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This Issue in the Journal

High calcium scores in patients with a low Framingham risk of cardiovascular (CVS) disease: implications for more accurate CVS risk assessment in New Zealand
Chris J Ellis, Malcolm E Legget, Colin Edwards, Niels Van Pelt, John A Ormiston, Jonathan Christiansen, Helen Winch, Mark Osborne, Greg Gamble

Trying to predict who is at higher risk of a heart attack or stroke is not easy for doctors. The currently used method is quite inaccurate, and relies on ‘risk factors’ such as a high cholesterol level and advancing age, to try to calculate who is more at risk. A calcium score test, which involves having a low dose of X-ray via a 'CT' scan, helps to improve this assessment, by measuring who actually has more calcified arteries than would be expected for their age. These patients are at higher risk. Hence a calcium score test can detect some of those people who may be best advised to take preventative medicines to protect their arteries at an earlier age, than would otherwise be suggested, using currently used methods. Of 942 patients in Auckland, we found that 27% of patients who were calculated to be at low risk, using the standard method, in fact had a markedly increased 'calcium score', meaning that they were actually at a much higher risk than had been calculated. These patients were then able to be more vigorously managed, rather than (inadvertently) falsely reassured.

Basing musculoskeletal curriculum changes on the opinions of practicing physicians
Thomas Pasley, Song Chan, Phillippa Poole, Martin Wild, Fiona McQueen

This study has involved a survey of practising doctors in the Auckland region including GPs and specialists. The aim has to be to define a list of musculoskeletal conditions which are felt to be the most important for the qualified doctor to know about (knowledge used frequently in day to day clinical practice). Thus we hope to influence the structure and emphasis of the medical curriculum so that it provides medical students with knowledge that they are most likely to need in clinical practice.

Needlestick injuries in a healthcare setting in New Zealand
Marie Fullerton, Veronique Gibbons

The level of underreporting of needlestick injuries was 33% for all respondents, which is consistent with internationally-reported figures. The main reason for non-reporting was a lack of time. Furthermore, the perception of a low risk of blood-borne viruses from a patient or procedure was another reason for non-reporting. This study serves to quantify the level and rationale for non-reporting of needle-stick injuries. Results from the study will inform policy and enable targeted education to reduce the unnecessary burden on health resources within the Waikato District Health Board.
Healthcare services funded by Counties Manukau District Health Board for people in the last year of life
Wing Cheuk Chan, Gary Jackson, Doone Winnard, Philippa Anderson

Consistent with the international literature, this study found that Counties Manukau District Health Board (CMDHB) residents in the last year of life have a high level of health service utilisation. People often have multiple chronic diseases in the last year of life. Decisions about the appropriate use of high-cost health services in people towards the end of life can be extremely challenging. These decisions are resource allocation decisions as well as clinical decisions and should be based on clinical factors, cost utilities, and patient, family, and society’s expectations.
Improving cardiovascular risk assessment

Harvey D White

Cardiovascular disease (CVD) remains the number-one cause of death in New Zealand and is part of an increasing global crisis in non communicable diseases.\(^1\) Although there have been impressive gains made, we have fallen behind Australia in reducing CVD deaths and the prevalence is expected to increase because of the increasing rates of obesity and diabetes.

The New Zealand cardiovascular risk assessment guidelines were updated in 2009\(^2\) but failed to take account of several recent advances. The guidelines remain conservative in not recommending screening for men until 45 years of age and 55 years in women, which is out of step with screening recommendations for mammography in women at the age of 45 years. Also it would be better if hard clinical endpoints such as total mortality, stroke, and myocardial infarction (MI) are used to predict risk and not soft endpoints such as the development of angina, transient ischemic attacks and heart failure. Furthermore 5-year risk prediction is used rather than 10-year risk.

A 10-year risk is favoured by most guidelines because of the cumulative effects of multiple risk factors over a longer time than 5 years.\(^3\) Also lifetime risk is favoured,\(^4\) as men currently classified as being at low risk (5–10% 5-year risk) have a lifetime risk of 50%, and similarly women classified at low risk have a lifetime risk of 25% for dying from CVD. In addition, total isolated cholesterol levels are used as the metric ($\geq 8$ mmol/L), to identify a 5-year risk $>15\%$ rather than low density lipoprotein (LDL) levels and the targets for LDL levels are conservative in the light of recent data.

The Canadian guidelines recommend,\(^3\) in primary prevention, an LDL level $<2.0$ mmol/L (New Zealand $<2.5$), and more recent data supports an even lower level in certain subgroups. For example in the Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin (JUPITER) primary prevention trial with entry LDLs $<3.4$ mmol/L and high sensitivity C-reactive protein (CRP) levels $\geq 2.0$ mg/L patients who achieved an LDL $<1.3$ mmol/L with rosuvastatin had a 41% reduction in mortality as compared with patients with LDLs $>1.3$ mmol/L.\(^5\)

There are several other deficiencies which include the following: lack of incorporation of socioeconomic factors which have been incorporated in other risk scores; lack of accounting for the effects of treatments, such as statins, antihypertensive agents, and antiplatelet drugs on modifying the extent of risk; lack of emphasis of the importance of the metabolic syndrome; lack of assessment for individuals with inflammatory diseases including rheumatoid arthritis, psoriasis, systemic lupus erythematosus, and human immunodeficiency virus (HIV); lack of discussion of high sensitivity CRP and novel biomarkers such as Lp-PLA\(_2\); and lack
of discussion of imaging modalities such as computed tomographic (CT) angiography, and calcium scoring.

The latter is the focus of a manuscript in this issue of the Journal. In a well performed study in 1000 patients Ellis and colleagues show, in the selected population studied, that the current New Zealand cardiovascular risk tool performs poorly as compared with risk assessed by calcium scoring. CT angiography was also performed. A notable finding was that the New Zealand risk tool misclassified approximately 10% of patients as being low risk when in fact they were at very high risk.

It is alarming that these individuals may have been reassured and may not have received intensive lifestyle advice and perhaps medications. It is also likely that knowledge of the test results may have had an effect on patient acceptance and adherence to lifestyle advice and appropriate pharmaceutical treatments.

The population in this study were selected and skewed from the general New Zealand population with only 1.7% being Māori. The patients were not asymptomatic as the Framingham group from which the risk score used in the New Zealand guidelines was derived, but had atypical symptoms of ischemia or equivocal stress tests. In addition they were probably from a higher socioeconomic group as they could afford to pay approximately $500 for the calcium score and an approximate further $1000 for the coronary artery imaging.

There are some important practical considerations with calcium scoring and CT angiography. The amount of radiation exposure is about 7 millisieverts (a mammogram exposure is about 0.5 millisieverts), equivalent to about 2½ years background radiation exposure and thus repeat imaging at regular intervals is not recommended. Newer equipment and acquisition protocols for calcium scoring and CT angiography have reduced the radiation exposure to <1 millisievert.

There are no New Zealand guidelines for calcium scoring or CT angiography. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines state that calcium scoring is reasonable for risk assessment in patients at low to intermediate risk (6–10% at 10 years) in order to re-stratify risk. The guidelines also state that in patients at <6% 10 year risk that calcium scoring is not recommended and CT angiography is not recommended in asymptomatic patients.

**Does calcium scoring add to risk assessment?**

The Framingham risk assessment tool was published in 1976. There are now over 120 cardiovascular risk scoring methods. New modalities of risk assessment should show improved risk prediction over current methods and improved risk stratification before incorporation into risk models. Calcium scoring achieves this. However, new risk assessment tools should also demonstrate that clinical decision making and outcomes are influenced by their measurement before incorporation into risk models and calcium scoring has not yet achieved this.

A limitation of calcium scoring is that most elderly patients have calcium in their coronary arteries and it is not helpful in patients older than 75 years. Conversely, not all plaques have calcium and it is the minor ‘soft’ plaques without calcium which are the ones that are prone to fissure and rupture and that cause myocardial infarction.
These plaques have a lipid-rich necrotic core and a thin overlying fibrous cap and little calcium. In addition, as these plaques develop the coronary arteries positively remodel and become larger. Therefore when a ‘luminogram’ from a coronary arteriogram or a CT angiogram is performed the lumen may appear normal. Also soft plaques with the potential to rupture may not cause angina or ischemia on stress testing but may cause sudden cardiac death, which may be the first indication of coronary artery disease (CAD) in approximately 30% of asymptomatic individuals, who may have been reassured that they are at low risk with the New Zealand risk assessment tool.

There is thus a paradox, in that with calcium scoring and current CT angiography techniques, these plaques can not be imaged and yet the test is good for predicting risk. This could be explained by the calcium score reflecting the total atheromatous burden, which correlates with the presence of non imaged soft plaques, or it could indicate the presence of previously ruptured plaques (and therefore propensity for rupture to occur again) that have healed with the laying down of calcium.

A low calcium score can reliably rule out significant CAD in patients with atypical symptoms or uninterpretable stress tests as in the current study. CT angiography is not as sensitive (85%) as invasive coronary angiography and has a specificity of approximately 90% for detecting stenoses ≥50% in patients with suspected CAD. CT angiography has enormous potential for ruling out CAD in the emergency department in conjunction with normal high sensitivity troponins.

**Role of myocardial ischaemia in deciding management**

The Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation (COURAGE) trial in 2287 patients showed that percutaneous coronary intervention (PCI) does not improve outcomes in patients with stable angina (without a markedly positive stress test or left main stenosis ≥50%) as compared with optimal medical therapy. In a substudy involving 314 patients of the COURAGE trial PCI reduced ischaemia as detected by positron emission tomography compared to optimal medical treatment. However reducing ischaemia did not reduce cardiac events. Thus the detection of CAD alone or ischaemia should not drive a decision to perform revascularisation with PCI or surgery, but the decision should be based on the potential for the relief of major angina symptoms in addition to the effects of optimal medical therapy.

**Genetic variants**

Although the Framingham risk factors, together with novel biomarkers and imaging modalities are important determinants of risk, genetic variants may account for more than 50% of the susceptibility to CAD. A locus on the 9p21 chromosome has been shown to have a robust association with CAD. Approximately 25% of the population carry two copies of the risk alleles and the risk of CAD is twice as high in carriers. The risk is independent of known risk factors implying a new biologic pathway probably involving smooth muscle cell proliferation. It is possible that a reliable genetic screening test may be able to be developed for this locus.

A future potential role for CT angiography is the characterisation of coronary artery plaques. Newer machines with appropriate software can assess plaque volume and
density which correlates with plaque composition. A recent study in 1000 patients determined whether soft plaques and positive remodelling was present in 1000 patients and followed them for 2 years. Patients with these features had a risk of the occurrence of an acute coronary syndrome of 22% compared to the risk if these features were absent of 0.5%.

Calcium scoring is a valuable risk tool but more evidence on hard clinical endpoints and cost-effectiveness is required. If we could identify soft ‘vulnerable’ plaques with imaging techniques such as CT angiography, and give treatments with statins and perhaps new plaque stabilising therapies such as Lp-PLA₂ inhibitors to stabilise these plaques, prevention of the biggest killer in our community will make a huge leap forward.

Competing interests: None.

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References:

Musculoskeletal undergraduate curriculum: what is required?

Jean-Claude Theis

The article by Thomas Pasley et al in this issue of the NZMJ rightly calls for development of a new undergraduate musculoskeletal curriculum within our medical schools to better prepare medical practitioners to manage the future tsunami of patients suffering from chronic pain and physical disability caused by musculoskeletal conditions.

This crippling burden is directly linked to the ageing of the New Zealand population. Currently 16% of our population is aged over 60 but this is expected to double over the next 50 years. As a result of an increase in longevity and number of elderly citizens, chronic conditions such as osteoarthritis, osteoporosis, back pain, and other degenerative conditions will increase exponentially and put significant pressure on primary and secondary healthcare resources.

The undergraduate musculoskeletal curriculum currently is inadequate and this has been well documented in articles by Freedman et al. One of their study has shown that 75% of medical school graduates failed a basic musculoskeletal competency examination. Woolf et al recommended that core competencies be introduced into musculoskeletal undergraduate courses so that students graduate with knowledge and skills in the management of chronic degenerative conditions and injuries.

Most musculoskeletal attachments during the medical course are limited to 4 weeks which is significantly less than most other similar disciplines. Curriculum committees allocate teaching time not based on educational evidence but influenced by the opinion of some powerful heads of department who argue strongly for their disciplines. This has led over the years to distortion of medical curricula to the detriment of disciplines such as orthopaedics, rheumatology, sports medicine, rehabilitation etc.

There is an urgent need to reform the musculoskeletal curriculum within our medical schools by developing a comprehensive and integrated course which teaches the basic clinical competencies necessary to diagnose and manage patients presenting with common musculoskeletal conditions. In the past most of the teaching has occurred in orthopaedic departments but a more integrated approach is required.

All disciplines dealing with musculoskeletal diseases have to be involved in the curriculum from the preclinical right through the clinical and trainee intern years. This will require increased curriculum time in the clinical years of at least 6 weeks purely dedicated to musculoskeletal teaching. Students need to be exposed to patients with fractures, osteoarthritis, osteoporosis, inflammatory conditions, back pain and others in a hospital as well as community setting.

Rehabilitation medicine is currently underrepresented in undergraduate teaching and this needs urgent attention. The core musculoskeletal competencies must become an
integral part of all summative assessments through the medical course in order to make sure that students are competent by the time they graduate.

As far as the New Zealand medical workforce is concerned there is currently a lack of rheumatologists and rehabilitation physicians which is partly due to insufficient exposure to those disciplines at an undergraduate level. The musculoskeletal skills of general practitioners is also lacking and this has been well documented in a paper by Clawson et al which surveyed 5000 GP trainees. This has contributed to an increasing number of patients being referred into the secondary sector leading to ‘choking’ of orthopaedic and rheumatology clinics.

Many musculoskeletal conditions are self-limiting but occasionally acute conditions need to be recognized early and acted upon promptly such as infections, fractures, tumours and spinal cord compression. Without appropriate training, doctors will miss the diagnosis resulting in poor outcomes for patients and medicolegal risk for the health practitioner. Recognising normal from abnormal is important: a false negative will result in a delayed diagnosis and poor outcome for the patient whereas a false positive can result in inappropriate investigations and treatment leading to unnecessary worries for patients and families.

The Bone and Joint Decade which finished last year has significantly increased the awareness of the musculoskeletal burden, but unfortunately little has filtered down to the undergraduate curriculum. In New Zealand alone it is estimated that one in four adults is affected by disability resulting directly from a musculoskeletal condition to the cost of over 500 million dollars a year. This should certainly rank very high amongst the government’s priorities when planning healthcare delivery in the future.

National disability prevention programmes are urgently needed to deal with the predicted crippling tsunami. A good place to start is at the medical school level through the introduction of a well integrated musculoskeletal course which will teach medical students the basic competencies to allow them to manage these conditions later on in the community and hospitals. This will as a consequence have a positive effect on the workforce as students will have greater exposure to musculoskeletal conditions and more likely to take up specialities like rheumatology, rehabilitation medicine etc.

We need to listen to the recommendations of Thomas Pasley et al and develop a nationally agreed musculoskeletal undergraduate curriculum with clear competencies to be achieved by the end of the trainee intern year through collaboration amongst our medical schools.

Musculoskeletal medicine deserves a prominent place in the medical curriculum equivalent to all the other clinical disciplines. The tsunami warning is loud and clear: we need to act now or it will be too late!

Competing interests: None.

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High calcium scores in patients with a low Framingham risk of cardiovascular (CVS) disease: implications for more accurate CVS risk assessment in New Zealand

Chris J Ellis, Malcolm E Legget, Colin Edwards, Niels Van Pelt, John A Ormiston, Jonathan Christiansen, Helen Winch, Mark Osborne, Greg Gamble

Abstract

Aims New Zealand (NZ) patients are recommended to undergo an ‘adjusted’ Framingham score to assess their cardiovascular (CVS) risk. The current (2009) NZ CVS Risk Guideline does not recommend the use of a ‘calcium score’ as an additional risk tool, although it has been shown to be powerfully predictive of CVS events above the predictive power of traditional Framingham risk factors. Calcium scores of >400 are very strongly predictive of a future CVS event and give direct evidence of atheromatous disease in the coronary circulation. Identification of people with advanced, premature coronary atheroma would allow early treatment of those who may benefit from more vigorous preventative strategies, including statin therapy.

Methods Using a prospectively acquired, comprehensive database we audited the first 1000 patients (7 August 2006 to 28 November 2008) to undergo a 64-slice computed tomographic (CT) cardiac angiogram (GE Light Speed), which included a scan for a ‘calcium score’, at the Mercy Hospital, Auckland. We excluded 58 patients who had experienced one or more of a previous myocardial infarction (MI) (n=21), coronary artery bypass graft (CABG) surgery (n=15), percutaneous coronary intervention (PCI) (n=13) or stroke (n=21) and who therefore already had definite evidence of vascular disease and would be automatically placed in a high risk strata. We calculated each patient’s Framingham risk from the original ‘Anderson’ equation, used by the 1996 NZ CVS risk Guideline, and the ‘adjusted’ Framingham 5-year CVS risk using the NZ Guidelines Group 2003/2009 recommendations, and then compared this with the observed calcium scores.

Results The mean patient age was 56 (SD 9) years; 364 (39%) patients were female, 82% patients were Caucasian. 41% were current (4.6%) or previous (36%) cigarette smokers, 35% had a history of hypertension, 44% hyperlipidaemia and 5.6% had diabetes mellitus. The percentage of patients at ‘low’ 5-Year CVS risk (0-10% 5-year risk), using the 1996 and 2003/2009 guideline methods, was 78% and 58% respectively. Of patients in these Framingham ‘low-risk’ groups, 10% and 8.8% had a calcium score of >400 Agatston units, indicating that they were actually at very high CVS risk, and 203 (28%) and 147 (27%) respectively had a calcium score of >100 Agatston units, indicating that they were actually at ‘high risk’ and not ‘low risk’.

Conclusion Approximately 10% to 27% of patients with a low CVS risk as assessed by the established Framingham equation have a markedly increased calcium score and hence a significantly increased risk of a CVS event. Currently promoted methods of risk assessment may be inadvertently, falsely reassuring these patients. Clinicians...
managing patients may consider a calcium score as an additional tool to the standard risk assessment strategies.

The traditional method of assessing cardiovascular (CVS) risk has been to measure baseline factors in an intermediate-sized cohort of a general population and, after some years, to then estimate which factors are subsequently found to be predictive of CVS events.

The Framingham epidemiological study pioneered many of the methods commonly used in risk estimation, and these have been widely used for the assessment of CVS risk. The major and independent CVS risk factors demonstrated by Framingham are cigarette smoking, elevated blood pressure, elevated serum total cholesterol and low-density lipoprotein cholesterol, diabetes mellitus, male gender and advancing age.

Other studies have been conducted using different population cohorts, such as the larger Prospective Cardiovascular Munster (PROCAM) project which enrolled German industrial employees and included additional potential risk factors. From these epidemiological studies, a variety of calculations have been developed to try to predict the risk of a CVS event.

Another important method of assessing CVS risk is to use an individuals family history of CVS disease, particularly a family history of ‘premature’ CVS disease (first degree relatives: male <55 years, females <65 years), which approximately doubles the risk for an individual. A range of ‘biomarkers' have also been shown to help define CVS risk in a population, including, homocysteine levels and lipoprotein (a). Inflammatory markers, which recognise that atherosclerosis is an inflammatory disease are also useful at assessing risk, with the 'highly sensitive' C-reactive protein (CRP) being widely studied, to date.

'Thrombogenic' risk factors have also been assessed, with the serum fibrinogen level being particularly predictive of CVS risk. Ethnic characteristics have also been assessed outside of the Framingham study, who were largely of white European descent, with the absolute CVS risk of Indians and Pakistanis living in a western society being about twice that of the white European population. In addition, Māori age-specific rates of death from CVS disease are two to three times higher than non-Māori in those aged less than 75 years of age in the New Zealand population.

Further, psychosocial factors have more recently been demonstrated to have a significant impact on the CVS risk of a population. However, with all of these additional risk factors which have been modelled and validated in a variety of populations, there is, as yet, no general agreement as to how they should be included in the CVS risk stratification dataset for daily clinical use.

The development of CT cardiac angiography (CTCA) has been a significant, recent medical development which now allows for direct, non-invasive imaging of an individual's coronary arteries. This ability to assess both the lumen, and also the degree of calcification and atheroma within the walls of the coronary arteries has brought a new dimension to CVS risk assessment. Intuitively, it would seem that there would be a major advantage in being able to determine the amount of atheroma which is actually present in an individuals coronary arteries, and with this, a better ability to predict risk.
The amount of calcification in the coronary artery walls, as assessed by the Agatston ‘calcium score’ has been shown to be highly predictive of CVS risk. A calcium score of ≥100 conferred a 10-fold increase in risk, in the St Francis Heart study of 4,613 asymptomatic people followed for 4.3 years compared with a calcium score of 0. Further, the coronary calcium score alone was superior to the Framingham Risk Score at predicting CVS events (area under the receiver-operating characteristic [ROC] curve of 0.79±0.03 vs 0.69±0.03, p=0.0006), and enhanced the stratification of those falling into the Framingham categories of low, intermediate, and high risk (p<0.0001).

We reviewed the first 1000 patients undergoing a CT cardiac angiogram, which included a calcium score, at the Mercy Hospital in Auckland. We wished to calculate patients’ baseline ‘CVS risk’, as assessed by the previous [1996] and current [2003/2009] New Zealand Guideline recommendations, and the patients’ actual calcium score, to determine if some apparently ‘low risk’ patients were actually ‘high-risk’, and were inadvertently being falsely reassured about their personal CVS risk.

Methods

Data collection—A prospective audit of all patients presenting for computed tomographic (CT) cardiac angiogram was performed. Data were prospectively collected by a practice nurse using a standardised data collection sheet from 07 August 2006 until 28 November 2008. The data collection form recorded patient demographics, personal and family history, medication use and the results from the CT cardiac angiogram and the calcium score. Referrals from cardiologists in Auckland were of patients principally with equivocal exercise test changes, and/or equivocal symptoms.

All CT cardiac angiograms, including the calcium score, were performed by one of three radiographers using a standardised procedure for the 64-slice CT machine (GE Light Speed). The calcium score was derived according to the method of Agatston, with these details incorporated into the CT machine. All patients gave informed consent to undergo the clinical investigation as a part of their clinical management. As an audit of current practice, individual patient consent was not required for this study.

Framingham CVS Risk Scores—The 5-year risk of a CVS event was calculated using two models. The first model was the basic model of Anderson developed using data from the Framingham study with the equation then used in the 1996 New Zealand Guidelines. This model was significantly extended for New Zealand conditions by the New Zealand Guideline Group (NZGG) in 2003 and minimally changed again in 2009. For the basic model, in 1990 Anderson et al used data from 5573 subjects in the original and ‘offspring’ Framingham Heart Study, aged 30 to 74 years. Requirements for inclusion were 1) age 30 to 74 years at the time of baseline examination (from 1968 through 1975); 2) measurements were available for systolic blood pressure (SBP), diastolic blood pressure (DBP), cigarette smoking status, total and high-density lipoprotein (HDL) cholesterol, and the diagnosis (yes or no) of diabetes mellitus, and electrocardiogram (ECG) criteria for left ventricular hypertrophy (LVH); and 3) freedom from CVS disease (stroke, transient ischaemic attack (TIA), coronary heart disease (CHD) [angina pectoris, unstable angina, myocardial infarction (MI), and sudden CHD death], congestive heart failure, and peripheral vascular disease (intermittent claudication) until the time of risk factor measurement. These criteria were also the CVS ‘endpoints’ used in Framingham. The basic Framingham equation used to predict these events incorporated patients’ age, gender, total and HDL-cholesterol, SBP, DBP, smoking, diabetes, and ECG-LVH. The equation allowed CVS risk to be determined from 4 to 12 years into the future. In 1996, the New Zealand Guidelines group developed the CVS risk stratification charts which simplified access to the Framingham risk calculation by the (now familiar) coloured, graphical approach. Although the 1993 New Zealand dyslipidaemia guidelines used a ‘10-year’ CVS risk
period (along with most International Guidelines), a 5-year CVS risk figure was subsequently adopted in 1996.

