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Prehospital thrombolysis

Harvey D White, Director of Coronary Care and Cardiovascular Research, Green Lane Hospital, Auckland.

New Zealand is a country with many rural communities, some at considerable distance from a base hospital. Twenty New Zealanders die from acute myocardial infarction every day, making it one of our commonest causes of death. It is well established that thrombolytic therapy reduces mortality and helps to preserve left ventricular function, but its efficacy attenuates markedly the longer that treatment is delayed after the onset of acute coronary occlusion.1-2 If patients receive treatment within the first (or ‘golden’) hour, the risk of mortality can be halved,1,3-4 but for every hour of delay, 2-5 lives are lost per 1000 patients treated.1,4,5 Patients further than one hour’s travel from hospital therefore have much to gain from prehospital thrombolysis.

In this issue of the Journal, Nunn and colleagues describe a meticulous pilot study of prehospital thrombolysis by general practitioners (GPs) in the Coromandel region. Comparison with a control group of patients from the same area who received thrombolysis in hospital between 1993 and 1998 showed that prehospital thrombolysis halved the delay from symptom onset to treatment (135 minutes versus 270 minutes), with no arrhythmias during transport and no hospital deaths. This was not a randomised study, but it shows that prehospital thrombolysis is logistically possible in this country, and results in markedly shorter ‘pain to needle’ times.

GPs in other parts of New Zealand (eg, Takaka, Westport, Queenstown and Wanaka) have also had experience in prehospital thrombolysis, using treatment algorithms tailored to local needs.

In the Coromandel study, patients were given thrombolytic therapy and then immediately transported to hospital by rescue helicopter or ambulance (without Advanced Care Officers). Elsewhere, patients have been given thrombolytic therapy and then observed for one hour before being transferred to hospital, or were accompanied by the GP during transfer. The thrombolytic agent used in Coromandel was reteplase, a deletion mutant of tissue plasminogen activator that can be administered as a bolus. Bolus thrombolytics have important advantages for prehospital administration, such as ease of dosing and administration without the need for intravenous lines, and a lower associated incidence of hypotension than older agents like streptokinase. In Coromandel the patient’s ECG was transmitted to Waikato Hospital for confirmation of the diagnosis. In other regions, the ECG was interpreted by the GP and thrombolytic therapy was administered according to the local treatment protocol.

Eight randomised trials have evaluated prehospital versus in-hospital thrombolysis.6 Overall, prehospital thrombolysis was shown to reduce mortality by 17%, saving sixteen lives per 1000 patients treated. The trials that showed the greatest benefit involved patients with more than one hour’s delay between pain onset and thrombolytic therapy. In particular, the GREAT Trial, where there was a two-hour delay in treatment (similar to the Coromandel experience), found that there was a 44% reduction in mortality with prehospital thrombolysis,4 which could translate into 7-8 lives saved per 100 patients treated per year.

It is encouraging to note that there were no episodes of ventricular fibrillation in the Coromandel study, as this has been a concern with very early administration of thrombolytic therapy, leading to the recommendation that ambulances should be double-manned and carry defibrillators.2

An alternative approach would be to transfer all patients to a hospital with angioplasty facilities, but this would mean that many patients would not receive any reperfusion therapy for at least 90 minutes.7 Besides, most hospitals in New Zealand do not have the facilities to perform primary angioplasty, and those that do sometimes find it difficult to maintain a 24-hour service. A recent meta-analysis of ten trials showed that patients treated by primary angioplasty had long-term mortality rates similar to those treated with thrombolytic therapy.8 Modern reperfusion strategies such as bolus administration of reteplase can produce full epicardial coronary artery reperfusion in 43% of patients by 30 minutes,9 and regimens combining a thrombolytic agent with a IIb/IIIa receptor antagonist achieve full reperfusion in about 80% of patients at 90 minutes, with enhanced myocyte reperfusion.10 The optimal strategy may include both thrombolytic therapy and angioplasty (known as facilitated angioplasty), which has been shown to be safe and to achieve earlier restoration of epicardial blood flow.11

While doctors continue to research and debate the merits of different reperfusion strategies, late patient presentation remains the greatest barrier to myocardial salvage, particularly in rural communities. Mass media campaigns have been run to encourage patients with suspected heart attacks to present earlier for treatment, with some initial success, but the message tends to be forgotten unless the campaigns are repeated every so often.12

Guidelines for the administration of thrombolytic therapy by New Zealand GPs are currently being developed. Patients with suspected acute myocardial infarction should initially be given pain relief, oxygen and an aspirin to chew. If the hospital is less than one hour away, the patient should be transported there immediately. If the expected transport time is greater than one hour, an ECG should be recorded and transmitted to the hospital for advice regarding the appropriateness of prehospital thrombolysis. But this initiative will all come to nothing if the funding agencies fail to ensure funding for bolus thrombolytic agents, as has unfortunately happened in Coromandel. It is to be hoped that rural New Zealanders suffering heart attacks will...
soon have access to the same standard of care as is currently available to their urban counterparts.

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Parasite genome publication

Rival approaches to the way in which genome sequences are published are creating growing tension between scientists at the sequencing centres and those who want to use the sequencing data to further their study of the organisms involved.

International consortia that are sequencing *Plasmodium falciparum*, a parasite that causes malaria, and *Trypanosoma brucei*, which causes sleeping sickness, are each currently engaged in heated arguments over the wisdom of publishing preliminary, annotated sequences in advance of the completion of full sequences.

In each case, prominent biologists who specialize in the organism are pushing for early publication. But genome sequencing centres say the publication of preliminary data could deprive their teams of the proper credit for the full, annotated sequence when it is completed.

Claire Fraser, president of The Institute for Genomic Research at Rockville, Maryland, which is part of both consortia, believes that if outside scientists publish preliminary annotations (proposed function of a stretch of DNA) based on raw sequencing data made available voluntarily by the sequencing centres, the final complete sequence may never be published.


No advantage in screening for endometrial cancer

No advantage exists in routine endometrial screening for patients with breast cancer who are being treated with tamoxifen, according to two new studies.

Although tamoxifen cuts the risk of breast cancer in some women, it also raises the risk of endometrial cancer. As a result, patients taking the drug often undergo invasive and somewhat painful biopsies and ultrasound examination of the uterine lining.

The new studies report that these commonly used screening methods are no more effective at diagnosing early endometrial cancer in these patients than watching for abnormal vaginal bleeding. Researchers found that with high rates of false positive results, the risks from both endometrial biopsy and transvaginal ultrasonography far outweigh the benefits.

Community thrombolysis in the Coromandel region. Audit of the “Cardiac Events in the Coromandel – Assessment Strategy and Triage” (CE-COAST) pilot program

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Abstract

Aim. To audit the experience of a pilot program for community thrombolysis undertaken within the Coromandel region.

Methods. Community thrombolysis for patients suffering acute myocardial infarction (MI) was undertaken in areas within the Coromandel peninsula greater than half an hour by road from Thames Hospital. Thrombolytic therapy (Reteplase) was given following discussion and review of a digitally transmitted ECG with the cardiology registrar. Treatment times and patient demographics were prospectively recorded. Subsequent clinical events were obtained by chart review. Comparison of treatment times were made with an historical cohort for the same population which had received in-hospital thrombolysis between 1993 and 1998.

Results. Between July 1998 and December 1999, nineteen patients received thrombolysis in the community. There were no arrhythmic events during transportation and no deaths or reinfarctions during hospital stay. Median time from pain onset to thrombolysis was 135 (mean 175.5 ± 144.9 SD) minutes which equated to a reduction in median time delay of 135 minutes compared to that experienced by the historical cohort (median 270, mean 316.7 ± 145.8 SD minutes), p=0.0003.

Conclusion. Community thrombolysis is logistically feasible within the New Zealand setting and results in major time reductions in the treatment of patients with acute MI.

Thrombolytic therapy has been shown, over the past ten years, to reduce mortality in patients suffering acute myocardial infarction (MI). However, the extent to which mortality is reduced attenuates with increasing delay from onset of infarction to delivery of treatment. Delays in accessing medical services, transportation to hospitals and hospital triage processes all contribute to total time to treatment. Rural communities are particularly disadvantaged due to the greater distances from both general practitioner (GP) services and base hospitals. A recent report revealed an average delay of over five hours from pain onset to thrombolysis for patients residing within the Coromandel region.

Several international studies have demonstrated the feasibility and safety of pre-hospital assessment and initiation of thrombolysis. 

Because of the considerable treatment delays experienced by Coromandel patients, a pilot program of community thrombolysis was established. We report the first eighteen months experience of this program.

Methods

Local populations greater than half an hour from Thames hospital were identified: Coromandel township, Whitianga, Pauanui, Whangamata, and Waihi. Following appropriate education, GPs from these areas were supplied with a Lifepak-11 machine (Physio Control) and Reteplase (Boehringer Mannheim). The Lifepak 11 ECG machine was used in preference to fax transmission because of convenience and improved clarity of the received ECG. Due to limited supplies of the Lifepak-11 machines, GPs in the same area shared machines on an on-call basis. Because of the distance between Whitianga and Waihi township, sharing the Lifepac was impractical and thus only Waihi township participated in the program.

Patients who presented to their GP with a possible acute MI had a 12 lead ECG which was transmitted via modem to the Waikato coronary care unit (CCU). A direct phone line was set up along side the Lifepac receiving station in CCU to allow unimpeded GP access. The on-call registrar reviewed the ECG and discussed appropriateness of thrombolytic therapy with the GP. In cases of diagnostic uncertainty the registrar could obtain a cardiologist’s opinion. Indications for thrombolytic therapy were: 1. A clinical history of ≥ 30 minutes of chest discomfort beginning ≤ 6 hours previously or > 6 hours if on-going pain present. 2. An ECG showing ST-segment elevation of ≥ 1 mm in two or more inferior leads, or ≥ 2 mm in two or more anterior leads. 3. Absence of contra-indications to thrombolysis.

Standard contraindication criteria were adhered to. Indications and contra-indications to thrombolytic therapy were discussed with the cardiology registrar before a final treatment decision was made.

All patients received 300mg soluble aspirin as soon as a diagnosis of acute MI was made. In those patients receiving thrombolysis, 5000 IU intravenous heparin was given as a bolus injection. Due to the lack of advanced care ambulance officers, intravenous infusions were unable to be administered during transportation. Heparin infusions were therefore commenced once the patient arrived at Thames Hospital. Thrombolytic therapy was administered using Reteplase according to the manufacturer’s guidelines. Two bolus doses were given 30 minutes apart.

The mode of patient transport was determined by the patient’s clinical condition. Road transport by ambulance (unaccompanied by a doctor) was preferred, but where there was haemodynamic instability and medical supervision was required, helicopter retrieval was undertaken usually to Waikato hospital.

GPs documented baseline demographic and clinical data including the timing of onset of chest pain and treatments. Subsequent hospital events and six month outcomes were obtained by phone and/or retrospective chart review. All ECGs were reviewed subsequently by a cardiologist to determine diagnostic accuracy.

An earlier historical review of acute MI management in the Coromandel region between 1993 and 1998 provided detailed information on treatment delays and this allowed comparisons to be made with patients receiving community thrombolysis. Only patients residing within the Coromandel region more than half an hour from Thames hospital in the historical cohort were included in these comparisons.

Results

Between July 1998 and December 1999, there were 75 ECG transmissions to Waikato CCU, nineteen of which met criteria for acute MI (CE-COAST patients). One patient with ST elevation anteriorly who was given thrombolytic therapy was subsequently found to have had an abnormal baseline ECG from an earlier infarct and in fact did not suffer a further infarction. These data
were included in the overall results to allow an intention-to-treat analysis.

Patient characteristics were typical of a cardiac care population (Table 1). There was a trend to reduced heart failure on admission in patients receiving community thrombolysis compared to the historical cohort (21.1 % vs 44.2% respectively, p=0.069). Patient transportation was uneventful following thrombolysis with no arrhythmic events requiring treatment. There were no deaths or reinfarctions during hospitalization. Average length of hospital stay was 5.2 ± 1.8 days. Total creatine kinase enzyme (CK) rise was only 1147 units. Only one patient required in-hospital revascularization.

