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*Michael Harkness*
This Issue in the Journal

Preventable medication-related events in hospitalised children in New Zealand
Desireé L Kunac, David M Reith

Few data are available regarding preventable medication-related events in hospitalised children in the New Zealand setting, yet such data is essential to effectively implement prevention strategies that reduce the risk of harmful events. Through identification of events using a variety of methods, and subsequent classification of events for preventability, we suggest optimal prevention strategies should target dosing errors, particularly systems and processes in prescribing medications, and use of antibacterial agents, particularly when administered by the intravenous route. As very few events were identified through the hospital incident reporting system, it should be supplemented by other methods that enhance event identification if patient care quality is to continually improve.

Allele frequency differences of cytochrome P450 polymorphisms in a sample of New Zealand Māori
Rod A Lea, Rebecca L Roberts, Michael R Green, Martin A Kennedy, Geoffrey K Chambers

Cytochrome P450 (CYP) enzymes play a key role in the metabolism of certain medicines—e.g. antidepressants and beta blockers. Variant CYP genes have been previously linked to different responses (by ethnicity) to medicines among patients. Our study determined the presence of functional CYP gene variants (alleles) in DNA from a sample of 60 Māori and compared the frequency of these variants to that seen in Europeans. Allele frequencies of differed between Maori and European groups warranting larger general population studies. These findings may also bear thinking about when conducting pharmacogenetic studies or clinical trials in New Zealand cohorts because patients with Māori ancestry may respond differently to certain medicines based on genotype.

Surveillance of vaccine breakthrough cases following MeNZB vaccination
Anne McNicholas, Yvonne Galloway, Diana Martin, Kerry Sexton, Jane O’Hallahan

The meningococcal B vaccination programme to control an epidemic of meningococcal disease in New Zealand was our largest ever immunisation campaign. It is important to find out how well the new vaccine worked by monitoring the total number of disease cases as well seeing whether children who had been fully vaccinated got sick with meningococcal disease. The number of epidemic disease cases has gone down dramatically since the MeNZB vaccine was introduced in New Zealand. Because the vaccine is not 100% effective, meningococcal disease cases occurred in some fully vaccinated children.
Capitation funding of primary health organisations in New Zealand: are enrolled populations being funded according to need?

Jennifer Langton, Peter Crampton

The aim of this study was to determine whether the three main funding formulas for Primary Health Organisations (PHOs) achieved a stated aim of the Primary Health Care Strategy to fund enrolled populations according to need. The study found that the greater funding for ‘Access’ PHO enrollees was notably eroded with the introduction of Access-level funding for those aged 65+ in ‘Interim’ PHOs. The rapid shift to Access-level funding for First Contact Services has seen a continued erosion of the redistributive effect of the original needs-based formulas. A system cannot be considered equitable if some members of society are not realising their health potential, and financing of primary care should remain redistributive until such a time as this objective is attained.

Correlation of physician seniority with increased emergency department efficiency during a resident doctors’ strike

Martyn Harvey, Mustafa Al Shaar, Grant Cave, Muir Wallace, Paul Brydon

We examined the efficiency of Emergency Department (ED) processing of patients during a week when junior doctors were on strike, and the department was staffed with specialists, and compared with a week of normal ED staffing. Waiting times, time seen to discharge/admission, and total time spent in the ED were reduced during the strike period. The increase in efficiency is likely to be at least in part due to the presence of more senior ED medical staff.

Review Article

Evidence-based recommendations for hand hygiene for health care workers in New Zealand

Peter J Larmer, Trish M Tillson, Faye M Scown, Philippa M Grant, Jamie Exton

Handwashing with soap and water is not the most effective method of hand hygiene for health care workers. Additionally a high percentage of health care workers suffer from dry/damaged hands which may be worsened by simple handwashing. The use of 70% alcohol-based hand rub with the addition of a moisturiser is not only the most effective hand hygiene method, but it can also assist in repairing damaged skin.
Medication error in New Zealand—time to act

Alan F Merry, Craig S Webster

Preventable adverse events involving medications have been repeatedly identified as a leading cause of iatrogenic harm internationally. They occur in hospitals, in primary healthcare, and notably at the interfaces between healthcare settings (e.g. on admission to and discharge from hospitals). They involve all routes of administration and all provider groups, and they are responsible for much serious and costly morbidity (and occasionally even mortality) in patients of all ages.

Thus far, there has been a disappointing lack of commitment to addressing them. In the end, these errors are simply an unacceptable failure in a process of only moderate complexity for which it ought to be possible to achieve the six sigma standard expected in high reliability organisations in other sectors of industry or service provision. Six sigma implies (in effect) no more than 1 failure in every 300,000 (or so) episodes of a process.

In this issue of the Journal, Kunac and Reith report a prospective observational cohort study of preventable medication-related events in children in a New Zealand university-affiliated urban general hospital. They identified 761 medication-related events arising from 3160 medication orders over 3037 patient days of admission for 495 children.

One probable reason for complacency in relation to medication errors is the fact that many are without consequence. However, Kunac and Reith report that of the preventable events (which might also be called “errors”, or perhaps simply “process failures”), 38 (5%) caused actual patient harm, and a further 75 (9.9%) were classified as potentially harmful. It is a little disappointing that no examples were provided to illustrate the nature of this harm, but clearly these data describe a situation that is a long way short of six sigma and a reason for considerable concern.

In light of current interest in establishing a unified national incident reporting system in New Zealand, it is sobering to see that less than 1% of the identified errors were captured by the hospital’s routine incident reporting system. A voluntary staff quality improvement system was more successful (capturing nearly 15%), but the vast majority of events (identified by chart review) were still missed. It is also interesting that nurses submitted more voluntary incident reports than any other professional group.

A recent report from the UK’s National Reporting and Learning System made a similar observation and emphasised the importance of engaging the key speciality groups involved with generating incidents in the process of reporting them. Their analysis of 2000 incidents related to anaesthesia found that many patients had been harmed, but was unable to identify any recommendations to reduce the likelihood of the same things happening again because of inadequate detail about the specifics of what had gone wrong, and why.
In general, this requisite in-depth information needs to come from those who generate the events. Clinicians’ engagement in incident reporting is likely to depend on their perception of the relevance of the information collected to their own field of practice, and of the likelihood of a constructive response. These concepts are sometimes embedded in the term “ownership”.

There is, therefore, a strong case for supplementing any overarching generic incident reporting system with a number of more targeted initiatives driven and owned by specific groups of practitioners. For example, in this part of the World, the Australian and New Zealand College of Anaesthetists, the New Zealand Society of Anaesthetists, and the Australian Society of Anaesthetists have combined resources to fund re-invigoration of a binational voluntary incident reporting system for anaesthetists with the aim of capturing sufficient detail to identify and promote effective ways of improving safety for their patients.

There is no point collecting information without acting upon it. Reluctance on the part of many workers and funders in healthcare to embrace simple commonsense measures that might make a substantial difference to drug safety is disappointing. For example, the New Zealand Medicines and Medical Devices Safety Authority (Medsafe) has indicated (in a personal communication with Dr Ahmed Engin) that it opposes colour coding by pharmacological class of drug because this may reduce the imperative to read the label. However, the point of colour coding is not to substitute for the essential task of reading the label, but to provide a supplementary safeguard to make between-class errors less likely, on the basis that within-class errors are likely to be less serious or injurious.

The whole thrust of error management developed along principles of cognitive psychology over the last 3 decades has been to add as many protective measures to the mix as possible, and there is certainly scope for this principle in the administration of medications. The real issue here is that “look-alike sound-alike” drug ampoules are repeatedly identified in incident reports as contributing to drug errors.

It is not necessary to colour code all drugs, but it makes obvious sense to identify high-risk examples (that Reason would call “latent factors”) and address these; the recent release in New Zealand of dopamine and magnesium in very similar presentations is just one example that could have been avoided, and it is one that has already lead to the administration of a 200 mg bolus of dopamine instead of magnesium (and very nearly a disaster). This was a virtual re-run of the infamous dopamine for doxapram drug swap which lead to a tragic patient death and the conviction for manslaughter of an anaesthetist in the early 1990s. Do we never learn? Similarly, there have been reports of incidents in which nursing staff have perverted the functioning of unit dose verification systems on the ward (see below), presumably in the interests of efficiency rather than safety. There is also anecdotal evidence that some individual practitioners provided with a system which uses barcodes to check the identity of intravenous drugs in anaesthesia by enunciating the name of the drug choose to swipe the drugs after administration rather than before, despite the fact that wrong drugs have in fact been identified on swiping after the event.
The astonishing thing about this example is that it is hard to see any way in which it is easier or more efficient than using the system properly. Objections have also been advanced to requests for double-checking of drugs (for example, from the New Zealand Medical Council following another patient’s death from yet another drug error), not only because double checking may occasionally fail (incident reports testify to this) but on the grounds that it might actually be counter productive (a highly improbable proposition for which we can find no empirical evidence).

Reluctance over patient safety initiatives sometimes reflects denial that a problem exists, or optimist bias (the belief that a problem applies only to other practitioners or organisations). In this case, we suspect that it also reflects a lack of awareness of the empirical and theoretical research which today not only continues to add to the increasingly indisputable evidence of the alarming magnitude of avoidable patient harm from medication errors, but also provides good guidance on many useful and sensible ways to improve the processes by which medications are presented, prescribed and administered (see below).

In all of these examples, one would surely expect constructive efforts to make sensible initiatives work rather than nihilist defeatism based on unrealistic demands for evidence and the notion that if we can’t achieve perfection it is not worth striving for any improvement on the status quo. In fact, pursuing small gains in a continuous iterative cycle of Plan Do Check Act (PDCA) is a widely accepted principle for quality improvement. In patient safety, every little bit helps.

Kunac and Reith’s observation that “Patients were supportive of the study” is not surprising. Of course patients want this problem addressed. It is very encouraging that the Quality Improvement Committee has identified Safe Medication Management as one of five priorities for healthcare in New Zealand, and that the Government has allocated $10.2 million to this end.

A national programme will encompass a standardised hospital medication chart with built-in safety features, medicine reconciliation, electronic prescribing with standardised information on medicines with appropriate links between hospital information systems, and the use of barcodes on unit doses of medications for bedside verification of administered drugs.

Medication error is an entrenched and multi-faceted problem and it will take time for these initiatives to work. That time would be considerably shortened if everyone concerned elevated this issue to the priority it deserves, stopped raising vexatious objections to common sense initiatives and engaged fully in the fundamental healthcare responsibility of always giving the right drug by the right route to the right patient in the right dose at the right time.

Competing interests: Professor Alan Merry has financial interests in improving safety in healthcare and chairs the ANZTADC which is promoting anaesthesia incident monitoring.

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Personalised medicine in New Zealand

Patrick Gladding

Pharmacogenomics is the branch of pharmacology which deals with the influence of genetic variation on drug response in patients by correlating gene expression or single-nucleotide polymorphisms with a drug's efficacy or toxicity. By doing so, pharmacogenomics aims to develop rational means to optimise drug therapy, with respect to the patients' genotype, to ensure maximum efficacy with minimal adverse effects. Such approaches promise the advent of "personalised medicine", in which drugs and drug combinations are optimised for each individual's unique genetic makeup (Wikipedia: [http://en.wikipedia.org/wiki/Pharmacogenomics](http://en.wikipedia.org/wiki/Pharmacogenomics)).

Whilst personalised medicine has gained considerable attention internationally, it has not entered the consciousness of the New Zealand public. This is likely to change, however, as it emerges as an important field in medicine with major implications for patient management. Indeed, innovative research in this field is being performed in New Zealand at the Carney Centre, which is a centre of excellence for pharmacogenomics, based in Christchurch.

Personalised medicine principally surrounds the field of molecular biology, and includes many of the 'omic' sciences such as genomics, transcriptomics, proteomics, and metabolomics.

A leading ‘omic’ science, which has already reached clinical practice, and been in existence for some decades, is pharmacogenetics/omics (the concept of genetic testing to determine pharmacological treatment). With the ability to test genomes instead of individual gene variants, pharmacogenetics has evolved into pharmacogenomics, which is proving in some circumstances, to be a powerful predictor of drug response and toxicity.

The article by Lea et al in this issue of the NZMJ provides some crucial information that needs to be extrapolated into clinical trials in Māori: *Allele frequency differences of cytochrome P450 polymorphisms in a sample of New Zealand Māori: [http://www.nzma.org.nz/journal/121-1272/3002](http://www.nzma.org.nz/journal/121-1272/3002).*

Ethnicity is an issue that arises frequently in any debate associated with predisposition to genetic disease. Although variation between individual human genomes differs by less than 0.1%, and that the variation within one ethnic group may be greater than between ethnic groups, there are some important interethnic genetic differences to consider.

Warfarin is the prototypic example of a drug that displays some inter-ethnic variation in dose response. Chinese individuals, for instance, have been shown to require lower doses of warfarin to achieve a therapeutic effect. This observation predated pharmacogenetic testing for warfarin but is now mostly explained by the higher carriage of VKORC1 variants (low-dose warfarin Group A Haplotype). This is despite the lower frequency of CYP2C9 loss of function variants in Chinese, which on their own would theoretically decrease warfarin metabolism and increase drug effect.
It is important to consider the combined effect of different genotypic variants when considering the therapeutic outcome of any medication.

Of considerable interest is the frequency of the known CYP gene variants, associated with metabolism of warfarin, in other ethnic groups. The work here provided by Lea et al looks at the frequency of functional CYP polymorphisms, and provides some important insights to interethnic response to drugs.

Of relevance to Māori should be their proposed genetic ancestry. The work by Whyte et al has suggested that Māori originated in indigenous Taiwan and Melanesia, which is supported by the presence of the CYP2C9 gene variant in Maori, that is also seen in South East Asians.\(^6\) The immediate extrapolation from this is that Māori may require different warfarin doses, as do South East Asians. However this may oversimplify things as it fails to take into account the complex effect of the other interacting genes (e.g. VKORC1).

Further research is required to establish in these populations a complete understanding of the relevant contribution of important gene variants in determining the response to drugs such as warfarin.

The response to clopidogrel, recently shown to be influenced by CYP2C19 variants,\(^7\)\(^–\)\(^12\) may be another example where Māori respond differently. A loss of function CYP2C19 gene variant occurs in higher frequency in Māori than Europeans and might mean that clopidogrel is less effective in Māori. This concern of interethnic differences in drug response continues to arise and needs to be urgently addressed in further studies.

A poor response to clopidogrel has been associated with adverse outcomes such as increased vascular events and death after coronary intervention.\(^13\)

Though there are hints of benefit, pharmacogenetic testing for warfarin has yet to be proven in a prospective trial to offer more clinical utility than warfarin dosing nomograms.\(^14\) However one must question the applicability of such nomograms when they have been developed using a Bayesian methodology in populations of only one ethnicity (usually European).\(^15\) Furthermore, warfarin nomograms may not be widely used, adding to the variability of drug response, caused by diverse prescribing habits (Table 1).

Table 1. Questionnaire measuring prescribing habits of health professionals at Auckland City Hospital (ACH)

<table>
<thead>
<tr>
<th>Questions</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you follow warfarin nomograms?</td>
<td>60%</td>
<td>40%</td>
</tr>
<tr>
<td>Do you think a nomogram is necessary?</td>
<td>67%</td>
<td>33%</td>
</tr>
<tr>
<td>Do you think nomograms are evidence-based?</td>
<td>55%</td>
<td>45%</td>
</tr>
<tr>
<td>Did you know there is a new nomogram at ACH?</td>
<td>74%</td>
<td>26%</td>
</tr>
<tr>
<td>What is your preferred loading dose (LD) regimen?</td>
<td>variable</td>
<td></td>
</tr>
<tr>
<td>When do you check INR during loading?</td>
<td>variable</td>
<td></td>
</tr>
</tbody>
</table>

42 respondents: House Officer (n=14), Registrar (n=17), Consultant (n=10), Medical Officer Staff Grade (n=1). Survey taken March 2008, 4 months after the introduction of a new warfarin nomogram to a major teaching hospital. Note that the Hawthorne effect\(^16\) may mean the usage of nomograms is even lower than that stated.
Although nomograms have been shown to be more effective than unguided clinician prescribing, doctors seem convinced that they can do it better. Cost-effectiveness studies assessing the benefit of warfarin pharmacogenetics, in terms of a one-off prediction of maintenance dose, may eventually provide the proof-of-concept of this technology.

Although only a handful of genetic variants appear to be important, when considering warfarin dosing, the importance of these variants needs validating in the diverse ethnic populations of New Zealand. However in a multicultural, genetically admixed, population such as New Zealand, warfarin pharmacogenetics offers an attractive alternative to nomograms, which cannot account for all the influencing variables.

The provision of drug treatment according to ethnicity has not been popular in the past. For example Bidil™ is a hydralazine/nitrate combination drug for heart failure which was targeted to African Americans based on ethnicity, rather than genetics. Treating to genetic determinants of drug absorption, metabolism, clearance, and drug effect makes more pragmatic sense, especially if there are reported admixture and ethnic variation in frequency of causal genotypes.

Of course the fear of genetic information superseding ethnic discrimination is real. In the United States, a Genetic Information Non-Discrimination Act (GINA) is in the process of being passed. It is believed that without this Act, personalised medicine will not gain traction with the public. Signs indicate that this is moving ahead. The era of Personalised Medicine is nearly with us and it is time to discuss how it should be best managed.

Footnote: The United States House of Representatives most recently passed the Genetic Information Non Discrimination Act (GINA) on 7 March 2008 as part of the Paul Wellstone Mental Health and Addiction Equity Act of 2007. It now has to pass through the US Senate.

Competing interests: None known.

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Health services research: an essential part of a wider spectrum of national health information?

Laurence Malcolm, Pauline Barnett

The viewpoint article by Brown and Ashton in this issue of the NZMJ presents a useful overview of the current state of health services research (HSR) in New Zealand. They note both that the potential for undertaking HSR is now greater than ever before and that it is more likely to make a significant contribution to improvements in the health sector and to health outcomes. However they also note that there are significant barriers to undertaking HSR. These include a marked gap between providers and academics, limited research capability, devolution of power to 21 district health boards (DHBs), and the challenges facing academics in making their research relevant to providers.

They propose two potential solutions. First is the possibility of setting up research centres to undertake independent work in broad areas of health policy concern—an example is the Australian Primary Health Care Research Institute. Second, they suggest that funders and providers might set up a separate independent body with more of an advisory and priority setting role. They cite the Kings Fund in the UK as a typical example.

While such models may have a place, it is important first to consider both the place of HSR within the wider spectrum of health research, and then its role in relation to other sources of health information in New Zealand.

First, the full spectrum of health research ranges from basic biomedical research through clinical, epidemiological, behavioural, and HSR including the evaluation of health services. The World Health Organization (WHO) takes a broad view of HSR and includes research into health systems and health policy, stressing the importance of seeking a researched understanding of the political environment within which health systems must function.

Not surprisingly there are significant overlaps and fuzzy boundaries between the above research categories. For example, a recent study of the use of echocardiography undertaken by cardiologists showed a threefold variation in its utilisation between DHBs. Was this a clinical study or HSR? It would seem the latter as its primary aim was to explore the pattern of use of such services.

This illustrates the extent to which many clinicians, both primary and secondary, are undertaking HSR in their search for better outcomes and making the best use of resources. It also illustrates the wide clinical variation in utilisation of health services, an almost universal finding in HSR studies. Particularly it confirms that health research in all its categories is not an end in itself and is the business of both funders and providers. Its utility must be judged against the extent to which it contributes to improved outcomes and the best use of health resources.
Research at the biomedical end of the spectrum has always been accorded a high priority. However international priorities in the past few decades have strongly stressed the need for balance in the distribution of health research resources. The change from the Medical to the Health Research Council in 1989 typified this broadening of priorities in New Zealand.

Research at the biomedical end of the spectrum is attractive because it is typically generalisable internationally. Health policy and services research is often only nationally or even locally relevant. Yet they have a major role is achieving better health outcomes and use of resources. For example the recent Auckland attempt to control laboratory expenditure of about NZ$15 million through contracting for price missed the possible savings of $40 million annually shown by HSR to be possible through engaging GP leadership in the much more important issue of volume control as has been achieved in Christchurch. 6

In terms of the role of HSR in New Zealand, health research in all its forms needs to be considered in the context of the totality of all the information needed to organise and manage a health system effectively. In other words it is one part of a national approach to health information. This totality also needs to include the New Zealand Health Information Service (NZHIS); data collection and analysis; research and development; information technology and standards; patient identity and security systems; library services and the many other databases and sources of information; and research projects, both large and small.

Considering this wider view of health information and the place of HSR within it, the two options proposed by Brown and Ashton have some limitations. Their suggestions may go some way to developing capacity in key areas, but may not address the need to integrate HSR into a national approach to health information and research.

HSR and related health policy research are undoubtedly much more effectively utilised when commissioned by executives and providers. A good New Zealand example is the establishment of the National Primary Health Strategy initiated in 2002. 7 Significant HSR and related studies commissioned by the Ministry of Health and the National Health Committee provided a firm basis for the strategy and contributed substantially to its success. 8

Similarly, the studies by Davis et al which have contributed significantly to action on treatment injury were undertaken as joint activities between clinicians and researchers and closely involved hospital executives. 9 Ashton and Brown point out that there is a need for closer relationships between information providers and users, but what is the framework in which this can take place?

It would appear that there is a critical leadership role for the Ministry Health in advancing health research generally and HSR in particular. The recent restructuring of the Ministry has established an information directorate at the deputy director level. However although it is responsible for ‘providing leadership and the capacity to manage the sector’s information systems,’ it is not clear that it has a role in advancing research as part of a national information system. (An earlier restructuring of the Ministry in the early 1990s did provide for such a role but this was disestablished, presumably because it was seen as inconsistent with the commercial adventurism of that time.)
An important ongoing initiative now coming under the leadership of the information directorate is the Ministerial Health Information Strategy Action Committee (HISAC)\(^{10}\) which was established to provide governance, oversight, and leadership for the implementation of the Health Information Strategy for New Zealand. It has 12 action zones, primarily concerned with national data collections, standards, and accessibility. However there appears to be no complementary HSR strategy to make use of this major initiative and to translate data into information and intelligence. As is well known, data analysis is the most critical factor in ensuring accuracy and reliability of the collection process.

In summary, therefore, some structures highly relevant to the future of HSR have recently been established. What especially might be needed is for the Ministry of Health Information Directorate to assume some leadership for a more comprehensive approach to national health information. This will include support for building a HSR research culture within provider organisations, a stronger perspective on research priorities, and the translation of these into the management and funding of a greater range of research and development activity within the sector.

**Competing interests:** None known.

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Preventable medication-related events in hospitalised children in New Zealand

Desireé L Kunac, David M Reith

Abstract

Aims To evaluate the frequency and characteristics of preventable medication-related events in hospitalised children, to determine the yield of several methods for identifying them and to recommend priorities for prevention.

Methods A prospective observational cohort study was conducted over a 12-week period on the paediatric wards at a university-affiliated urban general hospital in New Zealand. For all admissions of greater than 24 hours, medication-related events were identified using a multifaceted approach and subsequently classified independently by three reviewers (using a standardised reviewer form) by event type, type of error, stage of the medication process, and preventability.

Results There were 495 eligible study patients, who had 520 admissions and 3037 patient days of admission, during which 3160 medication orders were written. Of 761 medication-related events reported during the study period, 630 (83.3%) were identified by chart review; 111 (14.6%) by a voluntary staff quality improvement reporting system; 16 (2.1%) by interview of parents; and 4 (0.53%) events via the concurrent routine hospital-incident reporting system.

Excluding duplicate reports and practice-related issues, a total of 696 study patient-specific events were included in the analysis. Excluding the inconsequential events (trivial rule violation and ‘other’ categories), the majority [368/399 (92.2%)] of events were found to be preventable; comprising 38/67 (56.7%) ADEs, 75/77 (97.4%) potential ADEs, and all 255 (100%) harmless medication errors. Most commonly implicated in preventable ADEs and potential ADEs were, event rate (95%CI): improper dose and the prescribing stage—35 (29 to 42) and 74 (64 to 84) respectively per 1000 patient days; and antibacterial agents and the intravenous route of administration 21 (17 to 25) and 11 (10 to 13) respectively per 100 medication orders.

Conclusions Preventable medication-related events occur commonly in the paediatric inpatient setting, and importantly over half of the events that caused patient harm were deemed preventable. Voluntary staff reporting in a quality improvement environment was found to be inferior to chart review for identifying events, but a vast improvement on the conventional incident reporting system. Most commonly implicated in the harmful or potentially harmful preventable events, and hence the best targets for prevention are dosing errors, particularly during the prescribing stage of the medication use process, and use of antibacterial agents, particularly when administered by the intravenous route.

Medications are internationally found to be the leading cause of patient injury in the hospital setting,1–3 are costly, and are responsible for significant patient morbidity and mortality.4
The NZ Quality of Healthcare Study found medicines to be the third highest adverse event overall, associated with 15% of all adverse events. The impact of these events was considerable—reported to prolong hospital admission by a mean of 7.8 days, and 12.3% of cases were associated with permanent disability and death.

Data pertaining to the paediatric inpatient population is limited. In a North American population, the rate of preventable medication-related patient injury, termed preventable adverse drug events (ADEs), was found to be 1.8 per 1000 patient days. Whilst this rate was similar to that of a previous adult hospital study, the potential ADE rate (all deemed preventable) was three times higher in children than adults. The World Health Organization (WHO) Collaborating Centre for Patient Safety has released nine lifesaving patient safety solutions, several of which address the issues of medication safety. The issues include look-alike, sound-alike medication names; patient identification; concentrated electrolyte solutions; and assuring medication accuracy at transitions of care. However, medication safety issues in children may be different to those for adults and require different preventive measures.

In children there are issues of dosing and administration of medicines because of differences in scale (size), developmental changes in drug disposition, and the unsuitability of many drug formulations. In addition, there are issues of inadequate drug information and lack of drug registration and labelling for the paediatric population. Hence there is a need to identify the nature and incidence of preventable medication-related adverse events in the paediatric population.

**Aims**

To evaluate the frequency and characteristics of preventable medication-related events in hospitalised children, to determine the yield of several methods for identifying them and to recommend priorities for prevention.

**Methods**

**Study design and setting**—A prospective observational cohort study was conducted over a 12-week period from 18 March to 9 June 2002 at a university-affiliated urban general hospital in New Zealand. All admissions to the neonatal intensive care unit (NICU), postnatal ward, and paediatric ward during the study period were included and followed through to discharge from hospital. Patients were excluded if the hospital admission was for less than 24 hours or if medical staff deemed it inappropriate for a patient to be involved. The study was granted regional Ethics Committee approval.