In 2003, in response to criticism that the New Zealand 1996 Framingham equation did not account for individuals that were obviously at high risk, a set of additional risk factors was formed (Table 1) which permitted a one off 5% increase in the risk estimate in those individuals which had at least one of these risk factors. Very minor changes were then made by the New Zealand Guidelines group in 2009, with the CVS risk assessment being the same, except that 'metabolic syndrome' was removed from the 'additional risk factors' which could give an extra 5% risk, and diastolic blood pressure values were no longer a feature of the coloured charts (the systolic blood pressure was always the parameter actually used in the equation). The NZGG still opted for a 5-year CVS risk time scale.

Table 1. Variables included in CVS risk prediction models

<table>
<thead>
<tr>
<th>Variables</th>
<th>Framingham Anderson</th>
<th>NZGG Framingham Ad Hoc Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Gender</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Smoking Status</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Total Cholesterol/HDL Cholesterol</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>LVH</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

A 'one off' 5% increase if one of the following is present:

- Family history of premature coronary heart disease or ischaemic stroke in a first-degree male relative before the age of 55 years OR a first-degree female relative before the age of 65 years

- Māori OR Pacific* OR people from the Indian subcontinent

- People with both diabetes and microalbuminuria

- People who have had type 2 diabetes for more than 10 years OR who have an HbA1c consistently greater than 8%  

- People with the metabolic syndrome

- IF Total Cholesterol >8 then risk >15%

- IF Total Cholesterol/ HDL >8 then risk >15%

- IF BP consistently >170/100 then risk 15%

* Mostly of Samoan, Tongan, Niuean, or Cook Islands origin.

Statistics—Continuous data are summarised as median and interquartile range or mean and standard deviation as appropriate. Differences in frequencies were tested using Chi-squared procedures or Fisher's exact test as appropriate and differences between groups in continuous variables using the Wilcoxon independent groups test. SAS software (SAS Institute Inc, v9.1) was used to perform the analyses. All tests were two-tailed and a 5% significance level was used.

Results

We examined the first 1,000 patients undergoing CT cardiac angiography and a calcium score at Mercy Radiology, Auckland. We excluded 58 patients with one or more prior CVS events (previous MI (n=21), CABG surgery (n=15), PCI (n=13) or stroke (n=21)), and assessed 942 patients for their CVS risk.

Patient demographics—The mean patient age was 56 (SD 8.9) years, 364 (39%) patients were female, 82% were Caucasian. 381 (40%) were current (4.6%) or
previous (36%) smokers, 35% had a history of hypertension, 44% hyperlipidaemia and 5.6% had diabetes mellitus (Table 2).

Table 2. Baseline patient demographic data (n=942)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age [years] (SD)</td>
<td>56 (8.9)</td>
</tr>
<tr>
<td>Sex (female)</td>
<td>364 (39%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>776 (82%)</td>
</tr>
<tr>
<td>Māori</td>
<td>16 (1.7%)</td>
</tr>
<tr>
<td>Pacific</td>
<td>11 (1.2%)</td>
</tr>
<tr>
<td>Indian</td>
<td>45 (4.8%)</td>
</tr>
<tr>
<td>Asian</td>
<td>27 (2.9%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>30 (3.2%)</td>
</tr>
<tr>
<td>Others</td>
<td>37 (3.9%)</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>43 (4.6%)</td>
</tr>
<tr>
<td>Previous</td>
<td>338 (36%)</td>
</tr>
<tr>
<td>Never</td>
<td>559 (59%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>2 (0.2%)</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>416 (44%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>326 (35%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>53 (5.6%)</td>
</tr>
<tr>
<td>Family history of 1st-degree relative with CVS disease</td>
<td>449 (48%)</td>
</tr>
</tbody>
</table>

The distribution of total calcium scores is shown in Figure 1. 319 patients (34%) had a coronary circulation free from calcium, using the Agatston technique (12). 112 patients (12%) had scores >400 and are considered to both have considerable calcium deposition in the coronary circulation and to be at ‘very high’ risk of a vascular event.

Figure 1. Frequency distribution of total calcium scores (n=942)
The distribution of CVS risk scores (Figure 2a) shows that 45% of patients were predicted to be at ‘very low’ (<5%) and 4% of patients at ‘very high’ (>20%) CVS risk within 5 years, using the 1996 NZGG method, and 15% and 6.6% respective using the 2003/2009 NZGG adjustments (Figure 2b).

Agreement between risk predictors and total calcium score is shown in Figure 3. There is a statistically significant (P<0.0001), although poor correlation (Spearman) between total calcium score and the NZGG (1996) unadjusted (r=0.20) and NZGG (2003/2009) ‘adjusted’ (r=0.15) Framingham 5-year CVS event risk.
There is a wide spectrum of Framingham CVS risk scores in those with a total calcium score of zero. Similarly of the 110 patients with total calcium scores >400, 83 patients had an adjusted Framingham risk score <15% (including 48 patients <10% and 10 patients <5%). Reducing the unadjusted five year risk estimates to bands of risk: low (0-10%), moderate (10-15%) and high risk (>15%) showed 10% of patients calculated to be at low risk of a CVS event in five years to have total calcium scores in excess of 400, indicating very high risk (Figure 4). Application of the NZGG (2003/2009) adjustment reduced this to 8.8%.

Hoff\textsuperscript{19} has provided age and gender-specific percentiles for total calcium score. In our patient group of 942 patients, 109 (12%) had total calcium scores above the 90\textsuperscript{th} percentile for their age and gender, placing them at increased CVS risk, yet they had the same median adjusted Framingham risk score (9.0 [IQR 7.2, 12.0] v 8.7 [IQR 6.2, 12.7 P=0.35]).

For patients calculated by the NZGG 1996 and 2003/2009 CVS risk guidelines to be at ‘low risk’ (0-10%), 14% and 9.1% respectively actually had a calcium score above the 90th percentile for this age and gender matched population, indicating that they were, in fact, at very high risk.
Figure 4. Agatston calcium score by band of 5-year CVS risk estimated by: (A) the original Anderson equation (NZGG 1996) (B) with the NZGG 2003/2009 'adjusted' Anderson equation

A

10% (95% CI 8-12) of patients predicted to be at low 5 year CVS risk have Ca scores > 400

B

8.8% (95% CI 6-10) of patients predicted to be at low 5 year CVS risk have Ca scores > 400
Since an elevated calcium score has been recognised as a potential risk factor for a CVS event and is direct evidence of coronary atherosclerosis, we examined the ability of the NZGG (2003/2009) adjusted Framingham risk score to discriminate those patients with total calcium >100 and >400 Agatston units. Patients with scores >100 or >400 were not well predicted by the adjusted Framingham score with only a moderate ability to discriminate as assessed by the area under the ROC curve (0.57 and 0.61 respectively (Figure 5).

Figure 5. Receiver operating characteristics of the 5-year risk of a CVS event from the Framingham score (with NZGG 2003/2009 adjustments) to discriminate people with total Agatston calcium scores of >100 and >400

Discussion
We have assessed the CVS 5-year risk of a selected New Zealand population, as promoted by the current 2009 NZ Guideline Groups methodology. Within this population who are estimated to be at 'low risk' (0-10%), approximately 10% have a calcium score which at >400 Agatston units actually places them at 'very high' risk of a CVS event, up to 30 times the risk of a population with a calcium score of zero Agatston units.
Further, approximately 25% of this population have a calcium score of >100 Agatston units, which actually places them at 'high' risk of a CVS event, possibly up to 10 times the risk of the population with a calcium score of <100 Agatston units.\(^{f3}\)

This finding is of considerable concern, as the currently promoted methods of CVS risk assessment may, inadvertently, be falsely reassuring 10 to 25% of the 'low-risk' population. Hence, this finding sharply questions the currently employed method of CVS risk assessment in our New Zealand population.

With the new ability to accurately image the coronary arteries, both for calcium and for soft, mixed and fibrous atheromatous plaque, there is the potential to far more accurately assess CVS risk based upon the knowledge of how the very many 'risk factors' actually result in premature atheroma within the coronary arteries.

There has always been something incongruous about the epidemiological methods of CVS risk assessment, as currently promoted. If a clinician wishes to detect a colon cancer, a colonoscopy is undertaken, or a mammogram to identify a breast cancer as he ‘looks for disease’. However, if the premature development of coronary atheroma is to be determined, we have been encouraged to view charts of some major risk factors for the condition. Intuitively, the ability to visualise the degree of an individual's actual coronary atheromatous burden would seem to have the potential for more accurate, CVS risk assessment.

Although early studies comparing the prognostic accuracy of coronary calcium measurement by CT vs the Framingham Score alone, or risk factors alone, yielded conflicting results, subsequent larger reports have conclusively demonstrated the predictive value of a calcium score in CVS risk assessment.\(^{20–24}\) Budoff et al have published the largest cohort (25,253 patients) with the longest mean follow up (6.8±3 years) to date\(^{24}\). Using the end-point of all cause mortality, a calcium score of >10 predicted increased risk, with risk-adjusted relative risk ratios of 2.2 to 12.5 with calcium scores of 11 to 100 through to >1,000.

Ten-year survival (after adjustment for risk factors, including age) was 99.4% for a calcium score of 0 and worsened to 87.8% for a score of >1,000 (p<0.0001). In the St. Francis Heart Study\(^{13}\), in which over 4,500 patients were followed for 4.3 years, a calcium score of >400 was associated with a 30-fold increased risk for myocardial infarction or coronary artery disease death (coronary death, nonfatal myocardial infarction (MI), surgical or percutaneous coronary revascularization procedures, non-hemorrhagic stroke, and peripheral vascular (i.e., arterial) surgery).

In the Prospective Army Coronary Calcium Project, in which younger patients were evaluated with a calcium score and followed prospectively, the calcium score was associated with a 12-fold increased risk for hard coronary heart disease events (p=0.004), even after controlling for the Framingham risk score\(^{22}\).

A recent prospective European study\(^{23}\) enrolled 510 uncomplicated type-2 diabetic patients who underwent a calcium score assessment. The ROC analysis for CVS risk prediction showed that a calcium score had the best area under the curve (0.92), significantly better than the United Kingdom Prospective Diabetes Study (UKPDS) risk score (0.74) and Framingham score (0.60) (p<0.0001). The relative risk (RR) to predict a CVS event for a calcium score of 101 to 400 was 10.1 and increased to 58.1 for scores >1000 (p <0.0001).
Responding to these overwhelming data, that in asymptomatic patients there is an incremental prognostic value of a calcium score, an American College of Cardiology/American Heart Association clinical Expert Consensus Panel and the National Cholesterol Education Program (NCEP) Adult Treatment Panel have recommended coronary artery calcium score assessment in patients at intermediate risk (10% to 20% risk prediction at 10 years) to refine the risk assessment and adjust the intensity of treatment accordingly.

Other recommendations have been published by Hecht et al., and by the Screening for Heart Attack Prevention and Education (SHAPE) guidelines; particularly from the latter, the Texas State legislature has mandated insurance coverage for coronary artery calcium scoring for the intermediate-risk population.

Interest has also been given to patients who are calculated by the Framingham score to be at high CVS risk, but are found to have a low calcium score. The potential to reassign these patients to a lower risk has been discussed, and may have particular relevance to an older population, whose CVS risk in current epidemiological-based risk equations has been largely driven by their advanced age.

The concept that the degree of atherosclerosis actually found in the coronary arteries, rather than using a patients age as a surrogate for this finding, has also been suggested. In our cohort, using the 2003/2009 NZGG, we found 26% of an apparently 'high risk' (5-year CVS event risk ≥15) cohort with a calcium score of zero, which would place them in a low risk (although clearly not a 'no risk') category.

The advantage of using the calcium score to improve on CVS risk prediction has been reviewed in the Multi-Ethnic Study of Atherosclerosis (MESA). During a median follow-up of 5.8 years, a final cohort of 5878 participants experienced 209 coronary heart disease (CHD) events of which 122 were myocardial infarction, death from CHD, or resuscitated cardiac arrest.

The ‘net reclassification index’, which reflects the ability of a ‘new’ risk factor to predict risk over the established methods, was measured for calcium score at a high 25%. In contrast, the widely reported inflammatory marker: CRP has been less able to independently predict CVS events after correction for other risk factors.

One limitation to our study is the fact that some patients were offered a CTCA based on atypical symptoms, or had equivocal stress tests, whereas Framingham patients were reported to be asymptomatic. Therefore the Framingham risk assessment tool may underestimate the risk in our study population. However, these patients did not have clinical angina, as they would not have been offered a CTCA, which is very much used as a 'rule out' coronary disease procedure; the negative predictive power of the CTCA being it's major clinical benefit.

In an editorial article in the Journal of the American College of Cardiology in 2007, Alan Guerci stated that there was “a consistent record of incremental prognostic value of the coronary calcium score, which (then) comprised of more than 300,000 patient years of observation”. He felt that it was time to 'move on' to the “remaining important questions about calcified coronary plaques, prognostic accuracy in minorities, the effect of screening on outcomes, and cost-effectiveness”.
Our New Zealand cohort mimics the overseas experience, and with particular reference to our local methods of assessing risk, there are clearly issues of inaccuracy and false reassurance of significant numbers of the population, which could potentially be alleviated by the use of a coronary calcium score.

**Conclusions**

Approximately 10% to 25% of patients with a low CVS risk as assessed by the traditional Framingham equation, have a markedly increased calcium score and hence, actually, a significantly increased risk of a CVS event. Clinicians managing patients may consider a calcium score as an additional tool to the standard risk assessment.

Further work needs to be done to determine the relationship between standard risk factors and the total calcium score, and the utility of these tests alone, or in combination, to predict future CVS events in a New Zealand population. Indeed 26% of patients classified as high risk by the Framingham model have calcium score of zero suggesting a potentially lower ‘true’ risk.

This study has highlighted that the traditional CVS risk assessment based on age, gender, blood pressure, diabetes, a cigarette smoking history and blood lipids does not concord well with measured coronary calcium scores. In particular CT calcium scoring can identify a group of patients who are at a high likelihood of significant vascular events yet who may have inadvertently been falsely reassured by a low calculated Framingham CVS risk score.

**Potential Conflicts of Interest:** CE, ML, CEd, NvP, JO, JC, HW and MO all received payment for reporting of Cardiac CT scans, from the 'Auckland Heart Group' private cardiology practice, or for working at the Mercy Radiology Cardiac CT scanner. As a part of their private cardiology practice CE, ML, CEd, NvP and JO have a share-holding in the 'Auckland Heart Group', which itself has a minority share in the ownership of the Mercy Radiology Cardiac CT scanner.

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References:


Basing musculoskeletal curriculum changes on the opinions of practicing physicians

Thomas Pasley, Song Chan, Phillippa Poole, Martin Wild, Fiona McQueen

Abstract

Aim To establish which musculoskeletal conditions are deemed to be the most important in clinical practice. To use this information to inform the development of a new musculoskeletal curriculum, with emphasis on common and relevant conditions.

Methods A survey listing 29 musculoskeletal conditions was sent to 150 doctors in Auckland, New Zealand. Doctors from 5 specialties, including general practice, were asked to score each condition on a rating scale from 0 to 7, to determine which conditions were perceived to be the most important as encountered in day-to-day clinical practice.

Results The overall response rate to the survey was 36% and this was predominantly due to the low response rate from general practitioners. Fifteen conditions were given average ratings of 4.5/7 or greater and the top 5 of these were as follows: prolapsed intervertebral disc, hip fracture, mechanical back pain, gout at the great toe and osteoarthritis of the hip.

Conclusion This study has used a consensus approach to identify specific musculoskeletal conditions deemed to be the most important in clinical practice. The information obtained can be used for designing a contemporary and relevant musculoskeletal medical curriculum.

The current decade (2000–2010) has been named the “Bone and Joint Decade” by the United Nations in recognition of the increasing morbidity of musculoskeletal conditions. Recent surveys have shown that 40–67% of the New Zealand population suffers from musculoskeletal pain and that 20% of visits to general practitioners are due to musculoskeletal disorders.

The financial burden of these conditions is considerable. In New Zealand, it has been estimated that the direct health cost of arthritis alone in 2005 was $563 million and the net cost of disability and premature death due to arthritis was approximately $2.56 billion.

As the incidence of musculoskeletal conditions increases, there has been renewed interest in musculoskeletal education. A number of authors have noted that the teaching of musculoskeletal medicine is currently inadequate. This was highlighted by a study from the University of Pennsylvania where 82% of medical school graduates failed to demonstrate basic competency in musculoskeletal medicine.

Another study revealed that only 7% of students from Harvard medical school passed a musculoskeletal competency exam. Limited teaching time was identified as a problem at Canadian medical schools where, on average, only 2.3% of curriculum time was spent on musculoskeletal medicine.
Musculoskeletal curricula need to be reformed to address these deficiencies and to prepare future medical graduates for the range of musculoskeletal conditions they are likely to encounter once they enter clinical practice.\textsuperscript{12,13} To this end, the University of Auckland is currently reforming its musculoskeletal programme so that it will become more clinically relevant.

In this study we have surveyed practicing clinicians who manage these conditions, to find out their opinions on which disorders they regard as most important. This information will then be used to inform curriculum design.

**Materials and Methods:**

The survey questionnaire was designed by a statistician and ethics approval was obtained from the University of Auckland Ethics Committee. After collaboration between a rheumatologist and an orthopaedic surgeon, 29 adult musculoskeletal conditions believed to be the most important to clinical practice were included in the survey.

The survey required doctors to rate the 29 conditions on a rating scale of 1–7 ranging from “least important to clinical practice” (1) to “most important to clinical practice” (7), while 4 was described as “neither important nor unimportant”. A definition of importance was supplied based on “how often these are encountered in daily clinical practice, their significance in terms of impact upon patients’ health and other factors you deem important” (e.g. adverse consequences to the patient if condition is missed).

The study population to be surveyed was chosen from the five clinical specialties most likely to encounter the majority of musculoskeletal problems. The five groups chosen were general practitioners (GPs), general physicians, rheumatologists, orthopaedic surgeons and emergency medicine specialists. It was decided to divide the study population of 100 doctors into proportions which approximately match the five specialty numbers in New Zealand.\textsuperscript{14} It was initially decided to survey 76 GPs, 9 general physicians, 2 rheumatologists, 7 orthopaedic surgeons and 6 emergency medicine physicians.

Doctors included in the survey were chosen from the Auckland region. The surveys were mailed out between January – June 2008 and included a stamped self addressed envelope, a consent form and information on the survey. Reminder packs were sent after 6 weeks if there had been no response. If there was no response after 12 weeks, participants were termed non-respondents.

The initial GP response rate was under 10% and so, in an effort to increase GP numbers, an additional 50 GPs were surveyed. Every specialty had an average score calculated for each of the 29 conditions. These scores were then added together and divided by five to give an average score for each condition across all five specialties. Results were rounded to two significant figures.

**Results**

The response rate to the survey by all doctors was 36% (n=54). The response rate for rheumatologists (n=2), orthopaedic surgeons (n=8) and emergency physicians (n=6) was 100%. General physicians (n=6) had a response rate of 67% and GPs (n=32) had a response rate of 25%. Four surveys returned were incorrectly filled out and were excluded from the data analysis.

The condition which received the highest average score across all specialties was prolapsed intervertebral disc (score of 6.2/7) and this condition received a score of at least 6 by every specialty. It was also given the highest score in orthopaedics (6.4) and emergency medicine (6.2).

The top 5 conditions also included hip fracture, mechanical back pain, gout at the great toe and osteoarthritis of the hip. Fifteen conditions had an average score of 4.5 or greater.
Table 1. Top-15 conditions for “importance”, average weighting across all specialties

<table>
<thead>
<tr>
<th>Condition</th>
<th>GP AVG (n=32)</th>
<th>GM (n=6)</th>
<th>Ortho (n=8)</th>
<th>Rheum (n=2)</th>
<th>ED (n=6)</th>
<th>Average Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prolapsed Intervertebral Disc</td>
<td>6.0</td>
<td>6.2</td>
<td>6.4</td>
<td>6.0</td>
<td>6.2</td>
<td>6.2</td>
</tr>
<tr>
<td>2. Hip Fracture</td>
<td>4.7</td>
<td>6.2</td>
<td>6.1</td>
<td>6.0</td>
<td>6.2</td>
<td>5.8</td>
</tr>
<tr>
<td>3. Mechanical Back Pain</td>
<td>6.3</td>
<td>5.0</td>
<td>5.9</td>
<td>6.0</td>
<td>5.7</td>
<td>5.8</td>
</tr>
<tr>
<td>4. Gout at great toe</td>
<td>5.8</td>
<td>6.3</td>
<td>4.7</td>
<td>6.5</td>
<td>5.2</td>
<td>5.7</td>
</tr>
<tr>
<td>5. Osteoarthritis of the Hip</td>
<td>5.8</td>
<td>6.0</td>
<td>5.9</td>
<td>6.5</td>
<td>4.2</td>
<td>5.7</td>
</tr>
<tr>
<td>6. Neck Pain</td>
<td>6.2</td>
<td>5.0</td>
<td>5.8</td>
<td>5.5</td>
<td>5.7</td>
<td>5.6</td>
</tr>
<tr>
<td>7. Osteoporosis with fracture</td>
<td>5.5</td>
<td>6.2</td>
<td>5.4</td>
<td>6.0</td>
<td>4.5</td>
<td>5.5</td>
</tr>
<tr>
<td>8. Rotator Cuff Syndrome</td>
<td>6.2</td>
<td>4.0</td>
<td>5.9</td>
<td>6.0</td>
<td>5.2</td>
<td>5.5</td>
</tr>
<tr>
<td>9. Degenerative Spinal Disease</td>
<td>5.5</td>
<td>6.0</td>
<td>5.9</td>
<td>5.5</td>
<td>4.2</td>
<td>5.4</td>
</tr>
<tr>
<td>10. Carpal Tunnel Syndrome</td>
<td>5.9</td>
<td>5.2</td>
<td>5.6</td>
<td>6.0</td>
<td>4.3</td>
<td>5.4</td>
</tr>
<tr>
<td>11. Fracture of the Radius</td>
<td>5.3</td>
<td>5.5</td>
<td>4.6</td>
<td>5.0</td>
<td>6.0</td>
<td>5.3</td>
</tr>
<tr>
<td>12. Rheumatoid Arthritis</td>
<td>4.6</td>
<td>5.8</td>
<td>4.6</td>
<td>6.5</td>
<td>4.7</td>
<td>5.2</td>
</tr>
<tr>
<td>13. Ankle Sprain</td>
<td>6.0</td>
<td>3.7</td>
<td>5.5</td>
<td>5.0</td>
<td>5.8</td>
<td>5.2</td>
</tr>
<tr>
<td>14. Meniscal Tear at the Knee</td>
<td>5.7</td>
<td>3.7</td>
<td>5.1</td>
<td>5.5</td>
<td>5.3</td>
<td>5.1</td>
</tr>
<tr>
<td>15. Muscle pain and weakness</td>
<td>4.3</td>
<td>5.0</td>
<td>5.1</td>
<td>4.5</td>
<td>3.8</td>
<td>4.5</td>
</tr>
</tbody>
</table>


The 10 lowest ranked musculoskeletal conditions are shown in Table 2. These received average ratings of ≤ 4/7. There was marked variation between some specialties for some of these conditions. For example lateral epicondylitis was ranked as 5.7 by GPs but only 2.4 by general physicians, lowering its weighted average to 3.9.