Table 1. Patient characteristics.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Community Thrombolysis</th>
<th>Historical Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>69.4±9.4</td>
<td>66.2±11.2</td>
</tr>
<tr>
<td>Male</td>
<td>13 (68.4%)</td>
<td>59 (76.6%)</td>
</tr>
<tr>
<td>Diabetic</td>
<td>0</td>
<td>6 (7.8%)</td>
</tr>
<tr>
<td>Smokers</td>
<td>3 (15.8%)</td>
<td>21 (27.3%)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>7 (36.8%)</td>
<td>18 (23.4%)</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>1 (5.3%)</td>
<td>1 (1.3%)</td>
</tr>
<tr>
<td>Heart failure on admission</td>
<td>4 (21.1%)</td>
<td>34 (44.2%)*</td>
</tr>
</tbody>
</table>

*CE-COAST vs historical cohort, p=0.069. Data are mean ±SD or %.

All ECG transmissions were successful except three, which were faxed in the early phase of the program. These failures were due to problems with the use of an external modem at the receiving station. Review of all ECGs revealed only one inappropriate diagnosis as outlined earlier. No patient was denied thrombolysis due to failure to diagnose an infarct although two patients with equivocal ECGs initially had developed an infarct pattern by the time of hospital arrival and were given thrombolysis at that time.

Treatment Times. The initial mean delay to presentation was 122.9 ± 142.8 (SD) minutes (median 65 minutes). The other major delay was the time required for medical assessment (29.0 ± 20.1 minutes). The total delay from pain onset to thrombolysis was 175.5 ± 144.9 minutes, less than a third of which (30.0%) comprised delays after initially seeking medical attention (Table 2).

Historical Comparison. As previously reported,7 between 1993 and 1998, patients with acute MI from within the Coromandel region but living outside Thames and its environs experienced a mean delay of 5 hours and 16 minutes between pain onset and in-hospital thrombolysis. 53.5% of this time comprised delays subsequent to the initial medical assessment. The initial delay in seeking medical attention was unchanged over this time period (Table 2). However, patients thrombolysed in the community had reduced medical assessment (hospital and GP) time (40.2 ± 20.7 vs 96.8 ± 40.9 mins, p<0.0001) and reduced travel time (0 vs 72.6 ± 24.0 mins, p<0.0001) compared to the hospital thrombolysed cohort, respectively. Total time therefore, from pain onset to thrombolysis was reduced from 316.7 ± 145.8 minutes in the hospital treated patients to 175.5 ± 144.9 minutes in community treated patients, p<0.0003 (Figure 1). This resulted in a net median time saving of 135 minutes.

Table 2. Treatment delays prior to thrombolytic administration.

<table>
<thead>
<tr>
<th>Delay</th>
<th>Community Thrombolysis</th>
<th>Historical Cohort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain onset to GP visit</td>
<td>Median* 65.0 (45,130)</td>
<td>Mean† 122.89 ± 142.84</td>
</tr>
<tr>
<td>GP visit to ECG transmission or GP assessment</td>
<td>25.0 (15.45)</td>
<td>29.0 ± 20.05</td>
</tr>
<tr>
<td>ECG transmission to diagnosis or hospital triage time†</td>
<td>9.0 (5,15)</td>
<td>11.21 ± 9.61</td>
</tr>
<tr>
<td>Transport time</td>
<td>0</td>
<td>65 (39.95)</td>
</tr>
<tr>
<td>GP visit to lysis</td>
<td>50.0 (45,70)</td>
<td>52.63 ± 18.81</td>
</tr>
<tr>
<td>Pain onset to lysis</td>
<td>135.0 (85,60)</td>
<td>175.53 ± 144.86</td>
</tr>
</tbody>
</table>

* Median (25th,75th quartiles). † mean ± standard deviation. § GP visit to ECG transmission for community thrombolysis patients or GP assessment for historical group. ‡ ECG transmission to diagnosis for community thrombolysis patients or hospital triage time for historical group.

Discussion

Institution of thrombolytic therapy in the community removes two sources of treatment delay. The first is patient transportation, which is particularly important for areas at considerable distance from a base hospital, and averages 72.6 minutes for Coromandel patients not living in Thames or its environs.7 The second delay occurs once the patient reaches the base hospital and averages between 45 and 90 minutes.19-24 The experience at Thames hospital (60.5 ± 35.3 SD minutes) is consistent with this.7 By instituting community thrombolysis only one medical assessment is required. This is undertaken by the GP in consultation with the base hospital CCU. Repeat assessments are therefore avoided resulting in a reduction in total medical assessment time of 56.6 minutes.

Figure 1. Time differences between patients receiving community thrombolysis (CE-COAST) and historical cohort receiving hospital thrombolysis.7
The impact of the 135 minute total reduction in treatment delay can only be assessed by randomised trials of community versus hospital thrombolysis. A meta-analysis of the eight completed trials revealed a 17% reduction in mortality (p=0.02). Individually, however, the trials failed to show an early mortality benefit due to a combination of small sample sizes and relatively small time savings. Only three trials enrolled more than 300 patients and all but two had timesavings of less than one hour. The largest trial was EMIP (5649 patients) which demonstrated a time saving of 55 minutes with only a trend to reduced 30 day mortality. The GREAT study on the other hand had a time saving of 130 minutes, similar to the CE-COAST program, and demonstrated a divergence in mortality curves with a very substantial late mortality benefit from early thrombolysis. At 30 months, the benefit for patients who presented one hour after pain onset was 90 lives saved per 1000 per hour.

Clinical events during transportation and subsequent hospitalisation in this report were low. Hospital stay was only 5.2 ± 1.8 days, a reduction of two days compared to that experienced over the previous five years (5.2 ± 1.8 vs 7.1 ± 2.8 days respectively, p=0.006). One concern, however, that arises with community thrombolysis is early ventricular fibrillation (VF). Whilst the EMIP study found an increased incidence of early VF with community thrombolysis (2.5% vs 1.6% in hospital patients, p=0.02) the overall incidence was unchanged (6.2% vs 7.0%, p=0.23) due to the relative excess occurring during hospitalisation in patients receiving delayed thrombolysis. The increased incidence of early VF highlights the need of defibrillation facilities and trained personnel during initial GP assessment and subsequent transportation.

This study is limited by small patient numbers and use of an historical cohort. The numbers here, however, reflect the largest experience of community thrombolysis reported to date in Australasia. The use of an historical cohort does allow confounding variables to influence treatment delays independent of treatment location. The differences reported here, however, are of such magnitude that the impact of community thrombolysis is unlikely to be negated.

In conclusion, community thrombolysis is logistically feasible and results in major timesavings for patients otherwise disadvantaged by geography. At present the CE-COAST program is suspended due to difficulties funding thrombolytic agents for use in the community. Given the timesavings and potential impact on short and long term mortality, this is most unfortunate. New Zealand comprises many populated rural areas, which would benefit from this treatment. A national program should be instituted to identify these areas and provide funding to allow all acute infarct patients to receive appropriate therapy in a timely and equitable fashion.

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Adverse Events Regional Feasibility Study: methodological results

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Abstract

Aims. To assess the feasibility of research into the occurrence, causation and prevention of adverse events (AEs) in New Zealand public hospitals.

Methods. A two-stage retrospective review was carried out on 1575 medical records selected by systematic list sample from admissions for 1995 in three public hospitals in the Auckland region. Following initial screening, medical records were subject to structured implicit review using a standardised protocol. Feasibility measures, using international benchmarks where possible, were: adequacy of sample selection; completeness of medical records; reliability and validity of screener and reviewer judgements; internal consistency and face validity of AE determination and preventability assessment.

Results. The sample selection procedure was effective, although nearly 10% of records could not be secured. Information in medical records was sufficient for the identification and analysis of AEs. Adequate levels of agreement were achieved for screener and reviewer judgements, with kappa scores ranging between 0.302 and 0.622 and positive predictive values between 50.0% and 89.7%. The criteria for AE determination showed internal consistency and face validity, as did those for preventability.

Conclusions. Research into the occurrence, causation and prevention of AEs in New Zealand health care settings is methodologically feasible and meets international benchmark standards.

In New Zealand the question of patient safety has, to date, been the subject of relatively little systematic research. One of the first studies to use a standardised, epidemiological approach was a survey of adverse drug events among over 9000 admissions to Dunedin hospital in the early 1970s.1 While useful research since that time has been carried out on surgical audit2 and anaesthetic error,3 and while the Ministry of Health has published some standardised information across New Zealand hospitals,4 no generic, epidemiological data on adverse events (AEs) has been published in this country. The absence of such data has been recognised as an obstacle to developing proposals for the regulation of safety in health and disability.5

A major scientific stimulus to rigorous epidemiological research on patient safety has been the development of standardised procedures for the assessment of AEs using medical records. It was not until the Harvard Medical Practice Study (HMPS) that a measurably reliable, valid and generic definition of AEs was first established across a wide range of clinical settings.6 This approach has been replicated in the Quality in Australian Health Care Study (QAHCS).7

The object of this study was to test the feasibility of applying a standardised protocol to the analysis of medical records with a view to determining the occurrence, causation and prevention of AEs in New Zealand public hospitals.

Methods

Sampling and data collection. Three major public hospitals were selected for study in the Auckland region. The survey population was defined as all patient admissions to these hospitals for calendar year 1995 (excluding day and psychiatric cases). The sampling frame for each hospital was a list of all eligible admissions. New Zealand Health Information Service (NZHIS) selected a systematic list sample of 325 admissions from each hospital, with cases ordered by admission date. Each selected case signalled an index admission. To be included in the study an AE had to be related to, or occur during, the index admission.

The core data collection procedure was a two-stage retrospective review of medical records for selected cases using the Review Form 1 (RF1) and Review Form 2 (RF2), both closely modelled on the comparable instruments in the American and Australian studies.6,7 The first stage was the RF1 screen undertaken by Registered Nurses (RNs). The purpose of this stage was to ascertain if the hospitalisation in question - the index admission - met any of eighteen screening criteria selected as potentially indicative of an AE.8 The second stage undertaken by Medical Officers (MOs) used the RF2, an instrument relying on structured implicit review (that is, the guided exercise of professional judgement), and was designed to determine the presence and context of any AE. In two of the three hospitals an Expert Reviewer (ER) administered “blind” the full cycle of data collection on a one-in-ten sub-sample.

Definitions.8 An AE was operationally defined as (a) an unintended injury or unintended complication, (b) resulting in temporary or permanent disability, including increased length of stay and/or financial loss to the patient, (c) that was caused by health care management rather than the underlying disease process. A key part of AE determination was the assessment of the extent to which an identified injury resulting in disability was caused by health care management. In order to assist reviewers to make this judgement they were guided through a series of seven evaluation questions. Preventability of an AE was assessed as an error in health care management due to failure to follow accepted practice at an individual or system level. In order to assist reviewers in making a judgement about the preventability of AEs they were required to work through ten evaluation questions.

Evaluation of feasibility. (1) Adequacy of sampling – comprehensive sample frame and “success rate” in accessing records. (2) Completeness of records – information available for data collection. (3) Reliability – kappa and positive and negative predictive values (MO as criterion). (4) Validity – kappa and positive and negative predictive values (ER as criterion). (5) Internal consistency and face validity – assessment of AE determination and level of preventability against individual items using positive and negative predictive values (analysis of AE status against discharge mode, ICD external cause code and length of stay, was also carried out, but these results are not reported in full).

Where possible, benchmark comparisons will be made with the corresponding data drawn from QAHCS.3

Results

Adequacy of sampling. Of the 246 sampled records that could not be screened, 30.1% could not be retrieved, 24.4% had inadequate documentation, and 45.5%, mainly day stay, were incorrectly included in the sample by NZHIS. There

were also three records screened criteria positive but not available for further review. Excluding the mis-sampled admissions, the success rate was 90.8%.

Completeness of medical records (Table 1). For the first stage of the review procedure, the RN screen, the available information was judged to be sufficient to complete all aspects of the RF1 in nearly 95% of all sampled records. For the second stage, the MO review, the available information was deemed sufficient to complete all aspects of the RF2 for nearly 85% of all cases classed as AEs, and in the remainder was adequate to determine AE occurrence.

Reliability and Validity (Table 2). Reliability showed only moderate results. While agreement on criteria presence was high (89.7%), the positive predictive value for AE presence was a little over 50%. There was a similar pattern with moderate results. While agreement on criteria presence was high (89.7%), the positive predictive value for AE presence was nearly 85% of all cases classed as AEs, and in the remainder was adequate to determine AE occurrence. Completeness of medical records (Table 1).