**Medication use process**—At the time of the study, medication orders were handwritten by medical staff directly on to the patient’s medication chart which has an integrated medication administration record (MAR), thus eliminating the need for transcription of orders. A supply of medications is available on the wards, and patients are also encouraged to bring their current medications in to hospital, with nursing staff subsequently performing any dose calculations and administration. It is standard procedure for all paediatric drug administrations to be double checked (i.e. independent two-nurse check) prior to administration to the patient.

Clinical pharmacist activities on NICU and the paediatric ward included daily ward visits and medication chart review; regular attendance at weekly multidisciplinary ward meetings; but no active participation in ward-based rounds. There was no regular clinical pharmacy service provided for the postnatal ward.
**Event identification**—Medication-related events were identified by the principal investigator (DK) using a multi-faceted approach involving four methods to maximise data yield:

- Chart review for all admissions
- Attendance at multidisciplinary ward meetings
- Interview of parents/caregivers (and children) when further information or clarification of information was required
- Voluntary and verbally solicited reports from staff. Staff were instructed to follow the Hospital Incident Reporting system in the usual way. However, as this traditionally had such a low yield of events, all paediatric ward staff were educated about the study. We emphasised the role of systems problems in the origin of errors and encouraged voluntary reports of any actual events or potentially unsafe medication systems that they noted during their daily activities.

**Table 1. Medication-related event types: definitions and examples (adapted from Kaushal et al)***

<table>
<thead>
<tr>
<th>Event type</th>
<th>Definition</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adverse drug events (ADE)</td>
<td>Actual injuries resulting from medical interventions related to a medicine</td>
<td>Troublesome drug rash requiring intervention</td>
</tr>
<tr>
<td>Preventable ADE</td>
<td>Actual injuries resulting from the use of medication in error</td>
<td>The development of a rash after administration of flucloxacillin in a patient known to be allergic to penicillins</td>
</tr>
<tr>
<td>Non-preventable ADE (adverse drug reactions)</td>
<td>Actual injuries resulting from the use of a medication not associated with error, also termed adverse drug reactions (ADRs)</td>
<td>The development of a rash after administration of flucloxacillin in a patient with no known drug allergies</td>
</tr>
<tr>
<td>Potential ADE</td>
<td>Events that have a significant potential for injuring a patient but do not actually cause harm. This may be because they are intercepted before reaching the patient or, due to particular circumstances or chance, the patient is able to tolerate the event</td>
<td>A prescription order written for a 10-fold overdose of digoxin that is intercepted and corrected by the pharmacist before reaching the patient. A non-intercepted potential ADE would be the administration of a non-steroidal anti-inflammatory agent to an asthmatic patient who does not experience any adverse effects.</td>
</tr>
<tr>
<td>Harmless medication error</td>
<td>Harmless errors associated with the use of a medication</td>
<td>Administration of one regular dose of non-critical medication given more than two hours later than scheduled</td>
</tr>
<tr>
<td>Trivial rule violation</td>
<td>Faulty medication orders with little potential for harm or extra work because they are typically interpreted correctly by pharmacy and nursing staff without additional clarification</td>
<td>Prescription written for regular medication but not dated</td>
</tr>
<tr>
<td>Other events</td>
<td>Any reported events not classified as one of the above event types</td>
<td>Mild side effects that are tolerated without the need of intervention</td>
</tr>
</tbody>
</table>

**Review process and definitions**—All suspected medication-related events were reviewed by a panel of three health professionals (a clinical pharmacist, a clinical pharmacologist/paediatrician, and a neonatologist) who independently categorised the events using a standardised reviewer form. Prior to this process, the reviewers underwent a calibration exercise using simulated test cases. As a result of discussions regarding these test cases, reviewer guidelines were agreed which contained explanatory notes about the reviewer process and definitions with examples for the event types, as shown in Table 1. The term ‘injury’, as contained in these definitions is defined as damage or harm and includes the entire range of seriousness, from very minor injuries to life-threatening injuries.
The reviewers independently categorised each of the suspected medication-related events by type (Table 1) and for preventability on the basis of the practitioner’s presumed knowledge at the time the medication was prescribed. Harmless medication errors by definition were automatically deemed preventable events. Trivial rule violations, and ‘other’ events were excluded from any further analyses. The process of categorising the events has been evaluated and was found to have excellent inter-rater and intra-rater reliability. For preventable events, a further judgement was required by each reviewer to identify the:

- Stage(s) in the medication use process (prescribing, dispensing, administration, or monitoring) where the error occurred.
- Type of error, using a taxonomy of medication errors.
- Origin of the event, either prior to hospital admission, during hospitalisation or at discharge.

The Society of Hospital Pharmacists of Australia classification of drug categories was used to group medications.

Incidence rates and Poisson 95% Confidence Intervals (95%CI) were calculated using Stata® (version 8) software (College Station, TX, USA).

Results

A total of 676 patients were admitted to the paediatric wards during the 12-week study period, of which 181 patients were excluded (179 patients were excluded due to a length of stay less than 24 hours and 2 patients were judged by the medical team as inappropriate to be included). This resulted in 495 eligible study patients, who had a total of 520 admissions and 3037 patient days of admission, during which 3160 medication orders were written.

Just over half of the admissions were male (53.7%), ranging from newborn to almost 17 years of age and had a median length of stay of 3 days.

Identification of medication-related events

A total of 761 medication-related events were reported during the study period. The majority [630 (83.3%)] of events were identified by the principal investigator (DK); 111 (14.6%) identified by staff using the voluntary reporting process; 16 (2.1%) by parents/children via interview or voluntary report to investigator or a staff member, and 4 (0.53%) events via the concurrent routine hospital incident reporting system. Excluding 14 duplicate reports (same event independently reported by two or more individuals) and 51 practice-related issues, a total of 696 study patient-specific events were included in the analysis.

Of the events identified by staff or parents, nurses were the best reporters of events followed by hospital pharmacists and parents (Figure 1). Parents were supportive of the study; 110 were approached for interview and 106 (96.6%) gave consent for interview.

Suspected medication-related events were reported to occur in almost one-third of the study population. The number of reports per admission ranged from 1 to 78 (median 2). Of those admissions who experienced events, 49 (32%) of admissions experienced 1 event, 34 (24%) of admissions experienced 2 events, and 67 (44%) of admissions experienced 3 or more events (of these 14 (9%) of admissions experienced 10 or more events).
Classification of event types

Classification of events by type are shown in Figure 2. Two ‘potential ADEs’ classified by the reviewers were deemed ‘non-preventable’; in one case gentamicin trough levels were higher than anticipated despite adherence to hospital dosing guidelines, and in the second case, delays in antibiotic therapy occurred due to difficulties in gaining intravenous access. Excluding the inconsequential events (trivial rule violation and ‘other’ categories) the majority [368 (92.2%)] of events were found to be preventable; comprising 38 (56.7%) ADEs, 75 (97.4%) potential ADEs, and all 255 (100%) harmless medication errors.

Figure 2. Classification of events by type
Characteristics of preventable events

Type of error—For preventable ADEs and potential ADEs, improper dose was the most common error type, whereas most harmless medication errors were due to dose omissions, followed by improper dose (Table 2).

Stage of medication use process—Preventable events could be attributed to more than one stage of the medication use process. Of the total of 368 preventable events, 224 involved the prescribing stage, 34 involved the dispensing stage, 164 the administration stage, and 55 the monitoring stage (Table 3).

The most common stage of the medication use process involved in preventable ADEs and potential ADEs was prescribing then monitoring. For harmless medication errors, the most common stage was administration then prescribing (Table 3). For all preventable events, the stages most frequently involved were the prescribing and administration stages.

Among prescribing errors, improper dose was the most common, followed by wrong drug, dose omission, monitoring error and wrong route of administration. The most common dispensing errors were improper dose, dose omission and wrong time administration (due to delays in dispensing), and deteriorated drug error (which involved dispensing of an expired medication).

For the administration stage, dose omission was the most common error, followed by wrong time and improper dose. Monitoring stage errors related to drug-drug interaction, drug-disease interaction, and improper dose (due to inadequate monitoring).

Origin of event—The majority of preventable events were found to originate during hospitalisation 344 (93.5%), with only 10 (2.7%) occurring prior to admission and 14 (3.8%) at discharge.

Class of medication—Preventable events were associated with 25 of the 36 different classes of medication prescribed during the study period (Table 4). In terms of the impact of medicine classes upon the number and severity of adverse events, the most commonly implicated classes of medicine were antibacterial agents, narcotic analgesics and non-narcotic analgesics. In the context of frequency of prescription episode, the rankings were antidotes and cytoprotectives, enzyme replacement, drugs acting principally on blood forming, and antifungal and antiviral agents.

Route of administration—The intravenous and oral routes of medication administration were by far the most commonly implicated in all preventable events (Table 5). For harmless medication errors, the oral route predominated; however for preventable ADEs and potential ADEs, the intravenous route was the most frequently involved. The rate of events per 100 medication orders was highest for the nasogastric and inhaled routes.
Table 2. Error type for preventable events

<table>
<thead>
<tr>
<th>Type of error</th>
<th>Preventable ADEs</th>
<th>Preventable potential ADEs</th>
<th>Harmless medication errors</th>
<th>Total (%)</th>
<th>Rate/100 admissions (95% CI)</th>
<th>Rate/1000 patient days (95% CI)</th>
<th>Rate/100 medication orders (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>70.1 Dose omission</td>
<td>5</td>
<td>1</td>
<td>89</td>
<td>95 (25.8)</td>
<td>18 (15 to 22)</td>
<td>31 (25 to 38)</td>
<td>3.0 (2.4 to 3.7)</td>
</tr>
<tr>
<td>70.2 Improper dose</td>
<td>11</td>
<td>42</td>
<td>53</td>
<td>106 (28.8)</td>
<td>20 (17 to 25)</td>
<td>35 (29 to 42)</td>
<td>3.4 (2.7 to 4.1)</td>
</tr>
<tr>
<td>70.2.1 resulting in overdose</td>
<td>7</td>
<td>30</td>
<td>20</td>
<td>57 (15.5)</td>
<td>11 (8 to 14)</td>
<td>19 (14 to 24)</td>
<td>1.8 (1.4 to 2.3)</td>
</tr>
<tr>
<td>70.2.2 resulting in underdose</td>
<td>2</td>
<td>7</td>
<td>22</td>
<td>31 (8.4)</td>
<td>6 (4 to 8)</td>
<td>10 (7 to 14)</td>
<td>1.0 (0.7 to 1.4)</td>
</tr>
<tr>
<td>70.2.3 extra dose</td>
<td>2</td>
<td>5</td>
<td>11</td>
<td>18 (4.9)</td>
<td>3 (2 to 5)</td>
<td>6 (4 to 9)</td>
<td>0.6 (0.3 to 0.9)</td>
</tr>
<tr>
<td>70.3 Wrong strength / concentration</td>
<td>0</td>
<td>4</td>
<td>5</td>
<td>9 (2.5)</td>
<td>2 (1 to 3)</td>
<td>3 (1 to 6)</td>
<td>0.3 (0.1 to 0.5)</td>
</tr>
<tr>
<td>70.4 Wrong drug</td>
<td>5</td>
<td>3</td>
<td>21</td>
<td>29 (7.9)</td>
<td>6 (4 to 8)</td>
<td>10 (6 to 14)</td>
<td>0.9 (0.6 to 1.3)</td>
</tr>
<tr>
<td>70.5 Wrong dose form</td>
<td>0</td>
<td>1</td>
<td>7</td>
<td>8 (2.2)</td>
<td>2 (1 to 3)</td>
<td>3 (1 to 5)</td>
<td>0.3 (0.1 to 0.5)</td>
</tr>
<tr>
<td>70.6 Wrong technique</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3 (0.8)</td>
<td>1 (0 to 2)</td>
<td>1 (0 to 3)</td>
<td>0.1 (0.0 to 0.3)</td>
</tr>
<tr>
<td>70.7 Wrong route of administration</td>
<td>0</td>
<td>2</td>
<td>8</td>
<td>10 (2.7)</td>
<td>2 (1 to 4)</td>
<td>3 (2 to 6)</td>
<td>0.3 (0.2 to 0.6)</td>
</tr>
<tr>
<td>70.8 Wrong rate</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>7 (1.9)</td>
<td>1 (0 to 3)</td>
<td>2 (1 to 5)</td>
<td>0.2 (0.1 to 0.5)</td>
</tr>
<tr>
<td>70.9 Wrong duration</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>7 (1.9)</td>
<td>1 (0 to 3)</td>
<td>2 (1 to 5)</td>
<td>0.2 (0.1 to 0.5)</td>
</tr>
<tr>
<td>70.10 Wrong time</td>
<td>2</td>
<td>0</td>
<td>35</td>
<td>37 (10.1)</td>
<td>7 (5 to 10)</td>
<td>12 (9 to 17)</td>
<td>1.2 (0.8 to 1.6)</td>
</tr>
<tr>
<td>70.11 Wrong patient</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1 (0.3)</td>
<td>0 (0 to 1)</td>
<td>0 (0 to 2)</td>
<td>0 (0 to 0.2)</td>
</tr>
<tr>
<td>70.12 Monitoring error</td>
<td>5</td>
<td>11</td>
<td>8</td>
<td>24 (6.5)</td>
<td>5 (3 to 7)</td>
<td>8 (5 to 12)</td>
<td>0.8 (0.5 to 1.1)</td>
</tr>
<tr>
<td>70.13 Deteriorated drug error</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>4 (1.1)</td>
<td>1 (0 to 2)</td>
<td>1 (0 to 3)</td>
<td>0.1 (0 to 0.3)</td>
</tr>
<tr>
<td>70.14 Other</td>
<td>6</td>
<td>6</td>
<td>16</td>
<td>28 (7.5)</td>
<td>5 (4 to 8)</td>
<td>9 (6 to 13)</td>
<td>0.9 (0.6 to 1.3)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>38</strong></td>
<td><strong>75</strong></td>
<td><strong>255</strong></td>
<td><strong>368</strong></td>
<td><strong>71 (64 to 73)</strong></td>
<td><strong>121 (109 to 134)</strong></td>
<td><strong>11.6 (10.5 to 12.9)</strong></td>
</tr>
</tbody>
</table>
Table 3. Preventable events by type of error and stage of medication use process

| Preventable events by type of error and by stage of medication use process# | Stage of medication use process |
|---|---|---|---|---|
|  | Prescribing | Dispensing | Administration | Monitoring |
| Preventable ADEs n=38 | 32 | 2 | 10 | 18 |
| Potential ADEs n=75 | 66 | 4 | 9 | 19 |
| Harmless medication errors n=255 | 126 | 28 | 145 | 18 |
| 70.1 Dose omission | 20 | 4 | 83 | 8 |
| 70.2 Improper dose | 95 | 10 | 21 | 19 |
| 70.3 Wrong strength / concentration | 9 | 0 | 1 | 0 |
| 70.4 Wrong drug | 27 | 1 | 3 | 3 |
| 70.5 Wrong dose form | 7 | 3 | 4 | 0 |
| 70.6 Wrong technique | 1 | 0 | 0 | 1 |
| 70.7 Wrong route of administration | 10 | 0 | 3 | 0 |
| 70.8 Wrong rate | 7 | 0 | 1 | 1 |
| 70.9 Wrong duration | 6 | 0 | 1 | 1 |
| 70.10 Wrong time | 7 | 4 | 35 | 0 |
| 70.11 Wrong patient | 0 | 1 | 0 | 0 |
| 70.12 Monitoring error | 14 | 1 | 3 | 19 |
| 70.13 Deteriorated drug error | 0 | 4 | 4 | 0 |
| 70.14 Other | 21 | 6 | 5 | 3 |
| Total preventable events | 224 | 34 | 164 | 55 |
| Rate of preventable events/100 admissions (95% CI) | 43 (38 to 49) | 7 (5 to 9) | 32 (27 to 37) | 11 (8 to 14) |
| Rate of preventable events/1000 patient days (95% CI) | 74 (64 to 84) | 11 (8 to 16) | 54 (46 to 63) | 18 (14 to 24) |
| Rate of preventable events/100 medication orders (95% CI) | 7.1 (6.2 to 8.1) | 1.1 (0.7 to 1.5) | 5.2 (4.4 to 6.0) | 1.7 (1.3 to 2.3) |

# Preventable events could be attributed to more than one stage of process.
### Table 4. Preventable events by medicine class

<table>
<thead>
<tr>
<th>SHPA Code Group</th>
<th>Medication class</th>
<th>Preventable ADE</th>
<th>Potential ADE</th>
<th>Harmless medication error</th>
<th>Total events n</th>
<th>Number of medication orders, N (n/N%)</th>
<th>Event rate (95%CI) per 100 medication orders</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Parasympathetics</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>100 (1)</td>
<td>1 (0 to 6)</td>
</tr>
<tr>
<td>12</td>
<td>Sympathetic agents</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>6</td>
<td>101 (5.9)</td>
<td>6 (2 to 13)</td>
</tr>
<tr>
<td>14</td>
<td>Neuromuscular junction modifiers</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>42 (0)</td>
<td>0 (0 to 9)</td>
</tr>
<tr>
<td>18</td>
<td>Antitussives and antipolypeptides</td>
<td>0</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>33 (9.1)</td>
<td>9 (2 to 27)</td>
</tr>
<tr>
<td>21</td>
<td>Anaesthetics</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>70 (2.9)</td>
<td>3 (0 to 10)</td>
</tr>
<tr>
<td>22</td>
<td>Analgesics – non narcotic</td>
<td>4</td>
<td>5</td>
<td>23</td>
<td>32</td>
<td>269 (11.9)</td>
<td>12 (8 to 17)</td>
</tr>
<tr>
<td>23</td>
<td>Analgesics – narcotic</td>
<td>4</td>
<td>17</td>
<td>5</td>
<td>26</td>
<td>212 (12.3)</td>
<td>12 (8 to 18)</td>
</tr>
<tr>
<td>25</td>
<td>Psychotherapeutic agents</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>35 (2.9)</td>
<td>3 (0 to 16)</td>
</tr>
<tr>
<td>26</td>
<td>Sedatives and hypnotics</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4 (0)</td>
<td>0 (0 to 92)</td>
</tr>
<tr>
<td>27</td>
<td>Central Nervous System specific purpose agents</td>
<td>6</td>
<td>4</td>
<td>18</td>
<td>28</td>
<td>189 (14.8)</td>
<td>15 (10 to 21)</td>
</tr>
<tr>
<td>31</td>
<td>Drugs acting principally on blood forming</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>10</td>
<td>18 (55.6)</td>
<td>56 (27 to 102)</td>
</tr>
<tr>
<td>32</td>
<td>Drugs acting on blood clotting</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (0)</td>
<td>0 (0 to 123)</td>
</tr>
<tr>
<td>34</td>
<td>Drugs and factors affecting blood composition</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>22 (0)</td>
<td>0 (0 to 17)</td>
</tr>
<tr>
<td>36</td>
<td>Drugs acting on blood vessels</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5 (0)</td>
<td>0 (0 to 74)</td>
</tr>
<tr>
<td>42</td>
<td>Antibacterial agents</td>
<td>10</td>
<td>25</td>
<td>65</td>
<td>100</td>
<td>480 (20.8)</td>
<td>21 (17 to 25)</td>
</tr>
<tr>
<td>43</td>
<td>Antibiotics specific application</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>27 (11.1)</td>
<td>11 (2 to 32)</td>
</tr>
<tr>
<td>45</td>
<td>Antifungal and antiviral agents</td>
<td>0</td>
<td>1</td>
<td>14</td>
<td>15</td>
<td>49 (30.6)</td>
<td>31 (17 to 50)</td>
</tr>
<tr>
<td>48</td>
<td>Antineoplastics</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (0)</td>
<td>0 (0 to 184)</td>
</tr>
<tr>
<td>49</td>
<td>Antiseptics, disinfectants</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3 (0)</td>
<td>0 (0 to 123)</td>
</tr>
<tr>
<td>52</td>
<td>Electrolyte replacement</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>6</td>
<td>37 (16.2)</td>
<td>16 (6 to 35)</td>
</tr>
<tr>
<td>53</td>
<td>Enzyme replacement</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>36 (66.7)</td>
<td>67 (8 to 241)</td>
</tr>
<tr>
<td>54</td>
<td>Fluid replacement</td>
<td>3</td>
<td>4</td>
<td>16</td>
<td>23</td>
<td>458 (5.0)</td>
<td>5 (3 to 8)</td>
</tr>
<tr>
<td>55</td>
<td>Hormones replacement</td>
<td>1</td>
<td>5</td>
<td>17</td>
<td>21</td>
<td>123 (17.1)</td>
<td>17 (11 to 26)</td>
</tr>
<tr>
<td>57</td>
<td>Nutritional products</td>
<td>2</td>
<td>4</td>
<td>18</td>
<td>24</td>
<td>218 (11.0)</td>
<td>11 (7 to 16)</td>
</tr>
<tr>
<td>58</td>
<td>Vitamins and other metabolic agents</td>
<td>0</td>
<td>2</td>
<td>32</td>
<td>34</td>
<td>425 (8.0)</td>
<td>8 (6 to 11)</td>
</tr>
<tr>
<td>72</td>
<td>Eye (local only)</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>13 (7.7)</td>
<td>8 (0 to 43)</td>
</tr>
<tr>
<td>75</td>
<td>Dental only</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 (0)</td>
<td>0 (0 to 369)</td>
</tr>
<tr>
<td>74</td>
<td>Gastrointestinal tract</td>
<td>2</td>
<td>2</td>
<td>7</td>
<td>11</td>
<td>56 (19.6)</td>
<td>20 (10 to 35)</td>
</tr>
<tr>
<td>76</td>
<td>Locally acting only on individual subsystems</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (0)</td>
<td>0 (0 to 184)</td>
</tr>
<tr>
<td>77</td>
<td>Kidney and bladder</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>6</td>
<td>45 (13.3)</td>
<td>13 (5 to 29)</td>
</tr>
<tr>
<td>78</td>
<td>Respiratory system</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>16 (12.5)</td>
<td>9 (2 to 45)</td>
</tr>
<tr>
<td>81</td>
<td>Skin preparations</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>8 (25.0)</td>
<td>25 (3 to 90)</td>
</tr>
<tr>
<td>85</td>
<td>Galenicals</td>
<td>1</td>
<td>0</td>
<td>6</td>
<td>7</td>
<td>74 (9.5)</td>
<td>9 (4 to 19)</td>
</tr>
<tr>
<td>86</td>
<td>Irrigation and perfusion fluids</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>8 (0)</td>
<td>0 (0 to 46)</td>
</tr>
<tr>
<td>89</td>
<td>Miscellaneous</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7 (0)</td>
<td>0 (0 to 53)</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td></td>
<td>38</td>
<td>75</td>
<td>255</td>
<td>368</td>
<td>3160 (11.7)</td>
<td>12 (10 to 13)</td>
</tr>
</tbody>
</table>

**SHPA** denotes Society of Hospital Pharmacists of Australia.
Table 5. Preventable events by route of administration

<table>
<thead>
<tr>
<th>Route of administration</th>
<th>Preventable ADE</th>
<th>Potential ADE</th>
<th>Harmless medication error</th>
<th>Total number of events, n</th>
<th>Number of medication orders, N (n/N%)</th>
<th>Event rate (95% CI) per 100 medication orders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intravenous</td>
<td>20</td>
<td>53</td>
<td>93</td>
<td>166</td>
<td>1480 (11.2)</td>
<td>11(10 to 13)</td>
</tr>
<tr>
<td>Oral</td>
<td>13</td>
<td>14</td>
<td>120</td>
<td>147</td>
<td>777 (18.9)</td>
<td>19 (16 to 22)</td>
</tr>
<tr>
<td>Nasogastric</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>13 (38.5)</td>
<td>38 (12 to 90)</td>
</tr>
<tr>
<td>Intramuscular</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td>338 (1.2)</td>
<td>1 (0 to 3)</td>
</tr>
<tr>
<td>UAC or UVC</td>
<td>0</td>
<td>4</td>
<td>1</td>
<td>5</td>
<td>52 (9.6)</td>
<td>10 (3 to 22)</td>
</tr>
<tr>
<td>Eye or ear drops</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>3</td>
<td>166 (1.8)</td>
<td>2 (0 to 5)</td>
</tr>
<tr>
<td>Rectal</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>48 (6.3)</td>
<td>6 (1 to 18)</td>
</tr>
<tr>
<td>Topical</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>10</td>
<td>60 (16.7)</td>
<td>17 (8 to 31)</td>
</tr>
<tr>
<td>Endotracheal</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>17 (11.8)</td>
<td>12 (1 to 42)</td>
</tr>
<tr>
<td>Inhaled</td>
<td>2</td>
<td>1</td>
<td>17</td>
<td>20</td>
<td>87 (23.0)</td>
<td>23 (14 to 36)</td>
</tr>
<tr>
<td>Subcutaneous</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>27 (3.7)</td>
<td>4 (0 to 21)</td>
</tr>
<tr>
<td>PEG</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>11 (18.2)</td>
<td>18 (2 to 66)</td>
</tr>
<tr>
<td>No route specified</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>84 (n/a)</td>
<td>0 (0 to 4)</td>
</tr>
<tr>
<td>Totals</td>
<td>38</td>
<td>75</td>
<td>255</td>
<td>368</td>
<td>3160 (11.7)</td>
<td>12 (10 to 13)</td>
</tr>
</tbody>
</table>

PEG denotes gastrostomy; UVC, umbilical venous catheter; UAC, umbilical arterial catheter.

Discussion

Yield of events

The rate of preventable medication-related events is influenced not only by health systems and settings but also to a major degree by the methods of detection. In the present study, a multifaceted approach to identification of events was employed, as no single approach will identify all types of events.

As expected, the most effective method of identification was chart review, with a five-fold yield compared with voluntary reporting, and the least effective method was via the hospital incident reporting process which identified only a minority (0.5%) of events. Interestingly, little overlap occurred between the methods of identification, with only 14 duplicate reports, which reinforces the need for multiple methods of identification of events to obtain not only a higher yield of events but also different types of events.

Of the voluntary reports, nurses reported more events than any other health professional (59%) which is consistent with previous paediatric studies; 59% and 61%. This may be because nursing staff are at the end of the line in terms of the medication use process and are therefore able to identify events at any preceding stage. The present study highlights the value of involving the parents and the patients themselves as they were an important means of event detection (12%) and also highlights the need to investigate strategies to enhance reporting by medical staff.
Preventable event rates

The overall rate of preventable events reported in the present study is comparable to similar studies in children, also using chart review and similar definitions for events. Kaushal (2001) reported similar rates of events, at around 7 per 100 medication orders, 65 per 100 admissions, and 190 per 1000 patient days. However, as would be expected these rates are higher than those from studies using only voluntary reporting systems.

