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This Issue of the Journal

Anti-cyclic citrullinated antibodies: complementary to IgM rheumatoid factor in the early diagnosis of rheumatoid arthritis
K Solanki, M Spellerberg, P Chapman, P Moller, J O'Donnell

The detection of serum anti-citrullinated (CCP) antibodies is helpful in identifying patients with inflammatory joint symptoms who will develop rheumatoid arthritis. The antibody tends to appear earlier than the more commonly used rheumatoid factor. The identification of anti-CCP antibodies in selected patients will aid in the early diagnosis of rheumatoid arthritis and allow treatment to be commenced early.

The burden of rheumatic disorders in general practice: consultation rates for rheumatic disease and the relationship to age, ethnicity, and small-area deprivation
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Rheumatic disorders form a significant component of general practice workload, with 20.4% of people (consulting over a 12-month period) having one of 10 rheumatic disorders. Older people, men, those who lived in more deprived areas, and Europeans were more likely to consult. Maori with gout were more likely to see their GP, but less likely to do so with back pain or regional pain disorders. New Zealand health policy should be revised to reflect the burden of these diseases upon the New Zealand population.

Low back pain in young New Zealanders
D McBride, D Begg, P Herbison, K Buckingham

Fifty-four percent of individuals in a birth cohort (now aged 26 years) experienced low back pain (LBP) in the previous 12 months. The frequency, duration, and severity of LBP varied—but 13 individuals could not look after themselves because of pain, and 56 had to have time off work. The cost to the economy in terms of time off work is estimated at $NZ500 million per year. The natural history of the disorder is largely unknown, but workplace factors are important and need to be addressed.

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A Doyle, S Hawkins, J Tran

Vertebroplasty is a way of stabilising a fractured vertebra by putting a needle into the vertebral body and injecting cement. The aims are to relieve pain from the fracture and to prevent further fracture. Performed properly, vertebroplasty is safe and effective in most patients. The procedure has been used in the Northern Hemisphere

for several years but not very much in New Zealand. We are reporting our early experience to encourage use of, and research into, this procedure locally.

Ruptured abdominal aortic aneurysms: risk factors for mortality after emergency repair

A Lo, D Adams

Despite recent advances in anaesthetic and operative care, emergency repair for ruptured abdominal aortic aneurysm (AAA) is still associated with a high mortality rate. This has prompted many surgeons to question whether repairing ruptured abdominal aortic aneurysms should even be attempted in the subsets of patients with poor physiological reserve. Within the confinements of a retrospective study, simple preoperative variables with predictive values have been identified. These variables may be used to complement the surgeon's own patient selection criteria for emergency repair.

The role of ERCP in management of retained bile duct stones after laparoscopic cholecystectomy

S Anwar, R Rahim, A Agwunobi, J Bancewicz

Laparoscopic cholecystectomy has become the 'gold standard' for removal of diseased gall bladders. This procedure, however, carries a risk of residual common bile duct stones. These stones can become symptomatic and subsequently cause morbidity. In our series, the risk of residual stones is about 2.5%. The endoscopic retrograde cholangiopancreatography (ERCP) procedure was used to detect and treat these stones with very good results and minimal morbidity.

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Rheumatologic diagnostic serology: tests which test clinicians

Min Loke Wong

The performances of diagnostic tests are typically reported as sensitivities and specificities, which refer to their accuracy in groups of patients with known presence and absence of the diagnosis in question, respectively. However, in clinical practice, such tests are applied to patients whose diagnostic status are uncertain, and predictive values provide more useful information.

Positive (and negative) predictive values refer to the proportion or probability of patients with positive (and negative) results, having (and not having) the diagnosis. A highly specific test will have few false positives, and generally have a high positive predictive value (PPV). Likewise, a highly sensitive test with few false negatives, tends to have a high negative predictive value (NPV): thus a negative test result for antinuclear antibodies (ANA) is useful for excluding systemic lupus erythematosus (SLE).

In this issue of the *Journal*, Solanki and colleagues report on the performance of a new serological test for rheumatoid arthritis (RA): antibodies to cyclic citrullinated peptide.[1] The patients tested were known to all have one of a range of chronic inflammatory rheumatic diseases. In any such highly selected group there will usually be many with RA, for which the test is highly specific, and hence the PPV will be high. In less highly selected patient groups with a lower prevalence of RA, there will be a lesser number of true positives (when the same test is applied), which may be further diluted by an increased number of false positives—resulting in a reduced PPV.

Thus, PPVs generally decrease with decreasing prevalence of the diagnosis in the population group being tested. Rheumatoid factors (RF) and ANA have low PPVs for RA and SLE when applied in low prevalence settings (such as patients with common symptoms like arthralgia) without features more highly suggestive of each diagnosis. In low prevalence settings, a clinician may order a test in the hope of obtaining a negative result for reassurance.

The difficulty arises when the result is positive, which may be expected at a frequency approximating the false positive rate (1–specificity), which may be about 10% or more, for commonly requested tests such as RF and ANA. The clinician, if lacking the confidence to interpret the result as a false positive, may then unwittingly set off a cascade of further tests, referrals, and treatments—with the potential to cause alarm and harm to the patient. Outside of rheumatology, these same potential traps are to be found; for example, in screening for prostatic carcinoma with tests for prostate specific antigens.

Translated to the level of an individual patient, prevalence of a diagnosis becomes pre-test probability—as shown earlier, this influences predictive values, which become post-test probabilities. Post-test probability can be calculated from pre-test probability, sensitivity, and specificity using Bayes' theorem, more conveniently

presented in the form of a nomogram using likelihood ratios (LRs) for converting one probability to the other.[2,3]

LRs are calculated from the formulae sensitivity/(1–specificity), and (1–sensitivity)/specificity for positive and negative results, respectively. LRs <0.1 and >10 markedly modify pre-test probabilities, especially those in the intermediate range.[4]

In general, whether treatment would be offered in the first place as well as the level of post-test probability at which one would start therapy (or, at the other end, be confident about excluding a diagnosis) depend on factors such as disease severity and natural history, and risks and efficacy of treatment. Additional testing may be required to attain the desired level of probability.

Clinicians can estimate pre-test probabilities in their patients, from information on prevalence of the diagnosis being considered, in groups of patients with comparable demographics and clinical findings comparable to their own patients.[3,4] Thus, clinical findings act as test results and are subject to the same considerations. However, there is limited published information on predictive values of clinical findings[3,5]—and in relying on 'clinical judgement', pre-test probabilities tend to be overestimated.[6]

Further challenges arise—in that the tests available locally to the clinician may not perform the same as those reported. Indeed, performances may differ due to variations in test systems, kit manufacture, laboratory techniques, and the way cutoff values for positivity are derived (including the characteristics of any control group used for the purpose). Wise clinicians, therefore, take into account their experience of the performances of tests used locally.

Thus, diagnostic tests that reportedly perform well do not replace the need for a careful clinical assessment of the patient, selective application of tests (the greater the number of tests performed, the greater the chance of finding a false positive), and thoughtful interpretation of the results.

'Diagnostic' tests are indeed testing of clinicians.

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The end of the one-eyed surgeon? Time for more randomised controlled trials of surgical procedures

Angus Watson, Frank Frizelle

In a recent editorial in the *British Journal of Anaesthesia* entitled *In the Country of the Blind, the One-Eyed Man is King*,[1] the author points out that when there is a lack of data from randomised controlled trials (RCTs), doctors can only use the best currently available information to determine the correct patient management.

Indeed, trials of surgical methods have, until recently, been uncommon. Surgical decision-making has been modelled on the experience gained from case series and the authoritative statement. For too long, surgeons have relied on non-RCT data to determine the management of patients. Recently, as more data is becoming available, it is becoming increasing clear that some surgical procedures are at best of little use, and at worst potentially harmful.

A search of Medline reveals the growth in surgical RCTs. In the 10 years 1974 to 1984, there were 3415 RCT publications; from 1984 to 1994, there were 12,944 RCT publications; and in the 10 years since, there have been 27,251 RCT publications. Most of these trials, however, are not of surgical method—but instead relate to pain relief, anaesthetic variations, antibiotics, perioperative adjunctive therapy for cancers, and medical treatments used in association with surgical management. Indeed, there are few RCTs looking at the appropriateness and outcome of various surgical procedures.

Recently, however, there has been a sudden flurry of RCTs on surgical method. The results of which have sufficient influence to impact on how surgery is practised.

Some of the more recent trials include:

- A RCT of laparoscopic adhesiolysis published in 2003 in *The Lancet*.[2] 100 patients were randomly allocated to either laparoscopic division of adhesions or diagnostic laparoscopy. Both groups reported substantial pain relief and a significant quality of life (QOL) improvement early after the operation. At 12 months, there was no difference in pain or QOL in either group.
- A RCT of arthroscopic knee surgery for osteoarthritis published in the *New England Journal of Medicine*.[3] In this study, 165 patients with osteoarthritis of the knee were randomised to either arthroscopic debridement and lavage or placebo surgery. Outcomes were assessed at 24 months. The outcomes in regard to knee pain and function for those patients who had surgery were no better than those patients who had the placebo procedure.
- A RCT of laparoscopic colon cancer surgery.[4] The results from several trials are now available with the results of other trials pending. The COST trial from North America produced mature data showing no early or long-term benefit from laparoscopic resection. Other trials have produced similar results. In summary, the trials to date show no oncological disadvantage from having the operation

performed laparoscopically. There is probably an improved recovery time, a lower wound infection rate, and an early improvement in QOL. The results of further trials, including the Australia and New Zealand trial (ALCAS), are pending.

- The use of carotid endarterectomy in patients without neurological symptoms.[5] The use of carotid endarterectomy in patients following neurological events is now well established following a number of RCTs. However, the correct management of a patient with a recognised carotid stenosis without a prior neurological event is uncertain. This RCT involved 3120 patients who were randomised to carotid endarterectomy or no treatment. The stroke rate at 5 years was 6.4% vs 11.8% (net gain 5.4%)—however the authors point out that there is a variable stroke rate associated with the surgical procedure, and if this is increased by poor patient selection or poor surgery then the benefit could easily disappear. One always has to wonder that if a trial needs this number of patients to show a benefit, just how good the benefit really is, and what the effect of an aspirin might be?
- A RCT of laparoscopic inguinal hernia repair.[6] This trial looked at 2164 patients of whom 1983 had a hernia repair; patients were randomised to either an open mesh or laparoscopic mesh repair of inguinal hernia. Recurrences were more common in the laparoscopic group (10.1%) compared with the open group (3.2%); and the rate of complications was higher in the laparoscopic group (39.0% vs 33.4%). The laparoscopic group did, on average, return to work one day earlier.
- A RCT of the effectiveness of adenotonsillectomy in children with mild symptoms of throat infections or adenotonsillar hypertrophy.[7] In this study, 300 children aged 2–8 years were randomised to adenotonillectomy or watchful waiting. Episodes of fever, throat infections, upper respiratory tract infections, and health QOL were assessed. After a median follow-up of 22 months, children in the adenotonsillectomy group had 2.97 episodes of fever per person compared with 3.18 in the watchful waiting group; 0.56 throat infections per person year compared with 0.77; and 5.47 upper respiratory tract infections per person year compared with 6.00. No difference was found for health-related QOL.

Surgical trials, however, are necessarily different. Interestingly, most trials fail to show that the new treatment is better than the old treatment, and it is often no better than doing nothing. Trials of surgical procedures are well over due. Surgeons as a group have embraced evidenced-based medicine, and around Australasia, various surgical groups have sprung up to undertake RCTs or analyse them—such as Surgical Outcomes Research Centre (SOURCE) and Australian Safety and Efficacy Register of New Interventional Procedures – Surgical Royal Australian College of Surgeons (ASERNIP-S).

The results of RCTs could be of enormous benefit in directing patient management more effectively, and saving patients from the morbidity and mortality of unnecessary procedures.

The problems with surgical trials are:

- **Funding.** Many medical drug or device trials are readily funded by industry. Drug trials are regarded as being sexy by both industry and medical workers, many of whom dream of effecting a significant change in the management of cancer or

cardiovascular disease. Trials of surgical method are not considered to be sexy, and are often not funded by industry, however they can have a significant effect on healthcare expenditure and patient care. It is imperative that grant-giving bodies take the funding of research on surgical procedures seriously.

- **Recruitment of patients.** Surgeons perceive that when they are advising patients on their surgical management they should have the freedom to choose which surgical treatment they believe to be the most appropriate. Furthermore, surgeons hold very definite opinions, possibly engendered by the nature of their work or perhaps due to their selection and training. Patients, likewise, are perceived as unlikely to consent to procedures, especially if there is no treatment arm such as in the laparoscopic division of adhesions or the knee surgery trial. Operations have major and significant complications, and who wants an operation with its associated risk when the trial design suggests to the patient that they might not need surgery in the first place?
- **The influence of private practise on surgical recruitment.** Private surgical practice is as large in New Zealand as it is overseas. The nature of private surgical practice funding in New Zealand means that it is difficult to recruit patients into RCTs from private practise.
- **The attitude of surgeons towards RCTs.** Surgeons often put more emphasis on case reports and case series that RCTs. Whether this reflects a lack of research training or some other factor in the surgical mentality, it is difficult to fathom.
- **The lack of surgical academics.** Surgical academics need to lead. There is a worldwide shortage of surgical academics due to these multiple and intermixed reasons:
    - The income variation between private practice and academic life is cavernous,
    - It is difficult to attract research funding,
    - Academics are frustrated by university administration,
    - Making a research impact is problematic, and
    - The lack of posts means that many budding surgical academics last 2–3 years before moving on to a part-time hospital and private practice job, thus giving up any hope of future surgical research.

    (The *British Medical Journal* has recently published a theme edition on the problems of academic medicine, much of which is true to academic surgery.)

In summary, well-designed trials are urgently needed to improve patient care and to provide evidence on whether money spent on surgery is beneficial. In addition, the important issues listed above need to be addressed, especially with regards to the attitudes of funders to the funding of trials of surgical methods.

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Anti-cyclic citrullinated antibodies: complementary to IgM rheumatoid factor in the early diagnosis of rheumatoid arthritis

Kamal Solanki, Myfanwy Spellerberg, Peter Chapman, Peter Moller, John O'Donnell

Abstract

Aims To compare the diagnostic sensitivity of anti-cyclic citrullinated (CCP) antibodies and rheumatoid factor (RF) in rheumatoid arthritis (RA) within a general hospital setting.

Method Using the American College of Rheumatology (ACR) classification criteria as a gold standard, the frequency of RF and anti-CCP antibody positivity was compared between two groups of RA patients: those with disease duration less than 2 years (early RA, ERA) and those with disease duration more than 2 years (late RA, LRA).

Results In ERA, the diagnostic sensitivity of RF and anti-CCP antibodies was 57% and 79% respectively. In LRA, it was 81% and 84% respectively.

Conclusion Anti-CCP antibodies are 20% more sensitivity than RF in the diagnosis of early RA.

Rheumatoid arthritis affects 1% of the population and is one of the most common serious inflammatory arithritides.[1] It is characterised by chronic inflammation of the synovial membrane of diarthrodial joints. This inflammation results in joint damage leading to morbidity and premature mortality .[2,3] In many persons, erosion and damage occur within the first 2 years of disease onset.[2] Suppressing the inflammatory response early in the course of disease is a major goal of therapy, hence early diagnosis is important.

The American College of Rheumatology (ACR) classification criteria (1987) emphasise features of chronicity and, as such, have limited use in early diagnosis.[4] However they are still used as a 'gold standard' for research purposes.

IgM rheumatoid factor (RF), which is one of the ACR criterion, is the best known serological marker—however it has a relatively low diagnostic sensitivity (50 to 90%) particularly in early disease and relatively low specificity (87%).[5,6] Nevertheless in the appropriate clinical context, rheumatoid positivity is an important finding giving support to the diagnosis of RA.

Newer serological markers have included antibodies to cyclic citrullinated peptide (CCP), Sa protein, heavy chain binding protein (Bi P), and glucose-6-phosphatase isomerase.[2] Of these, anti-CCP antibodies seem promising as a diagnostic[5,6] and possible prognostic marker in RA.[7–10]

Citrullination (conversion of arginine residues to citrulline by the enzyme peptidyl arginine deaminase) of filagrin induces an autoantigen that was recognised previously by anti-keratin auto-antibodies. Profilagrin, present in the keratohyaline granules of

human keratinocytes, is proteolytically cleaved to filagrin subunits, which form the target autoantigen. Unfortunately these subunits are not stable enough to be used as a commercial substrate in diagnostic tests.

In their study, Schellekens and colleagues reported that 76% of rheumatoid arthritis sera contained autoantibodies reactive with linear synthetic peptides containing citrulline. These antibodies were 96% specific for rheumatoid arthritis[5,6]

An ELISA based on cyclic-citrullinated peptide (CCP), a more stable compound,[6] was shown to efficiently measure these antibodies in the sera of patients with rheumatoid arthritis.

Using ACR criteria as a 'gold standard', we assessed the diagnostic sensitivity of anti-CCP antibodies in early and late rheumatoid arthritis within a general hospital setting.

Patients and methods

Christchurch Hospital is both a secondary and tertiary referral centre in the South Island of New Zealand. It has an immediate catchment population of approximately 500,000. Canterbury Health Laboratories provides laboratory support to the hospital. The immunology section of this laboratory routinely stores sera for a minimum of 5 years. These stored samples allowed for the retrospective analysis of anti-CCP antibodies in some patients prior to and following the diagnosis of RA.

Patients referred by their general practitioner to the rheumatology clinic at Christchurch Hospital and subsequently diagnosed with RA formed the study subjects. All patients were given a diagnosis of RA once their caring physician considered that they fulfilled the ACR classification (1987) criteria for RA. **Patients were divided into two groups as follows:**

- **Group 1:** patients diagnosed with RA of less than 2 years duration who had stored serum available from the time of the initial diagnosis. These patients formed the early rheumatoid arthritis group (ERA)
- **Group 2:** patients with RA who had been diagnosed ≥2 years previously who had a contemporary serum sample available. These patients formed the established rheumatoid arthritis group (LRA)

RF was measured by rate nephelometry (Beckman array) with a cut-off at 40 IU/L. Anti-CCP antibodies were measured by the Quantalite CCP ELISA assay (INOVA Diagnostics, San Diego, CA, USA) with a cut-off at 20 units as specified by the manufacturer. All sera were tested in duplicate in the ELISA and a 5-point standard curve was derived for each microtitre plate.

Statistical Analysis

The McNemars Chi-square test was used for statistical comparison between diagnostic antibody tests within each group.

Results

Table 1 summarises the results of both rheumatoid factor and anti CCP antibodies in the two patient groups.

In group 1, the diagnostic sensitivity of RF and anti CCP antibodies was 57% and 79% respectively (p=0.009) while in group 2 it was 81% and 84% respectively (p=1.0).

Six patients within group 1 had more than one serum sample stored from the time of initial presentation. Of these, two patients were negative for both anti-CCP antibodies and RF at initial presentation. The other four patients were anti-CCP positive and RF negative at initial presentation.

Analysis of stored serum samples revealed that the two double negative patients seroconverted for anti-CCP between 6 and 48 months and seroconverted for RF

between 15 and over 48 months. In the four patients who were anti-CCP antibody-positive and RF-negative at initial presentation, RF seropositivity developed in three patients between 16 and 48 months and the fourth patient remained RF negative.

Table 1 Diagnostic sensitivity of rheumatoid factor and anti-cyclic citrullinated (CCP) antibodies in early and late rheumatoid arthritis

		GROUP 1 (ERA) n=66 (%)	GROUP 2 (LRA) n=32 (%)
RF	**+ve**	**38 (57)**	**26 (81)**
	-ve	**28 (43)**	**6 (19)**
Anti-CCP antibodies	**+ve**	**52 (79)**	**27 (84)**
	-ve	**14 (21)**	**5 (16)**

RF=rheumatoid factor; ERA=early rheumatoid arthritis; LRA=late rheumatoid arthritis.