AE Determination (Table 3 and Figure 1). The results of reviewer responses on AE determination are presented. The first question, whether there was a note in the record suggestive of the causal role of health care management, was strongly predictive of an AE (positive predictive value=92.7%). In the case of the second question – a note predictive of injury – the relationship was weak. The assessment of the timing of events was the only other item that was strongly predictive of a reviewer’s attribution of an AE. The remaining questions showed a moderate tendency to be predictive of an AE.

Following these seven evaluation questions reviewers were then required to make an assessment of the degree to which the outcome was ‘caused’ by health care management. The results of this exercise are outlined in Figure 1. For nearly half of all cases with both injury and disability or longer hospital stay there was virtually no evidence of health care management causation in the opinion of the reviewer. These were excluded from further analysis. The full protocol - RF2 - was administered to the remaining 142 cases. It is notable, however, that only 70 of these showed moderate, strong or virtually certain evidence of health care management causation.

Assessment of preventability (Table 4). The results of reviewer responses on assessment of preventability are presented.

Table 1. Adequacy of medical records.

<table>
<thead>
<tr>
<th>Medical Record Items</th>
<th>RN* Screening (RF1): Percentage of screened admissions</th>
<th>MO† Reviewing (RF2): Percentage of AE admissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial medical assessment</td>
<td>99.5 % (n=1326)</td>
<td>96.5 % (n=142)</td>
</tr>
<tr>
<td>If applicable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical progress notes</td>
<td>98.7 %</td>
<td>91.5 %</td>
</tr>
<tr>
<td>Nursing progress notes</td>
<td>99.4 %</td>
<td>97.9 %</td>
</tr>
<tr>
<td>Procedure documentation</td>
<td>99.2 %</td>
<td>97.2 %</td>
</tr>
<tr>
<td>Pathology reports</td>
<td>98.6 %</td>
<td>98.6 %</td>
</tr>
<tr>
<td>Discharge summary</td>
<td>96.3 %</td>
<td>94.4 %</td>
</tr>
<tr>
<td>All above items</td>
<td>94.1 %</td>
<td>84.5 %</td>
</tr>
</tbody>
</table>

* Registered Nurse. † Medical Officer. § Review Form 2.

Table 2. Reliability and validity of screener and reviewer judgements.

<table>
<thead>
<tr>
<th>Reliability</th>
<th>Kappa</th>
<th>Percent agreement</th>
<th>Positive predictive value</th>
<th>Negative predictive value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RN/MO†: criteria presence (n=553)</td>
<td>-</td>
<td>89.7%</td>
<td>89.7%</td>
<td>-</td>
</tr>
<tr>
<td>RN/MO: AE presence (n=548)</td>
<td>0.344</td>
<td>74.8%</td>
<td>51.4%</td>
<td>83.0%</td>
</tr>
<tr>
<td>Validity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RN/ER: criteria presence (n=74)</td>
<td>0.465</td>
<td>74.3%</td>
<td>71.4%</td>
<td>76.1%</td>
</tr>
<tr>
<td>RN/ER: AE presence (n=72)</td>
<td>0.302</td>
<td>86.1%</td>
<td>50.0%</td>
<td>89.4%</td>
</tr>
<tr>
<td>MO/ER: AE determination (n=28)</td>
<td>0.622</td>
<td>85.7%</td>
<td>62.5%</td>
<td>95.0%</td>
</tr>
</tbody>
</table>

* Registered Nurse. † Medical Officer. § Expert Reviewer.

Figure 1. Assessing health care management causation.

Assessment of preventability (Table 4). The results of reviewer responses on assessment of preventability are presented.
Whether there was consensus about diagnosis and therapy had little bearing on their judgement of preventability (positive predictive value=46.0%). Complexity, co-morbidity, degree of emergency, potential benefit, chance of benefit, and risk of an AE were other questions with little predictive value. By contrast, appropriateness of management, deviation of management from the accepted norm, and reflection on repetition were questions that were more predictive of reviewers’ judgements of high preventability.

**Table 4. -- High preventability* of adverse events by evaluation category: percent agreement and predictive value.**

<table>
<thead>
<tr>
<th>Evaluation Category</th>
<th>High Preventability of Adverse Events (n=142)</th>
<th>Percent agreement</th>
<th>Positive predictive value</th>
<th>Negative predictive value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GREAT/Deal, some,</td>
<td>56.4%</td>
<td>46.0%</td>
<td>73.6%</td>
<td></td>
</tr>
<tr>
<td>UNCOMPPLICATED/mode</td>
<td>58.2%</td>
<td>45.8%</td>
<td>64.5%</td>
<td></td>
</tr>
<tr>
<td>NONE/moderate, very</td>
<td>75.0%</td>
<td>75.7%</td>
<td>74.8%</td>
<td></td>
</tr>
<tr>
<td>NOT, POSSIBLY/probably, definitely</td>
<td>51.8%</td>
<td>38.2%</td>
<td>60.5%</td>
<td></td>
</tr>
<tr>
<td>SEVERE, MODERATE/little</td>
<td>69.8%</td>
<td>67.6%</td>
<td>70.6%</td>
<td></td>
</tr>
<tr>
<td>NONE/moderate, critical</td>
<td>58.6%</td>
<td>46.7%</td>
<td>64.2%</td>
<td></td>
</tr>
<tr>
<td>MINOR/major, life-saving</td>
<td>52.8%</td>
<td>23.8%</td>
<td>58.5%</td>
<td></td>
</tr>
<tr>
<td>HIGH/moderate, low</td>
<td>51.2%</td>
<td>38.8%</td>
<td>65.0%</td>
<td></td>
</tr>
<tr>
<td>HIGH, MODERATE/low</td>
<td>50.8%</td>
<td>41.3%</td>
<td>63.2%</td>
<td></td>
</tr>
<tr>
<td>NO, PROBABLY NOT/ probably, definitely</td>
<td>72.5%</td>
<td>72.7%</td>
<td>72.4%</td>
<td></td>
</tr>
</tbody>
</table>

* Preventability judged to be more likely than not.

**Discussion**

The primary objective of this study was to assess the feasibility of conducting research into the occurrence, causation and prevention of AEs.

In methodological terms the feasibility study was able, in the first instance, to establish the adequacy of the sample frame and the effectiveness of the sampling procedure. In QAHCS, for example, the sample frame had to be constructed for each hospital. Through NZHIS we were able to draw samples centrally. The success rate - that is, the proportion of sampled records which was screened - was, however, relatively low; 90.8% compared with the Australian rate of 96.8%.8

The level of the information available appeared to be adequate, and comparable to international results. Thus, the standards of medical documentation were sufficient to permit almost universal completion of the RF1. In the case of the RF2, while the determination of AE status was possible in virtually all cases, at least one data item was missing in a sixth of completed RF2s. These results are comparable to those achieved in the Australian study.8 The quality of the assessment process – that is, the level of agreement on the screening and reviewing tasks - was adequate when compared to the results from QAHCS.

More important to the validity of the study was the process of AE determination. In QAHCS and HMPS two MO reviewers were used, with arbitration in case of disagreement. Despite this, the overall level of agreement in these studies was low.9 This confirms the conclusion of one authority that physician agreement on the quality of care is often only slightly better than chance.10 Furthermore, research suggests that discussion between reviewers does not actually improve the reliability of peer review of hospital quality.11 In this investigation, as in the more recent Utah and Colorado studies (UTCOS), only a single MO was used, with ‘blind’ expert review of a 10% sub-sample. The positive predictive value for agreement between MO reviewers and ERs - admittedly on a small sample - was 62.5% (kappa=0.622). This equates to the level of agreement achieved between MOs in QAHCS (kappa=0.55)9 and between reviewers in a reliability study of a sub-sample in UTCOS (kappa=0.4).

The study broke relatively new ground in attempting to go beyond conventional measures of internal validity. Thus it was possible to use the evaluation items in the assessment of AE status and of preventability to establish the internal consistency and face validity of reviewer judgements. For example, of the records assessed for AE status and which had a note indicating that health care management was causative, over 90% were judged by reviewers to be AEs. Similarly, where the reviewer deemed management inappropriate, a high level of preventability was a likely assessment.

In a separate exercise not reported here, internal consistency was also assessed against routinely-collected hospital information. In this case, however, results did not prove to be useful. In particular, external cause codes signalling medical misadventure had low sensitivity, identifying only a quarter of AEs, although length of stay was more predictive (a greater than average stay was associated with half all AEs).

In conclusion, this study was designed to test at a regional level the feasibility of carrying out research on the detection and analysis of AEs. The investigation has demonstrated sufficient levels of performance in methodological terms, as judged by international benchmark standards for work of this kind.

**Acknowledgements.** This study was funded by the Health Research Council of New Zealand. We are very grateful to the management and clinical staff of the three participating hospitals and to the study’s Advisory and Monitoring Committee, chaired by Dr. David Richmond. Specifically, we thank Dr Colin McArthur of Auckland Hospital, Dr Ian Brown and Dr Peter Gow of Middlemore Hospital, and Dr Andrew Love of North Shore Hospital, for their assistance and comments. Valuable comments were also contributed by an anonymous reviewer. We wish to thank our medical review and data processing teams for their meticulous work, and hospital records staff for their willing co-operation. Finally, we acknowledge the assistance of statistics students supervised by Professor Scott.

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Adverse Events Regional Feasibility Study: indicative findings

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Abstract

**Aims.** To identify substantive findings of potential clinical and managerial significance from a regional feasibility study of adverse events (AEs).

**Methods.** A standardised protocol using structured implicit review was applied to 142 AEs generated in an audit study of three public hospitals in the Auckland region for admissions in 1995. Areas of potential significance addressed were: timing, location and impact of AEs; preventability; and clinical context and predictability.

**Results.** 142 cases were identified as AEs (10.7% of 1326 screened records). In 102 cases, 7.7% of all screened records, it was considered to be more likely than not that health care management contributed to the AE. About half the reported AEs occurred before the index admission, the majority outside hospital. Over half of all events resulted in disability that was resolved within a month. An average 6.7 extra days stay in hospital were attributable to AEs. For 60% of AEs the evidence for preventability was either low or non-existent. Areas of potential prevention were predominantly educational. Over half of all AEs occurred in a surgical context. Medical AEs were more likely to have occurred outside hospital, to be drug-related, to be associated with an acute admission, to be classified as highly preventable, and to have a greater impact on hospital stay.

**Conclusions.** Although the data generated by a feasibility study must be treated with caution, the pattern of results is consistent with comparable Australian findings and is of potential clinical and managerial significance.

The subject of patient safety, and the quality of health care, has gained increasing momentum. Although it has been over a decade since the publication of the first authoritative estimates of adverse events (AEs) in the Harvard Medical Practice Study (HMPS), within the last eighteen months there has been a report on patient safety from the Institute of Medicine and an issue of the British Medical Journal devoted to medical error. Other journals have also canvassed the question and studies on AEs and medical error have been published in other developed countries.

Interest in patient safety has also been evident in Australia, with some of the earliest work published on anaesthesia-related mortality. The first broad-based and representative investigation using internationally standardised and clinically generic procedures of AE determination was the Quality in Australian Health Care Study (QAHCS).

In New Zealand the question of patient safety has, to date, been little researched. The methodological results from a feasibility study designed to test the application of such standardised epidemiological techniques in the New Zealand setting is reported in the preceding article. This article presents some key substantive findings from the feasibility study that may be of clinical and managerial significance. These relate to the timing, location and impact of AEs, their preventability, and their clinical context and predictability.

The core data collection procedure of the study was a two-stage retrospective review of medical records for selected cases using the Review Form 1 (RF1) and Review Form 2 (RF2), both closely modelled on the comparable instruments in the American and Australian studies. In two of the three hospitals an Expert Reviewer (ER) administered “blind” the full cycle of data collection on a one-in-ten sub-sample. Fuller details on data collection are provided in the preceding paper.

Preventability of an AE was assessed as an error in health care management due to failure to follow accepted practice at an individual or system level. Potential for prevention of recurrence of particular AEs was assessed by MO reviewers identifying broad ‘areas of effort’.

Results

**Frequency.** Of 1575 medical records sampled, and allowing for missing and excluded data, 515 were screened criteria positive and went on to medical review. Of these, 142 cases were identified as AEs (10.7% of all screened records). In 102 cases, 7.7% of all screened records, it was considered to be more likely than not that health care management contributed to the AE.

