic GMS claim data held by Pegasus. This showed a very high correlation (r = 0.94) between expenditure per patient and expenditure per consultation. In other words the groups selected were low or high by both criteria.

Another question about the selection of the groups is the different types of practices represented. It is known that some low users of laboratory tests may be specialists in a particular type of practice such as sports medicine. Others may be high users because they have older patients or specialise in women's health and are hence high users, for example of cervical screening. Both practice and patient variability may explain some of the differences in the observed per consultation expenditure. However, there was no difference between the two Pegasus groups in expenditure per test. The variation was almost entirely explained by differences in the volume of tests ordered not their price.

Despite the observed variability in expenditure per consultation between the two groups there was remarkably little expenditure difference between the two groups or the academics in what they indicated they would have used for the vignettes. A perusal of the individual responses to vignettes relating to particular tests pointed to a consistency in the responses for some tests but wide variablility with regard to others. For example, in vignette number 1, regarding a woman with possible anaemia, there was almost universal use of complete blood count (CBC) and a higher use, particularly in the high expenditure group, of ferritin. However, there was also a scatter of requests ranging from plasma creatinine, ESR, glucose tolerance, iron binding, serum iron, liver function tests, midstream urine, Paul Bunnell test, and thyroid stimulating hormone.

It may be difficult to compare what doctors say they would use and what they actually use in practice. The case vignettes were a sample of genuine general practice problems likely to need laboratory tests. They are not a sample of consultations. The low group actual figure of $2.3 is a derived figure of laboratory expenditure divided by total number of all types of consultations and therefore cannot be compared with the calculated figure of $25.3 for the vignettes.

The wide variablility between the three groups may reflect both uncertainty as well as personal opinion in the choices made. On the other hand respondents were consistent in respect of their use of tests between vignettes with high users in one vignette also tending to be high users in others. This suggests that general practitioner factors are much more important than patient factors in determining laboratory test behaviour. A case vignette is an artificial experience, removing a great number of those skills which general practitioners bring to bear during a consultation. Although they produce a level playing field with each participant having the same information, they do not allow for testing of an individual practitioner's level of comfort in the clinical situation. The results of this study therefore do not appear to assist in defining under or over utilisation and raise doubts about the value of the vignette approach to assessing quality and efficiency.

There is an increasing need to define measures of quality in general practice. There is evidence that practices with low per capita expenditure on laboratory tests may also have low per capita expenditure on GMS and pharmaceuticals.[5] This may be linked to better quality general practice and to confidence, experience and other personal characteristics of the general practitioner. One measure of quality of laboratory utilisation could be the ratio between negative and positive laboratory tests. The numbers of positive diagnoses per consultation or per registered patient may be one indicator of quality and efficiency in laboratory test use.

There are a number of similarities in trends in the use of laboratory services and pharmaceuticals with budget holding. Both low expenditure prescribers as well as low expenditure users of laboratory services have tended to further reduce their expenditure, despite feedback indicating that they were already low users.[6] Given the need to develop indicators of quality it might be desirable to undertake a study of patient outcomes for a random sample of consultations from low and high per capita users of laboratory tests. Outcomes could be patient satisfaction with the consultation, length of the sickness period and time off work.

A search of the international literature was made for studies of laboratory utilisation behaviour. Only one study relating to the use of vignettes was found but this did not compare stated with actual use of tests.[7] However, one study of what general practitioners stated they did in the management of hypertension, compared with what they actually did as found from their practice records, showed considerable differences and raised doubts about the validity of the questionnaire approach to determining clinical behaviour.[8]

There were few studies reporting variation in laboratory test use.[1,9] The published studies show that there are three main factors which determine clinical decision making including the use of laboratory tests.[10,11] These are professional/personal factors, patient benefit and the wider societal good. Professional factors include uncertainty in

decision making and monitoring, attitudes to risk taking or risk avoidance, unawareness or disinterest in costs, the growth of science and technology in medicine and the influence of and standards set by clinical leadership.[1,5,7,9,12,13]

Personal factors include personal characteristics, tolerance of uncertainty in decision making, practice experience, fear of litigation, financial incentives, style of practice and practice setting.[3,5,7,9,14] Patient benefit factors include acting as the patient's agent in providing the best possible care, clinical factors, the personal characteristics, expectations and demands of the patient and the practice of "defensive medicine".[5,6,9,10] Third and least influential is acting as guarantor of the "social good". This includes the need to balance the needs of the individual patient against those of the wider society.[9]