Preventable event characteristics

The most common type of event detected in the present study was dosing errors and these occurred at a similar rate to that reported by Kaushal (2001) in a study undertaken in a US paediatric inpatient population using comparable methods.

Of the dosing errors, overdosing was more prevalent than underdosing in the present study, which is in contrast to previous work. Nevertheless, dosing errors have been found to be the most common type of medication error in many other paediatric inpatient studies, although there is great variation in the rates reported, likely due to different methodologies and settings. Furthermore, a recent review suggests that dosing errors are probably the most common type of error across the paediatric population (both for inpatients and outpatients).

The medication use process provides many opportunities for dosing errors in children. Doses for children are most often based on weight, clinical condition, and age. Miscalculations, particularly of the magnitude of 10-fold dosing errors are common in the paediatric population. Ten-fold dosing errors have recently been detected by chart review at a rate of 2 per 100 medication orders and have been identified as a contributor to serious adverse events.

The prescribing and administration stages were most commonly implicated in preventable events. This finding is consistent with previous studies where prescribing and/or administration stages have been highlighted as being the most error prone as compared to the dispensing and monitoring stages. However, this study has shown that it is within the prescribing stage that harmful or potentially harmful events arise.

The stage in the medication use process where transcription of medication orders occur has been shown to be error prone in studies undertaken in the US or Canada, but transcription errors did not occur in the present study because medications are prescribed directly onto the medication chart in New Zealand.

Almost all preventable events were found to originate during hospitalisation. Few events occurred prior to admission, resulting in a drug-related admission rate of 1.7% overall, and 4.5% if postnatal admissions are excluded. This is consistent with the findings of two studies investigating the frequency of drug-related admissions (which did not include postnatal admissions) to paediatric hospitals in Australia, where rates of 3.4% and 4.3% are reported.

In a review of studies of drug-related admissions to Australian hospitals this rate of paediatric drug-related admissions appears to be much lower than emergency admissions (6 to 7%), admissions to adult medical wards (12%), and emergency
admissions among the elderly (15 to 22%). This would seem reasonable as the paediatric population require fewer regular medications than adults, particularly the elderly where polypharmacy is a common problem.41

The medications most frequently implicated in medication-related events were analgesics and antibacterial agents. This supports the findings of Kaushal (2001)7 which found anti-infectives, followed by analgesics and sedatives, to have the highest rates of events. This pattern is different to that for adults, where antiplatelets, anticoagulants, diuretics, cardiovascular drugs, and cytotoxic agents are more frequently implicated in harm.40, 42

Consistent with previous work undertaken in the UK and the US7,21 we found that the intravenous route was associated with the highest overall number of events. However, the rate of events per 100 medication orders was highest for the nasogastric and inhaled routes. Adding to current knowledge, we also found that harmful or potentially harmful events were more commonly associated with intravenous medication administration whereas harmless medication errors were linked with oral administration.

Implications for prevention in clinical practice

Classification of medication-related events by type enables the distinction between those events due to error and those events that actually or potentially cause patient harm. The majority of medication-related events were found to be preventable and importantly, over half of the events that caused patient harm were deemed preventable. There is therefore a great opportunity to improve the safety of the paediatric inpatient medication use process.

Need for more effective routine detection mechanisms—An effective mechanism for routinely identifying and analysing medication-related events is fundamental to improving medicines safety. Spontaneous voluntary reporting, such as via incident reporting schemes, have traditionally been the main method of event identification in hospitals in New Zealand (NZ). Although an inexpensive method, as demonstrated in the present study, a major limitation is that it reveals a very low yield of events. Continued sole reliance on incident reporting for event identification in hospitals in NZ is inadequate and unlikely to have a major impact on improving the safety of medication use processes. Although effective in providing a high yield of events, the comprehensive chart review process utilised in the present study would be too expensive and time consuming to be used routinely in hospitals and is more suited to research purposes.

The strategy of the future should involve computerised surveillance systems that use detection programmes to search for events within electronic records that are likely to be associated with ADEs.43, 44 Until such time as prescription, medical, and administration records are automated within hospitals, the present study suggests incident reporting systems need to be supplemented with other methods such as the use of manual trigger tools45 and solicited staff and parent report.

Proactive risk evaluations of high-risk processes and procedures before events actually cause patient harm (by using methods such as failure mode effects analysis
[FMEA]) should also be employed.\textsuperscript{46} Indeed, a multi-pronged approach is clearly needed if improvements are to be achieved.

**Targets for prevention in clinical practice**—The American Academy of Pediatrics has produced comprehensive guidelines for medication error prevention in paediatric inpatients; including advice for hospitals, manufacturers, and health care professionals.\textsuperscript{47} Others have also provided recommendations for prevention of medication errors in neonatal care\textsuperscript{48} and in children in general.\textsuperscript{49} However, these recommendations are so comprehensive that it would be impossible for hospitals to implement all of these at once.

The findings of the present study provide further documentation of those areas in most need of attention. Dosing errors and the prescribing and administration stages of the medication use process are most commonly implicated in preventable events.

However the more serious events (ADEs and potential ADEs) were associated with dosing errors during the prescribing stage and when using antibacterial agents and narcotics, particularly when administered by the intravenous route so to reduce patient harm, prevention strategies should target these areas.

**Limitations**

The limitations of the present study include the generalisability of the findings to other health care systems; and the comparability of the methods of detection and classification of events with other studies. The findings of the present study are similar to those of Kaushal (2001) which used similar methods of detection and classification.\textsuperscript{7}

Although care was used in the study design to use methods that would enable comparison with previous work, because of the wide disparities of methods in the previous studies it was not possible to enable comparison with all the previously published studies.\textsuperscript{11–13,15} Despite a comprehensive approach to event identification, some errors would have gone undetected, particularly administration errors.

The direct observation method, using specially trained observers will reliably detect more errors at the point of administration than any other method.\textsuperscript{50} In addition, due to the study design reflecting a limitation of resources available, admissions of less than 24 hours duration were excluded from the study and any events experienced by these patients would have been missed. It is unlikely that any actual harmful event would have been missed as such events would have prolonged hospital stay.

The frequency of events may have been reduced over time, as it was the study protocol to advise of any serious practice problems so that corrective action could be taken. For the majority of events reported, the actual time of event was not recorded by the reporter, thus it was not possible to determine the diurnal variation of errors.

**Conclusions**

Preventable medication-related events occur commonly in the paediatric inpatient setting and importantly; over half of the events that caused patient harm were deemed preventable. Very few events were detected via the routine hospital incident reporting process; however staff (particularly nurses) supported a voluntary reporting system that had a quality improvement focus.
Continued sole reliance on incident reporting is inadequate and should be supplemented with other strategies that enhance medication-related event identification if patient care quality is to be improved.

Most commonly implicated in the harmful or potentially harmful preventable events (and hence the most likely targets for prevention) are dosing errors during the prescribing and monitoring stages of the medication use process, and use of antibacterial agents and narcotics, particularly when administered by the intravenous route.

**Competing interests:** None known.

Author information: Desireé L Kunac, Research Fellow, School of Pharmacy; David M Reith, Senior Lecturer, Department of Women’s and Children’s Health; University of Otago, Dunedin

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(This work was undertaken independent of the funding provided by CHRF. The CHRF had no involvement with the study design; collection, analysis, and interpretation of data; writing of the report; nor the decision to submit the paper for publication.)

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**References:**


Allele frequency differences of cytochrome P450 polymorphisms in a sample of New Zealand Māori

Rod A Lea, Rebecca L Roberts, Michael R Green, Martin A Kennedy, Geoffrey K Chambers

Abstract

Aims To determine the prevalence of functional alleles for drug metabolising genes in a sample of Māori and compare allele frequencies with Caucasians estimates.

Procedures DNA from 60 Māori volunteers was genotyped for cytochrome P450 polymorphisms—CYP2A6, CYP2C9, CYP2C19, and CYP2D6—and allele frequencies calculated and compared with Caucasian estimates.

Results Absolute allele frequency differences between Māori and Caucasian groups ranged from 1% to 16% for the polymorphisms tested.

Conclusions Functional allele frequencies of drug metabolising genes differed between Māori and European groups warranting larger general population surveys. These findings may also bear thinking about when conducting pharmacogenetic studies or clinical trials in New Zealand cohorts because patients with Māori ancestry may respond differently to certain medicines based on genotype.

The cytochrome P450 (CYP) enzymes play a central role in the metabolism of commonly prescribed drugs such as antidepressants, beta blockers, antipsychotics, and nicotine replacement therapy (NRT). Many of the CYP genes are known to contain polymorphisms that are associated with variation in drug response among individuals. Allele frequencies of some of these polymorphisms are known to vary considerably across different racial groups, and this may be important for understanding variation in drug response among patients with different ancestral backgrounds.

The Māori population of New Zealand (NZ) represents the final link in a long chain of island-hopping voyages stretching across the South Pacific Ocean. It is believed this population originated from small groups of common ancestors ~1000 years ago and, within the geographic isolation of NZ, underwent rapid growth until the arrival of European colonisers in the 1800s.

Considering this unusual genetic history it is reasonable to expect that the Māori population developed substantially different allele frequencies compared to other human populations. The intermarriage with Europeans over the last 8–10 generations has further modified the genomic structure of the Māori population, such that an estimated 40% of the modern Māori gene pool consists of Caucasian genes.1

Little research has been conducted on pharmacokinetics or pharmacogenetics of drugs in the Māori population. In 1995, Wanwimolruk et al, phenotyped CYP2D6 and CYP2C19 in a sample of Māori using debrisoquine and proguanil as substrate drugs, respectively. These researchers found that the prevalence of poor metabolisers (PMs)
for debrisoquine was not higher in Māori compared to Caucasians. However the frequency of the PM phenotype for proguanil was increased in Māori.\textsuperscript{2}

Recently, we assessed nicotine metabolism via the \textit{CYP2A6} enzyme in a sample of smokers and found evidence that Māori metabolise nicotine at a slower rate than Caucasians.\textsuperscript{3} These findings suggest that genetic variants, influencing \textit{CYP2C19} and \textit{CYP2A6} activity, may play a role in predicting relevant drug response in Māori.

Here we have determined the prevalence of functionally relevant alleles in the \textit{CYP2C9}, \textit{CYP2C19}, \textit{CYP2D6}, and \textit{CYP2A6} genes in a sample of Māori volunteers and compared these frequencies to those previously reported for Caucasians. We also discuss the findings in terms of possible clinical relevance to Māori as well as pharmacogenetic association studies in this admixed population.

\textbf{Methods}

The sample (n=60) was selected from a pre-existing bank of DNA housed at Victoria University of Wellington. DNA samples were originally collected through the Blood Transfusion Service in Wellington.

Participants were unrelated by first-degree and classified as “Māori” by self-report using:

- The 2001 census definition for ethnicity, and
- An ancestral definition—i.e. having 8 Māori great grandparents.

As such, this sample might be considered more representative of the ancestral (non-admixed) Māori population than the modern day general (and admixed) Māori population. Ethics approval was granted by the Wellington Ethics Committee in 2004.

We tested \textit{CYP2A6} variants that have been previously associated with slow nicotine metabolism and that have an allele frequency of greater than 2\% in Caucasians (i.e. \textit{CYP2A6*4}, \textit{CYP2A6*7}, and \textit{CYP2A6*9}). Genotyping of \textit{CYP2A6} variants was performed as described in Lea et al, 2005. We also genotyped the commonly studied variants in \textit{CYP2C9} and \textit{CYP2C19}, \textit{CYP2D6} genes according to the methods described in Sullivan-Klose et al, 1996; Roberts et al, 2006; Goldstein et al, 1996.\textsuperscript{4–6} P-values were determined using Fisher’s Exact Tests by comparing data from the Māori sample to previously published Caucasian data.

\textbf{Results}

Table 1 shows the CYP allele frequencies observed for the Māori sample as well as absolute frequency differences compared to previously published estimates in Caucasian samples (see references in Table 1). Across all variants tested the absolute difference values ranged from less than 1\% to 16\%. The largest differences were observed for \textit{CYP2C9*2}, \textit{CYP2D6*4}, and \textit{CYP2A6*9} (>11\%). For the Māori sample, the PM alleles (i.e. *2 and *3) were less prevalent for \textit{CYP2A9} and more prevalent for \textit{CYP2C19} compared to Caucasian (p<0.05).

The distributions for \textit{CYP2D6} alleles were different between the groups due to lower frequency of *4 and *41 alleles and higher frequencies of *10 alleles in Māori (p<0.05). For \textit{CYP2A6} variants the slow metabolising alleles (*4 and *9) were more prevalent in Māori compared to Caucasian (p<0.001).

\textbf{Discussion}

This study determined allele frequencies for functionally relevant \textit{CYP} gene variants in a sample of Māori selected to be fairly representative of the non-admixed Māori population. The rationale for the research is based on the premise that the unique genetic history of Māori has significantly modified the allele frequencies at these loci, particularly compared to Caucasian, and that this may partially explain variation in drug response of this indigenous population.
Our results showed that substantial differences exist for alleles of CYP2C9, CYP2C19, and CYP2D6 polymorphisms between Māori and Caucasian groups. The increased prevalence of PM alleles for CYP2C19 in Māori is consistent with the phenotypic results of Wanwimolruk et al (1995).² These findings may ultimately have implications for clinicians prescribing commonly used drugs metabolised via these enzymes such as fluoxetine and warfarin. For example, if a patient with Māori ancestry has a different likelihood of possessing variant CYP450 alleles this might alter their risk of adverse events or otherwise influence successful treatment outcomes.

We also found evidence for a higher frequency of CYP2A6 alleles (*4 and *9) in the Māori sample compared to Caucasian. These alleles have been previously associated with slower acting CYP2A6 and nicotine clearance in smokers: ~80% of nicotine is metabolised via CYP2A6. There is also evidence that slow-acting CYP2A6 may modify response to nicotine patch therapy and likelihood of smoking cessation.¹¹ Therefore, knowledge of increased frequency of the slow-acting CYP2A6 alleles in Māori might benefit the smoking-cessation programmes and clinicians when screening smokers for likely success using standard dose nicotine replacement patches.

It is important to note that due to the fact that the Māori sample studied here was selected to possess as little non-Māori ancestry as possible our allele frequencies should not be interpreted to be estimates of the general Māori population. Studies of much larger random Māori samples are required before evidence-based decisions can be made about these genetic markers in terms of clinical practice.

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**Table 1. Frequencies of variant cytochrome P450 alleles in Māori and Caucasian samples**

<table>
<thead>
<tr>
<th>Gene</th>
<th>Variant</th>
<th>Common Drugs Metabolised</th>
<th>Allele Frequency (%)</th>
<th>Difference</th>
<th>P-value*</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>CYP2C9</td>
<td>*2</td>
<td>NSAIDs, Angiotensin II</td>
<td>1.7  / 11</td>
<td>9.3</td>
<td>0.000</td>
<td>Suarez-Kurtz, 2005¹</td>
</tr>
<tr>
<td></td>
<td>*3</td>
<td>Blockers, Sulfonylates</td>
<td>0.8  / 7</td>
<td>6.2</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>CYP2C19</td>
<td>*2</td>
<td>Proton pump Inhibitors,</td>
<td>24.0  / 15</td>
<td>9</td>
<td>0.001</td>
<td>Xie et al, 1999²</td>
</tr>
<tr>
<td></td>
<td>*3</td>
<td>Anti-epileptics</td>
<td>1.7  / 0.4</td>
<td>1.3</td>
<td>0.012</td>
<td></td>
</tr>
<tr>
<td>CYP2D6</td>
<td>*5</td>
<td>Antidepressants,</td>
<td>0.9  / 1</td>
<td>0.1</td>
<td>ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*4</td>
<td>Antidepressants,</td>
<td>7.9  / 19.5</td>
<td>11.6</td>
<td>ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*5</td>
<td>Antidepressants,</td>
<td>1.8  / 4.1</td>
<td>2.3</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*6</td>
<td>Antipsychotics,</td>
<td>0.0</td>
<td>ns</td>
<td>ns</td>
<td>Griese et al, 2001³</td>
</tr>
<tr>
<td></td>
<td>*9</td>
<td>Beta blockers</td>
<td>0.0</td>
<td>ns</td>
<td>ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*10</td>
<td>Beta blockers</td>
<td>6.1  / 2</td>
<td>4.1</td>
<td>0.024</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*41</td>
<td>Beta blockers</td>
<td>3.5  / 20</td>
<td>14.5</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>CYP2A6</td>
<td>*4</td>
<td>Nicotine</td>
<td>9.5  / 1.2</td>
<td>8.4</td>
<td>0.000</td>
<td>Schoedel et al, 2004⁴</td>
</tr>
<tr>
<td></td>
<td>*7</td>
<td>Nicotine</td>
<td>1.1  / 0.3</td>
<td>0.8</td>
<td>ns</td>
<td></td>
</tr>
<tr>
<td></td>
<td>*9</td>
<td>Nicotine</td>
<td>19.0  / 7.1</td>
<td>11.9</td>
<td>0.000</td>
<td></td>
</tr>
</tbody>
</table>

*P values determined by comparing Māori allele frequencies to published Caucasian frequencies using Fisher's Exact Test; #Values are the lower of the range published for European (i.e. British, Italian, Spanish, and Swedish).
The present day Māori population exhibits significant genetic admixture largely as a result of intermarriage with Caucasians of European origin. This unique genetic structure presents both problems and advantages for pharmacogenetics studies involving this population. When conducting genetic association studies or clinical trials of drug response in NZ care should be taken to control for ancestry-specific genetic variation within the study cohort to avoid false or misleading results.

Depending on the study design this may be achieved by genotyping relevant functional polymorphisms such as the CYP markers investigated here (e.g. clinical trial) and/or by using genomic control markers—i.e. DNA markers that can estimate degree of Māori/European ancestry (e.g. genetic association studies of cases and controls).

If DNA is not available or difficult to obtain, as is the case for many clinical trials or epidemiological studies, we suggest self reported ancestry of the patient based on grandparental information could be used as a proxy for estimating and controlling for variation in genomic ancestry.

Recently admixed populations such as Māori also make it possible to map genes associated with differential drug response or disease susceptibility using a method known as mapping by admixture linkage disequilibrium (MALD). This method exploits the new allelic associations that are formed among adjacent polymorphisms when two genetically distinct populations mix and can identify ancestry-specific alleles that may contribute to ethnic differences in heritable traits like drug response.

In conclusion, this study is the first to report frequencies of functionally important CYP alleles in a sample of Māori and has shown that potentially important differences exist when compared to Caucasians. These data provide a compelling rationale for conducting further large-scale pharmacogenetic research involving Māori.

Competing interests: None known.

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References


Surveillance of vaccine breakthrough cases following MeNZB vaccination

Anne McNicholas, Yvonne Galloway, Diana Martin, Kerry Sexton, Jane O’Hallahan

Abstract

Aim To describe and investigate epidemic strain serogroup B meningococcal disease in recipients of the meningococcal vaccine, MeNZB.

Method Epidemic strain meningococcal disease cases in vaccine recipients were identified by matching disease notification and laboratory data against the National Immunisation Register. Descriptive analyses were undertaken for disease cases aged under 20 years and vaccine breakthrough cases (epidemic strain meningococcal disease cases with onset 28 or more days after receipt of the third MeNZB dose). Questionnaires were sent to hospital clinicians requesting medical histories and laboratory test results for vaccine breakthrough cases. A committee reviewed this information to assess immune competence in these cases.

Results From the start of the meningococcal B immunisation programme in July 2004 to the end of 2006, 34 vaccine breakthrough cases were identified. No underlying host factors were identified that explained disease occurrence for the 30 cases (88.2%) for whom questionnaires were completed. For 12 (35.3%) cases all requested laboratory tests to assess immune competence were performed and these subjects were judged to be immune competent.

Conclusion While epidemic strain meningococcal disease incidence has fallen dramatically with the introduction of the vaccine, these early results confirm the expectation that vaccine breakthrough cases will occur in immune competent individuals given the anticipated vaccine effectiveness of approximately 75%.

Following successful age-group trials\(^1,2\) demonstrating satisfactory safety and immunogenicity, a new group B meningococcal vaccine, MeNZB was introduced in New Zealand (NZ) to combat a meningococcal disease epidemic caused by serogroup B Neisseria meningitidis expressing the PorA P1.7-2, 4 protein. It was anticipated that the vaccine would have an effectiveness of approximately 75%.

The meningococcal B immunisation programme (hereafter termed ‘the Programme’) was gradually implemented from July 2004 with a three-dose vaccination series recommended for those aged 6 weeks to 19 years.\(^3\) Up to the end of 2006, the vaccine remained available for this age group. Since then it has remained available to children aged under 5 years. In January 2006, a fourth dose was introduced for infants who had started their vaccination series before the age of 6 months.\(^4\)

Based on data from the National Immunisation Register (NIR), approximately 1.02 million individuals received three or more doses of MeNZB up to the end of December 2006, with overall vaccine coverage for three or more doses of 82.3% for
all aged under 20 years: 84.8% for children aged 1 to 4 years and 86.8% for those aged 5 to 17 years.

MeNZB was specifically developed for epidemic control in NZ and, similar to other meningococcal vaccines, its licensure was approved without direct evidence of efficacy from Phase III trials. Therefore, the incidence of epidemic strain disease in vaccine recipients was closely monitored. Individuals with disease onset 28 or more days following receipt of the third vaccine dose (regardless of whether a fourth dose was received) were assessed for underlying factors such as compromised immune status.

The overall purpose of these surveillance activities was to measure the incidence and distribution of vaccine breakthrough cases, and (to the extent possible) identify factor(s) underlying the occurrence of epidemic strain-specific disease in fully vaccinated children.

Methods

Meningococcal disease is notifiable to Medical Officers of Health under the Health Act 1956, with case details recorded on a computerised database (EpiSurv) and managed nationally by the Institute of Environmental Science and Research (ESR). Patient specimens and meningococci or meningococcal DNA from disease cases are referred to ESR’s Meningococcus Reference Laboratory for confirmation and strain characterisation. Laboratory results were combined with the EpiSurv data and used to identify cases of meningococcal disease that were due to the epidemic strain.

Epidemic strain meningococcal disease incidence for the 2 years prior to the implementation of the Programme (2002 and 2003) was compared with the incidence for the 2 years post implementation (2005 and 2006). Data for 2004 were excluded because both prior and post periods occurred within that 1 year. Age-specific incidence rates for all epidemic strain meningococcal disease cases aged under 20 years at the time of onset were calculated for the combined 2-year periods 2002–2003 and 2005–2006 using census population estimates at the mid-point of each of the relevant years. Because age-ethnic-specific population estimates were unavailable for 2002 to 2006, these were extrapolated from 2001 census data and used to calculate ethnic-specific incidence rates of epidemic strain meningococcal disease for those aged under 20 years. Age-specific and ethnic-specific rates were similarly calculated for vaccine breakthrough cases occurring from July 2004 to December 2006. Prioritised ethnicity was used whereby each case was allocated to a single ethnicity, with the order of prioritisation Māori, Pacific people, and European/other. Annual incidence rates were compared using Fisher’s exact test.

The NIR holds details for MeNZB immunisations given to those aged 0–20 years. The majority of MeNZB vaccination records were electronically transferred to the NIR from general practice patient management systems and school-based vaccination systems maintained by public health units. Some data were entered manually from paper-based records including vaccinations administered through outreach services and also the NZ clinical trials; the latter being recorded retrospectively.

Vaccination information for meningococcal disease cases was verified by public health unit staff when they contacted the case/case’s family at the time of disease notification. Using the National Health Index (NHI) number, which is a unique identifier, the Ministry of Health’s Meningococcal Vaccine Strategy (MVS) Data Management Group matched EpiSurv data each week with an extract from the NIR to identify epidemic strain meningococcal disease cases who had received any doses of MeNZB.

A vaccine breakthrough was defined as a notified case of meningococcal disease that was laboratory-confirmed as group B with the P1.7-2,4 PorA and with symptom onset 28 or more days after the third dose of MeNZB. This time period was based on the minimum timing at which post-vaccination specimens were taken for measuring bactericidal antibody levels during the clinical trials. The Data Management Group sent a questionnaire to the responsible hospital clinician asking about factors that would indicate compromised immune competence, such as administration of immunosuppressive medication or presence of hyposplenism, and also about disease sequelae. Clinicians were requested to arrange laboratory tests for immunoglobulin levels (Total IgG, IgM, IgA); human immunodeficiency virus (HIV) status; complement deficiency (CH50 or classical pathway
activity); and to send acute and convalescent sera to ESR for meningococcal serology. ESR undertook enzyme linked immunosorbent assays (ELISA) and serum bactericidal assays to determine the presence of an epidemic strain-specific serum bactericidal antibody (SBAb) response. A committee independent of the Ministry of Health (with expertise in paediatrics, paediatric immunology, and infectious disease) reviewed the results to assess whether there were underlying host factors that explained disease occurrence despite completion of the vaccination course.

The Data Management Group reviewed vaccine batch numbers and administration dates for breakthrough cases to identify obvious overlaps. The vaccine distribution company was required to advise the MVS team of any breaches of the cold chain or complaints regarding vaccine batches. In addition, general practice and public health nurses advised the MVS team of any cold chain breaches that occurred at the practice or school level.

The Multi-Region Ethics Committee reviewed the proposal for investigation of meningococcal disease in MeNZB vaccine recipients and considered that it did not require approval per se as it was surveillance appropriate to the Programme rather than research.

**Results**

**Epidemic strain meningococcal disease in those aged under 20 years**—Between 2002 and 2006 there was a significant decrease (p<0.001) in the incidence of epidemic strain meningococcal disease. There were 218 (18.6 per 100,000) cases aged under 20 years reported in 2002 compared with 47 cases (3.9 per 100,000) in 2006.

During the 2002–03 and 2005–06 periods, the highest disease rates occurred in those aged under 1 year followed by those aged 1 to 4 years. Following introduction of MeNZB, rates declined in all age groups, however the rate in those aged under 1 year for the 2005/06 period remained relatively high at 22.3 per 100,000 (Table 1).

