CONTENTS

This Issue in the Journal

4 A summary of the original articles featured in this issue

Editorials

6 The Cartwright legacy: shifting the focus of attention from the doctor to the patient
Ron Paterson

11 The missing manuscript
Frank A Frizelle

14 A response to criticisms of The History of the 'Unfortunate Experiment' at National Women's Hospital
Linda Bryder

22 Acute coronary syndromes in New Zealand hospitals
Robin Norris

Original Articles

25 Patients admitted with an acute coronary syndrome (ACS) in New Zealand in 2007: results of a second comprehensive nationwide audit and a comparison with the first audit from 2002
Chris Ellis, Greg Gamble, Andrew Hamer, Michael Williams, Philip Matsis, John Elliott, Gerard Devlin, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

44 ACS patients in New Zealand experience significant delays to access cardiac investigations and revascularisation treatment especially when admitted to non-interventional centres: results of the second comprehensive national audit of ACS patients
Chris Ellis, Gerard Devlin, John Elliott, Philip Matsis, Michael Williams, Greg Gamble, Andrew Hamer, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

61 A programme of Enhanced Recovery After Surgery (ERAS) is a cost-effective intervention in elective colonic surgery
Tarik Sammour, Kamran Zargar-Shoshtari, Abhijith Bhat, Arman Kahokehr, Andrew G Hill
Audit of cervical screening in women with HIV infection in the Auckland and Northland regions of New Zealand

Jasmin Grewal, Michele Lowe, Hilary Gerrard, Rebecca Henley, Nicky Perkins, Simon Briggs

Clinical Correspondence

Homozgyous familial hypercholesterolaemia and treatment by LDL apheresis

Richard I King, Russell S Scott, Christopher M Florkowski, Andrew D Laurie, Nicola Reid, Peter M George

Angiomatosis: a case report

Hamesh Jina, Philippa Mercer, Malcolm Gordon, Harsh Singh, Martin Whitehead

A case of intestinal-type gastric adenocarcinoma metastatic to a caecal tubulovillous polyp

James McKay

Medical image. Refractory ascites due to ascites praecox

Pazhanivel Mohan, Jayanthy Venkataraman

100 Years Ago in the NZMJ

Report of St Helen’s Hospital, Dunedin

Medical History

Equipment of Yore: a contribution from the Cotter Medical History Trust

Max Abernethy

Proceedings

Proceedings of the Health Research Society of Canterbury Seminar Series, Friday 16 July 2010

Methuselah

Selected excerpts from Methuselah

Letters

The 1987 National Women’s Hospital (NWH) ‘Unfortunate Experiment’. Accusations of unethical experiments and undertreatment, resulting in excess deaths from cervical cancer. Facts and fables

Graeme Overton
106 Why did so many women develop cancer? Part 2 (with response by Linda Bryder)

Ron W Jones

109 Why won’t defenders of the Cartwright Inquiry provide evidence to justify their use of the term ‘conventional treatment’ for carcinoma in situ?

Iain Chalmers

113 A response to Ron Jones' letter of 30 April 2010

Helen Overton

116 A particular relationship

Paul Patten

117 Discussion of Morris and Jewell's editorial 'Array of hope for high-resolution genetic screening services in New Zealand' (with authors' response)

Clive Felix

118 Managing your student loan

Charles Ronaldson

Obituaries

120 John Cuthbert Parr

122 Irwin Bruce (Bill) Faris

Book Review


Jennie Connor
This Issue in the Journal

Patients admitted with an acute coronary syndrome (ACS) in New Zealand in 2007: results of a second comprehensive nationwide audit and a comparison with the first audit from 2002

Chris Ellis, Greg Gamble, Andrew Hamer, Michael Williams, Philip Matsis, John Elliott, Gerard Devlin, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

Heart and circulatory diseases are the commonest cause of death in New Zealand, accounting for approximately 40% of all deaths. A heart attack is a common cause of death, with 17 New Zealanders dying each day. Cardiac services can significantly improve patients outcomes if new treatments, available especially over the last 5 to 15 years, are available. The 2nd National comprehensive Cardiac Society Audit, run by busy Senior Doctors and Nurses have shown in 2002 and now again in 2007 that significant limitations exist at present. Improvements are needed, based on a 'Hub and Spoke' service, from each of the 5 Regional centres, with significant guidance and input from current clinicians: senior Doctors and Nurses who understand the complexities of Healthcare, and can improve on the current, fragmented service.

ACS patients in New Zealand experience significant delays to access cardiac investigations and revascularisation treatment especially when admitted to non-interventional centres: results of the second comprehensive national audit of ACS patients

Chris Ellis, Gerard Devlin, John Elliott, Philip Matsis, Michael Williams, Greg Gamble, Andrew Hamer, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

This is the second of 2 papers reporting on the 2nd National comprehensive Cardiac Society Audit, run by busy Senior Doctors and Nurses, which has shown in 2002 and now again in 2007 that significant limitations exist in the management of patients who present with a heart attack or unstable angina to a New Zealand hospital. This paper focuses on the time delays experienced by patients who 'wait' for tests and treatments; the delays are significantly worse for patients admitted to a 'Non-Interventional' (mainly rural) Centre. Improvements are needed, based on a 'Hub and Spoke' service, from each of the 5 Regional centres, with significant guidance and input from current clinicians: senior Doctors and Nurses who understand the complexities of Healthcare, and can improve on the current, fragmented service.
A programme of Enhanced Recovery After Surgery (ERAS) is a cost-effective intervention in elective colonic surgery
Tarik Sammour, Kamran Zargar-Shoshtari, Abhijith Bhat, Arman Kahokehr, Andrew G Hill

The enhanced recovery after surgery programme (ERAS) is a clinical pathway that has been implemented to improve patient recovery after colonic surgery. We compared the costs involved to put 50 patients through this programme, with the costs incurred in 50 historical controls. We identified a significant cost saving as a result of ERAS implementation, even when implementation costs are taken into account.

Audit of cervical screening in women with HIV infection in the Auckland and Northland regions of New Zealand
Jasmin Grewal, Michele Lowe, Hilary Gerrard, Rebecca Henley, Nicky Perkins, Simon Briggs

Women with HIV infection have an increased risk of cervical cancer. It is recommended that women with HIV infection receive yearly cervical smears. Only 56% of women with HIV infection who were seen by the Infectious Diseases and Sexual Health Services at Auckland City Hospital had received a yearly cervical smear. It is very likely that seven women in this audit had undiagnosed HIV infection at the time of their first abnormal cervical smear. Health professionals performing cervical smear tests should consider offering an HIV test to all women with an abnormal cervical smear who have resided in areas with high rates of HIV infection.
The Cartwright legacy: shifting the focus of attention from the doctor to the patient

Ron Paterson

In her 1988 *Report of the Cervical Cancer Inquiry*, Judge Silvia Cartwright noted that “old habits and attitudes” had provided a sense of security for the medical professionals and administrators “buffeted by the cold winds” of the Inquiry. Her far-reaching recommendations sought to change the regulatory landscape for patient care and research. They were a full-frontal challenge to the medical establishment, and to the model of professional autonomy and self-regulation that held sway in New Zealand. The judge said that “[t]he focus of attention must shift from the doctor to the patient”, and made detailed recommendations to effect fundamental change.

All too often, the recommendations of major inquiries garner brief media attention, before gathering dust and being quietly shelved by officials. Inaction may be well advised; lawyers undertaking a one-off inquiry, unfamiliar with the subject matter of the inquiry, may make recommendations that are ill-conceived or impractical. And even recommendations that “hit the mark” may still be ignored. Once the inquiry body is *functus officio*, and the media spotlight shifts to other issues, inertia, discrete lobbying from vested interest groups, and a lack of political nerve will often lead to cosmetic change but no significant reform.

Yet from the vantage point of over two decades later, it is clear that the Cartwright Inquiry resulted in major and enduring changes to the legal and health systems in New Zealand. Why did this inquiry lead to such a seismic shift in the relationship between doctors and patients, and the medical profession and the community? What is Cartwright’s legacy in the early 21st Century?

Changing times

In the 1970s and 1980s, feminism, women’s health activism, and a broad range of social movements were challenging traditional norms and changing the fabric of New Zealand society. This was fertile ground for the Cartwright Inquiry. As Joanna Manning notes, “public attitudes to the medical profession were undergoing a transformation”, and the Inquiry and *Report* “both reflected and accelerated these evolving attitudes”.

The teaching and practice of medicine was slow to react to societal changes, and the New Zealand legal system in the late 1980s provided no incentive for reform. Patients’ rights (in particular the right to make an informed choice about medical treatment) were slow to develop in New Zealand, in part due to the constraints of accident compensation legislation. Doctors (and other health professionals) were the beneficiaries of a system that looked to the state to compensate injured patients, and effectively barred claims for medical negligence.

The combined effect of social change and underdeveloped legal protections for patients in New Zealand meant that the time was ripe for reform.
Shocking revelations

But timing alone does not explain the impact of the Cartwright recommendations. The shocking revelations during the Inquiry had a profound impact on the New Zealand public, and made reform inevitable. As Silvia Cartwright has noted, “[T]his was a drama unfolding in the nation’s living rooms.” David Skegg recalls that “public attention was riveted by what seemed like the daily revelations from the hearings”. Helen Clark, who became Minister of Health six months after release of the Report, described the revelations as “truly shocking”. It was shocking (and, despite recent attempts to rewrite history, remains so) that so many women received inadequate treatment for cervical carcinoma in situ at New Zealand’s leading obstetrics and gynaecology hospital, and that some suffered needlessly and died; that patients and their families were kept in the dark; that medical colleagues failed to act; that the system for ethical approval and monitoring of research was woeful; and that abhorrent practices, including students practising the insertion of intra-uterine devices on anaesthetised women without their consent, and the taking of vaginal smears from babies without parental consent, were tolerated.

“Adverse events” are often accepted as a byproduct of a complex healthcare system, and the public becomes inured to news of avoidable harm to patients. Sometimes it takes a major scandal to ignite public outrage, compel government action, and defuse professional resistance. Richard Smith wrote of the inquiry into deaths from paediatric cardiac surgery at Royal Bristol Infirmary that “All changed, changed utterly” (quoting Yeats). The same was true of New Zealand post-Cartwright.

Putting the patient first

One other factor was critical. Judge Cartwright modelled, both in the Inquiry process and in her recommendations, an approach that put the patient first. In chapter 7 of the Report, “Ethics and Patient Rights”, she quoted extensively from the testimony of the women, criticised the prevailing views of doctors and administrators at National Women’s Hospital, and contrasted the weaknesses of the ethical and legal framework for patient care and research in New Zealand with developments overseas.

The judge made a compelling case for patients to be treated with dignity, to receive all relevant information about their condition and treatment options, and to be fully protected as research participants. Her wide-ranging recommendations targeted both the individual patient-doctor relationship, and the legal and health systems more generally.

Decades before the language of “patient-centred care” and “consumer perspective” became fashionable, Silvia Cartwright saw the need to make consumer voice central to the monitoring of health care delivery, via elected representatives to hospital boards, independent advocates, and an independent Health Commissioner; to enshrine patients’ rights in legislation; to have much greater lay involvement in a rigorous system of ethical review of proposed research; and to make the needs of patients pivotal to medical education and to the complaints and disciplinary process.
Cartwright’s legacy

The path to implementation of the Cartwright recommendations was not smooth. Despite the “public climate of expectation of change”, and vigilance by the women’s health movement, five years after the Report release there was still much “unfinished business” (as described by Sandra Coney and colleagues in their 1993 book of that title). But significant reforms did follow and have proved enduring.

The system for ethical review of clinical research and innovative treatments is now subject to a rigorous approval process, with independent regional ethics committees (with a lay chairperson and 50% lay membership) operating in accordance with a national Operational Standard, and using guidelines developed by a statutory National Ethics Advisory Committee. Mechanisms for monitoring compliance with ethical approval remain problematic, but Jan Crosthwaite concludes that “New Zealand now has good regulatory protections in place, although we should not think we are immune to the possibility of rogue researchers”.

The Code of Health and Disability Services Consumers’ Rights, enacted in 1996, gives patients legally enforceable rights, far surpassing the puffery of non-binding charters adopted in many other countries. The rights closely mirror the template proposed by Judge Cartwright, including rights to be treated with respect, to effective communication and adequate information, to make an informed decision, to receive care of an appropriate standard, and to make a formal complaint to an independent advocate or Commissioner. The Code has become ubiquitous, visible on the walls of hospitals and health clinics and, more importantly, familiar to health professionals and the public (with surveys showing much greater awareness of patients’ rights).

Most importantly, there has been an attitudinal shift within the medical profession. Communication skills, professionalism, and ethical reflection are now taught alongside clinical skills in the undergraduate medical curriculum. Informed consent, ridiculed by members of the profession in the immediate aftermath of the Inquiry, is now accepted as essential to securing the trust of patients and improving the outcomes of care. The focus has shifted to how to provide information in a way that meets the needs of patients, and has extended from the context of pre-surgery to the whole continuum of health care and disability service provision, including in the aftermath of an adverse event (“open disclosure”). Ensuring that patients are treated with dignity and compassion within an increasingly complex health system remains a challenge, with debate about how best to achieve this, but no one contests the need to do so.

The complaints and disciplinary system in 2010 is transformed from the unwieldy and health professional-dominated system of the past. A nationwide network of independent advocates is contracted by a statutory Director of Advocacy, and is highly effective in resolving consumer complaints in local communities.