Discussion

General practitioners and specialist physicians are often faced with evaluating patients with polyarthralgia and polymyalgia.

Typically, such patients eventually fall into four categories.

- Patients destined to suffer a self-limiting disorder (most often idiopathic, but occasionally associated with an infection).
- Patients suffering from a chronic diffuse pain disorder (such as fibromyalgia).
- Patients destined to develop degenerative joint disease.
- Patients ultimately destined to develop some form of chronic inflammatory joint disease.

Patients destined to develop rheumatoid arthritis form a large sub-group of the last category. The importance of identifying these RA patients early has been increasingly emphasised as strategies are developed to reduce disease morbidity.[2,11]

Early intervention with disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate, salazopyrin, and more recently anti-tumour necrosis factor α (anti-TNFα) therapy has been associated with more favourable long–term outcomes.[9,11]

Anti-CCP antibodies have a high positive predictive value for RA in patients with polyarthralgia, particularly if used in conjunction with clinical findings and rheumatoid factor.[12] Thus there is the potential to identify patients for therapy at an early stage. In addition, anti-CCP antibodies also have prognostic value in identifying those patients at greater risk of erosive disease.[6,7,13]

The results of our study support previous findings from reference and research centres that anti-CCP antibodies tend to appear earlier and are therefore of added value in the in early diagnosis of rheumatoid arthritis. In our study, diagnostic sensitivity was increased by 20% over IgM rheumatoid factor in the early RA group.

Prior to commissioning the anti-CCP ELISA in our laboratory, the specificity of the assay was evaluated using stored serum samples from 54 patients with non-RA rheumatic diseases: SLE (n=23); Wegener's granulomatosis (n=9); psoriatic arthritis (n=3); mixed connective tissue disease (n=2); and one patient each with giant cell arteritis, CREST syndrome, ankylosing spondylitis, and juvenile chronic arthritis.

Three of the 54 were positive giving a specificity of 94%, a result comparable with published data.[4] The three 'false positive' sera where from patients with SLE and all had low titre concentrations of anti-CCP antibodies.

In recognition of the benefits of early intervention in RA, there have been calls to establish new prognostic criteria.[2] Despite their demonstrated diagnostic and prognostic value[9] it has been suggested that anti-CCP antibodies should be excluded from such criteria on the pretext that the test is not widely available.[2]

Cost is the primary determinant of availability. In New Zealand, the price of tests varies from laboratory to laboratory. Rheumatoid factor measured by nephelometry is about $NZ15.00/test (exclusive of taxes) whereas currently the price of anti-CCP antibodies is about $NZ60.00/test. The cost of assay kits and the cost of labour are the main determinants of price. More general use, automation, and competition will see prices fall.

Anti-CCP antibodies have all the hallmarks of establishing themselves firmly in the diagnostic algorithm of rheumatoid arthritis providing additive sensitivity to rheumatoid factor.

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The burden of rheumatic disorders in general practice: consultation rates for rheumatic disease and the relationship to age, ethnicity, and small-area deprivation

William Taylor, Louis Smeets, Jason Hall, Kathryn McPherson

Abstract

Aims To estimate the burden of rheumatic disorders in the New Zealand population by calculating general-practice consultation rates—as well as the influence of age, gender, ethnicity, and small-area deprivation on these consultation rates.

Methods Cases were identified from the Royal New Zealand College of General Practitioners database using search-strings of typical words used in consultation notes (for each of 10 rheumatic disorders). Rates were calculated from a denominator of the number of people attending at least once over a 12-month period to any of 22 contributing general practices. The independent effect of age, sex, ethnicity, and small-area deprivation was modelled using multiple logistic regression.

Results Of 29,152 people attending their general practitioner, 20.4% consulted with a rheumatic disorder. Older people, males, people who lived in more deprived areas, and Europeans were more likely to consult with a rheumatic disorder. With all rheumatic disorders, age was a significant influence upon consultation—especially osteoarthritis, gout, osteoporosis, and joint surgery. Maori were more likely than Europeans to consult with gout, but they were slightly less likely to consult with back pain or regional pain disorders. Small-area deprivation had small influences upon people consulting with gout, regional pain, and back pain.

Conclusions Rheumatic disorders form a significant part of the workload of general practitioners and this is significantly influenced by local demographic factors. Most of these conditions seen in general practice are non-inflammatory and non-surgical. If a community-needs approach was taken, it is likely that the workload and associated costs would be even greater.

The decade 2000–10 is the ‘bone and joint decade’ with calls for increased recognition of the burden posed by musculoskeletal conditions.[1] Musculoskeletal disorders are common and disabling but are low priorities for public health resources[2] and medical education curricula.[3,4]

Osteoarthritis (OA) alone is the 4th leading cause of years lost to disability (YLD) in Australia (after depression, dementia and asthma).[5] In New Zealand, OA ranks as the 6th leading cause of YLD (after depression, anxiety, asthma, diabetes and chronic obstructive airways disease).[6] However, there is very little detailed knowledge about the epidemiology of musculoskeletal disorders in New Zealand, apart from gout.[7,8] The data on which to base measurement of YLD come largely from overseas, and may not necessarily be relevant for the New Zealand community.

The only peer-reviewed publication about a community survey of rheumatic diseases occurred in the 1960s[9]—and the 2002/03 New Zealand Health Survey looked at only

self-reported arthritis, back problems, or osteoporosis as specific musculoskeletal health conditions. That survey showed that 30% of people aged over 45 years report arthritis and 32.7% report back problems.[10]

A population-based survey of rheumatic diseases in Dunedin was carried out as a BMedSc thesis in 1984; it showed a very high prevalence of any prior peripheral-joint problem (76.5%).[11] A very high overall point prevalence of clinically defined osteoarthritis (71.2%) was also observed, but much of this may have been painless knee crepitus and not OA as is currently defined (symptoms plus radiographic changes). Rheumatoid arthritis (RA) was observed in 2.9% (modified New York criteria) or 2.0% (modified Rome criteria). The largest survey of general practitioner consultations in New Zealand came from the 1991/1992 Waikato Medical Care Survey.[12] Given that this is over a decade old, and provided relatively superficial coverage of specific morbidity, it seemed appropriate to investigate consultation rates for rheumatic disease in more detail.

As a first step to more accurately estimating the burden of musculoskeletal disorders in New Zealand, we obtained estimates of consultation rates with general practitioners for a range of rheumatic disorders using the Royal New Zealand College of General practitioners (RNZGP) database. In addition, we were able to assess the influence of sex, ethnicity, and geosocial deprivation using multivariate logistic regression models.

Methods

General practice data are extracted onto floppy disk from contributing member practices of the RNZCGP Computer Network, and sent by courier to the Research Unit, Department of General Practice, Dunedin School of Medicine. No information that could identify a patient is collected. The information is indexed by encrypted National Health Indices (NHI). The sole holder of the decryption algorithm is the New Zealand Health Information Service (NZHIS). Data that are consistently available for all practices include: textual information for each general practice encounter, demographic details, and pharmaceuticals prescribed.

Twenty-two general practices were selected for the study on the basis of having fully computerised their clinical records, and these practices were assigned valid NHIs to their patient register. The denominator was limited to those patients who had consulted at least once, for any reason, with one of the study practices over a 1-year study period. This represented 29,152 people out of a notional catchment population of 40,599 who had consulted at least once over a three-year period (1 July 1996 to 30 June 1999).

A file of encrypted NHI numbers of these people was sent to the NZHIS. New Zealand Deprivation Index 1996 (NZDep96) and ethnicity along with secondary care data was attached to each number. This individualised but non-identifiable data was returned to the Research Unit, engineered into a standardised format, and stored on a secure server.

People for whom ethnicity or NZDep96 was not available were excluded from the study. Age and gender were available from the general practice records. Ethnicity is sourced from the National Minimum Data Set and uses the Statistics New Zealand official definition as modified by the National Data Policy group, which states that ethnic group should be self-identified wherever possible.

NZDep96 is an updated version of the NZDep91 index—a small-area deprivation scale developed by the Health Services Research Centre.[13,14]. It combines nine variables from the 1996 census, which reflect eight dimensions of deprivation. The nine variables are: proportion of persons without access to a car, without access to a telephone, receiving a means-tested benefit, with a low income, living in a single parent family, without any qualifications, not living in own home, and living below a threshold of bedroom occupancy.

The index is designed to reflect the level of deprivation experienced by people living in a small area. A score of ‘1’ would mean that an individual is living in a small area that is in the 10% least deprived of small areas in New Zealand, a score of ‘10’ is the most deprived.

The GP population closely matches the New Zealand population in terms of age. The median age of the population was 34 years, identical to that of the 1996 New Zealand census. The median age for Maori was 23 years compared to the census median of 22 years. Overall, the percentage of people in each NZDep96 category is a good match for the expected 10% of the population in each ranking. There is slight under representation of 'NZDep96–category 1' in the study population and a slight over-representation of 'NZDep96–category 10'. The data set is not representative of the geographic distribution of New Zealand's population as only 7.6% of patients came from the Central region (34.0% Midland, 21.4% Northern, and 37.1% Southern). This compares to the New Zealand population (1996 Census) of 24.7% Central, 20.0% Midland, 33.7% Northern, and 21.6% Southern.

Seventy-two percent of the study population had general practice encounters (for any reason) during the year, compared to the figure of 79% self-reported consultations in the 1996/97 New Zealand Health Survey (NZHS). Women were 9.5% (NZHS 10.9%) more likely than men to have had at least one general-practitioner consultation during the year. Women were also 5.7% (NZHS 6.5%) more likely than men to have made six or more visits to a general practitioner. The percentage of costs of prescriptions written for the study group is similar to the percentage costs for prescriptions dispensed for the New Zealand population for the top five therapeutic groups.

In existing practice management systems, the diagnosis is often trapped inside the textual consultation notes. Analysis of these data is extremely time-consuming, as information processing requires searching for text strings and manual-checking for accuracy. Read codes were not available consistently for this study. Therefore, we devised search strategies of consultation text for each of 10 musculoskeletal conditions, based on typical words used in recording encounters with these disorders.

To judge the approximate positive predictive value of the search strategy method, we manually read consultation text from 100 randomly selected patient consultations identified as having gout or osteoarthritis.

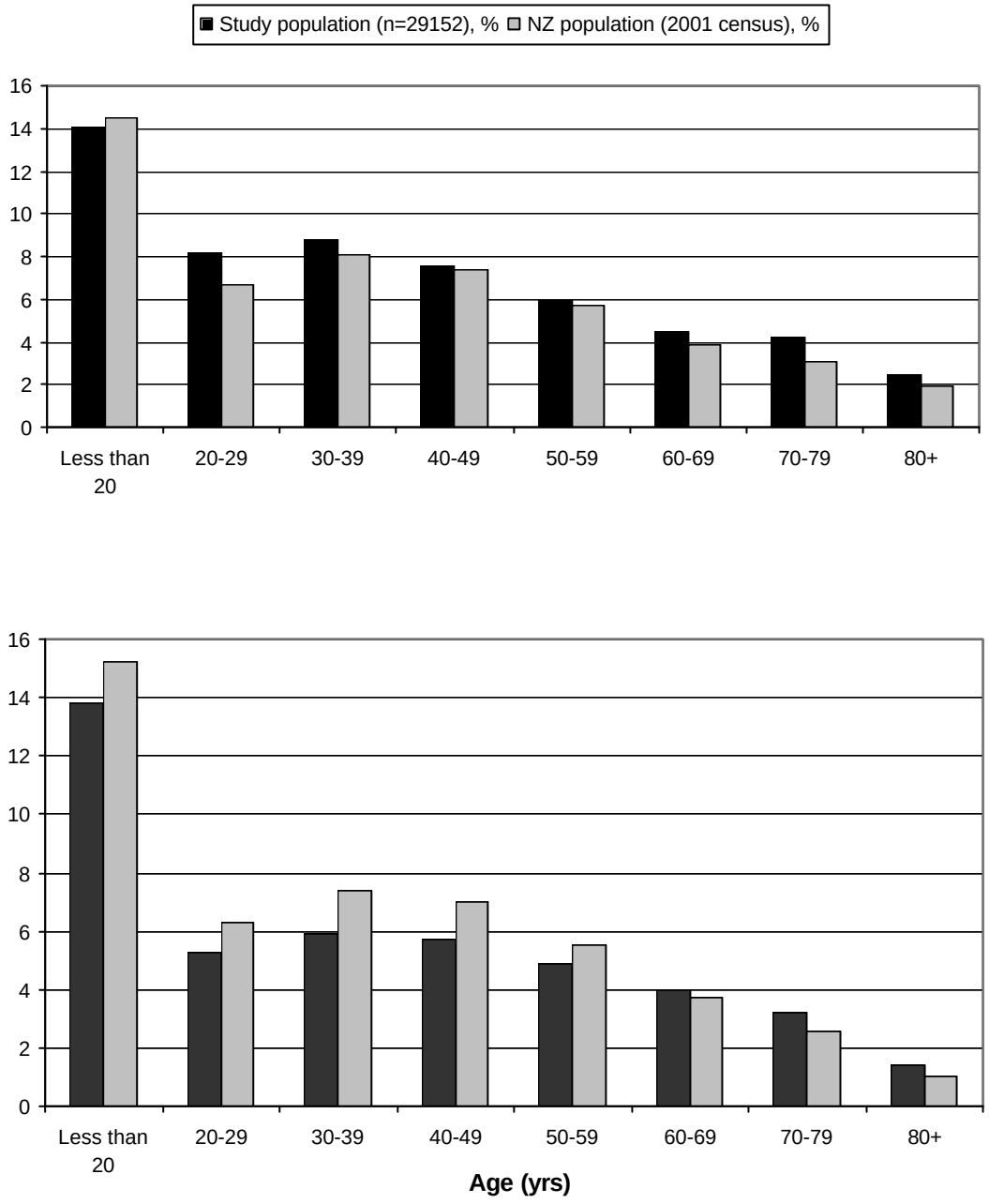
Results

The demographic characteristics of the population at risk (those people who had consulted with one of the participating practices at least once during the year) are shown in Figure 1 and Table 1—with comparisons to the NZ population (data from the Census 2001). The sample was older (Chi-squared=347, df=7, $p<0.001$), more likely to be female (Chi-squared=246, df=1, $p<0.001$), under-represented by Pacific peoples, and over-represented by 'other' ethnicity (Chi-squared=25,960, df=3, $p<0.0001$). This last observation is highly likely to be due (in part) to 'unknown' ethnicity being coded as 'other', and is a similar finding to other general practice data.[15]

A visual inspection of textual data by a consultant rheumatologist for 100 randomly selected consultations identified as 'osteoarthritis' and/or 'gout' by the text-word search strategy suggested that 80% of the identified consultations with gout were likely to be correctly diagnosed and 93% of the identified consultations with osteoarthritis were likely to be correctly diagnosed. An example of consultation text for a person assessed as likely to have gout is: *'Had an attack of acute gout over holidays. UA was high. Not sore now. stop frusemie. BP 140/82 See in a month with a further UA'*.

Figure 1. Age and sex structure of the study population compared to the general New Zealand (NZ) population

(The vertical scale represents percentages of the study populations; upper graph=female; lower graph=male)



Overall, 5940 people (20.4% of the study population) consulted with a musculoskeletal disorder over a 12-month period. The rates for the 10 specific disorders were: rheumatoid arthritis 0.79%, osteoarthritis 1.44%, gout 1.38%, regional pain 13.8%, back pain 7.18%, spondyloarthritis 0.14%, connective tissue disorder 0.66%, nerve compression syndrome 0.79%, joint surgery 0.45%, and osteoporosis 0.34% (Table 2).

Table 1. Sex and ethnicity profile of the study sample in comparison with the general New Zealand (NZ) population

Ethnicity	Sex	Number of people who consulted at least once over 12 months (%)	Percentage of the NZ population (Census 2001)	Number (percentage) who consulted their GP at least once over 12 months. N=6213 (1996/1997 Health Survey)
European/Pakeha	F	9682 (33.2)	41.1	4580 (73.7)
	M	6966 (23.9)	38.9	
Maori	F	1879 (6.4)	7.5	1000 (16.1)
	M	1456 (5.0)	7.2	
Other	F	4592 (15.8)	3.8	495 (2.2)
	M	4305 (14.8)	3.5	
Pacific people	F	146 (0.56)	3.3	138 (8.0)
	M	126 (0.43)	3.2	

Table 2. Identified cases consulting with a general practitioner at least once over 12 months

	Total number of cases	Consultation rate (95%CI) as percentage of the study population n=29152	Adjusted OR (95% CI) for Maori ethnicity	Adjusted OR (95% CI) for male gender
Rheumatoid arthritis	230	0.79 (0.69-0.89)	0.75 (0.43-1.30)	0.63 (0.47-0.83)*
Osteoarthritis	422	1.44 (1.31-1.58)	0.94 (0.62-1.42)	0.94 (0.77-1.15)
Gout	403	1.38 (1.25-1.51)	3.54 (2.84-4.42)*	3.54 (2.84-4.42)*
Regional pain	4014	13.8 (13.4-14.2)	0.79 (0.69-0.89)*	1.05 (0.98-1.13)
Back pain	2094	7.18 (7.48-6.89)	0.84 (0.72-0.98)**	1.07 (0.98-1.17)
Spondyloarthritis	43	0.14 (0.10-0.19)	0.65 (0.18-2.29)	1.27 (0.70-2.33)
Connective tissue disorder	192	0.66 (0.57-0.75)	0.84 (0.48-1.48)	0.61 (0.45-0.83)*
Nerve compression syndrome	230	0.79 (0.69-0.89)	0.63 (0.36-1.08)	0.81 (0.62-1.06)
Joint surgery	131	0.45 (0.37-0.53)	0.91 (0.45-1.84)	0.99 (0.70-1.41)
Osteoporosis	98	0.34 (0.40-0.27)	0.17 (0.02-1.28)	0.13 (0.07-0.26)*

*p<0.001,**p<0.05; OR=odds ratio; CI=confidence interval.

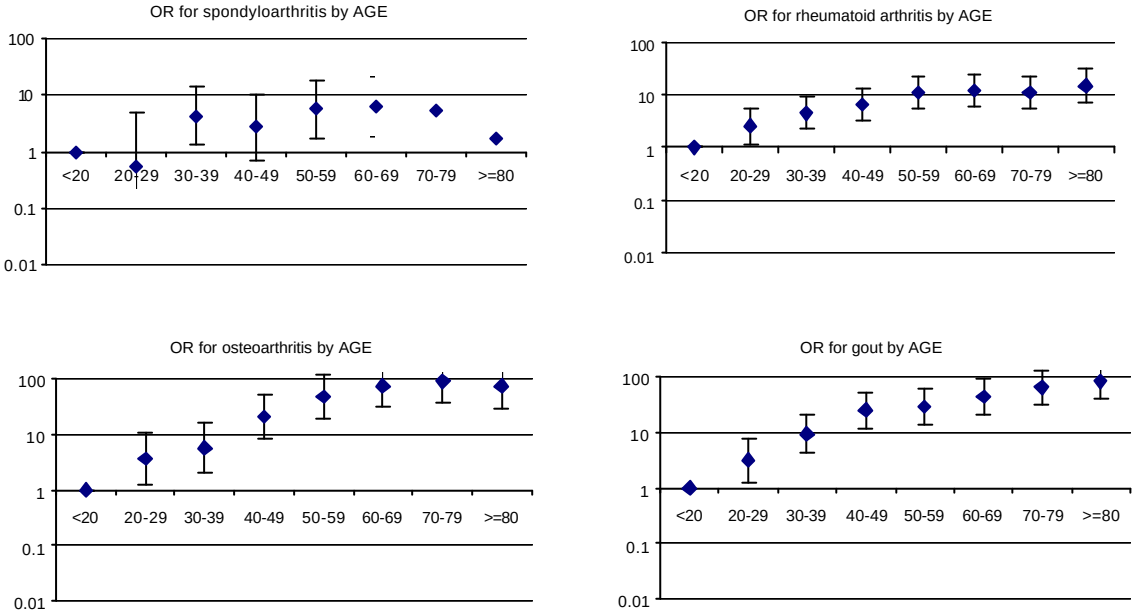
Regional pain disorders included soft tissue syndromes, fibromyalgia, and minor musculoskeletal injury and clearly account for the commonest musculoskeletal reason for consultation. Older people and those from more deprived NZDep96 categories were more likely to consult with any musculoskeletal disorder, whereas Maori and women were less likely to consult (Table 3).