Table 2. Lowest-10 ranked conditions.

<table>
<thead>
<tr>
<th>Condition</th>
<th>GP (n=32)</th>
<th>GM (n=6)</th>
<th>Ortho (n=8)</th>
<th>Rheum (n=2)</th>
<th>ED (n=6)</th>
<th>Average Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>20. Congenital Dislocation of the hip</td>
<td>4.9</td>
<td>2.8</td>
<td>5.3</td>
<td>4.0</td>
<td>3.2</td>
<td>4.0</td>
</tr>
<tr>
<td>21. Ankylosing Spondylitis</td>
<td>3.7</td>
<td>4.2</td>
<td>3.6</td>
<td>4.5</td>
<td>3.8</td>
<td>3.9</td>
</tr>
<tr>
<td>22. Rickets and Osteomalacia</td>
<td>3.3</td>
<td>4.6</td>
<td>4.6</td>
<td>5.0</td>
<td>2.0</td>
<td>3.9</td>
</tr>
<tr>
<td>23. Lateral Epicondylitis</td>
<td>5.7</td>
<td>2.3</td>
<td>4.4</td>
<td>4.0</td>
<td>3.0</td>
<td>3.9</td>
</tr>
<tr>
<td>24. Dislocation of the Knee Joint</td>
<td>3.1</td>
<td>2.4</td>
<td>5.8</td>
<td>3.5</td>
<td>4.2</td>
<td>3.8</td>
</tr>
<tr>
<td>25. Hamstring Injury</td>
<td>3.9</td>
<td>2.0</td>
<td>4.6</td>
<td>4.0</td>
<td>3.5</td>
<td>3.6</td>
</tr>
<tr>
<td>26. Brachial Plexus Injury</td>
<td>3.4</td>
<td>3.2</td>
<td>4.8</td>
<td>2.0</td>
<td>4.3</td>
<td>3.5</td>
</tr>
<tr>
<td>27. Trochanteric Bursitis</td>
<td>4.8</td>
<td>2.7</td>
<td>3.0</td>
<td>4.0</td>
<td>2.4</td>
<td>3.4</td>
</tr>
<tr>
<td>28. Umbilical Hernia*</td>
<td>3.6</td>
<td>2.3</td>
<td>4.4</td>
<td>2.0</td>
<td>2.8</td>
<td>3.0</td>
</tr>
<tr>
<td>29. Tarsal Tunnel Syndrome</td>
<td>3.4</td>
<td>1.3</td>
<td>4.2</td>
<td>1.5</td>
<td>2.2</td>
<td>2.5</td>
</tr>
</tbody>
</table>

*umbilical hernia was included as a “musculoskeletal condition” in this survey as the anatomy of the abdominal wall is taught as part of the musculoskeletal anatomy curriculum in the Auckland medical course.
Discussion

This study has used a collaborative approach to determine which areas of musculoskeletal medicine should be taught within a New Zealand medical school curriculum. We have designed a simple system to score “clinical importance” for a list of musculoskeletal conditions and have then surveyed clinicians from five medical specialties including general practice, for their opinions. We have deliberately included practitioners from primary, secondary and tertiary care in our sample to avoid a bias towards hospital-based practice.

The majority of medical curricula are designed by hospital or university based staff, with disproportionately little input from primary care physicians, yet approximately half of all senior doctors in NZ work in primary practice.

Using an arbitrary cut-off score of 4.5, we were able to identify 15 conditions regarded as moderately or highly relevant by our sample of doctors. We propose that case-based scenarios, exploring the diagnosis and management of these conditions, could provide a clinically relevant “backbone” for a new musculoskeletal curriculum.

Appropriately, spinal disorders figured prominently amongst the list of conditions regarded as most important. Prolapsed intervertebral disc was ranked at the top when scores were averaged across specialties, while neck and mechanical back pain were in the top 10.

Rotator cuff syndrome was also regarded as very important. These findings are consistent with those described by Taylor et al, who found that the commonest sites for musculoskeletal pain were the back, shoulder, and neck.

It is likely that the clinicians rated “importance” largely according to the frequency with which they encountered these conditions in clinical practice. However, when designing a curriculum, other factors need to be taken into account when deciding what is to be taught.

Thus, disorders such as ankylosing spondylitis, which received a score of 4 and was therefore “less important” on weighted average, would be regarded by most rheumatologists as an essential addition because of its significant impact on the patient and the potential for effective management with new biological therapies.

Basing curriculum design on physician opinion has been used in Britain, where the Regional Examination of the Musculoskeletal System (REMS) study aimed to develop a set of “core musculoskeletal examination skills” for medical students. Surveys with a list of examination skills were sent to a similar sub-group of specialists including rheumatologists, orthopaedic surgeons, general practitioners and geriatricians.

Doctors were asked to rate each skill from 1 (definitely not required) to 5 (essential). From this study, 50 clinical skills were identified as being “core” examination skills for British medical students. The trend in modern medical curricula is to move away from traditional didactic teaching methods and towards a clinically orientated, problem based approach. Problem based learning has recently been shown to have positive effects on physician competency and satisfaction as well as positively influencing medical students’ attitudes and perception of clinical practice.
Our study surveyed clinicians from the Auckland region but the results are likely to reflect medical opinions nationwide for most musculoskeletal conditions. The one exception may be the emphasis placed on gout, as this condition is particularly prevalent in the Auckland region due to a relatively high Maori and Polynesian population. To capture information from the entire medical community, future surveys should also incorporate practitioners practicing in the rural environment as well as smaller urban centres.

The overall response rate of 32% was mainly due to the low response rate from GPs. This has been recognized in previous studies and reflects the day-to-day pressures of clinical practice. Web-based or email-based surveys may help overcome this barrier and could improve response rates in future studies.

Another weakness of the current study was the relatively arbitrary way in which the list of conditions was drawn up, based on the opinions of 2 clinicians. However, the fact that our results identified a group of conditions similar to those described by Taylor et al as mentioned above, does give some validity to this approach. Future surveys should also include the opportunity for the respondent to suggest extra conditions that they felt should have been included in the list.

In summary, we present here the results of a survey of medical practitioners based in the Auckland region, ranking the perceived importance of musculoskeletal conditions encountered during their day-to-day clinical practice.

The 15 conditions proposed as the most important could form a “core set” around which a new musculoskeletal curriculum could be based, with extension to include other rheumatologic and orthopaedic conditions as deemed appropriate. Reforming the medical curriculum in this way should ensure that its content is more clinically relevant and will better equip future doctors to deal with the challenges of musculoskeletal medicine in primary, secondary and tertiary care.

Competing interests: None.

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Acknowledgement: We thank Professor David Thomas (Department of Community Health, University of Auckland) for helping to design the questionnaire.

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References:


Needlestick injuries in a healthcare setting in New Zealand

Marie Fullerton, Veronique Gibbons

Abstract

Aim The aim of this study was to quantify the extent of needlestick underreporting, to examine factors which may contribute to underreporting, and to optimise the relevant risk management strategy.

Method An 11-item structured postal questionnaire was adapted from an existing CDC design.

Results The survey results showed that 9% of respondents had experienced at least one needlestick injury in the past year, and three practitioners had five or more injuries in the same period. The overall underreporting rate for needlestick injuries was 33%, which is consistent with internationally-reported figures. More than one in six respondent doctors (17.8%) had sustained one or more needlestick injuries in the past year, compared with nurses (7.6%) or midwives (6.7%).

Conclusion The survey identified the level of underreporting and the factors that influence needlestick reporting. This has resulted in a series of recommendations that will help our DHB to formulate an appropriate strategy to manage needlestick incidence and impact.

Needlestick injuries are a potential risk to health professionals who use needles and syringes in their clinical work. The New Zealand Health Strategy includes a framework and action plan to reduce the incidence and impact of infectious disease in an occupational setting. Needlestick prevention protocols are an important element of the action plan since they aim to reduce the incidence and impact of blood borne virus (BBV) transmission in the healthcare setting.

Exposure to a BBV such as Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), and the Human Immunodeficiency Virus (HIV) are of concern due to the potentially serious consequences of contracting an infection. The incidence of occupational transmission following percutaneous exposure from an infected source for HIV, HCV and HBV are 0.3%, 3%, and 30% respectively.

The Waikato DHB implemented a needlestick injury prevention program consisting of staff education, safe work practices, point of use sharps disposal units, and the implementation of needle safe devices. Despite these interventions statistics showed that the needlestick injury reporting rate had stayed relatively consistent over the last 5 years. There was anecdotal evidence that some needlestick injuries went unreported by health professionals at Waikato DHB, however, the magnitude of the problem was unknown.

Underreporting of needlestick injuries are estimated at between 30-51%. Furthermore, there are suggestions of cultural differences in reporting behaviours.
between disciplines. Nurses were more likely to report, whereas the medical culture favoured ‘in-house’ incident management.

The aim of this study was to quantify the extent of needlestick underreporting, to examine factors which may contribute to underreporting, and to optimise the relevant risk management strategy.

Method

A postal questionnaire, adapted from an existing CDC design, was sent to all staff within Waikato DHB to be returned anonymously through internal mail. Five new questions were added and piloted before the final questionnaire was agreed. The questionnaire contained 11-items, with close-ended questions seeking dichotomous, multiple, and numeric responses.

The objective of the questionnaire was to establish the number of needlestick injuries sustained in the past 12 months, and the level of reporting. Respondents who had a needlestick injury which had not been reported had the option of identifying the reasons for not doing so. The questionnaire also asked about occupational group, age, gender, total number of years working with needles, types of needles that they are currently exposed to, and knowledge of the organisational policy.

The study population (N=2734) included all doctors, nurses and midwives employed at Waikato DHB in June 2007. Nurses and midwives were identified separately as they are recognised as different disciplines and identify with independent registration associations. At that time, the Waikato DHB employed doctors (n=576), nurses (n=2058), and midwives (n=100).

A sample-size analysis determined that a minimum of 521 nurses, 71 midwives and 133 doctors were needed to be surveyed to show disparity between reported and actual incidence of needle-stick injuries based on a 50% rate of non-reporting. However, the response rate from postal questionnaires can be as small as 5% to 30%, particularly for doctors. Therefore, a decision was made to sample the full population of medical, nursing and midwifery staff employed by the Waikato DHB.

Data were checked and edited before analysis. Descriptive data were analysed by profession. Logistic regression was used for the likelihood of needlestick injuries by years of working with needles and gender. Analyses were performed in Microsoft Excel (Microsoft, 2003) and SAS v9.1 software. Ethics approval was provided by the University of Auckland Human Subjects Ethics Committee.

Results

In total, 2734 questionnaires were sent out. The total number returned was 1346, a response rate of 49.2%. By profession the response rate was: midwives 75% (95% CI: 66.5, 83.5), Doctors 36.8% (32.8 - 40.7) and nurses 51.4% (49.52 - 53.57).

The age of respondents ranged from 20-60+years. The median age was 44 years. 84% of respondents were female. By profession, the majority of nurses (94%) and all midwives were female while 34% of doctors who responded were also female.

Familiarity with the needlestick policy—In total, 95.9% of respondents claimed an awareness of the Waikato DHB policy for reporting blood and body substance exposure. 2.6% were not aware of the policy and 1.5% did not know. The response by profession demonstrated that over 1 in 10 doctors (10.8%) were not familiar with or did not know about the policy compared with 2.9% of nurses and 1.3% of midwives.

Four out of five respondents (80.7%) were familiar with the process for reporting exposures, while 1 in 5 were not (19.3%). By profession, doctors (46.9%) were over three to five times less likely to be familiar with the process than nurses (14.5%) and midwives (9.3%).

When asked who their first line of contact would be following a needlestick injury which exposed them to blood or body fluid, the majority of respondents reported to...
their supervisor or manager (55.7%) followed by Health and Safety (23.9%). The emergency department (11.1%) and others (9.2%), including infection control, made up the remainder.

According to the relevant policy in 2007, the first point of contact after an exposure to blood or body fluid is the Health & Safety Service. Only 24% of all respondents were aware of this, despite 81% of respondents being familiar with the process for reporting exposures.

Needlestick frequency—A total of 123 out of 1346 respondents reported one or more needlestick injuries in the past 12 months (positive responders). By profession, 65 needlestick injuries occurred in 38 of 213 doctor respondents, 97 needlestick injuries occurred in 80 of 1058 nurse respondents and 6 needlestick injuries occurred in 5 of 75 midwife respondents.

This showed that the percentage of individuals that sustained a needlestick injury by profession was greatest for doctors at 17.8% (38/213), compared with nurses or midwives at 7.6% (80/1058) and 6.7% (5/75) respectively. This equated to over one in six respondent doctors sustaining one or more needlestick injuries in the past 12 months.

Of these 123 positive responders, a total of 168 separate incidents were recalled. These ranged from 1-6 needlestick injuries per person (Figure 1). The majority of positive responders had only one needlestick injury in the 12-month period (93/123, 76%).

Figure 1. Number of needlestick injuries by respondents in a 12-month period

Underreporting by profession—Underreporting varied according to profession with 50% of needlestick injuries being unreported by midwife respondents compared with 40% of doctor respondents and 26% for nurse respondents. (Table 1). In total, one-third of the needlestick injuries (55/168, 33%) were not reported.
Table 1: Rate of underreporting by profession

<table>
<thead>
<tr>
<th>Profession (n=total responders)</th>
<th>Total number of needlestick injuries (n=168)</th>
<th>No. of needlestick injuries unreported (n=55)</th>
<th>Percentage of needlestick injuries not reported by profession</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midwife (n=75)</td>
<td>6</td>
<td>3</td>
<td>50%</td>
</tr>
<tr>
<td>Doctor (n=213)</td>
<td>65</td>
<td>26</td>
<td>40%</td>
</tr>
<tr>
<td>Nurses (n=1058)</td>
<td>97</td>
<td>26</td>
<td>27%</td>
</tr>
</tbody>
</table>

**Reasons for not reporting**—Reasons for not reporting included a lack of time to carry out the reporting procedure which was cited by 16% of respondents. The perceived low risks of contracting a BBV from a patient or from using a particular procedure were cited by 16% and 11% of respondents respectively. Other reasons such as lack of importance, unaware of procedure, blame or trouble, ‘nothing more can be done’, and confidentiality concerns were also given.

**Multivariate analysis**—Logistic regression analysis revealed that females have an almost 50% lower likelihood of experiencing a needlestick injury in any particular year. There was also a reduction in the risk of needlestick injury of 18% for every 10 years of experience with handling needles.

**Discussion**

This study reports on the rate of non-reporting of needlestick injuries within a large District Health Board. The sample population included all of the doctors, nurses and midwives employed at Waikato DHB as of June 2007. There was no sub-sampling from this population, consequently the results are more likely to be transferable to other, similar settings within the health industry.

This study used a questionnaire that was adapted from a proven CDC design with demonstrated external validity. The overall response rate of this questionnaire was just over 49%. This compares to reported response rates as low as 5% to 30% for mailed questionnaires.

The relatively high response rate in this particular case is probably due to a combination of several factors:

- The questionnaire was short, with only eleven questions that took 2–3 minutes to complete.
- Departmental managers were informed of the questionnaire and its purpose in advance, and were asked to remind their staff to complete it.
- The subject matter involved personal safety in an area of recognised risk.
- The questionnaire was circulated in personalised envelopes with a typed name.
- All responses were anonymous.

It is interesting to note the high response from the midwifery group (75%). The majority of Midwives employed by the Waikato DHB are managed from a single central point, which may have helped to secure a good response rate. The response rate from nurses and doctors was 51.4% and 36.8% respectively.
Despite review by peer groups, a face validity test and a pilot trial, the questionnaire contained an ambiguity that was revealed during data analysis. While 123 individuals responded to having sustained any type of needlestick injury in the past 12 months, an additional 44 respondents who did not have a needlestick injury in the past 12 months, responded to the question which asked the reasons for not reporting their injury. These 44 ‘extra’ responses were excluded from the analysis.

Over 9% of the respondents (123 individuals) had experienced a needlestick injury in the past twelve months. The incidence rate varied according to profession, with around 17.8% of respondent doctors, 7.6% of respondent nurses and 6.7% of respondent midwives sustaining one or more needlestick injuries in the previous 12 month period. Doctors and midwives had a high underreporting rate (40% & 50% respectively) meaning both are an occupational group that are most at risk.

Of the 123 respondents who sustained a needlestick injury in the past 12 months, 93 experienced one event while 27 respondents sustained two or three needlestick injuries. A further 3 respondents sustained five or six injuries. The confidential and anonymous nature of the questionnaires makes it impossible to identify these individuals. However, follow-up dissemination of the data will provide encouragement for these particular individuals to come forward so that their individual risk factors can be better understood.

Our finding of a 33% non-reporting rate fits with current literature on this topic. Underreporting rates varied substantially between studies and occupational groups, however the consensus point to a rate of 30-35%. A lack of time for reporting is consistent with other studies, one which reported that only 9% of doctors reported their needlestick injuries and the main reason for not reporting was ‘too little time’.

A sample-size calculation was undertaken prior to the study which determined that a minimum of 521 nurses, 71 midwives and 133 doctors were needed to be surveyed to show disparity between reported and actual incidence of needle-stick injuries based on a 50% rate of non-reporting. We achieved these targets yet there may be sampling bias in the design of the study. As an anonymous survey, we are not privy to reasons that may have motivated individuals to participate in this survey. We therefore cannot comment on whether there may be underestimation or overestimation of results.

An underestimation of risk has previously been identified: in one study, 52% of doctors underestimated their risk of acquiring HIV infection, and 70% underestimated their risk of contracting Hepatitis B. Furthermore, another study reported that 70% of doctors and 39% of nurses cited a perceived low risk of transmission of BBVs. Directed education to address the perceptions of staff about risks of BBVs and to highlight the health benefits of reporting needlestick injuries may increase reporting compliance.

A New Zealand study reported that the best predictors of reporting compliance were the perceived severity of acquiring a disease, the perceived efficiency of the reporting system and overall motivation to maintain their health.

The needlestick-reporting process needs to be reviewed to ensure it is easy to access and risk assessments are carried out quickly. Only 24% knew that the first contact
after an exposure is the Health & Safety department suggesting familiarity with the relevant DHB policy is quite poor.

One reason may be confusion caused by the existence of a different policy for incident management, which has the supervisor as the first point of contact. It is vital that a review of the current needlestick policy is undertaken to align the reporting responsibilities with the organisations operational systems.

When examining policy familiarity by profession, almost half (46.9%) of the respondent doctors did not know how to report a needlestick incident. This compares with nurses (14.5%) and midwives (9.3%). It is tempting to invoke poor organisational communication as a possible explanation for the lack of familiarity among doctors. However, issues such as non-familiarity with surroundings (i.e. locum) may be involved.

In addition, reasons for high injury rates and high underreporting for doctors has been linked to ‘the medical culture’ favouring ‘in-house’ incident management; citing a culture that is less transparent and less resilient to directives and protocols. For midwives who did not report, they felt resigned to the fact that nothing further could be done.

This study used logistic regression modelling to establish the impact of various exploratory variables on the incidence of needlestick injuries. The use of combinations of these predictors in different models has reduced the possibility of non-causality, but absolute causality is notoriously difficult to establish. Females are reported to have an almost 50% lower likelihood of having a needlestick injury.

A reduction in the risk of needlestick injury of 18% for every 10 years of experience with handling needles was also identified. In a survey of 11,516 nurses, it was found that nurses with less than 5 years experience, those performing venepuncture, and perioperative nurses were more likely to sustain a needlestick injury. In addition, nurses learning new skills and their lack of experience increase their risk of needlestick injury.

The critical factors involved in lowering the risk of exposure were, support for safe practices, and using specialised staff to perform high risk high frequency procedures. These studies support the hypothesis that the number of years of experience is a predictor in the incidence of needlestick injuries. Educational programmes will need to ensure that those learning new skills are able to obtain a level of competency and have appropriate supervision and support as skills are developed.

**Conclusions**

The level of underreporting was 33% for all respondents, which is consistent with internationally-reported figures. Both needlestick incidence and high levels of underreporting demonstrate that doctors are a group that will require priority attention of future risk management strategies. The perception of low risk by patient or procedure is another area that requires improved educational information.

Furthermore, ensuring that staff are familiar with processes and ensuring these are accessible and timely are fundamental to improving reporting. These processes must work adequately throughout the 24-hour day.
This study served to quantify the level and rationale for non-reporting of needlestick injuries. Results from the study will inform policy and enable targeted education to reduce the unnecessary burden on health resources within the Waikato District Health Board.

**Competing interests:** None.

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**References:**
Healthcare services funded by Counties Manukau District Health Board for people in the last year of life

Wing Cheuk Chan, Gary Jackson, Doone Winnard, Philippa Anderson

Abstract

Introduction The last year of life is often associated with a high level of healthcare utilisation and cost. To date, little information is available regarding the healthcare utilisation patterns in the last year of life in New Zealand.

Aim To describe the healthcare utilisation patterns and costs of the residents of Counties Manukau District Health Board (CMDHB) region in the 1-year period prior to death in 2008.

Method CMDHB residents who died in 2008 were identified from the National Mortality Dataset. The health services utilisation patterns and costs in the last year of life were derived from National Minimum Dataset (NMDS), Pharmaceutical Collection, Laboratory Claims Collection, and National Non-Admitted Patient Collection via encrypted NHI linkage.

Results Forty percent of all deaths in 2008 in CMDHB occurred in a publicly funded hospital. Just over 80% of people had at least one inpatient hospital stay in the last year of life. More than 75% of the healthcare costs funded by CMDHB in the last year of life were related to inpatient hospitalisations. The average cumulative length of inpatient stay over the year in the people who had an inpatient event was 20.6 days. Outpatient, pharmaceutical, and laboratory services were received by 84%, 91%, and 86% of people respectively in their last year of life.

Conclusion Consistent with the international literature, this study found that CMDHB residents in the last year of life have a high level of health service utilisation. Decisions about the appropriate use of high cost health services in people towards the end of life can be extremely challenging. These decisions are resource allocation decisions as well as clinical decisions and should be based on clinical factors, cost utilities, and patient, family, and society’s expectations.

The last year of life is often associated with a high level of healthcare utilisation and cost.1–5 Indeed, a disproportionate share of healthcare funding is spent in the last of life in many developed countries.6–8 The observations from international literature suggest that providing a high level of care may not necessarily translate to improvement in health outcomes.9

In the context of the ageing population in New Zealand, the end-of-life issues in people with chronic disease will become more common. To date, little information is available regarding the healthcare utilisation patterns in the last year of life in New Zealand.

This study aims to describe the healthcare utilisation patterns and costs of the residents of Counties Manukau District Health Board (CMDHB) region in the 1-year...
period prior to death in 2008. CMDHB covers the southern third of Auckland City with a population of around 490,000, making up around 11% of the New Zealand population.

Method

The routinely collected administrative datasets were sourced from New Zealand Health Information Service (NZHIS). We examined the National Mortality Dataset to identify the CMDHB residents who died in 2008. Record linkage was made via encrypted National Health Index (NHI) to the Pharmaceutical Collection, Laboratory Claims Collection, National Minimum Dataset (NMDS, ‘inpatient hospital events’), and National Non-Admitted Patient Collection (‘outpatients’).

The encrypted form of NHI was used to ensure privacy and anonymity of individuals. As all datasets were entirely based on anonymous administrative data no formal ethical review was required as per New Zealand ethical guidelines.