The Health and Disability Commissioner (HDC) complaints system enables independent resolution of complaints, with a minority leading to published investigation reports in which substandard hospitals and rest homes are identified. The reports are generally welcomed by professional groups and used for education, providing guidance on complex issues such as follow-up of patient test results and co-ordination of primary and secondary care. The Commissioner has become a highly visible “patient watchdog”, commenting on problems in the health system.
A combination of much greater appreciation of the role of systems in patient safety, together with the rehabilitative approach fostered by HDC and the Medical Council (using tools such as competence reviews, under the Health Practitioners Competence Assurance Act 2003) has resulted in far fewer cases leading to disciplinary hearings before the Health Practitioners Disciplinary Tribunal (a multidisciplinary body entirely separate from the individual registration authorities). Clinical negligence cases seldom result in discipline; charges of unethical behaviour, such as sexual or financial exploitation of patients, and improper prescribing predominate.

Implementation of the national cervical cancer screening programme recommended by Judge Cartwright was particularly vexed, with flaws exposed in the Gisborne Cervical Cancer Screening Inquiry in 2001, but the current scheme has been described by David Skegg as “a triumph of preventive medicine”. The programme is estimated to be preventing at least 70% of the cases of cervical cancer that would otherwise be occurring in New Zealand, saving the lives of more than 100 women every year.

A complex story

The lessons to be learned from the Cartwright Inquiry remain contested territory. Yet even the revisionists, while seeking to downplay the significance of the Inquiry, hesitate to criticise the reforms described above. In retrospect, they can be seen to be timely and necessary.

It is, however, too simplistic to view the Inquiry as a triumph of external regulation over internal morality. As Charlotte Paul has described, the Cartwright story is more nuanced and complex, and over-reliance on the “blunt instruments” of external controls can undermine trust and be counterproductive to a “functioning internal morality”.

The role of the doctors who attempted to raise concerns with National Women’s Hospital (notably Bill McIndoe, Jock McLean and Ron Jones), and of the professional leaders who sought to make the reforms workable for patients and doctors (including Robin Briant as chair of the Medical Council in the 1990s), needs to be acknowledged. Sandra Coney applauds the “quiet but monumental shift in the attitudes” of doctors, who “on the whole … grasped the nettle and changed their practice”.

Challenges remain. Despite all the rhetoric about putting patients first, the current emphasis is on clinical leadership. There is still a need to strengthen consumer voice at all levels in the health system. It is remarkable that the new (albeit interim) Health Quality and Safety Commission has no consumer member. There is also a sense of complacency about the current framework for ensuring health practitioner competence, and lay involvement in registration authorities has fallen well behind regulatory reforms in some other countries (notably the United Kingdom).

Finding effective ways to raise concerns within the health system is a particular concern. Too often, health professionals who attempt to do so lack institutional support and are met by denial and resistance. Even external inquiry bodies learn to expect re-litigation of findings by interested parties, denigration by critics, and
revisionism by subsequent commentators who did not hear all the evidence and sometimes seem wilfully blind to it.

Much has been achieved, and the focus has largely shifted from doctor to patient, but Silvia Cartwright’s words of warning bear repeating:¹

“... [A]dministrators and health professionals need to listen to their patients, communicate with them, protect them, offer them the best health care within their resources, and bravely confront colleagues if standards slip. If this does not happen, then the kind of events disclosed during this Inquiry may well happen again.”

Competing interests: None known.

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References:

The missing manuscript

Frank A Frizelle

This edition of the NZMJ contains a number of letters about what became known as the ‘Unfortunate Experiment’. In many ways New Zealand has been fortunate as a result of these events in that they led to a complete revamp of our medical, legal, and ethical environment as outlined by Professor Ron Paterson’s editorial in the same edition.¹

Recent books²,³ have not surprisingly generated a lot of conflicting views and heated debate as they have tried to shed new light on the events that occurred. It has been the re-exploration of events by Professor Bryder that led to her book being published² followed by two book reviews with contrasting views that the NZMJ published.⁴,⁵

Then Professor Charlotte Paul contacted me wanting to write an editorial about the book review⁴ which was supportive of Professor Linda Bryder’s book. Given Professor Paul’s key role on the Cartwright Report and the career that she has since built at least in part on these issues, I suggested that she write either a:

• Letter to the editor which would be more appropriate if she wanted to disagree with a book review that we had published, or

• An article exploring issues around the Cartwright Report that had been raised by Professor Bryder’s book—but this must provide us with new insights into the issues and the article must not focus solely on criticism of the author, Bryder. (There are many other avenues, for example the Listener, for publicly disagreeing with a published book.)

The Journal received from Professor Paul a very wordy manuscript which didn’t fulfil the guidelines I had given her. We then spent some months reviewing her article and reflecting on whether it really did contribute anything to the debate. In the end I decided to publish it, but thought that perspectives should also be given by others and so I asked Professor Ron Jones and Professor Linda Bryder to separately write editorials, while pointing out that they would get to see each other’s editorials for comment pre-publication.

Linda Bryder produced an interesting and thoughtful editorial. Professor Jones refused the offer. Then Professor Paul withdrew her manuscript because she did not feel that it was reasonable to have Professor Bryder writing an editorial on this issue.

Amongst much media publicity (including lead items on national media) within a few days of withdrawal of this manuscript Professors Jones and Paul published an article in the Australian and New Zealand Journal of Obstetrics and Gynaecology.⁶ They reported:

“And our findings show that inclusion in this clinical study subjected women to many medical interventions designed to observe rather than treat their cervical intraepithelial neoplasia, and increased their risk of developing cancer of the cervix or vaginal vault. The greater numbers of subsequent biopsies that were performed on women in the core group (who received only a punch or wedge biopsy initially) attest to their assiduous follow-up…”
And “…Among women diagnosed with CIN3 in 1965–1974, the incidence of invasive cancer was ten times greater in the core group (who received only a punch or wedge biopsy initially) than in women treated initially with curative intent….”

Over the past few months the Journal received the letters on these issues (which are published in this edition) plus I asked for an editorial from Ron Paterson that outlines the medical-legal-ethical significance of the Cartwright Report.

We informed Professor Bryder that Professor Paul had withdrawn her manuscript and asked Professor Bryder to rewrite her editorial removing any reference to what Professor Paul had said in her withdrawn manuscript.

I have decided to publish these editorials and letters in the Journal despite my concern that the real issues are being increasingly lost over time since these events occurred. However the papers provide an interesting perspective and a somewhat insightful perspective.

Over this period I have received numerous emails from many self-interested parties offering advice on how to run the Journal and how I should deal with these issues. Choosing to publish these letters and editorials relates to the need for free discussion of the issues and not personal attacks on those who are the messengers.

With this current background it is important, amongst all this recent dialogue and the effect of various personalities, not to forget the real issues which are:

- Consent for studies at the time when these studies was undertaken were inadequate by today’s standards.
- People were harmed. This is described in the article by Professor Paul and colleagues in article in Lancet Oncology in 2008 where the authors state: “. . . 1229 women whose treatment was reviewed by the judicial inquiry in 1987–88 were included. Of these, 48 records (4%) could not be located and 47 women (4%) did not meet the inclusion criteria. At histopathological review, a further 71 (6% of 1134) women were excluded because the review diagnosis was not CIN3. We identified outcomes in the remaining 1063 (86% of 1229) women diagnosed with CIN3 at the hospital in 1955–76. In 143 women managed only by punch or wedge biopsy, cumulative incidence of invasive cancer of the cervix or vaginal vault was 31.3% (95% CI 22.7–42.3) at 30 years, and 50.3% (37.3–64.9) in the subset of 92 such women who had persistent disease within 24 months. However, cancer risk at 30 years was only 0.7% (0.3–1.9) in 593 women whose initial treatment was deemed adequate or probably adequate, and whose treatment for recurrent disease was conventional.”
- A key unresolved issue to some would appear to be whether the two groups had received the same treatment or not. In the Journal it is argued by some that they did [receive the same treatment] as the division is dependent on treatment outcomes decided only at 2 years.

The context in which these events occurred is the issue for discussion, not whether they happened. The result, as Professor Paterson states, is that “ most importantly, there has been an attitudinal shift within the medical profession.”

The New Zealand medical system has gained much from what happened. Some individuals have been blamed for what happened, while it is in reality institutional systems that resulted in the problem, not a “rogue doctor”.

URL: http://www.nzma.org.nz/journal/123-1319/xxxx/
Similar issues are worth exploring to avoid institutional blindness resulting in similar problems harming patients again.

**Competing interests:** None known.

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**References:**


A response to criticisms of The History of the 'Unfortunate Experiment' at National Women's Hospital

Linda Bryder

The publication of my recent book The History of the 'Unfortunate Experiment' at National Women's Hospital has generated a lively debate concerning the conclusions of the 1988 Cartwright Report. As the most trenchant criticism of my book appeared in Charlotte Paul’s chapter in The Cartwright Papers, I will use this editorial to address her criticisms.

Professor Paul states that I ‘insinuate’ that the Inquiry was biased by her involvement. I make no such claim. My book is not concerned with her involvement, but rather with understanding what happened at National Women's Hospital and how it was represented at the Inquiry and publicly. Her further claim that my book was the result of a ‘particular relationship’ I had formed with certain gynaecologists in the course of my research does not merit a response.

Charlotte Paul and I offer opposing interpretations of Green’s understanding of CIS and of his management compared to others working in the field, and it is these differences I will address here. In engaging with these debates, I will take issue with Paul’s criticisms of my use of evidence, and look critically at her own use of evidence. I will conclude by considering the contributions to the debate in the recent article in the Australian and New Zealand Journal of Obstetrics and Gynaecology by Paul and colleagues.

In The Cartwright Papers Paul states that Green ‘wrote repeatedly of his belief that CIS was a benign condition’. While she provides no reference following this statement, in the 2008 Lancet Oncology article, of which she was a co-author, the same claim is referenced to an article Green wrote in 1966. Yet what he actually wrote there was: ‘These then are still the two uncertain factors—the length of the pre-invasive phase and the proportion going on to invasion. Clinical evidence is tending to show, but cannot prove that the latter is small—probably much less than 10 per cent’. In other articles (1969 and 1970) he again addressed the current state of knowledge and the uncertainty as to whether ‘the invasive potential in in situ cancer is as high as has been claimed’. In addressing this uncertainty he was following authorities such as George Knox, Professor of Social Medicine at the University of Birmingham England, who stated in 1966 that population and pathological evidence could suggest not one but two diseases—a benign one and some hitherto unidentified lesion; they simply did not know. The role of the human papillomavirus was not identified until the 1980s.

Regarding Green’s 1966 proposal to the Hospital Medical Committee I do not accept Paul’s claim that I tried to misrepresent it as ‘conservative treatment by cone biopsy’. Indeed it was obvious that this was not the case, given that the proposal was to ‘diagnose and treat by lesser procedures than hitherto’; one of the ‘hitherto procedures’ had been cone biopsy. The important point was careful follow-up. As the
minutes of the meeting recorded, ‘If at any stage concern was felt for the safety of the patient a cone biopsy would be performed’.\(^{11}\)

As Green told the Medical Superintendent a few years later, ‘It was always a calculated risk that invasive cancer could be overlooked, although it was hoped that colposcopy, clinical examination, and repeated directed biopsies would minimise, if not actually avoid, this.’ He cited several authorities in support of this (see my book p25). Had Green believed that CIS was totally benign, as Paul suggests, he would not have insisted on such careful follow-up.

Paul incorrectly states that I ignored the considerable discussion at the Inquiry on the use of the word ‘invariably’ in the minutes of the meeting at which Green put forward his proposal (see my book p30). Furthermore, in her discussion of the 1966 proposal, Paul credits his colleagues with little intelligence. Green said at the Inquiry that, had he proposed that CIS was harmless when he took his protocol for conservative management to the Hospital Medical Committee in 1966, he was sure the committee would never have agreed to the proposal. Paul asks, ‘What does this mean? If he had told the committee that CIS was harmless, they wouldn’t have agreed to a trial of no treatment? Surely they would have agreed more willingly to no treatment for a benign condition.’\(^{12}\) What Green knew, and he was correct, was that no gynaecologist regarded CIS as invariably benign and they would clearly have thought him crazy if he had suggested it, and would not have approved his proposal.

Paul states that I misinterpreted the differences between a study by Norwegian Professor Per Kolstad and Green’s ‘trial’. The difference was, she said, that Kolstad followed patients after treatment and Green followed patients without treatment.\(^{13}\) Yet Cartwright criticised Green for following patients after treatment. She declared in her Report, ‘One outstanding fact ought to have been clear to [Green] and to others—following (without treating) patients with positive smears, whether after cone biopsy, or after hysterectomy, was unsafe, as a proportion of those women would subsequently be shown to have invasive cancer.’(my emphasis).\(^{14}\)

Like Green, in the 1970s Kolstad advocated a cautious approach to treatment of CIS. He hoped that the use of new technology, specifically colposcopy, would help reduce the need for cone biopsies as treatment. He advised caution as, ‘many of these [in situ lesions] occurred in young women of childbearing age, for whom hysterectomy and even cone biopsy could mean considerable trauma to the reproductive tract’.\(^{15}\)

Paul states that in the period 1965–74 Green, unlike Kolstad, was not using cone biopsies; elsewhere in the article\(^{16}\) she complains that in the same period he was doing too many cone biopsies; she can’t have it both ways. Nor can she retrospectively distinguish between cone biopsies for diagnostic purposes and for curative purposes, as is further discussed below in relation to the 2010 article.