**Timing, location and impact.** Information on the timing, location and impact of adverse events is presented in Table 1. Looking at all AEs, about a half occurred before the sampled (index) admission and an extra 6.7 days was added to hospital stay. A third of all AEs took place outside a public hospital (mostly in ambulatory settings) and had a greater than average effect in lengthening hospital stay. Over half of all events occurring before the index admission took place outside a public hospital. AEs occurring inside hospital and...
during the index admission had least impact on length of hospital stay.

The effect of AEs on the health status of patients is assessed in Table 2. For most patients - more than half - any disability suffered as a result of an AE resolved within a month. The impact on hospital workload - as measured by attributable bed days (ABD) - increased noticeably for more severe and more long-term disability.

Preventability. Information on reviewer assessments of the preventability of AEs is presented in Table 3. In a third of cases the reviewers judged there to be virtually no evidence of preventability. For another third of cases the evidence was weak to equivocal, while for the remainder the judgement of preventability was much more definitive.

In Table 4, ‘areas of effort’ - that is, the potential for the prevention of recurrence - are considered alongside impact and preventability. The largest category identified by reviewers was improved education, followed by improved resources, quality assurance, communication, and systems reorganisation. The area with the greatest adverse impact was poor quality assurance, while the area with the highest level of preventability was systems error. Improved education was the largest category for prevention, but its profile was an average one for both impact and preventability.

Clinical context and predictability. Reviewers classified AEs according to specialty and area of clinical application. This information, together with other features of clinical context, is presented in Table 5. Overall, AEs were reasonably evenly distributed across medicine and surgery. Operative and drug-related incidents were the commonest clinical areas involved. The former were more characteristic of surgery and of AEs internal to hospital, the latter of medicine and of AEs external to hospital. Events classified in medicine were also more likely - when compared (conservatively) to AEs overall - to occur outside hospital, to be associated with an acute admission, and to have co-morbidity present.

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|c|c|c|}
\hline
\textbf{Location} & \textbf{Before} & \textbf{During} & \textbf{All AEs} \\
 & \textbf{index admission} & \textbf{index admission} & \\
 & \textbf{Percent} & \textbf{Mean ABD*} & \textbf{Percent} & \textbf{Mean ABD} \\
\hline
\text{Inside Hospital} & 42.3\% & 8.9 & 100\% & 4.5 & 68.3\% & 6.1 \\
\text{Outside Hospital} & 57.7\% & 8.0 & - & - & 31.7\% & 8.0 \\
\hline
\text{All AEs} & 100\% & 100\% & & & & \\
\hline
\end{tabular}
\caption{Distribution of AEs – by location and timing of occurrence.}
\end{table}

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|c|c|}
\hline
\textbf{Disability} & \textbf{Percent} & \textbf{Mean ABD*} \\
\hline
Minimal < 1 month\textsuperscript{1} & 56.3\% & 4.1 \\
Moderate 1-12 months & 20.4\% & 11.0 \\
Permanent ≤50% & 3.5\% & 23.4 \\
Permanent >50% & 2.1\% & 27.5 \\
Death & 6.3\% & 3.9 \\
Unable to tell & 11.3\% & 4.3 \\
\hline
\text{All AEs} & 100\% & 6.7 \\
\hline
\end{tabular}
\caption{Impact of AEs – disability status by hospital stay.}
\end{table}

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|}
\hline
\textbf{Preventability*} & \textbf{Frequency} & \textbf{Percent} \\
\hline
1. Virtually no evidence & 45 & 31.7\% \\
2. Slight to modest evidence & 27 & 19.0\% \\
3. Close call, ≤50\% & 14 & 9.9\% \\
4. Close call, >50\% & 25 & 17.6\% \\
5. Moderate/strong evidence & 22 & 15.5\% \\
6. Virtually certain evidence & 8 & 5.6\% \\
Missing & 1 & 0.7\% \\
\hline
\text{All AEs} & 142 & 100\% \\
\hline
\end{tabular}
\caption{AEs – Attribution of preventability.}
\end{table}

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|c|c|}
\hline
\textbf{Area for Attention*} & \textbf{% All AEs} & \textbf{%Perm. Preventability} & \textbf{Mean ABD*} & \textbf{% High preventability} \\
\hline
Education & 41.6\% (59) & 13.6\% & 6.9 & 61.0\% \\
Resources & 10.6\% (15) & 13.3\% & 6.7 & 66.7\% \\
Quality assurance & 9.2\% (13) & 30.8\% & 10.2 & 61.5\% \\
Communication & 9.2\% (13) & 23.1\% & 7.5 & 61.5\% \\
System & 6.3\% (9) & 11.1\% & 6.5 & 88.9\% \\
Other & 14.8\% (21) & 4.8\% & 9.9 & 66.7\% \\
\hline
\text{All AEs} & 100\% (142) & 12.0\% & 6.7 & 38.7\% \\
\text{(n=142)} & & & & \\
\hline
\end{tabular}
\caption{Prevention of recurrence – areas of effort by impact and preventability.}
\end{table}

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|c|c|}
\hline
\textbf{Specialty and clinical area.} & \textbf{AEs Occurred} & \textbf{All AEs} & \textbf{Specialty} & \textbf{Other}\textsuperscript{1} \\
\text{Inside hospital} & 68.3\% (n=97) & 100\% & 51.4\% (n=73) & 44.4\% (n=63) & 4.2\% (n=6) \\
\text{Outside hospital} & 31.7\% (n=45) & & & & \\
\hline
\text{All AEs} & 100\% & 100\% (n=142) & 100\% (n=73) & 44.4\% (n=63) & 4.2\% (n=6) \\
\hline
\text{Operative} & 31.3\% & 9.6\% & 25.5\% (40) & 47.0\% & 1.4\% \\
\text{Drug} & 12.4\% & 36.5\% & 20.4\% (32) & 7.2\% & 37.7\% \\
\text{System} & 16.2\% & 17.3\% & 16.5\% (25) & 15.7\% & 17.4\% \\
\text{Other}\textsuperscript{1} & 38.1\% & 36.5\% & 37.6\% (59) & 30.1\% & 43.5\% \\
\hline
\text{Total mentions} & 100\% & 100\% (n=157) & 100\% & 100\% & \\
\hline
\text{% of AEs: Outside Hospital} & 31.7\% & 12.3\% & 50.8\% & \\
\text{% of AEs: Transfer admission} & 7.0\% & 8.2\% & 4.8\% & \\
\text{% of AEs: Acute admission} & 68.1\% & 48.0\% & 92.1\% & \\
\text{% of AEs: Co-morbidity present} & 47.2\% & 39.7\% & 55.6\% & \\
\hline
\end{tabular}
\caption{Specialty and clinical area.}
\end{table}

*Categories 4, 5 and 6 are classified as ‘high’ preventability.
The United Kingdom has become the first country to give official approval for the use of results from genetic tests for Huntington’s disease for insurance purposes.

Insurers, to use information from such testing.

Genetic tests for Huntington’s disease are sufficient for insurance companies to use the results when assessing applications for life insurance.

The approval was given in response to an application from the professional body representing the insurance industry, the Association of British Insurers, to use information from such testing.

Discussion

Many of the key substantive findings outlined in this paper are not only of potential clinical and managerial significance; they also add to confidence in the overall study because of their consistency with the comparable Australian results. For example, about half of AEs occurred before admission, a high proportion of events were regarded as not preventable, the great majority of events resulted in disability that was temporary, but resulted in an average of just under seven extra days hospital stay. These are all findings of intrinsic clinical and policy interest, but they are also of an order of magnitude comparable with results generated in QAHCS.

There are other findings reported here that were relatively unanticipated and that invite further attention. For example, a third of all AEs occurred outside the hospital setting. Similarly, and related to this, there appeared to be a pattern of drug-related events, many of which occurred outside hospital. Furthermore, routinely-collected hospital data showed some predictive power, with over 90% of medical AEs and about half of surgical AEs associated with an acute admission.

These two findings - the importance of adverse drug events (ADEs) and the possibilities of administrative data - provide examples, respectively, of potential clinical and managerial significance. They have been reported elsewhere in the literature and underline the potential for further development in these areas. Thus, Bates et al evaluated fifteen screening criteria for their sensitivity and specificity in predicting AEs, preventable AEs, and serious AEs. Although no set of administrative data was particularly sensitive - that is, able to predict a high percentage of AEs - using such data was much less costly than other methods of detection. Similarly with ADEs; a high proportion are preventable. Data of this kind in turn can lead to a search for causes.

In conclusion, this feasibility study has generated substantive results that not only engender confidence in the methodology - being generally consistent with findings reported from other studies - but are also of potential clinical and managerial application.

Acknowledgements

This study was funded by the Health Research Council of New Zealand. We are very grateful to the management and clinical staff of the three participating hospitals and to the study’s Advisory and Monitoring Committee, chaired by Dr David Richmond. Specifically, we thank Dr Colin McArthur of Auckland Hospital, Dr Ian Brown and Dr Peter Gow of Middlemore Hospital, and Dr Andrew Love of North Shore Hospital, for their assistance and comments. Valuable comments were also contributed by an anonymous reviewer. We thank our medical review and data processing teams for their meticulous work, and hospital records staff for their willing co-operation. Finally, we acknowledge the assistance of statistics students supervised by Professor Scott.

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Table 6. Impact and preventability - by specialty and clinical area.

<table>
<thead>
<tr>
<th>Specialty</th>
<th>% permanent disability/death</th>
<th>Mean ABD</th>
<th>% High Preventability</th>
</tr>
</thead>
<tbody>
<tr>
<td>All AEs (n=142)</td>
<td>12.0% (n=17)</td>
<td>6.7</td>
<td>38.7% (n=55)</td>
</tr>
<tr>
<td>Specialty</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Surgery (n=71)</td>
<td>8.2%</td>
<td>6.0</td>
<td>24.7%</td>
</tr>
<tr>
<td>Medicine (n=63)</td>
<td>14.5%</td>
<td>7.7</td>
<td>56.5%</td>
</tr>
<tr>
<td>Other (n=6)</td>
<td>33.3%</td>
<td>5.6</td>
<td>33.3%</td>
</tr>
<tr>
<td>Clinical Area</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operative (n=40)</td>
<td>7.5%</td>
<td>5.7</td>
<td>20.0%</td>
</tr>
<tr>
<td>Drug-Related (n=12)</td>
<td>15.6%</td>
<td>8.4</td>
<td>41.8%</td>
</tr>
<tr>
<td>System (n=26)</td>
<td>19.2%</td>
<td>7.0</td>
<td>76.0%</td>
</tr>
<tr>
<td>Other (n=59)</td>
<td>10.2%</td>
<td>6.4</td>
<td>42.2%</td>
</tr>
</tbody>
</table>

*Attributable bed days in the study hospital, spent over one or more admissions associated with an AE. An AE in a particular clinical area could be additionally classified as “system”.

The community services card: does it make a difference to pharmaceutical utilisation?

Frances Sutton, consultant, Wellington; Peter Crampton, Senior Lecturer, Department of Public Health, Wellington School of Medicine and Health Services Research Centre, Wellington.

Abstract

Aims. To estimate the utilisation rate, and amount of state subsidy of prescription items per head by age, sex and community Services Card (CSC) for the year ended June 1999.

Methods. Data from a market research company (IMS Health), Health Benefits Limited, Statistics New Zealand and Work and Income New Zealand were used to calculate average per head per year pharmaceutical utilisation rate and subsidy cost for CSC holders and non-holders.

Results. For both sexes, and for all age groups, CSC-holders tended to use more prescription items per head and incur higher subsidy cost than non-holders. The standardised CSC utilisation rate was 2.6 times the non-CSC rate. For children, average per-item subsidy cost for CSC-holders was lower than for non-holders; the reverse was true for adults.

Conclusion. CSC holders had higher pharmaceutical utilisation rates than non-holders at a national level (but not necessarily at a local level). If non-uptake of cards and health status were taken into account, however, it is possible that pharmaceutical utilisation rates were sub-optimal amongst those most in need of services. Analyses are urgently required to examine prescribing patterns at a regional level.