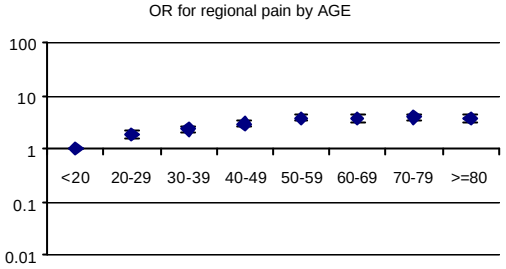
Table 3. Logistic regression model for the probability of being a person who consulted with any musculoskeletal disorder, showing the independent effects of sex, geosocial deprivation, ethnicity, and age (model Chi-squared=1486.13, df=20, p<0.001)

Variable	OR (95% CI)
Female sex	0.92 (0.87–0.98)**
NZDep96 (referenced to category 1)	
2 *(less deprivation)*	1.07 (0.93–1.22)
3	1.04 (0.91–1.19)
4	1.12 (0.98–1.29)
5	1.12 (0.97–1.28)
6	1.14 (1.00–1.31)*
7	1.19 (1.04–1.37)**
8	1.05 (0.91–1.21)
9	1.30 (1.14–1.50)**
10 *(more deprivation)*	1.41 (1.22–1.63)**
Ethnicity (referenced to European)	
Maori	0.84 (0.76–0.94)**
Other	0.79 (0.74–0.84)**
Pacific people	0.79 (0.59–1.11)
Age (referenced to less than 20)	
20-29	2.15 (1.92–2.41)**
30-39	2.56 (2.30–2.85)**
40-49	3.52 (3.17–3.92)**
50-59	4.24 (3.80–4.73)**
60-69	4.59 (4.09–5.15)**
70-79	5.05 (4.48–5.69)**
80 and over	4.61(3.97–5.36)**

*p<0.01,**p<0.05; OR=odds ratio; CI=confidence interval.

Figure 2. The influence of age upon consultation rates for rheumatic diseases. Each graph shows the adjusted odds ratios (95% confidence intervals) plotted on a log scale, referenced to the rate for age less than 20 years.




OR for regional pain by AGE
100
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0.1
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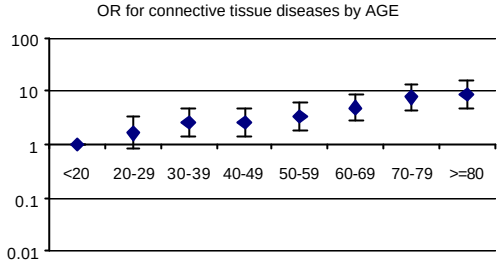
60-69
70-79
>=80


OR for connective tissue diseases by AGE
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70-79
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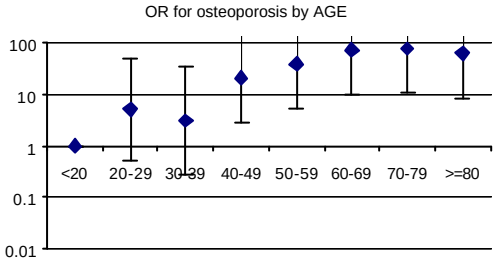
OR for osteoporosis by AGE
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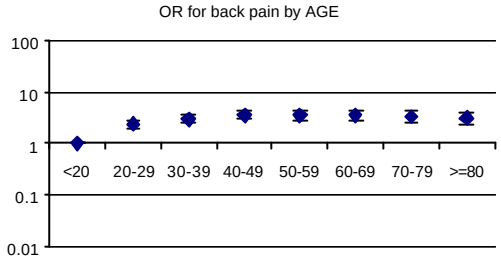
OR for back pain by AGE
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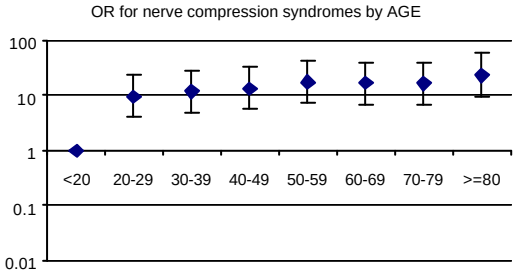
OR for nerve compression syndromes by AGE
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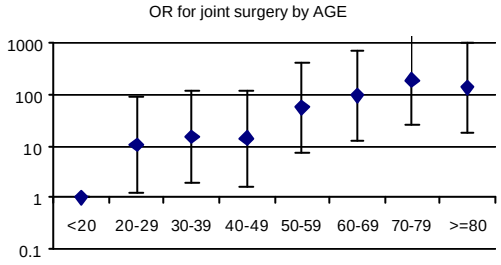
OR for joint surgery by AGE
1000
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40-49
50-59
60-69
70-79
>=80

Figure 3. The influence of ethnicity (upper panel) or geosocial deprivation (lower panel) upon consultation rates for selected rheumatic diseases. Each graph shows the adjusted odds ratios (95% confidence intervals) plotted on a log scale, referenced to the rate for Europeans (upper panel) or category 1 (lower panel).

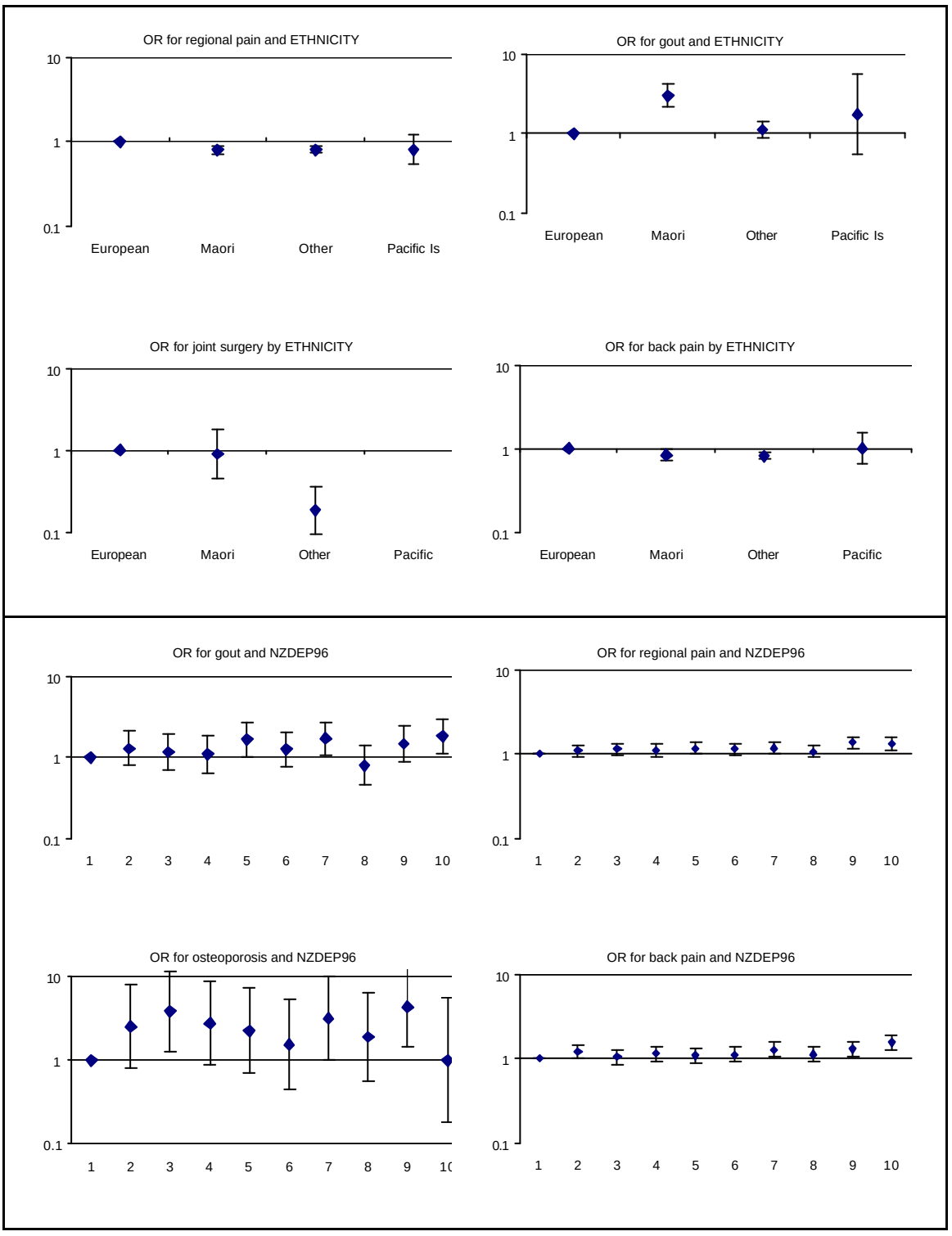
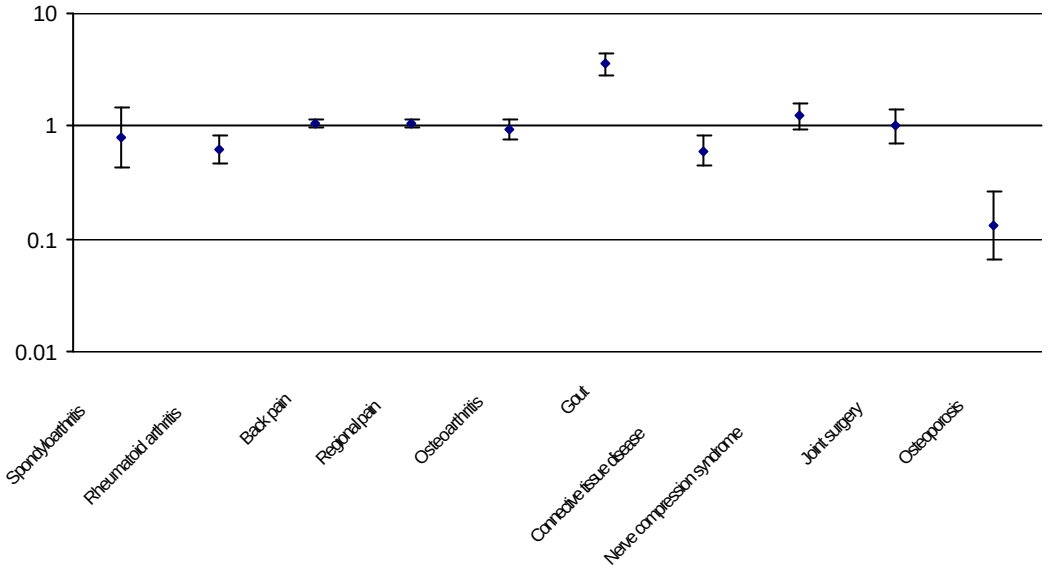


Figure 4. Influence of gender for consultation rates with rheumatic disorders. The odds ratios (95% confidence interval) for males is shown for each disorder on a log scale.



Ethnicity was not associated with spondyloarthritis, rheumatoid arthritis, connective tissues disease, or nerve compression syndromes. There was a trend towards fewer Maori consulting with osteoporosis (OR 0.17, 95%CI 0.02 to 1.28). Women were more likely to consult with rheumatoid arthritis (OR 1.59) and osteoporosis (OR 7.6) and less likely to consult with gout (OR 0.28). Increasing age was associated with increased likelihood of all disorders. People living in the most deprived areas were slightly more likely to consult with back pain, regional pain disorders, and possibly osteoporosis. Geosocial deprivation appeared to have little effect upon consultations for other disorders.

Discussion

The overall consultation rates for rheumatic disorders seen in this study were similar or a little higher than those observed in the three major surveys describing the content of general practice in New Zealand. The most recent of these was the 1991/1992 Waikato Medical Care Survey[12] which recorded a sample of encounter details of participating general practices over a 4-week time period (overall response rate of 68.6% of potential doctor recording weeks). Adjusting for the variable sampling fraction and practitioner non-response, the 11,888 encounters in the WaiMedCa survey were reported to represent 1% of all practice encounters over a 12-month period in this population (population of the sample frame of patients was 324,433).

Musculoskeletal (MSK) problems were managed in 17.4% of these encounters, second only to respiratory problems (21.7%). The most frequent individual MSK problems were osteoarthritis (1.6%), sprain/strain (2.8%), arthritis (1.2%), bursitis (1.2%), knee injury (1.1%), back complaint (1.0%), and neck injury (0.7%). A methodologically similar Waikato survey in 1981 found that MSK complaints accounted for 9.9% of all visits—and in a North Canterbury survey, 11.3% of visits were due to MSK complaints.[16]

Comparative data concerning consultation rates with general practitioners is also available from the United Kingdom.[17] This survey covered 1% of the population of England and Wales, from 60 NHS general practices that volunteered to take part. During the a 12-month period in 1991/1992, 78% of the population consulted at least once and 15% of the population consulted with a musculoskeletal disorder, which was the third most common problem after respiratory diseases (31%), nervous system diseases, and sense organs (17%). A comparison with a randomly selected evaluation of actual practice records suggested that the submitted diagnoses were correct in 93%.

The results from the 2002/03 NZHS have been reported in preliminary form.[10] This government-sponsored survey of population health status randomly sampled persons aged 15 years or over within randomly selected private dwellings and undertook face-to-face interviews using trained interviewers. The musculoskeletal section included these questions:*'Have you ever been told by a doctor that you have arthritis?'* and *'Have you ever been told by a doctor that you have a disorder of the neck or back?'*.

Comparison with the results presently available is difficult since the reported age bands (over 45 years) for back pain and arthritis are different from the age bands used in our study, and the denominators are different (general practice attendees rather than the general population). In the current study, 30.5% of the general practice population over 50 years consulted with a MSK disorder, and 28.8% of the general practice population over 40 years consulted with a MSK disorder. This suggests that a substantial proportion of the 30.0% of the community (sampled in the NZHS over 45 years with self-reported arthritis) may attend their general practitioner with related complaints.

If the notional catchment population for the general practices in this study is used as the denominator, then the 1-year prevalence of people who attend with a MSK problem amongst the general population is 14.6%. Unfortunately, the age-specific rates cannot be calculated from our study since the ages of people from the notional catchment population who did not attend with a MSK problem during the study year were not recorded. This makes direct comparison to the NZHS problematic. However, if we assume that the unadjusted odds ratios for effect of age in this general practice population (eg, 2.32 for age over 50 years) are similar to the relative risk in the notional catchment population, then we might expect about twice as many people over 50 years to have a MSK condition as those younger than 50 years. This is similar to the community population rates seen in the NZHS.

For individual rheumatic conditions, using the notional catchment population as the denominator will reduce the prevalence rates by a factor of 0.72, but community prevalence rates using this denominator are likely to be biased. Firstly, this is because not all people with a MSK condition will attend their GP and, secondly, the population of people who attend a GP at least once over a 3-year period may not be representative of the true community population, especially in terms of health status.

There are several substantial limitations to this study. Firstly, the identification of cases from the general practice database relied upon largely untested search strings, constructed by considering words typically used by doctors in consultation records for each of the 10 rheumatic disorders. A preliminary validity check of the free-text fields in two conditions amongst randomly selected records identified as being cases, suggested that the positive predictive value of this search strategy was 80% to 93%.

Since patient identifying data is not held by the database, it is not possible to manually review cases or non-cases to verify diagnostic accuracy. Nevertheless, the consultation rates and relationship of these to age and gender were within reasonable distance of what is reported in other general practice and population surveys, so it seems unlikely that the errors associated with this case-identification method is greatly biased in any one direction.

Secondly, the general practice population obtained in this study was not representative of the general community, especially with regard to Pacific people. It was not possible to generate risk estimates for this ethnicity because of sparse data-points. The recording of ethnicity from general practice records tends to over record 'other' ethnicity with proportionally fewer Maori or Pacific people, and this was also observed in our study.

Thirdly, the denominator in this study was a general practice population drawn from those people attending at least once over a 12-month period. This is likely to be different from the general population in several ways, especially with regard to the frequency of health problems. Translating consultation rates into population prevalence rates is very problematic since not all patients with rheumatic diseases will consult.[18]

However, in terms of determining the size of the problem as it presents to general practice, these data are potentially helpful. A significant proportion of the workload in general practice is due to rheumatic diseases and much of this relates to non-inflammatory and non-orthopaedic problems, particularly regional pain disorders (13.8% of patients) and back pain (7.18% of patients).

Gout also forms a significant part of the workload, being nearly as frequent as osteoarthritis. For Maori, gout is an important rheumatic disorder with adjusted odds ratio (approximate relative risk) of 3.54 (95% CI 2.84 to 4.42) compared to Europeans. Gout accounted for 7.8% of Maori general practice attendees over the age of 40 years, and 12.5% of Maori male general practice attendees over the age of 40 years. Ethnicity may also play a role in the presentation of osteoporosis, regional pain and back pain, which are less frequent in Maori. Patients coded as 'other ethnicity' presented much less frequently with joint surgery, which may be an indication of differences in the use of surgical services.

The influence of socioeconomic factors in the frequency of MSK disorders is confirmed in this study, with an adjusted odds ratio of 1.41 (95% CI 1.22 to 1.63) for consultation with any rheumatic disorder, for the most deprived versus the least deprived categories. This effect appears to be largely occurring in regional MSK pain, including back pain, and gout. A population survey in the UK found that a social deprivation index was associated with the self-report of MSK to a similar degree (relative risk of 1.16 for any MSK and 1.38 for back pain, for the most deprived versus the least deprived categories).[19]

It is interesting to observe a possible effect of geosocial deprivation upon consultation rates for osteoporosis, although the wide confidence intervals prevent firm conclusions about this association. It is possible to speculate that dietary factors, especially calcium intake could be a factor in any such association.

To more completely understand the epidemiology of rheumatic disorders in the New Zealand general population, it is necessary to ascertain cases from a large community-based sample. This poses considerable logistic problems, but an approach that combines screening by postal survey, telephone interview,[20] and then direct interview and examination[21] may be feasible.

It is interesting to note that there are no musculoskeletal disorders listed amongst the 13 population health objectives in the 2000 New Zealand Health Strategy,[22] which suggests that these diseases are 'hidden' to policy makers. Perhaps the relative prioritisation of rheumatic disorders should be reconsidered, given the relatively high frequency of these conditions amongst general practice attendees.

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Low back pain in young New Zealanders

David McBride, Dorothy Begg, Peter Herbison, Ken Buckingham

Abstract

Aims To describe the occupational implications and impact of low back pain (LBP) in a birth cohort now aged 26.

Methods The LBP data were collected by an interviewer-administered questionnaire. Study members were asked about the prevalence and frequency of LBP and, for the worst episode, details were sought on occupation, time off work or job limitation, the severity of the discomfort, and disability.