The numbers of community pharmaceutical and laboratory claims, and inpatient and outpatient events as well as their associated costs were collated for the last year prior to the date of death for each individual. Each of the inpatient events was categorised into one of the diagnostic groups as per Table 1.

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>ICD codes (version 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease (including coronary heart disease, stroke, peripheral vascular disease and heart failure)</td>
<td>I20x to I25x, E1053, E1153, E1059, E1159, E1453, E1459, I60x to I69x, and G45x to G46x, I70x to I79x and E1050, E1051, E1052, E1150, E1151, E1152, E1450, E1451, E1152. I50x, I11.0, I13.0, I13.2</td>
</tr>
<tr>
<td>Malignant cancer</td>
<td>C00 to C97</td>
</tr>
<tr>
<td>Chronic respiratory disease</td>
<td>J41 to J47, E84, Z942.</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>B18, B19, K70 to K77, I85, Q446-Q448, T864, Z944.</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>N01 to N19, N25-N29, T861, Z992, Q601, Q604, Q606, Q611, Q619</td>
</tr>
<tr>
<td>All other causes of hospitalisation</td>
<td>Any episode that did not have any of the other named ICD codes above in the primary or secondary diagnostic fields</td>
</tr>
</tbody>
</table>

These diagnostic groups were chosen to determine the relative burden of these common diseases in the last year of life. Formal diagnosis coding is not available in outpatient, pharmaceutical or laboratory databases. The diagnostic category refers to the discharge diagnosis of people who had a publicly funded inpatient event within the last year of life. Both primary and secondary codes were searched for a diagnosis related to the first five categories.

To avoid double counting, each hospital event was assigned with only one of the six categories listed. If a hospital event had more than one diagnosis from the first five categories then the diagnosis taken was based on the order of the hospital diagnostic codes in which they appear for the hospital event. One person may have more than one type of hospital admission during the last year of life. This means one person may be classified into multiple diagnostic categories.

All cost estimates were derived from the NZHIS datasets. Total healthcare costs refer to the inpatient and outpatient events (including ED attendance), community pharmaceutical dispensing and community laboratory tests in the year prior to death. Cost estimates were based on the national cost weights in 2008.
Results

Hospitalisation—A total of 2290 deaths occurred in 2008 in CMDHB. In the 12-month period prior to death there were 6296 inpatient hospital events associated with 1835 people. Therefore there were 455 (20%) people who did not have a public hospital admission in the year prior to death.

Malignant cancer had the highest number of hospitalisations partly because malignant cancer was associated with the highest number of disease specific readmissions (average 3.4) (see Table 2).

Table 2. The number of hospitalisations in the last year of life of people who died in 2008 by disease category

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Number of hospital admissions</th>
<th>Number of people</th>
<th>Average number of disease-specific hospitalisations per patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease</td>
<td>1519</td>
<td>818</td>
<td>1.9</td>
</tr>
<tr>
<td>Malignant cancer</td>
<td>2168</td>
<td>640</td>
<td>3.4</td>
</tr>
<tr>
<td>Chronic respiratory disease</td>
<td>308</td>
<td>149</td>
<td>2.1</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>63</td>
<td>33</td>
<td>1.9</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>375</td>
<td>213</td>
<td>1.8</td>
</tr>
<tr>
<td>All other causes</td>
<td>1863</td>
<td>945</td>
<td>2.0</td>
</tr>
</tbody>
</table>

As noted, this study assigned only one disease diagnostic category to each of the inpatient event based on the order of the discharge diagnostic codes. Table 3 describes the number of people who had different disease categories of inpatient hospitalisations. Many people have more than one disease that had contributed to the inpatient events. For example, 651 people had 3010 hospitalisations in the last year of life falling into two different disease categories. The more diagnoses a person has, the higher the number of hospitalisations occurring in the last year of life. For example, 10 people with 4 different diagnosis categories apiece had a total of 94 inpatient events (an average of 9.4 admissions per person).
Table 3. Hospitalisation number in the last year of life stratified by the number of different chronic disease categories per person

<table>
<thead>
<tr>
<th>Number of hospitalisations with different disease categories</th>
<th>Number of people</th>
<th>Total number of hospitalisations</th>
<th>Average number of hospitalisations per patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>455</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>1034</td>
<td>2292</td>
<td>2.2</td>
</tr>
<tr>
<td>2</td>
<td>651</td>
<td>3010</td>
<td>4.6</td>
</tr>
<tr>
<td>3</td>
<td>139</td>
<td>890</td>
<td>6.4</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>94</td>
<td>9.4</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>10</td>
<td>10.0</td>
</tr>
<tr>
<td>Total</td>
<td>2290</td>
<td>6296</td>
<td>2.7</td>
</tr>
</tbody>
</table>

Proportion of deaths that occurred in a publicly funded hospital—Overall, 40% of all deaths in CMDHB occurred in a publicly funded hospital. The proportion of deaths that occurred in hospital varies with age. The highest was in the under-5 age group, with more than 70% of all deaths occurring in a hospital.

Figure 1. Percentage of all deaths in CMDHB in 2008 that occurred in a publicly funded hospital stratified by age
Length of stay—Of those 1835 people who had a hospital admission in the last year of life, the mean and median cumulative length of stay over the year were 20.6 and 13.0 days respectively. There were 125 people who had hospital admissions but did not have an overnight stay, i.e. length of stay = 0. The total length of hospital stay in the year prior to death varies widely between individuals ranging from 0 to 313 with a standard deviation of 27.9 days.

Table 4. Statistical analyses of the total length of hospital stays in the last year of life of people who died in 2008

<table>
<thead>
<tr>
<th>Length of hospital stay analyses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of people</td>
</tr>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>Standard Error</td>
</tr>
<tr>
<td>Median</td>
</tr>
<tr>
<td>Standard Deviation</td>
</tr>
<tr>
<td>Minimum</td>
</tr>
<tr>
<td>Maximum</td>
</tr>
</tbody>
</table>

Outpatients, pharmaceutical and laboratory service utilisation—The number of people who utilised the service and the volume of service utilised are shown in Table 5. For those people who attended an outpatient clinic, the average number of clinics attended in the last year of life was 12.9.

Table 5. Outpatients, pharmaceutical and laboratory service utilisation in the last year of life

<table>
<thead>
<tr>
<th>Type of service</th>
<th>Number of people who utilised the service (% out of total 2290 people in study)</th>
<th>Volumes of service used</th>
<th>Average number of service per patient in the last year of life</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outpatients</td>
<td>1935 (84%)</td>
<td>24,866 clinics</td>
<td>12.9 clinics</td>
</tr>
<tr>
<td>Pharmaceutical</td>
<td>2092 (91%)</td>
<td>177,102 prescription items dispensed</td>
<td>84.7 prescription items dispensed</td>
</tr>
<tr>
<td>Laboratory</td>
<td>1967 (86%)</td>
<td>85,257 individual lab tests</td>
<td>43.3 individual lab tests</td>
</tr>
</tbody>
</table>

Healthcare cost in the last year of life—CMDHB spent a total of $51.2 million on health care for the 2290 people who died in 2008 during their last year of life. This consisted of $38.8 million on inpatient hospitalisations, $7.8 million on outpatients, $4 million on community-dispensed pharmaceuticals and $680,000 on community laboratory tests.

While the average health care cost per person in the last year of life was $22,376 per person, the range of health care costs spent varied widely (Table 6). There were 69 people who did not access any of health care services recorded by the study in their last year of life. On the other hand, there were 61 people (2.7%) who had health care
costs greater than $100,000 in the last year of life, accounting for a total of 8.7 million (17% of the total cost).

Table 6. The health care cost per person in the last year of life

<table>
<thead>
<tr>
<th>Total health care cost per person</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>$22,376</td>
</tr>
<tr>
<td>Standard Error</td>
<td>$666</td>
</tr>
<tr>
<td>Median</td>
<td>$13,412</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>$31,859</td>
</tr>
<tr>
<td>Range</td>
<td>$782,111</td>
</tr>
<tr>
<td>Sum</td>
<td>$51,200,000</td>
</tr>
<tr>
<td>Number of people</td>
<td>2,290</td>
</tr>
</tbody>
</table>

The total health care cost by age groups—The average health care cost per person in the last year of life varies by age (Figure 2). Children from 28 days to 9 years, and adults between the ages 50 and 79 years are associated with the highest health care cost (blue line in Figure 2). The health care cost in the last year of life progressively falls after 80 years of age.

Figure 2. The number of deaths in CMDHB in 2008 and the average health care cost per person in the last year of life stratified by age category (error bars: 95% confidence interval)
**Hospitalisation cost**—There were 1835 people (80%) who received inpatient care in the last year of life in 2007/08. CMDHB spent just under $38.8 million on inpatient hospitalisations for this group. Inpatient care related to cancer was associated with the highest average inpatient cost in the last year of life. Note that the overall average cost ($21,100) per patient who utilised inpatient services was higher than the average cost per patient associated with any of specific diagnostic category because one person can have multiple hospitalisations with different diagnoses (Table 7).

Table 7. The hospitalisation costs related to last year of life by disease category

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Average cost per hospital admission ($)</th>
<th>Cost over the last year of life per patient who had utilised inpatient services ($)</th>
<th>Number of patients</th>
<th>Total cost by disease category ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease</td>
<td>8242</td>
<td>15,300</td>
<td>818</td>
<td>12,520,000</td>
</tr>
<tr>
<td>Malignant cancer</td>
<td>5367</td>
<td>18,200</td>
<td>640</td>
<td>11,636,000</td>
</tr>
<tr>
<td>Chronic respiratory disease</td>
<td>5026</td>
<td>10,400</td>
<td>149</td>
<td>1,548,000</td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>7599</td>
<td>14,500</td>
<td>33</td>
<td>479,000</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>8284</td>
<td>14,600</td>
<td>213</td>
<td>3,106,000</td>
</tr>
<tr>
<td>All other causes</td>
<td>5093</td>
<td>10,000</td>
<td>945</td>
<td>9,488,000</td>
</tr>
<tr>
<td>Overall (people who had hospitalisation)</td>
<td>6159</td>
<td>21,100</td>
<td>1,835</td>
<td>38,777,000</td>
</tr>
</tbody>
</table>

**Outpatient, pharmaceutical and laboratory costs in the last year of life**—There were 1935 people who had utilised outpatient services in the last year of life. The average cost per patient over the year was about $4000.

Table 8. The average outpatient, pharmaceutical and laboratory costs per patient in the last year of life

<table>
<thead>
<tr>
<th>Type of service</th>
<th>Number of people who utilised the service</th>
<th>Total cost of service ($)</th>
<th>Average cost per patient who utilised the service ($)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outpatients</td>
<td>1935</td>
<td>7,785,000</td>
<td>4023</td>
</tr>
<tr>
<td>Pharmaceutical</td>
<td>2092</td>
<td>3,999,000</td>
<td>1911</td>
</tr>
<tr>
<td>Laboratory</td>
<td>1967</td>
<td>681,000</td>
<td>346</td>
</tr>
</tbody>
</table>

Outpatients, pharmaceutical or laboratory databases do not have formal diagnosis coding. The diagnostic category in Table 9 refers to the discharge diagnosis of people who had at least one inpatient hospitalisation event within the last year of life; linkage was then made by encrypted NHI to their outpatient, pharmaceutical and laboratory costs. For example, there were 818 patients who had an inpatient event with a diagnosis of cardiovascular disease, and the average outpatient, pharmaceutical and laboratory costs for these patients were $4700, $2000, and $340 respectively.
with chronic renal failure had the highest outpatient, pharmaceutical and laboratory costs.

Table 9. The average outpatient, pharmaceutical, and laboratory cost in the last year of life by disease category

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Number of patients</th>
<th>Average cost per patient ($)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Outpatients</td>
<td>Pharmaceutical</td>
<td>Laboratory</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>818</td>
<td>4700</td>
<td>2000</td>
<td>340</td>
<td></td>
</tr>
<tr>
<td>Malignant cancer</td>
<td>640</td>
<td>5400</td>
<td>2200</td>
<td>420</td>
<td></td>
</tr>
<tr>
<td>Chronic respiratory disease</td>
<td>149</td>
<td>7600</td>
<td>2500</td>
<td>290</td>
<td></td>
</tr>
<tr>
<td>Chronic liver disease</td>
<td>33</td>
<td>700</td>
<td>1300</td>
<td>360</td>
<td></td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>213</td>
<td>13,900</td>
<td>3900</td>
<td>620</td>
<td></td>
</tr>
<tr>
<td>All other causes</td>
<td>945</td>
<td>2900</td>
<td>1900</td>
<td>330</td>
<td></td>
</tr>
<tr>
<td>People who had inpatient hospitalisation</td>
<td>1835</td>
<td>4100</td>
<td>2000</td>
<td>330</td>
<td></td>
</tr>
<tr>
<td>Overall (total cohort)</td>
<td>2290</td>
<td>3400</td>
<td>1700</td>
<td>300</td>
<td></td>
</tr>
</tbody>
</table>

Discussion

People in their last year of life had a high level of health service utilisation in 2007/08. Just over 80% of people had at least one publicly funded inpatient hospital stay in the last year of life. Forty percent of all deaths in 2008 in CMDHB occurred in a publicly funded hospital. More than 75% of the health care costs in the last year of life were related to inpatient hospitalisations.

The average cumulative length of inpatient stay over the year was 20.6 days. Outpatient, pharmaceutical, and laboratory services were received by 84%, 91%, and 86% of people respectively in their last year of life. There were 69 people (3%) who did not attend any of the services examined in the study in the last year of life. This observation is likely to be related to people who had sudden deaths (e.g. car accidents), and/or people who did not engage with the health system at all.

The average health care cost per person in last year of life is high in CMDHB. A CMDHB report suggested that the age standardised health care cost in the last year of life may be seven or more times higher than the average annual health cost in people not in the last year of life. The proportion of all deaths that occurred in hospital (40%) in CMDHB is roughly similar to the other developed countries: 36.6% in the US and 50.3% in the UK.

As shown in Figure 2 and Table 7, the health care costs in the last year of life varied widely between individuals, disease categories and age ranges. People who had hospitalisations related to cancer had the highest average hospitalisation cost per person ($18,200). People who had hospitalisations with chronic renal failure had the highest outpatient costs, presumably related to outpatient dialysis. Overall in 2008 Counties Manukau District Health Board spent at least $51m on its residents in their last year of life – around 5% of its overall budget.

In the subgroup analyses by age range, the average health care costs in last year of life were highest in young children between 28 days and 9 years and adults in the 50–79
years age group, and the health care costs subsequently dropped with advancing age from 80 year old onwards. The common causes of death in young children in CMDHB include extreme prematurity, congenital anomalies, cancer, sudden unexplained death in infants (SUDI, formerly known as SIDS) and injury and poisoning. \(^\text{13}\)

The high end of life costs of the young children may be related to the former three conditions. The observed gradual fall in health care costs with advancing age is consistent with other international studies. \(^\text{5,14,15}\)

Reasons for this are likely to include:

- Age is a good proxy for prognosis. \(^\text{14}\) The balance between potential benefit and likely morbidity from an intervention is often less favourable in people who are older or have a poor prognosis.
- Individuals and their families being less likely to want significant interventions the later they are in life (“I’ve had a fair innings”).

If a simple cost utility analysis was undertaken, the results could be somewhat alarming. At a time when the quality of life is often far from perfect, a small proportion of people (2.7% of the cohort) who had health care cost greater than $100,000 accounted for significant proportion (17%) of the total cost of people in the last year of life. However, results from such cost utility analysis should not be taken on face value only, because the clinical decisions relating to any health service provision (including end of life care) are always made in a prospective manner.

Predicting prognosis and benefits from interventions is often uncertain and the decision to provide high cost health services might be appropriate when the decision was made given the available information at the time. Therefore, it would be inappropriate to use a retrospective analysis after the fact of death to solely determine whether the health care services provided were appropriate or not.

Nevertheless, evidence from overseas studies suggest that the end of life costs in people even with perceived poor prognosis still remained very high, even though the end of life spending is less in people who had a higher estimated risk of death. \(^\text{16}\)

Furthermore, the amount of health services utilised towards the end of life has been shown to be subject to variations in local clinical practice and/or policy that are not explained by disease prevalence and severity. The Dartmouth Atlas Project demonstrated there are marked variations in the length of stay in inpatient hospitalisations, and intensive care units during the last 6 months of life in the US. \(^\text{9}\)

The main reason for the variations in care between 93 integrated academic medical centres is related to the level of ‘supply sensitive’ care that was provided, while severity or prevalence of illness accounted for little of the variation. ‘Supply sensitive’ services refer to services where the availability of a specific resource had a major influence in utilisation rates, e.g. the number of intensive care beds. Disconcertingly, the project found little evidence to suggest the higher volume of services provided had lead to improvement in health outcomes.

In fact, aggressive management in people who have poor prognosis can be associated with more adverse outcomes such as more physical distress, and worse overall quality of death as reported by the caregiver. \(^\text{9,17}\) The provision of an intervention that has
little or remote chance of prognostic benefit may result in unnecessary suffering for
the patient in terms of side effects. In some cases, it may be more preferable to re-
direct the resources of such interventions to provide a better end of life care to the
individual.

In the context where the current trends of the health care expenditure increase in New
Zealand are not sustainable in the long term, high cost interventions provided
towards the end of life should be carefully considered based on clinical factors,
patients’ expectations and cost utility of interventions. Health care resources are
always finite. Every clinical decision is actually also a resource decision. The cost
utilities of various available treatments in different patient groups are often not
explicitly compared. However, the New Zealand Medical Council expects that as a
part of clinical practice, doctors will balance their duty of care to each patient with
their duty of care to the population.

The decisions to initiate a high cost or invasive treatment in patients with poor
prognosis can certainly be challenging. In some circumstances, the decision making
process may be assisted by guidelines. However, the decisions to withdraw treatment
are phenomenally difficult particularly in patients who would no longer be eligible to
have the treatment had the person not started treatment already or the decision to
withdraw treatment will lead to death in the immediate future, e.g. the decision to stop
dialysis.

As demonstrated by this study, people in their last year of life often have multiple
chronic conditions.Clinicians should be supported in taking a more integrated
approach in assessing the patients’ prognosis based on all the co-morbidities rather
than providing a prognosis for a specific condition. A regular forum with
contributions from multi-disciplinary and multi-speciality teams to discuss these
ethical questions may be appropriate in challenging cases.

The strength of this study is that the data analysed were derived from the routinely
collected national datasets in New Zealand. As it is a complete data set, there are no
sampling errors or difficulties with generalising to the whole population. The methods
can be easily replicable to provide a regional comparison within New Zealand or
provide time trends in health care utilisation in the future for monitoring or evaluating
purposes. However, a limitation of this study is that it did not include the health care
services that are not captured by the routinely collected datasets. The health care costs
related to primary health care, inpatient and outpatient hospice care, pharmaceutical
go-payments or health services that are funded privately in the last year of life were
not included. Furthermore, the significant costs of informal care provided by family
and friends were also not included.

The sub-grouping by clinical condition should be interpreted with caution as it was
created in an empirical manner from hospital disease coding rather than a true clinical
review. Since one person may be classified into more than one disease category, the
non-inpatient hospital costs should be interpreted with caution. For example, not all of
the outpatient, pharmaceutical, and laboratory costs for the 818 people discharged
with a prioritised diagnosis of cardiovascular disease would be related to
cardiocascular disease (Table 9).
Conclusion

Consistent with the international literature, this study found that CMDHB residents in the last year of life have a high level of health service utilisation. People often have multiple chronic diseases in the last year of life. Providing health services for patients in the last year of life is associated with high health care costs. The majority of the health care cost is related to inpatient care. Decisions about the appropriate use of high cost health services in people towards the end of life can be extremely challenging. These decisions are resource allocation decisions as well as clinical decisions and should be based on clinical factors, cost utilities, and patient, family, and society’s expectations.

Competing interests: None.

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References:


Neuro-ophthalmic manifestations and outcomes of pituitary apoplexy—a life and sight-threatening emergency

Sumu Simon, David Torpy, Brian Brophy, Peter Blumbergs, Dinesh Selva, John L Crompton

Abstract

Objective To report the neuro-ophthalmic manifestations and outcomes in patients with pituitary apoplexy.

Method Retrospective chart review.

Results 23 patients were identified (17 men, mean age 54.1 years (range 23–86 years). The onset was abrupt in 22 patients; one patient had a subclinical presentation. Headache was the commonest presenting symptom (82.6%, 19/23). Neuro-ophthalmic manifestations were present in more than three-quarters of the patients (82.6%, 19/23). At presentation, 55% (11/20), 47.6% (10/21) and 60.9% (14/23) of the patients had reduced visual acuity, field defects and cranial nerve palsies respectively. Management was conservative in 4 patients and surgical in 18 patients; one patient died shortly after presentation. The median follow up period was 10.5 months (22 patients, range 0.2–168 months).

At final follow up, improvement was present in 100% of the patients with reduced acuity (8/8) and ocular palsy (13/13) and 81.8% of patients with field deficits (9/11). Age, sex, presence of precipitating factors and timing of surgery did not have an impact on neuro-ophthalmic recovery.

Conclusion Pituitary apoplexy should be considered in any patient with abrupt onset of neuro-ophthalmic deficits. Prompt medical and surgical management is lifesaving and can lead to significant improvement in visual and cranial nerve deficits.

Pituitary apoplexy is a rare neurosurgical emergency resulting from an acute ischemic or haemorrhagic infarction within pre-existing pituitary adenomas. It differs from Sheehan’s syndrome in that in the latter, the postpartum pituitary necrosis occurs within a normal pituitary gland. It occurs in about 0.5%–17% of operated pituitary tumours.

Symptoms and signs are due to hypopituitarism, compression of adjoining structures and due to release of blood and necrotic products into the cerebrospinal fluid (CSF). The clinical presentation can mimic optic neuritis, ruptured arterial aneurysms, carotico-cavernous fistula and giant cell arteritis.

The mortality rate associated with pituitary apoplexy is 0.7–12.5%. Advances in neuro-imaging and prompt multidisciplinary management have significantly reduced apoplexy related visual sequelae and deaths.
Materials and methods

We present a retrospective case study of 23 pituitary apoplexy patients admitted to the Royal Adelaide Hospital (South Australia) from 1979 to 2009. All cases with a diagnosis of pituitary apoplexy confirmed by clinical findings, neuro-imaging and /or histopathology (in the surgical group) were included in the study. Data regarding clinical presentation, precipitating factors, investigations, management and follow up were reviewed. Neuro-ophthalmic data pertaining to visual acuity, ocular motility, visual fields and ocular examination were collected. Complete recovery of visual function was defined as vision of 6/6 or a return to baseline acuity prior to apoplexy. The numbers of patients used in each analysis are provided throughout. Data analysis was done using SAS version 9.2 software.

Results

Twenty-three patients presented with pituitary apoplexy. Four patients in the series did not have any neuro-ophthalmic manifestations. A postoperative histo-pathological diagnosis of pituitary apoplexy was made in one unsuspecting patient with pituitary macro adenoma and partial third nerve palsy. Their ages ranged from 23 to 86 years with a mean age of 54.1 years. The male to female ratio was 2.8:1.

The diagnosis at initial presentation was accurate in only 21.7% of patients (5/23) with meningitis, upper respiratory infection and migraine being the common misdiagnoses. The median delay in referral was 1 day (range 0–14 days). The median follow up period was 10.5 months (22 patients, range 0.2–168 months). Headache was the commonest symptom and was present in 82.6% of the patients (19/23).