The plan to introduce community services cards (CSCs) and high use health cards (HUHCs) was first announced in the July 1991 National Government Budget along with planned changes to the New Zealand health system.1 From 1/2/1992 low income families and some chronically ill people became eligible for CSCs and HUHCs respectively. Possession of a card entitled the holder to a higher rate of subsidy for GP consultations and prescriptions, and to lower user charges for certain hospital and secondary services. The overall aim of the CSC was to target more effectively state assistance for certain primary care services.2 The estimated CSC holding rate for the year ended June 1999 was 39.3%.3

As the principal tool aimed at reducing financial barriers to access for primary care services, the CSC has been criticised on several counts. Problems such as low uptake, the ‘poverty trap’ effect around the eligibility threshold (the abrupt cut-off for CSC eligibility creates a ‘poverty trap’ at the low end of the non-eligible population), lack of indexing of subsidy levels to take account of inflation, as well as the actual subsidy level (which typically results in a part-charge to the patient), have meant that significant barriers to access (ie, part-charges) have persisted for many families.4-6

The aim of this paper is to assess whether the targeted subsidy regime has benefited CSC holders by comparing, at the national level, average utilisation and subsidy cost of prescription items for CSC holders and non-holders. Utilisation is defined in terms of prescription items processed by Health Benefits Limited (HBL), and costs are defined by government-funded (ie, excluding user charges) costs of these items.

Methods

Data and methods for calculating the utilisation rates are described elsewhere.1

Data sources. Data were from the following sources: IMS Health (a market research company) and HBL (prescribing data); Statistics New Zealand (population projections); and Work and Income New Zealand (WINZ) (CSC population data). IMS provided from their Medical Index numbers of prescription items by age group, sex, and CSC/HUHC status, collected from a stratified sample of 300 general practitioners (GPs). The stratification was based on the four Health Funding Authority (HFA) regions. The GPs in the sample reported for seven consecutive days. The IMS panel did not consist of the same doctors for the whole year; IMS bulked up the sample so that the number of prescription items represented New Zealand GP usage. The IMS data counted each repeat prescription as one item, and in this respect matched HBL data. Unlike HBL data, the IMS data were intended to capture all prescription items, unsubsidised as well as subsidised.

Calculation of IMS-derived utilisation rates. The 10% of prescription items with missing values of the categorical variables (eg, age, sex) were distributed over categories without missing values using proportionality assumptions. The IMS and population data were used to compute utilisation rates (prescription items per head per year by age, sex, CSC and HUHC status). Denominator populations for the utilisation rates were estimated using Statistics New Zealand population projections at 30/6/1998 and 30/6/1999, CSC population data from WINZ, information on HUHC-holding from HBL, and HUHC prevalence rates (probability of HUHC-holding, by age, sex and CSC status) estimated from a sample of 1 million patients from practice registers of primary care organisations participating in an HFA pilot project on capitation funding for primary care.

Adjustments had to be made to the utilisation rates for some of the elderly population. First, rates for age 85+ years appeared inconsistent with each other and with younger age groups, probably because of small sample size and denominator problems. The latter arose because a material proportion of the population aged 85+ years is resident in a geriatric hospital, and should be (but was not) excluded from the population denominators. IMS stated that such residents were not included in their survey (test-home residents were included). For males and non-HUHC females, utilisation rates derived from the IMS data were replaced with estimates defined thus: [Rate for age 85+] = 1.3 x [Rate for age 75-84].

The factor of 1.3, though somewhat arbitrary, was based on the following evidence: 1) the relationship in the IMS data between rates for ages 65-74 and 75-84 years; 2) the relationship of discharge rates for ages 75-84 and 85+ years for discharges from publicly-funded hospitals. Secondly, for HUHC females aged 75-84 and 85+ years, the utilisation rates calculated from the IMS data were implausibly low. These two rates were replaced with the rate for non-HUHC females of age 65-74 years. There was no alternative to an essentially arbitrary estimate.

Utilisation rates for the whole population by age, sex and CSC were obtained by assuming that age- and sex-specific utilisation rates for HUHC holders were the same regardless of CSC status. Utilisation rates for HBL-processed items and subsidy cost per head per year. Some items for non-CSC/HUHC adults are not processed by HBL, as they cost less than the $15 prescription charge. The IMS data, however, include such items. The first step of the method, therefore, involved reducing by 20% IMS-derived numbers of items per head for non-CSC/HUHC adults. This percentage is the estimated proportion of items for this population that were dispensed, but not processed by HBL. The estimate is based on the assumption that for adults, prescriptions with a gross cost under $15 as a proportion of all prescriptions dispensed is the same for CSC holders and non-holders. Gross cost is defined as the subsidy plus the prescription charge (which excludes the manufacturers’ surcharge). This step resulted in IMS-derived utilisation rates by age, sex and CSC/HUHC status, reducing as described for non-CSC/HUHC adults.

The second step involved scaling these rates to match numbers of items from HBL. The scaling was such that when the scaled rates...
Results
For both sexes, and for all age groups, CSC-holders tended to use more prescription items per head and incur higher subsidy cost than non-holders (Tables 1 and 2). The difference (measured by the ratio) was greatest for females in the childbearing years. This may arise because of associations between maternity and utilisation, and maternity and the probability of CSC-holding. Because the age/sex profile of CSC-holders and non-holders differs, overall rates by CSC were age/sex standardised. The standardised CSC utilisation rate was 2.6 times the non-CSC rate. For children, average per-item subsidy cost for CSC-holders was lower than that for non-holders; the reverse was true for adults (Table 3). Interpreting these findings on unit costs is difficult because the subsidy system is complex and the denominators in the averages exclude items dispensed, but unprocessed by HBL.

Discussion
The results of this study, based on HBL data and a national sample of GPs, show that CSC holders used more prescription items per head and had higher per head subsidy cost than non-CSC holders. This finding is consistent with a retrospective survey of GP records carried out by Gribben which found that the mean number of prescription items per year for CSC holders was 6.74 compared with 4.89 for non-holders (p<0.0001). Gribben also found that CSC holders had 0.9 more GP consultations per year than non-holders, and CSC holding remained a significant predictor of consultation rate after controlling for age, sex, HUHC status and five common chronic conditions. More generally, New Zealand and United Kingdom studies have shown people on low incomes, or with low socioeconomic status, or living in deprived areas have overall higher GP utilisation rates. However, studies of GP utilisation amongst Maori have generally demonstrated utilisation rates that are similar to or lower than those of non-Maori. Five important points must be noted in interpreting the results of this study. First, data coding errors and errors arising from assumptions may have introduced bias into the analysis. It is not possible to quantify the magnitude or direction of any such bias. Second, there is evidence that not all eligible people apply for a CSC. Non-uptake of cards limits the usefulness of CSCs as a means of targeting those most in need, and results in inequitable resource allocation. Other methods of resource allocation might be preferable, for example capitation-based funding of primary care pharmaceutical budgets, weighted for the socioeconomic deprivation of a practice population. Third, the results are national averages which may hide considerable regional variation. Other studies have demonstrated lower levels of pharmaceutical expenditure in poor areas compared with the national average. More emphasis should perhaps be focused on addressing barriers to access at a local level in low-income areas.

Fourth, the health status of various population groups, particularly Maori and those living in socioeconomically deprived areas, is comparatively poor. Reducing health inequalities requires a range of strategies; improving access to primary health care services is undoubtedly important, especially as research suggests that significant cost barriers to access remain for many CSC holders. Fifth, this study did not take health status (as a measure of need for health services) into account. If health status were taken into account it is possible that pharmaceutical utilisation rates were sub-optimal amongst those most in need of services. In conclusion, this study suggests that CSC holders had higher pharmaceutical utilisation rates than non-holders at a national level (but not necessarily at a local level). If non-

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### Table 1. Number of HBL-processed items per head and ratio of rates (CSC / non-CSC) for the year ended June 1999.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age group</th>
<th>Rate per head</th>
<th>Rate per head ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>0</td>
<td>11.4</td>
<td>7.2</td>
</tr>
<tr>
<td></td>
<td>1-4</td>
<td>9.9</td>
<td>6.9</td>
</tr>
<tr>
<td></td>
<td>5-14</td>
<td>3.2</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td>15-24</td>
<td>11.2</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>25-44</td>
<td>15.3</td>
<td>3.6</td>
</tr>
<tr>
<td></td>
<td>45-64</td>
<td>22.1</td>
<td>7.8</td>
</tr>
<tr>
<td></td>
<td>65-74</td>
<td>30.9</td>
<td>11.8</td>
</tr>
<tr>
<td></td>
<td>75-84</td>
<td>33.8</td>
<td>17.3</td>
</tr>
<tr>
<td></td>
<td>85+</td>
<td>43.3</td>
<td>23.3</td>
</tr>
<tr>
<td>M</td>
<td>0</td>
<td>14.4</td>
<td>7.9</td>
</tr>
<tr>
<td></td>
<td>1-4</td>
<td>9.9</td>
<td>7.5</td>
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<tr>
<td></td>
<td>5-14</td>
<td>3.1</td>
<td>1.5</td>
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<td></td>
<td>15-24</td>
<td>3.0</td>
<td>1.4</td>
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<td>5.6</td>
<td>2.4</td>
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<tr>
<td></td>
<td>45-64</td>
<td>17.3</td>
<td>5.4</td>
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<tr>
<td></td>
<td>65-74</td>
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<td>13.0</td>
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<td></td>
<td>75-84</td>
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<tr>
<td></td>
<td>85+</td>
<td>40.9</td>
<td>26.6</td>
</tr>
<tr>
<td>NZ*</td>
<td></td>
<td>13.3</td>
<td>5.1</td>
</tr>
</tbody>
</table>

*Age/sex standardised using NZ 1998/99 population.

### Table 2. Cost ($ex GST) per head, and ratio of rates (CSC / non-CSC) for the year ended June 1999.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age group</th>
<th>Rate per head</th>
<th>Rate per head ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>F</td>
<td>0</td>
<td>108</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td>1-4</td>
<td>94</td>
<td>62</td>
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<td></td>
<td>5-14</td>
<td>45</td>
<td>20</td>
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<td></td>
<td>15-24</td>
<td>176</td>
<td>47</td>
</tr>
<tr>
<td></td>
<td>25-44</td>
<td>247</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td>45-64</td>
<td>359</td>
<td>141</td>
</tr>
<tr>
<td></td>
<td>65-74</td>
<td>502</td>
<td>216</td>
</tr>
<tr>
<td></td>
<td>75-84</td>
<td>551</td>
<td>316</td>
</tr>
<tr>
<td></td>
<td>85+</td>
<td>710</td>
<td>429</td>
</tr>
<tr>
<td>M</td>
<td>0</td>
<td>136</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>1-4</td>
<td>94</td>
<td>67</td>
</tr>
<tr>
<td></td>
<td>5-14</td>
<td>43</td>
<td>18</td>
</tr>
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<td></td>
<td>15-24</td>
<td>47</td>
<td>23</td>
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<tr>
<td></td>
<td>25-44</td>
<td>90</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>45-64</td>
<td>280</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td>65-74</td>
<td>442</td>
<td>201</td>
</tr>
<tr>
<td></td>
<td>75-84</td>
<td>305</td>
<td>153</td>
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<td></td>
<td>85+</td>
<td>674</td>
<td>488</td>
</tr>
<tr>
<td>NZ*</td>
<td></td>
<td>209</td>
<td>86</td>
</tr>
</tbody>
</table>

*Age/sex standardised using NZ 1998/99 population.

### Table 3. Cost ($ex GST) per item, and ratio of unit costs (CSC / non-CSC) for the year ended June 1999.

<table>
<thead>
<tr>
<th>Age group</th>
<th>CSC</th>
<th>No CSC</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>0, 1-4</td>
<td>9.5</td>
<td>8.9</td>
<td>1.1</td>
</tr>
<tr>
<td>5-14</td>
<td>11.8</td>
<td>11.7</td>
<td>1.0</td>
</tr>
<tr>
<td>15-24</td>
<td>15.7</td>
<td>16.6</td>
<td>0.9</td>
</tr>
<tr>
<td>25-44, 45-64</td>
<td>16.1</td>
<td>18.1</td>
<td>0.9</td>
</tr>
<tr>
<td>65-74, 75-84</td>
<td>16.2</td>
<td>18.2</td>
<td>0.9</td>
</tr>
<tr>
<td>85+</td>
<td>16.4</td>
<td>18.2</td>
<td>0.9</td>
</tr>
<tr>
<td>NZ*</td>
<td>15.2</td>
<td>16.2</td>
<td>0.9</td>
</tr>
</tbody>
</table>

*Age/sex standardised using NZ 1998/99 population.
uptake of cards and health status were taken into account, however, it is possible that pharmaceutical utilisation rates were sub-optimal amongst those most in need of services. More analyses are urgently required to examine prescribing patterns at a regional level.