Both Kolstad and Green were opposed to an aggressive approach to the management of cases of CIS. They were supported in this by a textbook written by a British Professor of Obstetrics and Gynaecology, Sir Norman Jeffcoate. Paul claims I distort his advice, since he was only advising caution during pregnancy.\(^{17}\) She is mistaken; as I explain on page 41 of my book, he advised, ‘When cervical smears repeatedly contain cells indicative of malignancy or severe dyskaryosis [dysplasia], the next step is to carry out cervical biopsy ... This operation is not free from immediate and late
hazards and fatalities are reported; so it is wise to be sure that it is really necessary before proceeding to it. (his emphasis). He then went on to discuss the particular concerns during pregnancy.  

In Australia, Malcolm Coppleson became renowned for advocating conservative treatment for CIS, and was a major influence on Green. Like Green and Kolstad, Coppleson hoped that with the use of colposcopy-directed punch biopsy, the use of cone biopsies could be reduced. Yet again, Paul argues that I misinterpret his ‘conservative’ approach. She states that Coppleson’s ‘punch biopsies’ were to be followed by ‘definitive treatment’ which she defined as ‘excisional biopsy, cryosurgery, therapeutic conisation, hysterectomy, or irradiation’. This was not necessarily the case; in his 1977 contribution to Recent Advances in Obstetrics and Gynaecology, Coppleson made the following statement:

Preliminary results from several sources indicate that many such lesions, which would have previously been treated by the more expensive and more hazardous sequence of diagnostic conisation followed by hysterectomy or by therapeutic conisation, have been thoroughly evaluated and safely treated by either cryosurgery, electrocautery or electrodiathermy, or multiple punch biopsy...

Indeed, in 1970 Coppleson lamented that ‘much present-day management of cervical lesions is imbalanced, illogical and too radical’ because the clinician abdicated his ‘traditional captaincy to the pathologist or exfoliative cytologist’.  

Charlotte Paul notes that I refer to Sir Richard Doll in chapter 4 on patient consent, and opines that his comments did not apply to Green’s ‘trial’. The important thing to remember about Doll is that he supported Green in his research until Green retired in 1982, and wrote references for him to the Medical Research Council (see p.206, n121 of my book). He also praised Green highly during a symposium in Auckland in 1973 for not automatically adopting a radical approach to CIS. Regarding Professor Archie Cochrane and Paul’s surmise that he would have disapproved of Green, Paul should know otherwise if she attended the Cartwright Inquiry—Cochrane’s invitation to Green to collaborate in a research proposal was discussed there.  

Paul states that Sir Graham Liggins acknowledged at the Inquiry that Green was ‘doing a prospective study that entailed withholding treatment’. She provides no reference for this, and Liggins did not give evidence at the Inquiry. What can be referenced, however, is Liggins’s published oration to the 1990 General Scientific Meeting of the Royal Australasian College of Surgeons in Wellington, in which he commented on the fact that the 1984 McIndoe article on which ‘the cervical cancer enquiry was based, was misinterpreted by the authors of the Metro article and by the judge’. This misinterpretation consisted of regarding it as a prospective study rather than a retrospective study.

Discussing the 1984 McIndoe paper Paul writes, ‘Bryder’s claim that there was no difference in treatment between Groups 1 and 2 also leaves her with no explanation for the fact that the rate at which cancer appeared in Group 2 was twenty-five times higher than in Group 1. Again, she has made an illogical and incoherent claim’. Yet it is Paul herself who is not being logical here. These women were placed in Group 2 by McIndoe and his co-authors retrospectively because they had persistent positive smears and so they would be more likely to develop cancer than those whose smears
had returned to normal. The groups were constructed according to outcomes.
Treatment did not enter the study, as McIndoe himself told Sandra Coney in 1985 (see my book p33).

Despite Paul’s claims to the contrary, I do distinguish between follow-up of CIS and dysplasia; see for instance, page 45 of my book: ‘Some follow-up studies were designed to follow mild lesions only, though the categorisation of smears was far from a precise science’; I then refer the reader to the more detailed discussion in chapter five. Here, among others, I cite (p.75) an American authority, Professor Leopold Koss, who declared in 1978, ‘Truly it can be repeated that one man’s dysplasia is another man’s carcinoma in situ ... There is no publication on this subject where one could not reshuffle the photographs and substitute pictures labelled as dysplasia for those labelled carcinoma in situ and vice versa.’ The reclassification of slides which Paul refers to was discussed at the Inquiry, and pathologist Jock McLean said this was ‘quite open’ and the result of disagreements; it was not dishonest manipulation (see my book, pp78-9).

In other places in her critique, Paul distorts or misrepresents what I have written. When I responded to Ron Paterson’s claim that no empirical evidence of the ‘revisionists’ had been published,28 I was referring to the evidence of the uncertainties and debates surrounding the meaning, interpretation and treatment of CIS,29 discussed in my 2008 article in the *Journal of Epidemiology and Community Health*. When she addresses my coverage of the 1990 Medical Council decision Paul writes that I provide no evidence that there were any debates in the 1960s. My 2008 article and the earlier chapters of my book provide this in abundance. Also in her discussion of the Medical Council findings, she claims that I overestimated McIndoe’s grasp of colposcopy, citing Coppleson that McIndoe ‘only briefly attended his colposcopic clinic in Sydney and was keenly aware of his own lack of expertise’.31 She fails to note that in 1968 McIndoe visited America and Europe to study the current use of colposcopy, funded by the Auckland Medical Research Fund and the Cancer Society of New Zealand, and in 1972 he attended the First World Congress of Colposcopy in Argentina.32

Paul criticises me for ignoring the medical survey reported in Appendix 3 of the Cartwright Report.33 She refers specifically to Table 2. It is difficult, however, to see how meaningful the increases during the period 1966-1974 really are, as presented here, when the numbers are so small—one case in 1965, reaching a maximum of four women in 1968 and 1974, declining to three in 1975. Moreover, as an epidemiologist surely Paul should be wary of attributing any trends to one single factor. Such changes could be related to the prevalence of the human papillomavirus, or the use of the contraceptive pill, or, as Professor Skegg later suggested in 1985, the ‘sexual revolution’.34 The new retrospective analysis of data reported in the 2010 article is addressed below.

I also take issue with Paul’s use of evidence in her critique of my book. She writes, ‘Cartwright commented on the “remarkable degree of unanimity” among the overseas authorities in their conclusions from reviewing patients’ files.’ This looks less impressive when it is viewed together with the immediately preceding sentence in which Cartwright explained, ‘Some of the overseas authorities who gave evidence were invited to carry out case studies on selected patient files...’ (my emphasis).
Paul states that Kolstad concluded at the Inquiry that some women suffered ‘severe’ and ‘terrifying mismanagement’. In his evidence the ‘terrifying mismanagement’ comment applied to one woman, not ‘some’ women. That woman was Clare Matheson and under questioning at the Inquiry Kolstad admitted that his source of information on this was primarily the Metro article.

The 2010 Australian and New Zealand Journal of Obstetrics and Gynaecology article continues along the same theme as previous publications by Paul, i.e. that Green ‘withheld treatment of curative intent’ and thereby engaged in unethical behaviour. I have several reservations with the arguments of this article. The first reservation relates to the definition of ‘treatment of curative intent’. The authors define this as hysterectomy, amputation of the cervix and cone biopsy, and exclude wedge and punch biopsy. Yet Professor Koss stated in 1963 that even a small punch biopsy could be curative. Coppleson also saw punch biopsy as a possible treatment, as noted above. Similarly in Scandinavia by the mid-1970s a debate was launched as to whether cone biopsy was ‘over-treatment’ for CIS.

If ‘treatment of curative intent’ meant doing at least a cone biopsy, then my second reservation relates to the period defined for the ‘clinical experiment’ in the 2010 article. The 1984 McIndoe paper stated that no-one at National Women’s Hospital practised less than cone biopsy after 1970 (‘the few clinicians who initially performed punch or wedge biopsy alone had abandoned the practice by 1970’), so the period in which doctors at National Women’s were ‘experimenting’ by not giving cone biopsies should presumably have been 1965 to 1970. On the other hand, one of the authors of the 1984 and the 2010 articles, Dr Ron Jones, stated in a recent letter to the New Zealand Medical Journal, that if only McIndoe had succeeded in publishing his views in 1974 he might have been able to stop the ‘experiment’, suggesting that he did not see 1974 as the cut-off point for the ‘experiment’. In other words, choosing the period 1965 to 1974 as the period of the ‘trial’ is an artificial contrivance on the part of the 2010 authors.

Thirdly, there is no evidence in the article that the patient records reviewed were Green’s patients. The authors write of an ‘excess of inappropriate follow-up interventions for women diagnosed in 1965-74’—these follow-up interventions presumably continued well beyond 1974 (the article states that follow-up continued until death or 31 December 2000). These follow-up interventions were provided by a wide range of doctors at the hospital, including Jones himself (Green retired at the beginning of 1982). Jones told the 1987–8 Inquiry that from the time he arrived in the hospital, the mid-1970s, treatments at the hospital were in accord with international standards; now he is saying otherwise.

Fourthly, it is not true to say that women in the early 1960s were more likely to be unscreened than in the period 1965–74. The early 1960s saw a number of screening programmes initiated and reported in the New Zealand Medical Journal, in Thames and elsewhere, which were short-lived, and from 1960 all women attending clinics at National Women’s Hospital were given smear tests.

Fifthly, a retrospective study cannot prove unethical behaviour, which implies intent not to do best for one’s patients. Management choices in medicine might not always be the right ones in retrospect, and Green was the first to acknowledge this.
Finally, where is the authors’ evidence that ‘follow-up biopsies were often intended to exclude invasive cancer rather than to diagnose and treat CIN3’? Again they are attributing ‘intent’ with no evidence to support this.

The management of CIS following the introduction of Pap smears in the 1950s caused considerable debate, and these debates were not merely academic. As one commentator explained as late as 1991, the 'medical dilemma' in relation to CIS was 'to know when to treat the abnormality and when to leave it alone because no harm would result from doing so, whereas intervention could lead to a variety of unintended negative consequences'. These were serious issues which Green amongst others was attempting to grapple with.

The recent article does not, as has been alleged, settle the debates. The debates about what happened at National Women's Hospital raise wider issues about medical practice which are overlooked by those who try to present the past as one-dimensional (i.e. portraying Green as a 'villain', and McIndoe as a 'hero'). The wider issues, still relevant for medical practice today, are concerned with how medicine deals with uncertainties, how doctors are more likely to be criticised for undertreatment than overtreatment, even if the latter does more harm than good, and how medicine and society interact, particularly where there is any hint of the possibility of the dreaded disease, cancer.

**Competing interests:** None known.

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**References:**

3. Ibid, p118.
17. Paul 2009. p123.
33. Paul 2009. p132


45. Bryder 2009, p17-18


Acute coronary syndromes in New Zealand hospitals

Robin M Norris

In this issue of the Journal Ellis et al\textsuperscript{1,2} report a repeat, 5 years later, of their original 2002 study\textsuperscript{3,4} in which they examined the treatment of acute coronary syndromes (ACS) both nationally, and by comparison of hospitals with and without facilities for invasive cardiac examination and treatment. They found that little had changed over the period 2002–7.

While the use of coronary angiography and percutaneous balloon angioplasty had increased in New Zealand, neither was used as frequently as in Australia, Europe or the USA. Patients treated in non-intervention hospitals had equal access to invasive procedures, but access was delayed for those in the smaller hospitals. Most important, prescription of most state of the art drugs for secondary prevention was widely followed both in 2002 and 2007. The exception, due to funding restrictions, was clopidogrel.

Ellis et al are to be congratulated for their pioneering efforts in bringing the audit process to bear on the most numerous and important group of medical patients admitted as emergencies to New Zealand hospitals; also on their recognition of the principle that audits should be compared over time and that individual performances should be compared so that those who do badly can learn from those who do well.

In this respect, clinicians in the smaller hospitals performed as well as their colleagues in the larger hospitals by referring patients for coronary angiography, albeit with inevitably increased delay. The authors suggest that this delay might be reduced by adoption of a formalised “hub and spoke” approach whereby patients from smaller hospitals are referred to the five major hospitals which are fully equipped for invasive investigation and treatment.

There are, however, caveats to interpretation of the study by Ellis et al. First, international comparisons of the use of invasive study and revascularisation do not necessarily imply that New Zealand hospitals should strive to achieve the highest rates. Although invasive revascularisation can be lifesaving, is the treatment of choice for acute ST elevation myocardial infarction, and is efficacious for relief of angina, it does not prevent reinfarction\textsuperscript{5} because sites of vulnerable atherosclerotic plaques do not correspond with the sites of critical stenoses as defined by angiography\textsuperscript{6}.

Decisions to revascularise based on angiographic appearances rather than on real clinical need do not result in benefits to patients\textsuperscript{7} nor does opening of a chronically occluded infarct-related artery\textsuperscript{8}. Anecdotal evidence suggests that revascularisation can be overused, particularly in parts of the USA. Moreover, coronary atherosclerosis can regress with secondary prevention, particularly with statins\textsuperscript{9}.