A number of strategies can influence the ordering of laboratory tests. These include education and transfer of knowledge, feedback on performance related to test use and costs of tests ordered, redesigning test forms to limit the tests available, and the use of incentives and/or penalties.[15-18] The most successful is a multi-faceted approach, i.e. a combination of several of the above strategies.[13,14,19,20]

Important themes which emerge include creation of a desire for change by leaders, participation by doctors in agreeing on the nature of the problem and developing the solution, individual feedback from a respected colleague, brevity of written information, the use of recognised leaders and the long term nature of successful interventions.[15,21,22]

In conclusion previous observations and the present study findings have important implications for the future management of clinical activity in general practice. The Health Funding Authority's move to population-based, equitable funding of laboratory and pharmaceutical services highlights the need to clarify the nature and basis for the wide variation reported in laboratory and pharmaceutical utilisation. Of particular importance is the need to clarify the relationship between utilisation and quality. If better quality care leads to lower utilisation this provides a powerful incentive to invest in complementary measures to promote both better quality and lower cost.

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Cricketing injuries in children: from the trivial to the severe

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Abstract

Aim. To describe the nature of acute cricketing injuries in children presenting to the emergency department of a tertiary level children's hospital. Two cases of severe injuries during a cricket game are reported.

Method. A retrospective review of presentations to the emergency department from 1993 to April 1998.

Results. Sixty cases of cricketing injuries were reviewed. Injuries to the head, hands and forearms were most common. Most injuries were caused by being hit by a ball. A high proportion of cases required operative intervention. Length of stay in hospital was only overnight in most cases. The two case reports highlight unusual but severe injuries that caused significant morbidity to the patients involved.

Conclusion. Although cricket is, by and large, a safe sport, this report will raise awareness of the variety of injuries that can be suffered by children playing the game.

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Each summer thousands of school children enjoy playing cricket. Although traditionally regarded as a relatively safe sport, cricket has been identified as having a moderate risk of injury. Injuries to the eyes, face, head and hands are the major concerns associated with playing cricket at an adult level.[1-3] Chronic spinal injuries have been described in young fast bowlers[4] but the literature is scarce when describing the sorts of acute injuries suffered by children playing cricket. We describe the nature of injuries sustained by children playing cricket who presented to the emergency department of a tertiary level children's hospital from 1993 to March 1998. We also present two case reports of severe, life-threatening injuries that occurred while playing cricket.

Methods

A retrospective analysis of the notes of children who had presented to Starship Children's Hospital between 1993 to April 1998 with a coding

for injury secondary to playing cricket (ICD E-8897) was performed. There were 91 patients identified as having this coding. Thirty-one patients were excluded as their injuries occurred as a result of being hit with a bat, ball or wicket, but not while playing a game of cricket. The remaining 60 patients were reviewed with respect to type of injury, mode of injury, age and length of stay in hospital, and need for operative intervention.

Results

Major categories of injury analysed were: closed head injury with concussion, skull fracture, nasal fracture, abdominal blunt trauma, thoracic blunt trauma, fractured fingers, fractured forearms and 'other' (Table 1). The category 'other' included assorted injuries including soft tissue contusions, ligamentous injuries, minor lacerations and abrasions that did not require admission into hospital.

Table 1. Types of injuries suffered by children playing cricket.

Injury	Number	Age (median) years	Length of stay (median) days
Closed head injury	8	11	1
Head injury + skull fracture	2	9	2.5
Blunt abdominal trauma	6	12	1
Blunt thoracic trauma	2	12.5	2
Fractured finger(s)	7	13	1
Fractured forearm	11	11	1
Fractured nose	6	11	1
Other	18	12	1

The category of blunt abdominal trauma included two cases of severe injury. One case had a grade IV renal rupture that led to functional loss of the left kidney. The other patient required bowel resection for a haematoma of the ascending colon. Both cases are described further below.

In the category of thoracic trauma, a 14-year-old boy sustained a pneumomediastinum whilst diving for a ball during fielding. A case similar to this has been previously noted in the literature.[5] The pneumomediastinum resolved itself with conservative management and the patient had no further problems at follow-up.