Diplopia and defective vision were present in 43.5 % (10/23) and 34.8 % (8/23) respectively. None of the patients were aware of a pre-existing pituitary adenoma. A history of precipitating factors was present in 17.4% of cases (4/23). These included coronary angiography and cardiac surgery in two patients each.

Table 1. Symptoms and signs of pituitary apoplexy

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>% (No. of cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>82.6% (19/23)</td>
</tr>
<tr>
<td>Nausea &amp; vomiting</td>
<td>47.8% (11/23)</td>
</tr>
<tr>
<td>Diplopia</td>
<td>43.5% (10/23)</td>
</tr>
<tr>
<td>Defective vision</td>
<td>34.8% (8/23)</td>
</tr>
<tr>
<td>Altered consciousness</td>
<td>26.1% (6/23)</td>
</tr>
<tr>
<td>Fever</td>
<td>8.6% (2/23)</td>
</tr>
<tr>
<td>Vertigo</td>
<td>8.6% (2/23)</td>
</tr>
<tr>
<td>Seizures</td>
<td>4.3% (1/23)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th>% (No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual acuity defects</td>
<td>55% (11/20)</td>
</tr>
<tr>
<td>Visual field defects</td>
<td>47.6% (10/21)</td>
</tr>
<tr>
<td>Cranial nerve palsies</td>
<td>61% (14/23)</td>
</tr>
<tr>
<td>Visual acuity and field defects</td>
<td>38.9% (7/18)</td>
</tr>
</tbody>
</table>

Whilst all patients at presentation had data regarding cranial nerve functions, information pertaining to acuity and field defects was available in 20 and 21 patients respectively. Neuro-ophthalmic involvement was present in 82.6% of the patients (19/23). One patient had unilateral disc oedema at presentation.
Deterioration of visual acuity was present in 55% (11/20) of the patients with the involvement being bilateral in more than half of them (54.5%). Only one patient in the study had bilateral absent light perception at outset (1/20). Of the 14 patients with cranial nerve palsies, the involvement was unilateral in 92.8% (13/14). Bilateral ophthalmoplegia and pupil sparing III nerve palsy were rare and present in one patient each.

Table 2. Cranial nerve involvement in patients with pituitary apoplexy

<table>
<thead>
<tr>
<th>Nerve palsies</th>
<th>% ( No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>III nerve palsy</td>
<td>28.6% (4/14)</td>
</tr>
<tr>
<td>VI nerve palsy</td>
<td>21.4% (3/14)</td>
</tr>
<tr>
<td>III, IV &amp; VI nerve palsies</td>
<td>14.3% (2/14)</td>
</tr>
<tr>
<td>III &amp; IV nerve palsies</td>
<td>7.1% (1/14)</td>
</tr>
<tr>
<td>V &amp; VI nerve palsies</td>
<td>7.1% (1/14)</td>
</tr>
<tr>
<td>III &amp; VI nerve palsies</td>
<td>7.1% (1/14)</td>
</tr>
<tr>
<td>IV &amp; VI nerve palsies</td>
<td>7.1% (1/14)</td>
</tr>
<tr>
<td>II-VI nerve palsies</td>
<td>7.1% (1/14)</td>
</tr>
</tbody>
</table>

Field disturbances were present in 47.6% of the patients (10/21) at presentation. Visual field analysis was by automated perimetry (Humphrey Field Analyser) in 14 patients and confrontation in eight cases. The defect was bilateral in 80% of the patients (8/10). Bitemporal hemianopia was the commonest field defect (50%, 5/10) followed by homonymous hemianopia (20%, 2/10) and generalised depression (10%, 1/10). Unilateral temporal field loss and generalized depression was present in one patient each.

In the eighteen patients who had data pertaining to visual acuity, visual fields and cranial nerve palsies, the commonest presentation was the mixed type with both cavernous and chiasmatic involvement (38.8% [7/18]), followed by the chiasmatic (27.7%, [5/18]) and the cavernous types (16.6%, [3/18]). Preoperative hypopituitarism was present in 76.5% (13/17). Hypothyroidism was the commonest (58.8%, 10/17) followed by hypocortisolism (52.9%, 9/17) and testosterone deficiency (23.5%, 4/17). The majority of the adenomas were non-functional (19/21); the rest were prolactinomas.

Neuro-imaging was done in all cases; computerised tomography (CT) and magnetic resonance imaging (MRI) were done in 17 and 19 patients respectively, whilst plain X-ray was done in 2 cases. CT demonstrated a sellar mass in all patients with suprasellar and parasellar extensions noted in 10 patients and 2 patients respectively. The mass was described as hyperdense in 3 cases, whilst peripheral enhancement was noted in 2 cases (Table 1). On MRI, suprasellar and parasellar extensions were present in 13 and 4 patients respectively. Fluid-fluid level within the mass was present in one case (Table 2).

Optic chiasm involvement was present in eight cases. MRI was superior (89.5%, 17/19 patients) to CT (5/17 patients, 29.4%) in diagnosing pituitary apoplexy. X-ray demonstrated enlarged pituitary fossa with double floor appearance in one case. Three patients with suspected ruptured aneurysm underwent angiography with negative
outcomes. CSF studies were done in 4 patients with one of them showing xanthochromia. The management was medical in 4 patients and combined medical and surgical in 18 cases.

Table 3. Comparison of demographic data and neuro-ophthalmic signs in patients with pituitary apoplexy managed conservatively and surgically

<table>
<thead>
<tr>
<th>Variables</th>
<th>Conservative (total n=4)</th>
<th>Surgical (total n=18)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years) range</td>
<td>46.5 (25-79)</td>
<td>54 (23-75)</td>
<td>0.44</td>
</tr>
<tr>
<td>Male/females</td>
<td>3:1</td>
<td>2.6:1</td>
<td>1.00</td>
</tr>
<tr>
<td>Decreased visual acuity</td>
<td>1</td>
<td>10</td>
<td>0.56</td>
</tr>
<tr>
<td>Visual field defect</td>
<td>1</td>
<td>9</td>
<td>0.60</td>
</tr>
<tr>
<td>Ocular palsy</td>
<td>3</td>
<td>10</td>
<td>0.61</td>
</tr>
</tbody>
</table>

One patient died while awaiting management with post mortem examination revealing extensively infarcted invasive pituitary macro adenoma and death due to cardiac tamponade following myocardial infarction. There was no difference in the proportion of patients with visual acuity defects, field defects and ocular palsy in the two management groups. Neuro-ophthalmic deficits were present in three patients in the conservative group. The mean follow up period of patients managed conservatively was 5.6 months (2.5–10.5 months). Two patients with cavernous presentation made a full recovery whilst the third patient with a mixed presentation had partial recovery of nerve palsy; No data was available regarding visual recovery. One patient showed worsening from normal baseline field.

The surgical approach was trans-sphenoidal in 15 cases, trans-cranial in 2 cases whilst a combined trans- sphenoidal and ethmoidal approach was done in one patient. The mean duration of surgical delay was 15 days (range 1–120 days). Surgeries were done within and later than 1 week in 12 and 6 patients respectively. The mean follow up period was 10.4 months (range 0.26–168 months). Preoperatively 62.5% of the patients (10/16) had deterioration in visual acuity with all of them being operated within the first week of presentation. Recovery was partial in 75% of the patients (6/8) and complete in the rest. Field defects were present in 44.4% (8/18) of the surgical patients; 7 of them were operated within the first week.

Postoperatively improvement was present in 88.9% (8/9) with the recovery being partial in 55.5% (5/9); worsening from normal baseline field was observed in 1 patient. Of the 10 patients with cranial nerve involvement, 9 were operated within the initial week; recovery was complete in 70% and partial in the rest.

No statistically significant difference was shown between the timing of surgery (less than or more than a week) and visual acuity, field or nerve palsy recovery nor between age (above or below 50 years), sex and neuro-ophthalmic recovery; Presence of precipitating factors did not adversely affect the visual acuity and field recovery. Total or partial hypopituitarism was present in 90.5% of the patients (19/21) at follow up.
Table 4. Neuro-ophthalmic outcomes in pituitary apoplexy patients with conservative and surgical management

<table>
<thead>
<tr>
<th>Variables</th>
<th>Conservative</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Visual acuity defects</strong></td>
<td>1*</td>
<td>8*</td>
</tr>
<tr>
<td>Complete recovery</td>
<td>–</td>
<td>2 (25%)</td>
</tr>
<tr>
<td>Partial recovery</td>
<td>–</td>
<td>6 (75%)</td>
</tr>
<tr>
<td><strong>Ocular palsy</strong></td>
<td>3*</td>
<td>10*</td>
</tr>
<tr>
<td>Complete recovery</td>
<td>2 (66.6%)</td>
<td>7 (70%)</td>
</tr>
<tr>
<td>Partial recovery</td>
<td>1 (33.3%)</td>
<td>3 (30%)</td>
</tr>
<tr>
<td><strong>Visual field defect</strong></td>
<td>1*</td>
<td>8*</td>
</tr>
<tr>
<td>Complete recovery</td>
<td>–</td>
<td>3 (30%)</td>
</tr>
<tr>
<td>Partial recovery</td>
<td>–</td>
<td>5 (60%)</td>
</tr>
<tr>
<td>Worsening from normal</td>
<td>–</td>
<td>1 (10%)</td>
</tr>
</tbody>
</table>

*Defects at presentation.

Histopathology revealed haemorrhagic infarction in 11 cases (Table 3), acute haemorrhage in 2 cases and ischemic infarction in 1 case (Table 4) whilst infarction of unspecified type was present in another 5 cases. Adjuvant radiation therapy was given to two post surgical patients for residual tumour. Tumour recurrence was noted in one patient 4 years after surgical management.

**Discussion**

Pituitary apoplexy was first described by Pearce Bailey in 1898 in an acromegalic patient. The term “pituitary apoplexy” was coined years later by Brougham in 1950. Pituitary tumour apoplexy is a clinical syndrome due to haemorrhage or infarction into the pituitary tumour leading to sudden expansion of the sella turcica. The exact pathogenesis of pituitary apoplexy is still unclear. It has been proposed that the compromise of the hypophyseal portal vasculature and/or the arterial supply particularly the inferior hypophyseal artery is responsible for the apoplexy.

Rapid tumour growth outstripping the blood supply, structural abnormalities of the pituitary adenoma blood vessels, arteriosclerotic embolisation of blood vessels as well as abrupt alterations in the perfusion pressure of adenomas have been implicated. The true incidence and prevalence of pituitary apoplexy is unknown. However the incidence of this clinical syndrome in surgically treated patients has been reported in about 17%. No age is immune from this vascular catastrophe with the ages ranging from 6–90 years (mean of 50.9 years).

Various studies show similar gender distribution with the male to female ratio ranging from 1:1 to 2.1:1. Although spontaneous in the majority, precipitating factors are present in 25–30% of patients; these include malignant hypertension, diabetic ketoacidosis, trauma, radiotherapy, cardiac surgery, anticoagulant therapy, coronary angiography, dynamic testing of pituitary function, hypotension, head trauma and administration of dopamine antagonists. The signs and symptoms of this syndrome are acute onset of headache which is of abrupt onset and frontal or retro bulbar location, altered sensorium, seizures, meningism, visual impairment, ophthalmoplegia, facial pain/paraesthesia, and those related to endocrine deficiencies.
Rarely, hemiplegia can occur due to compromise of the internal carotid artery. Orbital bruit, proptosis, lid oedema, isolated Horner’s syndrome and light near dissociation are rare orbital manifestations of pituitary apoplexy. The differential diagnoses for pituitary tumour apoplexy are subarachnoid haemorrhage from ruptured aneurysm, meningitis, acute cerebral infarction, midbrain infarction, cavernous sinus thrombosis, optic neuritis, hypothalamic lymphoma, haemorrhage into Rathke’s cyst, carotico-cavernous fistula and ruptured posterior communicating artery aneurysm.

Different series have reported the visual deterioration and oculomotor paresis from pituitary apoplexy as ranging from 40–100%. In our study, the commonest manifestation was cranial nerve deficits (61%) followed by visual acuity (55%) and field defects (42.8%). The majority of patients have hypopituitarism at presentation; in this case series, endocrine deficiencies were observed in 76.5% (13/17 patients).

Computerised tomography (CT) and magnetic resonance imaging (MRI) are useful diagnostic tools. CT is most useful in the acute stage (<72 hours) when haemorrhage appears as a focal, multifocal or diffuse hyper density in the pituitary mass. MRI is superior to CT in diagnosing and delineating tissue involvement in pituitary apoplexy (89.5% vs. 29.4%); MRI/MRA also has the added advantage of ruling out aneurysms. The MRI appearance varies with the evolution of haemorrhage. In the hyper acute stage (<3 hours) findings may not be present. Haemorrhage less than a week old appears as hypo-intense on T1 and T2 weighted images; The signal intensity increases on T1 weighted images from 7–14 days. Fluid–fluid level within the pituitary mass is typical of pituitary apoplexy. Rim enhancing lesion on contrast enhanced T1 weighted image is suggestive of tumour infarction.

Management of pituitary apoplexy is multidisciplinary requiring close co-operation between neurology, neurosurgical, endocrine and neuro ophthalmologic specialities. After establishing the diagnosis, medical management is aimed at correcting the endocrine abnormalities and stabilising the patient. Clear cut management guidelines are lacking due to the rarity of this condition. Favourable outcomes have been reported in the conservatively and surgically managed patients. This result needs to be cautiously interpreted bearing in mind the selection bias for the two treatment groups. Though there is no consensus regarding ideal time for surgery, urgent surgical management is indicated in those patients with rapidly deteriorating visual and neurological defects. In our series, improvement was present in all the patients with decreased acuity (8/8) and ocular palsies ((13/13) whilst and in 88.8 % (8/9) of the patients with field defects.

Similar outcomes have been reported by Nawar et al (visual acuity and field recovery 60–100%, ocular palsy recovery 65–100%) and Bills et al (visual acuity -88%, field recovery -95%,ocular palsy recovery -100%). The absence of precipitating factors, preserved light perception and early decompression have been reported to be associated with favourable recovery. Our study however, did not find any significant association between age, sex, presence of precipitating factors, timing of surgery and neuro-ophthalmic recovery; the visual recovery was marginal in the patient with bilateral absent light perception at presentation. Long term monitoring is required in these patients for the management of hypopituitarism and detection of tumour recurrence.
In conclusion, pituitary apoplexy can mimic a wide spectrum of clinical conditions due to its myriad signs and symptoms. The constellation of signs must alert the clinician to the possibility of pituitary tumour apoplexy. Appropriate intervention is associated with good neuro-ophthalmic recovery.

**Competing interests:** None.

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Citizenship, work, welfare, education and health in New Zealand

Des Gorman

Abstract
Access to excellent, unconstrained and timely health care is considered a birthright by most New Zealanders. However, there are shortcomings in some health services, especially in mental health and rehabilitation, and these not only have an adverse personal impact but also challenge the sustainability of the national welfare system. There are insufficient well-trained people who can manage ‘care’ in line with either generic best practice or Whānau Ora ideology. An important and core reform is for a coordination of relevant programmes in education, health, justice and welfare, and for both shared accountabilities and linked governance.

The Welfare Working Group
I recently had the privilege of being a member of Minister Bennett’s Welfare Working Group (WWG). The WWG, which was led by Ms Paula Rebstock, recommends substantive reform. This outcome arises from a recognition that our welfare system is often disabling and that it is neither sustainable nor fit-for-purpose. As an illustration of the cassis belli for reform, 31% of working age Māori are State welfare beneficiaries of one sort or another. There is no comfort for any New Zealander in such a rate of joblessness.

This essay is a personal review of the milieu of the WWG’s report and the knock-on implications for our health services.

Citizenship
In my opinion, New Zealanders have a sense of citizenship, which is in part based on a commonly held set of values. Ironically, those values are probably most appreciated by us when we are confronted by dissimilar viewpoints. For example, many New Zealanders were bemused by much of the argument against President Obama’s programme to increase health access in the USA. The concepts and values of mutuality, welfare and altruism were largely missing from the oppositional argument.

It is hardly surprising that we have common values; after all, we are all ‘boat people’ who have come to New Zealand for a better way of life. Some of my ancestors (the Māori) arrived about 1000 years ago, whereas my father arrived on his boat from Australia in 1952. We also have defining legislation, such as the Social Security Act of 1938, and a unique treaty between the British colonisers of the nineteenth century and Māori (The Treaty of Waitangi).

The WWG was frequently told by commentators and those making submissions that social welfare was the centre-point of the national social contract. The more I thought about this, the more it seemed to me that this was not the case; surely the central
social contract is that those of us who can work do so, for pay or not, and by way of this work we contribute both directly and indirectly to our society. In the absence of such a core commitment to work, our society cannot exist.

For reasons that are unclear, work and work-related schemes are often regarded pejoratively and both are frequently seen as being punitive in rehabilitation programmes. The sadly commonplace nature of these views and the resultant distortion of medical practice are such that the Royal Australasian College of Physicians has felt the need for a public campaign to argue that work is good for our spiritual, mental and physical health and that without work, people often experience consequent ill health. 5 Indeed, positive vocational rehabilitation outcomes should become a key accountability for our health services. At present, they are not and the welfare system ‘inherits’ the poor outcomes of what are somewhat deficient rehabilitation and mental health services.

If work is at the core of our social contract, then the welfare system can be seen as consequential and to exist to support those who cannot work until they can do so. In this way, a sensible relationship between this analogous dog and its tail is affirmed.

Some expectations inevitably arise from this citizenship. These include employment opportunities, which I will not discuss further, and unlimited access to education and health care, which will be the subject of the balance of this essay.

Citizenship and education

New Zealanders assume certain egalitarian birthrights: these include access to the highest quality of education; and, similarly, access to the health care that they need, when and where they need it, again of the highest quality, and without constraint. These expectations are essentially not negotiable, are passive (as compared to being consumer-owned and proactive in a health setting) and set a very high bar for service provision.6

Despite considering themselves to being “Better British”, 4 which is reasonably argued to have been a common feature of many early settlers from the UK, education is the basis of social mobility in New Zealand and the principal ‘vehicle’ used since European colonisation began in earnest here to prevent a repetition of the restrictive social classes of the immigrants’ countries of origin. Our Prime Minister is an example of such mobility. Similarly, I went to a lower socioeconomic (decile 1) secondary school and have not experienced any related externally-imposed limitations on my ambition and/or employment.

Educational equity has nevertheless been eroded since my school days, as evidenced by recent university-entrance attainment rates of only 13% for decile one, two and three secondary school students.7 This relative educational failure has self-evident and adverse employment, health and welfare, and justice-system impacts.

Measures are in place to remedy the imbalance. The one that has the greatest appeal to those of us in the health system is the advent of health sciences academies at lower-decile and predominantly Polynesian (e.g. Otahuhu College) and Māori (e.g. James Cook High School) secondary schools. Cohorts of students are admitted to the academy for the last three years of secondary schooling (years 11, 12 and 13).
In addition to core academic subjects, such as English and Chemistry, students have work experience exposures through joint ventures with local providers and medical societies, and undertake programmes and courses that ensure they have (potential) access to the entire range of tertiary health worker education programmes.

The direct benefits are three-fold by way of positive education, health and employment outcomes. First, the academies render education purposeful and are likely to increase student retention and to improve both attendance and performance. Second, the students’ health literacy is enhanced and these students will carry a health debate deep into their families and Whānau, and communities. Third, there can be few more guaranteed industries for employment than health.\textsuperscript{8,9}

**Citizenship and health**

New Zealand shares a health service demand-supply-affordability mismatch with most of the industrialised World.\textsuperscript{8-10} Some of our health services are especially vulnerable and most of these are community- as compared to hospital-based.

Those with the greatest adverse impact on the welfare system are the shortcomings in rehabilitation and mental health services, and the essential absence of a managed care (Whānau Ora) workforce. Treasury estimates that unmet mental health need is the single greatest contributor to long term injury- and illness-related disability and consequent welfare-dependency.\textsuperscript{11,12}

Health Workforce New Zealand is well aware of these vulnerabilities and appropriate clinician-led service reviews are underway.\textsuperscript{13} These reviews are largely vignette-based and predicated to resolve the conundrum of meeting a significant growth in demand for health services (perhaps a doubling over the next decade) in a way that both maintains overall quality and access and closes access and outcome ‘gaps’,\textsuperscript{14,15} and that slows the rate of increase in the costs of health care to something closer to the likely growth in wealth of our country over the same period (about 40%).

A disruptively innovative reform of service configurations and models of care is necessary and will need to be underpinned by a similarly extensive reform of funding schema and rewards systems. The latter must include the consumer if there is to be a meaningful shift to patient-centred and -owned care. The landscape will inevitably involve both primary-secondary care and public-private partnerships and integration.

Funding, management, provider and education integrated models of care that include the health sciences academies cited above are also being developed in partnership with Māori (see Figure 1); this recognises and attends to current health outcome inequities. For example, the difference in life-expectancy between European New Zealanders/Pakeha (as well as other New Zealanders) compared to Māori is greater than the equivalent gap between North American Indians and the European colonisers of that continent.\textsuperscript{14}

**Across-sector alignment and governance**

Education, health, welfare, along with the accident compensation and the justice systems are inexorably linked at a functional level and a failing in one has knock-on effects for most of the others; and yet, governance of these services is dislocated.
If I were to select any aspects of the WWG’s recommendations for highlight here, it would be for a whole-of-sector conjoint governance, for aligned and long-term outcome accountabilities (e.g. positive employment outcomes as a headline health KPI) and for a consequential shift in the management of our rehabilitation system to the sort of long-term actuarial logic employed by the ACC. All are both overdue and necessary.

Figure 1. Schematic of Iwi Health Plan showing integration of secondary school health sciences academy

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ACC and back injuries: the relevance of pre-existing asymptomatic conditions revisited

Peter A Robertson, O Ross Nicholson

Abstract

The application of the New Zealand Accident Compensation Corporation (ACC) legislation in the management of patients who sustain back injuries requires a detailed knowledge of the pathogenesis of tissue injury, and the natural history of ageing and related conditions, so that the application of the ACC Act(s) is appropriate. We have reviewed the new information published in the last decade, and updated the previous knowledge basis in these fields, so as to assist the interpretation of the Act(s).

A decade ago we published a viewpoint article that focused on ACC entitlements for ACC patients with lumbar spine conditions who had a sustained a personal injury by accident. Our concern was that a number of ACC claimants were being declined on the basis of radiological findings (often discovered on then newer imaging modalities such as MRI scans) rather than an appropriate focus on the claimant’s history.

Since that time changes have occurred to the ACC Acts, and their administration and the increased medical knowledge in relation to the prevalence and natural history of common spinal conditions has questioned accepted dogma. Over the same period a vast number of opinions have been published. These range from independent expert medical opinion, ACC medical advisory opinion, independent legal opinion, ACC review decisions, and up to District Court judgements. The latter of these can be considered expert legal opinion that establishes case precedents.

Our observation is that the quality of these opinions is highly variable, and this is reflected in the downgrading of the weight given to expert medical evidence. Evidence based medicine and evidence based observational literature is now given greater weight than the expert opinion of an individual. From the outside, one might see parallels in the legal system where the higher courts form decisions with panels of judges but the lower courts rely on the expertise of a judge sitting alone whose judgement creates precedents for the future.

In New Zealand most ACC appeals end at the District Court, and it has been our observation that there are occasions when the judge has not clearly understood the medical details of the case, or given apparently different decisions in similar cases thus creating both precedent and confusion.