Results The cohort comprised 980 individuals, and 969 individuals answered the questionnaire. Of these 969 individuals, 524 (54%) experienced LBP in the previous 12 months, with a modal frequency of three or more times a year. For the 448 individuals with a current occupation, there was no difference in the distribution of LBP between those with professional, clerical, and technical jobs and those with production or trades jobs. Fifty-six individuals had to have time off work (the majority for less than 7 days), and 13 individuals could not look after themselves because of pain. Based on data gathered on 26 year olds, the cost to the economy is estimated as $NZ500 million per annum.

Conclusion LBP is prevalent and disabling even in young people, and has high costs. The natural history of the disorder is still largely unknown, but work does play a part. Although getting a person back to work may still be the best treatment, it is important to address the workplace factors.

Low Back Pain (LBP) has been described as the most common, most costly and disabling musculoskeletal condition.[1] Since World War 2, a dramatic increase in LBP disability has been observed, at a rate disproportionate to all other health conditions.[2] In New Zealand, this is reflected by the numbers of, and costs to, the Accident Compensation Corporation (ACC) for back pain claims—in 2000–2001, 10,968 new claims cost $30 million and 6,660 ongoing claims cost $68 million.[3]

The majority of persons with LBP recover within 2 months, but 2–3% eventually develop disabling chronic LBP (DCLBP)[1]—these patients accounting for 80% of the costs of compensation. LBP, therefore, has major socioeconomic implications; with many of the tangible costs related to disability and compensation.

This aim of this study was to describe the frequency, severity, and economic impact of LBP in a birth cohort of individuals now aged 26.

Method

The study was part of the Dunedin Multidisciplinary Health and Development Study, which has been described in detail elsewhere.[4] In summary, it is an ongoing follow-up of approximately 1,000 young people who were born in Dunedin over a 12-month period in 1972 and 1973. The cohort has been studied at birth, at age 3, every 2 years to age 15, then at ages 18, 21, and 26.

The demographics of this group, and their physical and mental wellbeing, have been carefully documented. The LBP data were collected by interviewer-administered questionnaire during which study members were asked about the number of episodes of LBP occurring in the previous 12 months and consultations with health professionals. For the worst episode of LBP, details were sought on occupation, time off work or job limitation, the severity of the discomfort (as measured on a visual analogue scale with a maximum score of 100), and disability (as measured by the modified Oswestry questionnaire).[5]

Results

Of the 980 study members interviewed, 969 completed the LBP questionnaire. 524 individuals, 244 of the 477 females (51% of females), and 280 of the 492 males (57% of males) indicated that they had experienced LBP in the previous 12 months, the frequency of occurrence of which is shown in Table 1.

Table 1, How often LBP (low back pain) was experienced in the previous 12 months.

How often LBP occurred in previous year	Number of individuals	% of total with LBP
Once	52	10
Twice	54	10
Three or more times	305	58
On most days	113	22
At any time (total)	524	100

LBP severity showed a bimodal distribution with a median of 49 and modal values of 25 and 65 (Figure 1). There was some evidence that those who had more frequent episodes suffered from higher reported discomfort (Figure 2).

The answers to the disability questions indicated a small but significant number of individuals with severe impairments. Thirteen indicated that they could not look after themselves because of pain and seventeen avoided walking. Eleven indicated that pain prevented them from sleeping at all. One individual was chronically disabled by back pain and unable to work for most of the year.

There was a similar proportion of individuals with back pain in the employed and non-employed groups—and for the 448 with a current occupation, there was no difference in the distribution of LBP between those with professional, clerical and technical jobs and those with production or trades jobs.

Fifty-six individuals with LBP had to have time off work because of discomfort. These individuals reported more severe pain (mean score of 71) than those who did not take days off (mean score of 46); the difference being statistically significant (t=9.6, p=000) Of these 56 individuals, 45 consulted a health professional. The 11 who did not consult took 3 days or less off work. The majority had relatively short periods off work; 26 (43.3%) had 1 or 2 days, 41 (72%) taking less than 7 days. There were, however, 11 individuals (28%) who took more than 1 week off from work. Of those that did not have time off work, 158 indicated that LBP affected their work by either slowing them down or necessitating a change in their duties. There was evidence (Figure 3) that those who had more frequent episodes tended to take more time off work.

Figure 1. Distribution of pain severity

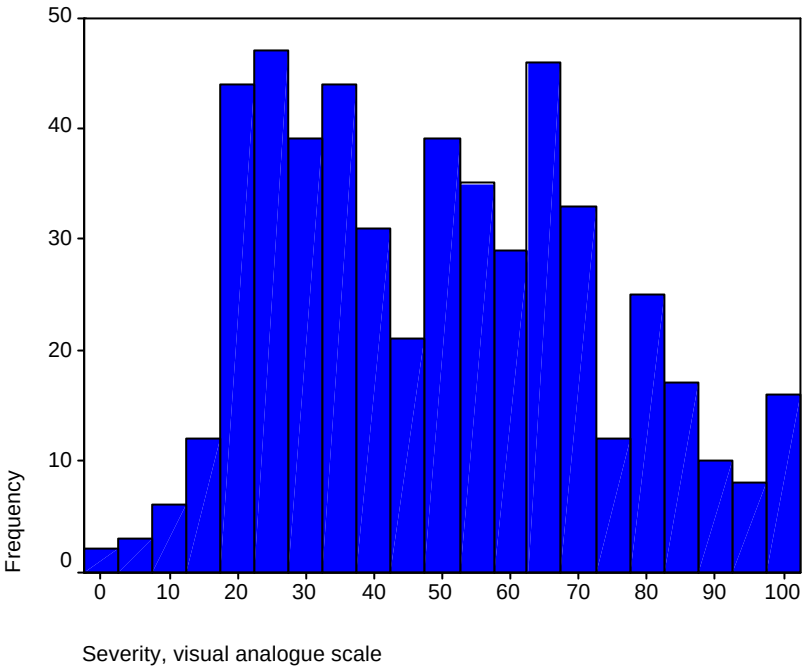


Figure 2. Relationship between the mean severity of discomfort (visual analogue scale) and the number of episodes of LBP in the previous year

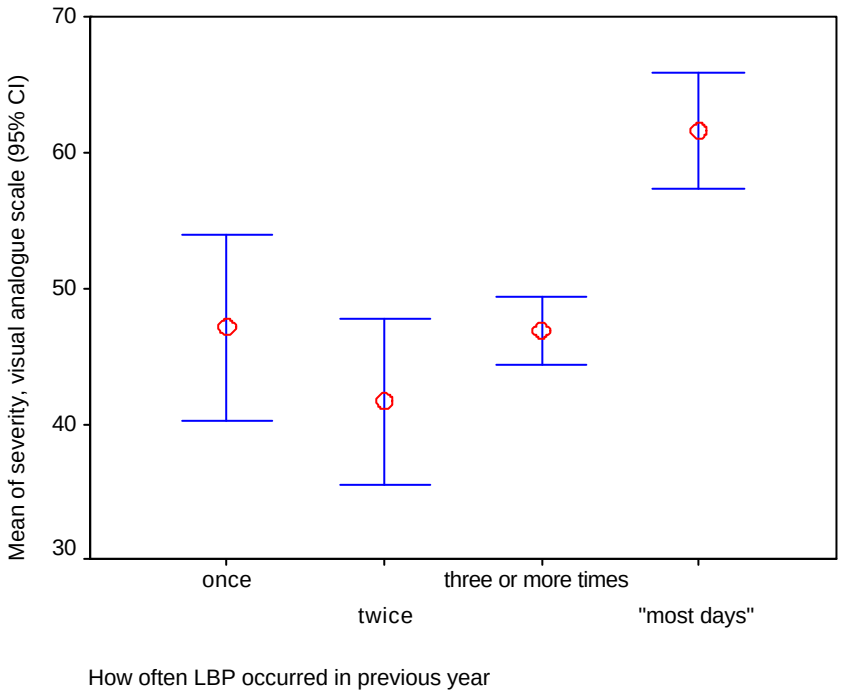
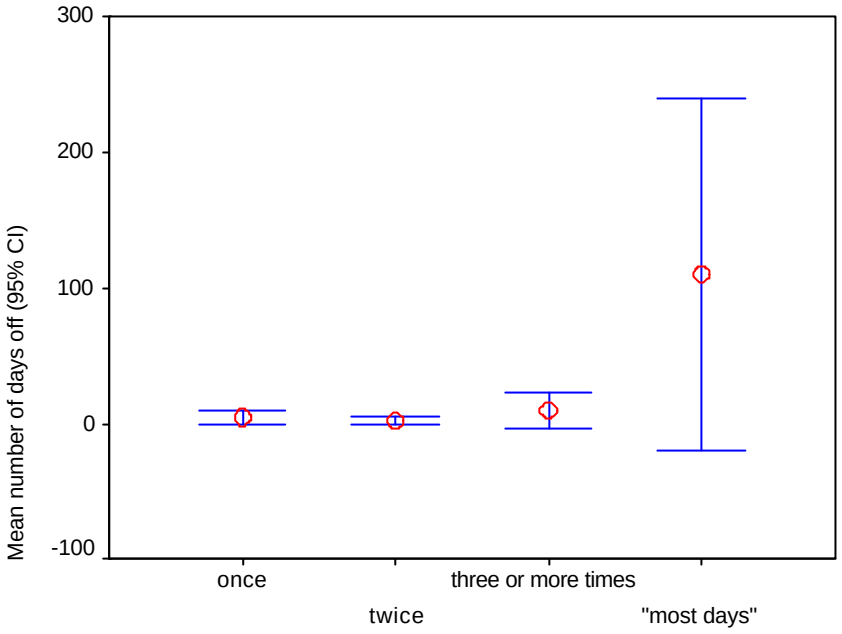


Figure 3. Relationship between the mean number of days off work and the number of episodes of LBP in the previous year



The indirect costs to the economy can be estimated in that the 448 respondents (who had a job in the year prior to assessment) lost a total of 665 days of work through illness due to back pain, implying a total number of days lost per working person as approximately 1.5 days per year. The average weekly earnings of 25–29 year old New Zealanders in paid employment was $676 (before tax) in June 2003.[6] Assuming a 5-day working week, daily earnings for this age group are $135 (before tax), and the value of the annual loss of working days per person in employment is approximately $203.

Of those individuals with symptoms, 150 (29%) sought treatment—with 37 individuals consulting a physician as sole treating practitioner, 11 consulting a chiropractor, 8 consulting a physiotherapist, and 3 consulting an osteopath. There were 15 'other' health professionals (masseurs) providing the sole source of advice Fifty-one individuals sought advice from a treatment team, most commonly a physician/physiotherapist combination. The remainder sought advice from multiple sources.

Discussion

In the planning phase of this study, evidence from an earlier cross sectional study in New Zealand[7] suggested that there would be at least a 20% prevalence of LBP during the previous year. The high rate of LBP in this young group might therefore be

viewed as a source of concern, but the prevalence alone does not give the whole picture because it varies according to the question asked and the target population.

In international studies, the peak frequency of lifetime LBP occurs in the age range 30–55 years,[8] which differs according to the prevalence period, possibly illustrating a 'recall' effect for more recent events.[9]

In the Life in New Zealand (LINZ) study,[7] the annual prevalence was highest for young males aged 15–18 (44%); the figures for the 19–24 and 25–44 year old groups being 20% and 17% respectively. The prevalence of 'ever' having LBP was, however, highest in the 45–64 year old group (67%). There is no satisfactory explanation of why this pattern of reporting should appear, but it seems likely that different biomechanical factors are responsible in the different age groups.

At the most simplistic level, there are a number of 'pain generators' in the lower back, the disc being thought responsible for approximately 36% of back pain cases; facet joints for 15%; the sacroiliac joint 30%; and the remaining 19% assumed to be soft tissue injuries or a combination of pathologies.[8]

The cumulative trauma model[8] was developed due to increasing acknowledgement that acute and chronic LBP are different (with soft tissue injuries being more likely in the younger age groups and degenerative changes more likely in the older age groups). This model fits with what is known about occupational LBP, because the onset is gradual and frequently not related to an 'accidental' cause.[8]

It is important, because of the cumulative trauma model, to focus on more outcome-specific measures rather than the simple presence or absence of pain. Thus, LBP can be described in terms of duration, frequency, and disability—attributes which describe the 'severity' or importance of an attack.

Duration of the attack is obviously important, and restricting the outcome to LBP that lasts for at least 2 weeks obviously indicates a more significant event. Such LBP has a lower prevalence of around 14%.[10] Duration of time off work is also a predictor of chronicity; those off work for 1 month having a 20% risk of long-term disability.[11] The frequency of attacks is also indicative of chronicity: the 22% prevalence of attacks occurring 'most days' in this study seems high until one looks at the daily reporting in LINZ for the 15–18, 19–24, and 25–44 year age groups (which was 28%, 33%, and 11% respectively).[7]

Disability is arguably the most important occupational indicator, and the fact that a small but significant proportion of such a young population experienced severe disability should be cause for concern. LBP with onset early in life is thought to be indicative of a poor long-term outlook, especially if associated with a long initial episode.[12] The reaction to a first episode of LBP is also important, with growing evidence that pain associated with fear leads to a poor outcome.[13]

An inability to work has consequences not only for the individual but also for society. Although the cost per working individual was only NZ$203, there were 1,956,000 individuals in employment in the March 2004 quarter,[14] so a current estimate of the annual loss to the economy in this age group is NZ$396 million. If ACC costs are estimated at NZ$100 million, then the total (excluding the costs of healthcare not funded by ACC) will be nearly NZ$500 million.

Due to the social and economic consequences, it should be important to identify those persons 'at risk', but the search to identify individual risk factors has been very disappointing to date.

Although our data do not support any clear association between occupation and risk, occupational factors are important. A National Institute of Occupational Safety and Health review panel[15] identified 40 research papers looking at the relationship between LBP and physical workplace factors. They found 'strong' evidence that LBP was linked to whole-body vibration and work-related lifting/forceful movements, and that LBP is associated with heavy physical work and work-related awkward postures. The review also emphasises the point made earlier—LBP is not a uniform entity, it is complex, it means different things at different ages, and it has to be looked at in different contexts.

For future epidemiological studies, we will have to define what 'clinically significant' LBP is, and develop standardised tools and questionnaires to detect this outcome. The results of studies can then be directly compared. We must also be very clear what we are looking for, and specific factors (for example, vibration or posture) must be carefully defined and measured prospectively to clarify dose-response relationships and to 'unmask' occupational effects.

What should be done about it? LBP is a common life experience, but can become costly and disabling. Because of the lack of clear risk factors, primary prevention has been disappointing, and the positive benefits of any single treatment modality have been uncertain. As a result, some researchers have suggested that occupational factors are relatively unimportant and that LBP is a symptom not a disease.[16,17] Current advice from the Accident Compensation Corporation and the New Zealand Guidelines Group, outlined in the New Zealand Acute LBP Guide[18] is that the best treatment is to get the individual back to work. There is, however, a danger in this approach.

Because of all the confusion about what LBP is, or means, one can be led to ignore the biomechanical model. This is where the conundrum lies: because the trauma is cumulative the occupational link is not clear, and although the best treatment for someone in employment may be to get the individual back to work, the work may have caused the condition in the first place and needs to be assessed. The New Zealand Acute LBP Guide[18] states: *It is important to discuss work activities, especially those involving heavy lifting, bending or twisting, that may have contributed to the original problems. Alternative duties and/or workplace design may need to be discussed with the worker and/or employer.*

Some LBP 'patients' show fear-avoidance beliefs, and avoid activities which are predicted to cause an increase in pain and suffering.[19] Graded exposure to work activities in the acute phase may help to avoid chronic pain in some individuals,[19] but this obviously requires active workplace intervention. The busy family physician will not usually have time for this and should be willing to refer. Our data indicates that this does seem to happen relatively often, a physician and physiotherapist 'team' being the commonest combination. We advocate that at least one member of the team should be familiar with the work.

Work-related and physical factors are, however, low in the hierarchy of LBP risk factors. The psychological and social elements are pre-eminent, and undoubtedly important in maintaining disability. We now propose to look at these factors

(particularly their temporality) in relation to the occurrence of back pain within the cohort. Some of the most important elements will be previous psychiatric disorders identified using the Diagnostic Interview Schedule,[20] and the social variables which have been measured using instruments such as the life history questionnaire.[21]

The value of the cohort in looking at the natural history of LBP will now be clear: the high prevalence of LBP at this early stage does not seem not to bode well for the future. It is, in our view, very important to define exactly what we mean by LBP and to identify what we think is causing it. The natural history can then be investigated properly and preventive measures designed.

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Percutaneous vertebroplasty for osteoporotic fracture: preliminary experience at Middlemore Hospital

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Abstract

Aims To describe our initial experience with percutaneous vertebroplasty in a New Zealand teaching hospital setting.

Methods Five patients suffering osteoporotic vertebral fractures were treated with percutaneous vertebroplasty.

Results Three patients experienced improvement in pain after the procedure. The other two experienced documented improvement in mobility. There were no significant complications.

Discussion This small series shows that percutaneous vertebroplasty has been successfully performed in a New Zealand teaching hospital. The definitive role of this technique in the management of patients with osteoporotic fractures remains to be determined.

Osteoporotic vertebral fractures are common in the elderly New Zealand population. The National Health Committee report on osteoporosis[1] indicates that such fractures are an integral part of the osteoporotic syndrome, although their incidence is difficult to determine. Steroid treatment and malignancy can also cause vertebral fractures. Whatever the aetiology, vertebral compression fractures can be very painful, causing immobility and difficulty with normal activities.

Conventional treatment involves analgesics and, sometimes, bisphosphonate therapy. Percutaneous vertebroplasty is a treatment technique that has been used successfully in Europe since first reported in 1987[2,3] and for 10 years in North America.[4] We report on our initial experience with the technique in Middlemore Hospital.

Methods

Five patients were treated with percutaneous vertebroplasty over a 10-month period. Pre-procedurally, the patients provided written informed consent. Coagulation studies were normal and vertebral infection excluded. All five patients had typical osteoporotic compression fractures after minor trauma (falls while walking in three patients, trip without fall in one, and low speed motor vehicle accident in one). There were no complex fractures, burst fractures or pathological fractures secondary to malignancy in the series.

All five patients had preliminary magnetic resonance imaging (MRI) showing bone marrow oedema within the relevant vertebral body on short tau inversion recovery (STIR) images, confirming the presence of an acute fracture (Figure 1). In one of these patients, MRI was initially performed on clinical suspicion of a pelvic fracture but in fact showed a fracture of L5. In another patient for whom vertebroplasty was not performed, pre-procedure MRI revealed that the patient actually had a sacral insufficiency fracture. Patient age ranged from 79 to 83 years with an average of 81 years. There were four females and one male in the series. Symptom duration prior to treatment and length of follow-up were recorded (see Table 1).

In all five patients, vertebroplasty was performed using the following technique. After sterile preparation and local anaesthetic, C-arm fluoroscopy was used to place either unilateral or bilateral 13-

or 11-gauge needles (Osteo-Site™, Cook Inc, Bloomington IN, USA) into the vertebral body via the pedicle.

Once needle placement in the anterior body was established, polymethyl methacrylate cement, mixed with barium sulphate to provide radio-opacity, was injected until cement reached the posterior vertebral cortex or extravasation was seen (Figure 2).

The cement comprised 40 g of polymethyl methacrylate plus 5 g of barium sulphate powder mixed with methyl methacrylate to a toothpaste-like consistency (all components DePuy CMV, Blackpool, England).

Three patients received unilateral injections in the L1 vertebra and one of these patients also received bilateral injections in the T12 vertebra during the same procedure. The two other patients received bilateral injections in the T12 and L4 vertebrae respectively. The average amount of cement per injection was 4.1 cc (range, 1.5 cc to 6.5 cc).

No periprocedural or intra-cement antibiotics were used, and vertebral venography was not employed. All patients were given intravenous midazolam and fentanyl during the procedure but remained awake and able to report pain and/or neurological symptoms.