**Table 1. Incidence of epidemic strain meningococcal disease and vaccine breakthrough cases by age group, 2002/03 and 2005/06**

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>1 January 2002 to 31 December 2003</th>
<th>1 January 2005 to 31 December 2006</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All cases</td>
<td>All cases</td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>Rate*</td>
</tr>
<tr>
<td>&lt; 1</td>
<td>72</td>
<td>65.0</td>
</tr>
<tr>
<td>1–4</td>
<td>123</td>
<td>27.2</td>
</tr>
<tr>
<td>5–9</td>
<td>62</td>
<td>10.5</td>
</tr>
<tr>
<td>10–14</td>
<td>49</td>
<td>7.9</td>
</tr>
<tr>
<td>15–19</td>
<td>101</td>
<td>17.4</td>
</tr>
<tr>
<td>Total</td>
<td>407</td>
<td>17.3</td>
</tr>
</tbody>
</table>

*Age specific rate per 100,000 using Statistics New Zealand population estimates as the denominator.

During both periods the highest disease rates by ethnic group in those aged under 20 years were found in Pacific people, followed by Māori and European/other (Table 2). Of the 407 cases that occurred during 2002/03, eight (2.0%) deaths were reported; of the 129 cases for the 2005/06 period, three (2.3%) deaths were reported.

**Epidemic strain meningococcal disease in vaccine recipients**—From the Programme start in July 2004 to December 2006, 72 individuals developed epidemic strain disease after receiving any dose of MeNZB: 24 of these individuals had completed only one MeNZB dose; 13 individuals had completed two doses; and 35
had completed three or more doses. Of these 35 cases that developed disease after the receipt of the third dose, 34 met the case-definition for a vaccine breakthrough. The remaining case developed disease 26 days after receipt of the third dose. Of the five vaccine breakthrough cases in infants aged under 1 year, two were overdue for their fourth MeNZB dose at the time of disease onset. For one of the 34 cases classified as a vaccine breakthrough, the strain identified was a variant of the epidemic strain as it was PorA deficient, although otherwise identical to the epidemic strain by clonal type, capsule type, PorB, and FetA genotypes. A conservative approach to assessment of vaccine effectiveness was taken and the case was categorised as a vaccine breakthrough case.

The highest rate of vaccine breakthrough cases was in those aged under 1 year with a rate of 4.3 per 100,000 (Table 1). The highest rate by ethnicity occurred in Pacific people at 4.0 per 100,000 (Table 2). Of the 34 breakthrough cases, one (2.9%) death was reported.

**Table 2. Incidence of epidemic strain meningococcal disease and vaccine breakthrough cases in those aged under 20 years, by ethnic group, 2002/03 and 2005/06**

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>1 January 2002 to 31 December 2003</th>
<th>1 January 2005 to 31 December 2006</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All cases</td>
<td>All cases</td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>Rate*</td>
</tr>
<tr>
<td>Pacific people</td>
<td>68</td>
<td>35.6</td>
</tr>
<tr>
<td>Māori</td>
<td>149</td>
<td>28.2</td>
</tr>
<tr>
<td>European/other</td>
<td>190</td>
<td>11.3</td>
</tr>
<tr>
<td>Total</td>
<td>407</td>
<td>17.1</td>
</tr>
</tbody>
</table>

*Age specific rate per 100,000 using 2001 census population extrapolations as the denominator

The time interval from receipt of the third dose to the onset of epidemic strain disease ranged from 49 days to 27.1 months, with a median of 9.7 months (Table 3). The shortest interval was in a child aged 6.5 months and the longest interval was in a teenager who was vaccinated during Phase II clinical trials undertaken in 2002/03.
Table 3. Vaccine breakthrough cases by age and interval (in months) from receipt of the last MeNZB vaccine dose to disease onset date, for the period ending 31 December 2006

<table>
<thead>
<tr>
<th>Age group at disease onset (years)</th>
<th>Time in months (m) from receipt of the last MeNZB dose to disease onset</th>
<th>&lt; 6 m</th>
<th>6–11 m</th>
<th>12–17 m</th>
<th>18–23 m</th>
<th>24–29 m</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1</td>
<td></td>
<td>4</td>
<td>1</td>
<td>n/a*</td>
<td>n/a*</td>
<td>n/a*</td>
<td>5</td>
</tr>
<tr>
<td>1–4</td>
<td></td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>3</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>5–9</td>
<td></td>
<td>0</td>
<td>3</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>10–14</td>
<td></td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>15–19</td>
<td></td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>8</td>
<td>14</td>
<td>8</td>
<td>3</td>
<td>1</td>
<td>34</td>
</tr>
</tbody>
</table>

*Not applicable as the maximum period an infant aged under 1 year can be vaccinated with three doses before they move into the next age group is 7 months

Completed questionnaires were returned for 30 (88.2%) of the 34 vaccine breakthrough cases. None of the 30 cases was reported to have a chromosomal, other genetic, or congenital disorder; a known malignancy or to be receiving immunosuppressive medication; a known immunoglobulin or complement pathway deficiency or other immunoglobulin condition; or a history of frequent infections. For 22 of the 30 (73.3%) cases the child’s attending clinician indicated that the case did not have hyposplenism; and for the remaining eight (26.6%) cases, the presence of hyposplenism was reported as unknown.

Information was available for 31 of 34 (91.2%) cases regarding length of hospital stay due to meningococcal disease, with a median stay of 4 days (range 2–25 days) reported. Of these 31 cases, 12 (38.7%) were admitted to intensive care.

Five (16.6%) of the 30 vaccine breakthrough cases with completed questionnaires were reported to have disease sequelae, with one report of each of the following: possible epilepsy; conductive hearing loss; abnormal audiometry results (but with concurrent ear infection); transient behavioural regression with nocturnal enuresis; and residual left-sided limp. For a further four cases, audiometry tests were awaited; and for three cases, the respondents recorded “unknown” to at least one of the following sequelae: deafness, epilepsy, neurological deficit, or behaviour changes.

Of the 34 breakthrough cases, 12 (35.3%) had results available for the full range of tests requested to assess immune competence—i.e. total IgG, IgM and IgA, HIV serology and CH50 or classical pathway activity levels. For all 12 cases the results were within normal ranges and the HIV serology results were negative, and the Committee concluded that the cases were immune-competent (although it was noted that for two cases the presence of hyposplenism was recorded as unknown).

Eight (23.5%) of the 34 breakthrough cases had partial laboratory results for assessing immune competence (including one case for whom a questionnaire was not completed); six (17.6%) of the 34 cases had results available for only total IgG, IgM, IgA, and HIV serology; one (2.9%) for only total IgG, IgM, IgA, and CH50 or classical pathway activity levels; and one (2.9%) for only total IgG, IgM, and IgA. The Committee concluded that the results indicated that the cases were immune-
competent, but the unlikely event of terminal complement deficiency or underlying HIV infection could not be excluded where these tests were not undertaken.

For the remaining 14 of the 34 cases (41.2%), laboratory test results to assess immune competence were not provided, and thus the committee was unable to make a final decision. However, epidemic strain-specific antibody testing was completed on acute and convalescent sera for three of these cases and convalescent serum for one case. The Committee noted that the serum antibody testing results for these four cases indicated an ability to mount an IgG response.

ESR received both acute and convalescent serum samples for epidemic strain-specific antibody testing for 15 of the 34 (41.1%) breakthrough cases (including one for whom a questionnaire was not completed and the three cases mentioned above). Convalescent serum only was received for a further seven cases (20.6%), including the one case mentioned above. Eleven of the 15 acute serum specimens did not have elevated antibody-specific IgG ELISA levels. SBAb levels could not be measured for these 15 specimens because of the presence of antibiotics. Twenty-one of the 22 convalescent serum specimens had an elevated epidemic strain-specific SBAb antibody response (range log$_2$ 4.5–>10) and an elevated IgG ELISA (>80 Units) response. The one individual who did not show a convalescent SBAb response at a protective level (log$_2$ 3) or an elevated IgG ELISA level was considered by the Committee to be immune competent based on overall immunological investigations.

For the 30 (88.2%) vaccine breakthrough cases with information on their medical history, the Committee concluded that, based on the information available (and noting that immunological investigation results were not available for a number), it was reasonably confident that the cases were not due to pre-existing host factors. For the remaining four (11.8%) cases, the Committee was unable to draw conclusions regarding the factors that led to disease. One additional case that occurred post receipt of dose three, but with disease onset at 26 days post vaccination, was also reviewed and judged to have no pre-existing host factors.

The Data Management Group found no evidence implicating particular batch numbers or vaccination dates for breakthrough cases. No programme errors such as cold chain errors were identified through the vaccine distribution database, or through individual health providers, that could explain any of the breakthrough cases.

Discussion

When any new vaccine is introduced, it is important to establish vaccine-effectiveness through monitoring disease trends and vaccine breakthrough cases in addition to undertaking formal analyses to estimate vaccine efficacy and/or effectiveness. Since NZ is the only country using the MeNZB vaccine, moreover without direct evidence of efficacy, such results are essential to inform policies regarding ongoing use of the vaccine in NZ as well as use of similar vaccines in other countries.

Based on experience with other group B meningococcal vaccines, a number of vaccine breakthrough cases were expected. A prospective observational study for the period ending June 2006, 2 years after the Programme started, estimated the MeNZB vaccine effectiveness to be 73%.
It is possible some vaccine breakthrough cases may not have been detected, either because they were not laboratory confirmed as being due to the epidemic strain or because their vaccination details were incorrectly recorded. There were 13 children notified with meningococcal disease from July 2004 to December 2006 who had received three or more vaccine doses but their disease was not laboratory confirmed as due to the epidemic strain. Of these 13 cases, 10 had no laboratory confirmation at all and three were laboratory confirmed as meningococcal disease but the strain was not identified. An audit carried out 8 months after the Programme started found that approximately 95% of MeNZB vaccinations were recorded on the NIR and approximately 98% of fields were correct. In addition, public health staff were asked to confirm the vaccination status of cases when they contacted case families regarding prophylaxis.

The definition of a vaccine breakthrough (disease symptom onset 28 or more days after the third dose of MeNZB) was implemented at the start of the Programme before a decision was made to vaccinate infants with a fourth dose. While this approach of using a single definition means we have overestimated vaccine breakthrough cases in infants, it was agreed that this more conservative approach to monitoring the effect of MeNZB on disease was preferable. A similarly conservative approach meant we included the one breakthrough case where the B strain was PorA-deficient even though antibody generated by the MeNZB vaccine could not be protective as antibodies target the PorA protein.

While the expert committee that reviewed the available information on vaccine breakthrough cases concluded that there was no evidence of pre-existing host factors to explain epidemic strain meningococcal disease in fully vaccinated individuals, it noted that complete results (particularly immunological investigations) were not available for a substantial proportion. Therefore, the unlikely event of factors such as HIV infection or terminal complement deficiency cannot always be excluded. Susceptibility to meningococcal disease has been reported in almost every form of complement deficiency, particularly those of the late complement components. However, in some instances, only C3 and C4 measurements were undertaken, whereas methods such as haemolytic titration of the classical pathway by CH50 procedures or ELISA are required to detect a wider range of complement deficiencies.

Some immunological investigations were not completed because patients and/or caregivers did not attend follow-up appointments or were unwilling to undergo blood tests. However, some clinicians indicated that since an individual was clinically well, immunological tests were not necessary. This response was disappointing, and the Advisory Committee considered that good clinical practice would see full immunological assessments of antibody, splenic, and terminal complement function undertaken whenever invasive bacterial infections from vaccine preventable diseases occurred in fully vaccinated patients. In particular, it has been recommended that all patients with invasive meningococcal disease be screened for complement function as individuals with complement deficiency and their families may benefit from clinical advice and prophylactic treatment. These investigations are even more important where a child has developed disease despite vaccination.
The possibility of detecting systemic Programme errors is more difficult to evaluate, however these would seem improbable at the national level with, for example, the national vaccine distributor required to follow strict operating procedures, including cold chain monitoring. Individual errors are possible, however, and those at the provider level in particular less likely to be detected.

It is possible that disease severity may be reduced in individuals fully vaccinated with MeNZB. While information was collected on length of hospital stay, intensive care admission, and ongoing sequelae, investigation of this hypothesis is not possible with data from this surveillance system. Instead it would require a rigorous and systematic comparison of medical records from both vaccinated and unvaccinated children with meningococcal disease.

Over the same period that the vaccine breakthrough cases were observed, epidemic strain meningococcal disease incidence decreased dramatically. Rates of disease remain highest in those aged under 1 year and are likely explained by a number of factors. In particular, not only are children aged under 1 year historically at most risk of disease, but a fourth MeNZB dose is required in young infants to elicit a rise in bactericidal antibodies similar to the levels achieved with three doses in older infants and children. As this fourth dose is not administered until 10 months of age, there is a period when a proportion of infants may not have the same degree of protection as older children who have been vaccinated. Indeed, the importance of infants receiving their fourth vaccine dose was highlighted by the occurrence of two vaccine breakthrough cases overdue for their fourth dose at the time they developed meningococcal disease.

Given the vaccine’s reported effectiveness of 73%, vaccine breakthrough cases will continue to occur in immune-competent children. Further, epidemic strain breakthrough cases numbers may equal or exceed cases in unvaccinated individuals. This situation does not represent vaccine ineffectiveness, but is a reflection of the high vaccine coverage achieved. The Ministry of Health and ESR will continue to monitor vaccine breakthrough cases and meningococcal disease trends to inform policy decisions related to ongoing vaccine use. Given the monitoring results to date, the Ministry of Health’s collation of individual level clinical information and immunological measurements for expert committee assessment of immune competence has ceased for vaccine breakthrough cases that have occurred after 31 December 2006.

Funding: Funding for the surveillance was provided by the Ministry of Health, New Zealand. The Ministry of Health received funding from Chiron Vaccines (now Novartis) toward the cost of establishing the Data Management Group to manage and analyse data collected on MeNZB vaccine safety and effectiveness.

Competing interests: In the past, authors YG, DM, and JO received funding from the vaccine manufacturer Chiron Vaccines (now Novartis) to attend meetings and conferences. However Novartis took no part in the writing of this paper and did not review it.

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References:

Capitation funding of primary health organisations in New Zealand: are enrolled populations being funded according to need?

Jennifer Langton, Peter Crampton

Abstract

Aim To determine whether the three main funding formulas for Primary Health Organisations achieved a stated aim of the Primary Health Care Strategy to fund enrolled populations according to need.

Methods National data were obtained from the Ministry of Health for a 12-month period beginning in April 2004: these included demographic characteristics of the enrolled Primary Health Organisation population, plus rates tables for: First-Contact Services, Services to Improve Access, and Health Promotion. Funding for Access and Interim practices for four-quarters was calculated for each of these three funding streams. Analysis of the demographic characteristics of Access and Interim practices was undertaken.

Results Māori and Pacific peoples made up a greater proportion of the Access population than the Interim, had higher rates of deprivation than the non-Maori/non-Pacific population, and demonstrated a younger age distribution. The first quarter (April 2004–June 2004) showed there was preferential funding for Access PHOs and in particular high-needs groups. In quarter two, this level of preferential funding had diminished, coinciding with the introduction of increased government funding for all Interim enrollees aged 65 and over.

Conclusions The greater funding for Access enrollees was notably eroded with the introduction of Access-level funding for those aged 65+ in Interim PHOs. Since these data were analysed all remaining Interim age groups have shifted to Access-level funding, benefiting non-Māori /non-Pacific in Interim PHOs. The rapid shift to Access-level funding for First Contact Services has seen a continued erosion of the redistributive effect of the original needs-based formulas. A system cannot be considered equitable if some members of society are not realising their health potential, and financing of primary care should remain redistributive until such a time as this objective is attained.

The Primary Health Care Strategy was published in 2001. It articulated a vision for primary health care which the government believed would help to achieve the population health objectives identified in the New Zealand Health Strategy (2000).1 In particular, the government wanted to reduce health inequalities through the provision of primary health care services that are community centred and emphasise preventative and health promotion as well as curative services. A key component of the Primary Health Care Strategy was the development of Primary Health Organisations (PHOs).
Capitation funding formulas were developed as a ‘needs-based’ mechanism for delivering healthcare funds to PHOs in such a way that populations with the greatest need benefit most. The Primary Health Care Strategy described a primary health care system that is central to the whole health system and founded on the principles of the Alma Ata Declaration of 1978. The values inherent in this vision of primary health care include responsiveness, fairness, and comprehensive family and community-oriented services, including (but not limited to) restorative health care.

A needs-based method of funding PHOs is consistent with this vision, and is a necessary tool in an environment of escalating healthcare costs if real health gains for the most disadvantaged in New Zealand are to be realised.

Two different PHO types (Access and Interim) were developed so that initial increases in primary health care financing could be targeted to those groups in the population with greatest need. Access PHOs were those organisations that had an enrolled population with more than 50% identified as high need as determined by deprivation and ethnicity. All other PHOs were Interim.

The formulae for financing these organisations differentiated between the two PHO types so that Access PHOs saw their enrolees funded at a higher rate than their counterparts in Interim organisations. The funding mechanism is one of capitation, where the PHO is funded at a predetermined rate for each enrolee regardless of whether contact is made during the period.

Several different funding tools have been established to finance PHOs, however three key funding formulas—First Contact, Services to Improve Access (SIA), and Health Promotion (HP)—are the major contributors to public financing of primary health care.

The First Contact formula provided the bulk of PHO funding and was intended as the major means of purchasing primary health care for the population. It funded PHOs to provide first contact services such as GP or practice nurse consultations. The SIA formula financed PHOs to develop mechanisms for making their services more accessible to the enrolled population they serviced and HP was paid for health promotion activities carried out by PHOs. To receive SIA and HP funding, the PHO needed to demonstrate how this money was to be spent.

This paper examines the three main funding formulae to determine how well and by how much they preferentially targeted primary health care funding to high needs groups for the 12-month period April 2004 to March 2005.

Methods

The data supplied by the Ministry of Health consisted of a Microsoft Access database containing the quarterly-reported demographic variables for all PHO enrolees over the 12-month period. Data were reported quarterly and are referred to in this article as quarter 2 2004 (April–June 2004), quarter 3 2004 (July–September 2004), quarter 4 2004 (October–December 2004), and quarter 1 of 2005 (January to March 2005).

The demographic table was linked with the quarterly funding rates for each of the funding streams (First-Contact, Services to Improve Access, and Health Promotion) to determine the income generated for Access and Interim funded practices.

Data were analysed at the practice level in order to accurately gauge the impact of the needs-based formulas. This was necessary because a number of PHOs had evolved as “Mixed” organisations where
some practices within the PHO received Access funding for pockets of high-need. All analyses were carried out using Microsoft Access. Data were then transferred to Microsoft Excel for graphical presentation.

The outputs derived from the funding data were the total quarterly income for Access and Interim funded practices; quarterly per capita income for Maori, Pacific, and all other ethnicities combined; and the increase in per-capita income between quarter 2 2004 and quarter 3 2004 as a result of increased funding for ages 65+ in Interim PHOs. These outputs were examined for First Contact funding only and also combined First-Contact, Services to Improve Access, and Health Promotion.

Table 1 shows the demographic variables examined.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
<td>Analysed to the level of Māori, Pacific, and all other ethnicities (i.e. 3 ethnic categories examined)</td>
</tr>
<tr>
<td>Age</td>
<td>Age was analysed in 6 different funding categories: 1 = 0–4 years, 2 = 5–14 years, 3 = 15–24 years, 4 = 25–44 years, 5 = 45–64 years, 6 = 65+ years</td>
</tr>
<tr>
<td>Gender</td>
<td>The proportion of male and female enrolees for each ethnic group were analysed</td>
</tr>
<tr>
<td>Deprivation</td>
<td>The NZDep2001 scale was used to measure socioeconomic deprivation, deciles were combined to create quintiles—i.e. deciles 9 and 10 became NZDep 5, and deciles 1 to 8 became NZDep &lt;5. This was in keeping with the raw data as funding for deprivation was only given for quintile 5</td>
</tr>
<tr>
<td>Community Services Card (CSC)</td>
<td>The proportion of enrolees holding a CSC in both Access and Interim practices</td>
</tr>
<tr>
<td>High-User Health Card (HUHC)</td>
<td>The proportion of enrolees holding a HUHC in both Access and Interim practices</td>
</tr>
</tbody>
</table>

Results

Enrolled population—Most growth in PHO enrolment between April 2004 and March 2005 occurred in the Interim population and the majority of new enrolees were non-Maori, non-Pacific (i.e. other); see Figure 1. It should be noted that a significant minority of enrolees did not have ethnicity coded, particularly in Interim practices, although ethnicity recording improved throughout the year. For example, in quarter 2 2004, 5% of Access and 18% of Interim enrolees did not have ethnicity recorded compared with 4% of Access and 12% of Interim enrolees in quarter 1 2005.

Age distribution—Figure 2 demonstrates that the non-Maori/non-Pacific population in both Access and Interim funded practices had an older age profile when compared with the Māori and Pacific populations.

Deprivation—Māori and Pacific peoples exhibited higher levels of deprivation in both Access and Interim PHOs compared with non-Maori/non-Pacific enrolees.

Table 2 shows the percentage of the enrolled population residing in NZDep2001 quintile 5 (most deprived) for Access and Interim funded practices.
Figure 1. Enrolled population and ethnic distribution in Access and Interim funded practices*

Figure 2. Age distribution by funding age categories*

Table 2. Percentage of population residing in NZDep2001 quintile 5 between April 2004 and March 2005

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Access %</th>
<th>Interim %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Māori</td>
<td>55.75</td>
<td>27</td>
</tr>
<tr>
<td>Pacific</td>
<td>68.75</td>
<td>31</td>
</tr>
<tr>
<td>Non-Maori/non-Pacific</td>
<td>25</td>
<td>8</td>
</tr>
</tbody>
</table>

Gender—For all ethnicities there were slightly more female enrolees than male in both Access and Interim funded practices. Male enrolees made up 48% to 49% of the enrolled population.

First Contact funding—Between April and June of 2004, Access funded practices received a greater proportion of total funding from this stream compared with the proportion during the following two quarters (Figure 3).
The per-capita income each quarter (Table 3) shows that high-need groups did generate greater funding; although this comparative advantage was reduced at quarter 3 2004 when the Interim population, and in particular non-Maori/non-Pacific saw a marked increase in funding per person. The percentage increase observed between these two quarters is presented in Table 4.
Table 3. Quarterly income ($) per capita (First Contact services) for Interim and Access funded practices

<table>
<thead>
<tr>
<th>Variables</th>
<th>Q2 2004</th>
<th>Q3 2004</th>
<th>Q4 2004</th>
<th>Q1 2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total population-Access</td>
<td>28.37</td>
<td>28.86</td>
<td>28.82</td>
<td>28.72</td>
</tr>
<tr>
<td>Total population-Interim</td>
<td>16.02</td>
<td>19.14</td>
<td>19.21</td>
<td>19.08</td>
</tr>
<tr>
<td>Māori-Access</td>
<td>28.95</td>
<td>29.35</td>
<td>29.27</td>
<td>29.15</td>
</tr>
<tr>
<td>Māori-Interim</td>
<td>21.47</td>
<td>22.17</td>
<td>22.30</td>
<td>22.14</td>
</tr>
<tr>
<td>Pacific-Access</td>
<td>29.09</td>
<td>29.67</td>
<td>29.76</td>
<td>29.67</td>
</tr>
<tr>
<td>Pacific-Interim</td>
<td>20.07</td>
<td>21.36</td>
<td>21.65</td>
<td>21.57</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Interim</td>
<td>15.55</td>
<td>18.89</td>
<td>18.95</td>
<td>18.80</td>
</tr>
</tbody>
</table>


Table 4. Increase in per capita First Contact funding between quarter two and quarter three 2004

<table>
<thead>
<tr>
<th>Variables</th>
<th>Increase ($) per capita income</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Population-Interim</td>
<td>3.12</td>
<td>19.48</td>
</tr>
<tr>
<td>Total Population-Access</td>
<td>0.49</td>
<td>1.73</td>
</tr>
<tr>
<td>Māori-Interim</td>
<td>0.70</td>
<td>3.25</td>
</tr>
<tr>
<td>Māori-Access</td>
<td>0.40</td>
<td>1.40</td>
</tr>
<tr>
<td>Pacific-Interim</td>
<td>1.29</td>
<td>6.43</td>
</tr>
<tr>
<td>Pacific-Access</td>
<td>0.58</td>
<td>1.99</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Interim</td>
<td>3.33</td>
<td>21.42</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Access</td>
<td>0.51</td>
<td>1.82</td>
</tr>
</tbody>
</table>

Services to Improve Access (SIA) and Health Promotion (HP)—Both these funding streams carried a weighting for high-need as measured by ethnicity and deprivation. The data for these funding streams are not presented separately but their effect can be observed when combined with First Contact income.

Combined Services to Improve Access, Health Promotion and First Contact funding—Figure 4 shows the combined income for Access and Interim PHOs for quarter 2 2004 was very similar. There was a smaller difference between the two than when comparing First Contact funding alone (Figure 3). A substantial increase in funding for Interim PHOs in quarter three occurred, followed by only a slight rise in the following two quarters.

SIA and HP contributed noticeably to funding for high-need groups, however it is also apparent that in quarter 3 2004 the increase in income for the Interim population was still sizeable (Table 5). Table 6 highlights the increase in per capita income between quarter 2 2004 and quarter 3 2004.
Figure 4. Combined First Contact, Services to Improve Access and Health Promotion funding for Access and Interim PHOs between April 2004 and March 2005*


Table 5. Combined First Contact, SIA, and HP per capita quarterly income ($)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Q2 2004</th>
<th>Q3 2004</th>
<th>Q4 2004</th>
<th>Q1 2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total population-Access</td>
<td>33.78</td>
<td>34.33</td>
<td>34.29</td>
<td>34.14</td>
</tr>
<tr>
<td>Total Population-Interim</td>
<td>17.52</td>
<td>20.67</td>
<td>20.73</td>
<td>20.62</td>
</tr>
<tr>
<td>Māori-Access</td>
<td>38.36</td>
<td>38.91</td>
<td>38.83</td>
<td>38.67</td>
</tr>
<tr>
<td>Māori-Interim</td>
<td>29.33</td>
<td>30.15</td>
<td>30.24</td>
<td>30.10</td>
</tr>
<tr>
<td>Pacific-Access</td>
<td>39.32</td>
<td>40.05</td>
<td>40.16</td>
<td>40.06</td>
</tr>
<tr>
<td>Pacific-Interim</td>
<td>28.19</td>
<td>29.60</td>
<td>29.89</td>
<td>29.79</td>
</tr>
<tr>
<td>Non-Māori/non-Pacific-Access</td>
<td>29.70</td>
<td>30.24</td>
<td>30.19</td>
<td>30.10</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Interim</td>
<td>16.47</td>
<td>19.82</td>
<td>19.88</td>
<td>19.72</td>
</tr>
</tbody>
</table>
Table 6. Increase in quarterly per capita income for combined First Contact, SIA, and HP funding between quarter 2 2004 and quarter 3 2004

<table>
<thead>
<tr>
<th>Variables</th>
<th>Increase ($) per capita income</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Population-Interim</td>
<td>3.15</td>
<td>17.97</td>
</tr>
<tr>
<td>Total Population-Access</td>
<td>0.55</td>
<td>1.63</td>
</tr>
<tr>
<td>Māori-Interim</td>
<td>0.81</td>
<td>2.77</td>
</tr>
<tr>
<td>Māori-Access</td>
<td>0.55</td>
<td>1.43</td>
</tr>
<tr>
<td>Pacific-Interim</td>
<td>1.41</td>
<td>5.00</td>
</tr>
<tr>
<td>Pacific-Access</td>
<td>0.73</td>
<td>1.86</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Interim</td>
<td>3.35</td>
<td>20.33</td>
</tr>
<tr>
<td>Non Māori/non-Pacific-Access</td>
<td>0.54</td>
<td>1.83</td>
</tr>
</tbody>
</table>

Over the 12 months examined, the Access population fell by almost five percentage points (from 33.6% to 28.7% of the total population) while the proportion of the total income generated fell by almost twice that (from 49.4% of total funding to 40%). The shift to Access-level funding for enrollees 65 and over in Interim practices in July 2004 is the likely explanation for this.