The most common mechanism of injury was being hit by a ball, either while batting or fielding (Table 2).

Table 2. Mechanism of injury in childern playing cricket.

Mechanism	Number
Hit by bat	12
Hit by ball	31
Fall	12
Collision with player	3
Fall on to bat handle	1
Fall on to cricket stumps	1

There were 19 cases which required operative intervention. Ten were closed reduction of fractured fingers or forearms, four were open reduction and internal fixation of fractures, five were suturing of lacerations and there was one laparotomy and resection of bowel.

Case 1.

A 14-year-old boy was running between the wickets during a cricket game when he tripped and fell onto his bat handle. The handle of the bat caused blunt trauma to the left upper quadrant of the abdomen. He proceeded to have urgent abdominal CT with intravenous contrast. This revealed grade IV rupture of the left kidney, (Figure 1) with a large perinephric haematoma. The spleen and the rest of the abdominal viscera were normal. He was treated conservatively with bed rest, intravenous antibiotics and nasogastric tube. He continued to make good progress and was discharged 18 days after injury. At a follow-up clinic six weeks later, he had no complaints of any haematuria and his blood pressure was stable at 126/60 mmHg. A DMSA scan showed that there was no residual function in the left kidney and the right kidney had undergone compensatory hypertrophy. He remains on long-term follow-up under the renal physicians in case he develops hypertension.

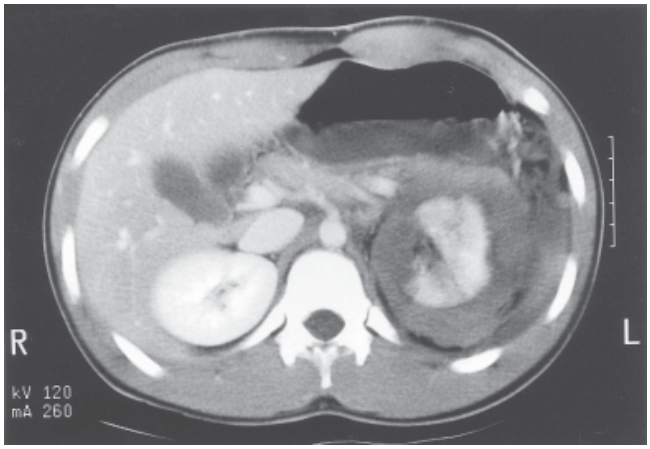

Figure 1. Abdominal CT scan showing grade IV renal rupture secondary to blunt abdominal trauma from a cricket bat handle.

Case 2.

A 14-year-old boy was chasing a ball during a game of cricket at school. He collided with a fellow fielder and sustained blunt trauma to the abdomen when they fell on top of each other. He had to leave the field soon after and was brought to the emergency department complaining of abdominal pain. On examination he was pale and looked unwell with a temperature of 36.2°C, blood pressure 138/68 mm Hg and a heart rate of 92/min. There was a large tense swelling in the right lower quadrant of the abdomen, which was very tender to palpation but the rest of the abdomen was soft. Rectal examination was normal. Haemoglobin, coagulation screen, serum urea, electrolytes and amylase were all within normal limits. CT scan of the abdomen showed a 5 x 6 x 5 cm mass consistent with a haematoma which was situated below the right lobe of the liver around the caecal wall and an active bleeder was demonstrated in the middle of it during the contrast phase (Figure 2).

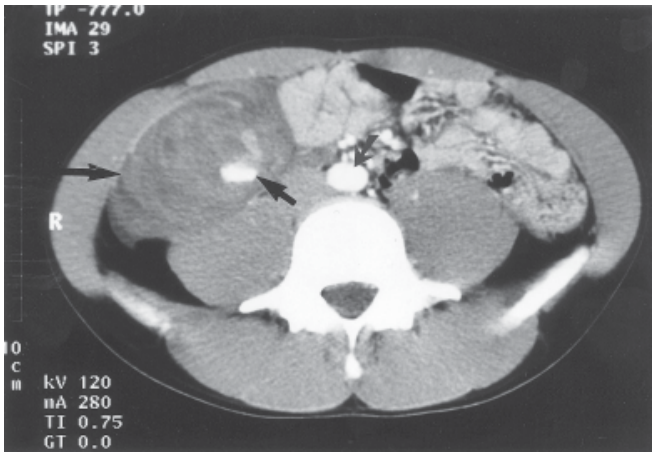

Figure 2. Abdominal CT scan showing caecal wall haematoma. Note the arrow pointing to the brightly contrasting bleeding vessel, of the same intesity as the aorta (small arrow).