Blood pressure, pulse, and pulse oximetry were monitored. Haemostasis was achieved using simple pressure, and post-procedural follow-up consisted of regular observations of vital signs both in Middlemore Hospital's Radiology Department and on returning to the hospital ward.

An attempt was made to obtain pain scores and indices of activity before and after the procedures. Pain scores were recorded on a scale of 0 (no pain) to 10 (worst pain imaginable). A nurse assisted the patient in conveying pain scores. Four patients were able to provide pain scores before and after the procedure. Activity scores were recorded using the standard Barthel ADL index.[5] The fluoroscopy and room time taken for each procedure was recorded.

Figure 1A. Lateral lumbar spine radiograph and
Figure 1B. Sagittal short tau inversion recovery (STIR) magnetic resonance imaging (MRI) image show an acute fracture of L1 vertebral body
(Note old T11 fracture also)

Fig 1A

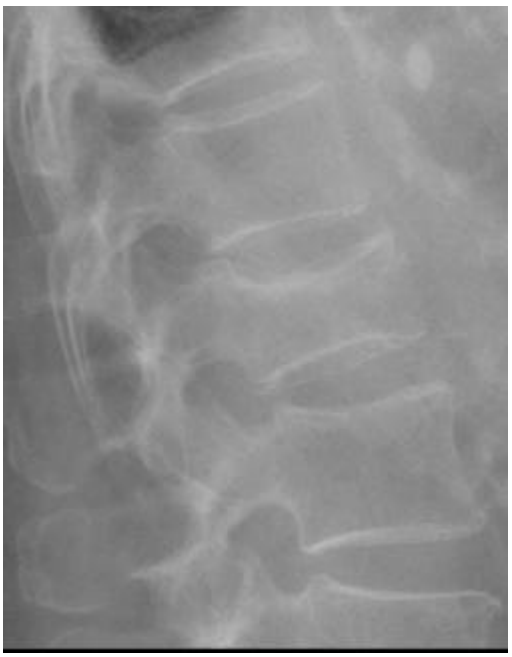

Fig 1B

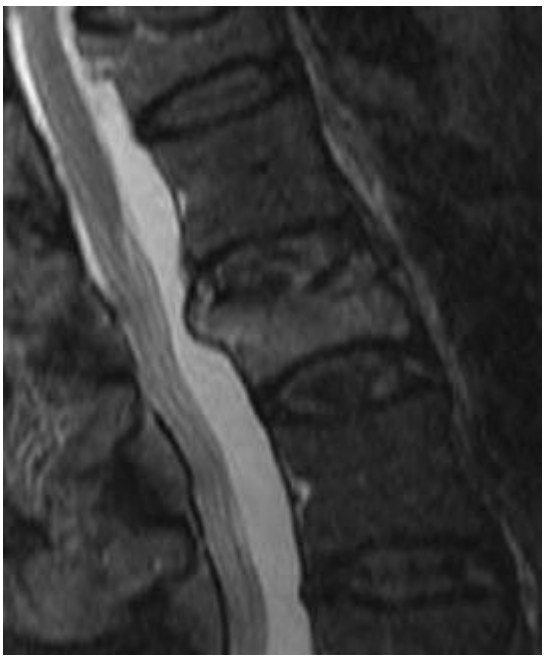

Figure 2A and Figure 2B. Under fluoroscopy, a needle is inserted into the vertebral body and cement injected to fill the marrow cavity

Fig 2A

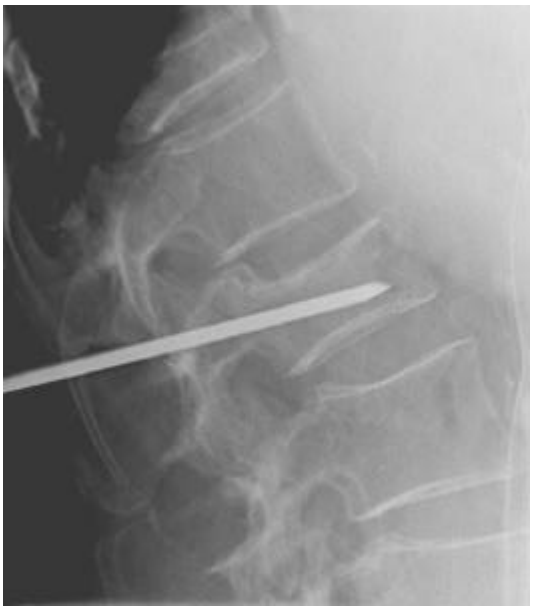

Fig 2B

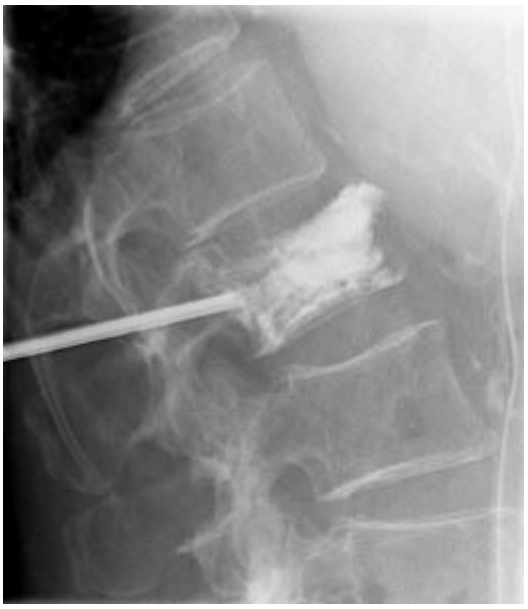

Table 1. Patient and procedure details

Variable	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Symptomatic*	18 days	24 days	30 days	13 days	45 days
Level 1	L4 both†	L1 left	L1 right	L1 left	T12 both
Level 2			T12 both		
Screening time	13.9minutes	3.4 minutes	8.7 minutes	9.2min	12 minutes
Cement total	5 cc	3.5 cc	6.5 cc/9.9 cc	4cc	1 cc
Pain pre/post	5/0	10/0	10/9	10/6	Not available
Barthel pre/post	11/18	13/16	13/17	12/10	Not available
Complication	Pain	Pain	Extravasation‡	Extravasation	Extravasation
Follow-up	16 days	20 months	14 days	19 months	7 days

*This row indicates the duration of symptoms prior to vertebroplasty; †This refers to whether the right, left or both pedicles were cannulated; ‡In all three patients with extravasation, this was minor and limited to the paravertebral veins or intervertebral disc.

Results

Of the five patients treated, three experienced immediate pain relief and increased mobility. Two of them indicated post-procedure pain scores of zero while the third experienced a decrease in worst pain from 10/10 to 6/10 and in best pain from 9/10 to 0/10.

In the fourth patient, two levels were addressed. Pain relief was not immediate and pain scores decreased only minimally but the patient's activity improved from 12 to 17 points on the Barthel score and they were discharged walking with a frame 2

weeks after the procedure. The fifth patient did not have pain scores recorded, partly because of a chronic cognitive deficit and an acute stroke (which happened before the procedure). This patient was able to transfer with the aid of one person (rather than two persons) after the procedure, but died from a perforated viscus 1 week later. This was thought not to be related to vertebroplasty but to multiple comorbidities.

Complications were limited to transient increase in pain during the procedure (in all patients) and to small amounts of cement extravasation into paravertebral veins in three patients. No symptoms could be attributed to this cement extravasation. No radiculopathy, other neurological compromise, or distant emboli were demonstrated.

Procedure time ranged from 60 to 110 minutes (average: 85 minutes). Screening (fluoroscopy) times ranged from 3.4 to 13.9 minutes (average: 9.4 minutes.) A radiologist, medical radiation technologist, and nurse were usually involved in the procedure. The consumable supplies for each procedure cost approximately NZ$600. Further details are given in Table 1.

Discussion

The role of percutaneous vertebroplasty in managing patients with painful osteoporotic vertebral fractures is evolving.[3,4,6,7] As demonstrated in our small series, the procedure can be successfully performed if care is taken with technique. However, there remains disagreement as to which patients should have the procedure and at what point in the natural history of vertebral fracture it should be performed.

Some practitioners advocate using percutaneous vertebroplasty early to maximise the effect on mobilisation and/or rehabilitation,[4,7,8] while others reserve percutaneous vertebroplasty for patients who have not responded to conservative therapy.[3,6]Anecdotal evidence certainly suggests that, at least in some patients, percutaneous vertebroplasty produces an immediate and quite profound relief of pain, with improved activity tolerance and decreased analgesic use. In our small series, vertebroplasty was clearly beneficial for four out of the five patients.

From our experience, it is clear that MRI (including a STIR or similar sequence prior to the procedure) is desirable to confirm the presence of an acute vertebral fracture. Many osteoporotic patients have more than one flattened vertebra upon viewing their radiographs, and it may be very difficult to decide which of these are chronic fractures and which are acute. It is also possible, as in one case mentioned above, that the patient may not have an acute vertebral fracture and therefore would not benefit from vertebroplasty. MRI helps to resolve these problems in practice although its role has not been scientifically validated.

A significant impediment to understanding the role of percutaneous vertebroplasty in fracture management is the lack of any randomised controlled trials comparing it with medical therapy.

Zoarski et al[9] evaluated pain relief in a prospective non-randomised study and found that 22 out of 23 patients were satisfied at 15–18 months follow-up. McGraw[10] prospectively followed 100 patients and found a statistical improvement in pain. Other retrospective studies show good pain relief by percutaneous vertebroplasty. There are no reports of further vertebral height loss following percutaneous vertebroplasty. The results of percutaneous vertebroplasty in malignant disease have

been similarly encouraging though pain relief may not be as great as in osteoporotic fracture.[11]

Some of the costs involved are mentioned above. These include the preliminary MRI scan, the consumables used, the cost of 85 minutes in the fluoroscopy suite and the time of the radiologist, nurse, and technologist. We have not attempted a comparison with the estimated cost of conservative therapy in our five patients.

Complications occur in around 1% of patients. These complications consist chiefly of a transient increase in general pain and radicular pain, both usually treatable with simple analgesia and occasionally anaesthetic injections. Infection and significant neurological complications are rare. Some practitioners recommend the use of antibiotic in the cement but many do not.[3,4,6–12]

Reports of infectious complications are rare; one case reported recently was in the context of incompletely treated infection (presumed urinary tract but possibly spinal) immediately prior to the vertebroplasty.[13] Those authors indicated they had treated 200 other patients without using antibiotics in the cement and that it is not normally necessary.

Both intra-cement and intravenous antibiotics are probably advisable in immunocompromised patients.[4] Pulmonary emboli can occur and deaths have been reported in two multi-level procedures probably due to respiratory compromise secondary to fat and/or cement emboli.[12] Malignant disease is associated with a higher complication rate, probably due to higher cement leakage.

The only complications experienced in our series were the minor ones of transient increase in pain and minor cement extravasation. Our technique, complications, and results are in compliance with the only published guideline we know of for this procedure, issued by the American College of Radiology.[14]

Several hundred cases of vertebroplasty guided by fluoroscopy have now been described in the literature. There is general agreement that fluoroscopy (provided it is of adequate quality) is the guidance method of choice because the injection and distribution of cement can be monitored in real-time. Most practitioners do not use computed tomography (CT) to guide the procedure or to verify cement placement.[2–4, 6-12] Technical developments in this arena continue.

An interesting extension of vertebroplasty is the use of expandable balloons to develop a cavity in which to place the cement and to provide for some correction of kyphosis. That procedure, known as balloon kyphoplasty, has certain claimed advantages compared to simple cement vertebroplasty but it is relatively early in its clinical development and requires general anaesthesia plus more expensive consumable equipment.[15]

Percutaneous vertebroplasty is gaining rapid acceptance worldwide as a safe and reliable tool in the treatment of pain due to vertebral fracture, principally as an aid to early and rapid mobilisation and rehabilitation of patients. Our early experience concurs with this assessment. Where interventional and orthopaedic radiologists have access to good quality fluoroscopy facilities, we believe that percutaneous vertebroplasty can provide effective treatment for osteoporotic and malignant vertebral fractures not responding to conservative therapy.

In future, percutaneous vertebroplasty may become the primary treatment modality in these cases. A randomised trial including a cost-benefit analysis would help to establish the proper place of the procedure in the management of vertebral fractures.

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Ruptured abdominal aortic aneurysms: risk factors for mortality after emergency repair

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Abstract

Aims Emergency repair for ruptured aneurysm is associated with a high mortality rate. From our experience of treatment of ruptured abdominal aortic aneurysms (AAA), we evaluated the morbidity and mortality rates, and identified preoperative variables that may be predictive of mortality after emergency repair.

Methods Retrospective review of 41 patients who had emergency ruptured AAAs repair during July 1996 to April 2002 in Middlemore Hospital, Auckland, New Zealand. Demographic, preoperative, intraoperative, and postoperative data were recorded and statistically analysed to identify predictors for mortality.

Results The mean age of the population was 72.9 years (range 57–89 years). Fifteen percent of patients died during the operation, and 26% died postoperatively. Bleeding was the most common cause of postoperative death (45%), followed by myocardial infarction (36%) and multisystem organ failure (27%). The overall surgical mortality rate was 41%. Serum creatinine of ≥0.15, age ≥80 years, 3 or more medical comorbidities and preoperative hypotension (systolic <90 mmHg) were identified to be significant risk factors for mortality ($p<0.05$) by univariate analysis. Serum creatinine of ≥0.15 mmol/L was the only variable multivariately ($p<0.05$) associated with mortality with an odds ratio of 9.3.

Conclusions Selection of patients with ruptured AAAs for emergency repair can be a complex and emotionally charged process. Simple preoperative variables with predictive values have been identified, which may be used to complement the surgeon's own patient selection criteria for emergency repair.

Rupture of an abdominal aortic aneurysm (AAA) is a lethal event, carrying a mortality rate of between 32% and 95% dependent on access to surgical intervention.[1,2] Rose et al recently reported a 46% hospital mortality rate in Auckland.[3] They also noted an increasing incidence of ruptured AAA over a 5-year period (1993–1997).[3]

Ruptured AAA is a common problem, resulting in a significant workload for vascular surgery and intensive care services. In Scotland, Best et al reported that the proportion of AAA patients admitted as an emergency increased from 40.4% in 1981 to 54.6% in 2000.[4] Furthermore, in 2000, ruptured AAA was the documented cause of 4028 deaths in England and Wales (accounting for 1.4% of all deaths in men over 65 years).[5]

In New Zealand, most vascular surgeons contribute their data to NZVASC, a national audit database administered by the New Zealand Society of Vascular Surgeons. A recent retrospective study by Rosaak et al using the NZVASC database identified

1868 patients who had an AAA repaired between 1993 to 2000, of whom 30.8% had emergency repair of their aneurysms.[17]

Despite recent advances in anaesthetic, operative, and postoperative care, the high mortality figure has prompted many surgeons to question whether repairing ruptured AAAs should even be attempted in the subsets of patients with poor physiological reserve (such as elderly patients with multiple medical comorbidities). However, if the patients recover from their emergency repair, they have a good long term survival.[18] Recent studies have demonstrated that life expectancy in operative survivors with ruptured AAAs is comparable to those who have undergone elective repair.[14]

The aims of this study are to evaluate the morbidity and mortality rates after emergency ruptured AAA repair in a single institution and to identify preoperative variables that may be predictive of higher mortality.

Methods

We conducted a retrospective analysis of 41 patients with ruptured AAAs who had undergone emergency repair between 4th July 1996 and 9th April 2002 at Middlemore Hospital, Auckland. Middlemore Hospital is a high-volume tertiary referral hospital serving a population of around 400,000 people in the southern part of the greater Auckland region, New Zealand.

Patients were identified from ICD-9 diagnosis and computerised surgical service records. All patients with the clinical diagnosis of ruptured abdominal aortic aneurysms (and who had emergency repair) were included. A ruptured aneurysm, identified radiologically or during surgery, was indicated by association with a retroperitoneal haematoma or free intraperitoneal bleeding. Those patients with thoracoabdominal aneurysms, isolated iliac artery aneurysms, pseudoaneurysms, and chronic contained aneurysms were excluded.

Demographic, preoperative, intraoperative, and postoperative data were collected retrospectively through individual questionnaires for multivariate analyses.

These data were classified as:

Patient demographics and preoperative data—comprised source of referral, mode of transport, vital signs on arrival to emergency department, and significant medical comorbidities (diabetes, hypertension, ischaemic heart disease, chronic obstructive airway disease, CVA, peripheral vascular disease, and chronic renal failure). Occurrence of cardiac arrest (asystole or pulseless ventricular fibrillation), hypotension (systolic blood pressure <90mmHg), loss of consciousness and cardiopulmonary resuscitation prior to surgery were also noted. Blood results (eg, haemoglobin, haematocrit, platelets, and serum creatinine) were documented. The duration of symptoms prior to admission and time interval between admission and start of surgery were also recorded.

Intraoperative data—Duration and timing (day [0800–1800] or night) of surgery; extent of rupture; estimated blood loss; requirement of blood transfusion; urine output during surgery; hypotension (systolic BP <90 mmHg for >20 minutes); cardiac arrest; aneurysm location (infrarenal or suprarenal); site of placement of aortic cross clamp; type of graft (straight or bifurcated); size of aneurysms; inotropic drug use; and simultaneous secondary operations (eg, femoral embolectomy, colectomy).

Postoperative data—Postoperative destination (ICU or wards); need for mechanical ventilation and its duration; and length of stay in ICU. Postoperative complications recorded included respiratory failure (mechanical ventilation >5 days), tracheostomy, renal failure (defined as oliguria or serum Cr >0.16mmol/L, but excluding patients with preoperative elevated creatinine), sepsis (positive blood culture), cardiac complications (perioperative myocardial infarction or cardiac failure), bleeding, ischaemic colitis (diagnosed by colonoscopy and requiring colectomy), stroke, lower extremity ischaemia (in which vascular intervention was required), graft thrombosis and need for re-operation (eg, to control bleeding). Destination after discharge from the Surgical Service and follow-up period were also recorded.

Mortality data included the cause(s) of death and the time-interval after surgery in the case of postoperative deaths.

Statistical analysis of various clinical variables was carried out to give rates of mortality and morbidity. Comparison of clinical parameters was performed by using a Chi-squared test in all cases. Statistical significance was assumed for $p<0.05$. Multiple logistic regression was used during multivariate analysis to simultaneously evaluate the effects of significant risk factors identified in univariate analysis. The odds ratio, 95% confidence interval, and p values were calculated.

Results

During the study period, 61 patients were admitted with the ruptured AAA. However, emergency surgical interventions were not offered to 19 patients for various reasons as outlined in Table 1.

Table 1. Reasons for exclusion from emergency repair

Number of patients	Reasons
9	Multiple medical comorbidities
5	Moribund status on arrival in ED
4	Suprarenal rupture (2 had previous infrarenal AAA repair)
1	Personally elected not to undergo surgery

ED=emergency department; AAA=aortic abdominal aneurysm.

Forty-two patients had emergency AAA repair but one patient was excluded from the study because the medical records department was unable to retrieve his clinical notes. Thus a total of 41 patients were included in this study.

Demographic and preoperative data

The mean age of the sample population was 72.9 years (range 57–89 years) with a male:female ratio of 4:1. The majority, 80% (33/41) of the patients were of European ethnicity, while the remainders were of Maori (3/41), Pacific Islanders (4/41), and Chinese (1/41) origin.

Self referral to emergency department (ED) accounted for 54% (22/41) of these patients, while 39% (16/41) of patients were urgently referred by their family doctors. One patient was transferred from a peripheral district hospital for further investigation intractable back pain. The source of referral was not documented for two patients. Eighty percent (33/41) of patients were transported to the hospital by ambulance.