Discussion

Age is the primary demographic variable influencing funding. Although age distribution varies across ethnic groups, the First Contact formulas cost weights do not reflect the premature morbidity and mortality experience of Māori. Age distributions differ little between Access and Interim funded practices.

Figure 2 demonstrates that for Māori and Pacific peoples the population is young with a very small percentage aged 65 years and over. In comparison, non-Māori/non-Pacific people are older with a significant proportion aged 65 years and over and a much smaller percentage of the population in the two youngest age groups. In 2001, the median age for Māori was 22 and for Pacific it was 21, this is compared to a median age for the total New Zealand population of 35.8–10

The PHO funding formulas for First Contact and Services to Improve Access allocated different sums of money to enrollees’ depending on age. The original First Contact formula also preferentially funded Access enrollees over their Interim counterparts.

As time progressed this preferential funding was eroded as more age groups in Interim practices shifted to Access-level funding. The first shift occurred prior to the data analysed here and increased First Contact funding for all children and young people up to the age of 18. The second change saw all people 65 and over in Interim practices allocated increased funding (in July 2004) and is captured in the data analysed here.

Since then, funding has increased at yearly intervals for all other age groups so that by July 2007 all enrollees were funded at the Access rate for First Contact services. This has resulted in less redistribution of total primary health care funding to high-need groups (as defined by ethnicity and deprivation). This occurred while the inequities in health outcomes for these groups are yet to be addressed.
The reason the difference in per capita income between Access and Interim reduced in July 2004 was the increase in First Contact funding for those aged 65 and over enrolled in Interim PHOs. Interim organisations have a much smaller number of Māori and Pacific peoples than other ethnicities and a greater proportion of non-Māori/non-Pacific are people aged 65 and over. This means non-Māori/non-Pacific in Interim PHOs benefited most from this funding change (as indicated by the greater increase in their per-capita income).

The combined per-capita income in Access PHOs was appreciably higher than First Contact Services alone in all four quarters, by over $5. This differential was even more marked for Māori and Pacific peoples (approximately $10.00). In Interim PHOs the per-capita income did not grow as strongly with an overall increase of about $1.50. When the data are looked at by ethnicity however, there is a much more discernible rise for Māori and Pacific of around $8.00. This is primarily explained by the allocation of SIA funding for these populations.

SIA clearly redistributed money to high-need groups, which should serve as an incentive for providers to develop innovative services and for District Health Boards to require PHOs to have a clear plan for how SIA monies are to be used to increase access for high-need groups.5

The primary issue is that over the year observed in this study there was no growth in income generated per head of the Access population, whether examined as the total population or by ethnicity. At the same time, there was a strong rise for Interim enrollees.

As of July 2007, all people enrolled with a PHO were funded at the same rate according to their age for First Contact Services. While increased funding in these other age categories will benefit some high-need groups (such as Māori and Pacific enrolled in Interim PHOs), they make up a small percentage of the Interim population.

The most significant beneficiaries are non-Māori/non-Pacific in Interim PHOs who demonstrate low levels of deprivation (less than 10% reside in NZDep2001 quintile 5) and therefore have lower need from a population perspective. The rapid shift to Access-level funding for First Contact Services has seen a continued erosion of the redistributive effect of the original needs-based formula.

A significant point is that while SIA and HP do favour high-need groups, they are targeted funding streams, and need to be used for clearly defined purposes. Developing services that achieve greater access is a critical step toward improving the health status of high-need groups; SIA funding will help accomplish this but the money involved will be used for the actual service (e.g. staff salary, rental of premises, development of resources, etc) rather than directly for patient care. If access is improved and utilisation rates increase, health practitioners will need to be reimbursed at a level commensurate to the level of care demanded.

This research used an administrative dataset as its main source of data. As with all administrative datasets, issues related to data quality should be considered carefully. These data were collected from PHO reports made to HealthPAC as a requirement for payment. The accuracy of the data is therefore dependent upon consistent and complete recording of enrollee characteristics by all PHOs. It is also reliant on precise data inputting and is vulnerable to human error in transcription of information.
A limitation of this research lies in the fact that it has only examined the distribution of financing and is unable to provide answers regarding what happens with the money once it reaches PHOs. It is important to not only look at how funding for primary care is spread throughout the population but also the ways in which it is used to provide care in the community.

An evaluation of the Primary Health Care Strategy assessed the impact of funding changes on access to primary care services by measuring, over time, changes in user charges and changes in utilisation. However more research is needed in this area, particularly the associations between user charges and changes in utilisation, given that reducing financial barriers to access was a key objective of the Primary Health Care Strategy.

The Access and Interim capitation structure could be regarded as a crude tool for distribution of health dollars resulting in individuals benefiting to a greater or lesser degree depending on the areas in which they reside. More information is needed on the impact the Primary Health Care Strategy has made on individuals in Access and Interim areas.

Also of importance is the impact the shift to capitated funding for enrolled populations has had on GP income. Within the scope of this research this question was not addressed; no comparison was made between funding solely through the General Medical Subsidy and the PHO funding formulas. Further research is warranted to determine the extent to which GP incomes increased during this period.

It is imperative that New Zealand strives to eliminate the significant disparity in health outcomes for its populations to achieve equity in health for all. This may mean unequal access for unequal need, with funding allocated accordingly. Thus, fairness in financing does not necessarily mean universal low-cost care as a first priority but rather implies preferential funding for high-need communities, at least until significant reductions in inequality occur. With the rapid move toward universal low-cost access to primary health care it is possible that the original ambitions of the Primary Health Care Strategy are being diluted.

Further research is needed to evaluate the effects of the Primary Health Care Strategy on access to primary care for Maori, Pacific, and low-income groups as well as any effects on health status. A system cannot be considered equitable if some groups in society are not realising their health potential, and financing of primary care should remain redistributive until such a time as this objective is attained.

**Competing interests:** None known.

**Author information:** Peter Crampton, Dean and Head of Campus, University of Otago, Wellington; Jennifer Langton, Dispensary Manager, Pharmacy Department, Capital and Coast District Health Board, Wellington

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Correspondence: Professor Peter Crampton, Department of Public Health, University of Otago, PO Box 7343, Wellington, New Zealand. Fax: +64 (0)4 3895319; email: peter.crampton@otago.ac.nz

References:

Correlation of physician seniority with increased emergency department efficiency during a resident doctors’ strike

Martyn Harvey, Mustafa Al Shaar, Grant Cave, Muir Wallace, Paul Brydon

Abstract

Aim Physician seniority has increasingly been shown to correlate with improved clinical outcomes. However few studies examine the relationship between treating doctor experience and the efficiency of emergency care systems. We explored the hypothesis that increased seniority of emergency department (ED) medical staff would result in improved ED efficiency.

Method This was prospective observational study conducted at the ED of Waikato Hospital, a 650-bed university-affiliated teaching hospital. All patient presentations during a 5-day resident doctors’ strike when the ED was staffed by senior physicians, and the corresponding normally staffed days of the subsequent calendar week were examined. Patient waiting times, time seen to disposition, and total ED length of stay were recorded according to Australasian Triage Score (ATS).

Results 608 and 683 patient presentations were recorded during the strike and non-strike period respectively. Waiting times were reduced for ATS3 (43.8 vs 73.6 minutes, p<0.001) and ATS4 (53.7 vs 82.0 minutes, p<0.001) during the strike period. Time seen to disposition were reduced for ATS2 (147.9 vs 255.1 minutes, p=0.001) and ATS3 (119.9 vs 165.0 minutes, p<0.001) during the strike period. ED length of stay was reduced for ATS2 (162.6 vs 278.6 minutes, p<0.001), ATS3 (161.9 vs 238.4 minutes, p<0.001), and ATS4 (134.1 vs 179.2 minutes, p<0.001) during the strike period. No difference was observed in patient walkout, ED mortality, 48-hour mortality, or 30-day unscheduled representation rates.

Conclusions Increasing seniority of front line ED staff during a period of resident doctors’ strike action was associated with increased efficiency of ED patient processing. Early specialist involvement with ED patients may replicate these efficiencies during periods of normal departmental operation.

Emergency departments within Australasia and the United Kingdom are staffed predominately with resident medical doctors in training working under the supervision of a specialist consultant. Provision of verbal or bedside advice on patient management and disposition by emergency physicians and inpatient specialists occurs largely at the discretion of the attending resident doctor.

The ad-hoc nature of this consultation may result in inefficiency due to delays in diagnosis, initiation of appropriate management, and disposition decision.

Access block and emergency department (ED) overcrowding are well recognised impediments to emergency care systems on every continent.1–3 Attempting to de-clutter EDs and facilitate patient flow to disposition has become a reality for both clinicians and hospital managers.4,5
Physician experience has increasingly been shown to correlate with improved clinical outcomes in both emergent and non-emergent settings. Additionally, perceived doctor experience has been linked with improvements in patient satisfaction. Few studies however exist examining the relationship between treating doctor seniority and the efficiency of emergency care systems. Whilst it might seem intuitive that more experienced physicians respond to a given clinical scenario with more rapid and accurate decision-making as compared less experienced colleagues, data supporting this assumption is limited, and largely based on observational experiments. The potential to improve ED patient flow and minimise ED overcrowding through speeding decision-making processes requires evaluation.

An estimated 2500 junior medical doctors working in New Zealand District Health Board (DHB) hospitals launched strike action during the period 15/6/2006 to 19/6/2006 pursuing improved working conditions and remuneration. During this period, service delivery by all hospital departments was provided by consultant specialists, career medical officers, and non-striking junior doctors.

We were interested to explore the hypothesis that increasing seniority of front-line ED medical staff during the resident doctors’ strike would result in improvements in commonly measured markers of ED activity. Specifically, we intended comparing patient waiting times, time seen to disposition, ED length of stay, and utilisation of diagnostic investigations during the period of the resident doctors’ strike, with a corresponding interval of normal ED staffing.

Methods

Design—This was a prospective observational study comparing ED activity during the period of a resident doctors’ strike with a corresponding non-strike period of the same calendar month.

Study setting—Waikato Hospital is a 650-bed university-affiliated teaching hospital located in the city of Hamilton, New Zealand. The hospital provides tertiary-level adult and paediatric care and serves as the regional trauma centre for a local population of 190,000 and total regional catchment of 650,000. All major specialties are represented on campus. The ED has an annual census of 51,000 of which 20% are paediatric. Admission rate is approximately 35%.

The ED serves as the major admission portal for the hospital, with initial assessment of all self presenting and externally referred patients occurring within the facility. All patient presentations are initially triaged by a senior nurse according to the Australasian Triage Scale (ATS) as defined by the Australasian College for Emergency Medicine. This triage tool comprises five categories denoting the clinical urgency of presentation (1–5). Adherent to ATS categorisation is a maximum recommended time to initiation of medical assessment and treatment (ATS1 0 min, ATS2 10 min, ATS3 30 min, ATS4 60 min, ATS5 120 min), and institutional performance indicator thresholds representing the recommended percentage of patients seen within the stated target (ATS1 100%, ATS2 80%, ATS3 75%, ATS4 70%, ATS5 70%).

ED doctors, in conjunction with the appropriate inpatient service, initially manage patients of triage category 1 and 2 regardless of referral status. ED doctors additionally assess and manage all self-presenting patients of category 3, 4, and 5. Externally-referred patients (originating from general practitioners or referring hospitals) are managed by the designated specialty service. Usual ED staffing is by 9 consultant emergency physicians, 13 registrar-level (postgraduate year 4–10) doctors, and four senior house officer (SHO) level (postgraduate year 3) training emergency doctors. Standard ED staffing during the non-strike period provided a daily average of 111.2 clinical hours (total hours: consultant 216, registrar 323, SHO 75). Non-strike specialty medical staffing of the ED was via the hospital pool of specialty registrars and provided a daily average of 124.8 clinical hours (total hours: medical registrar [general, cardiology, respiratory] 264, paediatric medical registrar 120, ...
general surgical registrar 120, orthopaedic surgical registrar 120) with additional on-call consultant back-up. Additional specialty registrars attend on an on-call basis.

During the strike period, ED medical staffing was via 10 consultant emergency physicians, 1 career medical officer (CMO), and 3 non-striking registrars providing a daily average of 98.6 clinical hours on a rostered basis (total hours: consultant 359, CMO 20, registrar 114).

Specialty medical staffing of the ED was via the hospital pool of medical and surgical specialists providing a daily average of 128 clinical hours (total hours: physician [general, cardiology, respiratory] 280, paediatrician 120, general surgeon 120, orthopaediac surgeon 120).

**Study participants**—We utilised the hospital’s computerised registration and coding system (HOSPRO) to collect data on all patient presentations during the strike period (0730 hours on 15/6/2006 to 0730hrs on 20/6/2006), and the non-strike period defined as the corresponding days of the subsequent calendar week (0730 hours on 22/6/2006 to 0730 hours on 27/6/2006).

**Definitions and outcome measures**—Patient records were examined for:

- Date and time of presentation,
- Australasian Triage Score,
- Waiting time until medical assessment (defined as time from patient registration until time seen by a doctor),
- Time seen until disposition (defined as time seen by doctor until time of exit from the ED), and
- ED length of stay (defined as time from registration until exit from the ED).

These key times are recorded routinely on our computerised record. Recordings of requested laboratory haematologic and biochemical blood analysis, in addition to plain film radiology, ultrasound, CT scan, and MRI imaging requests from the ED during the study period were obtained retrospectively. Patients who did not wait until medical assessment; death within the ED and death within 48 hours of hospital admission; and 30-day non-scheduled representation were also recorded retrospectively.

**Statistical analysis**—Presentation rates according to triage category were computed for five 24-hour periods (commencing at 0730 hours of the first day and corresponding with strike onset) and covering the six calendar days of the strike and non-strike periods (Thursday–Tuesday inclusive). Statistical analysis of all variables were preformed using SPSS for Windows (version 10.0) software (SPSS, Chicago, IL). Two-tailed students-t and Mann-Whitney U testing were used as appropriate to evaluate differences in continuous variables. Chi-squared testing and Fisher’s exact testing were used were appropriate to compare dichotomous outcomes. A p value less than 0.05 was deemed significant.

**Ethics**—Ethical approval for the study was obtained from the Regional Y committee of the New Zealand Health and Disabilities Ethics Committee.

**Results**

A total of 1291 patient presentations were identified during the strike (SP) and non-strike periods (NSP) (Table 1). No difference was observed in patient age (median 35 years, range 0-91 SP; median 32 years, range 0-97 NSP, p=0.291), or male/female ratio (1.06:1 SP; 1.01:1 NSP, p=0.723). Patient:clinical hours worked ratio for the strike period and non-strike period were 0.54 and 0.55 respectively.

Admission rate according to ATS category were: ATS1 100% for both the SP and NSP; ATS2 81.6% SP vs 89.6% NSP (p=0.188); ATS3 56.4% SP vs 65.1% NSP (p=0.028); ATS4 34.8% SP vs 38.5% NSP (p=0.372); ATS5 10.7% SP vs 11.4% NSP (p=1.0).

Hospital inpatient numbers and bed availability as at 0700 hours for the strike period and non-strike period are presented in Figure 1. Daily bed availability during the strike period was less than the non-strike period (33.0 beds SP vs 48.8 beds NSP, 95% CI difference 4.9 to 26.7).
Table 1. Emergency Department presentations

<table>
<thead>
<tr>
<th>Presentations</th>
<th>Strike period</th>
<th>Non-strike period</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>Rate per 24 hours</td>
<td>Number</td>
</tr>
<tr>
<td>ATS1</td>
<td>3</td>
<td>0.60 (0.49%)</td>
<td>4</td>
</tr>
<tr>
<td>ATS2</td>
<td>76</td>
<td>15.2 (12.5%)</td>
<td>96</td>
</tr>
<tr>
<td>ATS3</td>
<td>298</td>
<td>59.6 (49.0%)</td>
<td>301</td>
</tr>
<tr>
<td>ATS4</td>
<td>203</td>
<td>40.6 (33.4%)</td>
<td>247</td>
</tr>
<tr>
<td>ATS5</td>
<td>28</td>
<td>5.6 (4.6%)</td>
<td>35</td>
</tr>
<tr>
<td>Total</td>
<td>608</td>
<td>121.6</td>
<td>683</td>
</tr>
</tbody>
</table>

Figure 1. Waikato Hospital occupancy and bed availability during strike (SP) and non-strike periods (NSP)

Wait times according to triage category for all patients are presented in Table 2. The percentage of patients seen within recommended waiting times during the strike period were 0%, 63%, 48%, 66%, and 96% for ATS category 1 to 5 respectively. The percentage of patients seen within recommended waiting times during the non-strike period were 25%, 53%, 38%, 47%, and 91% for ATS category 1 to 5 respectively.
Table 2. Wait times per Australasian Triage Score (ATS) in minutes

<table>
<thead>
<tr>
<th>Presentations</th>
<th>Strike period</th>
<th>Non-strike period</th>
<th>95% CI difference</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Number</td>
<td>Mean (SD)</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATS1</td>
<td>8.0 (12.1)</td>
<td>3</td>
<td>4.0 (6.7)</td>
<td>4</td>
</tr>
<tr>
<td>ATS2</td>
<td>15.6 (25.9)</td>
<td>76</td>
<td>23.5 (38.0)</td>
<td>96</td>
</tr>
<tr>
<td>ATS3</td>
<td>43.4 (46.2)</td>
<td>298</td>
<td>73.6 (85.9)</td>
<td>301</td>
</tr>
<tr>
<td>ATS4</td>
<td>53.7 (48.3)</td>
<td>203</td>
<td>82.0 (74.5)</td>
<td>247</td>
</tr>
<tr>
<td>ATS5</td>
<td>47.6 (42.4)</td>
<td>28</td>
<td>50.6 (43.6)</td>
<td>35</td>
</tr>
</tbody>
</table>

Table 3. Time seen to disposition per Australasian Triage Score (ATS) in minutes

<table>
<thead>
<tr>
<th>Presentations</th>
<th>Strike period</th>
<th>Non-strike period</th>
<th>95% CI difference</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Number</td>
<td>Mean (SD)</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATS1</td>
<td>57.7 (38.5)</td>
<td>3</td>
<td>165.0 (90.9)</td>
<td>4</td>
</tr>
<tr>
<td>ATS2</td>
<td>147.9 (129.3)</td>
<td>76</td>
<td>255.1 (246.8)</td>
<td>96</td>
</tr>
<tr>
<td>ATS3</td>
<td>119.9 (124.3)</td>
<td>298</td>
<td>165.0 (176.4)</td>
<td>301</td>
</tr>
<tr>
<td>ATS4</td>
<td>85.5 (78.3)</td>
<td>203</td>
<td>99.7 (115.9)</td>
<td>247</td>
</tr>
<tr>
<td>ATS5</td>
<td>28.9 (35.6)</td>
<td>28</td>
<td>79.8 (125.9)</td>
<td>35</td>
</tr>
</tbody>
</table>

Time seen to disposition for all patients is presented in Table 3. Subgroup analysis of time seen to disposition, according to admission or discharge, demonstrates reduction for ATS category 2 patients who were admitted (95% CI difference 42.6 to 184.9 minutes, p=0.002) and ATS category 3 patients who were admitted (95% CI difference 19.1 to 93.7 minutes, p=0.003) during the strike period. No difference was observed in time seen to disposition for discharged patients of any ATS score.

Table 4. Emergency Department length of stay per Australasian Triage Score (ATS) in minutes

<table>
<thead>
<tr>
<th>Presentations</th>
<th>Strike period</th>
<th>Non-strike period</th>
<th>95% CI difference</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Number</td>
<td>Mean (SD)</td>
<td>Number</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATS1</td>
<td>65.7 (42.3)</td>
<td>3</td>
<td>169.0 (90.9)</td>
<td>4</td>
</tr>
<tr>
<td>ATS2</td>
<td>162.6 (128.8)</td>
<td>76</td>
<td>278.6 (247.5)</td>
<td>96</td>
</tr>
<tr>
<td>ATS3</td>
<td>161.9 (127.2)</td>
<td>298</td>
<td>238.4 (190.6)</td>
<td>301</td>
</tr>
<tr>
<td>ATS4</td>
<td>134.1 (86.6)</td>
<td>203</td>
<td>179.2 (131.0)</td>
<td>247</td>
</tr>
<tr>
<td>ATS5</td>
<td>74.9 (51.9)</td>
<td>28</td>
<td>126.1 (133.0)</td>
<td>35</td>
</tr>
</tbody>
</table>

ED length of stay is presented in Table 4. Length of stay in the ED according to disposition was reduced for ATS category 2 patients who were admitted (95% CI difference 48.3 to 191.5 minutes, p=0.001), ATS category 3 patients who were admitted, and discharged (95% CI difference 51.2 to 128.1 minutes, p<0.001 and 95% CI difference 12.3 to 63.5 minutes, p=0.004 respectively), and ATS category 4 patients who were admitted, and discharged (95% CI difference 15.1 to 102.0 minutes, p=0.009 and 12.8 to 48.7 minutes, p=0.001 respectively) during the strike period.
No difference was observed in ED mortality (2 patients SP; 1 patient NSP, p=0.513), 48-hour mortality (2 patients SP; 4 patients NSP, p=0.419), patient walkout (11 patients SP; 17 patients NSP, p=0.219), or 30-day unscheduled representations (43 patients SP; 64 patients NSP, p=0.119).

Utilisation of commonly ordered laboratory investigations and radiologic imaging modalities are presented in Table 5.

### Table 5. Clinical investigations during strike (SP) and non-strike periods (NSP)

<table>
<thead>
<tr>
<th>Presentations</th>
<th>Strike period</th>
<th>Non-strike period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test/patient</td>
<td>Total (n)</td>
</tr>
<tr>
<td>Haematology</td>
<td>0.54</td>
<td>331</td>
</tr>
<tr>
<td>Biochemistry</td>
<td>0.54</td>
<td>326</td>
</tr>
<tr>
<td>Plain film XR</td>
<td>0.45</td>
<td>272</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>0.025</td>
<td>15</td>
</tr>
<tr>
<td>CT</td>
<td>0.066</td>
<td>40</td>
</tr>
<tr>
<td>MRI</td>
<td>0.0016</td>
<td>1</td>
</tr>
</tbody>
</table>

**Limitations**—This study was by design observational and as such suffers from deficiencies inherent with this methodology. Unavoidable errors of the administrative dataset include reliance on electronic patient registration, and data entry by both clerical staff and clinicians to determine key outcome variables. Delays in patient registration or inaccurate clinician reporting of time seen may have contributed to erroneous computer entries. These are more likely to have resulted in significant discrepancies for presentations of high acuity, where initial assessment and patient management take priority over data entry which is often completed retrospectively. Specifically, chart review of ATS category 1 patients revealed doctor attendance on patient arrival (and as such wait time equaling zero minutes) in all cases, yet electronically recorded waiting times are significantly longer. We were unable to ascertain the impact of this systemic error on presentations of other triage categories.

The observational nature of this study dictates that the relative contributions of multiple interacting factors contributing to the observed efficiencies in ED patient processing can be inferred only. Further evaluation of the many factors inherent in patient transit through the ED (patient complexity, number of consultations, waiting times for investigation results, evaluation of response to treatment, time to disposition decision, access block, time of presentation) are required to accurately characterise these.

We attempted to reduce potential confounding secondary to markedly different hospital occupancy, and the adherent efficiencies of scale, by electing the non-strike period during the week immediately following the strike. As such, potential exists for patients to have delayed presentation to the week of the non-strike period in accordance with media and public information directives, thus increasing non-strike period patient numbers and potential acuity. No significant difference was observed however in the number of strike and non-strike presentations. Furthermore the mean
strike and non-strike daily presentations were both lower than the seasonal average of 145 patients daily.

The existence of a “strike culture” with heightened awareness of the requirement for expeditious patient processing and disposition by senior clinicians during the strike period, in addition to organisational streamlining in anticipation of expected demand may have contributed to the observed differences in efficiency.

Elective admissions and surgeries were cancelled during the strike period in anticipation of increased demand on available staff. Research and teaching activities were curtailed. Conversely up-regulation and the return to ‘normal’ hospital function in the non-strike period (with the adherent reliance on doctors returning from strike action) may have resulted in inefficiencies not usually present in patient-processing times. We have been unable to quantify the effect of these potential confounders.

Finally this study examines patient processing and measures of activity within the ED only. Whilst the results of this study indicate efficiencies in emergent patient processing, the effect on the organisation as a whole has not been examined. This would require a whole of hospital analysis.

**Discussion**

ED length of stay is a key measure of ED throughput and a marker of overcrowding. In the present study we have observed reduced ED length of stay to be associated with increased treating clinician seniority during the period of a resident doctors’ strike. This increase in ED efficiency was observed during a period of comparable patient attendance and acuity. Triage during the strike and non-strike periods were via experienced senior nursing staff thus mitigating possible bias in triage categorisation. Similar patient walkout; death within the ED and death within 48 hours; and 30-day non-scheduled readmission rates suggest the observed reductions in ED transit occurred with no decrease in the quality of patient care.

Reduced strike period waiting times are likely resultant on the compounding effect of reduced patient transit times on ED bed occupancy, and subsequent bed availability for newly presenting patients, and the reduction (albeit non statistically significant) in number of patient presentations between the two periods. Additionally, the described ‘strike culture’ may have engendered increased urgency amongst senior clinicians thus prompting efforts to ensure reduced wait times. Notably, however, only ATS category 5 patients from the strike and non-strike periods met advocated performance thresholds for patients seen within recommended waiting times (96% SP and 91% NSP respectively). As such, generalisation of study findings to departments where these performance indicators are routinely met must be interpreted with caution.