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Heart disease and diabetes risk factors in Pacific Islands communities and associations with measures of body fat

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Abstract

Aims. To describe the prevalence of obesity and other coronary heart disease and Type 2 diabetes risk factors by age and ethnic group in Pacific Island communities and to determine the associations between these risk factors and body mass index.

Methods. Cross-sectional data from two community-based intervention projects were combined to provide anthropometric, blood sample and blood pressure data on 1175 Pacific Islands people (467 men, 708 women) aged 20 years and over from church communities in South, Central and West Auckland. Self-reported data on diabetes status and leisure-time physical activity were also collected.

Results. Based on an ethnic-specific body mass index (BMI) cut-off (≥ 32 kg/m²), 45% of men and 66% of women were obese. The age-standardised prevalence of known diabetes was 12%. Men and women aged 40 - 60 years had the highest risk factor levels and were the most sedentary. Tongans had higher risk factor levels than Samoans. In men, BMI and waist circumference were associated (p<0.05), in the direction of greater disease risk, with blood pressure and concentrations of total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, and blood glucose. In women, these associations were similar but less consistent.

Conclusions. While these data are not representative for all Pacific people living in New Zealand, they do show an extremely high prevalence of obesity and significant associations between obesity and other cardiovascular risk factors. These communities warrant a very high priority as part of public health efforts to address New Zealand's growing obesity epidemic.


Pacific populations in New Zealand carry a heavy burden of coronary heart disease (CHD) and diabetes. Several CHD risk factors are more prevalent amongst Pacific people than they are amongst European New Zealanders. They have higher mean blood pressures and a higher prevalence of hypertension, microalbuminuria is more prevalent, physical activity levels are lower and smoking rates are higher. Also, Pacific people consume large quantities of food than Europeans and their diets contain more meat and less fruit and vegetables. Moreover, Pacific populations are among the most obese populations in the world and obesity is a strong independent risk factor for both CHD and Type-2 diabetes. In contrast, some CHD risk factors are not as prevalent as might be expected in such obese populations. For example, serum cholesterol levels tend to be lower for Pacific people compared to Europeans. This may be due to genetic differences and to diet, but also, weak associations between high levels of body fat and other CHD and diabetes risk factors have been described.
Much of the information available on the prevalence of obesity and other CHD risk factors in Pacific populations in New Zealand is based on the Workforce Diabetes Survey, or the 1997 National Nutrition Survey (NNS97). Unfortunately, both surveys have their limitations in providing the full picture of obesity in Pacific populations. The NNS97 over-sampled Pacific people but interpretation is hampered by a poor response rate (less than 50%) and low sample size (273 with anthropometric data). The workforce survey had larger numbers (n=650) and was able to examine for differences in CHD risk between Pacific ethnic groups, rather than assuming that risk is homogeneous. However, the sampling frame was restricted to the older (40+ years), employed Pacific workforce in Auckland and Tokor;oa.

This present study pools baseline data from two large community-based intervention projects in Auckland and describes variations in the prevalence of obesity and other CHD risk factors by age and ethnic group. It also investigates the associations between these risk factors and measures of body fatness. While this study also has limitations of extrapolation to the New Zealand-wide Pacific communities, the overall aim is to provide information from these two large studies to build a clearer picture of these important risk patterns.

Methods

Participants. Participants came from two community-based intervention programs, the South Auckland Diabetes Project (SADP) and the Samoan Ola Fa’a’utata Project (SOFP). The SADP was established in 1991 as a multi-faceted program that aimed to reduce the incidence of diabetes in those with diabetes. The church community was defined as those people who were invited to take part. There was no specific selection for those who were obese or those with diabetes. The church community was defined as those people whose names were on the church roll plus their household members. From these membership lists, the baseline response rate for the SADP was 60% and for the SOFP it was 81%.

This analysis includes self-identified Pacific Islands people, aged 20 years and over who had complete anthropometric data at baseline. A total of 1175 people, 725 (287 men, 438 women) from the SADP and 450 from the SOFP (180 men, 270 women). Where our data from these Church Intervention Surveys (CIS) were compared with the National Health and Nutrition survey, we included BMI data from an additional 117 adolescent’s aged 15 - 19 years. The SOFP was given ethical approval by the University of Auckland Human Subjects Ethics Committee and the SADP by the Auckland Area Health Board Ethics Committee.

Data collection. Both projects collected data at a series of health surveys on church premises between 1991 and 1996. Participants received information sheets, translated if necessary, and a brief presentation explaining the project. Consent was obtained from all participants (interpreters were available). Standardised techniques were used to measure weight, height, waist, and hip circumference. The SOFP used Seca electronic scales (model 708) with an attached stadiometer to measure weight and height after removing heavy clothing and shoes. The SADP used the same standardised techniques, a portable stadiometer (CMS, London) and Salter spring scales. The scales were calibrated regularly. Non-stretch fibreglass tapes were used to measure waist and hip circumference. Waist circumference was measured horizontally through a point midway between the top of the iliac crest and the bottom of the ribs. Hip circumference was measured at the largest posterior extension of the buttocks.

Non-fasting venous blood samples were collected, stored and analysed for blood lipids, glucose and fructosamine levels by Medlab Ltd. Samples were measured using Roche Diagnostic protocols. Blood pressure was measured twice in the sitting position with a standard mercury sphygmomanometer using Korotkoff phase 1 and 5 sounds for systolic and diastolic blood pressure respectively. Oversize cuffs were used for large arms. Participants were considered sedentary if they did no moderate or vigorous activity during a normal week.

Statistical Analysis. BMI was calculated as weight (kg) divided by height (m) squared. Pacific specific BMI cutoffs were used to define overweight (26 kg/m2 ≥ BMI < 32 kg/m2) and obesity (BMI ≥ 32 kg/m2). The data were stratified by gender and analysis of variance was used to calculate adjusted age- and ethnic-specific means. Age-standardised means (Table 1) were calculated by the direct method using Segi’s world population for those aged ≥ 20 years. Multiple regression was used to test for linear associations between age and blood cholesterol, triglyceride, glucose and blood pressure. A second model that included a quadratic term (age-squared) was used to test for curvilinearity. With the exception of the ethnic group analysis, indicator variables were used to adjust for confounding by ethnic group and to adjust for systematic differences in risk factor levels between the two projects.

Table 1. Association between age and anthropometric, biochemical and blood pressure measurements, mean (SEM), in Pacific Islands men. The prevalence of obesity, sedentary leisure time activity and known diabetes is also given.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>P for linear term1</th>
<th>P for quadratic term1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthropometry, n</td>
<td>467 106 97 120 94 50</td>
<td>0.0006</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>97.6 (8.9) 96.7 (1.9) 98.3 (2.0) 99.9 (1.8) 99.0 (2.1) 94.7 (2.6)</td>
<td>0.69</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172.9 (3.0) 171.7 (0.6) 174.1 (0.7) 171.5 (0.6) 170.9 (0.7) 168.9 (0.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Body mass index, kg/m^2</td>
<td>32.6 (2.7) 30.8 (0.6) 32.3 (0.6) 33.9 (0.5) 33.9 (0.6) 33.2 (0.8)</td>
<td>0.0004</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>101.9 (6.9) 96.7 (1.5) 101.0 (1.5) 107.2 (4.4) 108.2 (4.6) 108.3 (2.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hip, cm</td>
<td>110.7 (5.6) 109.9 (1.2) 109.7 (1.3) 112.1 (1.1) 111.2 (1.3) 111.4 (1.6)</td>
<td>0.33</td>
</tr>
<tr>
<td>BMI &gt; 32 kg/m², %</td>
<td>45 32 37 59 56 47</td>
<td>-</td>
</tr>
<tr>
<td>Non-fasting lipids, n</td>
<td>352 67 69 94 79 43</td>
<td>0.0001</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.80 (0.49) 5.28 (0.11) 5.87 (0.13) 6.27(0.11) 6.08(0.11) 5.74(0.16)</td>
<td>0.0001</td>
</tr>
<tr>
<td>HDL, mmol/L</td>
<td>1.10 (0.14) 1.14 (0.04) 1.11 (0.04) 1.09(0.03) 1.05(0.03) 1.07(0.04)</td>
<td>0.0004</td>
</tr>
<tr>
<td>LDL, mmol/L</td>
<td>3.67 (0.47) 3.17 (0.12) 3.68 (0.13) 4.08(0.11) 3.94(0.12) 3.74(0.14)</td>
<td>0.0002</td>
</tr>
<tr>
<td>Total:HDL ratio</td>
<td>5.56 (0.78) 4.83 (0.21) 5.67 (0.21) 5.98(0.18) 6.03(0.20) 5.65(0.25)</td>
<td>0.0003</td>
</tr>
<tr>
<td>Triglyceride, mmol/L</td>
<td>2.40 (0.60) 2.17 (0.28) 2.65 (0.28) 2.77(0.25) 2.51(0.27) 2.00(0.34)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Non-fasting glucose, mmol/L</td>
<td>402 81 84 105 88 44</td>
<td>-</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>6.37 (1.61) 4.96 (0.39) 5.36 (0.40) 6.42 (0.35) 7.60 (0.39) 8.47 (0.50)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Blood pressure, n</td>
<td>414 86 87 110 87 44</td>
<td>0.0001</td>
</tr>
<tr>
<td>Systolic, mmHg</td>
<td>136.2 (8.4) 126.4 (2.0) 130.0 (2.0) 134.1(1.8) 145.3 (2.0) 152.1 (2.6)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diastolic, mmHg</td>
<td>80.8 (5.6) 80.9 (1.3) 84.2 (1.4) 87.9 (2.2) 92.2 (1.4) 92.9 (2.8)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Leisure time activity, %</td>
<td>342 94 75 75 66 32</td>
<td>-</td>
</tr>
<tr>
<td>Sedentary, %</td>
<td>22 14 19 31 24 28</td>
<td>-</td>
</tr>
<tr>
<td>Known diabetes, %</td>
<td>12 2 3 7 21 36</td>
<td>-</td>
</tr>
</tbody>
</table>

Age standardised to Segi’s world population. 1Model 1. Age was the independent continuous variable and variables for ethnic group and study were included in the model.

1Model 2. Same as model 1 with the quadratic term (age-squared) included. 2Total n for LDL = 310 because of triglyceride levels > 4 mmol/L. 3The natural logs of triglyceride and glucose were used in the models. 4Leisure time activity data were not collected from 1995 onwards in the South Auckland Diabetes project.
Associations between body size and lipids, glucose and blood pressure were tested using analysis of variance. Multiple regression was used where the variables were treated continuously. Triglyceride and glucose variables were log transformed to improve the normality of the distributions. Analyses were carried out in SAS version 6.10 (SAS Institute, Cary, NC, USA).

Results

Participants in this CIS study were older (mean age 42 years for men, 41 years for women) and more likely to be female (60% of participants) than the general Pacific Islands population (1996). Most were Samoan (64%) or Tongan (26%) whereas approximately 50% of the general Pacific Islands population identify themselves as Samoan and 14% as Tongan. Compared to the overall distribution of occupations in New Zealand (1991), the distribution of occupations for Samoans in the SOFP project was shifted to the lower income end (p<0.001).

Based on our results, 81% and 86% of Pacific Islands men and women aged 15 years and over were either overweight (26 kg/m² ≥ BMI < 32 kg/m²) or obese (BMI ≥ 32 kg/m²), with 4% of men and 58% of women being obese (note: these are not age-standardised). We compared these percentages with results from the National Nutrition Survey in Figures 1 and 2. Compared to the NNS97, we observed a considerably lower combined prevalence of overweight and obesity for young Pacific males (87.6% vs 55.8%). On the other hand our estimates of obesity were higher (44.8% vs 27.4%) for males aged 25-44 years and overweight lower than for the NNS97. Differences were also noted for females aged 25-44 years (61.5% obese vs 34% obese; Figure 2).

Differences were also noted for females aged 25-44 years (61.5% obese vs 34% obese; Figure 2).