The second caveat applies, in the opinion of this reviewer, not only to the study by Ellis et al, but also to most if not all other audits of ACS. We pointed out some years ago\textsuperscript{10} that, although defibrillation prevented four times as many deaths as
thrombolytic treatment in ACS, it had been undervalued in the literature because it was not “evidence based”; it had never been subjected to clinical trial.

Evidence for the greater efficacy of defibrillation came from two UK audits during the 1990s,\textsuperscript{11,12} which also showed that numbers of deaths prevented by defibrillation were critically dependent on delay from onset of symptoms to coming under care, and that 30–40% of successful defibrillations were performed outside hospital by ambulance personnel. If this evidence is accepted, audits of ACS should include success of defibrillation and should also examine delay in coming under care and the vitally important pre-hospital phase of ACS. A pilot study of delay has recently been reported from Middlemore Hospital in Auckland.\textsuperscript{13}

What should be the future of audit of ACS in NZ? Because the structure of the British National Health Service (NHS) is in many ways most comparable with the NZ Health Service, the British (England and Wales) National Audit of Myocardial Ischaemia (MINAP),\textsuperscript{14} although surprisingly not referred to by the authors, probably offers the best guide. MINAP, involving 210 hospitals, has been operating continuously for 10 years, has recorded data on more than 700,000 episodes of ACS, and has been associated with remarkable improvements in delivery of treatment, at first in reductions in delay to thrombolytic treatment, and more recently in the organization of the “hub and spoke” principle to primary angioplasty (PPCI).

It is anticipated that in 2010 the majority of patients with ST elevation MI will be treated by PPCI. Classification of individual hospitals by their performance in treatment of ACS is recorded and is in the public domain. Similarly, information on performance of individual cardiac surgeons is available to the public,\textsuperscript{15} and it is planned to extend this to performance of invasive cardiologists. This culture of transparency is now an integral part of the NHS. Should a similar culture be adopted in New Zealand?

What MINAP does not do, is to document the characteristics of the victims and the circumstances of death of the great majority of fatalities from ACS which happen outside hospital. Surprisingly, there are few or no data on this since the international MONICA study of 25 years ago\textsuperscript{16} and our own UK Heart Attack Study (UKHAS) of 15 years ago\textsuperscript{11,17} which showed that 74% of fatal events in people under 75 years of age happened outside hospital.

The good news, according to official figures, is that mortality from coronary heart disease has fallen by more than 50% in the UK over the last 15 years.\textsuperscript{18} We are currently planning a repeat of UKHAS, which we hope will cast light on the mechanisms by which this has happened.

\textbf{Competing interests:} None.

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References:


11. The United Kingdom Heart Attack Study Collaborative Group. Effect of time from onset to coming under care on fatality of patients with acute myocardial infarction: effect of resuscitation and thrombolytic treatment. Heart 1998;80:114-120.


Patients admitted with an acute coronary syndrome in New Zealand in 2007: results of a second comprehensive nationwide audit and a comparison with the first audit from 2002

Chris Ellis, Greg Gamble, Andrew Hamer, Michael Williams, Philip Matsis, John Elliott, Gerard Devlin, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

Abstract

Aims To audit all patients admitted to a New Zealand (NZ) Hospital with an acute coronary syndrome (ACS) over a 14-day period, to assess their number, presentation type and patient management during the hospital admission and at discharge. To compare patient management in 2007 with the 1st NZ Cardiac Society ACS Audit from 2002.

Methods We updated the established NZ ACS Audit group of 36 hospitals to 39 hospitals now admitting ACS patients across New Zealand. A comprehensive data form was used to record individual patient information for all patients admitted between 00.00 hours on 14 May 2007 to 24.00 hours on 27 May 2007.

Results 1003 patients, 9% more than in 2002 (n=930), were admitted with a suspected or definite ACS: 8% with a ST-segment-elevation myocardial infarction (STEMI), 41% with a non-STEMI (NSTEMI), 33% with unstable angina pectoris (UAP), and 17% with another cardiac or medical condition. In 2007 non-invasive risk stratification following presentation remained similar to 2002 and was suboptimal: exercise treadmill tests (21% vs 20%, p=0.62), echocardiograms (19% vs 20%, p=0.85). An increase in utilisation of coronary angiography was noted (32% vs 21%, p<0.0001). In hospital revascularisation rates remained low in patients with diagnosed ACS (n=828): STEMI (45%), NSTEMI (23%) and UAP (7.3%). In comparison to 2002, changes were noted in revascularisation techniques with percutaneous coronary intervention (PCI) performed in 19% vs 7% (p<0.0001). The use of coronary artery bypass grafting (CABG) remained extremely low: 2.8% vs 3.5% (p=0.20). The use of hospital and discharge medication of proven benefit was also limited.

Conclusions A collaborative group of clinicians and nurses has performed a second nationwide audit of ACS patients. Despite a small increase in access to cardiac angiography, guideline recommended risk stratification following the index suspected ACS admission with a treadmill test or cardiac angiogram occurred in only 1 in 2 (48%) patients. Furthermore, in patients with a definite ACS, levels of revascularisation are low. (PCI 19%, CABG 2.8%). These aspects of care remain of significant concern and have not substantially changed in 5 years. There remains an urgent need to develop a comprehensive national strategy to improve all aspects of ACS patient management.
Cardiovascular disease remains the commonest cause of death in New Zealand, being responsible for 11,293 (39%) of the 28,636 total deaths in 2004.\(^1\) Ischaemic heart disease was responsible for 6313 (22%) of these deaths. An acute coronary syndrome (ACS) is an unstable and potentially life-threatening presentation of ischaemic heart disease, and is a spectrum of clinical conditions: unstable angina pectoris (UAP), non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

Effective treatment strategies, summarised in international\(^2-5\) and local\(^6,7\) guidelines, exist and are able to significantly improve the morbidity and mortality of this condition.

In May 2002, the Cardiac Society of New Zealand ACS Audit Group performed a comprehensive audit over a 14-day period which described patient numbers, presentation type and management during hospital admission. This audit demonstrated low levels of investigations, evidence-based treatments, and revascularisation.\(^8\) In addition, there was inequitable management as patients admitted to a hospital without cardiac interventional facilities received fewer investigations and less revascularisation than patients admitted to interventional centres.\(^9\)

Significant efforts were subsequently made by clinicians to improve both the medical and invasive management of patients with ACS. Local practice consensus guidelines were written,\(^6,7\) and efforts were made to try to improve access to treatments of proven benefit.\(^10-15\) Clinicians from interventional and non-interventional centres in New Zealand aimed to facilitate the transfer of appropriate patients from Non-Interventional to Interventional hospitals: the so-called ‘hub and spoke’ approach to management.\(^16\)

In May 2007, the Cardiac Society of New Zealand ACS Audit Group performed a further 14-day assessment of ACS in New Zealand to record current management, to discover if significant changes had been made from 2002, and to identify areas where further improvements in service delivery may be indicated. Once again we chose to undertake the Audit during a 2-week autumn period, to be consistent with the 2002 Audit, and to minimise the known influence of seasonal change on the number of ACS patients.\(^17\)

**Methods**

**Data collection**—The established ACS Audit Group network from 2002 was used, consisting of one physician for every hospital in New Zealand that admitted ACS patients. Most centres also co-opted one or more research nurses or registrars to assist with data collection for the study.

Since 2002, the ‘Green Lane Hospital’ Cardiovascular Service in Auckland had moved to the site of the ‘Auckland Public Hospital’, and these two cardiac services had combined as the ‘Auckland City Hospital’ service. In addition, Waitakere Hospital in Auckland had opened a coronary care unit, and Kaitaia, Dargaville and Rawene in Northland were now actively planning to admit patients with an ACS presentation.

Other smaller hospitals in New Zealand were not actively trying to admit such patients. Therefore, in 2007 there were 39 hospitals admitting ACS patients, compared to 36 hospitals in 2002.

The data collection form recorded patient demographics, initial and discharge diagnosis, medication use in hospital and at discharge, as well as investigations undertaken and invasive treatments received by patients. The dataset collected in 2007 was similar to 2002, with additional information obtained to help assess pre-hospital presentation and aspects of PCI practice. The inclusion criterion for the audit was ‘a patient admitted overnight with a suspected or definite acute coronary syndrome’.
Following admission and investigations, a ‘discharge diagnosis’ was subsequently determined by the local clinical team who confirmed the diagnosis of an ACS, as a STEMI, NSTEMI or UAP, or determined a ‘non-ACS’ presentation resultant on investigations undertaken in hospital and the patients clinical course.

A 2-week audit period was accepted as a compromise between the need to collect sufficient patient numbers to obtain an accurate representative cohort versus the ability of mainly unfunded clinicians and nurses to collect the consecutive patient data. We collected data from 0000 hours on Monday 14 May to 2400 hours on Sunday 27 May 2007 (13–26 May in 2002).

Following input from all 39 centres, ethical approval was obtained from the Multi-region Ethics Committee. As an audit of current practice, individual patient consent was not required. The Ethics Committee permitted the collection of patient names and National Health Index (NHI) numbers to assist with accurate data collection.

Data (including revascularisation procedures) from patients subsequently transferred to another institution are ‘attributed’ to their original admitting hospital. Patients readmitted within the 2 weeks have all admissions included in the data; they only represented a small percentage of the overall patient number. Ethnicity was self-reported at hospital admission.

Troponin was measured at all 39 hospitals. In May 2007 there were 11 different analysers across New Zealand provided by 5 major companies: Roche (5), Abbott (3), Bayer (1), Dade Behring (1) and Beckman Coulter (1).

In order to divide NSTEMI and UAP by means of a ‘positive’ troponin we defined ‘normal’ or ‘abnormal’ troponin levels using the ‘cut-off’ for ‘positive’ troponins as troponin T [Roche]: ‘Modular E170’, ‘Elecsys 1010’, ‘Elecsys 2010’, ‘COBAS 601’, ‘Cardiac Reader’, ≥0.03ug/L, troponin I [Abbott]: ‘Axysm’ ≥0.04ug/l, troponin I [Abbott]: ‘Architect’ ≥0.03ug/l, troponin I [Abbott]: ‘i-stat’ ≥0.08ug/l, troponin I [Bayer]: ‘Advia centaur’ ≥0.04ug/l, troponin I [Dade Behring]: ‘Dimension’ ≥0.1ug/l.

Hypertension and dyslipidaemia were defined as patients on treatment, or with a previous clinical diagnosis. Patients with diabetes mellitus were those on diet control, oral hypoglycaemic, or insulin treatment. Cardiogenic shock was defined as: a systolic blood pressure of <90 mmHg for at least 30 minutes, or the need for supportive measures to maintain a systolic blood pressure of ≥90 mmHg with end organ hypoperfusion. Sustained ventricular tachycardia was defined as >30 seconds of ventricular tachycardia, or requiring electrical cardioversion.

Statistics—Continuous data are summarised as median and interquartile range. Differences in frequencies were tested using chi-squared procedures or Fishers exact test as appropriate. SAS (SAS Institute Inc, v9.1) was used to perform the analyses. All tests were two-tailed and a 5% significance level was used.

Results

1003 patients with a suspected or definite ACS were admitted to 39 New Zealand hospitals and enrolled in the ACS audit over the 14-day period (Figure 1). Eight patients (7 once and 1 twice) were readmitted within the 2 weeks, all to the same hospital. 134 patients were transferred from their admitting hospital to another institution for further management (128 [96%] to an intervention centre). Over the 2 weeks, 3 hospitals had no ACS admissions, 8 hospitals admitted 40 or more patients of which 2 hospitals (Auckland City and Christchurch) admitted more than 120 patients, from their own catchment area.

Patient demographics: The median age was 67 (IQR 56-78) years. Forty-two percent of patients were female, 77% Caucasian, 9.2% Maori, 3.3% Pacific Islander, 2.7% Indian, 2.0% Asian, 0.9% from another ethnic group and in 4.8% the ethnicity was unspecified. Patient demographics were slightly changed from those in 2002 except that there were fewer Caucasian patients (77% vs 83%, p<0.05), more Maori (9.2% vs 6.7%, p<0.05) and more Pacific Island ethnicity patients (3.3% vs 1.5%, p<0.05) in
In addition, more patients were now identified as being ‘dyslipidaemic’ (50% vs 35%, \(P<0.05\)) and more had previously undergone CABG surgery (9.9% vs 4.6%, \(p<0.05\)) (Table 1).