A significant reduction in time seen to disposition was observed across a broad range of patient acuity. Reductions in time seen to disposition for patients of moderate to high acuity (ATS category 3 and 2) who were admitted, contributed significantly to the observed efficiencies in ED transit. This suggests disposition decisions were arrived at earlier in the course of patient assessment by senior doctors, with more rapid initiation of the admission process during the strike period.

Such decisions are likely to have been made with greater reliance on clinical acumen than pending investigation results. Additional reduction in time seen to disposition for
admitted patients may have resulted from strategies to improve access to ward beds. The strike period was, however, universally characterised by a reduction in absolute bed availability as compared the non-strike period (Figure 1).

A reduction in the admission rate for ATS category three patients during the strike period was observed. This suggests a subgroup of patients of moderate acuity who may have their discharge from the ED facilitated by more senior clinicians, thus avoiding hospital admission. This may have resulted from earlier institution of appropriate therapy, or the ability of senior clinicians to arrange early outpatient follow-up. Alternatively, the apparent increased admission rate during the non-strike period may be consequent on patients who were erroneously assigned a lower triage category, or patients who have delayed appropriate presentation for the duration of the strike period and thus attended with more fulminent illness during the non-strike period.

Conversely, no significant difference was observed in time seen to disposition for discharged patients of any ATS category. This finding is somewhat counter-intuitive to what would be expected with more experienced clinicians wherein early discharge might be predicted. Several factors in addition to the disposition decision, however, are inherent in the discharge process (input from supportive agencies [social work, physiotherapy], routine nursing tasks, transport arrangements) which are likely to have impacted on time seen to disposition for discharged patients irrespective of study interval. Additionally, increased discharge for patients of ATS category 3 during the strike period suggests patients of moderate acuity who were treated and successfully discharged from the ED by senior clinicians, potentially contributing to these data intervals.

Requests for laboratory and radiologic investigations were similar during the strike and non-strike periods in the present study. Previous investigators have demonstrated reduced investigation ordering by more senior clinicians.\textsuperscript{14,15} The failure of this study to replicate such differences is likely to be secondary to the protocolised initiation of simple haematologic and biochemical blood analysis, in addition to plain film radiography requests, by senior nursing personnel at our institution. This process was unaffected during the strike.

Few previous studies have investigated the effect of treating doctor seniority on the efficiency of emergency care systems. Studies examining the introduction of ‘hospitalists’ in the North American health system clearly document enhanced efficiency when inpatients are cared for by senior clinicians.\textsuperscript{16–18} Salazar et al\textsuperscript{12} have similarly reported reductions in patient wait times and increased departmental efficiency during a Spanish junior doctors’ strike. That study, however, compared striking departmental operation when staffed with specialist doctors, with a period of usual complement characterised by training doctors in the absence of a recognised emergency medicine faculty. Conversely, the non-strike period of the current study examines a period of normal staff complement with greater than 16 hours daily of specialist Emergency physician supervision in a department staffed by doctors training in emergency medicine and by inpatient specialty trainees.

Travers et al\textsuperscript{19} have demonstrated reduction of waiting times and more rapid discharge for walk-in patients triaged by a senior emergency physician as compared standard
nursing triage. Similarly, waiting times improved and a trend toward decreased access block was observed by O’Connor et al with consultant emergency physician presence in a regional referral hospital ED.\textsuperscript{20}

The creation of acute admission units led by consultant physicians has further demonstrated increased efficiencies and earlier patient discharges with senior clinician input early in the course of medical emergencies.\textsuperscript{21} Notably, however, McNamara et al reported that when EDs were left in the hands of senior physicians during the 1975 strike by house officers in New York City, they performed as well as (but not better than) junior doctors.\textsuperscript{22}

Increasing senior clinician input early in the course of ED presentations may result in similar efficiencies to those observed in this study when effected in emergency care systems more heavily staffed with resident doctors in training. Additionally, results from the present study suggest increased numbers of patients with illness of moderate acuity may have their discharge facilitated by the experience of more senior clinicians.

The potential to reduce ED length of stay and thus overcrowding via accelerating decision-making, and the role of senior clinicians in this process, requires further evaluation.

**Conclusion**

Significant reductions in ED patient processing times were observed during the period of a resident doctors’ strike. This was associated with increased seniority of front-line ED medical staff in addition to departmental and institutional streamlining measures. Strategies to increase senior clinician involvement early in the course of ED presentations may result in similar efficiencies during periods of usual operation.

**Competing interests:** None.

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**References:**

Evidence-based recommendations for hand hygiene for health care workers in New Zealand

Peter J Larmer, Trish M Tillsom, Faye M Scown, Philippa M Grant, Jamie Exton

Abstract

Aim The aim of this systematic review is to establish evidence-based recommendations for hand hygiene for health care workers in New Zealand.

Methods Using a systematic approach to literature searching, relevant studies were retrieved and evaluated using a standardised tool. The 23 studies that met the inclusion criteria were categorised into subgroups depending on the type of comparison: hand hygiene product; skin condition; hand drying method. A ‘best-evidence synthesis’ was utilised for classifying the evidence.

Results Included studies provided evidence to support the use of alcohol-based hand rub as the preferred hand hygiene product. There was conflicting evidence for the use of medicated or plain soap, or any particular method to dry hands.

Conclusions Hand hygiene is a crucial component of risk management for both health care workers and their patients. It is important that hand hygiene practice is based on the best current evidence. As a result of a systematic review, evidence-based recommendations for hand hygiene for health care workers are proposed.

Ever since we were toddlers our parents have taught us to wash our hands. It is as much a social and cultural act as it is a hand hygiene procedure. As health care professionals, the significance of hand hygiene greatly increases in importance as we attempt to prevent cross-infection.

In 2005, the World Health Organization (WHO) launched the Clean Care is Safer Care campaign, bringing together experts from around the World to formulate and globally implement hand hygiene guidelines in health care settings. Hand hygiene is defined as the reduction of harmful infectious agents by the application of alcohol-based hand rubs (ABHR) without the addition of water, or by handwashing with plain or medicated/antimicrobial soap and water.

Health care workers (HCW) can readily contaminate their hands with transient flora by touching the intact skin of patients. Multiresistant organisms capable of causing serious infections such as methicillin-resistant Staphylococcus aureus (MRSA), facilitate cross-infection by surviving well on skin and inanimate surfaces.

MRSA is now as much a problem for the community HCW as it is for those in the hospital environment, with 52.3% of new MRSA infections in New Zealand (NZ) in 2006 occurring in the community in people without established risk factors, and 48.7% in hospitals.
Aside from the personal suffering, infected patients incur health costs on average 2.9 times greater than those of uninfected patients, resulting in an estimated NZ$136 million expenditure in the year 1999. Rates can be significantly reduced with a sustained improvement in hand hygiene in both the health care setting and the community.

It is the responsibility of each HCW to contribute to the communities’ endeavours to reduce infection and the responsibility of all employers under the Health and Safety in Employment Act 1992, to have infection control policies that protect their staff.

Hand hygiene policies, usually based on a combination of evidence from randomised controlled trials (RCT) and expert opinion, vary between countries and between health care institutions within NZ.

A comparison of international hand hygiene guidelines concluded that most countries recommend handwashing with medicated or plain soap, except in central European countries, where an ABHR is the preferred method. The (American) Guideline for Hand Hygiene in Healthcare Settings and the WHO Guidelines on Hand Hygiene in Health Care, strongly recommend the use of ABHR for routine decontamination in all health care settings.

The NZ Ministry of Health similarly recommends ABHR as the preferred method for hand hygiene in situations where MRSA is an issue, and states that as many carriers are asymptomatic, all patients in hospitals and the community must be treated as though infectious. However there is variation in NZ district health board (DHB) policies, with some DHBs specifying ABHR be used for routine decontamination while others do not prioritise one method over another.

Knowledge and education do not ensure adherence to such recommendations, with compliance to hand hygiene guidelines varying from 4% to 51%. Infection control is inevitably adversely affected by such poor compliance.

As a result of the variation in hand hygiene guidelines within NZ, it was decided to conduct a systematic review. The aim of this systematic review was to establish evidence-based recommendations for hand hygiene for HCW in NZ.

Method

Medline, Cochrane Library, CINAHL, and AMED databases were searched for the years 1985 through May 2006, limited to human studies and the English language.

Keywords and phrases used were: handwashing or hand washing; hand hygiene; and hand contamination. Reference lists from retrieved articles were further hand-searched for relevant papers. Excluded from the review were studies investigating surgical hand asepsis, in vitro suspension studies, case studies, observational studies, and studies within the community, day-care centres, or schools.

To rate the methodological quality of each study, four reviewers (PG, JE, TT, FS) blinded to the others’ assessment, critically appraised each study using the Generic Appraisal Tool for Epidemiology (GATE) Checklist for Randomised Controlled Trials, and used a modified quality scoring system based on the methods of the Cochrane Musculoskeletal Injuries Group.

The scoring system evaluated the extent to which the study minimised bias by rating 11 aspects of internal and external validity, and allocating a score of 0, 1, or 2 to each criterion based on rating guidelines. The 11 scores were summed to give a total quality score out of a maximum of 22. Studies that scored 19 or greater were defined as ‘high quality’ and 15–18 as ‘moderate quality’. These were included in the final systematic review.
A study with a score ≤14 was rated ‘low quality’ and was excluded from this systematic review. The principal author was contacted to clarify methodological details that were unclear from the published paper. Any discrepancy between study scores was discussed and a consensus reached with a fifth reviewer (PL) brought into the discussion when necessary.

Significance levels were set at \( p<0.05 \) for each of the review outcome measures. Outcomes were extracted and defined as positive (+) if the intervention outcome was statistically significantly more effective than the comparison group, neutral (0) if there was no significant difference, and negative (-) if the intervention outcome was statistically significantly less effective.

A best-evidence synthesis was utilised and the conclusions based on five levels of scientific evidence. ‘Generally consistent’ was defined as two-thirds or more of the studies reporting the same finding.

<table>
<thead>
<tr>
<th>Level</th>
<th>Evidence Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1</td>
<td>Strong evidence—provided by generally consistent findings in multiple, relevant, high quality RCT.</td>
</tr>
<tr>
<td>Level 2</td>
<td>Moderate evidence—provided by one relevant, high quality RCT and/or generally consistent findings in multiple, relevant, moderate quality RCT.</td>
</tr>
<tr>
<td>Level 3</td>
<td>Limited evidence—provided by one RCT of moderate quality rating.</td>
</tr>
<tr>
<td>Level 4</td>
<td>Conflicting evidence—inconsistent findings among multiple RCT.</td>
</tr>
<tr>
<td>Level 5</td>
<td>No evidence—no RCT.</td>
</tr>
</tbody>
</table>

**Results**

**Antimicrobial efficacy**—Within the 12 studies that met the inclusion criteria there were 35 comparisons of the antimicrobial efficacy of ABHR to plain and/or medicated soap (Table 1). Twenty-four demonstrated statistically significant-positive results and the remaining 11 comparisons demonstrated no significant differences between ABHRs and medicated and/or plain soap (Table 1). No comparison demonstrated superior efficacy of medicated or plain soap over ABHR.

Three laboratory studies investigated various hand hygiene methods following heavy contamination of specific bacteria.

Kjolen and Andersen found a 70% ethanol, 0.5% chlorhexidine gluconate (CHG) ABHR to significantly reduce heavy contamination of *Staphylococcus aureus*, and found ABHRs containing 70% ethanol with or without CHG to reduce *Enterobacter cloacae*. Plain soap did not reduce the growth of either bacterial species. Heavy contamination of *Acinetobacter baumannii* was significantly reduced with ABHR and the medicated soap povidine-iodine compared with the medicated soap CHG.

Guilhermetti et al reported the same results in the presence of light and heavy contamination of MRSA. Trick et al using a lower concentration of alcohol significantly reduced the frequency of hand contamination for gram-negative bacilli and transient organisms, but not for *Staphylococcus aureus* or *Candida*. Larson & Bobo found ABHRs to be microbiologically more effective than medicated or plain soap in the presence of blood, although the blood was not physically removed.

Consistent findings in greater than two-thirds of moderate quality studies provided level 2/moderate evidence that ABHRs have greater antimicrobial efficacy than medicated and/or plain soap.

Within the 12 studies there were 22 comparisons of medicated soap to plain soap: 10 were statistically significant-positive, 11 had no significant differences, and 1 study demonstrated superiority of plain soap over CHG for the removal of MRSA (Table 1).
### Table 1. Summary of trials comparing the antimicrobial efficacy of hand hygiene products

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Score /22</th>
<th>n</th>
<th>Outcome: ABHR &gt; Med or Plain soap</th>
<th>Conclusion</th>
<th>Outcome: Med soap &gt; Plain soap</th>
</tr>
</thead>
<tbody>
<tr>
<td>Girou et al 2002</td>
<td>20</td>
<td>23</td>
<td></td>
<td>ABHR (45% 2-propanol, 30% 1-propanol, 0.2% mecetronium, emollient) &gt; 4% CHG.</td>
<td></td>
</tr>
<tr>
<td>Trick et al 2003</td>
<td>18</td>
<td>66</td>
<td>+</td>
<td>Gram-ve bacilli &amp; transient organisms: ABHR (62% ethanol) &gt; plain soap = medicated wipes (0.1% benzalkonium chloride).</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Staphylococcus aureus &amp; Candida: ABHR = plain soap = medicated wipes.</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ring wearing associated with 10x &gt; risk for contamination with Staphylococcus aureus, gram-ve bacilli, or Candida. Stepwise increase in risk with increased number of rings worn.</td>
<td></td>
</tr>
<tr>
<td>Kac et al 2005</td>
<td>18</td>
<td>50</td>
<td>+</td>
<td>ABHR (45% 2-propanol, 30% 1-propanol, 0.2% mecetronium, emollient) &gt; plain soap.</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No transient flora present after ABHR.</td>
<td></td>
</tr>
<tr>
<td>Larson et al 1989</td>
<td>18</td>
<td>80</td>
<td>N/A</td>
<td>End day 5 (6 washes/day): 2% CHG = 0.3% triclosan = 0.6% parachlorometaxylenol = plain soap.</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>End day 5 (18 washes/day): 2% CHG &gt; 0.3% triclosan = 0.6% parachlorometaxylenol &gt; plain soap.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CFU increased in plain soap group after 1x wash.</td>
<td></td>
</tr>
<tr>
<td>Guilhermetti et al</td>
<td>18</td>
<td>5</td>
<td>+</td>
<td>MRSA light &amp; heavy contamination: ABHR (70% ethanol) = 10% povidone-iodine &gt; plain soap &gt; 4% CHG.</td>
<td>0</td>
</tr>
<tr>
<td>2001</td>
<td></td>
<td></td>
<td></td>
<td>(alc = P.I)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(alc &gt; CHG &amp; P.S)</td>
<td></td>
</tr>
<tr>
<td>Butz et al 1990</td>
<td>17</td>
<td>48</td>
<td>+</td>
<td>End day 1 (15 washes): 4% CHG &gt; plain soap &gt; alcohol wipes (30% ethanol) &gt; 1% triclosan.</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>End day 5 (75 washes): CHG &gt; triclosan &gt; alcohol wipe = plain soap.</td>
<td></td>
</tr>
<tr>
<td>Larson et al 1987</td>
<td>17</td>
<td>40</td>
<td>+</td>
<td>End day 1 (15 washes): ABHR a (unspecified) &gt; ABHR b (unspecified) &gt; 4% CHG &gt; plain soap.</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(alc a &gt; CHG &amp; P.S)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+(alc b &gt; CHG &amp; P.S)</td>
<td></td>
</tr>
</tbody>
</table>
End day 5 (15 washes/day): ABHR a (unspecified) = ABHR b (unspecified) = 4% CHG > plain soap.
3ml > 1ml for all test agents

Zaragoza et al 1999
17 43 ABHR (45% 2-propanol, 30% 1-propanol, 0.2% meclotronium, emollient) > plain soap.
30% of plain soap had increased CFU after handwashing. Nil with ABHR.

Larson et al 2001
16 46 2%CHG = ABHR (61% ethanol, emollients)

Cardoso et al 1999
15 5 Acinetobacter baumannii heavy contamination: ABHR (70% ethanol) = 10% povidone-iodine > 4% CHG = plain soap.
Acinetobacter baumannii light contamination: ABHR (70% ethanol) = 10% povidone-iodine = 4% CHG = plain soap.

Larson & Bobo 1992
15 71 With blood on hands: ABHR (70% ethanol & 0.5% CHG) = ABHR (70% isopropanol) > 7.5% povidone-iodine = 4% CHG = plain soap.
Without blood: ABHR (70% isopropanol) > 7.5% povidone-iodine = 4% CHG = plain soap.
Without blood: Increase in CFU with plain soap.
Although ABHR microbiologically effective, blood was not physically removed.

Kjølen & Anderson 1992
15 3 S. aureus heavy contamination: Significant reduction only with ABHR (5% CHG, 70% ethanol) > ABHR (70% ethanol) = ABHR (40% isopropanol) = plain soap.
E. cloacae heavy contamination: ABHR (5% CHG, 70% ethanol) = ABHR (70% ethanol) > ABHR (40% isopropanol) > plain soap. Do not eradicate.
Successive contamination of all species: ABHR (5% CHG, 70% ethanol) most effective. Did not eradicate E. cloacae with repeated handwashing.
Heavy contamination: plain soap did not reduce growth of any bacterial species.

ABHR Alcohol based hand rub; CHG Chlorhexidine gluconate; CFU Colony forming unit; P.I Povidone-iodine; tri Triclosan, PMX parachlorometaxylenol; P.S plain soap; alc Alcohol; = Equivalent efficacy; > Statistically significantly more antimicrobial efficacy; < Significantly less antimicrobial efficacy; N/A Not Applicable; + Product statistically significantly more effective; 0 No significant difference between products; – Product statistically significantly less effective.
With heavy contamination, the medicated soap povidine-iodine but not CHG was superior to plain soap.\textsuperscript{30,32} In contrast, Larson and Bobo found no significant difference between povidine-iodine, CHG, and plain soap with or without blood on the hands.\textsuperscript{33} It was noted that the significant-positive results were demonstrated in studies that were of higher methodological quality than the neutral results.

There is level 4/conflicting evidence whether medicated soap is more efficacious than plain soap at reducing hand contamination.

A moderate quality RCT by Trick et al provided level 3/limited evidence that there is a proportional increase in hand contamination with the number of rings worn before and after hand hygiene.\textsuperscript{31}

**Skin condition**—The statistically significant-positive findings of all five\textsuperscript{38,39,42–44} moderate quality trials provide level 2/moderate evidence that ABHR are less irritating than medicated and/or plain soap (Table 2).

### Table 2. Summary of trials comparing skin condition changes with various hand hygiene products

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Score/22</th>
<th>n</th>
<th>Study duration</th>
<th>Conclusions</th>
<th>Outcome: ABHR &gt; med/plain soap</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butz et al 1990</td>
<td>17</td>
<td>48</td>
<td>5 days</td>
<td>End day 5 (75 washes): alcohol wipes (30%) / improved &gt; 4% CHG / worsened &gt; 1% triclosan / worsened.</td>
<td>+</td>
</tr>
<tr>
<td>Larsen et al 2001</td>
<td>16</td>
<td>46</td>
<td>4 weeks</td>
<td>From 3 weeks: ABHR (61% ethanol, emollient) / improved &gt; 2% CHG / worsened. 2% CHG group applied lotion 50% more often.</td>
<td>+</td>
</tr>
<tr>
<td>Grove et al 2001</td>
<td>15</td>
<td>18</td>
<td>5 days</td>
<td>ABHR (61% ethanol, 1% CHG, emollient) / improved &gt; 4% CHG / worsened. Difference in condition became more pronounced as wash cycles increased.</td>
<td>+</td>
</tr>
<tr>
<td>Pederson et al 2005</td>
<td>15</td>
<td>19</td>
<td>10 days</td>
<td>Repeated application: ABHR (78% ethanol, 5% isopropanol, emollient) &gt; alternate application ABHR &amp; plain soap &gt; plain soap. No significant difference in inflammatory response between products.</td>
<td>+</td>
</tr>
<tr>
<td>Boyce et al 2000</td>
<td>15</td>
<td>29</td>
<td>2 weeks</td>
<td>ABHR (62% ethanol, 10% isopropanol, emollient) / no change &gt; plain soap / worsened.</td>
<td>+</td>
</tr>
</tbody>
</table>

Product tolerated by skin significantly better > product tolerated by skin significantly less well; N/A Not Applicable; + ABHR’s statistically significantly less irritating than soap.

A high quality study by McCormick et al\textsuperscript{45} provided level 2/moderate evidence that skin condition improves with regular, liberal application of hand moisturisers.

**Hand drying**—Two studies\textsuperscript{37,46} of moderate methodological quality met the inclusion criteria (Table 3). Gustafson et al found no significant difference between drying the hands with a paper towel for 15 seconds, a cloth towel for 15 seconds, an air dryer for 30 seconds, or allowing spontaneous evaporation until the hand felt dry.\textsuperscript{37} However, Yamamoto et al\textsuperscript{46} found that when hands were held stationary for 30 seconds under a
warm air dryer, it was significantly more effective than up to three paper towels in reducing the number of bacteria from the whole hand.

Fifteen seconds of warm air drying was more effective than three paper towels on fingers and fingertips. Ultraviolet light in a warm air dryer increases the effectiveness of the dryer, whereas rubbing the hands together while drying, decreases the effectiveness of drying.

Therefore, the evidence for whether any particular method of hand drying is more effective than another, is conflicting/level 4.

Table 3. Summary of trials comparing hand drying methods

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Score /22</th>
<th>n =</th>
<th>Conclusions</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gustafson et al 2000</td>
<td>16</td>
<td>99</td>
<td>Drying by cloth towel (15s) = paper towel (15s) = air dryer (30s) = spontaneous evaporation until dry.</td>
<td>0</td>
</tr>
<tr>
<td>Yamamoto et al 2005</td>
<td>16</td>
<td>30</td>
<td>Warm air dryer with stationary hands, +/- U.V, 30(s) whole hand &gt; 1,2,3 paper towels. Warm air dryer with stationary hands, +U.V, 15(s) &gt; 1,2,3 paper towels. Warm air dryer whilst rubbing hands, +U.V. 30(s) whole hand &gt; 1,2,3 paper towels &gt; -U.V. 30(s) whole hand 1,2,3 paper towels do not significantly reduce bacteria on palms or fingers.</td>
<td>For air dryer + (+/- U.V.) + (+ U.V.)</td>
</tr>
</tbody>
</table>

= Equivalent efficacy; Statistically significantly more antimicrobial efficacy > Significantly less antimicrobial efficacy; 0 No significant difference between methods; + Air dryer statistically significantly more effective.

Discussion

It is important to consider the concentration of the active ingredient of the ABHR when interpreting the results. Alcohol wipes, which contain 30% ethanol, have no greater efficacy than plain soap. With the exception of the three laboratory-based heavy contamination studies, the other three studies that demonstrated no significant difference between ABHR and medicated soap used a 61–62% ethanol handrub. Greater efficacy was demonstrated by handrubs that contained 70% alcohol and 70% alcohol with CHG (Table 1).

In the intensive care setting efficacy results may be affected by the average time spent on hand hygiene. When subjects spent significantly less time performing an ABHR (12.7 seconds) compared to a medicated hand wash (21.5 seconds), they demonstrated equivalent efficacy. Whereas Girou et al found ABHR to have greater efficacy and noted there was no difference in the median time spent on either method (30 seconds). All other intensive care setting studies utilised a standard 30-second hand hygiene method and similarly demonstrated ABHR to have superior efficacy.

Although Larson and Bobo found that ABHR were microbiologically more effective than medicated or plain soap in the presence of blood, it is important to note ABHR did not remove the blood, necessitating handwashing to remove the visible soiling.
This evidence suggests that when there is blood on the hands, it may be worthwhile considering the use of handwashing followed by ABHR.

Three studies noted that there was an increase in hand contamination after washing with plain soap but not with medicated soap or ABHR. Additionally, when efficacy was compared after 15–18 washes over one day or several days, all three studies found medicated soap to have superior antimicrobial efficacy to plain soap. This suggests that in the clinical situation when handwashing frequency is high, it may be advantageous to use medicated soap rather than plain soap.

Trick et al provided evidence for recommending HCW minimise the number of rings they wear, by establishing a step-wise increased risk of hand contamination with the number of rings worn. Additionally, poor skin condition is a known risk factor for hand contamination and a significant barrier to hand hygiene.

Seventy-four percent of NZ and Australian HCW report dry/damaged hands, with 13.7% reporting symptoms severe enough to seek medical treatment, necessitating consideration of products that have good antimicrobial efficacy whilst minimising skin damage.

Grunewald et al established that significant deleterious changes such as alteration of stratum corneum moisture content, skin barrier function, skin surface pH, skin blood flow, and the amount of skin surface lipids occurred with repetitive washing over 1 week using either a mild or strong soap. Skin condition progressively worsened as the number of handwashing cycles increased, however (in contrast) repeated use of ABHR tended to improve skin condition (Table 2). Additionally, skin condition can be preserved and improved by moisturising.

McCormick et al concluded that liberal application of an oil-based lotion or a barrier cream significantly improved skin condition in HCW with severe hand irritation within 1 week, and that the improvement was sustained throughout the 4-week study period.

Lack of time, high workload, understaffing, inaccessibility of product, inconvenience of sink location, and prioritising patients needs, have also been identified by HCW as barriers to hand hygiene. As a result of ABHR being easier to apply and more accessible, some of these issues are addressed with the introduction of ABHR, improving hand hygiene compliance significantly to rates of 60% to 84%.

Pittet et al utilised a novel flat bottle designed to be easily carried in a pocket. Other advantages noted in the trials of ABHR include: that they are rapidly effective on transient/contaminating flora; have a wide antimicrobial spectrum; spread easily on hands lessening skin trauma; and dry by evaporation negating the need for a drying facility.