When the ACC legislation came into operation in 1974 it was the clear intention of the legislators to provide treatment, rehabilitation and compensation for patients suffering injury caused by accident. This intent has continued, but the ACC system does not cover congenital or developmental disorders, nor infective (de novo) nor malignant, nor chronic musculoskeletal conditions that occur other than resulting from trauma.
Given the increased diversity of opinions, viewpoints, and expertise paralleled by increasing knowledge of the prevalence of various symptomatic and asymptomatic spinal conditions, it is appropriate to review the medical knowledge in relation to recent Acts. As in the previous article we will focus on the Act(s) and the current and past relevant literature.

Changes in the Act through 2000, 2001, 2005, 2008 and 2010 have focused upon different areas of ACC administration and changes to the definition of personal injury and accident have been relatively minor.

**Accident**

The definition of accident in the current Act is unchanged from the 2001 Act:

**Accident**

(1) Accident means any of the following kinds of occurrences:

(a) a specific event or a series of events, other than a gradual process, that—

(i) involves the application of a force (including gravity), or resistance, external to the human body; or

(ii) involves the sudden movement of the body to avoid a force (including gravity), or resistance, external to the body; or

(iii) involves a twisting movement of the body:

This definition leaves a wide scope for the interpretation of actions or activities that fulfil this definition. It does not comment on intention, although the 2010 Act excludes the effects of deliberate self harm (Section 119). Perhaps more importantly, the definition of accident does not require assessment of the magnitude of the force. There is no exclusion on the basis of a trivial injury.

**Personal Injury**

The current Act has modified Section 26 of the 2001 Act, although there is no change to the relevant clause discussed here.

(1) Personal injury means—

(a) the death of a person; or

(b) physical injuries suffered by a person, including, for example, a strain or a sprain; or ....

Personal injury requires a physical injury yet the nature of such an injury is not described but includes a sprain or a strain. Presumably physical injury would include laceration, haematoma, muscle or tendon rupture, fracture, and neural or vascular injury.

The Act does not require imaging or histological abnormalities to establish the diagnosis of an injury and does not give any other guidelines. It does not exclude changes at a cellular or neural level, which are the mechanisms now considered to be involved in pain syndromes, with the biochemical and neural changes occurring after initiating traumatic events.
The Act does specify that cover includes a sprain or a strain. A sprain is stretched or torn ligament while a strain is a stretched or torn muscle or tendon (US National Institute of Health). These are soft tissue injuries, diagnosed from the history of injury and the examination findings. X-rays may be taken to exclude a bony injury and may demonstrate soft tissue swelling. Newer forms of imaging may demonstrate tissue discontinuity but this is not required for the diagnosis. Medically, we accept that the diagnosis of a sprain or strain involves a history of injury, pain, some restriction of motion and tenderness which will be more noticeable the more superficial the site of injury.

It is important to note the lack of any prescription of the force magnitude at the time of injury, nor any requirement for high tech imaging or histologic examination of the affected area to confirm the diagnosis, nor any limitation to the duration of cover that relates to "normal" times for recovery from specific injuries.

**Exclusion**

The Act includes provisions for exclusion of ACC entitlement in sections that relate both to the definition of accident and personal injury. In Section 25 of the Accident Compensation Act 2001 an accident is not a gradual process (accident means ... a specific event or series of events, other than gradual process...).

In Section 26 personal injury is excluded by the statement "personal injury does not include personal injury caused wholly or substantially by a gradual process, disease or infection unless ...". "Personal injury does not include a personal injury caused wholly or substantially by the aging process".

In summary, the diagnosis of personal injury by accident cannot include personal injury caused wholly or substantially by gradual process, disease, infection or the aging process. The intent of the legislation is clear. However, as the aging process is universal, the implications of these exclusion criteria warrant further attention. First, it is appropriate to consider the modifying descriptors for the exclusion criteria. "Wholly or substantially" defines the contribution of disease, gradual process, or aging that would lead to exclusion for cover.

The phrase "wholly" is unequivocal. "Substantially" leads to considerable arguments. In our 2000 article we used the Webster's dictionary for the word 'substantial', for which 'substantially' is "being largely but not wholly that which is specified" which translates to "in the most part" or "significantly". Thus for ACC cover to be excluded the major component of the ongoing personal injury needs to relate to gradual process, disease or the aging process. Thus, if the major component of the personal injury is the accident, and not gradual process, disease or aging on the balance of probabilities, (the legal standard of proof required by the Act,) then ACC coverage occurs.

In practical terms this raises the question as to what symptoms or personal injury the patient would have had had the accident not occurred? If the patient would likely have been symptomatic with a gradual process, disease or the aging process then the accident cannot be the whole or substantial cause of the symptoms or personal injury.
Conversely if the patient would likely have been free of symptoms had the accident not occurred, yet is subsequently suffering from symptoms after an accident, then it is the accident that is the whole or substantial cause of the subsequent personal injury. Using this latter consideration to determine whether personal injury is covered requires a clear understanding of the natural history of disease and the aging process. [Significantly there have been legal arguments and opinion that conclude that a mere component of aetiology, rather than more than half of the aetiology, can still be "substantial"].

**Aging/spondylosis/degeneration**

As gradual process, disease and the aging process are reasons for exclusion for cover it is essential to understand the natural history of these conditions. It is clear from the literature that it is very difficult to differentiate aging and degenerative disease in the lumbar spine. The concept of aging from a medical perspective is relatively clearcut. When tissues age there are changes at the cellular level, causing biochemical and tissue changes which change tissue behaviour and, importantly, biomechanical behaviour. These normal changes of age are manifest by skin thinning and wrinkling, the stiffening of the lens the eye, and loss of hair pigment, which are expected to occur.

It is unlikely that the founders of the ACC legislation wished to exclude cover for victims of personal injury just on the basis of age. ACC would accept a laceration caused by trauma in an elderly person with thinner skin, and a fracture of the hip in an elderly person with likely age related osteopenia. So what is the significance of aging changes in the lumbar spine? First it is necessary to clarify medical terminology.

The changes of lumbar spondylosis include disc space narrowing, osteophyte or spondylophyte formation and vertebral end-plate sclerosis shown on plain x-rays. MR scans will show more detail including disc desiccation, annular disruption with disc bulging, disc prolapse, annular tears, and end plate changes. These are considered as disc degeneration or spondylosis. If they are not symptomatic, then this can be clarified by the addition of the qualifying adjective "asymptomatic". If, however, the patient has mechanical axial pain in this setting then the changes can be considered as a disease and the term degenerative disc disease is appropriate.

Again by way of clarification, we would emphasise that an asymptomatic, normally functioning individual is not diseased and therefore does not suffer from degenerative disc disease.

Over the last two decades it has become clear that the lumbar spine shows increased MR abnormalities with increasing age in asymptomatic individuals. More recently and most importantly, it has become clear that these changes are not predictive of current or subsequent disability.

Boos et al demonstrated the MR findings were much less predictive of subsequent disability than psychological and physical aspects associated with work. Borenstein et al noted that the development of new low back symptoms in patients with previously abnormal MR scans was not related to the degree of MRI abnormality that predated the onset of symptoms or to changes in MRI appearance at a later stage.
Jarvik et al\textsuperscript{11} concluded that depression was a more important predictor of low back pain than any MRI finding, except where new disc herniation had occurred when that herniation was clinically relevant. In essence, these authors showed that the common aging changes in the lumbar spine are not predictive of subsequent pain and disability and therefore the concept that a person with pre-existing MRI abnormality would have a high likelihood of going on to develop significant pain and disability is incorrect.

Asymptomatic spondylosis should not be regarded as a pending clinical problem. In this situation the diagnosis of any new state after injury might include a sprain to the back or new pathology may be considered to have occurred with new onset of symptoms, e.g. disc prolapse.\textsuperscript{11} Thus the sudden onset of new pain and disability after an accident (in a person previously asymptomatic) is not likely due to the aging process / degeneration / disease, but at least substantially due to the accident. Stated another way, had the patient not sustained the accident, they would have likely remained asymptomatic.

For these reasons it remains our viewpoint that a patient who is symptom free prior to a clearly defined event should not be denied ACC cover. It is the accident that is the whole or substantial cause of the symptoms and not gradual process, aging or disease.

**Post-traumatic imaging abnormalities**

There are now improved experimental models that demonstrate how a normal disc may be damaged. Axial load produces increased pressure within the disc and may be combined with flexion, rotation and impulse creating a combination of annulus, end plate and nuclear damage. These changes reflect the clinical history and the events causing the symptoms. They probably can occur in previously normal spines and experimental models certainly favour this environment as facilitating disc damage.\textsuperscript{6,12-18}

Given the clear evidence in large animal models of progressive disc degeneration after injury,\textsuperscript{19} these changes are reasonably considered as post traumatic if they fit the clinical presentation (normal function prior to injury, clearcut accident and later discovery of spondylotic changes). We are unaware of any credible evidence in clinical practice that demonstrates that disc degeneration or spondylosis can be reversed.\textsuperscript{6} The time for the development of injury changes is difficult to determine. Widespread osseous changes are likely to take time to develop, but loss of nuclear hydration alone is likely to occur rapidly. Loss of hydration is an almost universal finding in acute disc prolapse, and as noted, it is unlikely that a significant number of these discs were abnormal prior to the injury.

In summary, as with most musculoskeletal presentations, the history and examination are paramount and the imaging findings are supportive. Previously normal patients who develop low back pain and disability after accident should not be denied ACC cover based on the current Act and the current medical knowledge. However it must be acknowledged that the history given by the patient is not always accurate,\textsuperscript{20} and evidence of pre injury medical or other consultation with pre injury imaging on file would cast doubt over a history of being "previously normal".
Spondylolysis and isthmic spondylolisthesis

Spondylolysis and isthmic spondylolisthesis represent a further area of challenge for the legislation. Again, we see people who have been asymptomatic and unaware of any existence of an abnormality, who subsequently sustain an accident with resulting symptoms and are denied cover. Again, it is important to look at the aetiology of a spondylolysis and its natural history.

Dysplastic spondylolisthesis in childhood can be considered developmental in the medical sense\(^\text{21-23}\) - (developmental, pertaining to the development of a condition during growth, either in the intrauterine phase or early childhood (Dorlands Medical Dictionary). This form of developmental spondylolisthesis may be excluded from cover, being a gradual process. Degenerative spondylolisthesis occurs with facet joint arthritis and variable degrees of disc degeneration resulting in spondylolisthesis. It is generally a condition that occurs over time as a gradual process and is typically symptomatic over a variable duration so it could be excluded from cover, being both a gradual process and a degenerative disease.

Spondylolysis (and isthmic spondylolisthesis) is generally accepted to be a failure or stress fracture of the pars interarticularis under flexion and that fails to heal.\(^\text{24,25}\) This is an acquired condition usually occurring in late adolescence. Once acquired, it is usually stable and asymptomatic. Conventional orthopaedic teaching has been that the incidence of spondylolysis is 6% in the community and 3% of adults have a low grade spondylolisthesis. A recent observational study from the Framingham Heart Project indicates that the incidence of pars defects may be as high as 11% in a cross sectional study.\(^\text{26}\)

Clearly all of these people do not present for treatment. Studies in the Scandinavian literature suggest that there is no evidence that patients with a spondylolysis or low grade isthmic spondylolisthesis have increased risks of back disability through life.\(^\text{27-28}\) This has been confirmed by Frederickson et al whose long-term study did not find any increase in problems throughout life for spondylolysis and isthmic spondylolisthetic patients when compared with normals.\(^\text{30}\) The Framingham Study also found that patients with spondylolysis or isthmic spondylolisthesis had no increase in lumbar spine symptoms when compared with the non spondylolytic or non isthmic spondylolisthetic patients.

In summary, there is no high quality observational literature that suggests that spondylolysis or isthmic spondylolisthesis predisposes to increased back pain or disability in adult life, but there is now good quality evidence that it does NOT predispose to an increased risk or rate disability. Given that spondylolysis (and isthmic spondylolisthesis) is not a gradual process (yet may occur after a specific event or series of events - the normal fracture or stress fracture aetiology), and neither can it be considered a disease in the asymptomatic individual (which may represent 10% of the community) and it is clearly not part of the aging process, there seems no justification for exclusion of these patients from cover if they have been previously asymptomatic and have a clear history of new symptom and a personal injury caused by accident.

The notion that “the accident has brought the condition to light, and the effects of the accident might now be spent leading to the underlying spondylolysis and
spondylolisthesis as being the whole or substantial cause of the symptoms or personal injury” has no support from the current quality observational literature.

**Conclusion**

As in all branches of medicine, and none more so than in the diagnosis and management of back pain a detailed history is fundamental to forming a diagnosis and optimising treatment. Over-reliance on modern high quality imaging increases the chances of unnecessary or inappropriate treatment. The practitioner must consider the widespread tissue changes that can occur with age.

In medicolegal decision-making where there is a need to apportion weight to the contribution of accident to subsequent symptoms or personal injury, it is essential that the decision-making relies is on quality observational population studies, rather than expert opinion that may be generated from skewed referral patterns in a previous practice life. As we have remarked before, the history of accident and the history of pre accident status must be the foundation for the correct application of the ACC Act.

**Competing interests:** None.

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What does degeneration mean? The use and abuse of an ambiguous word

Richard Wigley, Christopher Walls, David Brougham, Peter Dixon

Abstract

The use of the word *degeneration*, particularly in the compensation arena, is not recommended. It is imprecise and is interpreted in different ways by radiologists, clinicians and insurers. Insurers use the word to conclude that any so called *degenerative* changes mean that there is age causation so that compensation can be denied. These changes can be caused by single or multiple injuries continuing heavy work and other causes. Each risk factor should be carefully assessed in each case.

Interpretation of the imprecise and pejorative terms *degeneration* and *degenerative* can be misleading and confusing. It is often assumed by clinicians that degeneration implies an *age relation* and then some insurers make a false assumption that a statistical relation to age equates age to causation. Dorland’s Medical Dictionary defines degeneration as deterioration, change from a higher to a lower form, especially to a lower or less functionally active form.

In the compensation arena it is often stated that “the observed changes are degenerative and therefore due to age and so are not caused by injury or a gradual process from chronic overload or other possible causes.” This can lead to inappropriate refusal of insurance entitlement. Those with osteoarthritis (OA), spinal disc disease or tendinopathy may be refused compensation on an assumption of age causation when age is not the substantial cause or has only a minor role. All possible risk factors should be considered in assessing the causation of all musculoskeletal conditions.

When radiologists use the word *degeneration* they understand that such appearances can result from the cumulative effect of repeated minor and major impacts and physiological use, not just age. Usually changes called degenerative do not cause any symptoms and so are of no clinical importance.

Radiologists expect clinicians to assess, what they report as *degenerative* changes, together with the clinical observations, as part of the normal clinical path leading to diagnosis, but some clinicians and insurers interpret *degeneration* as meaning the ageing process. These conditions would be more precisely described as osteoarthritis for synovial (diarthrodial) joints, spondylosis for the spine and tendinopathy for the tendons as these terms do not imply causation. Insurers may still take these terms to imply age causation.

It is important to distinguish immutable risk factors such as sex, age and genetics which predispose to injury but do not cause injury. An accident or other environmental change is necessary to cause the injury.
The New Zealand Accident Compensation Corporation (ACC) often deems injuries to be aggravating factors of pre-existing degenerative and so age-related conditions, no matter how minor the changes. More logically, these radiological changes should be regarded as being caused by the interaction of several causes (risk factors). The effects of these are cumulative and so will increase with age. This is a statistical age relationship which does not indicate age causation. This association can be coincidental. Minor OA usually does not cause any symptoms at all.\(^4\)

For example it is inappropriate that a 28-year-old nurse with 10 years exposure to heavy lifting as a nurse is described as having degenerative changes at one level, only but is told by her employer and/or the insurer that her pain and loss of function were due to age-related degeneration. More likely, lifting heavy patients and to a lesser extent gardening and playing net ball have combined to produce the lumbar disc protrusion. She was too young to have the loss of tissue resilience of later life which could lead to multi-level changes. So, the decision not to grant compensation in this case was neither logical nor just.

**Spinal disorders**

In the spine degeneration is often used to describe loss of disc height, traction spurs and annular osteophytes. The loss of disc height puts an abnormal load on the facet joints causing secondary OA. Though there are many papers written under the title degenerative disk disease we have not found an explicit definition of this term. Radiological reviews of degenerative diseases of the spine\(^2,3\) consider that the main pathogenic factor is chronic overload and that such changes may not cause symptoms. The name spondylosis is more satisfactory as it does not imply a cause. Degenerative changes in the posterior synovial (diarthrodial) joints are better labelled as osteoarthritis, which may not be caused by age.

Freemont\(^5\) explains that changes said to be degenerative in the spinal discs may result from the interaction of one or more of the following risk factors:

- Diffusion of nutrients and oxygen across the inter-vertebral disc matrix
- Soluble regulators of cell function
- Mechanical load including and
  - acute, repeated and gradual process injuries
  - excessive spinal loading or obesity
- Genetic influences\(^*\)
- Ageing and senescence\(^*\)

\(^*\)Immutable predisposing factors

Others have suggested that micro-fractures in the subjacent bone lead to breakdown of the disc.\(^2\) This suggests injury causation.

Seidler et al\(^6\) found a strong dose-related relationship of cumulative physical load, lifting/carrying or extreme forward bending to lumbar spondylosis (osteoochondrosis) in 229 men attending orthopaedic clinics compared with 197 controls. The same result was found for 135 cases who also had disc herniation.
Battie et al\textsuperscript{7,8} in a large magnetic resonance imaging (MRI) study of twins noted that there is no agreed definition of degenerative disc degeneration. They did not state their inclusion and exclusion criteria nor did Sambrook et al\textsuperscript{9} in a similar twin study.

In a more recent paper\textsuperscript{10} Battie et al define degenerative disease of the spine (spondylosis) as decreased disc height and disc dessication on MRI scan. This restricted definition precludes comparison with other studies. Battie et al\textsuperscript{7,8} found that genetic predisposition had more effect than occupational workload.

Battie et al\textsuperscript{7,8} did not show a relation to age for disc height narrowing, disc herniations or upper end plate changes and only showed a moderate increase for signal intensity, disc bulging, osteophytes or fatty infiltration. These MRI changes are usually described as \textit{degenerative} but reduced disc height, disc herniations, disc bulging and end plate changes can occur in spinal injuries and chronic overload. We have not found direct evidence that age alone can cause such changes.

Using a summative “\textit{degenerative}” scale to assess spinal MRI changes\textsuperscript{10} in 120 subjects, over 40 years old, with chronic back pain, there was a relation to age and global “\textit{degenerative}” change (disc height loss, number of narrowed discs, spinal stenosis, and spondylolisthesis). They found that physical occupational exposure, a heavier work load, pain duration and disability were associated with \textit{degeneration}.

Some disc \textit{degenerative} changes in the spinal discs could be more accurately labelled \textit{internal disruption of the disc (IDD)}.\textsuperscript{11,12} Annular tears usually show on MRI. Though this suggests injury causation this can be labelled “\textit{age related degeneration.”}

Discography induces pain and indicates which disc causes the pain but this is not usually done as it is not without risk.

Schmorl’s nodes\textsuperscript{13} which are heritable,\textsuperscript{14} are often disregarded as irrelevant as they are usually asymptomatic but acute injuries can produce the same appearances with a break (fracture) in the end plate with herniation of disc material into the vertebral body and so can cause back pain. It is not clear whether such cases, or cases of IDD have been excluded in papers entitled \textit{disc degeneration}. Presumably cases with nerve root involvement were excluded.

In a study of diagnostic labels and perceived diagnosis in chronic low back pain Sloan and Walsh\textsuperscript{15} found that the use of degenerative terms, such as wear and tear, were associated by patients with a poor perceived prognosis.

\textbf{Synovial joints—osteoarthritis}

Osteoarthritis of the synovial joints also results from the combination a number of causes (risk factors)\textsuperscript{17,18} so it is misleading to call this \textit{degenerative} arthritis. For example, osteoarthritis of the knee results from a combination of many causes such as:

- Fracture through joints
- Chondral injuries
- Meniscus tears
- Repeated heavy loading, prolonged bending, crouching and squatting\textsuperscript{18}
- Repeated injury
• Obesity
• Knee deformity
• Inflammatory arthritis
• Heredity
• Hypermobility*
• Some rare hereditary conditions* and
• Sex*

(* Immutable factors)

The cumulative interaction of these factors determines the age of onset of symptoms and so there will be an increased prevalence and severity with age at least to retirement age. Studies will then show a statistical relation to age which is coincidental and so does not imply age causation. Yiuqin et al\(^{19}\) have shown that the incidence of osteoarthritis falls after the age of 70 years.

This suggests that age alone is not an important cause. For instance knee cartilage and cruciate ligament injuries, which are so common in footballers, lead to a very high rate of secondary osteoarthritis.\(^{20}\) The prevalence of this will increase with age though it is clearly not caused by age.

Linear (bucket handle) tears in the knee menisci result from acute injuries. More complex partial thickness meniscus tearing has been attributed to “degeneration” and so to age but this could also be due to chronic overload or repeated injuries.

**Tendon disorders—tendinopathy**

Tendon disorders are often described as degenerative though there is no evidence of age causation. This applies to the tendons at the wrist, ankle, hip, rotator cuff tendons and elbow. Tendinopathy of the extensor origin tendons at the elbow (epicondylitis) is common. The suffix *itis* suggests inflammation which is usually not evident so the term tendinopathy is preferred as this does not imply inflammation. If the synovial sheath of the tendon is inflamed the name tenosynovitis is appropriate.

Repetitive tendon overload in athletes causes tendinopathy and sudden excess load can rupture the tendon. Age-related muscle atrophy is associated with elasticity changes in the tendon\(^{21}\) implying greater susceptibility to injury.

The alleged degenerative changes in tendons may be caused by a combination of factors such as:

• Repeated overload
• Single injuries
• Multiple injuries
• Sport injuries
• Vibration
• Obesity\(^{22}\)
Cefloxacin
Age (immutable)
Genetic\textsuperscript{2,3}

Tendinopathy may be found in the absence of symptoms in the shoulder. The study by Allander\textsuperscript{24} showed a decrease in the prevalence and incidence of shoulder pain and epicondylitis past the age of sixty. This is contrary to expectation if age was the main cause and suggests that occupation, and/or the other factors listed above are the explanation.

**The rotator cuff**

Acute injuries and sustained overloads can cause partial or complete tears of the rotator cuff tendon without causing symptoms. Again a relationship to age may be assumed to imply age causation without considering the alternative causes.

In a 20-year prospective study of 883 asymptomatic subjects,\textsuperscript{25} 63 developed chronic shoulder disorders. Work exposure to repetitive shoulder movements increased the risk (odds ratio [OR] 2.3) and vibration (OR 2.5) of developing shoulder disorders. For three of these risks, lifting heavy loads and working in awkward postures the risk increased to an odds ratio (OR) of 4. \textit{“The effects seem to be long-term so that the accumulation of damage in shoulder tissues can be seen several years after work life has ended.”}

Age relationship was only significant for women and only body mass for men. Highly repetitive arm activity and sustained 60 degrees flexion or abduction can cause rotator cuff injuries.\textsuperscript{26} In asymptomatic volunteers\textsuperscript{27} full thickness rotator cuff tears increased with age up to 50 years but did not increase past 50. Shoulder tendinitis was more common in bricklayers, rock blasters and with those with vibration exposure compared to foremen.\textsuperscript{28}

Similarly tendinopathy and/or rupture of the Achilles and other tendons can be caused by acute injuries and repeated overload in sport and similar occupational activities.\textsuperscript{29}

**Conclusion**

Radiologists, clinicians and insurers frequently put different interpretations on the word \textit{degeneration} leading to confusion. It is suggested that this ambiguous word should be abandoned and replaced by osteoarthritis, spondylosis and tendinopathy as these terms do not imply causation. This would prevent false assumptions of age causation leading to flawed legal decisions in the New Zealand environment hindering early rehabilitation to the disadvantage of the patient. The various risk factors for each disorder should be carefully assessed in each case. Prevention, control strategies and compensation decisions would then be more logically based.