**Figure 1. New Zealand ACS hospitals and patient numbers: 1003 patients admitted to 39 hospitals**

**Table 1. Baseline patient demographic data for 2007 (n=1003) and comparison with the 2002 Audit baseline demographic data (n=930)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>2002 (n=930)</th>
<th>2007 (n=1003)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age [years] (IQR)</td>
<td>70 (58-78)</td>
<td>67 (56-78)</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>535 (58%)</td>
<td>580 (58%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>768 (83%)</td>
<td>775 (77%)*</td>
</tr>
<tr>
<td>Māori</td>
<td>62 (6.7%)</td>
<td>92 (9.2%)*</td>
</tr>
<tr>
<td>Pacifica</td>
<td>14 (1.5%)</td>
<td>33 (3.3%)*</td>
</tr>
<tr>
<td>Indian</td>
<td>17 (1.8%)</td>
<td>27 (2.7%)</td>
</tr>
<tr>
<td>Asian</td>
<td>8 (0.9%)</td>
<td>20 (2.0%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>56 (6.0%)</td>
<td>47 (4.8%)</td>
</tr>
<tr>
<td>Others</td>
<td>5 (0.5%)</td>
<td>9 (0.9%)</td>
</tr>
</tbody>
</table>
### Smoking
- Current: 171 (18%)
- Previous: 379 (41%)
- Never: 347 (37%)
- Not reported: 33 (4%)

### Hypertension
- 442 (48%)

### Diabetes mellitus
- 161 (17%)

### Dyslipidaemia
- 326 (35%)

### Prior MI
- 325 (35%)

### Prior angiogram
- 325 (31%)

### Prior PCI
- 105 (11%)

### Prior CABG
- 27 (4.6%)

### Prior PVD
- 93 (10%)

### Prior TIA/Stroke
- 119 (11%)

### Prior AF
- 126 (13%)

*P<0.05

IQR: Interquartile range; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; PVD: Peripheral vascular disease; TIA: Transient ischaemic attack; AF: Atrial fibrillation.

### Patient diagnosis:
Using both the admission clinical diagnosis and the measurement of a positive troponin level, we found that 86 (9%) patients presented with a STEMI, 413 (41%) with a NSTEMI, 329 (33%) with UAP, and 175 (17%) with another cardiac or medical diagnosis (Tables 2A, 2B, 2C, 2D). In 2007, compared with 2002, there was a higher percentage of NSTEMI patients (413 (41%) vs 287 (31%), P=0.0025) compared to UAP patients (Table 2B).

### Patient management:
69% of STEMI patients received reperfusion therapy. The majority received fibrinolytic therapy with only 13 (15%) of STEMI patients treated with primary PCI. This rate had, however, increased from 2002 (13 (15%) vs 3 (3%), p=0.0046) (Table 2A).

### Table 2A. Treatments and investigations of STEMI patients: 2002 and 2007

<table>
<thead>
<tr>
<th>Variables</th>
<th>STEMI 2002</th>
<th>STEMI 2007</th>
<th>P: 02 vs 07</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>101 (11%)</td>
<td>86 (8.6%)</td>
<td>0.18</td>
</tr>
<tr>
<td>Treatments in hospital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrinolytic therapy</td>
<td>56 (55%)</td>
<td>47 (55%)</td>
<td>0.87</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>3 (3.0%)</td>
<td>13 (15%)</td>
<td>0.0046</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>33 (34%)</td>
<td>42 (49%)</td>
<td>0.014</td>
</tr>
<tr>
<td>Daltafiban</td>
<td>6 (5.9%)</td>
<td>0</td>
<td>0.024</td>
</tr>
<tr>
<td>UF heparin</td>
<td>28 (28%)</td>
<td>28 (33%)</td>
<td>0.72</td>
</tr>
<tr>
<td>No heparin***</td>
<td>40 (40%)</td>
<td>18 (21%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>5 (5.0%)</td>
<td>2 (2.3%)</td>
<td>0.37</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>2 (2.0%)</td>
<td>1 (1.2%)</td>
<td>0.68</td>
</tr>
<tr>
<td>Abciximab</td>
<td>1 (1%)</td>
<td>2 (2.3%)</td>
<td>0.45</td>
</tr>
<tr>
<td>Aspirin</td>
<td>87 (88%)</td>
<td>81 (94%)</td>
<td>0.082</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>14 (14%)</td>
<td>63 (73%)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>
Overall in 2007, enoxaparin was more widely used in ACS patients than unfractionated heparin (UFH): 52% vs 5.6%, p < 0.0001. However only 339 of 752 (45%) of NSTEMI/UAP patients were treated with any heparin. Glycoprotein 2b/3a receptor blocking agents were used very rarely, in only 13 (1.6%) of ACS patients.

**Cardiac investigations:** Of the 1003 patient admissions, 195 (19%) underwent an echocardiogram, 214 (21%) received an exercise treadmill test, and 317 (32%) received a cardiac angiogram. Compared to 2002, there was no change in the percentage of patients receiving an echocardiogram or exercise test, but more patients in 2007 accessed cardiac angiography (317 (32%) vs 199 (21%), p<0.0001) (Table 3). However, in 2007 of 828 ‘definite’ ACS patients (STEMI, NSTEMI, UAP), half did not undergo either non-invasive or invasive risk stratification with an exercise test or cardiac angiography: 391 (47%) (Table 2D).
## Table 2B. Treatments and investigations of NSTEMI patients: 2002 and 2007

<table>
<thead>
<tr>
<th>Variables</th>
<th>NSTEMI</th>
<th>P: 02 vs 07</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2002 (N (%))</td>
<td>2007 (N (%))</td>
</tr>
<tr>
<td><strong>Treatments in hospital</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrinolytic therapy</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>156 (54%)</td>
<td>271 (66%)</td>
</tr>
<tr>
<td>Dalteparin</td>
<td>33 (12%)</td>
<td>0</td>
</tr>
<tr>
<td>UF heparin</td>
<td>25 (8.8%)</td>
<td>16 (3.9%)</td>
</tr>
<tr>
<td>No heparin***</td>
<td>92 (32%)</td>
<td>132 (32%)</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>6 (2.1%)</td>
<td>5 (1.2%)</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>2 (0.7%)</td>
<td>2 (0.5%)</td>
</tr>
<tr>
<td>Abciximab</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aspirin</td>
<td>228 (79%)</td>
<td>321 (78%)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>35 (13%)</td>
<td>195 (47%)</td>
</tr>
<tr>
<td><strong>Investigations in hospital</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>265 (92%)</td>
<td>360 (87%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>61 (22%)</td>
<td>91 (22%)</td>
</tr>
<tr>
<td>Exercise test</td>
<td>52 (18%)</td>
<td>51 (12%)</td>
</tr>
<tr>
<td>Angiogram</td>
<td>71 (35%)</td>
<td>188 (46%)</td>
</tr>
<tr>
<td>No ETT/Angio</td>
<td>180 (63%)</td>
<td>197 (48%)</td>
</tr>
<tr>
<td>No Echo/Angio</td>
<td>178 (62%)</td>
<td>201 (49%)</td>
</tr>
<tr>
<td>PCI</td>
<td>24 (8.4%)</td>
<td>94 (23%)</td>
</tr>
<tr>
<td>CABG surgery</td>
<td>8 (2.8%)</td>
<td>15 (3.6%)</td>
</tr>
<tr>
<td>In-hospital deaths</td>
<td>10 (3.5%)</td>
<td>8 (1.9%)</td>
</tr>
<tr>
<td><strong>Discharge medications</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>228 (83%)</td>
<td>353 (85%)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>26 (9.5%)</td>
<td>164 (40%)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>177 (63%)</td>
<td>298 (72%)</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td>127 (45%)</td>
<td>230 (56%)</td>
</tr>
<tr>
<td>ARB</td>
<td>0</td>
<td>29 (7%)</td>
</tr>
<tr>
<td>ACE-I or ARB</td>
<td>127 (45%)</td>
<td>255 (62%)</td>
</tr>
<tr>
<td>Statins</td>
<td>153 (55%)</td>
<td>308 (75%)</td>
</tr>
<tr>
<td>Fibrates</td>
<td>7 (2.5%)</td>
<td>9 (2.2%)</td>
</tr>
</tbody>
</table>

**Revascularisation:** Of the 828 ‘definite’ ACS patients, 291 (35%) patients underwent a cardiac angiogram. The majority of these (62%) subsequently underwent revascularisation prior to hospital discharge, more often using percutaneous techniques: 157 (19%) PCI vs 23 (2.8%) CABG surgery (Table 2D). In 2007 more ‘definite’ ACS patients received a cardiac angiogram (35% vs 22%, p=0.0001), and a PCI (19% vs 6.9%, p<0.0001), but there was no increase in patients receiving CABG surgery (2.8% vs 3.5%, p=0.20, Table 2D) compared to 2002.
Table 2C. Treatments and investigations of UAP patients: 2002 and 2007

<table>
<thead>
<tr>
<th>Variables</th>
<th>UAP</th>
<th>2002</th>
<th>2007</th>
<th>P: 02 vs 07</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treatments in hospital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrinolytic therapy</td>
<td></td>
<td>0</td>
<td>1</td>
<td>0.3%</td>
</tr>
<tr>
<td>Primary PCI</td>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Enoxaparin</td>
<td></td>
<td>126</td>
<td>120</td>
<td>0.78</td>
</tr>
<tr>
<td>Daltaparin</td>
<td></td>
<td>39</td>
<td>0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>UF heparin**</td>
<td></td>
<td>22</td>
<td>2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No heparin***</td>
<td></td>
<td>159</td>
<td>207</td>
<td>0.0001</td>
</tr>
<tr>
<td>Tirofiban</td>
<td></td>
<td>1</td>
<td>0</td>
<td>0.33</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0.33</td>
</tr>
<tr>
<td>Abciximab</td>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td>268</td>
<td>210</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td></td>
<td>21</td>
<td>53</td>
<td>0.0002</td>
</tr>
<tr>
<td><strong>Investigations in hospital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td></td>
<td>269</td>
<td>290</td>
<td>0.009</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td></td>
<td>54</td>
<td>25</td>
<td>0.0002</td>
</tr>
<tr>
<td>Exercise test</td>
<td></td>
<td>86</td>
<td>131</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Angiogram</td>
<td></td>
<td>57</td>
<td>53</td>
<td>0.27</td>
</tr>
<tr>
<td>No ETT/Angio</td>
<td></td>
<td>203</td>
<td>165</td>
<td>0.0002</td>
</tr>
<tr>
<td>No Echo/Angio</td>
<td></td>
<td>241</td>
<td>261</td>
<td>0.55</td>
</tr>
<tr>
<td>PCI</td>
<td></td>
<td>13</td>
<td>24</td>
<td>0.57</td>
</tr>
<tr>
<td>CABG surgery</td>
<td></td>
<td>13</td>
<td>4</td>
<td>0.033</td>
</tr>
<tr>
<td>In-hospital deaths</td>
<td></td>
<td>2</td>
<td>0</td>
<td>0.16</td>
</tr>
<tr>
<td><strong>Discharge medications</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td>266</td>
<td>236</td>
<td>0.009</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td></td>
<td>17</td>
<td>52</td>
<td>0.0002</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td></td>
<td>193</td>
<td>170</td>
<td>0.045</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td></td>
<td>128</td>
<td>123</td>
<td>0.66</td>
</tr>
<tr>
<td>ARB</td>
<td></td>
<td>0</td>
<td>18</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ARB or ACE-I</td>
<td></td>
<td>128</td>
<td>141</td>
<td>0.29</td>
</tr>
<tr>
<td>Statins</td>
<td></td>
<td>172</td>
<td>193</td>
<td>0.15</td>
</tr>
<tr>
<td>Fibrates</td>
<td></td>
<td>9</td>
<td>9</td>
<td>0.67</td>
</tr>
</tbody>
</table>

**Discharge medications**: For ‘definite’ ACS patients discharged in 2007 (n=815), compared to patients discharged in 2002 (n=695), the use of aspirin and beta-blockers was unchanged (Table 2D). However, more patients were discharged with an angiotensin converting enzyme-inhibitor/angiotensin receptor blocker (ACE-I/ARB) (55% vs 43%, p<0.0001), and with a statin (70% vs 55%, p<0.0001). In addition, clopidogrel was much more frequently used for ACS patients in 2007, with 269 (33%) of 828 patients being treated with this drug, compared to 57 (8%) of 721 patients: p≤0.0001, in 2002.

**Hospital outcomes**: 16 (1.6%) patients died during their hospital admission: 5 (5.8%) of STEMI patients, 8 (1.9%) of NSTEMI and UAP patients, and 3 (2%) of ‘other cardiac or medical diagnosis’ patients. 34 (3.4%) patients had a recurrent or subsequent myocardial infarction, and 124 (12%) had recurrent angina. Cardiogenic shock developed in 11(1.1%) patients. Three (0.3%) patients received an intra-aortic balloon pump, 2 (0.2%) received a temporary pacemaker, and 4 (0.4%) patients received a permanent pacemaker. 1 patient suffered a stroke and 19 (1.9%) sustained...
ventricular tachycardia. Only 1.5% of patients were enrolled in a research project whilst in hospital.