Although ABHR have no persistent antimicrobial activity, low concentrations of CHG can be added, resulting in greater residual activity than alcohol alone. Concerns have been raised about the potential flammability of the alcohol in some situations, which emphasises the need for hands to be rubbed together until all alcohol has evaporated. Some authors have described a residual film on the hands after repeated use of ABHR necessitating an intermittent handwash.
It has been clearly established that moisture significantly increases microbial transmission, implying that thorough hand drying is an essential component of hand hygiene.\textsuperscript{53,54} Although the hand drying studies had conflicting results, consideration must be given to the risk of cross-infection with cloth towels,\textsuperscript{2} the correct disposal of contaminated paper towels,\textsuperscript{55} and the potential abrasiveness to the skin of paper towels.\textsuperscript{38} Taylor et al established that warm air dryers are no more likely to contaminate the environment with airborne micro-organisms than drying the hands with paper towels.\textsuperscript{55}

The problems with comparing study results are generic to all evidence based reviews. There was wide variation in protocols such as: type, volume, and concentration of active ingredient; technique and duration of hand hygiene method; test organism; and method and frequency of contamination—all of which affect efficacy results.\textsuperscript{56} The considerable variety of outcome measures utilised was a problem particular to skin condition studies.

All of the studies also had methodological limitations. Outcome assessor blinding reduces the potential for bias but was not consistently used, and the statistical intention to treat principle was only utilised in one study.\textsuperscript{54} Other study limitations were due to the difficulty creating experimental conditions in institutions, such as differences in care programmes other than the intervention, or problems inherent in hand hygiene research such as the inability to blind subjects to the intervention.

For this review there was no attempt to search non-English journals, conference proceedings, or unpublished studies inevitably thus resulting in a degree of publication bias. In a best-evidence synthesis where judgements are made on study quality, there may be a risk of reviewer bias, compared to a meta-analysis that purports to avoid it by including all studies regardless of quality.\textsuperscript{26} Reviewer bias was addressed in this review by the application of a comprehensive scoring system that rated the study on the basis of the extent to which the study design minimised bias. Additionally the studies were independently reviewed and scored by four reviewers.

**Conclusions**

**Evidence-based recommendations for hand hygiene for health care workers**— Based on the weight of the evidence the following recommendations for hand hygiene are proposed:

*Level 2 Evidence*

- ABHR containing 70% alcohol are the preferred products for hand hygiene. They must contain an emollient, and preferably contain 0.5% CHG.
- If using soap instead of ABHR, hand moisturisers should be used liberally and at regular intervals.
Level 3 Evidence

- Hands must be washed with soap and water when visibly soiled.
- The number of rings worn should be minimised.

Level 4 Evidence

- Hands must be thoroughly dried, however there is no evidence that any particular method (i.e. handtowels, air dryer) is more effective than another.
- Due to the lack of data it is not possible to make recommendations on the use of either medicated or plain soap.

Ensuring clinical staff comply with hand hygiene guidelines could be considered an important component of risk management. Indeed, the Health and Safety in Employment Act 1992 requires employers to identify all hazards to which employees could be exposed, and to eliminate, isolate, or minimise the hazard.

To lessen the likelihood of harm being caused by a hazard such as cross-infection, employers are obliged to have clear, workable policies and procedures in place and to monitor compliance. Evidence shows that when implementing new hand hygiene guidelines to address barriers to adherence such as lack of knowledge, forgetfulness, and lack of belief in the efficacy of protocols, multifaceted programmes combining education, reminders, and feedback are necessary to gain sustained compliance.

Ultimately, however, the responsibility lies with each practitioner in their own healthcare setting: ‘First, do no harm’.

Competing interests: None known.

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A rare cause of early post-partum haematuria secondary to uterovesical fistula

Tahir Basheer, Dominic Lee, Warren Davis, Rahul Rindani

Uterovesical fistulae are uncommon, constituting 1–4% of all urogenital fistulae.\textsuperscript{6,8} Although the least common type of urogenital fistulae, they cannot be considered a rarity as there are about 800 published cases.\textsuperscript{11} Most Units report 1–5 cases over 5–15 year periods.\textsuperscript{3} Presentation is variable, ranging between 3–7 years post-delivery. There are few case reports about this condition following vaginal birth after caesarean (VBAC).\textsuperscript{8}

In this article we outline the immediate diagnosis and the management of a case involving this complication.

Case report

A 26-year-old woman, Gravida 2 Para 1, underwent labour. She had a previous delivery by emergency lower-segment caesarean section at 40 weeks gestation after a failed attempt at vacuum delivery. There was no other significant past medical or surgical history.

The current pregnancy was complicated by intrauterine growth restriction (IUGR) and multiple presentations for management of possible antepartum haemorrhage in the last trimester. She was induced at 39 weeks by artificial rupture of membranes (ARM) and syntocin infusion. She had an epidural and in her third stage of labour she developed decelerations in her cardiotocograph (CTG), which became deeper and wider. She progressed to a vacuum-assisted and Wrigley’s forceps delivery. She had persistent frank haematuria post-delivery with haemodynamic instability but mild pain and generalised abdominal tenderness. A 50 ml saline bladder wash was performed; unexpectedly the saline discharged from the vagina.

Dehiscence of the previous caesarean scar with formation of a uterovesical fistula was the initial diagnosis. This was confirmed by cystogram, ultrasound scan, and cystoscopy after involvement of the Urology Team (see Figure 1). Conservative management with transurethral catheter was instituted which failed after 4 months with persistent intermittent urine leak per vaginum, and menouria.

Further discussion of surgical treatment by laparotomy was conducted; a total abdominal hysterectomy and bladder repair with omental patch was decided after considerable counselling.

Intraoperatively, dense fibrous adhesions were noted between the lower segment of the uterus and the bladder. A 2–3 cm fistula was located in the lower segment scar area towards the left side of the uterus. The trigone and ureters were not involved. The hysterectomy was completed and the bladder was repaired with an omental patch. A transurethral catheter was left \textit{in situ} on free drainage for 2 weeks. At 6 weeks post-surgery, she was continent.
Discussion

While injury to the lower urinary tract is uncommon, occurring in only 0.1–0.3% of births, however it is expected to increase worldwide because of an increase in caesarean section rates. This complication is important from clinical, social, and medicolegal aspects.

The causes of peripartum bladder and uterine injury resulting in fistulae formation are nearly always iatrogenic. Risk factors include severe dystocia, instrumental deliveries, manual removal of the placenta, placenta accreta, uterine rupture, and previous caesarean section. Labour induction with prostaglandins, in particular ARM and IV syntocin, may be associated with an increased risk of scar dehiscence in comparison to a spontaneous onset of labour this should be emphasized in the consent process with women who have previously delivering via caesarean section.

The development of a fistula is believed to relate to higher attachment of the bladder relative to the lower segment, usually secondary to scarring from previous surgery. With an unrecognised bladder injury or suture transfixion of the bladder, a tract may develop between the bladder and uterine wall. Other risk factors associated with uterovesical fistula are malignancy, irradiation, and intrauterine devices.

Józwik and Józwik have proposed a classification for uterovesical fistula which is based on the route of menstrual flow. Type 1 (Youssef syndrome) is menouria,
amenorrhoea, and complete continence of urine. Type 2 is dual direction menstrual flow via bladder and vagina. Type 3 is normal vaginal menses but lack of menouria. The management of vesicouterine fistula can be either conservative or surgical.

Conservative management is indicated when the fistula is diagnosed early and is
small.\textsuperscript{1,2,10} Spontaneous healing is reported in 5\% of women.\textsuperscript{1} Surgical treatment is indicated when conservative treatment has failed or in cases involving a large fistula. This is either through laparoscopy or laparotomy immediately after the diagnosis (in 48 hours) or 3–4 months after diagnosis.

The operation can be transperitoneal or retroperitoneal. Either repair of the uterus and the bladder with excision of the fistulous track; or total abdominal hysterectomy and repair of the bladder if the patient has completed her family; can be performed. A transvesical approach involving fulguration of vesical opening\textsuperscript{6} has also been described but with a high recurrence rate. The pregnancy rate after repair has been reported to be 31.25–37.5\% with a rate of term deliveries of 25\%.\textsuperscript{1,14}

Fistulae are prevented by meticulous practice of surgical principles at caesarean section with investigations for intraoperative haematuria, caudal retraction of the bladder, and identification of the anatomical landmarks with suturing.

As authors, we recommend that the management should be tailored by the size of the fistula. With small fistulae, conservative management is recommended with expectation of spontaneous closure in 4 months, while big fistulae need surgical treatment.

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\textbf{References:}


Impacted radio-opaque glass in the oesophagus of a child

Titus S Ibekwe, James A Fasunla, Moronke D Akinola, Onyekwere G B Nwaorgu

Abstract
Most ingested foreign bodies in the aero-digestive tracts in both children and adults are not radio-opaque, and as a result, a preoperative plain radiograph may not be helpful. However, incorporating radio-opaque markers into such potential foreign bodies like toys, beads, and dentures during manufacture (as illustrated in this case of a 6-year-old boy who ingested a piece of glass mirror which was easily seen on X-ray) would obviate this problem. While this may constitute some financial burden on manufacturers, the cost effectiveness on health management is non-negotiable. We suggest that appropriate authorities and regulatory bodies should enforce this through legislation.

Most toys and objects that stand the risk of being ingested by children are mainly non-radio-opaque. Most fish bones and dentures, which form the bulk of foreign bodies ingested by adults, are radiolucent. As a result, radiological identification of such foreign bodies and their localisation, unlike the radio-opaque ones, can pose a diagnostic and management dilemma.

It is thus the aim of this paper to highlight the need to encourage inscription of common non-radio-opaque objects that stand the chance of being ingested, with radiological markers.

Case report
A 6-year-old boy presented to our Accident and Emergency (A&E) Department with a 9-hour history of ingestion of a piece of glass while playing. There was no eye witness; however his mother noticed sudden drooling of saliva and pointing at the neck. No dyspnoea, bouts of cough, haematemesis, vomiting, or swelling of the neck was observed. Academic performance and behaviour at home did not suggest attention deficit/ hyperactive disorder (ADHD).

On physical examination he was drooling saliva but not in any obvious respiratory distress (respirations: 31 per minute). There was no pallor, fever, trauma to the oral mucosa, or cervical emphysema. The complete blood count, serum electrolyte, and urea were within normal limits.

The plain radiographs of the neck and upper chest (anteroposterior and lateral views) showed a triangular radio-opaque substance (Figures 1 and 2) within the oesophagus at the level of first to third thoracic vertebrae (T1 – T3). The laryngo-tracheal air column appeared normal.

An emergency rigid oesophagoscopy was carried out to retrieve the foreign body (a triangular piece of glass mirror measuring 4 ×3 × 2 cm) with a reflective surface and a coated dull reverse surface (Figures 3 and 4). There was no mucosal injury noted and postoperative management was uneventful.
Discussion

Foreign body (FB) impaction in the aero-digestive system is an emergency and as such requires prompt intervention. This is more commonly seen in extreme young and elderly ages, the mentally deranged, and some disease conditions of the aero-digestive tract.¹,²

A variety of FBs have been documented including unusual ones. In children, little toys as well as smooth and colourful objects are regularly ingested due to the explorative nature of children resulting in probing the orifices in their bodies.³
The mentally deranged ingest limitless varieties of foreign bodies. Most often, the history of FB in the throat among children is vague especially in the absence of eyewitness account of caregivers. As such, investigations like the simple plain radiograph become invaluable in the establishment of the level of impaction, especially in the developing World where sophisticated equipment is not affordable.

Metallic coins used to be a common FB encountered in children in Nigeria but economic conditions have led to their replacement with plastic and paper money. As a result, most ingested FBs are radio-luscent which poses a diagnostic challenge to the clinician who now depends on the level of air entrapment and increased prevertebral soft-tissue shadow alone to characterise/establish the level of the FB. These features as diagnostic tool for oesophageal foreign bodies have a low specificity.

Our patient was able to identify the object swallowed as a piece of glass which was confirmed by oesophagoscopy. However, common household glass is composed of 60–75% silica, 12–18% soda, and 5–12% lime and as such is radiolucent. This was why the radiopacity on the radiograph created diagnostic doubts.

A mirror is a silvery-coated household glass and in this case the silver nitrate coat acted as a “radiologic marker” thus aiding easy identification (via X-ray) of the level of impaction, and subsequent removal.

Therefore, inclusion of radio-opaque markers into objects like little toys, beads, and dentures during manufacture will aid their identification and removal when inadvertently swallowed or inhaled. This may constitute some financial burden on the manufacturers of such products; however, the health cost effectiveness is non-negotiable. We suggest that appropriate authorities and regulatory bodies should enforce this through legislation.

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What is the state of health services research in New Zealand?

Paul M Brown, Toni Ashton

Abstract
The article reflects on the current state of health services research (HSR) in New Zealand. A recent HSR conference held in Auckland highlighted a number of issues regarding the practice of HSR, suggesting that while there is some good research currently being conducted on issues pertaining to access, quality and costs of health services, more needs to be done. There is limited capacity in the public sector to conduct this work but barriers exist to commissioning academic and private researchers. Academic and private researchers have additional capacity, but often face constraints in producing policy-relevant research. We discuss ways that we might better coordinate and conduct HSR in the future.

Background
The 5th Health Services Research and Policy Conference was held in Auckland in early December. With over 230 delegates, it represented the largest gathering of health services researchers in Australia and New Zealand. There was much talk about the current state of health services research, including several panel discussions with researchers, funders, policymakers, and providers from both sides of the Tasman. So it would seem like an appropriate time to ask “What is the state of health services research in New Zealand?”

For those unfamiliar with the term, health services research (HSR) is concerned (broadly) with three areas: quality of health care and health services (including examining whether care is effective, timely, and appropriate); equity of access to care and the distribution of health gains; and the cost and financing of health services.

Two factors tend to distinguish health services research from other disciplines; health services researchers place greater emphasis on making their research relevant to policymakers and practitioners than is done in other fields, and results are often required quickly and can have a short life span. As a result, the research needs to be rigorous and of a high quality but researchers are often under pressure to ensure that the results are relevant and accessible to ‘stakeholders’ (be they providers, funders, policymakers, or the public) in a timely fashion.

The recent restructuring of the Ministry of Health is encouraging a greater focus on innovation and development within the health system. The health sector has also seen a rapid growth in the amount of data that is routinely collected about variables such as patient pathways and outcomes, utilisation of services, and costs of service provision. The opportunity to conduct health services research is therefore greater than ever, and the potential is there to make a significant contribution to the health sector and to patient outcomes. For instance, many of the quality of care issues arising from
mistakes or misadventures in hospitals and primary care practices might have been picked up had closure scrutiny been paid to the available data.

**Lessons from the HSR Conference**

So what is the status of health services research in New Zealand? Judging by some of the views expressed by participants at the conference, there are currently some significant barriers to undertaking and learning from health services research in New Zealand. One lesson to emerge was that personnel within the provider sector (e.g. district health boards [DHBs]) along with academics are mostly working in parallel rather than in collaboration. Each group seems to have its own agenda, with separate lines of dissemination.

While personnel from the sector were conspicuous by their absence from the health services research conference, few academics attended the DHB Research Fund Workshop on Innovation for Health in October 2007. While there is an increasing amount of commissioned work from DHBs, it has historically been piecemeal and somewhat disjointed.

A second lesson from the conference is that there are too few people on the ground able to do the work that is required by the sector. People who know how to translate routinely collected data into information that is useful to managers and planners are in short supply. Historically, much of this work was done centrally by the (one) Health Funding Authority or Ministry of Health. But as power has been devolved to the (21) DHBs, so too has the responsibility for conducting health services research.

While some DHBs have access to research units (such as decision-support units) which have the capability to do some of this work, their capacity is insufficient to examine issues of quality or access in depth.

There are, however, several barriers to expanding the workforce, including attracting our able and brightest away from clinical fields into health services research. But another lesson gleaned from the conference was that there are challenges in utilising research workforce capacity from universities and (to some extent) the private sector.

Panellists at the conference made it clear that policymakers and providers desire researchers who can provide high-quality analysis, and present the results simply (often on the topic de jour), very quickly (e.g. within a day), and confidentially. In other words…they want employees.

External researchers (particularly those from universities) might be willing and able to contribute, but they come with constraints. Academic researchers often produce results that are complicated and difficult to condense into a sound-bite, require time to do the research well, and will want to publish the results independently.

Unlike employees (who can be directed to frame their conclusions in line with their organisation’s strategic considerations), academics must guard against tailoring the conclusions lest they lose their credibility. In a political environment, agencies may find it difficult to ignore the published advice of their experts or risk being challenged in a public arena. This creates barriers to bringing in outside academics.

Which leads to a third lesson to emerge from the conference—the current structure of research funding does not foster or sustain the development of health services
research in New Zealand. Granted, it is not unusual for researchers to bemoan the lack of funding for their particular area of interest.

The twist with health services research is that funding cycles for agencies such as the Health Research Council—which are over 12 months from project development to potential funding—are often too slow to allow researchers to respond to topical issues (e.g. management and clinical relations in a DHB) in a timely manner. In addition, most research projects are initiated by the investigators themselves, often without much consultation or involvement with the broader community of clinicians, funders, and stakeholders. Furthermore, unlike other fields, a common response to applications for funding for health services research projects is “Shouldn’t the DHBs be funding this?”

Options for the way forward

So the current state of health services research might be summarised as follows:

There is good work being undertaken both internally and externally, but providers and funders do not have the capacity or funding to undertake health services research to a greater extent. While they might be interested in commissioning work from external researchers, they have no assurance that the results will be treated with confidentiality. External researchers, on the other hand, might be interested in working on topics that are of relevance to providers and funders, but may not know what topics are of high priority and may balk at tailoring their conclusions should the results prove controversial.

What is the solution? The setting up of the DHB Research Fund was an excellent first step towards addressing the funding of health services research. Although the quantum of funding is currently inadequate to address many of the pressing research issues, it does provide a funding mechanism to begin to examine issues of importance.

But the limitation of provider commissioned research (including the DHB Research Fund) is that there is no assurance that the research priorities will incorporate the views of parties outside the commissioning organisations or that challenging conclusions from the research will be received in a constructive manner. Thus, what is needed is a way to involve both external researchers and providers/funders to establish research priorities, to ensure that work is of a high quality, and to allow challenging research findings to be produced without creating a political dilemma.

In looking at other countries, there are several options for improving health services research in New Zealand. One option is to provide funding for research centres to undertake independent work in broad areas of concern to the government. For example, there are research centres in Australia that look primarily at issues of quality in hospitals. The centres have the freedom to examine issues that arise (such as medical errors in a particular region) and have independence in publishing their findings but are expected to produce work that contributes to the public good. This allows the government some degree of separation from the research results (e.g. if the results do not reflect government objectives or priorities) but it requires a degree of confidence on the part of the government in the expertise and sensitivity of the researchers. More importantly, it also requires confidence that the centres will choose topics that are of interest to the government and the public.

The option described above was (not surprisingly) the preferred option of conference attendees. But another option that was discussed is for the research community to
work with policymakers, funders, and providers to set up an independent body to identify the most important topics of the day and provide clear and independent advice (such as done by the King’s Fund in the UK).

In some cases, simply identifying the priorities would be sufficient as work could be conducted with routinely collected data (provided researchers are granted access). And in cases where the priorities led to research being commissioned, the body could act as an independent source to ensure that the results are of a high quality and challenging conclusions can be placed in context.

Both options have merits and limitations. But a clear theme that emerged from the conference is that, although there is much good health services research currently being undertaken in New Zealand, it is fragmented and of limited scope. And while there are signs that the health system is beginning to recognise the limitations that currently exist, the situation will not improve without positive action and greater collaboration between researchers and stakeholders within the sector.

Competing interests: None known.

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Gangrenous foot


The next case which I wish to mention to you is of interest only on account of its rarity.

On October 10th., 1907, I saw in consultation with Dr. Watson, of Bulls, a woman, aged about 32, who a fortnight before had been attended in confinement by a slatternly nurse of the type that those of us who do country work have learned to love. Any history previous to Dr. Watson’s first visit on or about 6th. October is under the circumstances unreliable.

Dr. Watson found her suffering acute pain in the region of the right sacro sciatic notch; her temperature was high, her pulse rapid, and she had some abdominal tenderness, and an offensive vaginal discharge.

Next day, phlegmasia dolens had developed in the right leg, and she had severe pain over the left sacro sciatic notch, and on the same evening agonizing pain, unchecked by morphia, over the dorsum and outer side of the left foot, with coldness of the toes. The pain in the foot continued next day, when the toes and dorsum of the foot became dusky in hue, and later on anaesthetic.

When I saw her she had well-marked gangrene of about the anterior half of the foot with no distinct line of demarcation. For a few days it appeared as if the circulation was going to be re-established, as the dusky hue became paler, and in spots sensibility quite definitely returned. Upon 22nd. October, however, a well marked line of demarcation developed and extensive blistering occurred, and on the 23rd. I amputated the foot by Symes’ method.

Union occurred by first intention, and in four weeks the “white leg” had disappeared. The rarity of gangrene following septic infection is my reason for mentioning this case.
Acute lower gastrointestinal bleeding: multidetector-row CT findings
Kuei-Feng Tsou, Wei-Chou Chang, Shih-Hung Tsai, Ching-Feng Chang

Clinical
A 32-year-old man presented with rectal bleeding. On examination he was tachycardic and normotensive, with a haemoglobin of 8.6 g/dL. The patient was treated with a blood transfusion. The emergency colonoscopy failed to localise the bleeder due to active oozing with a large amount of fresh blood clot accumulation in the rectum. Multidetector-row CT (MDCT) was performed (Figures 1A and 1B).

What is the abnormality and what are the treatment options?
Answer

The CT shows a focal area of high attenuation, which represents arterial bleeding with the extravasated contrast (Figure 1A and B, white arrow) over the distal rectum. Selective right iliac arteriogram confirmed that the active bleeding with contrast extravasation was from the lateral sacral artery (Figure 2, white arrow indicates active bleeding), while selective inferior mesenteric arteriogram showed no evidence of active bleeding.

Bleeding ceased after embolisation of right lateral sacral artery by gelform pieces. The patient made an uneventful recovery and discharged on the sixth hospital day. At a 1-month follow-up, there is no sign of bowel ischemia or recurrent bleeding. A benign healed rectal ulcer at the rectum was seen on the follow-up sigmoidoscopy.

Figure 2. Selective right iliac arteriogram showed a serpiginous arterial branch supplying the area of contrast extravasation (white arrow). The active bleeder was lateral sacral artery

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Portomesenteric venous gas and pneumatosis intestinalis

Selim Doganay, Mecit Kantarci, S Selcuk Atamanalp

Hepatic portal venous gas (HPVG) was first reported by Wolf and Evans in 1955 in infants with necrotising enterocolitis. It is more common in neonates than adults. The first adult case was reported in 1960 by Susman and Senturia.

There are two types of pneumatosis intestinalis: primary pneumatosis intestinalis (15% of cases) and secondary pneumatosis intestinalis (85%). The cause of primary pneumatosis intestinalis is idiopathic. Intestinal ischaemia/infarction, intestinal trauma, intestinal obstruction, infection, inflammation, and chronic obstructive bronchopulmonary disease are the causes of secondary pneumatosis intestinalis. Primary pneumatosis intestinalis is usually asymptomatic and the prognosis good whereas the prognosis for secondary pneumatosis is relatively poor.

The patient whose images are illustrated below was a 23-year-old man who was admitted to hospital because of acute continuous abdominal pain, increasing abdominal distension, and gross rectal bleeding. He was known to have chronic renal failure for 3 years.

Figure 1

Figure 2
An axial-plane computed tomography (CT) scan revealed bubble-like pneumatosis and band-like pneumatosis in the wall of small bowel and stomach (arrows) (Figure 1), although there were distension of small bowel, bowel wall thickening, and mesenteric oedema.

A coronal oblique-plane CT angiography scan shows gas in portomesenteric venous system (P) (Figure 2), total occlusion of celiac truncus, and partial thrombosis of superior mesenteric artery.

After a day, the patient died, probably due to disseminated intravascular coagulation.

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Cannabis smoking and periodontal disease

Periodontitis is one of the most common chronic diseases in adults; it is bacterially mediated inflammation that extends deep into the tissues, causing loss of supporting connective tissue and alveolar bone. Tobacco smoking, through its systemic effects, is recognised as the primary behavioural risk factor for the condition.

A prospective study of a cohort over a 32-year period is reported as showing that cannabis smoking may be a risk factor for periodontal disease that is independent of the use of tobacco. A pleasure to see a paper from Dunedin published in *JAMA*.


Is the Medical Course in New Zealand too long?

In the context of the shortage of junior doctors, one could speculate on their length of training. At 6 years we lead (or trail) the pack. In Australia it is 5 years and in North America usually 4 years. Furthermore in Canada there is an ongoing 30-year natural experiment, where 2 Canadian universities, namely, McMaster University and the University of Calgary, opted for a 3-year curriculum while all other medical schools retained the customary 4 years of training. In contrast to many US schools, Canada’s two 3-year schools have not condensed 4 years of classes into 3 by teaching on Saturday and across summer.

Those trained in 3-year programmes do not appear to be any less competent than graduates of 4-year programmes. Licensing authorities have not signalled any concerns about inferior test scores, at least not publically. Food for thought.

*CMAJ. 2008;178:11.*

“Practising medicine without a licence”

Who said that? It was the editor of the *NEJM* some 10 years ago (NEJM 1997;336:1747). He was complaining about the continuing intrusion of legislators into the practice of medicine. Such interference is becoming a worldwide phenomenon and the editor of the *Medical Journal of Australia* has resurrected the expression in a recent editorial. He goes on to say health is a top priority for governments, and politicians increasingly dictate how health care is delivered in hospitals and the community. In this process, politicians assume the mantle of “de-facto doctors”.

Their past treatments have included: an intentional reduction of hospital beds, fuelling the current chaos in our emergency departments; the capping of medical school places, causing our present chronic dependence on overseas-trained doctors etc. He is referring to Australia of course, but it has a familiar ring.

Idraparinux—safer than warfarin?

Patients with atrial fibrillation at risk for thromboembolism are often anticoagulated with warfarin. Such treatment is tedious and places the patient at risk of haemorrhage. Idraparinux is a synthetic pentasaccharide that specifically inhibits activated factor X. It is given by subcutaneous injection once a week and requires no coagulation monitoring. Excellent, but is it effective and safe?

Well yes and no. In this randomised trial involving 4576 patients it was shown to be at least as good as warfarin in stroke prevention. But the trial was stopped because of excess of clinically relevant bleeding in the idraparinux arm.

The search for a safe anticoagulant continues.


Antioxidant supplementation and cancer prevention?

The high levels of antioxidants (particularly beta carotene and vitamin E) in fruits and vegetables are believed to contribute toward cancer prevention, possibly by inhibiting oxidative stress.

The authors of this paper doubted the validity of this proposition. Hence, a systematic review of the literature—12 trials were reviewed. In addition to beta-carotene and vitamin E, other popular antioxidant candidates (selenium, zinc, and vitamin C) were considered.