Sixty-six percent (27/41) of patients were hypotensive (systolic BP <90 mmHg) on arrival, and 41% (17/41) patients had documented episodes of loss of consciousness (LOC). The average systolic blood pressure on admission was 100 mmHg (ranging from unrecordable to 199 mmHg). Three patients had received cardiopulmonary resuscitation (CPR) in the emergency department. None of these survived the perioperative period. Two died during surgery, while the third succumbed on the first postoperative day.

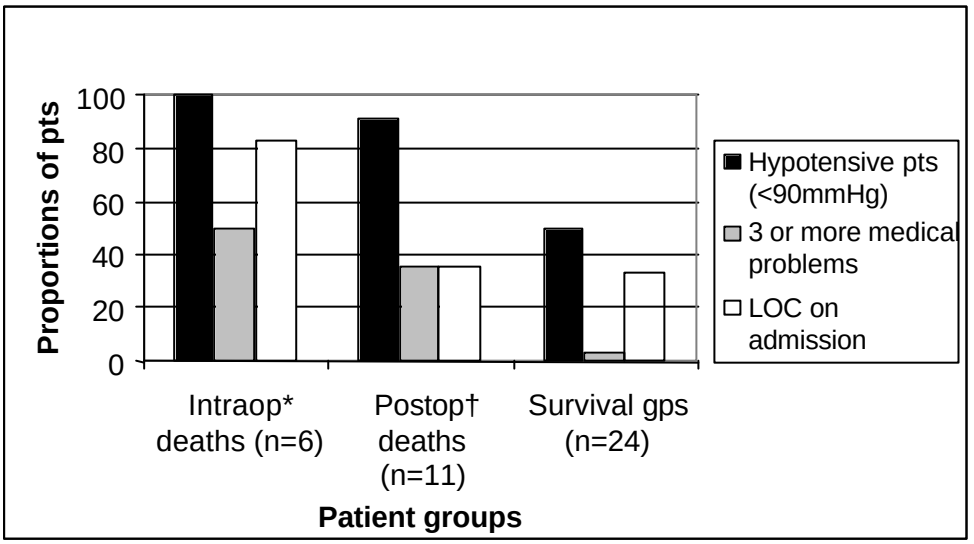
In terms of medical comorbidities, 17% (7/41) of patients had three or more medical illnesses. The mean initial haematocrit and creatinine level were 0.33±0.089 and 0.145±0.084 mmol/L respectively. Blood tests results (both paper and electronic records) were not available for four patients (two died, two survived). Hence, the denominator for calculations of mean values and statistical analyses relating to blood tests results was 37 patients.

Patients were categorised into three groups:

- Intraoperative death.
- Postoperative death.
- Survivors.

Differences in risk factors among the three patient groups are illustrated in Figure 1. All of those patients (100%) who died intraoperatively and 91% of postoperative deaths had preoperative hypotension (systolic blood pressure <90 mmHg), while only 50% of survivors were hypotensive preoperatively. A similar trend of higher incidence of medical comorbidities and documented episodes of LOC were noted in patients who did not survive.

Figure 1. Patient groups characteristics



*Intraoperative; †Postoperative; pts=patients; gps=groups; LOC=loss of consciousness.

Intraoperative data

All 41 patients underwent emergency abdominal aortic aneurysm repair with 85% (35/41) surviving the operation. All procedures were performed through midline laparotomy. The mean duration of operation for all those patients who survived the repair was 3 hours 1 minute±89 minutes. The timing of surgery (day, 0800–1800 or night) was nearly equally divided, 56% of operations occurred at night while remaining 44% of operations were done during the day.

Free intraperitoneal rupture was present in 29% (12/41) of patients. In terms of the neck of the aneurysms, 93% of patients had an infrarenal neck, 5% (2/41) patients had suprarenal aneurysms, and 2% (1/41) patients had a juxtarenal aortic neck. The two patients with suprarenal aneurysms died intraoperatively, while the only patient with juxtarenal aortic neck died postoperatively. Four patients with infrarenal aortic necks required initial suprarenal cross-clamping before identification of an infrarenal aortic neck.

The mean intraoperative blood transfusion requirement was 10 units (range 2 to 50 units). Of those 35 patients who survived the operations, 10 patients experienced prolonged intraoperative hypotension (systolic blood pressure <90 mmHg for more than 20 minutes).

Postoperative data

For the 35 patients who survived operation, the mean intensive care unit (ICU) stay was 6 days (range 1 to 28 days) with a mean duration of postoperative mechanical ventilation of 5 days (range 1 to 26 days). Respiratory failure and sepsis were the most common complications, followed by myocardial events and renal failure. Major postoperative complications are summarised in Table 2. Bleeding was the most common cause of postoperative death (45%), followed by myocardial infarction (36%) and multisystem organ failure (27%). The mean length of hospital stay was 13.5 days (range 1 to 62 days).

Table 2. Postoperative complications

Complications	% of patients
Respiratory failure	31
Sepsis	31
MI/CHF	29
Renal failure	26
Bleeding	20
Tracheostomy	14
Graft Thrombosis	6
Ischaemic Colitis	6
Leg ischaemia	14
Paraplegia/paralysis	3

MI=myocardial infarction; CHF=chronic heart failure

Mortality rates

The overall operative mortality rate was 41%. Six patients (15%) died during the operation and 11 (26%) died postoperatively. Seven of the postoperative deaths occurred within the first 24 hours, with the remainder occurring between 2 and 12 days postoperation.

Univariate relationship between preoperative clinical variables and mortality rates

After univariate analysis, three significant preoperative risk factors ($p<0.05$) for overall death were identified. These risk factors were age, preoperative hypotension (systolic BP <90mmHg), serum creatinine level (≥0.15 mmol/L) and patients with three or more comorbidities (Table 3).

The mortality rates for patients aged 80 years was 75%, significantly higher than for those between 70–79 years and those <70 years (47% and 14% respectively, $p=0.016$). Patients with preoperative hypotension had a mortality of 55%, compared with 14% in normotensive patients ($p=0.01$). The respective mortality rates with regards to creatinine level ≥0.15 vs <0.15: (77% vs 21%, $p=0.0009$), and for 3 or

more medical comorbidities vs <3 co-morbidities: (86% vs 33%, p=0.008), were also statistically significant.

Table 3. Mortality rates

Clinical Variables		Total number of patients N	Mortality rates n (%)
Age*	≥80	8	6 (75%)
(years)	70-79	19	9 (47%)
	<70	14	2 (14%)
BP on admission*	<90	27	15 (55%)
(mmHg)	≥90	14	2 (14%)
Creatinine level*	≥0.15	13	10 (77%)
(mmol/L)	<0.15	24	5 (21%)
Comorbidities*	3 or more	7	6 (86%)
	<3	34	11 (33%)

*p<0.05; BP=blood pressure.

Multivariate analysis

Those significant risk factors identified by univariate analysis were entered into multivariate analysis by logistic regression with death as outcome to predict mortality. As illustrated in Table 4, a creatinine level ≥0.15 mmol/L was the only significant (p<0.05) preoperative risk factor—with an odds ratio of 9.3.

Table 4. Logistic regression

Variable	Odds ratio	95% CI	P value
Creatinine level >0.15	9.3	1.18–73.90	0.03*
Preoperative hypotension	11.8	0.96–146.5	0.06
Age (years)	1.1	0.99–1.30	0.07
3 or more medical problems	3.8	0.25–58.0	0.3

*p<0.05

Conclusions

Emergency repair of ruptured abdominal aortic aneurysm (AAA) remains a challenge to even the most experienced vascular surgeon. AAA has a death rate of 4.1 per 100,000 in New Zealand.[20] In this series, we achieved a 41% overall operative mortality rate. This result is comparable with mortality rates reported by other institutions.[6] A meta-analysis by Bown et al demonstrated that the overall operative mortality rate of ruptured AAA repair from 1955 to 1998 reported in published literatures was approximately 48%.[21]

Some institutions advocate an 'all comers' approach—offering surgery irrespective of patient's presenting status and comorbidities.[22] In our institution, patients were selected for repair after consideration of age, presentation, and medical comorbidities.

In doing so, 19 patients received comfort care only, thus sparing them (and their families) the pain and false hope of unsuccessful surgical intervention. A recent survey showed that 97% of UK vascular surgeons practise a similar selective approach.[23]

Given the high operative mortality rates for ruptured AAA, objective predictors of eventual outcome could assist with both the humane treatment of critically ill elderly patients and the effective delivery of limited healthcare resources. Indeed, the potential of a protracted and undignified death after major surgery should be avoided if there is no reasonable chance of success. Furthermore, costs incurred during prolonged intensive treatment are substantial, and ideally these resources should not be wasted on futile endeavours.

The results in this study suggest that simple preoperative variables may be predictive of overall mortality. Creatinine level ≥0.15mmol/L is multivariately associated with overall mortality. Preoperative hypotension, 3 or more medical comorbidities, and advanced age were only associated with mortality in the univariate analysis. Although this is a retrospective study, these variables were accurately documented in all cases, and our findings are consistent with results reported from other studies.[6,10,12,19] Once the clinical decision is made to proceed with emergency surgery, our institution's policy is to rapidly transfer ruptured AAA patients to the operating theatre without delay for fluid resuscitation (to maintain patient's haemodynamic stability).

Many papers have attempted to identify independent predictors for mortality. A recent report from the Mayo Clinic associated advanced age, high APACHE II score, low initial haematocrit, and preoperative cardiac arrest multivariately with increased mortality rates.[6] Hardman et al reviewed 154 patients and identified 5 independent preoperative risk factors that were associated with mortality: age >76 years, an ischaemic ECG, haemoglobin <9 g/dL, creatinine > 0.19 mmol/L, and loss of consciousness.[12] They also reported that all patients who presented with three or more variables died.[12] In addition, Johnston et al found that hypotensive patients with raised creatinine had only a 20% chance of survival.[24] All of these findings strongly suggest that mortality is determined by the severity of physiological insult and the patient's premorbid physiological reserve.

An increased preoperative serum creatinine level (≥0.15 mmol/L) has been suggested as a predictor for mortality in both ruptured and non-ruptured AAAs.[12,13] As with other organ systems, there is a progressive decrease in the baseline function of the kidney with advancing age. Rowe et al first showed a sequential fall in standardised glomerular filtration rate (GFR) in an aging population.[26]

Excluding individuals with pre-existing renal disease, it is generally accepted that there is a 50% to 63% decline in GFR from the ages of 30 to 80 years.[27] Despite this decrease, the serum creatinine concentration remains within the normal limits in the healthy aged person. This paradox is due to the decrease in the muscle mass that accompanies the ageing process. An increased creatinine level in an elderly patient indicates that this patient has a much lower GFR than in a younger patient with a similar creatinine level. Hence, any small physiological stress could cause acute renal failure in the elderly patients. Although GFR is directly depressed by inhalation anaesthetics[28], the more deleterious effects on the GFR and renal blood flow arise from a decrease in the cardiac output with hypovolaemic shock. The most important

principle in preserving the renal function of elderly surgical patients is to maintain their intravascular volume.[29]

The incidence of AAAs increases with ageing of the population. Patients over 80 years old have been reported to comprise between 3.7% and 13% of those undergoing repair of both ruptured and non-ruptured AAA.[8,9] In our study, 19% of the sample population were aged 80 years or more, although this observation may reflect the small sample size. Old age is known to be associated with an increase in mortality following emergency repair[4]. It was the strongest predictor of survival for 1480 patients who had ruptured AAAs repaired in North Carolina hospitals, where age >65 years was associated with a mortality rate of >50%, which was significantly higher than younger patients.[25] Our study also suggests that age may have an effect on mortality although this trend was not statistically significant.

Despite the findings of our study and others, there is still no consensus on how to use these pre-operative variables. Indeed, there is no universal agreement regarding the use of a selective approach for treating patients with ruptured AAA. Such selective approaches can present a complex moral dilemma for the medical team. While these clinical variables may prove useful, they must be interpreted with caution and should only act as an adjunct to clinical decision making.

The ideal treatment of ruptured AAA is prevention. The implementation of a screening program has been suggested to reduce the incidence of ruptured aneurysms[11] and the mortality rate from aneurysm related causes in men.[15] The cost-effectiveness of such a screening program remains controversial.[16] However, selective screening in younger high-risk patients can be considered for detection and subsequent elective repair of aneurysms. There are two important factors in such screening: (a) very low rate of mortality for elective repair; and (b) younger patients tend to achieve more rapid recovery. The mortality rate for elective repair is 3.4% in New Zealand.[20]

In conclusion, the natural history of an untreated ruptured abdominal aortic aneurysm is invariably death. Early diagnosis, prompt operative treatment, and expert postoperative care are essential to a successful outcome. While it was hoped that this study may produce some useful predictors for mortality within the confinements of a retrospective review, its small sample size may limit the validity of the results, and the ultimate decision to proceed with emergency repair rests with the individual patient and surgeon.

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The role of ERCP in management of retained bile duct stones after laparoscopic cholecystectomy

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Abstract

Background Retained bile duct stones after cholecystectomy are an established entity. To find out the incidence of retained common bile duct (CBD) stones after laparoscopic cholecystectomy (LC) in our hospital, we conducted a retrospective study of patients who presented with symptomatic retained stones in the biliary system after a LC.

Methods Between the period 1992–2000, 824 LCs were performed in our hospital. Twenty-five of these returned to the hospital with symptoms and signs suggestive of CBD stones.

Results Prior to LC, ultrasound scans of all the patients showed gall stones. Alanine transaminase (ALT) was raised in 15 patients. All of these patients underwent LC. On readmission, ALT was raised in 20 patients, bilirubin was raised in 9 patients, and alkaline phosphatase (ALK) was raised in 16 patients. Ultrasound showed common bile duct dilatations in 16 patients, with 6 of these dilated bile ducts having stones. All 25 patients underwent endoscopic retrograde cholangiopancreatography (ERCP)—with successful removal of stones in 16 cases, failure in 5 cases, and no stones in 4 cases. A second ERCP was successful in removing stones in 4 of the 5 failed patients.

Conclusions In our hospital, the incidence of symptomatic retained stones after a LC is about 2.5%. Ultrasound is poor in visualising common bile duct stones although it detects CBD dilatations in majority of cases (76%). ERCP is an effective technique for diagnosis and treatment of retained post-LC stones, with minimum morbidity and no mortality in our small series.

Background

The incidence of choledocolithiasis amongst patients undergoing cholecystectomy is reported to be about 15%.[1] However the retention of CBD stones after open cholecystectomy is reported between 5–15%.[2] Since the acceptance of laparoscopic cholecystectomy (LC) as a gold standard for removing gall bladders the problem of retained biliary stones still persists and an incidence of 0.5–2.3% has been quoted in various series.[3,4] Both the open and laparoscopic methods for exploration of common bile duct carries a mortality and significant morbidity.

Aim and Methodology

The aim of this study was to review the incidence of retained CBD stones in patients who have had a LC done in our hospital. We were also interested in the course, treatment, and eventual outcome of these patients.

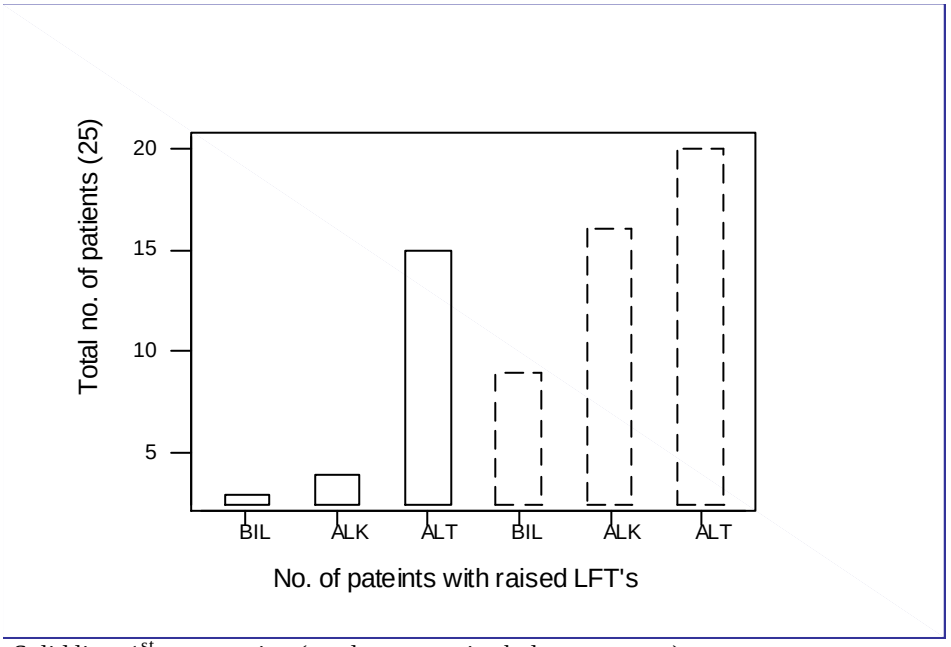
Between the period 1992-2001, 824 LC were done in our hospital. 25 patients returned with subsequent signs and symptoms suggestive of CBD stones. These patients underwent liver function tests (LFT), ultrasound (U/S), and endoscopic retrograde cholangiopancreatography (ERCP). Notes were also reviewed to check the operation note and to compare the original blood tests and the ultrasound scans between the first and the second admission.

Results

The age range was 20-75 years with a mean of 51 years. The male-to-female ratio was 5:20. On the first admission, ultrasound picked up gallstones in all 25 patients. Bilirubin and ALK was raised minimally only in 3 and 4 patients respectively. ALT, however, was raised in 15 patients. ALT is generally not regarded as an important marker for hepatobiliary obstruction.

In our unit, we run a policy of selective intraoperative cholangiography, which is mainly carried out to delineate any abnormal biliary anatomy or when there is a high suspicion of stones in CBD intraoperatively (ie, dilated CBD). All these patients underwent a laparoscopic cholecystectomy. In one case, the dissection was difficult due to dilated cystic duct and intraoperative bleeding, therefore the gall bladder was divided above the Hartman's pouch which was left *in situ.* There were no other complications reported.

Figure 1. Graph depicting a rise in liver function test levels (LFTs). In the post-laparoscopic cholecystectomy group, ALT was raised in 20 patients.



Solid line: 1st presentation (pre-laparoscopic cholecystectomy)
Broken line: 2nd presentation (post-laparoscopic cholecystectomy)
BIL=bilirubin; ALK=alkaline phosphatase; ALT=alanine transaminase

Figure 1 represents the LFTs of these 25 patients on first admission (pre-LC) and on the subsequent admission when retained stones were clinically suspected. The median time to return was 48 weeks (range: 2–364 weeks). The median time was skewed to 48 weeks due to the presence of a few outliers. The main presenting complaints, in majority of these patients were right upper quadrant and epigastric pain. Twenty-three patients had abnormal LFTs, 16 of these 23 patients had dilated CBD on U/S scan. Patients underwent U/S and ERCPs.

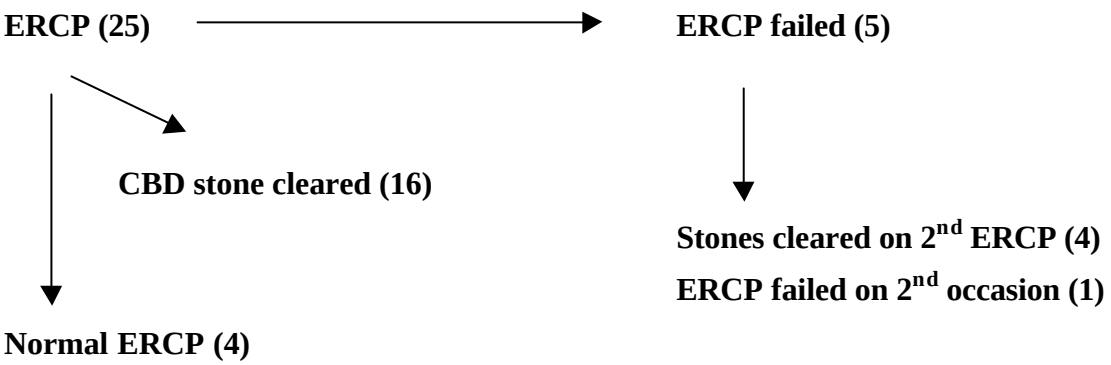
Results in comparison to the initial presentation are presented in Table 1. The result of ERCP is shown in Figure 2. The only patient that failed a 2nd ERCP is currently under follow-up.