A provisional diagnosis of a caecal wall haematoma without perforation was made and it was elected to treat the patient conservatively initially. A nasogastric tube and indwelling bladder catheter were placed, intravenous fluids were given and the patient was allowed nil by mouth. Serial measurements of haemoglobin showed a drop to 101g /L over eight hours after which it stabilised. Forty-eight hours after admission the patient's temperature rose above 38°C, his pain level increased and the haemoglobin dropped further. A decision was made to perform a laparotomy to prevent impending rupture of the large bowel.
At operation large amounts of free intraperitoneal blood were found. There was a huge haematoma involving the caecum and ascending colon (Figure 3) extending into the retroperitoneum and superiorly to the liver. The tension on the caecal wall was such that the visceral peritoneum had split. A right hemicolectomy and anastomosis of the transverse colon to terminal ileum was performed. He made an uneventful recovery and was discharged on the seventh postoperative day. He was reviewed in outpatient clinic one month later and was fully recovered.

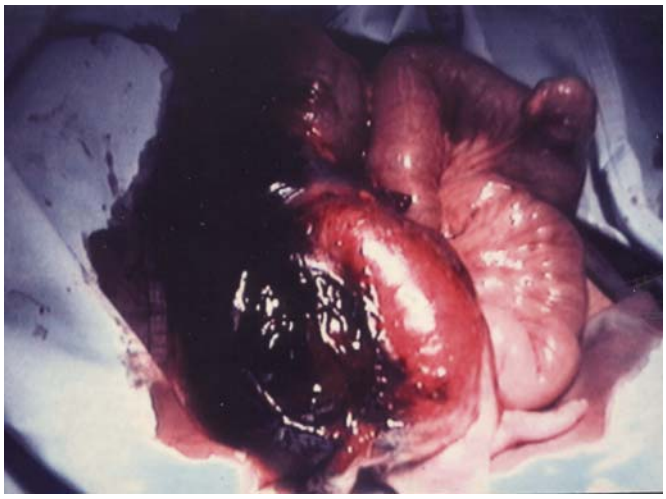

Figure 3. Operative photograph of the caecal wall haematoma.

Discussion

We have shown that injuries that require medical attention occur from time to time in children playing cricket. Although the majority of these injuries are not life-threatening, a significant number of them may require operative intervention under general anaesthetic as illustrated in our review. Our results agree with studies of cricketing injuries in adults,[1] with a high proportion involving areas of the body that are recognised to be at risk during the game: hands, forearms, face and head. Protection in the form of guards and helmets are available with these injuries in mind. We have also reported two unusual modes of cricketing injury in children that both resulted in blunt trauma to the abdomen. In the first case, the patient's injury resulted in loss of function of one kidney, with the potential for life-threatening haemorrhage during the acute phase. This patient will require life long follow-up to monitor his blood pressure and renal function. Long-term problems in the remaining kidney after childhood nephrectomy have been reported, such as progressive glomerulosclerosis and proteinuria, which increase the risk of premature renal failure.[6] Case report 2 illustrates the dangers of fielders colliding while chasing for a ball. This injury resulted in severe blunt abdominal trauma and led to a resection of the terminal ileum and ascending colon. The long-term sequelae of ileocolic anastomosis described in the literature include loose stools, vitamin B12 and folic acid deficiency[7] and perianastomotic ulceration.[8]

Conclusion

This is the first report of its kind describing the variety of acute cricketing injuries suffered by children. It also shows that significant and severe injury is possible from cricket as illustrated by the two case reports. This report helps document such injuries in children and with better accessibility of indexed journals raises awareness in general of these injuries.

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PROCEEDINGS

Waikato Academic Division, School of Medicine, University of Auckland, Biannual Research Seminar, 17 March 1999.

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The impact of polymerase chain reaction (PCR) on the laboratory diagnosis of community acquired pneumonia. J Haines, C Coles, G Mills, N Karalus, R Cursons. Respiratory Department, Waikato Hospital, Hamilton and Department of Biological Sciences, Waikato University, Hamilton.