Their conclusion was that antioxidant supplementation, particularly with beta carotene and vitamin E, does not reduce primary cancer incidence or cancer mortality. Beta carotene supplementation might increase the risk of smoking-related cancers, as well as cancer mortality, and should be avoided by tobacco users. Selenium supplementation might reduce cancer incidence, especially in men.

Alcohol and breast cancer

Jennie Connor has provided a timely and comprehensive review of the widespread harmful effects of alcohol on both society and the individual in her recent NZMJ editorial entitled *The knock-on effects of unrestrained drinking*.\(^1\) In addition to the numerous problems listed, alcohol is also a carcinogen that increases the risk of many cancers including cancer of the mouth, pharynx, larynx, oesophagus, colo-rectum, liver, and breast. These findings were reaffirmed in a recent report from the World Cancer Research Fund (WCRF) entitled *Food, Nutrition, Physical Activity and the Prevention of Cancer* released in October 2007.\(^2\)

Numerous studies have identified alcohol as a cause of breast cancer in particular.\(^3^-^5\)

Proposed mechanisms include a genotoxic effect of acetaldehyde which is the main metabolite of ethanol, alterations in folate metabolism and raised levels of oestrogen secondary to liver damage.\(^6\)

A study in the International Journal of Cancer reports an increased risk in women who drink alcohol while also taking hormone replacement therapy.\(^7\)

A study published in March 2008 suggests that recent alcohol consumption may be associated with increased breast cancer risk in young women.\(^8\)

The use of this information on alcohol as a significant and modifiable risk factor for cancer should be used in harm minimisation approaches to discourage excessive alcohol consumption and should be included in public awareness campaigns and individual consultations. This is particularly appropriate in New Zealand in terms of achieving the objectives of the *New Zealand Cancer Control Strategy*.

Recommendations from the WCRF are that men should consume no more than two units of alcohol a day and that women should limit consumption to no more than one unit a day.\(^2\) These levels are significantly less than the quantities currently recommended by The Alcohol Advisory Council in New Zealand.\(^9\)

Achieving these lower levels will require a significant shift in public attitudes, but could have a major positive impact on health and wellbeing as well as cancer statistics.

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References:

An update on tobacco smoking among New Zealand health care workers, the current picture, 2006

Smith and Leggat requested—via their letter published in the 12 October 2007 issue of the NZMJ—that information on smoking and health care workers (HCW) be analysed and disseminated from more recent national Census data. They highlighted the importance of updating trends in smoking among HCW in New Zealand (from as early as 1963) given that there have been two national Census’s since 1996.

There has been no specific survey of health care workers’ smoking habits in New Zealand since 1981. The Census allows this information to be derived, but using various data sources diminishes the comparative value of interpreting trends in time. The information presented in this paper provides a cross-sectional indication of the “current” picture of smoking amongst HCW in New Zealand. These questions were omitted in the 2001 Census, hence data are not presented from that year. These data are not strictly comparable to the earlier survey estimates so the time trend that emerges is a general (rather than detailed) picture of what has happened over time.

We analysed data on smoking behaviour and HCW from the 2006 Census of Population and Dwellings, which provides the best estimates of smoking rates in New Zealand. HCW have been placed into three categories: health therapy professionals (including pharmacists, dental practitioners, physiotherapists, etc); medical practitioners; and midwifery and nursing professionals. Further information around occupational coding can be found elsewhere.

<table>
<thead>
<tr>
<th>HCW type</th>
<th>Regular smokers</th>
<th>Ex smokers</th>
<th>Never smoked regularly</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>female</td>
<td>male</td>
<td>total</td>
</tr>
<tr>
<td>Health therapy professionals</td>
<td>5.6%</td>
<td>6.1%</td>
<td>5.7%</td>
</tr>
<tr>
<td>Medical practitioners</td>
<td>2.6%</td>
<td>3.9%</td>
<td>3.4%</td>
</tr>
<tr>
<td>Midwifery and nursing professionals</td>
<td>13.2%</td>
<td>19.5%</td>
<td>13.6%</td>
</tr>
</tbody>
</table>

Source: Census 2006.

Midwifery and nursing professionals have the highest rate of smoking across the three groups; males have higher rates than females in all three groups. The prevalence of male ex smokers among medical practitioners and midwifery and nursing professionals is higher compared to female ex smokers—reflecting historically higher male smoking rates.

Smoking rates are highest among people identifying with Māori and Pacific peoples in all three HCW groups. This is consistent with higher smoking rates in these two ethnic groups in the wider population—reflecting inequalities in the use of tobacco and the corresponding burden on health outcomes. Of note also are higher smoking...
rates among health therapy professionals and medical practitioners identifying with Middle Eastern/Latin American/African (MELAA) ethnic groups.

Table 2. The prevalence of regular smoking health care workers (HCW) in New Zealand (15+ years), by ethnic group

<table>
<thead>
<tr>
<th>HCW type</th>
<th>European</th>
<th>Māori</th>
<th>Pacific*</th>
<th>Asian</th>
<th>MELAA</th>
<th>Other ethnicity</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health therapy professionals</td>
<td>5.5%</td>
<td>15.3%</td>
<td>9.7%</td>
<td>4.3%</td>
<td>8.8%</td>
<td>4.5%</td>
<td>5.7%</td>
</tr>
<tr>
<td>Medical practitioners</td>
<td>3.1%</td>
<td>8.8%</td>
<td>8.3%</td>
<td>3.0%</td>
<td>7.3%</td>
<td>4.0%</td>
<td>3.4%</td>
</tr>
<tr>
<td>Midwifery and nursing professionals</td>
<td>13.9%</td>
<td>30.7%</td>
<td>17.9%</td>
<td>3.4%</td>
<td>6.3%</td>
<td>12.0%</td>
<td>13.6%</td>
</tr>
</tbody>
</table>

Source: Census 2006
Ethnicity (total response) includes all the people who stated each ethnic group. Where a person reported more than one ethnic group, they have been counted in each applicable group

*Mostly of Samoan, Tongan, Niuean, or Cook Islands origin; MELAA=Middle Eastern/Latin American/African.

There are limitations to the data worth noting. The Census asks two questions to derive ‘current’ and ‘ever’ smoking status, and no further information around smoking behaviours can be inferred other than smoking status at the time of the Census.

Between 1996 and 2006 there was a change from use of the New Zealand Standard Classification of Occupations 1999 to the Australian and New Zealand Standard Classification of Occupations (ANZSCO) for occupational coding. These changes hinder higher level comparisons of occupation, although not at the level that we have used here.

In conclusion, the rate of smoking among HCW in New Zealand is not high, and it is encouraging that such a high number of HCW have never smoked regularly in their lifetime—a shift within a generation.

The particularly low rates of smoking among medical practitioners shows what is “possible”, even under the current tobacco control environment. We contend that it reflects, at least in part, the intimate knowledge that doctors have of the huge health risks to smokers, half of whom die early as a result of their smoking. This emphasises the obligation on medical practitioners to share this knowledge, to treat smoking as a vital sign, and to provide evidence-based interventions to help smokers quit.

Finally we propose that any future analyses of smoking HCW include health therapy professionals who play an internal role in the delivery of healthcare and in influencing both smoking initiation and cessation.

We trust that the public dissemination of this information will be useful.

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References:


“We must discuss suicide”

Students of the disciplinary processes invoked when doctors err may be inclined to think that from time to time these processes are expensive, unfair, tedious, tardy, and secretive. Many of the reports handed down after the enquiries made by the Health and Disciplinary Commissioner are so steeped in anonymity that they lose most of their impact and they rarely make financial recompense for injuries sustained.

When several people, or maybe a whole hospital, are involved in a bad event, the episode becomes a “systems failure”. Nobody gets named and nobody other than the patient gets hurt. If just one person can be identified as the practitioner whose actions had an unfortunate outcome, and the problem passes from the Health and Disability Commissioner to the Medical Practitioners Disciplinary Tribunal, the presumed offender can expect the authorities to drop on to him or her like a ton of bricks.

The case of Dr John Angus Marks, a psychiatrist charged with professional misconduct, was reported by the Tribunal in the New Zealand Medical Journal on 14 March 2008. The events under scrutiny occurred in 1999, and the penalties ordered by the Tribunal were appealed in the District Court, where the appeal was partially successful.

Dr Marks qualified in Edinburgh in 1972, and had been admitted as a FRANZCP in 1998. He was “assigned” the management of a high-risk patient who committed suicide, and in effect it was he who got the blame for that, it being held that he failed the patient in a number of ways.

The Director of Proceedings is a government appointee, Ms Theo Baker. Nobody other than Dr Marks was named in the written decision. Thus we do not know the composition of the Tribunal, nor the names of Dr Marks’ supervisor and the two expert witnesses called by the Tribunal. The Tribunal fined Dr Marks $5,000 and, according to an item on the website www.accforum.org, his costs came to almost $44,000. The District Court in Wellington cancelled an order requiring the doctor to pay for his own supervision for a period of 3 years.

A number of recent news items show that suicide is common and that it is not being well handled. There has been a slight decline in the New Zealand statistics in recent years, but they are high by international standards. The figures were almost unchanged from 1960 until 2000, and I am personally of the view that psychiatric interventions don’t do much good. Several investigations into suicides and a presumed suicide are pending.

The message for the GPs is clear. If a patient so much as hints at suicide, write out a note, hand it to a friend or relation, and tell them to take the patient to the nearest hospital. The staff will do what they can, but, as the figures show, success in the prevention of suicide is by no means assured.

Roger M Ridley-Smith
Retired GP
Wellington
Response

Dr Ridley-Smith suggests that: i) “suicide is common”; ii) “it is not being well-handled”, and iii) “psychiatric interventions don’t do much good”. These pessimistic views are at odds with current international evidence and efforts in suicide prevention.

First, suicide is not common. New Zealand’s suicide rate (13.1 per 100,000 in 2005 the year for which most recent data are available) is in the mid-range for comparable OECD countries, for which suicide rates range from the low rates observed in Greece (2.9, in 2004) and Italy (5.6) to the highest rates of 20.3 in Japan and 18.7 in Korea.

Worldwide, suicide rates, as reported to the World Health Organization, are highest in Eastern European countries including Lithuania, Estonia, Belarus, and the Russian Federation. These countries have suicide rates of the order of 45 per 100,000—far higher than New Zealand’s suicide rate.

Second, suicide is being addressed in New Zealand in ways similar to those adopted in countries with which New Zealand is typically compared in terms of medical, public health, and social issues. The challenge in preventing suicide in all countries is that there are multiple causes of suicidal behaviour and no single, readily identifiable high-risk group accounting for the majority of suicides. This implies that many different types of programmes and activities are needed to prevent suicide, with each focused on addressing specific areas of risk. This complexity stands in contrast to the limited causal factors involved in road traffic crashes, for example, where efforts to reduce deaths by focussing on just two factors, speed and drink-driving, have been remarkably successful.

To address suicide prevention, New Zealand has developed a national suicide prevention strategy, and under this framework is developing a series of specific evidence-based suicide prevention programmes. These programmes include efforts to:

- Restrict access to means of suicide;
- Enhance training, recognition, assessment, treatment, and management of depression by medical practitioners (particularly in primary care);
- Increase public awareness of depression by public programmes;
- Improve assessment, treatment, and follow-up care of people who make suicide attempts; and
- Enhance access to mental health services and improve the care of people with serious mental illness.

These approaches are based on evidence of effective reductions in suicide from similar programmes (Beautrais et al 2007; Mann et al 2005).

Thirdly, there is evidence that some interventions with psychiatric patients are effective in reducing suicide. These include efforts to restrict access to means of suicide; psychosocial interventions with people who have made suicide attempts; psychopharmacological approaches, including lithium for patients with bipolar disorder and antipsychotics such as olanzapine; cognitive behavioural therapy for
those who have made suicide attempts, and dialectical behavioural therapy with patients with borderline personality disorder; the better recognition, treatment, and management of depression; and ‘chain-of-care’ approaches to support people discharged from psychiatric care (Hegerl et al 2008; Mann et al 2005).

Dr Ridley-Smith also suggests we must discuss suicide. There is no good reason to believe that the mere discussion of this complex problem can prevent it, and an increasing volume of evidence to suggest that incautious publicity about suicide is inadvisable. More than 50 studies now provide evidence that incautious media reporting of suicide is associated with increased risk of suicide in vulnerable individuals, and the impact of public messaging about suicide is unknown (Chambers et al 2005).

Finally, however, more can, and must, be done, not only to prevent suicide in patients in psychiatric care, but more widely. Although New Zealand has now developed a sound and evidence-based suicide prevention action plan, the extent to which this plan will be effective will depend on the adequacy of policy implementation.

Current international guidelines suggest that such implementation is likely to be most effective if it is led by an expert led consensus based approach rather than being controlled and led by Government agencies (United Nations, 1996; World Health Organization, 1993).

At the present time, the model being used in New Zealand appears to involve the Ministry of Health putting out various components of the action plan for tender. This approach is not generally consistent with best practice recommendations for the implementation of suicide prevention strategies and it remains to be seen how successful a tender-based approach to policy implementation will be.

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References:


Barefoot running and walking: the pros and cons based on current evidence

In response to the recent debate on barefoot running and walking of children published in the New Zealand Herald we have put together an argument relating to the pros and cons based on current evidence.

There is very limited evidence specifically relating to barefoot running and walking in children. One study from Germany reported that the increased prevalence of flatfoot and hallux valgus (bunions) in modern societies may be the consequence of inadequate footwear in childhood. The German study postulated that barefoot walking represents the best condition for the development of a healthy foot.

Walking and running on different types of surfaces such as grass, sand, and artificial running tracks may indeed enhance healthy foot development. However, the problem of barefoot walking on hard surfaces such as pavements may alter the biomechanics of walking and running. This may lead to potential arthritic changes and consequently a reduction in foot function.

A more worrying concern is the impact of obesity and overweight on children’s feet. A recent study from New Zealand suggested that three lifestyle risk factors related to obesity: low physical activity, skipping breakfast, and insufficient sleep on weekdays.

To prevent children undertaking physical activity may exacerbate a major issue already within New Zealand. A recent study from Australia suggests that the function of the arches of the feet in overweight and obese children may change and this might worsen if excess weight impacts on the foot throughout childhood and into adulthood.

Another problem to address relating to barefoot walking is the issue of children with diabetes. The long-term complication of diabetes on the foot includes infection, ulceration, and a loss of peripheral sensation. A UK study by Karabouta found over 50% of adolescents with Type 2 diabetes had peripheral neuropathy and weak posterior tibial pulses. The authors recommend that all children with Type 2 diabetes need podiatric surveillance for complications from the time of diagnosis. A study from rural Australia suggested that walking barefoot is a risk factor for diabetic foot disease.

It is interesting to note that certain types of footwear may cause injuries in children. A study from Ireland showed that there was an increasing trend in orthopaedic injuries using Heelys and Street Gliders. Another study using a cloth sport shoe showed inferior cushioning capability but the same lateral stability as the other sports shoes for children. However, a study from Germany reports shows that slimmer and more flexible children's shoes do not change foot motion as much as conventional shoes and therefore should be recommended for children of all ages.
Painful feet in children are often caused by flatfeet or mechanical instability of the arches of the foot. A recently conducted New Zealand review on children’s shoes found no evidence to support the suggestion that different types of footwear reduced pain in children’s flatfeet.9

Finally, a study from Australia found significant structural differences between the feet of European and Australian children.9 The German children displayed significantly longer and flatter feet relative to their Australian counterparts, whereas the Australian children reveal a significantly smaller ball angle, implying that the forefoot of the Australian children is squarer in shape.

These findings imply that footwear must be designed to cater to the unique foot dimensions of children in different continents to ensure that shoe shape matches foot shape. Most footwear companies do not vary the dimensions of their shoe lasts to accommodate intercontinental differences in foot morphology based on racial and/or environmental factors. The results of this study will have immediate implications for the design of comfortable footwear suitable for the developing feet of children.10

In summary, further research is required in this area.

Keith Rome
Professor of Podiatry

Dene Hancock
Senior Lecturer

Daniel Poratt
Head

School of Podiatry
Division of Rehabilitation & Occupation Studies
AUT University, Auckland

References:


Arthur Newton Talbot

9 September 1917–2 February 2008; Mb ChB, DO, FRACS; Captain 2nd NZEF, NZMC, WW2. No. 299280.

Arthur was born in Timaru, the second son of Leonard Talbot who became the Eye and Ear Nose and Throat (ENT) Surgeon at Timaru Hospital.

Arthur attended Timaru Boy’s High School, before proceeding to Dunedin to attend the Medical School, and reside in Selwyn College.

After graduating, Arthur worked as a house surgeon in Wellington and Timaru in 1940 and 1941, before enlisting in the army to join his father and elder brother who had preceded him.

Arthur used to say that that the 3 Talbots were recognised in the army as the father, son, and holy ghost! (Arthur being the latter).

Initially in the islands campaigns, Arthur found time to return briefly to New Zealand and marry Margaret Mitchell in 1944, before being posted to Seniglia, Italy to join the New Zealanders during that intense battle up Italy to Florence and the final German capitulation.

After the war Arthur did postgraduate study in Melbourne, achieving his diploma in ophthalmology, and completing the FRACS, before coming to New Plymouth with his family in 1947 to the post of Eye and ENT Specialist for Taranaki. He was relieved of the latter field in 1954, and pursued ophthalmology until his retirement.

In his work Arthur showed meticulous precision and dedication, and was held in high regard by his numerous grateful patients, while with his colleagues his high ethical understanding held the respect of all. He was particularly considerate to newcomers at the Hospital, and could be prompted to offer an impecunious newly arrived medical family the use of his bach at Mokau, or give his advice and sympathy to a fellow specialist in some of those difficulties which beset the beginner in a surgical field.

He was on the committee of the Foundation for the Blind and for a period its President, and served on the National Committee of the New Zealand Ophthalmological Society, serving as the President in 1972. On a less serious note he was renowned for a puckish sense of humour and had a fund of stories which could brighten the most dreary of occasions. This gift never left him, and only 3 months before his death he could still find an up-to-date, slightly risqué joke, on the current political situation.
He was very involved in the community at large, being a Rotarian, and part of the Mt Egmont search and rescue team for many years. Many injured trampers and climbers remained forever in Arthur’s debt, although his innate modesty kept him out of the limelight on such occasions.

He enjoyed the outdoors and was a member of the Pukeiti Rhododendron Trust, but his favourite activity would have been fresh and saltwater fishing. He was an expert in either medium, and was generous enough to share his knowledge of either the Taranaki rivers, or the Tongariro, with anyone who was prepared to accompany him and try to match his qualities of patience and enthusiasm. On a Saturday morning he might arrive late for his ward round, but the boot of his car would contain several beautiful brown trout, taken in the local streams before his Hospital visit.

During the financially and surgically difficult 1970s, Arthur was Chairman of the Iona Private Hospital, which staggered along on a shoestring budget, without the current benefit of the many Medical Insurance subsidies which support such institutions today. He spent hundreds of unpaid hours as the virtual ‘superintendent’, overseeing the day-to-day running, and at any medical assembly proved a forthright outspoken advocate of the need for his colleagues to keep up their support for the continuance of both private and public hospital systems.

Thanks to his efforts his successors inherited a viable Hospital, which could to be taken forward to reach the vital place it has in Taranaki health today, and it is a great legacy of his hard work.

Arthur’s wife Margaret died in 1998. He is survived by his 3 children: Jim, Brian, and Barbara; 10 grandchildren; and 5 great-grandchildren.

Dr Victor Hadlow wrote this obituary and the Taranaki Daily News supplied the photograph.
David Whitney Cannings Dove
9 August 1928—11 December 2007

David Dove was the quintessential GP. Born in Auckland he received his early education at Kings Preparatory school and Kings College. His father felt that he should study medicine in the UK, something that he himself had wished to do, but was unable because of the war. David obtained a place at Trinity College Oxford where he did his pre-clinical studies before proceeding to St Thomas’ London for the clinical component of his degree. He graduated MA. BM.BCh. Oxon in 1958.

While at Oxford he made a name for himself as a middle distance athlete and was acting as an official when Roger Bannister broke the 4 minute mile.

After graduation there followed a spell in general practice in West Malling, Kent before returning to New Zealand with his dearly loved English wife, Cynthia, who he had married in 1955.

Together with their first two children they travelled to New Zealand in 1960 with David serving as ship’s doctor.

In the Pacific the ship had to divert to Pitcairn Island where he successfully treated a displaced shoulder.

Shortly after his return to New Zealand, he bought the practice of Dr Bill Harrison of Remuera and he remained there until his retirement in 1990. In 1966 he obtained the Auckland Diploma of Obstetrics and practiced obstetrics for many years.

He was a foundation Member of the NZ College of General Practitioners and was made a Fellow in December 1982. David was outstanding in that he epitomised the traits that mark a truly well rounded family doctor.

When general practice teaching was introduced at the Auckland School of Medicine, David was one of the first to offer his assistance. He soon became the most popular student preceptor and accepted an enormous number of student attachments.

This was no accident. Students could recognise quality and appreciated the time that he was prepared to devote to helping them develop and put into use a philosophy of practice. They could also see that David was dedicated to the task of assisting those in need of help both physically and emotionally.

He had an uncanny ability to understand the human condition and recognise the subtle interplay of mind and body. He had a natural skill in identifying the hidden tensions in the home life or work of his patients and how those tensions could trigger or
aggravate impairment of their health. Moreover, he recognised the importance of family and showed great wisdom in helping his patients to cope with the crises that occurred within their families. Above all he showed great empathy with, and great compassion for all his patients. He was always generous with his time and if he felt it necessary, counselled his patients and their families well into the night.

Many medical students have been inspired by David and today aspire to emulate his approach to general practice. Not a few have decided to follow a career in general practice as a result of learning from him the challenges and satisfactions which exist in this discipline if one troubles to look below the surface of the presenting problems.

Out of the early days of academic general practice a small group of interested doctors and their spouses came to meet regularly to discuss issues surrounding the care of patients and provide some measure of support for themselves. I had the good fortune to be part of that group from its inception and it still meets. David has been the de facto leader of the group and we all learnt much from each other, but particularly from David.

Latterly his perceptions and acceptance of his final illness was a lesson in itself.

He never sought the public eye but on those occasions when he was asked to address a gathering could command their attention and speak eloquently from his life experience.

All was not hard work, however, and for some 20 years David had a launch, appropriately named Isis, which gave the family much pleasure. When he sold this he acquired a Farr 10.20 yacht which he taught himself to sail, becoming a competent yachtsman who loved the sea.

He did not neglect his family life and greatly valued time spent with his four children Sarah, William, Alan, and Michael. There was also the family cottage at Mahurangi where David, Cynthia, and family spent many restful holidays. We extend our deepest sympathy to Cynthia and family in their loss. He will be sadly missed.

John Richards (Retired Associate Professor of General Practice) wrote this obituary.
Henry Keith Watt

Former general practitioner of Milford (North Shore); born 1923; qualified MB ChB at Otago in 1949; died 24 March 2008 of a cerebral haemorrhage.

Keith was educated at New Plymouth Boys’ High School and Takapuna Grammar School. After his house surgeon years in Auckland Hospital and a year in Rotorua Hospital as a registrar, during which time he became interested in (and proficient at) anaesthetics, he moved to Milford with his wife Gwen, whom he had married in 1951.

In 1953 when his father retired from his Milford practice Keith took it over.

His general practice grew as the population on the North Shore expanded, especially after the completion of the Harbour Bridge. By 1985 the group consisted of five doctors with special interests in anaesthetics, musculoskeletal medicine, obstetrics, and diving medicine.

As well as his general practice, Keith provided an anaesthetic service to the RNZN Hospital in Devonport, the Lister Hospital in Takapuna, and the North Shore Obstetric Hospital. Eventually, with the opening of North Shore General Hospital the appointment of fulltime specialist anaesthetists replaced the service supplied over the previous 40 years by Keith and his fellow general practitioner anaesthetists.

Despite a potentially fatal waterskiing accident in 1967 with several complications during his convalescence, he recovered to return to fulltime practice as if nothing had happened. After 42 years in the practice he retired in 1995.

Keith was a reserved dignified man; as colleagues and friends we appreciated his logical thinking, his integrity, his dry sense of humour, and his equable temperament. He was always prepared to appreciate the other person’s point of view. During the 32 years I worked with him I never heard him raise his voice in anger or lose his temper.

He had several interests apart from his medical practice. He was a keen and accomplished skier, and after several years as an enthusiastic member of the committee of Ruapehu Ski Club, he served as President from 1980 to 1983. His other main interests were tramping, woodworking, and his large garden which he maintained meticulously until shortly before his death. His three grandchildren and their activities were a constant source of interest and pleasure.
Keith is survived by Gwen, his wife for the past 56 years; his children: David and his wife Wai-I and Margaret and her husband Doc; and his three grandchildren, Mark, Alice, and Amy.

Dr Murdoch M Herbert, Keith’s colleague at the Milford general practice from 1964 to 1995, wrote this obituary with the help of Keith’s wife, Gwen.
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Pickerill: Pioneer in Plastic Surgery, Dental Education and Dental Research


Henry Percy Pickerill was a remarkable man. This book by Harvey Brown, a former editor of the New Zealand Dental Journal (1975–2002), describes in some detail Pickerill's life from his upbringing in Hereford to his death in 1956. Pickerill was a complex, energetic, intelligent, and talented man.

The author tells us of Pickerill’s private life, his pioneering research, and his wartime service as a plastic surgeon. During the First World War, Pickerill was the Officer in Command of the New Zealand Section at The Queen’s Hospital at Sidcup. He and a number of other surgeons were responsible for rebuilding the faces of the many young soldiers injured in the war. It was inevitable that many young men would need further facial surgery by Pickerill when they returned home. Pickerill established a section in Dunedin for this purpose.

Pickerill resigned from his positions as Dean and Director in 1927, left his family in Dunedin and moved to Sydney to became the Australia's first full-time plastic surgeon. Shortly after, he married his former house surgeon. When the Pickerills returned to New Zealand a few years later, they established a small private hospital in Lower Hutt, which for a time served as the national centre for treatment of babies with clefts and other major congenital conditions. The fact that Cecily (his second wife) did not receive any recognised training in plastic surgery, and Pickerill's promotion of mother nursing of babies (which cut the prevalence of cross-infection) caused some conflict with the medical profession.

The book has 20 chapters, 24 pages of black-and-white illustrations and a comprehensive list of references at the rear. Harvey Brown has done an excellent job sifting through the documents, and infusing the book with much interesting detail on the life and work of a controversial and undervalued man. I strongly recommend this book to anyone interested in the history of plastic surgery, maxillofacial surgery, and dentistry in New Zealand.

Michael Harkness
Editor, Australian Orthodontic Journal