Table 1. Ultrasound findings in comparison with endoscopic retrograde cholangiopancreatography (ERCP)

Time of presentation	U/S findings			ERCP findings
	Gall Stones	Dilated CBD without stones	Dilated CBD with stones	Stones in CBD
1st presentation (Pre-LC)	25 (no. of pts)	0	0	ERCP not done
2nd presentation (Post-LC)	0	10	6	21

LC=laparoscopic cholecystectomy; U/S=ultrasound; pts=patients; CBD=common bile duct.

Figure 2. Results of patients after ERCP (number of patients)



Discussion

In our study, the incidence of symptomatic retained common bile duct stones was 2.5%. However we have only taken into account the patients that returned to our hospital with sign and symptoms suggestive of retained stones, therefore the actual percentage could still be higher.

As shown in Figure 1, alanine transaminase (ALT) was raised in both the pre- and post-LC groups. Overall, out of the 25 that returned with a suspicion of stones, 15 had raised ALT pre-operatively. Other studies[5] have also highlighted the importance of ALT in making a diagnosis of retained bile duct stones.

As we cannot comment on the ALT of the patients that did not return with retained stones, our conclusion has to be inferred with some caution. Ultrasound, which is

reliable for diagnosing gall stones, is a poor modality for detecting CBD stones. In our study, U/S detected 6 patients out of 21 (28%) who were proved to have stones later on ERCP. However it has to be emphasised that ultrasound is operator-dependent and in our series, picked up 16 dilatations on readmission.

ERCP has been proven to be a safe and effective method for dealing with retained CBD stones in various studies.[6] However, a mortality rate of less than 1% has been reported.[7] In our series, ERCP successfully dealt with retained CBD stones in 16 patients in the first attempt, and a further 4 were cleared in the 2nd attempt.

Stents were used if the CBD was not fully cleared on the first attempt, which helped with the decompression till the stones were cleared. There was no mortality or significant morbidity recorded as a result of this procedure. Four patients had no stones detected on ERCP. According to the ultrasound scans, all patients had raised LFTs and dilated CBDs so underwent the ERCP procedure. There is a possibility that these patients passed stones spontaneously.

Conclusions

The incidence of retained bile duct stones post-laparoscopic cholecystectomy in our hospital is 2.5%. ALT seems to be a sensitive marker for detection of CBD stones—as 86% of patients with raised ALT in the preoperative group were admitted as retained stones, whereas U/S scans detected stones in only 28% of the cases.

ERCP is very safe and effective in dealing with retained CBD stones; however at present, the availability of the magnetic resonance (MR) cholangiogram has challenged the role of the ERCP procedure (which carries a mortality of 1% in many large series).

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Angiosarcoma in father and son: a case report and literature review

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We describe the case of a father and son, both dying from angiosarcoma. The son died from angiosarcoma of the pleura about 1 year before his father. His father died from metastatic angiosarcoma of the liver and spleen. There was no history of exposure to the known carcinogens for this malignancy, and neither had any of the predisposing chronic medical conditions.

We believe that this was a case of primary hereditary angiosarcoma, which claimed the son before the father.

Case report

A 69-year-old man was admitted with a 2-week history of abdominal bloating and a dull ache in the left flank. He denied any other symptoms. There was no history of anorexia or weight loss. Twenty-five years earlier he had surgery for carcinoma of the bowel; and 5 years after that, he had a repeat laparotomy for adhesiolysis. He had a recent right inguinal hernia repair, a hiatus hernia, and Barrett's oesophagus. He had smoked for 30 years and had a morning cough productive of white phlegm.

On examination, he had a distended abdomen with scars from his previous laparotomies. There was ascites, and the spleen was enlarged and tender. Routine haematology and biochemistry were normal (apart from raised liver function enzymes).

A computed tomography (CT) scan of the abdomen showed diffuse infiltration of both the liver and spleen, with low attenuation lesions consistent with metastatic deposits. A peritoneal tap showed haemorrhagic ascites. Cytology did not show any malignant cells. In view of his previous history of carcinoma of the bowel, he had upper and lower bowel endoscopies, which were normal. At laparoscopy multiple cystic nodules were seen in the liver and splenic hilum. A wedge biopsy from the liver was taken, the ascitic fluid tapped, and a drain left *in situ.*

His Casoni test and hepatitis screen were negative. Serum ferritin was normal. Alpha fetoprotein and anti-mitochondrial antibodies were not detected. Histology of the liver was consistent with angiosarcoma.

The patient had no previous exposure to Thorotrast®, vinyl chloride, arsenic, anabolic steroids, or radiation (which are known pre-carcinogens for this malignancy)—but he did say that his son had died the previous year from angiosarcoma of the pleura. Detailed history, clinical examination and preliminary X-rays and lab tests also did not show any evidence of predisposing hereditary syndromes to this condition.

Once the diagnosis was made, further scans for certain syndromes (such as Von Hippel Landau disease) were not performed as it would not have affected his management. In addition, the patient requested palliative care only.

He was diagnosed as a case of metastatic angiosarcoma with liver as the more likely primary site, and transferred to a hospice for palliative care. He died 2 months after the diagnosis.

Discussion

Angiosarcoma is responsible for about 2% of primary liver tumors,[1] and is considered to be the most common of the mesenchymal liver tumours. About 25 cases occur each year in the United States. Males (ratio 3:1) in their fifth or sixth decade are most often affected[2] but it can also occur in children.[3,4] It often presents with abdominal discomfort and distension, weight loss, and fatigue. On examination, the patient may have jaundice, hepatomegaly, and ascites.[5] Liver function tests are usually abnormal. Thrombocytopenia, microangiopathic haemolytic anaemia, and disseminated intravascular coagulation may also be present.[6] There are no tumour markers.

CT images classically show multiple hypodense areas. After contrast, the lesions become partly or completely isodense with the normal hepatic tissue.[7] On T2 weighted MRI imaging, there are areas of high signals with central regions of low signals. Liver biopsy is hazardous as it may cause severe haemorrhage.[8] Macroscopically, 'blood lakes' may be seen as the tumour is angioinvasive. On histological examination, there are typical spindle shaped hyperchromatic cells with nucleoli, which on immunostaining are positive for endothelial cells markers.[9]

More than 30% of cases has been linked to exposure to environmental agents including Thorotrast® which was used as a radiological contrast agent in the past. Evidence of previous exposure is seen on a CT scan, and the latency period can be longer than 30 years.[10,11]

Angiosarcoma is also associated with exposure to vinyl chloride, which is used in rubber and plastic processing.[12,13] Again the latency period can be very long. The risk ratio is 400:1 compared to the general population[14] and may be due to increased frequency of P53 mutations in these people.[15] The incidence in this group has decreased since the acceptable exposure level to vinyl chloride has been reduced.

Angiosarcoma has also been reported to be associated with exposure to arsenic,[16] anabolic steroids,[17] and oral contraceptives.[18] It has been reported in a previous haemangioma,[19] with Von Recklinghausen's neurofibromatosis,[20] and in patients with certain hereditary conditions such as congenital hereditary lymphoedema (Milroy's disease)[21] and Von Hippel Landau disease.[22] The condition is invariably fatal and is unresponsive to surgery, chemotherapy, radiotherapy, or transplantation.

Detailed history from our patient failed to reveal previous exposure to any of the known carcinogens or predisposing medical conditions. Our case is the first report of primary angiosarcoma in a parent and a sibling. The primary sites involved were different in the father (probably liver) and son (pleura).

To our knowledge, the only other case of primary angiosarcoma in two members of the same family were in two brothers who developed primary renal angiosarcoma of the kidney.[23] As it is a very rare tumour in the general population, the son may have inherited a genetic defect from the father. No other cases were reported in over three generations after tracing the family pedigree.

Another less likely possibility is that both the father and son may have been exposed to some unknown carcinogen.

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Stapled haemorrhoidectomy—no pain, no gain?

Andrew Hill

Abstract

Stapled anopexy/haemorrhoidectomy (SH) was introduced in 1993 and first described by Longo in 1998. In New Zealand, more than 700 stapled haemorrhoidectomies have been performed. The procedure is one of the most studied of all recent new surgical technologies, and the literature is surveyed in this paper to assess the procedure's safety and efficacy.

From review of the current literature is seems appropriate to conclude that SH is a safe procedure. It is probably not the answer for all haemorrhoids, especially those that are extremely large or are associated with a very significant external component. The procedure certainly has a sound theoretical basis and is likely here to stay. Patients like it because it is less painful than conventional techniques but they need to be counselled that its durability is not known.

Stapled anopexy/haemorrhoidectomy (SH) was introduced in 1993 and first described by Longo in 1998.[1] In New Zealand, over 700 stapled haemorrhoidectomies have been performed and at the procedure's 10th anniversary it seems timely to review the literature on this procedure. The literature is substantial and this procedure is one of the most studied of all recent new surgical technologies.

The rationale of SH is that haemorrhoids are a result of fragmentation of Park's ligament; this results in submucosal tissue that lines the anal canal, along with the anal mucosa, sliding downwards. This prolapse obstructs venous outflow hence causing the clinical entity known as haemorrhoids.[2] Hence the rationale of excising a ring of rectal mucosa, thereby reducing the mucosal prolapse. Thus the operation is perhaps better known as a stapled rectal mucosectomy.[3] The operation has been given several other names including stapled anoplasty, stapled circumferential mucosectomy, Longo's haemorrhoidectomy, stapled anopexy, stapled prolapsectomy, and stapled haemorrhoidopexy.[4–9]

Many studies have looked at the efficacy and safety of the procedure. Several disturbing cases of serious complications have also been reported. Overall, however, the procedure seems safe and well-tolerated, and appears to be effective—at least in the short term. Should New Zealand surgeons be performing this procedure? Certainly the procedure has proven popular with several surgeons in New Zealand but has yet to gain general acceptance. Several significant arguments have been raised against the use of the post partum haemorrhage (PPH) device. These arguments can be divided into four groups:

- The first group of arguments concern the **validity of the trial**s themselves.
- The second group of arguments concern the **safety** of the procedure.
- The third group of arguments concern the **cost**.
- The fourth group of arguments concern the **efficacy and durability** of the procedure.

The validity of the trials

A significant issue has been the large number of patients accrued in the trials. Most colorectal surgeons only operate on a few haemorrhoid patients per year, as the majority of haemorrhoid patients can be dealt with in the outpatient clinic using rubber-band ligation or injection sclerotherapy.[10] Thus, where do the trials get all their patients from? Are they operating on people that would be better dealt with by non-operative measures? A study from Singapore, which compared SH and rubber-band ligation for Grade III and small Grade IV haemorrhoids, supports this theory.[11] Apart from studies from Singapore, the majority of the larger studies are multicentre. All other studies are relatively small.[12,13]

Is stapled haemorrhoidectomy safe?

Apart from a few isolated severe complications, the SH-operation complication rates are comparable to other conventional haemorrhoid (CH) operations.[14–16] In a large multicentre study from Italy there was a complication rate of 15%. The commonest complications were severe pain and bleeding, each at 5% or less.[17]

Interestingly, in this study, 65% of complications occurred after the surgeon had done 25 or more cases—suggesting that the learning curve phenomenon does not apply. Septic complications in this study, and others, have been very rare. Bacteraemia after haemorrhoidectomy is more common with SH rather than after CH but this does not seem to have clinical relevance.[18]

Very little work has been done on anorectal physiology after SH. In a study (from the Middle East) it was shown that anorectal pressures were decreased after CH but not after SH—but this did not translate into clinical benefit.[19] However, in Italian[20] and UK[21] studies, the differences in resting and squeeze pressures were not confirmed. Long-term follow-up will be important as with CH impairment of anal continence that the patient relates to the operation is as high as 29% at long-term follow-up.[22]

Despite the large amount of work done on efficacy and safety, the procedure has not been adopted widely in the United States or Canada with only a few reports coming from North America.[23]

Cost

The PPH instrument (from Ethicon Ltd) is expensive. SH is consistently faster to perform than CH, but this is unlikely to offset the cost of the instrument.[19,24] No studies are available looking at costs, but this needs to be done if the operation is to be accepted universally.

Efficacy and durability

SH is at least as good as conventional haemorrhoidectomy and is less painful.[25] Some have been skeptical—and in a recent paper from the UK studying a small group of patients, it was shown that while the SH is less painful, it was not associated with an earlier return to work than CH, and it failed to deal with external haemorrhoids.[26]

On the other hand, in the Middle East, a dramatic improvement in return to full activity was shown.[19] Other studies have raised questions about the long-term results

of the procedure despite the initial results being promising.[6,13] In one study from the UK, good results have persisted up until 33 months.[27]

Conclusions

A recent systematic review from Australia has concluded that SH is probably at least as safe as CH.[28] However they also concluded that the long-term outcome for the procedure has not been determined, and that studies of long-term outcome need to be performed before the procedure is adopted more widely.

What does the New Zealand general surgeon make of all of these data? Currently it can be justifiably concluded that SH is a safe procedure. It is probably not the answer for all haemorrhoids, especially those that are extremely large or are associated with a very significant external component. The procedure certainly has a sound theoretical basis and is probably here to stay. Patients like it because it is less painful than conventional techniques but they need to be counselled that its durability is not known.

Is this a case of 'no pain–no gain'? Probably not. In our search for painless effective treatment for haemorrhoids (that are unresponsive to non-operative measures), this is one important step towards that goal.

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THE NEW ZEALAND MEDICAL JOURNAL
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The birth of the New Zealand Dental Association

This Editorial Note was published in the New Zealand Medical Journal 1905, Volume 14(17&18), p281.

We are glad to welcome the birth of the New Zealand Dental Association, and to congratulate it on the evidence of vigorous vitality which it has put forward in the shape of a very successful congress held in Wellington in June, 1905, and in the form of a very attractive journal. There is no necessity in this place to labour the advantages of such an association. It is bound to make for efficiency of its members and for the security of their best interests.

It is very true, as Mr. F. W. Thompson pointed out at the congress, that "dentistry to-day is something more than mere tooth-extraction; it is an exact science only to be practised successfully by men of culture and learning."

We believe that dental practice has already reached a very high standard in New Zealand, but, at the same time, we are of opinion that New Zealand offers special facilities for research in dental science. It has been stated that dental caries is more prevalent in New Zealand than in other countries, and that the age-incidence of the complaint is more serious. If this be the case, then New Zealand should offer a specially suitable field for inquiry into the causation of the disease.

There is a movement at the present day towards regular medical inspection of schools, and one of the leading features of such an inspection will consist in an examination of the teeth of the children. This will, no doubt, lead to the discovery of many remediable defects, but it appears to us desirable to go a step further back, and to institute a system of preventive dentistry. Napoleon is credited with the statement that his army marched on its stomach; and in the same way the progress of a nation must depend in no trifling degree on the efficiency of its digestive apparatus. "Efficiency" must be the watchword of modern nations; and the enormous dissipation of energy which results from minor and chronic forms of indigestion hardly makes for efficiency.

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Pneumoconiosis and a pulsatile mass

A 76-year-old man with occupational lung disease, asthma, and bronchitis was noted to have an abdominal aortic aneurysm 6 cm in diameter as shown in the aortogram (Figure 1). This was treated percutaneously, and a plain abdominal radiograph after treatment is shown (Figure 2).

Figure 1

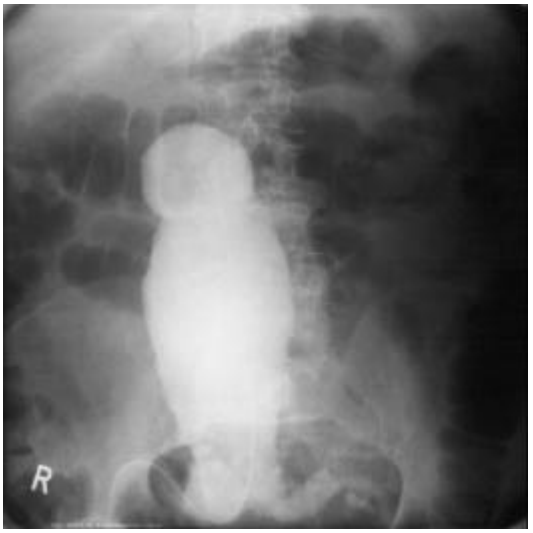
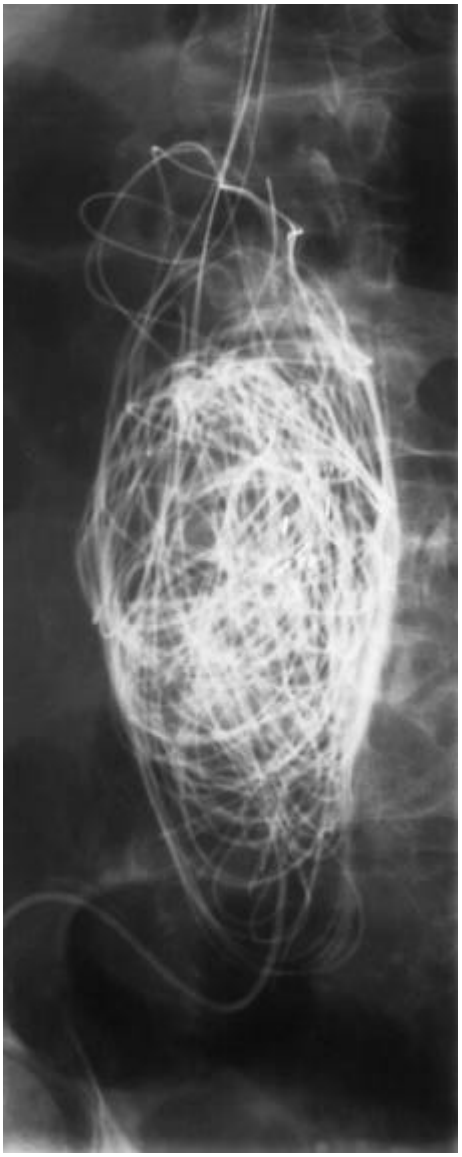


Figure 2



Question

What is the treatment, and why was it thought to be effective?

See the next page for the answer

Answer

Introducing a long radiological guide wire into an aneurysm sac for those patients deemed inoperable due to comorbidity was a practice in vogue in the 1960s. The aim of the treatment was to thrombose the aneurysm sac—except for a small central lumen, which would be preserved to allow perfusion of the lower body. This thrombus was thought to be protective against rupture.

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Gulf War Syndrome

Up to 20% of the 700 000 U.S. troops who were in the Persian Gulf in 1990–1991 have reported debilitating symptoms that as yet have no definitive explanation. The most common are fatigue, musculoskeletal pain, and neurocognitive problems. This complex of symptoms is clinically indistinguishable from that of the chronic fatigue syndrome fibromyalgia, and the resulting disorders have been called Gulf War veterans' illnesses (GWVIs).

It has been hypothesized that certain *Mycoplasma* species may have caused this problem and this led to a randomized, double-blind, placebo-controlled trial with 12 months of treatment with 200 mg of doxycycline daily. Adherence to treatment after 6 months was poor and treatment with doxycycline did not improve outcomes of GWVIs at 1 year.

Ann Intern Med 2004:141:85–94.