Table 2D. Treatments and investigations of STEMI, NSTEMI and UAP patients in 2002 (n=721) and in 2007 (n=828)

<table>
<thead>
<tr>
<th>Variables</th>
<th>All “Definite” ACS Pts</th>
<th>P : 02 vs 07</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2002</td>
<td>2007</td>
</tr>
<tr>
<td></td>
<td>N (%</td>
<td>N (%)</td>
</tr>
<tr>
<td><strong>Investigations in hospital</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>623 (86%)</td>
<td>727 (88%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>150 (21%)</td>
<td>169 (20%)</td>
</tr>
<tr>
<td>Exercise test</td>
<td>156 (22%)</td>
<td>192 (23%)</td>
</tr>
<tr>
<td>Angiogram</td>
<td>159 (22%)</td>
<td>291 (35%)</td>
</tr>
<tr>
<td>No ETT/Angio</td>
<td>440 (61%)</td>
<td>391 (47%)</td>
</tr>
<tr>
<td>No Echo/Angio</td>
<td>469 (65%)</td>
<td>478 (58%)</td>
</tr>
<tr>
<td>PCI</td>
<td>50 (7%)</td>
<td>157 (19%)</td>
</tr>
<tr>
<td>CABG surgery</td>
<td>25 (3.5%)</td>
<td>23 (2.8%)</td>
</tr>
<tr>
<td>In-hospital deaths</td>
<td>26 (4%)</td>
<td>13 (1.3%)</td>
</tr>
<tr>
<td><strong>Discharge Medications</strong></td>
<td>721 – 26 less deaths; n=695</td>
<td>828 – 13 less deaths; n=815</td>
</tr>
<tr>
<td>Aspirin</td>
<td>571 (82%)</td>
<td>668 (82%)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>57 (8%)</td>
<td>269 (33%)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>438 (63%)</td>
<td>539 (66%)</td>
</tr>
<tr>
<td>ACE-Inhibitors</td>
<td>298 (43%)</td>
<td>413 (51%)</td>
</tr>
<tr>
<td>ARB</td>
<td>– (0%)</td>
<td>48 (6%)</td>
</tr>
<tr>
<td>ACE –I or ARB</td>
<td>298 (43%)</td>
<td>457 (55%)</td>
</tr>
<tr>
<td>Statins</td>
<td>383 (55%)</td>
<td>577 (70%)</td>
</tr>
<tr>
<td>Fibrates</td>
<td>16 (2%)</td>
<td>18 (2%)</td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>– (0%)</td>
<td>10 (1%)</td>
</tr>
<tr>
<td>Other lipid drug</td>
<td>3 (0%)</td>
<td>6 (1%)</td>
</tr>
<tr>
<td>Warfarin</td>
<td>47 (7%)</td>
<td>62 (8%)</td>
</tr>
</tbody>
</table>

Table 3. Investigations and revascularisation treatments in 2002 and 2007

<table>
<thead>
<tr>
<th>Variables</th>
<th>All Patients</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2002</td>
<td>2007</td>
</tr>
<tr>
<td></td>
<td>(n=930)</td>
<td>(n=1003)</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>794 (85%)</td>
<td>880 (88%)</td>
</tr>
<tr>
<td>Pulmonary oedema **</td>
<td>96 (10%)</td>
<td>78 (8%)</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>184 (20%)</td>
<td>195 (19%)</td>
</tr>
<tr>
<td>Exercise treadmill test</td>
<td>190 (20%)</td>
<td>214 (21%)</td>
</tr>
<tr>
<td>Cardiac angiogram</td>
<td>199 (21%)</td>
<td>317 (32%)</td>
</tr>
<tr>
<td>Exercise test and cardiac angiogram</td>
<td>42 (4.5%)</td>
<td>50 (5.0%)</td>
</tr>
<tr>
<td>Exercise test or cardiac angiogram</td>
<td>347 (37%)</td>
<td>481 (48%)</td>
</tr>
<tr>
<td>Neither exercise test or cardiac angiogram</td>
<td>583 (63%)</td>
<td>522 (52%)</td>
</tr>
<tr>
<td>PCI</td>
<td>69 (7.3%)</td>
<td>158 (16%)</td>
</tr>
<tr>
<td>CABG</td>
<td>35 (3.8%)</td>
<td>25 (2.5%)</td>
</tr>
</tbody>
</table>
Discussion

In a 2-week period in May 2007, 1003 New Zealanders were admitted to hospital with a suspected or definite ACS, of whom 828 patients, after investigations and review, had a final diagnosis of an ACS. If a similar number of ACS patients were admitted throughout the year, approximately 21,500 patients would present with this life-threatening illness. The optimal management of these patients is accepted and widely promulgated in local and international guidelines. As a collaborative group of clinicians we have once again collected data on the management of these patients.

Patient management

In-hospital diagnosis: The New Zealand ACS Audit group data collection is unique, as it collects extensive data from all hospitals in one country and collects all patients admitted within a two-week period. We identified 1003 patients admitted with STEMI (9%), NSTEMI (41%), UAP (33%) or other cardiac or medical diagnoses (17%). In 2007, in comparison to 2002, there was a higher percentage of NSTEMI patients (413 (41%) vs 287 (31%), P=0.0025) probably as a result of the use of lower ‘cut off’ levels for a ‘positive’ troponin which had been adopted for biochemical analysers, as compared to 2002. Hence more patients crossed the ‘threshold’ for a ‘positive’ troponin T and the diagnosis of a NSTEMI.

In-hospital investigations: For STEMI and NSTEMI (heart attack) patients (n=499), with myocardial damage and at the highest risk, the use of guideline recommended echocardiography (29%) and angiography (48%) was low with 43% of patients receiving neither as a method of assessing left ventricular systolic function, an important determinant of long-term prognosis. Furthermore, for the same group, the use of an exercise treadmill test (12%) or a cardiac angiogram (48%) as methods of risk assessment was also low, with 45% of patients not receiving either test. These levels of investigation contrast markedly with the recommendations of guidelines which recommend that all STEMI and NSTEMI patients be considered for assessment in these ways.

Clearly there are some patients with significant co-morbidities, in whom a non-invasive management strategy is appropriate for their care, and who would not be expected to undergo such investigations. In addition, in some patients with a recent admission when cardiac angiography did identify the coronary anatomy, further angiography may not be felt to be required. However, these issues are not unique to New Zealand patients, and to try to assess our rates, particularly of cardiac angiography, a comparison with similar studies in overseas ACS patients is warranted.

International comparisons, Europe—The most recent European Society of Cardiology Survey of 3004 patients from 32 countries in 2004 demonstrated that the rate of investigations was higher in Europe as compared to New Zealand in 2007 (Figure 2) with European STEMI patients receiving a higher rate of echocardiography (83% vs 62%), angiography (70% vs 58%) and PCI (58% vs 45%). NSTEMI patients in Europe, compared to patients in New Zealand, also received a higher rate of echocardiography (72% vs 16%), angiography (63% vs 32%) and PCI (37% vs 16%).
**International comparisons, Australia:** The Australian audit of ACS patients was undertaken over seven months (November 2005 to May 2006) from 39 selected hospitals across all States and territories of Australia. A total of 3402 patients were enrolled: most from ‘metropolitan’ centres, although ‘regional’ (21% patients) and ‘rural’ (3% patients) admissions were included. Australian STEMI patients receiving a similar rate of echocardiography (59% vs 62%), more cardiac angiography (89% vs 58%) and more PCI (68% vs 45%) than New Zealand patients (Figure 2).

**Figure 2. Comparison of New Zealand (2007), European (2004), Australian (2005-2006) and USA (2002-2003) ACS patient surveys for ST-segment-elevation ACS and No ST-segment-elevation ACS patients: investigations and revascularisation treatments**
Non-ST-Elevation ACS-High Risk (NSTEACS-HR) and Non-ST-Elevation ACS-Intermediate Risk (NSTEACS-IR) patients in Australia, compared to NSTEMI and UAP patients in New Zealand, also received a higher rate of echocardiography (30% vs 16%) and cardiac angiography (48% vs 32%) with a similar rate of PCI (18% vs 16%).

**International comparisons, United States of America (USA)**—The CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of the ACC/AHA Guidelines) audit from the USA has reviewed the data from 400 participating hospitals and 43,317 high-risk NSTEMI ACS patients with positive cardiac markers and/or ischaemic ST-segment changes. NSTEMI patients in the USA, compared to patients in New Zealand (Figure 2), received a higher rate of cardiac angiography (66% vs 32%), PCI (37% vs 16%) and CABG (12% vs 2.6%).

**International comparisons, GRACE Registry**—A comparison can also be made with the 44, 372 patients enrolled in the International GRACE Registry in 2005, which included patients from 95 hospitals in 14 countries in North and South America, Europe, Australia and New Zealand (two sites). GRACE STEMI patients in 2005 received a higher rate of angiography (80% vs 58%) and PCI (64% vs 45%) compared to New Zealand patients in 2007, and GRACE NSTEMI patients also received a higher rate of angiography (63% vs 32%) and PCI (35% vs 16%).

With this high level of invasive management, STEMI patients from 1999 to 2005 in the GRACE cohort have shown a decline in in-hospital death (8.4% to 4.6%) and heart failure (19.5% to 11%). In addition, for NSTEMI patients from 1999 to 2005, the GRACE cohort has also been able to detect a decline in in-hospital death (2.9% to 2.2%) and heart failure (13% to 6.1%).

**Medical management of NSTEMI patients**—We found low levels of use of heparin for NSTEMI patients, with 32% not accessing this treatment. In comparison, 27% of European NSTEMI patients did not receive heparin, and neither did 17% of USA patients. The use of a glycoprotein 2b/3a inhibitor was very low in New Zealand NSTEMI patients with only 1.7% of patients receiving this therapy in 2007. The numbers were also low in 2002 (2.8%). In comparison, 21% of European NSTEMI patients and 35% of USA patients were treated with this effective strategy.

**Discharge medications**—The European, Australian and USA ACS patient surveys have all shown a higher uptake of evidence-based, prognostically advantageous, secondary prevention medication (Figure 3). The use of clopidogrel was particularly low in New Zealand patients.

In 1996, the Clopidogrel versus Aspirin in Patients at Risk of Ischaemic Events (CAPRIE) trial reported that in 19, 185 patients clopidogrel was superior to aspirin over 1 to 3 years of treatment, reducing ischaemic stroke, MI, or vascular death by 8.7% (p=0.04). In 1999, Australian patients were able to access this therapy, but in New Zealand, not even patients strongly allergic to aspirin, could access funding for clopidogrel. Subsequent, large randomised controlled clinical trials have demonstrated the benefit of clopidogrel treatment in NSTEMI patients, patients undergoing a coronary stent, and STEMI patients receiving fibrinolytic therapy. As a result of these and
other data, the European,\textsuperscript{2,3} USA\textsuperscript{4,5} and New Zealand\textsuperscript{6,7} ACS Guidelines recommend clopidogrel use for ACS patients for 9 to 12 months after presentation, and for 12 months after a coronary stent. However, from 1996 to 2007, New Zealand patients could only access clopidogrel funding [via the hospital service] for 3 weeks after a stent, and not for an ACS presentation.\textsuperscript{12}

Figure 3. Comparison of New Zealand (2007), European (2004), Australian (2005-2006) and USA (2002-2003) ACS patient surveys for ST-segment-elevation ACS and No ST-segment-elevation ACS patients: discharge medications

![Figure 3](image)

Then, in October 2006, ACS patients were given limited funding for 3 to 6 months and stent patients for six months. The low numbers of patients receiving clopidogrel,
both in the 2002 and the 2007 Audit will have been significantly influenced by this limited funding by PHARMAC.

Most recent USA data from the ‘Get with the Guidelines’ ACTION Audit—The most recent USA Audit data comes from the American Heart Association’s ‘Get with the Guidelines (GWTG) ACTION’ Audit from data collected from 1 January 2007 to 31 March 2009 from 343 sites. 46,245 STEMI and 71,536 NSTEMI patients are represented, with 93% and 88% patients receiving a diagnostic cardiac angiogram, of whom 89% and 55% of patients respectively received this within 24 hours of presentation (Personal correspondence: Drs Harrington and Roe, Duke University, USA). 72% and 42% of patients received a GP2b/3a receptor antagonist, and 90% and 86% of patients received heparin treatment, respectively.

At hospital discharge, the rates of use of proven medications for STEMI and NSTEMI patients were aspirin (98%, 97%), clopidogrel (91%, 74%), beta-blocker (97%, 95%), ACE-inhibitor/ARB (78%, 70%), and statins (92%, 86%). The clear message from this data is that with vigorous use of Guidelines and efficient hospital processes, major improvements in the use of proven medication can be achieved.

Improving quality of care

Access to investigations and treatments—Data from the second NZ ACS Audit Group have identified limitations to the care of ACS patients in New Zealand. The major issue in 2007 appears to be the limited availability of modern invasive management, although with a small, but encouraging improvement from the 2002 audit. The access to CABG surgery, however, remains very limited.

In addition, the availability of non-invasive assessment tests, especially echocardiograms and exercise treadmill tests, have not improved from 2002. Further there are areas of patient management, such as the use of proven medication both within hospitals, and at hospital discharge, where significant improvements could be delivered.

Organisation of ACS services—In light of our data, one proposed solution would be to organise National ACS services based upon a Regional strategy from each of the 5 major cardiothoracic centres: Auckland City Hospital, Waikato Hospital, Wellington Hospital, Christchurch Hospital and Dunedin Hospital.

An integrated approach to providing service should be considered. In particular necessary resource should be provided centrally to improve rates of angiography, PCI and CABG surgery, and to peripheral centres, for provision of echocardiography and exercise treadmill testing in particular. The concept of the ‘hub and spoke’ approach to care of ACS patients also requires coordination of patient transfer in a timely manner, to access invasive assessment and management.

Planning for acute and semi-acute transfers, similar to that already available for trauma patients in New Zealand, has the potential to markedly improve patient outcomes, and the health of many thousands of New Zealand ACS patients a year.

Governance of ACS services—There appears to be a need for Cardiologists and Physicians in New Zealand to have a major role in the planning of ACS patient services. That New Zealand’s health system is subject to ‘government rather than
governance’ is critically explored and discussed by respected academics: Professors Gorman and Scott from the University of Auckland, who, along with cardiologists have outlined the need for practicing clinicians to be a major part of health service management.