Table 1 and 2 present associations between age and CHD and diabetes risk factors for men and women aged 20 years and over. Pacific men had an age-standardised mean BMI of 32.6 kg/m². Most risk factors were higher in the older age groups, although mean weight, BMI, obesity prevalence, total cholesterol, LDL, the total: HDL ratio, triglycerides and sedentary leisure time activity were lower in the 60+ age group than the younger two decade groups. Women (Table 2) had a higher aged- standardised mean BMI (34.8 kg/m²) than men and a higher prevalence of obesity (60% with a BMI ≥ 32kg/m²). As with men, most risk factor levels were lower in the 60+ age group than the younger age groups. Overall, 12% of men and women reported having Type-2 diabetes and approximately one-quarter did no leisure time physical activity during a normal week.

A comparison of risk factors by ethnic group is given in Table 3. Tongans had the highest mean BMI and the highest prevalence of obesity. Their lipid profiles were significantly more atherogenic than Samoan profiles. Also, Tongan men had significantly higher mean glucose levels and Tongan women were significantly more likely to do no leisure time physical activity.

Table 4 shows relationship between BMI and waist circumference with other CHD and diabetes risk factors. For men, each risk factor, with the exception of HDL (where the association was inverse), was higher at higher quartiles of BMI. A similar, although less consistent pattern was observed between these risk factors and quartiles of waist circumference. Where BMI and waist circumference were treated continuously, both were positively (p<0.05) associated with each risk factor. There was an inverse association with HDL. These models were re-run (not shown) including squared terms for BMI or waist. None of the squared terms was significant.

For women, the variation in these risk factors with quartiles of BMI and waist was not as marked as it was for men. Mean total and LDL cholesterol and glucose levels differed little although mean systolic and diastolic blood pressure was higher with each quartile of BMI. Using continuous data, only the total:HDL cholesterol ratio, (log) triglycerides and systolic and diastolic blood pressure were positively associated with BMI. There was a significant negative association between HDL and BMI. Total cholesterol, the total:HDL cholesterol ratio, (log) triglycerides, glucose, and diastolic blood pressure were all positively associated with waist circumference in women. When the squared terms for BMI and waist were included in these models, negative associations (p<0.05 for BMI) were observed for cholesterol, the total:HDL cholesterol ratio, LDL and (log) triglyceride concentrations (not shown).

Discussion

These cross-sectional analyses of CHD and diabetes risk factors came from church-based Pacific populations and are not, therefore, a representative sample. However, the sample size was large (total = 1175) and the results are probably as characteristic of New Zealand’s Pacific Islands population as...
Pacific populations are amongst the most obese in the world living in the Pacific Islands. The review indicated that age range and amongst the most obese.

Table 2. Association between age and anthropometric, biochemical and blood pressure measurements, mean (SEM), in Pacific Islands women. The prevalence of obesity, sedentary leisure time activity and known diabetes is also given.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>n</th>
<th>Men</th>
<th>Tongan</th>
<th>Other PI*</th>
<th>Women</th>
<th>Tongan</th>
<th>Other PI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthropometry, n</td>
<td>708</td>
<td>187</td>
<td>169</td>
<td>164</td>
<td>111</td>
<td>77</td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>91.2 (9.6)</td>
<td>87.8 (6.0)</td>
<td>93.3 (6.0)</td>
<td>96.8 (1.7)</td>
<td>93.7 (1.9)</td>
<td>85.6 (2.3)</td>
<td>0.84 &lt; 0.01</td>
</tr>
<tr>
<td>Height, cm</td>
<td>161.5 (2.9)</td>
<td>164.0 (0.5)</td>
<td>163.0 (0.5)</td>
<td>161.4 (0.5)</td>
<td>160.0 (0.6)</td>
<td>157.4 (0.7)</td>
<td>&lt; 0.01 0.06</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>34.8 (1.1)</td>
<td>32.1 (0.5)</td>
<td>34.9 (0.6)</td>
<td>36.4 (0.7)</td>
<td>34.2 (0.8)</td>
<td>&lt; 0.01 &lt; 0.01</td>
<td></td>
</tr>
<tr>
<td>Hip, cm</td>
<td>115.5 (6.9)</td>
<td>118.1 (1.1)</td>
<td>115.0 (1.2)</td>
<td>118.9 (1.2)</td>
<td>119.3 (1.4)</td>
<td>119.4 (1.7)</td>
<td>&lt; 0.01 &lt; 0.01</td>
</tr>
<tr>
<td>BMI &gt; 32 kg/m², %</td>
<td>60</td>
<td>41</td>
<td>64</td>
<td>72</td>
<td>73</td>
<td>58</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 3. Ethnic differences in anthropometric, biochemical and blood pressure measurements, mean (SEM). Differences in the prevalence of obesity, sedentary leisure time activity and known diabetes are also given.

<table>
<thead>
<tr>
<th>Ethnic group</th>
<th>n</th>
<th>Samoan</th>
<th>Tongan</th>
<th>Other PI*</th>
<th>Samoan</th>
<th>Tongan</th>
<th>Other PI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthropometry, n</td>
<td>286</td>
<td>140</td>
<td>41</td>
<td>474</td>
<td>167</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>94.5(1.1)</td>
<td>101.9 (1.8)</td>
<td>96.7(2.8)</td>
<td>89.4(0.9)</td>
<td>95.9(1.7)</td>
<td>80.0(2.4)</td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>171.4(0.4)</td>
<td>174.4 (0.6)</td>
<td>171.7(0.9)</td>
<td>160.9(0.3)</td>
<td>162.8(0.5)</td>
<td>159.8(0.7)</td>
<td></td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>32.1(3.3)</td>
<td>33.5 (6.0)</td>
<td>32.7(0.8)</td>
<td>34.5(0.3)</td>
<td>35.6(0.6)</td>
<td>34.2(0.8)</td>
<td></td>
</tr>
<tr>
<td>Waist, cm</td>
<td>103.1(0.8)</td>
<td>104.0 (1.4)</td>
<td>105.9(2.1)</td>
<td>101.0(0.6)</td>
<td>105.7 (1.3)</td>
<td>102.6(0.7)</td>
<td></td>
</tr>
<tr>
<td>Hip, cm</td>
<td>108.0(0.7)</td>
<td>111.5 (1.1)</td>
<td>112.2(1.7)</td>
<td>115.0(0.6)</td>
<td>114.7(0.3)</td>
<td>115.6(0.7)</td>
<td></td>
</tr>
<tr>
<td>Obesity, % BMI ≥30 kg/m²</td>
<td>63</td>
<td>68</td>
<td>33</td>
<td>73</td>
<td>73</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>Obesity, % BMI ≥32 kg/m²</td>
<td>46</td>
<td>49</td>
<td>42</td>
<td>60</td>
<td>59</td>
<td>66</td>
<td></td>
</tr>
</tbody>
</table>

*Other Pacific Islands (PI) ethnic groups, Cook Islands Maori (n=51), Niuean (n=20), and mixed Pacific Islands ethnic group (n=39). †Total n for LDL = 514 because of triglyceride levels > 4 mmol/L. ‡The natural logs of triglyceride and glucose were used in the models. Leisure time activity data were not collected from 1995 onwards in the South Auckland Diabetes project. a,b,c Significantly different from Ap < 0.05, bp < 0.01, cp < 0.0001 adjusted for age group and study.
Table 4. Association between non-fasting blood lipids, blood glucose and blood pressure with quartiles of body mass index (BMI) and waist for Pacific Islands men and women. Multivariate regression co-efficients (β) and the variation explained by each model (R²) are also included.

<table>
<thead>
<tr>
<th></th>
<th>Cholesterol mmol/L</th>
<th>Chol:HDL Ratio</th>
<th>HDL mmol/L</th>
<th>LDL mmol/L</th>
<th>Triglyceride mmol/L</th>
<th>Glucose mmol/L</th>
<th>Systolic BP mm Hg</th>
<th>Diastolic BP mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men: BMI, n</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quartile (mean kg/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1(26.1)</td>
<td>5.56</td>
<td>5.42</td>
<td>1.17</td>
<td>3.60</td>
<td>1.87</td>
<td>5.74</td>
<td>128.8</td>
<td>81.0</td>
</tr>
<tr>
<td>2(31.3)</td>
<td>5.82</td>
<td>5.62</td>
<td>1.11</td>
<td>3.67</td>
<td>2.49</td>
<td>6.41</td>
<td>155.5</td>
<td>86.1</td>
</tr>
<tr>
<td>3(35.0)</td>
<td>5.96</td>
<td>5.85</td>
<td>1.05</td>
<td>3.84</td>
<td>2.60</td>
<td>6.39</td>
<td>137.2</td>
<td>88.5</td>
</tr>
<tr>
<td>4(41.2)</td>
<td>6.19</td>
<td>6.18</td>
<td>1.02</td>
<td>3.95</td>
<td>3.10</td>
<td>7.14</td>
<td>142.9</td>
<td>94.1</td>
</tr>
<tr>
<td>Linear, β (R² %)</td>
<td>0.04(3.2)</td>
<td>0.10(5.3)</td>
<td>-0.01(1.9)</td>
<td>0.02(1.2)</td>
<td>0.04(8.8)</td>
<td>0.01(2.5)</td>
<td>0.83(8.3)</td>
<td>0.81(2.5)</td>
</tr>
</tbody>
</table>

| **Women: BMI, n**|                    |                |            |            |                    |               |                   |                   |
| Quartile (mean kg/m²) |                  |                |            |            |                    |               |                   |                   |
| 1(26.1)      | 5.23               | 5.11           | 1.21       | 3.33       | 1.44               | 5.76          | 129.9            | 81.2              |
| 2(31.3)      | 5.71               | 5.51           | 1.37       | 3.70       | 2.44               | 5.75          | 132.1            | 84.0              |
| 3(35.0)      | 6.02               | 5.95           | 1.05       | 3.84       | 2.67               | 6.75          | 138.6            | 88.4              |
| 4(41.2)      | 6.11               | 6.19           | 1.04       | 3.87       | 3.03               | 6.68          | 137.0            | 91.8              |
| Linear, β (R² %) | 0.02(2.1)          | 0.04(3.1)      | -0.004(2.0)| 0.01(1.3)  | 0.02(5.0)          | 0.005(1.8)    | 0.30(7.0)        | 0.30(1.9)         |

| **Men: Waist, n**|                    |                |            |            |                    |               |                   |                   |
| Quartile (mean, cm) |                 |                |            |            |                    |               |                   |                   |
| 1(84.90)     | 5.51               | 4.73           | 1.21       | 3.53       | 1.44               | 5.76          | 129.9            | 81.2              |
| 2(97.10)     | 5.71               | 5.51           | 1.37       | 3.70       | 2.44               | 5.75          | 132.1            | 84.0              |
| 3(106.3)     | 6.02               | 5.95           | 1.05       | 3.84       | 2.67               | 6.75          | 138.6            | 88.4              |
| 4(120.5)     | 6.11               | 6.19           | 1.04       | 3.87       | 3.03               | 6.68          | 137.0            | 91.8              |
| Linear, β (R² %) | 0.01(0.3)          | 0.02(1.3)      | -0.005(1.4)| 0.005(0.1)| 0.01(3.8)          | 0.004(1.2)    | 0.53(7.7)        | 0.49(0.8)         |

| **Women: Waist, n**|                    |                |            |            |                    |               |                   |                   |
| Quartile (mean cm) |                  |                |            |            |                    |               |                   |                   |
| 1(82.60)     | 5.23               | 4.21           | 1.30       | 3.28       | 1.41               | 6.39          | 127.6            | 78.3              |
| 2(96.70)     | 5.48               | 4.73           | 1.22       | 3.30       | 1.76               | 6.62          | 128.6            | 80.5              |
| 3(105.9)     | 5.47               | 4.74           | 1.20       | 3.45       | 1.85               | 6.66          | 130.6            | 82.2              |
| 4(119.7)     | 5.44               | 4.80           | 1.18       | 3.43       | 1.88               | 6.69          | 136.6            | 86.5              |
| Linear, β (R² %) | 0.00(0.3)          | 0.02(1.3)      | -0.005(1.4)| 0.005(0.1)| 0.01(3.8)          | 0.004(1.2)    | 0.53(7.7)        | 0.49(0.8)         |

*The natural logs of triglyceride and glucose were used for the regression models. †‡Significantly different from quartile 1, 2 or 3 respectively at p<0.01 adjusted for age, ethnic group and study. *Significant association with BMI or waist circumference at *p<0.05 or ‡p<0.001 adjusted for age, ethnic group and study.