Doped athletes

Supporters of athletes who received steroids under East Germany's state-controlled doping programme are turning up the heat on the pharmaceutical company alleged to have supplied the drugs. The extent of doping is unknown, but thousands of athletes are thought to have been given muscle-boosting steroids during the 1970s and 1980s. Some, such as Olympic long-jumper and sprinter Heike Drechsler, say they were told these were vitamins.

Pressure on Jenapharm, based in Jena, Thuringen, has been mounting since July 2003, when a former employee alleged on television that the company supplied sports scientists in the German Democratic Republic (GDR) with substances used in the doping programme. Jenapharm's management disputes the allegations, saying the company produced the drugs for medical purposes and was not involved in doping studies.

I don't believe either of them!

Nature 2004;430(12 August):713.

Tobacco and the lungs

The association between cigarette smoking and chronic obstructive pulmonary disease (COPD) has been well established. But for unknown reasons only 15 to 20% of smokers acquire COPD.

In a recent paper from Germany it has been shown that COPD subjects have a significantly higher production of tumour necrosis factor alpha than smokers without airflow limitation. This results in increased bronchial activity with more neutrophils and secretions. Not too surprising really.

Chest 2004;125:1706–13.

Across the ditch

The Medical Journal of Australia became a nonagenarian in July and this has caused some editorial navel gazing. And there have been some outstanding papers to rejoice over. These include *"Q" fever a new fever entity: clinical features, diagnosis and laboratory investigation* in 1937, and *Congenital defects in infants following infectious diseases during pregnancy* in 1943.

Cade's 1949 paper on *Lithium salts in the treatment of psychotic excitement* attracted well justified international attention. Furthermore, the 1985 paper of Marshall and colleagues on *Pyloric campylobacter infection and gastroduodenal disease* has caused a quantum leap in the management of upper gastrointestinal diseases.

Long live the MJA!

Med J Aust 2004;181:9–12.

Out with the baby and the bathwater?

Reduction of information overload and replacement of traditional "didactic" teaching with problem based learning are two key features of the undergraduate curriculum reforms in the UK (and NZ). This will result in students having control over their own learning and make undergraduate training a platform for lifelong learning.

Pious intentions. The Dean of the Bristol Medical School, and a 4th year student, contest the reforms. They point out that there is no evidence that the "new" strategies will produce better doctors, and a risk that students with inadequate knowledge will become poor clinicians. They conclude that a rigorous comparison of "traditional" versus "new" curriculums is urgently needed to determine the best strategy for training doctors. A conclusion which appeals to Methuselah.

BMJ 2004:329:92–4.

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The bioavailability of coenzyme Q_{10} supplements available in New Zealand differs markedly

In New Zealand, at least 10 brands of coenzyme Q_{10} (CoQ_{10}) supplement are available over the counter from health food shops, pharmacies, and the Internet. These products claim that supplementation with coenzyme Q_{10} increases energy, wellbeing, stamina and muscle performance, strengthens the heart, and scavenges free radicals. The evidence for these effects is equivocal and well-controlled studies are needed. It is also necessary to confirm the bioavailability of the available CoQ_{10} supplements.

Coenzyme Q_{10} is an essential cofactor in the mitochondrial electron transport chain and also acts as an antioxidant, sparing, the α-tocopheroxyl radical.[1] In mammals, CoQ_{10} is synthesised in all cells—and the diet is also a source, with meat being the biggest contributor.[2]

It is unlikely that many healthy New Zealand adults are frankly deficient in CoQ_{10}, but CoQ_{10} deficiency has been associated with various diseases including Alzheimer's disease and Parkinson's disease. It is also possible that diseases producing oxidative stress may result in CoQ_{10} depletion. HMG-CoA reductase inhibitor (statin) therapy also decreases CoQ_{10} synthesis[3] and causes a potential CoQ_{10} deficiency, due to inhibition of the common biosynthetic pathway for cholesterol and CoQ_{10}. Thus, CoQ_{10} is relevant to at least 100,000 New Zealand patients currently on statin therapy.

The available CoQ_{10} supplements have different formulations, which may affect absorption.[4–6] In particular, supplements in which CoQ_{10} is dispersed in oil generally have higher bioavailability than those formulated as dry powder tablets.[4,6]

Therefore we have compared the bioavailability of seven different coenzyme Q_{10} supplement brands, and provide a basis for selecting brand(s) for clinical use.

Ten healthy adult male volunteers were enrolled in a study approved by the Canterbury Ethics Committee. Participants were excluded if they had taken CoQ_{10}, any vitamin supplements, or medications within the previous 4 weeks. The mean age was 24.2 years (range 21–28 years), the mean height was 179.8 cm (range 173–187 cm), and the mean weight was 71.8 kg (range 60–100 kg). The study was completed between November 2003 and January 2004.

Baseline blood samples were obtained after a 10-hour overnight fast, and CoQ_{10} supplements were administered as a single nominal dose of 150 mg, with supplement brands given in a different randomised order for each participant and a 1-week washout period between trial days. After administration of the supplement, a standardised vegetarian breakfast and lunch were provided, containing approximately 3 μg of coenzyme Q_{10}.[2] Lunch was provided as a takeaway package, and participants were permitted to leave the study centre after breakfast. A second blood sample was collected after 6 hours.

The brands investigated were selected because they are 'popular' brands that contain differing excipients and are outlined in Table 1, which also shows the measured CoQ_{10} content (n=6 capsules or tablets).

Table 1. The excipients, formulation, and actual CoQ_{10} content of the seven CoQ_{10} supplement brands investigated for bioavailability

Brand	Excipients	Capsule/tablet type	% Yield CoQ_{10} per capsule/tablet
Q-Gel	Vitamin E, Annato seed extract, Biosolv® base (lecithin, polysorbate, sorbitin monoleate, and medium chain triglycerides)	Softules containing liquid dispersion	137 ± 4
Radiance	Rice bran oil, lecithin, selenium, and vitamin E	Softgels containing liquid dispersion	125 ± 4
Blackmores	Soy lecithin	Capsules containing liquid dispersion	121 ± 8
Solgar	Vegetable cellulose, vegetable magnesium stearate, and silica	Vegetable capsules containing dry powder	130 ± 15
Kordel's	Evening primrose oil and salmon oil	Capsules containing liquid dispersion	127 ± 7
Thompson's	Vegetable oil	Vegetarian capsules containing liquid dispersion	121 ± 6
Good Health	Glucose, sucrose, magnesium stearate, calcium phosphate, and natural orange flavour	Chewable tablets	100 ± 7

Table 2. The median change in CoQ_{10} after supplementation with the different brands

Brand	Total CoQ_{10} (µmol/L)	CoQ_{10} to LDL cholesterol ratio (µmol/L)	CoQ_{10} to total cholesterol ratio (µmol/L)
Q-Gel	0.586 (0.349–1.424)	0.275 (0.218–0.500)	0.125 (0.100–0.225)
Radiance	0.321 (0.218–1.118)	0.140 (0.120–0.373)	0.065 (0.048–0.210)
Blackmores	0.229 (0.109–0.531)	0.130 (0.072–0.253)	0.045 (0.028–0.090)
Solgar	0.203 (0.094–0.295)	0.075 (0.048–0.188)	0.045 (0.020–0.075)
Kordel's	0.177 (0.102–0.274)	0.075 (0.050–0.173)	0.040 (0.018–0.078)
Thompson's	0.173 (0.106–0.442)	0.080 (0.060–0.150)	0.028 (0.060–0.073)
Good Health	0.139 (0.105–0.297)	0.095 (0.040–0.165)	0.040 (0.018–0.053)

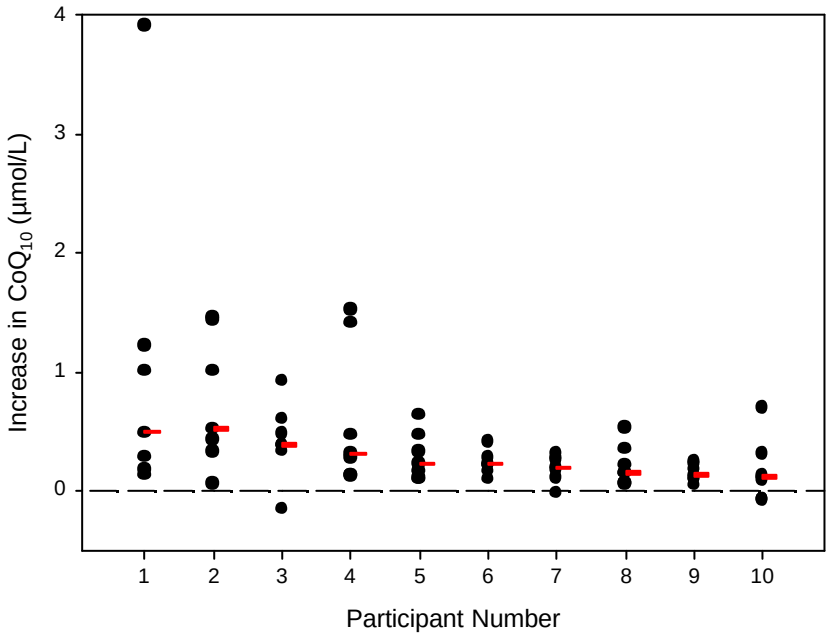
Values shown are median values, with brackets showing the inter-quartile range.

Blood specimens were collected and lithium heparin plasma was stored at -80°C until analysis. CoQ_{10} was analysed using a method similar to that used by Tang et al.[7] The within- and between-run coefficients of variation (CV) for the CoQ_{10} assay are approximately 3.3%. Plasma lipids were determined by routine clinical methods. The differences between CoQ_{10} supplements were tested using either the non-parametric Friedman test or Wilcoxon signed-rank test (as appropriate), with statistical significance inferred when $p<0.05$. All CoQ_{10} supplement brands tested contained at least the claimed CoQ_{10} (Table 2).

Mean baseline lipids (±SD) for all participants were 4.81±1.04, 2.78±0.75, 1.18±0.30, and 1.28±0.44 mmol/L for total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides respectively. Mean baseline CoQ_{10} (±SD) was 0.85±0.25 µmol/L. During the trial there was no significant change in baseline levels of CoQ_{10}, direct LDL cholesterol, HDL cholesterol, triglycerides, or total cholesterol, thus confirming that the wash-out period was sufficient.

There was no significant effect of CoQ_{10} supplementation on total cholesterol (p=0.539), triglycerides (p=0.128), or direct LDL and HDL cholesterol (p=0.910 and 0.587 respectively).

Figure 1: The change in coenzyme Q_{10} concentration for individual participants and all supplement brands (n=7). Horizontal lines show median increase in CoQ_{10} for each participant



There was a significant difference (p=0.003) in CoQ_{10} absorption between the 10 participants (Figure 1). Some participants efficiently absorbed CoQ_{10} from most

brands of the supplements, while others showed inefficient absorption. There was no correlation (p=0.56) between baseline CoQ_{10} levels and absorption of CoQ_{10}.

There was a significant difference in bioavailability between the seven CoQ_{10} brands (p<0.001), with Q-Gel being significantly better than any other supplement (p=0.013). This is summarised in Table 2.

There was a significant difference in the delta CoQ_{10} to direct LDL cholesterol and CoQ_{10} to total cholesterol ratios between the supplement brands (p=0.001 for both), thus mirroring the differences in total CoQ_{10} (Table 2).

There was a significant correlation between baseline LDL concentrations and change in CoQ_{10} (p=0.004; R=+0.343), between total cholesterol levels and change in CoQ_{10} (p=0.004; R=+0.338), and also between baseline triglycerides and change in CoQ_{10} (p=0.035; R=+0.253). Therefore, higher LDL cholesterol or triglyceride concentrations may aid absorption of CoQ_{10}. There was no correlation between HDL cholesterol, weight, or body mass index and mean CoQ_{10} absorption.

Although there are many different CoQ_{10} supplements available, there are little data on the prevalence and effect(s) of CoQ_{10} deficiency, or the benefits of CoQ_{10} supplementation. It is also necessary to confirm that the available CoQ_{10} supplements do in fact increase CoQ_{10} levels before advocating clinical use or attempting clinical trials.

There is also controversy about whether plasma (and hence dietary) CoQ_{10} is delivered to the mitochondria, but it is much easier to measure plasma levels than tissue level, and it is often assumed that tissue levels mirror those of plasma. However, Niklowitz et al[8] found a positive correlation between CoQ_{10} in plasma and platelets, which contain mitochondria, implying that raising plasma levels of CoQ_{10} by diet and supplementation also raises tissue levels.

CoQ_{10} supplementation is well tolerated and dosages as high as 1200 mg/day have been administered with minimal side effects.[9]

We found important differences in the bioavailability of these supplements. However, the mean increase in plasma total CoQ_{10} of 0.41 µmol/L equates to about 0.7 mg of CoQ_{10} being absorbed into the blood from the 150 mg supplied. This can be compared to the normal diet in which CoQ_{10} is limited to about 3 to 5 mg per day, mainly via the consumption of meats rather than fruits and vegetables.[2]

Because CoQ_{10} is lipid soluble, it is likely that administration as a dispersion (or solubilised) in oil will aid absorption, as found in our study. The high bioavailability of Q-Gel compared to other coenzyme Q_{10} supplement brands supports the findings of Chopra et al[5] who found the absorption of Q-Gel to be 319% better than that from a standard softgel capsule containing Q_{10} in oil, after 3 weeks of a daily 120 mg dose. Chopra et al[5] also found the absorption from powder-filled hardshell capsules and powder-based tablets to be higher (125% and 128% respectively) than that from a standard softgel capsule.

Miles et al[4] found the increase of plasma total CoQ_{10} by 'solubilised' supplemental CoQ_{10} to be 858%–1058% higher than that from a dry powder formulation. Furthermore, Wahlqvist et al[6] found that the bioavailability of CoQ_{10} in a complex micelle emulsion (in a soft gelatine capsule) was 927% higher than a crystalline

CoQ_{10} supplement with magnesium stearate (as an excipient and a hard gelatine capsule).

Thus it is clear that there are important differences. There is at least a four-fold variation in the increase in plasma CoQ_{10} achieved by different supplements, and some people get no increase when they take the less effective supplements at typical doses.

The high bioavailability of Q-Gel may be due to the presence of both non-ionic surfactants and the natural surfactant lecithin. The Radiance and Blackmores brands showed the next highest bioavailability, and these brands also contain lecithin.

Significant between subject differences in absorption have been previously reported,[10–12] and highlight a need for monitoring of CoQ_{10} levels during supplementation. There was no correlation between weight, body mass index or baseline CoQ_{10} and CoQ_{10} absorption—hence there are no simple clinical indicators that accurately predict response to different supplement formulations. Therefore, monitoring of plasma CoQ_{10} concentration appears to be the only method to estimate CoQ_{10} absorption.

There are important differences in bioavailability between the available CoQ_{10} supplements and also significant inter-individual differences. We therefore recommend monitoring of plasma CoQ_{10} levels during supplementation, and that differences in bioavailability are considered when selecting a supplement. In this study, the Q-gel brand showed significantly better bioavailability than the six other CoQ_{10} supplements tested.

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Performance indicators: primary health, secondary care, and diabetes

Rigorously measured outcomes are scarce in local healthcare systems, especially for cost-effectiveness in prevention of common disorders.

A seminal paper by Crampton and colleagues (published in the 2 April 2004 issue of the NZMJ)[1] is valuable—although it went largely unnoticed. It thoroughly canvassed important criteria for primary care indicators. Diabetes is used as an example. Diabetes also crosses into a secondary care prevention indicator of high-cost 'open-ended' hospital admissions being an objective international indicator.[2]

There are other compelling reasons why diabetes is New Zealand's best performance indicator. For example, in USA a well as New Zealand, diabetes is ranked first because of its killing propensities.[3] In addition, in a New Zealand survey, 210,000 diabetes patients and their families are involved with 14% of the overall hospital costs.

Prevention and better quality care could reduce these costly hospitalisations[5,6] and half of the cases of kidney failure and dialysis could be prevented,[4,5] by better use of diabetes performance indicators. The same applies for coronary disease, blindness, and amputations if there was more investment in moving upstream.[3,7]

Insulin resistance and pre-diabetes may be present for 20 years before a diabetes diagnosis is made—often itself delayed 12 years or more. Upstream screening of blood sugars thus becomes a key indicator in a common disorder.[8]

Eight percent of adults had undiagnosed diabetes in the Ausdiab Study; and unknown rates of diabetes will prove to be an unsustainable cost in New Zealand.[9] Raised blood glucose is also a key indicator for morbidity, and all cause mortality as well as the earliest predictor of preventable cardiovascular diseases.[10]

New Zealand's yearly 'get checked' programme (3 years' stored results) yields a unique continuing data set. Those persons with diabetes want these 12 digitally transmissible items used.

Health systems should maintain wellness in the clash between financially imposed environments and our 'hunter-gatherer' genes. Diabetes can be used (via these health systems) as an ideal performance indicator of accomplishment spanning primary, secondary, and tertiary preventions—and to monitor social community and educational inputs to health preservation.

Crampton and colleagues clearly listed four excellent key performance indicator uses. A fifth would be the *educative function of encrypted peer-reviewed rankings from the 'get checked' results* thus allowing improvement and changes to practice.

Indeed, with 40 patients per doctor in New Zealand, diabetes is an invaluable and unique performance indicator.

Don W Beaven
Patron

Murray Dear
President

Sarah Thomson
CEO

Diabetes New Zealand Inc

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Sport and Alcohol: Understanding the Mix (Conference)

The Centre for Studies in Sport and Exercise at Massey University are running a conference entitled *'Sport and Alcohol: Understanding the Mix'* on February 8–10, 2005, in Palmerston North, New Zealand. The Conference is a joint undertaking between Massey staff involved in sport and exercise teaching across the colleges of Business, Science and Education.

The Conference will critically analyse and debate the relationship between New Zealand sport and alcohol especially in relation to:

- Social issues (eg, youth, gender, culture, socialisation)
- Health issues (social marketing, holistic health issues)
- Performance issues (the effects of alcohol on sport performance)
- Business issues (sponsorship, management, marketing, legal issues, event management, media)

Speakers include:

- *Dave Currie* – NZ Olympic team Chef De Mission
- *Professor Wray Vamplew* – Researcher from Stirling University in Scotland
- *Greg Cox* – Australian Institute of Sport
- *Professor David Gerrard* – Scholar and former NZ Olympic team Chef De Mission
- *Andrew Martin* – Former All Black Manager
- *Glenda Hughes* – Sports Agent and former sport manager
- *Professor Gary Hermansson* – NZ Olympic team sport psychologist
- *Andrew Dawson* – Sydney Olympic Stadium Manager
- *Dr Farah Palmer* – Scholar and dual World Cup Winning Captain of Black Ferns
- *Graham Seatter* – Commonwealth Games Athlete/Coach and Lion Nathan Sponsorship Director
- *Norm Hewitt* – Former All Black
- *Doug Rollerson* – Former All Black and North Harbour Rugby CEO
- *Hugh McGahan* – Kiwi Rugby League Great and former administrator
- Representatives from other relevant groups like Alcohol and Liquor Advisory Council (ALAC), the New Zealand Rugby Union (NZRU), and Lion Nathan.

A light-hearted debate between high-profile athletes, sports management personnel and media personalities is being organised for the conference dinner.

Early Bird (3-day conference registration) Cost: NZ$570 GST inclusive (before December 15, 2004).

Full Cost: NZ$680 GST inclusive (After December 15th, 2004).

Single Day Registrations: NZ$300 GST inclusive.

Group registration discounts will be negotiated depending upon specific details.

For more general and registration information please check out our website: http://www.sport-alcohol.co.nz

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The Royal Australasian College *of* Physicians

Written Examination 2005

Tuesday 1 March 2005

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