Sixty two patients were originally enrolled in this pilot study. After a review of chest x-rays and consent requests, the number of patients was reduced to 44. Eighteen of these had received antibiotics prior to admission. The mean patient age was 61 years and there was an equal gender ratio. DNA amplification for Streptococcus pneumoniae, Mycoplasma pneumoniae, Chlamydia pneumoniae and Legionella spp. was investigated using different fractions of blood (plasma, buffy coat, blood). No DNA amplification was detected for Chlamydia, Mycoplasma or Legionella, whereas serology detected two cases of Legionella and one case of Mycoplasma. Blood culture for *S. pneumoniae* was positive for six cases whereas PCR was positive for 11 cases. Of the six positive blood cultures PCR detected *S. pneumoniae* in five cases. Some specimen inhibition was noted retrospectively and the use of a housekeeping gene to monitor specimen inhibition is recommended.

Management of heart failure in the elderly and very elderly at Waikato Hospital. Sonja Crone, Cathy Callagher, Clyde Wade, Gerry Devlin. Department of Cardiology, Waikato Hospital, Hamilton.

Congestive cardiac failure is a common cause of morbidity and mortality with increased prevalence in the elderly. Readmissions are frequent and are multifactorial. The aim of our study was to compare

the management of congestive cardiac failure in the elderly (E) (>65yrs <75yrs) and the very elderly (VE) (>75yrs) at Waikato hospital to examine if differences existed which could be addressed to improve patient management.

A retrospective review of hospital records was made for all patients over 65 yrs admitted with a discharge diagnosis of heart failure from 1/01/97 to 30/06/98. A total of 354 patients were identified, 63% female. All were managed by cardiologists. The majority, 63% (223) were in the VE group. The commonest aetiology was ischaemic heart disease in both groups (42v45%, p=ns). The incidence of co-existing renal failure was similar (24% v 25%, p=ns). Echocardiography was utilised more frequently in E (56% v 46%).

Mean length of hospital stay was 6.1 v 6.5 days (p=ns). Sixty three percent of E were on ACE inhibitors on admission compared with 55% of VE (p=ns). A trend was noted to more frequent use of ACE inhibitors on discharge in E (81% v 67% p=ns), with a higher mean dose of drug administered. Mortality rates were similar (5.3% v 7.2% p=ns). VE patients remained significantly more symptomatic on discharge with 61.5% New York Heart Association class III or IV compared with 41% in the E group (p=0.001). However 30 day readmission rates were similar (5.3% v 8.1% p=ns).

We conclude that in elderly patients presenting with congestive cardiac failure ACE inhibitors appear to be used less frequently in the VE and at lower doses despite similar occurrence of chronic renal failure. Length of hospital stay is no different but the VE are more likely to have significant symptoms on discharge and this may possibly be related to under-use of ACE inhibitors. Readmission rates are, however, similar.

A theoretical model of the EEG changes caused by general anaesthesia. Jamie Sleigh, D Alistair Steyn-Ross,[2] Moira L Steyn-Ross,[2] David TJ Liley.[3,1] Department of Anesthesia, Waikato Hospital.[2] Department of Physics, University of Waikato, Hamilton.[3] School of Biophysical Sciences and Electric Engineering, Swinburn University of Technology, Melbourne, Australia.

This paper describes a model of the effects of general anaesthetics on the cerebral cortex. It explains some of the changes in EEG that are observed at induction and recovery of general anaesthesia. Coherent activation of assemblies of ~105 cortical pyramidal neurons generate most of the observed EEG signal. Thus the EEG uniquely reflects the cortical information flow and its disruption by general anaesthetic agents providing a window into neuronal function.

Recently several theoretical models of the function of the human cerebral cortex have been proposed.[1] The theoretical formulations consist of a system of equations that describe all possible interactions between inhibitory and excitatory cortical neurons within a functional assembly. These equations realistically model the generation of action potentials dependant upon the neuronal membrane potential, and the time course and amplitudes of the resultant post-synaptic potentials. Assuming that the predominant effect of general anaesthetic agents is to prolong the duration of the inhibitory post-synaptic potentials,[2] it is possible to derive the expected changes in the EEG power spectrum that occur during induction and recovery from general anaesthesia. The theoretical EEG spectrum is found to agree nicely with the biphasic phenomenon observed in practice showing an initial increase in spectral power and a shift to higher frequencies followed by a decrease in power and lower frequencies predominating with increased depth of anaesthesia. The steady-state solutions of our formulation provide an elegant description of the relationship between anaesthesia, aesthesia and convulsive cortical states.