Tongan and Niuean.11 We found that Tongan men and women were bigger than their counterparts from other islands.

The lipid profiles of Pacific men and women in the current study were less athrogenic than those reported in the National Nutrition Survey,13 and the Workforce Diabetes Survey.11 The attenuation of risk factor levels in older Pacific people has previously been described23,24 and probably reflects selective mortality of high risk individuals or the cohort effect of a relatively lower risk group of individuals now reaching older age.2 The age-standardised prevalence of known diabetes (12%) was similar in these church communities to the prevalence observed in a household survey of inner urban South Auckland.25 Systolic and diastolic blood pressure levels were comparable to those observed in Pacific members of a Seventh-Day Adventist church.24 Also, similar increases in blood pressure with age have been observed in Pacific Islands people in the workforce.15-26 The number of people who were sedentary during leisure-time was high in these communities. Previous New Zealand studies found that Pacific people were less involved in leisure-time activities than Maori or European and that Pacific women were less active than men.27 Body size was adversely associated with other CHD and diabetes risk factors. However, the associations were not as strong as those observed for European New Zealanders,28 and other less obese populations.27 Moreover, for Pacific women, there was evidence of attenuation between BMI and blood cholesterol and triglycerides at the upper end of the BMI distribution. Similar findings have been observed in studies of Samoans in Western Samoa and American Samoa, and Micronesian Nauruans.28,29 The poor associations observed were attributed to extreme obesity in these populations. This suggests that these populations have reached a level of obesity above which the impact of total fat or intra-abdominal fat on CHD and diabetes risk factors becomes less apparent.

There are a number of limitations to this study. As mentioned above, neither the church communities nor the participants were randomly selected and therefore the results may not readily be generalised to New Zealand’s wider Pacific population. Numbers in the studies were lower than they could have been for several variables because providing a blood sample was voluntary and up to 30% declined. Finally, combining the results of two separately conducted studies is not ideal methodology although we tried to overcome this limitation by adjusting for risk factor differences between the projects.

The SADP and the SOFP were both designed to reduce CHD and diabetes risk factors in these church communities and both had some success.10,11 The high prevalence of obesity and other risk factors at baseline suggests that these interventions were not only warranted, but long overdue in the effort to bring the health status of these communities in line with that of other New Zealanders. Future efforts to contain the rising prevalence of obesity in New Zealand need to give priority to Pacific People.

Acknowledgements. The Samoan Ola Fa’auta Project was supported by the Health Research Council of New Zealand and the National Heart Foundation of New Zealand. Material support for the Pacific Islands Church program of the South Auckland Diabetes Project was provided by the Lotteries Board, North Health, South Auckland Health, Boehringer Mannheim / Roche Diagnostics, ASB Trust, Novo Nordisk, Eli Lilly, Tegal, New Zealand Dairy Board and Sanitarium. Colin Bell was supported by a National Heart Foundation Postgraduate Scholarship. We thank the pastors and their wives for their tremendous contribution to these studies and the congregations of the churches for their participation.

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With the escalation of treatment costs, who to treat and when to treat become increasingly important questions for the clinician and health care provider. For example, lipid lowering therapy using statins has been shown to reduce cardiovascular events even in asymptomatic individuals. However, widespread use of these drugs in asymptomatic low risk individuals is costly and the magnitude of benefit is likely to be small. We have previously compared the use of relative risk reduction (RRR) and number needed to treat (NNT) to describe coronary heart disease (CHD) event prevention following long-term intervention with statins under different circumstances in similarly aged adults.\(^1\) NNTs indicate how many individuals must incur the expense and inconvenience of treatment, and risk of adverse effects to save one person from experiencing the defined event over a given period of time. The higher the baseline risk, the smaller the corresponding NNT.

Consensus treatment guidelines drawn up by experts in recent years have adopted the concept of baseline risk in combination with the projected RRR to assess the necessity and urgency of treatment. The baseline risk can be estimated from the Framingham equation\(^2\) or from tables.\(^3,4\) With regard to the benefits of statins and antihypertensives, RRRs are fairly similar at different levels of baseline risk. The risk of a patient having a cardiovascular event without treatment can be used as a basis for deciding whether somebody needs drug treatment or not. For example, a young woman with hypertension or hyperlipidaemia only may not require drug treatment or not. For a 60-year old man who is a smoker and has a family history of cardiovascular disease may very well require treatment of hypertension and hyperlipidaemia even if the blood pressure and cholesterol levels are only moderately elevated.

Consideration of absolute risk reduction (ARR) has also been a key innovation in the new British Hypertension Society Guidelines for the management of hypertension.\(^5,6\) One of its recommendations is a formal estimation of a subject’s ten year CHD risk in order to decide whether or not to treat patients with mild hypertension. Recourse to ARR in preference to RRR is certainly a laudable development. However, this approach does not take age into account. As elderly patients have inherently higher risks of disease and death, treatment decisions based on risk alone will always favour the elderly. An elderly person with very mild hypertension readily attains the 1.5% or so annual risk of events that mandates treatment. A young person even with multiple risk factors, may not reach the same level of annual risk. If treatment decisions are to be based solely on risk, then implementing this strategy will lead to therapeutic decisions that run contrary to common sense. The problem is that knowing the baseline risk, (i.e. the risk of no treatment), is not enough. One also needs to know the benefits of treatment. In this regard, the benefit is directly related to life expectancy. A cardiovascular death at 50 represents, say 20 years of life lost, whereas a death at 75 could represent only five lost years.

Unfortunately, ARR has been confused with absolute benefit. The two are not equivalent. Risk is the likelihood of the occurrence of an unfavourable event whereas in preventive medicine, the benefit is the magnitude of the loss or harm averted. One is a probability; the other is a quantity. Total benefit depends on ARR and the value of preventing one unfavourable event. The total amount of benefit is more important than how frequently the instalments come. A minimal benefit can never be attractive, even if there is a 99% chance of gaining it. On the other hand, a tiny risk, say 1%, cannot always be ignored, especially if the penalty is something unpleasant. We take out fire insurance on our home because we cannot afford the consequences of a fire even if the risk is extremely remote. What is actually at risk matters just as much as how great the risk is.

For moral, ethical and religious reasons, we tend to value life and well-being as the same in everyone. Although the immediate consequences of a stroke are equally devastating regardless of age, the long-term consequences, however, are much worse when it afflicts a 40-year old. Hence, prevention of the same event gives rise to different magnitudes of benefit depending on a person’s age and circumstances. Any decisions on treatment based on absolute risk alone will miss this important dimension in a therapeutic decision. Clinicians routinely make use of such information instinctively and intuitively. Guidelines that exclude such considerations are mechanical, imperfect and misleading.

There is also the view that treatment of hypertension or hyperlipidaemia can be delayed until the absolute annual risk crosses a certain threshold. Most patients as consumers and clinicians concerned with preventive medicine, would reject such a view. Untreated hypertension or hypercholesterolaemia damage the heart, vasculature and kidneys so that early preventive measures are warranted. Coronary artery disease, heart failure and renal failure are often asymptomatic until an advanced stage.

In the absence of a full assessment of risk and benefit, basing treatment decisions on the cardiovascular risk alone is inadequate. This policy will tend to prolong life in the extremely elderly whilst forgoing the prevention of premature deaths through withholding treatment in the young. One solution might be to use the NNT to gain one year of life (or better still, one quality adjusted life year or QALY), to evaluate the effectiveness of an intervention. The elderly have a lower life expectancy and hence the life-years gained will be fewer compared to younger persons. The paradox arising from equating death in the elderly with premature death in the young can therefore be resolved. There are already plans to express clinical trial results in terms of average duration of life gained (ADLG).\(^7\) As benefits resulting from medical intervention vary with age, increasingly we will have to consult life tables as a fairer basis for the allocation of resources and priorities.

Pioneering attempts have been made at calculating the gain in event-free life years arising from lipid lowering therapy.\(^8\) As soon as one considers the lifetime risk of cardiovascular disease, it becomes apparent that most events occur before people reach

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**VIEWPOINT**

**Should decisions on treatment be based on absolute benefit rather than absolute risk?**

Bernard M Y Cheung, Associate Professor; Cyrus R Kumana, Professor, Division of Clinical Pharmacology and Therapeutics, Department of Medicine, The University of Hong Kong, Hong Kong.
an age at which they have a high annual cardiovascular risk. Individuals at high risk are mostly elderly and are only the tip of the iceberg. Using a hypothetical cohort of 100 non-smoking men as illustration, Ulrich et al showed that the gain in life years per treatment year was maximal at age 40, which was therefore thought to be the optimal age of starting treatment.\(^8\) However, this 'optimal' age may vary from person to person, depending on cardiovascular risk factors and life expectancy. There are many imponderables involved in calculating life expectancies and risks, so we do not necessarily expect this method of therapeutic decision to gain currency. On the other hand, the event-free life gained from treatment varies little from age 20 to 70.\(^9\) \(^5\) Treating 30 year olds will save fewer lives but gain as many life years as treating 70 year olds. Viewed from this perspective, using the absolute risk to determine necessity or priority of treatment is a seriously flawed backward step in the prevention of cardiovascular disease. The way forward must be to base therapeutic decisions on the benefit the patient is likely to derive from the treatment.

**Acknowledgements.** BMY Cheung and CR Kumana are members of the Institute of Cardiovascular Science and Medicine. BMY Cheung has received grant support from the Health Services Research Committee of Hong Kong.

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**MEDICOLEGAL DIARY**

**Recommending particular treatment options: the vitamin K experience**

Jonathan Coates, Senior Solicitor, Buddle Findlay, Wellington.


This column has previously discussed what information must be given to a patient in order for the patient to be sufficiently informed about an intervention, and for the consent to be valid. What, however, are a health professional's obligations where there are diametrically opposed treatment options? Should the health professional simply discuss the benefits and risks associated with the various options, or should he recommend one? How far should the patient be encouraged to accept a particular treatment option?

These issues can be answered with reference to a New Zealand case involving the death of a twelve day old baby. The baby's parents had conducted their own research (including using the internet) into the appropriateness of vitamin K administration. At the parent's request, no vitamin K was administered. The baby died from haemorrhage.

The law allows parents to either consent to or refuse administration of vitamin K to their baby. In reaching that decision, parents have the right to be provided with information that a reasonable parent, in the circumstances, would expect to receive. This will include an explanation of the options available together with expected risks, side effects, benefits and costs of each option.\(^1\) Studies have raised the possibility of a link between intra-muscular vitamin K and increased risk of acute lymphoblastic leukaemia. The link has not been proven and would not need to be raised by the health professional if the parents did not question the appropriateness of vitamin K. If the parents however indicate a concern about vitamin K, the risks associated with refusing vitamin K are sufficiently material to require the health professional to warn the parents.

Should the health professional do more than just explain the risks, and actually encourage the parents to consent to administration of vitamin K? On the face of it, the law only requires health professionals to recommend a treatment option if they are expressly asked to do so by the parents (or patient).\(^2\) So if a patient does not ask for a recommendation, the general rule is that a recommendation need not be offered. However, in order to ensure that the patient-parent is fully informed, the health professional must also provide “any other information required by legal, professional, ethical, and other relevant standards”.\(^3\)

Professional and ethical standards probably dictate that ‘other information’ which must be provided includes encouragement that vitamin K should be administered. At this point in time, the weight of research supports such a conclusion. Such encouragement would be consistent with a consensus statement issued by the Paediatric Society of New Zealand, together with a number of other professional organisations, on the subject.\(^4\)

Health professionals who encourage parents to consent to the administration of vitamin K must be careful that they do not go too far. An essential element of a valid consent is that it is given voluntarily. Any duress will invalidate the consent. The final decision must rest with the parents.

Issues such as this will become more frequent with the dissemination of information on the internet. The quality of information on the internet is variable and lay people will not always be able to assess its accuracy. Health professionals must get used to discussing treatment options with patients. They should consider asking the patient whether he or she would like a recommendation on the treatment options. Health professionals will want to help their patients make decisions that will lead to the most appropriate treatment, without becoming paternalistic and invalidating the consent. It will not always be an easy tightrope to walk.

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1. Code of Health and Disability Services Consumers' Rights, right 6(1)(b).
2. Code of Health and Disability Services Consumers' Rights, right 6(3)(b).
3. Code of Health and Disability Services Consumers' Rights, right 6(1)(b).

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