Management programmes—At a hospital level, observations among 64,775 patients drawn from 350 USA centres showed that higher rates of adherence to guidelines correlated with lower rates of in-hospital mortality. At the patient level, a clear gradient of increasing mortality risk can be observed among patients with acute coronary syndromes discharged on fewer evidence-based secondary prevention therapies.

In a study of 1385 patients, being discharged on all guidelines-advocated therapies, there was a 10-fold lower risk of mortality by six months compared with the risk in those discharged on none (OR 0.10, 95% CI: 0.03–0.42; p<0.0001). In addition to the need for improved health promotion strategies aimed at encouraging earlier presentation to hospital, specific local programmes facilitating implementation and ongoing compliance with life-saving evidence-based therapies offer a substantial capacity to reduce mortality, and could be readily introduced to the New Zealand environment.

Study limitations—As with 2002, we made significant efforts to enrol all ACS patients into the audit. In 2002 we estimated that the number of patients ‘missed’ was 4%. We expect that similar numbers of patients were ‘missed’ in 2007; these patients are not included nor further considered in this audit. The scope of the NZ ACS Audit did not include an assessment of individual hospital risk tools, availability or attendance of rehabilitation, nor more in-depth data on contra-indications for investigations or therapies. Neither did we collect data on defibrillator device use, nicotine patch use, or on the acute care of diabetes mellitus patients which was varied across centres.

Conclusions

The Cardiac Society of New Zealand Acute Coronary Syndrome Audit Group has undertaken a second nationwide audit of the management of ACS patients. The audits main finding is that there are still low levels of investigations, evidence-based treatments and revascularisation undertaken for New Zealand ACS patients in comparison to overseas experiences. The reasons for this are likely to be multifactorial.

Despite some small, but encouraging increased access to angiography and subsequent revascularisation with PCI from 2002, there has been no improvement in the access to non-invasive testing. CABG surgery remains a very limited management option. There has been an increase in the use of some, but not all, proven medications. There remains a need for a comprehensive national strategy to improve all aspects of ACS patient management.
Competing interests: None known.

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We thank these audit leaders and assistants in the following hospitals—from north to south by region (patient numbers in the study are given inside brackets; #Chairman; *Steering Committee member).

Auckland/Northland (North Island)

Kaitaia Hospital: Dr E Jeffrey (9). Dargaville Hospital: Dr D Gibbons (5). Rawene Hospital: Dr K Blattner (0). Kawakawa Hospital: Dr A Murray, Ms S August (3). Whangarei Hospital: Dr N Harrison, Dr B Wong, Ms K O’Keefe (37). North Shore Hospital, Auckland: Dr H Hart, Dr T Scott, Ms E Fairhurst, Ms W Young (66). Waitakere Hospital: Dr H Hart, Dr T Scott, Ms J Hewlett (24). Auckland City Hospital: Dr C Ellis #*, Prof H White*, Mr G Gamble*, Dr A Chateleine (122). Mercy Private Hospital, Auckland: Dr C Ellis, Ms C McGarrigle (6). Ascot Private Hospital, Auckland: Dr A Maslowski (2). Middlemore Hospital, Auckland: Dr A Kerr, Dr M Lund, Dr J Goh (35).

Waikato (Central North Island)

Thames Hospital: Dr J Lennane, Dr Aftabuzzaman (19). Tauranga Hospital: Dr J Tisch, Dr G Porter, Dr C Young, Ms W Bryson, Ms J Goodson (44). Waikato Hospital, Hamilton: Dr G Devlin*, Ms B Killion, Ms A Silverstone, Ms L Boenders (56). Whakatane Hospital: Dr E Edwards, Dr R Steeper, Ms D Garner (12). Rotorua Hospital: Dr N Crook, Ms A Morley (28). Tokoroa Hospital: Dr N Thornton, Dr F Kanan (2). Te Kuiti Hospital: Dr N Thornton, Dr K Buswell, Ms T Te Wano (0). Taupo Hospital: Dr K Logan (11). Gisborne Hospital: Dr C Duffy, Ms K Weytmans, Ms T Low (16). Taumarunui Hospital: Dr N Thornton, Dr H Wahid (1). New Plymouth Hospital: Dr I Ternouth, Dr T Boswell (17).
Wellington (Southern North Island, Upper South Island)

Hawkes Bay Regional Hospital, Hastings: Dr R Luke, Ms J MacKenzie (76).
Wanganui Hospital: Dr T Thompson, (26). Palmerston North Hospital: Dr D Tang (25). Masterton Hospital: Dr T Matthews, Ms K Lee (10). Hutt Hospital: Dr T O’Meeghan, Ms J Dewar, Ms M Klientjes (25). Wellington Hospital: Dr P Matsis*, Ms D Middleditch, Ms E Walsh (57). Wakefield Private Hospital, Wellington: Dr M Abernethy (0). Nelson Hospital: Dr A Hamer, Ms R Price (14). Blenheim Hospital: Dr M Heynike, Ms M Udy (12).

Christchurch/Canterbury (Central South Island)

Greymouth Hospital: Dr U Bopitiya, Ms L Skeats (6). Christchurch Hospital: Assoc Prof J Elliott*, Prof M Richards*, Ms L Skelton, Ms L Frost (141). Ashburton Hospital: Dr A Obafemi, Ms A Smart (3). Timaru Hospital: Dr M Hills, Ms Maria Hammond, Ms C Barker (31).

Dunedin/Otago (Southern South Island)

Oamaru Hospital: Dr D Phillips, Ms S McCulloch (10). Dunstan Hospital, Clyde: Dr G Nixon, Ms J Coutts (3). Dunedin Hospital: Assoc Prof MJA Williams*, Ms M McLelland (28). Invercargill Hospital: Dr A Maloney, Dr R Anand (21).

Correspondence: Dr Chris Ellis, Chairman of the NZACS Audit Group, Cardiology Department, Green Lane CVS Services, Level 3, Auckland City Hospital, Grafton, Auckland 1023, New Zealand. Email: chrise@adhb.govt.nz

References:


ACS patients in New Zealand experience significant delays to access cardiac investigations and revascularisation treatment especially when admitted to non-interventional centres: results of the second comprehensive national audit of ACS patients

Chris Ellis, Gerard Devlin, John Elliott, Philip Matsis, Michael Williams, Greg Gamble, Andrew Hamer, Mark Richards, Harvey White; for the New Zealand Acute Coronary Syndromes (NZACS) Audit Group

Abstract

Aim To compare the management of acute coronary syndrome (ACS) patients presenting to interventional versus non-interventional New Zealand hospitals, with emphasis, on access delays for invasive assessment and revascularisation treatments.

Methods Using data collected by the New Zealand Cardiac Society ACS Audit Group over 14 days from each hospital in New Zealand (n=39) that admits ACS patients, patient management at intervention centres (6 public, 3 private) was compared with non-intervention centres (30 public). Investigations and revascularisation procedures performed on transferred patients were attributed to the referring centre.

Results From 00.00 hours on 14 May 2007 to 24.00 hours on 27 May 2007, 1003 patients were admitted to a New Zealand hospital with a suspected or definite ACS: ST-segment-elevation myocardial infarction [STEMI] (8%), non-STEMI [NSTEMI] (41%), unstable angina pectoris [UAP] 33%, or another cardiac or medical diagnosis (17%). Patients admitted to a non-intervention centre (n=556) were older (median age 70 vs 66 years, p=0.0097), with similar risk factors, and were more likely to be of Māori (12% vs 5.8%, p<0.0001), and less likely to be of Indian (1.3% vs 4.5%, p=0.0026) or Pacific Island (2.0% vs 4.9%, p=0.012) ethnicity. Patients admitted to a non-intervention centre were less likely to have a chest X-ray performed (84% vs 93 %, p<0.0001), but, as likely to have an echocardiogram, exercise test, or cardiac angiogram for cardiac risk assessment as patients admitted to an intervention centre (n=447). However, only 1 in 2 patients overall underwent either treadmill testing or angiography, and only 1 in 3 underwent angiography. Time delays to access cardiac angiography were evident with only 23% of all patients receiving this test within 48 hours of hospital admission. Patients at non-intervention centres had a significantly longer median wait for cardiac angiography than those admitted to an intervention centre (5.1 vs 2.5 days, p<0.0001).

Conclusions Patients admitted to a New Zealand hospital with an acute coronary syndrome experience delays in accessing investigations and subsequent revascularisation. Furthermore, inequity exists with delays being significantly longer for patients admitted to a non-intervention centre. A comprehensive national strategy is needed to improve access to optimal cardiac care.
In New Zealand there has been a more than doubling of hospital discharges for a heart attack from 1989 to 2002/2003.\(^1\) Hence there is a significant clinical problem which mandates an efficient management strategy. Current management of ‘high-risk’ ACS patients includes optimal medical treatment and an invasive revascularisation strategy guided by cardiac angiography, as recommended by international\(^2\)\(^-\)^\(^5\) and local\(^6\)^\(^,\)^\(^7\) clinical guidelines. A previous comprehensive nationwide audit of ACS patients undertaken in 2002,\(^8\) found inequitable management across New Zealand, as patients admitted to a hospital without cardiac interventional facilities received fewer investigations and less revascularisation treatment than patients admitted to intervention centres.\(^9\)

Smaller local studies in Taranaki/Waikato,\(^10\) Invercargill/Dunedin\(^11\) and North Shore Hospital/Auckland City Hospital, Auckland\(^12\) have all demonstrated a limited access to invasive investigations and revascularisation of ACS patients admitted to non-interventional centres. Patients from Invercargill\(^11\) experienced a 2.5-fold lower rate of angiography and revascularisation than those admitted directly to Dunedin hospital. In-hospital mortality differed by 3.3% (10.7% vs 6.4%) and then widened to approximately 10% at both 6 months (19.1% vs 9.6%), 12 months (22.1% vs 12.1%) and up to 5 years.\(^13\)

We aimed to determine the current management of ACS patients presenting to interventional and non-interventional New Zealand hospitals. The primary goal of this study, in particular, was to assess cardiac investigations and treatments received by patients and examine time delays experienced by patients in accessing management.

**Methods**

**Data collection**—The development of the New Zealand Acute Coronary Syndrome (NZACS) Audit Group and the methodology for the national audit, which was supported by the Cardiac Society of New Zealand, has been published elsewhere.\(^14\) The inclusion criterion for the audit was ‘a patient admitted overnight with a suspected or definite acute coronary syndrome’.

Following admission and investigations, a ‘discharge diagnosis’ was subsequently determined by the local clinical team who confirmed the diagnosis of an ACS, as a STEMI, NSTEMI or UAP, or determined a ‘non-ACS’ presentation resultant on investigations undertaken in hospital and the patients clinical course. An extensive four-page case report form was used to obtain patient demographics, initial and discharge diagnosis, medication use in hospital and at discharge, as well as investigations undertaken and invasive treatments received by patients. Ethnicity was self-reported at hospital admission.

Data from the NZACS Audit were used to compare patients’ presentation and management at intervention centres (6 public hospitals and 3 private hospitals), with non-interventional centres (30 public hospital) [Table 1]. One centre (Middlemore Hospital, Auckland), was able to undertake cardiac angiograms and PCIs throughout the working week (08.00 to 16.00, Monday to Friday) but not “out of hours”, and did not perform CABG operations, and was classified as an intervention centre. One centre (Nelson Hospital) was able to undertake some angiography and a limited number of PCI procedures, but had a limited operation with only one interventional cardiologist performing PCI, and was also unable to perform CABG operations, and hence was classified as a non-interventional centre.
Table 1. Admissions and transfers to intervention (n=447) and non-intervention (n=556) centres

<table>
<thead>
<tr>
<th>Facilities</th>
<th>No. of own patients</th>
<th>Transferred in</th>
<th>Total Admissions</th>
<th>Transferred Out</th>
<th>Angiogram</th>
<th>PCI</th>
<th>CABG</th>
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**Non-Intervention Centres (n=30)**

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<th>Total Admissions</th>
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<th>Angiogram</th>
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</tr>
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</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting; Y: yes; N: no; *Acquired angiography and PCI capability during working hours since the Audit in 2007.

An additional four public hospitals (Tauranga, New Plymouth, Hastings and Palmerston North) were classified as non-intervention centres as they had the ability to perform some cardiac angiograms, but not PCI or CABG surgery. Cardiac angiography, PCI and CABG surgery was also performed at a private hospital in Christchurch; however this hospital does not plan to admit ACS patients and hence is not further considered with this audit. Investigations and revascularisation of transferred patients was performed during working hours.
attributed to the referring centre. Data were collected from 0000 hours on Monday 14 May to 2400 hours on Sunday 27 May 2007.

Statistics—Continuous data were summarised as median and interquartile range (IQR) and compared using the Wilcoxon rank sums test. Differences in frequencies were tested using standard chi-squared procedures or Fishers exact test as appropriate. All analyses were conducted using SAS (SAS Institute Inc v9.1). All tests were two-tailed and a 5% significance level was maintained throughout.

Results

Admissions and transfers—Over the 14-day period, 1003 suspected or definite ACS patients were admitted to an intervention centre (447) or to a non-intervention centre (556). Eight patients were re-admitted within the 2 weeks (seven once and one twice), all to the same hospital. One hundred and thirty-four patients were transferred to another institution for further management (128 (96%) to an intervention centre). Data from patients transferred were attributed only to the hospital to which they were initially admitted (Table 1).