**Competing interests:** None.

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Ulnar artery ischaemia following corticosteroid injection for carpal tunnel syndrome

Syed S Hussain, Chris Taylor, Rupert Van Rooyen

Abstract

A 36-year-old female with known bilateral carpal tunnel syndrome was admitted to hospital and given a steroid injection on the ulnar side in her right hand. She suffered immediate ischaemia of the 3rd, 4th, and 5th fingers. Imaging showed decreased flow in the 4th and 5th phalangeal arteries. Treatment with iloprost infusion commenced 7 days after the injury, with moderate improvement and further managed with a carpal tunnel release. This case report sheds light on an unusual yet very important complication of carpal tunnel management.

Carpal tunnel syndrome (CTS) is the most commonly seen peripheral neuropathy. Prevalence of clinically proven and electro-physiologically confirmed disease is about 3%, with a male to female ratio of 1:3. Characteristic symptoms include numbness, tingling and pain involving the radial 3.5 digits. Paresthesia in CTS can radiate to the forearm, elbow and shoulder. Weak grip and thenar muscle hypotrophy may be observed in long standing or severe form of the disease.

Treatment options include oral medication; for example, NSAIDs, local corticosteroid injections and surgical carpal tunnel release. Local corticosteroids have a proven efficacy over placebo, as displayed in a study conducted by Dammers and Girlanda et al. In this study follow-up visits of CTS patients 1 month post injection showed improvement of symptoms in 75% of the cases. Graham et al identified that 10% of patients with CTS are likely to benefit from local steroid injection, and remain symptom free for at least 1 year.

Case report

A 36-year-old, right-hand-dominant female, with no known comorbidities was admitted to hospital. Her only significant past history was allergy to cows for which she had been taking cetrizine 10 mg for 9 months, as her job involved milking cows.

Patient had symptoms consistent with carpal tunnel syndrome for about 5 years and a history of receiving corticosteroid injection in the left hand. Due to similar symptoms in the right, the patient was given a corticosteroid injection with an ulnar approach in the right hand.

The patient reported a gush of blood from the insertion site as soon as the needle was removed after steroid delivery. Soon after this her 3rd, 4th and 5th phalanges became pale, then black, associated with severe tenderness and subsequent numbness.

On examination the patient was found to have bluish discoloration of the tip of right 4th and 5th digit (Figure 1). She demonstrated painful and restricted flexion of the
same with severe tenderness to touch. The 3\textsuperscript{rd} digit appeared less affected as compared to the little and ring finger. The right index finger and thumb were normal on examination.

**Figure 1. Bluish discoloration of left middle ring and little finger (ischaemia more evident in lateral two digits as compared to middle finger)**

The patient’s lab results were all within normal limits, including coagulation profile. No pathology was observed on plain X-ray. With the impression of thrombosis, a digitally subtracted arteriogram was performed. DSA showed poor distal filling of the 4\textsuperscript{th} and 5\textsuperscript{th} digit (Figure 2). All other digital arteries along with the palmar arch displayed normal flow.

**Figure 2. Poor distal filling of the 4th and 5th digits**

The patient was started on iloprost (prostacyclin I\textsubscript{2}, infusion, an arterial dilator, and after 24 hours of treatment, a change in colour of the skin with decreased pain was noted. Keeping the initial presenting complaint in mind, a carpal tunnel release was also performed, following which further improvement was noted.
Gradually the colour of the fingers, paresthesias and pain reduced. The patient was discharged after 1 week of hospital stay (Figure 3).

**Figure 3.** No noticeable discoloration at the time of discharge (no colour difference between the marked areas and surrounding skin)

### Discussion

CTS is a common finding General Practitioners in New Zealand come across. Steroid injections are of proven benefit with a reasonably safe profile. Although rare, there are possible complications that can further deteriorate the condition. Bleeding and infection are common complications and can be avoided by using careful techniques.4

Ozturk et al used 150 cadaveric wrists and three different approaches to find the best technique for steroid injection in CTS.7 It was seen that 1cm proximal to the wrist crease, through the FCR tendon was the safest approach. This was further supported by an anatomical data analysis of median nerve variation by Thierry Dubert and Otilia Racasan.8 Ulnar approach, however, has only been shown to be beneficial when done sonographically.9

There have been a few case reports10–12 of worsening symptoms secondary to direct injection of the median nerve, which were surgically confirmed.

One such case report also showed synovitis in response to the injection that further deteriorated the condition and took more than a year of medical and physiotherapy to relieve the symptoms.13 From our literature search, however, we only found one case report where a corticosteroid injection caused ischaemia.14

Reasons for the ischaemia and thrombosis are currently unclear, and research so far does not reveal the role of steroids in causing ischaemia, vasospasm or thrombosis. Considering other constituents of the steroid suspension possibly responsible, would be a guess worth investigating.

In conclusion, although steroids injections are considered safe, caution needs to be observed while performing the procedure. Practitioners must be aware of possible complications, and their management options especially in crucial cases such as the one discussed.
The safest location of the injection remains controversial. A comparative study of 124 carpal tunnel releases performed by O’Racsan and TH Dubert\textsuperscript{15} in 2004 showed that Median nerve can be injured by the injecting on either side of the Palmaris Longus tendon. Ulnar approach can injure ulnar artery and nerve. They concluded the safest approach for a carpel tunnel injection was through the FCR tendon.

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A rare cause of chest pain

Goutam Datta, Dipankar Mukherjee, Bhuban Majhi

A 26-year-old nondiabetic, nonhypertensive male patient presented with acute coronary syndrome. He had absent pulses in both upper limbs. Pulse volume in left carotid was less than right carotid. Electrocardiogram showed ST-segment depression and T-wave inversion in inferior leads. Quantitative troponin T was 16 ng/ml. Coronary angiogram revealed significant stenosis in ramus intermedius, obtuse marginal as well as in left circumflex artery. There was 75% stenosis at midsegment of right coronary artery. Aortogram showed an occluded right subclavian artery after the origin of right vertebral artery and left subclavian artery was occluded from its origin. There was also stenosis in the left common carotid artery. Abdominal aorta and its branches were found to be normal. See Figs 1–3. What is the diagnosis?

Fig 1. Right anterior oblique caudal view showing lesion in left circumflex and left anterior descending artery

Fig 2. Left anterior oblique view showing lesion in right coronary artery

Fig 3. Aortic arch angiogram showing both subclavian artery cutoff and lesion in midpart of left common carotid artery
Answer and Discussion

*Takayasu’s arteritis* is a chronic idiopathic large vessel vasculitis involving the aorta and its major branches mostly affecting young females in their 2nd and 3rd decades. It evolves through two phases—early prepulseless or active phase and late pulseless or ischaemic phase.

In Takayasu’s arteritis, arterial stenoses occur three to four times more often than aneurysms. Patient may present with cerebrovascular disease, ocular disorder and renovascular hypertension. Mechanism of vessel involvement is thickening of vessel secondary to fibrosis of all three layers. Claudication (more than 60% upper versus approximately 30% lower extremities) is the most common complaint and bruits (approximately 80%), blood pressure and pulse asymmetries (60 to 80%) are the most common findings. Coronary artery involvement particularly ostial and of proximal segment are known to occur in 9–10% of cases. Skip lesions and diffuse triple vessel involvement has also been reported.

Coronary artery involvement has been classified into three groups:

- Type-I Stenosis and occlusion of coronary ostium and proximal segment.
- Type-II Diffuse or focal coronary arteritis, may have skip lesions.
- Type-III Coronary aneurysm.

Patients may suffer from angina, acute myocardial infarction or congestive heart failure. Even sudden cardiac death may sometimes be the first manifestation of Takayasu’s arteritis.

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Deliverance from exophthalmic goitre deaths

Dr Coulquhoun’s 1910 account of the deaths of two teenage girls and a teenage boy from exophthalmic goitre, vividly demonstrates Medicine’s wonderful progress in the last 100 years.

Out of the mass of mysterious diseases of that time, Robert Graves in Ireland and von Basedow in Germany had discovered the often-lethal syndrome of goitre, exophthalmos, tachycardia, tremor, weight loss and increased appetite. The cause was unknown, until surgeons found that excising the thyroid gland cured the disease.

Triumph at the success of this operation was abolished by the finding that the patients became vegetables, suffering from *Caehexia Strumipriva*, myxoedema. However, it was swiftly found that this was cured by ingestion of pills of thyroid siccum, from meat animals. This demonstrated that the thyroid gland produces a hormone that is essential for life and guided surgeons to performing sub-total thyroidectomies as therapy for Graves’ disease. Sir Charles Harington isolated thyroxine, the thyroid hormone, the first hormone to be isolated, finding it to contain 4 atoms of iodine. This explained the aetiology of endemic goitre, which is caused by iodine deficiency.

In 1928, Chesney and colleagues at Johns Hopkins found they could produce goitre in rabbits by feeding them a cabbage diet. This was the forerunner of medical treatment for thyotoxicosis. Cabbage contained a positive goitrogenic substance, as opposed to the negative goitrogenic effect of iodine deficiency.

In research set up by Sir Charles Hercus, HD Purves, a brilliant scientist, knowing that thyroxine production is controlled by the pituitary gland’s thyroid-stimulating hormone (TSH), with negative-feedback by thyroid hormone blood levels, realised that goitrogens act not by stimulating the thyroid, but by inhibiting thyroid hormone production, so that the goitre is caused by excessive TSH from the pituitary gland.

Therefore, Purves, Griesbach and Kennedy set about finding an anti-thyroid substance for treating thyrotoxicosis. Kennedy succeeded in 1942, a year before Astwood, independently, had the same success in the United States from exploring side effects of sulpha drugs.

As Dean of the Otago Medical School, Sir Charles Hercus, fostered research by setting up a Medical Research Council that funded highly-successful full-time research units, ranging from Endocrinology to Autoimmunity, with solution of the pathogenesis of Graves’ disease and autoimmune disease in general, discovering the principles needed for effective immunotherapy and prophylaxis, a major contribution to human welfare.

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Pathognomonic rash

Pandey et al have reported a patient in whom they have diagnosed erythema multiforme. However, the diagnosis may be incorrect. Figure 2 shows a target lesion, as described by the authors. Figure 1, showing rash on the pelvis, reveals urticaria.

A target lesion is a fixed round erythematous plaque less than 3 cm in diameter, with a well-defined border and consisting of three distinct zones; two concentric rings of colour change surrounding a central circular zone. Target lesions are not pathognomonic of erythema multiforme, which despite its name usually results in a symmetrical eruption of papules and plaques mainly distributed on distal limbs. Individual lesions have a relatively monomorphic clinical appearance.

Similar target-like lesions may also arise in urticaria, viral exanthems, drug eruptions, toxic epidermal necrolysis, polymorphous light eruption, cutaneous lupus erythematosus, bullous pemphigoid, linear IgA bullous dermatosis, erythema annulare centrifugum and vasculitis.

Erythema multiforme is frequently overdiagnosed by nondermatologists in patients presenting with acute urticaria. In urticaria, the lesions last less than 24 hours in one site and have normal overlying skin, whereas the target lesions of erythema multiforme persist 7 to 10 days and display central epidermal damage in the form of bullae or crusts.

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Reference:

Anterior cruciate ligament reconstructions, the debate continues—with another response by Assoc Prof Hooper

In 2005 the Cochrane Collaboration analysed all randomised and quasi-randomised trials comparing surgery with conservative treatment of ACL rupture in adult. They concluded that there was no evidence to determine whether current surgical techniques or conservative management was best treatment for ACL injuries. They recommended that good quality randomised trials were required to remedy this situation. In 2009 this paper was updated with no changes to its conclusions.

In his reply to my previous letter Mr Hooper promotes ACL reconstruction, claiming it "improves knee function in greater than 90%, with the majority of patients returning to aggressive sporting and working activities, especially those activities that involve twisting or rapid change of direction." Neither of the papers he quotes support his opinion. The first paper is a non-randomised retrospective study promoting ACL reconstruction in the non-athlete. The second paper is an assessment of 70 randomised controlled trials in regard to surgical technique and rehabilitation of ACL injuries. The authors concluded that none of the papers were high quality and they stressed the need for further studies.

I found five papers on the natural history of an ACL rupture. The most rigorous of these studies involved 50 patients with complete ACL ruptures. The MRI findings were that 21 (42%) had a normal ACL and 20 (40%) had a partial repair at 3 months. Mr Hooper claims that full thickness midsubstance tears (of the ACL) rarely heal. He is wrong. Nor do the papers he cite support his opinion. The first paper explores the effect of anterior cruciate ligament trauma and bracing on knee proprioception. The other follows 49 patients who had ongoing problems with their knees following ACL ruptures.

Mr Hooper justifies ACL reconstruction by stating there is no evidence that it is responsible for the development of long-term osteoarthritis. He misses the point of my letter. The onus is on the surgeon to prove that ACL reconstruction doesn’t cause long-term osteoarthritis and that study hasn’t been done. He also states “Instability is the commonest indication for ACL reconstruction” without supporting evidence. In my clinical experience instability has never been the indication for an ACL repair, but rather the patient has an ill-defined fear of future problems with the joint if they don’t have surgery.

Mr Hooper claims my concerns regarding ACL reconstructions are inappropriate and misinformed. How so? The papers he cites to support his opinion are irrelevant to his claims. His “Best Practice” is based on an ACC document written in 2002 that has been superseded twice by the Cochrane Collaboration.

ACL reconstruction surgery has no randomised controlled trial (RCT) support.

Dr Nicholas Cooper
General Practitioner
Epsom, Auckland
Assoc Prof Hooper’s response

The anterior cruciate ligament (ACL) is not a redundant structure. Rupture causes anterolateral rotary instability which can be disabling, even in routine activities of daily living. Some patients can cope with life changes to accommodate for this but most continue to experience episodes of instability. The overwhelming literature and clinical experience supports reconstruction in these patients to improve knee stability.

ACL reconstruction became popular in the late 1970s when the instability pattern was recognised and procedures that produced less morbidity were advanced. This was popularised because ACL ruptures rarely healed. Today the treatment of ACL rupture is being directed towards methods of manipulating the environment to enable ACL healing. The millions of dollars directed towards the genetic engineering to achieve this tissue modification would not be spent if the ACL healed.

Constructing a RCT at this stage in the evolution of ACL treatment would be impossible. Firstly, gaining ethical approval would be unlikely and secondly, recruitment into the non-operative group in a young active cohort would be difficult at best.

It is true that there are few RCTs to support reconstruction but this is not uncommon in surgical practise where a procedure has produced such a profound improvement in function. Few would disagree that total hip replacement has been a successful operation but there have never been any RCTs to prove this.

Does this mean that hip replacement is not a proven procedure?

Assoc Professor Gary Hooper
Head of Department, Orthopaedic Surgery and Musculoskeletal Medicine
University of Otago, Christchurch
Is the quality of evidence for air quality standards adequate?

Longley and Hales,\textsuperscript{2} when commenting on the conclusion of Palmer and Mann\textsuperscript{1} that the evidence shows that “Lowering the concentrations of PM$_{10}$ by reducing the emissions from home fires may not ameliorate the adverse effects from to (sic) PM$_{10}$ pollution...”, write “\textit{This sounds like a testable hypothesis, given that such reductions in wood fire emissions and PM$_{10}$ are currently happening in Christchurch...}”

Indeed, yes! There is already a large amount of pertinent Christchurch data. Hales et al\textsuperscript{3} reported on the association between variations the number of daily deaths and concentrations of PM$_{10}$ in Christchurch. There were considerable variations in PM$_{10}$ concentrations between years, but no correlation between these concentrations and yearly death rates were noted.

McGowan et al\textsuperscript{4} reported finding increased respiratory admissions after days of high PM$_{10}$ pollution in Christchurch from 1988 to 1998. During the first 5 years of the study the median winter time concentration of PM$_{10}$ averaged 33 micrograms per cubic metre. During the last 5 years it averaged 21 micrograms.\textsuperscript{5} Respiratory admissions did not decrease over the years, but persistently increased to almost double during the 11 years, with no indication of synchronisation of concentrations and admissions.

This feature of the study was pointed out for comment by Palmer,\textsuperscript{6} but none has been forthcoming.

Since then, the HAPiNZ Christchurch Pilot Study has measured the air quality in the hill suburbs (such as Cashmere) where about 8\% of the city's people live.\textsuperscript{7} As we all knew, the air there was of pristine quality. However the study assumed that “there is likely to be a generally homogeneous exposure to similar PM$_{10}$ levels for all city residents”, and did not ask whether the people living on the hills were healthier than those on the plain.

The large differences in PM$_{10}$ concentrations from two major different sources in summer and winter, and distinct geographical regions with markedly different PM$_{10}$ concentrations but demographically similar populations make the Christchurch data interesting. It is high time the available data were examined by some group not imbued with the ideology that all PM$_{10}$ whatsoever, wheresoever, is equally harmful. It will not suffice to have the same cohort of incumbent epidemiologists studiously and selectively not addressing the pertinent questions which I and others have raised.

Longley and Hales agree that it is highly plausible that “woodsmoke and other urban particles are involved in different biological responses leading to different health endpoints. However, as Kingham illustrates (quoting from an extensive international review\textsuperscript{8}), international research on this issue is not currently consistent”. Then, despite the inconsistency, they conclude that we must accept, along with WHO, EU and USEPA, that all PM$_{10}$ is equally toxic, their own good Christchurch evidence to the contrary notwithstanding.
I do not see the need to offer a quantitative solution, which would run into hundreds of millions of dollars, for what on the available evidence may well be a minor problem.

The “one size fits all” statistical straight-jacket into which the regulators would like to squeeze all communities in New Zealand and around the world may make for easy law and administration, but it is not good science, and may not lead to improved health outcomes.

As Karl Popper used to tell us in Canterbury, one proven exception disproves the rule. There may be more exceptions than Christchurch.

Pat Palmer
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References:

Government response to air pollution articles in NZMJ

Dear Sir

We are writing on behalf of the Ministry for the Environment and the Ministry of Health with regard to the air pollution articles published on 4 March 2011 in the NZMJ. This is intended as a formal comment on the standard of the viewpoint articles written by John Hoare and Peter Moller.1,2

As noted in your [Simon Kingham’s] editorial, each author presents a one-sided perspective questioning “the effect wood smoke from domestic home heating has on health, without presenting any new primary data.”2 Our concern is that these papers offer unsubstantiated criticism of the Government’s national environmental standards for air quality (Resource Management (National Environmental Standards Relating to Certain Air Pollutants, Dioxins and Other Toxics) Regulations 2004).

Without detailing each inaccuracy here, in general terms, the papers use the discredited method of data-mining and are inappropriately and/or selectively referenced.3 Neither paper can be regarded as balanced critical appraisal of the many hundreds of well-designed studies that have been published in this area and which have formed the basis of government development of air quality standards not only in New Zealand but in Australia, the United States and Europe.

While the authors are free to be selective in their choice of data to support their views, governments must adopt the ‘weight of evidence’ approach when it comes to determining public policy. There is always room for healthy skepticism on the epidemiology of air pollution, but one or two studies do not represent a consistent finding, and should not detract from the overwhelming weight of evidence.

As noted by Dockery and Pope—who are internationally recognised for their seminal work on particulate matter and health—it is remarkable that studies of mortality and short-term changes in particulate matter are capable of observing such small effects. They do so, however, and consistently do so over multiple cities and countries on different continents, using various methods including alternative time series analytic approaches and case-crossover designs.

When these results are coupled with a) the much larger observed chronic health effects from long-term exposure to particulate matter, b) the findings of laboratory human exposure studies, and c) increasing recognition of biologically plausible mechanisms by which particulate matter causes adverse health impacts, the margins for this skepticism become very small. Indeed given the weight of evidence, there is an imperative to act to protect public health.

Policies and actions taken to improve air quality should indeed leave home owners able to keep warm in winter. The authors appear to have overlooked the Government’s $347 million Warm Up New Zealand: Heat Smart programme which has funded insulation and clean heat appliances in 19,500 homes since 2009. In addition, the $1.1 million per annum EnergyWise Clean Heat programme fits clean heat appliances in around 600 low-income houses in polluted airsheds each year.
Finally, while the paper seeks to criticise central government policy, no opportunity was given to either Ministry to comment on the articles prior to going to print. This would have helped to ensure an objective assessment and avoid obvious errors which might mislead the reader. Publishing this letter in your journal will assist to redress this.

Kevin Currie  
Director  
Environmental Protection  
Ministry for the Environment

Dr Darren Hunt  
Acting Director of Public Health  
Clinical Leadership, Protection & Regulation  
Ministry of Health

Wellington, New Zealand

References and endnote:


4. For example Moller reference to The Economist for percentage contribution of road transport (presumably to PM$_{10}$ – the paper does not say). Moller reference to only three papers relating to (presumably again PM$_{10}$ – the paper does not say) Christchurch when in fact there are many more published. Similarly Moller references to Pelucchi et al and Hartog et al are inconsistent with hundreds of other papers finding mortality and morbidity associations with PM$_{10}$. 
Obituary on A L Napier Maclean (1866–1912) by Philip James

Published in NZMJ 1912 May;11(42):139.

The subject of this notice who died on February 23rd, 1912 after a long illness, was the son of an old Indian Army Doctor who served during the Mutiny. He was born in India but spent his childhood and early youth in France, where the family resided and where the children were educated.

He did not visit England nor could he speak English until he was fifteen or sixteen years old. He spoke English without the least trace of a foreign accent but the writer has often noticed and been amused at his use of certain quaint idioms and phrases which indicated his early French training and habits of thought.

After his retirement from the Indian Medical Service Maclean's father settled in Sydney, N.S.W., and was a physician to the Sydney Hospital, where he remained until his death, after which the family removed to England. Maclean was not brought up to any business or profession but devoted himself to music and sport, in both of which he attained high proficiency. It was not until 1897 or 1898 that he determined to enter the medical profession, and accordingly he entered at the "university of Aberdeen and qualified in 1903.

After spending a short time in London studying Tropical diseases he came to New Zealand in 1904 and settled in Wellington, where he remained until his death. For the first two or three years the writer's acquaintance with him was only casual and slight, "but in later years the acquaintanceship ripened into friendship -a friendship that was: never broken or interrupted but continued to grow until death, put an end to it. It is difficult for one friend to write of another without laying himself open to the charge of undue bias, but it can be truthfully said of Maclean that in his professional relations he was the very incarnation of honour.

For humbug and for everything that was false and dishonest he had the greatest abhorrence and intolerance—an intolerance which was apt to find expression in words more forcible than polite. He was popular with all that he came in contact, as he had a peculiarly lovable disposition, but it was only the few to whom his whole self was revealed. These few had to be "straight." During his few years in Wellington he had taken a great interest in sport, more especially in boxing, of which he had a thorough knowledge.

He rendered invaluable services to the local Boxing Association, frequently acting as referee and was much sought after by other Associations in the same capacity. As a referee it may safely be said that he had not his peer in New Zealand, and he always commanded the implicit confidence of the competitors. They knew they were going to get a "square deal." His loss to sport is incalculable and it will be long before his like again arises.
Professionally, in spite of long absences, he was succeeding, and if his life had been spared there is no doubt that he would gradually have built up an honourable reputation founded on that, best of all foundations—Honesty.

But alas! this was not to be. During the closing weeks of his life the writer was in almost daily contact with him and a witness of the calm and brave manner in which he contemplated the inevitable end. Despite the weariness and lassitude incidental to his disease, and despite frequent periods of acute distress and suffering, he was always bright and cheerful, although from the first he fully recognised his condition and was not buoyed up by illusive hopes of recovery.