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2. Franks NP, Lieb WR. Molecular and cellular mechanisms of general anaesthesia. Nature 1994; 367: 607-14.

Cognitive-behavioural intervention in fibromyalgia; sustained improvement in health outcome. M Lean, T Hocquard, P Jones. Queen Elizabeth Hospital for Rheumatic Disease and Rehabilitation, Rotorua.

We describe the short- and long-term health outcome of a multidisciplinary rehabilitation program for fibromyalgia. We investigated all patients who attended the program between 1.7.97 and 31.10.98 and who satisfied ACR criteria for primary fibromyalgia. The 3-week program was based on the biopsychosocial model in which fibromyalgia was viewed as a bodily state arising as a reaction to environmental factors. It consisted of education (cognitive restructuring, understanding pain, stress management), relaxation, physical training, lifestyle review based on the occupational model, and counselling using rational emotive behavioural therapy techniques. The only medical intervention used was low dose (10-30 mg) amitriptyline (in 30%). Health outcome measures (HOM) were scored on admission to and at discharge from the program, and 3-6 months later. The measures used were: SF-36, Fibromyalgia Impact Questionnaire (FIQ), McGill Pain Questionnaire (MPQ), Canadian Occupational Performance Measure (CPOM) and Wellness 1-10 anchored numeric scale. Student t tests were used to test the significance of changes.

Sixty nine patients (5 male) completed the program and follow up. Median age was 46 years, range 16-80. Significant (p<0.01) improvement was seen between admission and discharge in all scales of the SF-36 except role physical, general health and role emotional. The improvements were maintained at 3-6 months. Other HOM were (admission; discharge; 3-6 months) mean (95% CI): FIQ: 52.8 (52.2-55.4)); 36.7 (33.1-40.3); 38.8 (35.1-42.5). MPQ: 26.7 (24.1-29.2); 16.0 (13.6-18.4); 20.6 (16.8-24.4). COPM-performance: 3.4 (3.1-3.7); 5.7 (5.3-6.1); 6.0 (5.5-6.5). COPM- satisfaction: 2.6 (2.2-3.0); 5.7 (5.1-6.3); 6.1 (5.6-6.6). Wellness: 4.6 (4.0-5.2); 7.2 (6.7-7.7); 5.7 (4.9-6.4). All changes between entry scores and discharge/follow-up were significant at p<0.01. Alternative anaylsis showed that patients either had a marked response or remained unchanged. Substantial improvement was seen in 48% of the patients.

We conclude that patients in the fibromyalgia program showed clinically significant and sustainable improvements in health outcome.

Amylin and metabolic regulation in lactating goats. SR Davis, SH Min, VC Farr, J Lee, K Loomes,[1] GJS Cooper[1] AgResearch, Ruakura Research Centre, Hamilton and [1]Dept. Biological Sciences/School of Medicine, University of Auckland.

Amylin is a 37-amino acid peptide which has structural homology to the calcitonin family of hormones. Amylin is co-secreted with insulin and has been shown to have a wide range of metabolic actions in rodents. Several of these actions are similar to metabolic changes which occur in ruminants in early lactation. This study was performed as part of a wider investigation of the role of amylin in ruminants in regulating lactation and/or metabolism in support of lactation.

Five lactating goats were infused with rat amylin (320 pmol/kg/h) or saline via an external pudic artery into one mammary gland for 18h of a 24h period of 2-hourly milking. (The amylin dose was chosen on the basis of its ability to achieve an acute 10% increase in mammary blood flow. Previous work has shown that this dose results in a 5-10 fold increase in plasma amylin concentrations). Treatments were reversed one week later. Jugular blood samples and milk samples were taken at 2h intervals. Milking was performed with the aid of oxytocin (2x100 mIU, i/v). Mammary blood flow was measured using transit-time blood flow probes chronically implanted around the external pudic artery.

Amylin infusion had no effect on milk yield but milk lactose and protein contents showed small (<10%) but significant (p<0.05) reduction. Amulin increased plasma glucose (3.4 to 3.8 mM; p<0.05)) and free fatty acid concentrations. Plasma amino acid concentrations (essential and non-essential) were also reduced, but only after 6h of amylin infusion. For some amino acides, notably ornithine, arginine and lysine, the reduction was substantial (40%; p<0.05). Effects on glucose, cholesterol and mammary blood flow (30% increase by 4h) were not sustained beyond 8h, while other responses lasted for the duration of infusion.

Amylin infusion produced a wide range of responses in fat, mineral, protein and glucose metabolism. These changes, particularly in plasma amino acides, may have served to prevent any milk yield response to amylin. For some parameters (glucose and cholesterol) there was evidence of an endocrine counter-regulatory response, the mediator of which has yet to be identified. The fall in plasma cholesterol concentration was almost exclusively associated with the HDL lipoprotein fraction suggesting enhanced hepatic clearance. Effects on plasma amino acids may be mediated through reduction in

gut motility restricting amino acid absorption from the gut. This and other studies in sheep and goats have shown a particular sensitivity of plasma calcium to amylin infusion, which is likely to be mediated via a direct effect of amylin on bone resorption.

In conclusion, while the physiological and pharmacological actions of amylin in ruminants have not been distinguished in this experiment, the results demonstrate that amylin may play multiple roles in metabolic regulation.

Autidory recall and response-to-command during recovery from propofol anaesthesia. Murray Williams, Jamie Sleigh. Waikato Hospital, Hamilton.

Most studies of awareness under general anaesthesia use the ability to respond to a verbal command as the primary measure of consciousness. The aim of this study was to discover whether it was possible for subjects who were recovering from a propofol general anaesthetic, to experience conscious awareness, without being able to respond to verbal command.

Ten healthy volunteers received EEG monitoring and an intravenous propofol infusion (150 ml/hr) until they were no longer conscious. The infusion was then stopped and they were given verbal commands interspersed with random numbers from a recorded tape until they were able to respond appropriately. Seven subjects were able to remember numbers corresponding to times 10 to 40 secs before they responded to verbal command. In none of these subjects was there recall of the number 30 min later. The Bispectral Index amongst the different subjects was in the range 86 to 97 at the point at which they became amnesic, 49 to 92 at the point they lost consciousness on induction, and from 68 to 96 when they responded to verbal command on recovery.

We conclude that there is an ability to have conscious awareness of auditory input without necessarily being able to demonstrate this by responding to verbal command. Though the subjects were aware at the time, however, they did not seem to be able to consolidate the information into long-term explicit memory.

Total occlusion angioplasty in the real world: the Waikato hospital experience. Wanda Visser, Gerry Devlin, Chris Nunn, Hamish Charleson, Raewyn Fisher, Spencer Heald, Hugh McAlister, Clyde Wade, Department of Cardiology, Waikato Hospital, Hamilton.

Total occlusion angioplasty (TOA) is associated with lower primary success rates and a high clinical restenosis rate which has been improved by intracoronary stenting in high volume experienced research centres. Whether these results are generally reproducible is unclear. We report the experience at Waikato Hospital, a moderate volume interventional unit, with TOA over an eight year period.

A retrospective analysis from chart review and an interventional database was performed of all patients undergoing TOA (excluding primary angioplasty). Procedural outcome and complications were reviewed. Late symptomatic status and the need for further revascularisation was assessed by conducting a phone survey.

There were 274 TOAs performed in 253 patients, representing 14% of all angioplasties. Seventy two per cent were male, mean age 58 years. Sixty eight per cent of procedures were successful. Emergency coronary artery bypass grafting was necessary in three patients (1%). Sixty four per cent of patients responded to the phone survey with a mean time of follow-up of 36 months. The vast majority, 72%, required no further revascularisation procedure and were either asymptomatic (46%) or suffered infrequent CCS I symptoms (31%). In the last two years stent insertion has become common. During this period 51 successful TOAs were performed, 30 (59%) stented vs 21 (41%) unstented. Thirty five (69%) were available to follow-up (20 stented (57%) v 15 non-stented (71%). Stent usage resulted in a significant reduction in the need for further revascularisation (15% v 53% p=0.02). A trend was noted to reduced readmissions (10% v 33% p=NS) and improved late symptomatic status (CCS 0-II 95% v 86% p=NS) in stented patients.