To those around him who knew how rich he was in all those things that make life attractive there was something deeply pathetic in the spirit of resignation he displayed. In him the writer mourns the loss of a clear friend whose place can never be filled.

"Farewell! a word which must and hath been"
"A sound which makes us linger, yet Farewell!"

PHILIP JAMES.
Proceedings of the 207th Scientific Meeting of the Otago Medical School Research Society, Wednesday 18 May 2011

Activin C: a possible therapeutic target in prostate cancer progression. E Ottley, E Gold. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Prostate cancer (PCa) is a worldwide health concern. Broadly, two forms of PCa exist, latent organ-confined and aggressive metastatic. Treatment options for latent PCa are available and effective, whereas treatment options are limited for metastatic PCa. Activin A is a negative growth regulator in the prostate thereby inhibiting PCa progression. Activin A bioactivity is normally tightly regulated via antagonists; activin C is an antagonist of activin A and over-expression is associated with prostate hyperplasia. Thus we proposed that increased activin C maybe associated with PCa progression.

To address our hypothesis, we assessed proliferating cell nuclear antigen (PCNA) as a marker of proliferating cells, Smad-2 (an activin A signalling molecule) and p53 in the prostate of transgenic mice (TG) aged 9 months over-expressing activin C compared with wild-type (WT) controls (WT = 4; TG = 6). Significant increases were evident for PCNA (mean ± SEM, WT, 32.4% ± 4.8 vs. TG, 42.9% ± 2.2, \( P < 0.05 \), unpaired \( t \)-test) and p53 (WT, 27.6% ± 0.43 vs. TG, 38.5% ± 3.4, \( P < 0.05 \)), while Smad-2 decreased (WT, 48.53% ± 2.29 vs. TG, 25.17% ± 1.39, \( P = 0.001 \)). Low-grade prostatic intraepithelial neoplasia lesions, a precursor to PCa, were found in 30% of the TG mice.

Human prostate sections were assessed for activin staining intensity (where 0 = nil staining and 3.5 = intense staining), and Smad-2 signalling. Activin C was increased in PCa compared to benign prostatic hyperplasia (BPH) (PCa 3.1 ± 0.3, BPH 1.8 ± 0.4, \( P < 0.001 \)) and Smad-2 positive nuclei decreased (27% ± 0.6 vs. 16% ± 1.3, \( P < 0.001 \)).

Decreased Smad-2 in association with increased activin C indicates antagonism of activin A in the development of mouse and human prostate pathology, therefore activin C may be a novel therapeutic target to modulate PCa progression.

Identification of friend and foe: metagenomics of the oral cavity in health and disease D Sundaresan1, M Cullinan1, B Drummond1, G Seymour1, J Stanton2, N Heng1. 1Sir John Walsh Research Institute, Faculty of Dentistry, 2Department of Anatomy & Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

The human mouth is home to more than 700 microbial species, collectively known as the ‘oral microbiota’. Many species, mainly bacterial, cause oral infections such as dental caries and periodontitis (gum disease). However, only a small fraction of oral inhabitants are culturable. This project aimed to characterise, using next-generation
DNA sequencing technology: (a) the oral bacterial diversity in human participants at different stages of life, and (b) identify any shifts in the bacterial population in individuals with caries or periodontitis.

Samples from five intraoral sites were taken from 18 children and 15 adults, including individuals who were either periodontally healthy or those that had caries or periodontitis. Genomic DNA was purified and then subjected to polymerase chain reactions targeting hypervariable regions of the bacterial 16S ribosomal RNA gene. High-throughput DNA sequencing utilised the GS-FLX Titanium System and the data were processed by the Ribosomal Database Project Pyrosequencing Pipeline. A list of the ten most abundant species, i.e., the “Top 10 List”, was then compiled for each oral sample.

Analysis of the Top 10 Lists revealed that there was a significant species shift from healthy to diseased states, i.e., from a mixture of Gram-positive (e.g., *Streptococcus*) and Gram-negative genera to a predominantly Gram-negative population. Surprisingly, bacterial pathogens such as *Streptococcus mutans* and *Porphyromonas gingivalis* were conspicuously absent in individuals with caries and periodontitis, respectively. Whereas *Prevotella denticola* was commonly encountered in periodontitis samples, the picture was less clear with dental caries in that there appeared to be a reduction in *Leptotrichia* species (a Gram-negative genus) with a concomitant increase in streptococci.

This project demonstrates that next-generation sequencing technology is a powerful tool to study the oral microbiota. Furthermore, results show that some of the bacterial culprits in caries and periodontitis may have less of a pathogenic role than previously believed.

**Patterns of α-MSH-induced neuronal activation in the pregnant rat brain.** E Scherf, S Ladyman, D Grattan. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

During pregnancy, food intake is increased despite an increase in plasma leptin levels, which would be expected to suppress food intake. At least part of leptin action is mediated by α-melanocyte stimulating hormone (α-MSH), a peptide released by pro-opiomelanocortin neurons. In response to elevated leptin, α-MSH is released, activating melanocortin receptors on target neurons in the paraventricular, ventromedial and arcuate nuclei of the hypothalamus to mediate leptin’s anorectic effect. Therefore, it was hypothesised that leptin resistance during pregnancy may be associated with a loss of response to α-MSH in distinct hypothalamic regions. To test this hypothesis, we used immunohistochemistry for c-Fos, a marker of neuronal activation, to examine the response to α-MSH in pregnant and non-pregnant rats.

Pregnant (n = 6) and non-pregnant (n = 7) rats had indwelling cannulae surgically implanted to allow injections into the lateral cerebral ventricle. On day 14 of pregnancy (or diestrus, in non-pregnant rats), animals were injected with α-MSH (10 µg) or saline. Ninety minutes later, they were anaesthetised and transcardially perfused, and the brain collected and processed for immunohistochemistry for c-Fos.
In response to α-MSH, non-pregnant rats showed a significant increase in numbers of neurons expressing c-Fos in the arcuate nucleus and ventromedial hypothalamus (VMH) (43 ± 1.5 to 77 ± 14 and 33 ± 5 to 106 ± 7, respectively) compared with the vehicle controls (ANOVA, Neuman-Kewls post hoc test, \( P < 0.05 \)). Pregnant rats did not show this increase, with no significant change in numbers of neurons expressing c-Fos following α-MSH injection.

These data support our hypothesis that there is a loss of response to α-MSH in the arcuate and VMH hypothalamic nuclei during pregnancy. This is likely to contribute to the state of leptin resistance during pregnancy, facilitating increased food intake and weight gain.

**Effects of aging on arginine metabolism in the striatum and spinal cord in rats.**  
M Fleete, P Liu. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Aging is a multi-factorial process, and leads to cognitive decline. Recent evidence suggests that altered arginine metabolism in memory-related brain structures contributes to cognitive decline during aging. Arginine, a semi-essential amino acid, is metabolised by nitric oxide synthase (NOS), arginase and arginine decarboxylase (ADC) to produce a number of active molecules. The striatum is the brain region important in reward-association learning and visual-recognition-memory. The present study investigates the effects of aging on the three enzymes involved in arginine metabolism in the striatum, as well as the spinal cord as a comparison.

Male Sprague-Dawley rats, 3 (young, \( n = 9 \)) and 24 (aged, \( n = 9 \)) months old, were euthanised. The anterior and posterior portions of the striatum and cervical spinal cord from each animal were collected. Radioenzymatic and spectrophotometric assays were used to measure the levels of NOS and arginase activities, respectively. The western blot technique was used to determine the protein levels of neuronal NOS (nNOS), endothelial NOS (eNOS), arginase I (AI) and ADC.

There were no significant differences between groups in NOS activity in the anterior or posterior portions of the striatum. However, a significant decrease with age in NOS activity was observed in the cervical spinal cord (Student’s \( t \)-test, unpaired \( t(15) = 3.18, P < 0.01 \)). Arginase activity and the protein levels of nNOS, eNOS, AI and ADC did not differ between the young and aged groups in any region examined.

The present study found no alteration in the activity or protein levels of NOS, AI or ADC with age in the striatum. Interestingly, there was a dramatic decrease in NOS activity with age in the cervical spinal cord. This finding merits further investigation to understand the effects of aging on arginine metabolism in the spinal cord and the functional significance it may hold.
Plasticity-related down-regulation of microRNA regulators of gene expression. B Ryan¹, D Guévremont¹, M Ryan¹, B Logan², W Abraham², J Williams¹. Brain Health Research Centre, ¹Department of Anatomy and Structural Biology, Otago School of Medical Sciences, ²Department of Psychology, Division of Sciences, University of Otago, Dunedin.

The persistence of long-term potentiation (LTP), a widely accepted model for memory, depends on new gene expression. Recently, using microarray expression profiling, our laboratory found that newly-discovered molecules termed microRNA (miRNA) are likely to contribute to the control of gene expression in dentate gyri 20 min after LTP induction in awake rats. The aim of this research was to validate the connection between a select group of these microRNAs and LTP persistence.

This study was undertaken in three parts. First, a literature search was performed to link the potentially LTP-related miRNAs with synaptic activity in previous studies, in order to determine which miRNAs warranted further investigation. Second, real-time reverse transcription-quantitative PCR (RT-qPCR) analysis was used to confirm the microarray data for the miRNAs of interest (n = 4). Finally, we performed real-time RT-qPCR analysis of additional miRNA samples (n = 5) to further pursue the relationship between the miRNAs of interest and LTP.

The literature search revealed that three of the miRNAs that were differentially expressed in the microarray had previously been linked to synaptic plasticity: miR-132, miR-181c and Let-7d. RT-qPCR confirmed that miR-132 was down-regulated by 0.77 ± 0.06 fold (mean ± SEM, \( P = 0.03 \) one-tailed \( t \)-test; n = 4). However, using the current technology, miR-181c and Let-7d were not found to be differentially expressed (\( P > 0.05; \) n = 4).

MiR-132 regulates dendritic morphogenesis in an activity-dependent manner by decreasing synthesis of p250GAP, which results in activation of an actin remodelling pathway. Thus, regulation of miR-132 in response to LTP, as observed in our study, may regulate dendritic morphogenesis, a process thought to contribute to LTP persistence. Our results indicate that miR-132 is a promising candidate for further study of LTP-related gene expression.

Crystallisation and preliminary X-ray diffraction of an ATP-bound Hsp70 from Escherichia coli. A Gommans¹, M Mayer², S Wilbanks¹. ¹Department of Biochemistry, Otago School of Medical Sciences, University of Otago, Dunedin. ²Centre for Molecular Biology, University of Heidelberg, Germany.

The molecular chaperone Hsp70 mediates protein folding and stability. Malfunction of molecular chaperones leads to failure of proteostasis, contributing to diseases as diverse as cancer and Alzheimer’s. The ATP-bound structure of Hsp70 has not been characterised; understanding this form will give crucial insight into how the two domains of Hsp70 interact to protect client proteins in proteostasis. We have produced crystals of a modified Hsp70 bound to ATP and collected X-ray diffraction data for structural characterisation.

Modifications of Hsp70 were inspired by the structure of Hsp110, a distant homologue that is neither a molecular chaperone nor ATPase. Modified Hsp70 lacks...
the carboxyl-terminal 33 amino acids, contains both a T199A mutation that abolishes ATPase activity, and two additional cysteines that form a cross-link designed to lock the two domains of Hsp70 together in the ATP-bound state. Modified Hsp70 was expressed in *Escherichia coli* BB1553 (ΔDnaK).

Vapour diffusion hanging-drop crystallisation was used to produce crystals from polyethylene glycol. Diffraction data were collected on beamline MX2 at the Australian Synchrotron. Their space group is C121, with unit cell parameters a = 203 Å, b = 78 Å, c = 183 Å, α = 90°, β = 102°, γ = 90°. The Matthews coefficient of 2.68 Å³/Da, indicates 4 or 5 copies of Hsp70 per asymmetric unit in the crystal. Diffraction extended to 2.8 Å, with moderate intensity (overall I/σ of 7.2, I/σ of 2.0 at the highest resolution). Data were 99.7% complete and were collected with average multiplicity of 7.0. Merging multiple measurements of each reflection gave an R_merge of 0.20 overall (0.88 at the highest resolution).

The statistics indicate that larger crystals are required for a high resolution structure, but that the available crystals are sufficient to solve a low resolution model of the conformation of Hsp70 in complex with ATP.

The cardiac response to β-adrenergic stimulation is reduced in the obese rat heart: the role of adenosine monophosphate-activated protein kinase (AMPK). A Thaung, R Lamberts. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.

During surgery, patients with obesity have increased incidence of hypotension, requiring higher dosage of catecholamines (adrenaline). This may be responsible for their increased cardiovascular complications. However, the specific effects of catecholamines on blood pressure and its signaling pathway appear to differ in obesity. Adenosine monophosphate-activated protein kinase (AMPK), a key regulator of cardiac energy metabolism, contributes to the disturbed metabolic regulation in obesity. Therefore, the aim of this study was to determine the cardiac response to β-adrenergic stimulation in the obese rat heart and investigate the involvement of AMPK.

Isolated Langendorff-perfused hearts of sixteen-week-old male lean and *fa/fa* obese Zucker rats (n = 24) were used to determine cardiac function. Normalised percentage of left ventricular developed pressure was used to quantify the response to adrenergic stimulation. All the hearts were exposed to accumulating doses of β-agonist, isoproterenol (10⁻¹⁰ to 10⁻⁷ M), followed by random assignment to either AMPK antagonist, compound C (CC) (10 µM), or Krebs-Henseleit buffer for control group.

In the isolated hearts of lean and obese Zucker rats, no significant differences in basal cardiac characteristics were observed. However, the response to β-adrenergic stimulation was depressed in the obese compared with the lean group, indicated by increased EC₅₀ (half maximal effective concentration) values (lean vs. obese: -8.53 ± 0.04 vs. -8.38 ± 0.05 (mean ± SEM); \( P < 0.05 \), two-way ANOVA followed by a Bonferroni post-hoc test). AMPK inhibition significantly decreased sensitivity to adrenergic stimulation in lean and obese (lean vs. lean + CC: -8.53 ± 0.03 vs. -8.21 ±
0.03; obese vs. obese + CC: -8.34 ± 0.05 vs. -8.15 ± 0.06, both $P < 0.05$) diminishing
the difference in $\beta$-sensitivity between both groups.

AMPK is involved in the cardiac adrenergic stimulation and may be responsible for
the attenuated cardiac response to adrenergic stimulation in the obese heart.

**Human papillomavirus virus-like particles and their potential as gene delivery vectors in the skin. C Burn, J Leong, M Hibma. Virus Research Unit, Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Human papillomavirus (HPV) infection has been causally linked to the development of cervical cancer and, more recently, head and neck cancers. Virus-like particles (VLPs), morphologically and immunologically similar to the natural virion, have been developed to vaccinate against HPV infection of the skin. VLPs, made up of the L1 and L2 capsid proteins of HPV, self-assemble into virions that encapsidate plasmid DNA. The purpose of this study was to generate HPV VLPs that can be used as a delivery vector for DNA vaccines in the skin.

The production and gene delivery capability of HPV VLPs was tested using VLPs containing red fluorescent protein (RFP) or the model antigen, ovalbumin (OVA). 293TT cells were transfected with plasmids containing the genes for L1, L2 and RFP or OVA. The resultant VLPs were used to transduce 293TT cells in culture or carry out *in vivo* assays. OT-1 CD8$^+$ T cells that proliferate in response to the OVA peptide were used as a measure of the cytotoxic T cell response. OT-1 proliferation following the subcutaneous immunisation of mice with OVA peptide was compared with epidermal and subcutaneously administered VLPs, and an untreated control (n = 8 for all groups).

VLPs were successfully assembled in 293TT cells, as confirmed by transmission electron microscopy. The VLPs efficiently transduced 74% to 100% of cells in culture. Expression of the gene of interest was detected by fluorescence *in vitro* and by OT-1 proliferation in mouse models. Dermal abrasion prior to administration of VLPs into the epidermis was found to induce significantly greater proliferation of OT-1 cells than subcutaneous delivery of the VLPs (14% versus 10%, $P = 0.0458$, Student’s $t$-test).

The ability of HPV VLPs to deliver a gene of interest *in vivo* provides evidence for the potential usefulness of the vectors in the administration of DNA vaccines.

**Pulmonary hypertension is accentuated in heart failure – assessed using synchrotron radiation microangiography. M Beard, D Schwenke, E Gray, I Campillo. Department of Physiology, Otago School of Medical Sciences, University of Otago, Dunedin.**

Patients with chronic heart failure (CHF) often develop pulmonary hypertension (PH) due to the impact left ventricular failure has on the pulmonary circulation. Dysfunction of the pulmonary endothelium acts as a trigger in many etiologies of PH. To date, the role of the endothelium during the onset of CHF-induced PH is unclear.
This study assessed pulmonary endothelial function in a rat model of CHF and determined whether endothelial dysfunction is a leading contributor to the onset of PH.

Dahl-salt sensitive rats (D-Sen, n = 7, CHF-model) and Dahl-salt resistant rats (D-Resis, n = 7) were used. However, since both groups were subjected to a high salt diet for 6 weeks prior to experimentation, Sprague Dawley rats were the control (n = 7). Rats were anaesthetised (pentobarbital, 60 mg/kg) and utilising synchrotron radiation microangiography, we assessed changes in vascular responsiveness to i) acetylcholine (ACh, 3.0 µg/kg/min for 5 min), an endothelium-‘dependent’ vasodilator, ii) the nitric oxide (NO) donor sodium nitroprusside (SNP, 5.0 µg/kg/min for 5 min), an endothelium-‘independent’ vasodilator, iii) endothelin-1 (ET-1, 1 nmol/kg), a vasoconstrictor and iv) BQ-123 (1 mg/kg), an ET-1A receptor antagonist.

The dilatory response to ACh was impaired in D-Sen and, partly, in D-Resis rats (internal diameters (ID) increased 11.1 ± 1.7% and 13.8 ± 1.9%, respectively) compared with an 20.8 ± 2.2% increase in controls (two-sided t-test, P < 0.05). The vasodilatory responses to SNP, however, were similar for all groups (ID increased ~17%). The vasoconstrictor response to ET-1 was accentuated in all Dahl rats, although the vasodilatory response to BQ-123 was enhanced only in D-Sen rats compared with D-Resis and control rats (ID increased by 15.3 ± 4.3%, 4.9 ± 2.1% and 8.8 ± 1.5%, respectively, P < 0.05).

In conclusion, these results demonstrate that in CHF, endothelial dysfunction, which impairs NO release and enhances ET-1 sensitivity, plays a significant role in the secondary development of PH.
Cardiovascular safety of non-steroidal anti-inflammatory drugs (NSAIDs)

Conventional NSAIDs are often contraindicated because of dangerous gastrointestinal bleeding. Hence the development and promotion of the cyclo-oxygenase-2 selective inhibitors (COX-2) for the treatment of musculoskeletal pain. However, all of the COX-2 inhibitors studied in large placebo controlled trials have been found to confer an increased risk of serious cardiovascular disease.

The riposte from the COX-2 enthusiasts is that the conventional NSAIDs are also potentially cardiotoxic. This meta-analysis includes 31 trials with over 100,000 patients and more than 100,000 patient years of follow-up and attempts to clarify matters. It involves consideration of cardiovascular morbidity when naproxen, ibuprofen, diclofenac, celecoxib, etoricoxib, rofecoxib, and lumiracoxib have been studied in comparison with a placebo. You might think that that we now have the answer. However, the authors conclusion is that “evidence is lacking to suggest that any of the investigated drugs are safe in cardiovascular terms. However naproxen seemed least harmful.

Disappointing. Maybe all NSAIDs should be avoided in those at risk?

BMJ 2011;342:c7086.

Rapid assessment of chest pain in the emergency department

Central chest pain, suspicious of an acute coronary artery syndrome, is a common presentation at the emergency department. Identification of those who do not have a serious cardiac problem is important as they may not require admission or prolonged observation.

This international study, based in Christchurch, grapples with the feasibility of sorting out those with low risk by the use of a 2-hour diagnostic protocol. 3582 patients seen in 14 emergency departments in 9 countries were studied and followed for 30 days. The protocol included a probability score based on accepted risk factors, evaluation of the electrocardiograph and measurement of serum troponin, creatine kinase MG, and myoglobin. These factors, known as the accelerated diagnostic protocol (ADP) enabled 352 (9.8%) patients to be identified as low risk and suitable for early discharge. The follow-up revealed that 3 (0.9%) of this cohort subsequently had a major cardiac event within 30 days.

Sounds good, but requires a very efficient emergency department and a very speedy laboratory service.

Angiotensin receptor blockers and the risk of cancer

A recent meta-analysis of randomised trials suggests that use of angiotensin receptor blockers (ARBs) may be associated with a modestly increased risk of incident cancer, particularly lung cancer. This would be very unfortunate for the millions of patients whose diabetes, hypertension and heart failure is currently being treated with one of these drugs. This report from Denmark examines this suggestion; the data is derived from Danish registries of filled drug prescriptions of ARB users between 1998 and 2006. The cancer incidence in this cohort of over 100,000 ARB users for over 300,000 person years is reassuring. They report that ARB use was not significantly associated with an increased risk of cancer overall or lung cancer. I suppose that the cautious would prefer a longer follow-up.


Percutaneous repair of mitral regurgitation (MR)

Severe MR leads to left ventricular failure and congestive heart failure; it is often lethal. Surgical repair of the diseased valve is recommended in severe cases. This paper notes there is a non-surgical alternative that involves the percutaneous implantation of a clip that grasps and approximates the edges of the mitral leaflets at the origin of the regurgitant jet. The authors report on a randomised trial comparing percutaneous repair with conventional surgery in 279 patients with moderately severe MR. At 12 months, the rates of the primary end point for efficacy were 55% in the percutaneous-repair group and 73% in the surgery group (P=0.007). Subsequent surgical repairs was needed in 20% of the percutaneous cohort and 2% of the surgical group required a second operation. Major adverse events occurred in 15% of patients in the percutaneous-repair group and 48% of patients in the surgery group at 30 days. The overall outcome was that the percutaneous procedure obviated the need for open heart surgery in 80% of those who received it.


Are the elderly at increased risk of adverse drug reactions?

Adverse drug reactions (ADRs) are common and comprise a major source of morbidity in older adults. Multiple medications (polypharmacy) are a recognised risk factor but are there others? This paper considers whether 7 common geriatric conditions of daily living (dementia, incontinence, falls, difficulty ambulating, malnourishment, depression, and prolonged bed rest) can be incriminated. 808 elderly subjects aged 65 years or more were studied. They had a mean score of 2.9±1.2 geriatric conditions recognised at the outset of the study. Over the 12-month follow-up period, 497 ADRs occurred in 269 participants, including 187 ADRs considered preventable and 127 considered severe.

Rather surprisingly there were no associations shown between any of the conditions and an increased rate of ADRs. Very good, but the researchers observe that judicious prescribing is still recommended in the elderly.

University of Otago Faculty of Medicine
Freemasons Postgraduate Fellowships in Paediatrics and Child Health for 2012

The above Fellowships or Scholarships are open to University graduates who intend long term to pursue work in Paediatrics or Child Health within New Zealand. The Fellowships include full-time salary for one year with provision for a further year.

Applications close on **15 July 2011** with the Department Manager, Department of Women’s & Children’s Health, Dunedin School of Medicine, PO Box 913, Dunedin 9054, from whom further details may be obtained ([wch.admin@otago.ac.nz](mailto:wch.admin@otago.ac.nz))
Medical Benevolent Fund

NZMA Members, and families of deceased Members, may apply for aid when in situations of financial hardship or distress.

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161