We conclude that total occlusion angioplasty at Waikato Hospital can be performed with moderate expectation of success, acceptable repeat revascularisation rates and good late symptomatic status. Stent insertion has impacted significantly on the need for repeat revascularisation. These results compare favourably with reported series.

Mitral valve replacement surgery at Waikato Hospital. H Keown, RG Talbot. Waikato Academic Division, Faculty of Medicine and Health Science, University of Auckland, Department of Cardiology, Waikato Hospital, Hamilton.

This retrospective study was designed to assess whether valve replacement surgery for mitral regurgitation (MR) at Waikato Hospital is being performed at the optimal time.

All patients undergoing mitral valve replacement (MVR) for dominant MR over the ten-year study period were included. End systolic diameter (ESD), end diastolic diameter (EDD), end diastolic diameter (EDD), fractional shortening (FS), ejection fraction (EF) and NYHA functional class were recorded pre-operatively. The study showed a reduction in the number of patients with a pre-operative. ESD > 5.0 cm and a concurrent increase in those with an ESD 4-5 cm. Additionally there has been a reduction in the number of patients without adequate pre-operative assessment. Seventeen patients went to surgery with ESD > 5.0 cm, of whom ten died or developed heart failure and seven had a good outcome.

Previous studies suggest the MVR in patients with pre-operative. ESD > 5.0 cm is predictive of poor outcome. The number in this category has declined at Waikato Hospital but better uniformity in patient selection might be achieved if pre-operative echo assessment was an invariable prerequisite. The majority of patients with a poor outcome (death/failure) had pre-operative ESD >5.5 cm, suggesting that patients with such dimensions should possibly be declined surgery on the basis of high likelihood of poor post-operative outcome.

A serum amyloid protein homologue is expressed by the mammary gland. A Molenaar, G Rajan, K Stelwagen. Dairy Science, AgResearch, Ruakura research centre, Hamilton.

Serum amyloid proteins are of interest to the medical community because they can be used as indicators of inflammation in, for example, bacterial infection, renal allograft rejection and acute myocardial infarction. We are interested in the identification of non major-milk-protein mRNAs that are differentially expressed between lactating and involuting mammary tissue. Using representational difference analysis (RDA), many RDA products were cloned and sequenced. One of these proved to be homologous to the serum amyloid. A family of proteins and thus named bovine mammary amyloid protein (BMA). The expression of serum amyloid proteins has not been reported in the mammary gland. Northern analysis showed BMA to be expressed in the mammary gland in a similar temporal pattern to lactoferrin and was induced within 24 hr of milk stasis. In-situ hybridisation analysis showed that its mRNA was spatially co-localised with that of lactoferrin and it was expressed in the 'non lactatin' or vesicle engorged alveoli and highly induced in the epithelial cells during pregnancy and regression. A 600 bp mRNA was isolated from a lambda cDNA library. Its sequence was ~83% homologous to the rabbit serum amyloid A (SAA3) mRNA over a 374 nucleotide region.

The SAA family are apolipoproteins whose gene sequences reside on human chromosome 11p15. Their hepatic synthesis can be induced 1000x by pro-inflammatory cytokines during the acute phase response. No null mutants described suggesting that they are of fundamental importance in infection and trauma. Many putative roles of the serum amyloid proteins have been described. These include cholesterol transport and metabolism particularly of HDL3, modulation of host protective functions such as some immunological responses during inflammation and the acute phase response to infection, trauma or stress; induction of collagenase expression in joints; and participation in tissue repair and regeneration. Chronically elevated SAA levels can result in fatal secondary amyloidosis (plaques) and hence the genes contain certain transcription binding factor sites to allow rapid on/off modulation.

We are interested in determining the role of BMA in the mammary gland. By inference from the roles of the SAAs, it may have a protective effect in the immediate response to the stress of stretching in the unmilked mammary gland alveoli. In the longer term, its elevation may induce collagenase to loosen the tight junctions between engorged alveoli so relieving the pressure, and to promote remodelling of the gland during stasis / involution. Lastly, it may protect the gland against over-inflammatory responses during infection.

Potential applications for BMA exist in agriculture as a marker for disease resistance / susceptibility / production characteristics and in medicine and a 'natural' anti-inflammatory agent.