Table 2. Baseline demographic data of patients admitted to an intervention or non-intervention centre (n=1003)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hospital type</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n=447)</td>
<td>Non-intervention (n=556)</td>
</tr>
<tr>
<td>Age median <a href="range">years</a></td>
<td>66 (18-97)</td>
<td>70 (23-95)</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>265 (59%)</td>
<td>315 (57%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>340 (76%)</td>
<td>435 (78%)</td>
</tr>
<tr>
<td>Māori</td>
<td>26 (5.8%)</td>
<td>66 (12%)</td>
</tr>
<tr>
<td>Pacifica</td>
<td>22 (4.9%)</td>
<td>11 (2.0%)</td>
</tr>
<tr>
<td>Asian</td>
<td>13 (2.9%)</td>
<td>7 (1.3%)</td>
</tr>
<tr>
<td>Indian</td>
<td>20 (4.5%)</td>
<td>7 (1.3%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>18 (4.0%)</td>
<td>29 (5.2%)</td>
</tr>
<tr>
<td>Other</td>
<td>8 (1.8%)</td>
<td>1 (0.18%)</td>
</tr>
<tr>
<td>Tobacco smoker</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>83 (19%)</td>
<td>96 (17%)</td>
</tr>
<tr>
<td>Previous</td>
<td>157 (35%)</td>
<td>232 (42%)</td>
</tr>
<tr>
<td>Current and Previous</td>
<td>240 (56%)</td>
<td>328 (62%)</td>
</tr>
<tr>
<td>Never</td>
<td>188 (42%)</td>
<td>199 (36%)</td>
</tr>
<tr>
<td>Not reported</td>
<td>19 (4%)</td>
<td>29 (5%)</td>
</tr>
<tr>
<td>Hypertension (drug treatment)</td>
<td>231 (52%)</td>
<td>291 (52%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>93 (15%)</td>
<td>89 (16%)</td>
</tr>
<tr>
<td>Type 1</td>
<td>3 (3%)</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Type 2</td>
<td>89 (96%)</td>
<td>86 (97%)</td>
</tr>
<tr>
<td>Not defined</td>
<td>1 (1%)</td>
<td>0</td>
</tr>
<tr>
<td>Dyslipidaemia (drug treatment)</td>
<td>217 (49%)</td>
<td>257 (46%)</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>138 (31%)</td>
<td>180 (33%)</td>
</tr>
<tr>
<td>MI within 1 month</td>
<td>13 (2.9%)</td>
<td>20 (3.6%)</td>
</tr>
<tr>
<td>Prior angiogram</td>
<td>151 (34%)</td>
<td>146 (26%)</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>72 (16%)</td>
<td>78 (14%)</td>
</tr>
<tr>
<td>within 6 months</td>
<td>16 (3.6%)</td>
<td>25 (4.5%)</td>
</tr>
<tr>
<td>Prior CABG</td>
<td>49 (11%)</td>
<td>46 (8%)</td>
</tr>
<tr>
<td>Prior TIA/Stroke</td>
<td>56 (13%)</td>
<td>58 (10%)</td>
</tr>
</tbody>
</table>

MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting
TIA: Transient ischaemic attack.
Baseline demographics—Patients admitted to a non-intervention centre were older (median age 70 vs 66 years, p=0.0097), with similar risk factors except were more likely to have smoked (232[42%], vs 157[35%], p=0.037), and more likely to be of Māori (12% vs 5.8%, p=0.0009), but less likely to be of Pacific Island (2% v 4.9%, P=0.012) or Indian (1.3% v 4.5%, p=0.0026) ethnicity. They were less likely to have had a prior cardiac angiogram (26% vs 34%, p=0.012, Table 2).

In-hospital investigations—Overall, patients admitted to a non-intervention centre were less likely to have a chest X-ray performed (84% vs 93 %, p<0.0001), but as likely to have an echocardiogram, exercise test, or cardiac angiogram for cardiac risk assessment as patients admitted to an intervention centre (Tables 3, 4).

Time-delays for invasive investigations—For the entire cohort (n=1003), similar numbers of patients at non-intervention and intervention centres received diagnostic cardiac angiography (33% vs 30%) (Table 3). There was generally an important delay for patients awaiting cardiac angiography, with a median time to angiography of 4.0 (IQR 2.0–6.1) days with only 23% patients receiving an angiogram within 48 hours (Table 5).

Table 3. Investigations and revascularisations (all patients: n=1003)

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Hospital type</th>
<th>Intervention (n=447)</th>
<th>Non-Intervention (n=556)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td></td>
<td>414 (93%)</td>
<td>466 (84%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td></td>
<td>92 (21%)</td>
<td>103 (19%)</td>
<td>0.41</td>
</tr>
<tr>
<td>Exercise test</td>
<td></td>
<td>88 (20%)</td>
<td>126 (23%)</td>
<td>0.25</td>
</tr>
<tr>
<td>Angiogram</td>
<td></td>
<td>133 (30%)</td>
<td>184 (33%)</td>
<td>0.26</td>
</tr>
<tr>
<td>Exercise test or angiogram</td>
<td></td>
<td>207 (46%)</td>
<td>274 (46%)</td>
<td>0.35</td>
</tr>
<tr>
<td>Exercise test and angiogram</td>
<td></td>
<td>14 (3.1%)</td>
<td>36 (6%)</td>
<td>0.016</td>
</tr>
<tr>
<td>Neither exercise test nor angiogram</td>
<td></td>
<td>240 (54%)</td>
<td>282 (51%)</td>
<td>0.35</td>
</tr>
<tr>
<td>PCI</td>
<td></td>
<td>68 (15%)</td>
<td>90 (16%)</td>
<td>0.67</td>
</tr>
<tr>
<td>CABG</td>
<td></td>
<td>11 (2.5%)</td>
<td>14 (2.5%)</td>
<td>0.95</td>
</tr>
</tbody>
</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting

The delay to angiography for non-intervention centre patients at 5.1 (IQR 3.6–7.1) days was more than for intervention centre patients at 2.5 (IQR 1.2–4.6, p<0.0001) days (Table 5, Figure 1). However by region the median time to angiography for Christchurch (3.6 [IQR 1.8–5.8] days) and Otago (2.1 [IQR 2.0–6.1] days) was significantly lower than for other regions (P=0.0008) (Figure 2). Patients admitted to hospital on a Friday or Saturday waited longer for an angiogram, than those admitted on a Sunday to Thursday with 13% vs 29% (P=0.0036) accessing angiography within 48 hours (Figure 3).

In-hospital medical treatments—For STEMI patients (n=86), reperfusion treatment rates were similar between non-intervention and intervention centres (69% vs 70%) although primary PCI was less often received in non-intervention centres (3.7% vs 34%, p<0.05), compared to thrombolytic therapy. For NSTEMI patients (n=413),
non-intervention centres were more likely to use heparin (71% v 65%, p<0.05) (Table 6).

Figure 1. Days from admission to angiogram

![Figure 1](image1)

Figure 2. Median time to angiogram (± IQR) from admission by region.

![Figure 2](image2)
For UAP patients, non-intervention centres used more aspirin (77% vs 51%, p<0.0001), clopidogrel (22% vs 10%, p=0.0035), and heparin (57% vs 18%, p<0.0001) treatment (Table 7).

**Time delays for invasive treatments**—For definite ACS patients (n=828), similar numbers at non-intervention and intervention centres received PCI (20% vs 18%) and CABG (2.7% vs 2.9%) (Table 4). Non-intervention patients waited longer for PCI: 131 vs 48 hours, p<0.0001. For patients waiting for in-hospital CABG surgery, the median delay following cardiac angiography was 6.9 (IQR 5.4, 15.6) days and was not different for patients from either centre (10.5 vs 6.7 days, p=0.56).

**Table 4. Investigations and revascularisations (patients with a ‘definite’ ACS: STEMI, NSTEMI, UAP n=828)**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Intervention (n=376)</th>
<th>Non-intervention (n=452)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest X-ray</td>
<td>349 (93%)</td>
<td>378 (84%)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>82 (22%)</td>
<td>87 (19%)</td>
<td>0.36</td>
</tr>
<tr>
<td>Exercise test</td>
<td>80 (21%)</td>
<td>112 (25%)</td>
<td>0.23</td>
</tr>
<tr>
<td>Angiogram</td>
<td>125 (33%)</td>
<td>166 (37%)</td>
<td>0.30</td>
</tr>
<tr>
<td>Exercise test or angiogram</td>
<td>191 (51%)</td>
<td>246 (54%)</td>
<td>0.30</td>
</tr>
<tr>
<td>Exercise test and angiogram</td>
<td>14 (3.7%)</td>
<td>32 (7.1%)</td>
<td>0.036</td>
</tr>
<tr>
<td>Neither exercise test nor angiogram</td>
<td>185 (49%)</td>
<td>206 (46%)</td>
<td>0.30</td>
</tr>
<tr>
<td>PCI</td>
<td>68 (18%)</td>
<td>89 (20%)</td>
<td>0.56</td>
</tr>
<tr>
<td>CABG</td>
<td>11 (2.9%)</td>
<td>12 (2.7%)</td>
<td>0.81</td>
</tr>
</tbody>
</table>

PCI: Percutaneous coronary intervention; CABG: Coronary artery bypass grafting.
Table 5. Number of procedures and time delays for New Zealand centres

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Intervention</th>
<th>Non-Intervention</th>
<th>P</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of stay (days)</td>
<td>3 (1, 4)</td>
<td>4 (2, 7)</td>
<td>&lt;0.0001</td>
<td>3 (1, 6)</td>
</tr>
<tr>
<td>No. transfuse (complete data)</td>
<td>-</td>
<td>128</td>
<td>-</td>
<td>131</td>
</tr>
<tr>
<td>Length of stay (onset of base line)</td>
<td>-</td>
<td>4.5 (3.7)</td>
<td>122</td>
<td>4.5 (3.7)</td>
</tr>
<tr>
<td>Length of Stay (transfer time)</td>
<td>-</td>
<td>7.2 (1.11)</td>
<td>130</td>
<td>7 (5.11)</td>
</tr>
</tbody>
</table>

Discharge medications—The use of aspirin, clopidogrel, beta-blockers, angiotensin converting enzyme (ACE)-inhibitors/angiotensin receptor blockers and statins was broadly similar for STEMI and UAP patients. However, fewer NSTEMI patients from a non-intervention centre were discharged with aspirin (83 vs 88%, p< 0.05) or clopidogrel (22% vs 43%, p< 0.05) (Tables 6, 7).

Table 6. Investigations and treatments of ST-segment-elevation myocardial infarction (STEMI) (n=86) and NSTEMI (n=413) patients according to hospital type

<table>
<thead>
<tr>
<th>Investigation</th>
<th>STEMI Intervention</th>
<th>STEMI Non-Intervention</th>
<th>P</th>
<th>NSTEMI Intervention</th>
<th>NSTEMI Non-Intervention</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>32</td>
<td>54</td>
<td>178</td>
<td>235</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrinolytic</td>
<td>11 (35%)</td>
<td>36 (67%)</td>
<td>0.0067</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>11 (34%)</td>
<td>2 (3.7%)</td>
<td>&lt;0.0001</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Aspirin</td>
<td>30 (94%)</td>
<td>54 (94%)</td>
<td>0.14</td>
<td>143 (80%)</td>
<td>178 (76%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>26 (81%)</td>
<td>37 (68%)</td>
<td>0.22</td>
<td>77 (43%)</td>
<td>118 (50%)</td>
<td>0.17</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>15 (47%)</td>
<td>27 (50%)</td>
<td>0.83</td>
<td>109 (61%)</td>
<td>162 (69%)</td>
<td>0.12</td>
</tr>
<tr>
<td>UF Heparin</td>
<td>10(31%)</td>
<td>18 (33%)</td>
<td>0.99</td>
<td>9 (5.0%)</td>
<td>7 (3.0%)</td>
<td>0.31</td>
</tr>
<tr>
<td>No Heparin*</td>
<td>8 (25%)</td>
<td>10 (18%)</td>
<td>0.59</td>
<td>63 (35%)</td>
<td>69 (29%)</td>
<td>0.20</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>0</td>
<td>2 (3.7%)</td>
<td>0.53</td>
<td>3 (1.8%)</td>
<td>2 (0.9%)</td>
<td>0.66</td>
</tr>
<tr>
<td>Epitifibatide</td>
<td>1 (3.3%)</td>
<td>0</td>
<td>0.37</td>
<td>2 (1.2%)</td>
<td>0</td>
<td>0.19</td>
</tr>
<tr>
<td>Abciximab</td>
<td>2 (6.6%)</td>
<td>0</td>
<td>0.14</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>28 (88%)</td>
<td>49 (91%)</td>
<td>0.72</td>
<td>168 (94%)</td>
<td>192 (82%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>24 (75%)</td>
<td>29 (54%)</td>
<td>0.067</td>
<td>48 (27%)</td>
<td>43 (18%)</td>
<td>0.042</td>
</tr>
<tr>
<td>Exercise test</td>
<td>1 (3.1%)</td>
<td>9 (17%)</td>
<td>0.083</td>
<td>16 (9%)</td>
<td>35 (15%)</td>
<td>0.096</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th></th>
<th>UAP: Intervention</th>
<th>UAP: Non-intervention</th>
<th>P‡</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>166</td>
<td>162</td>
<td></td>
</tr>
<tr>
<td><strong>Treatments</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>85 (51%)</td>
<td>125 (77%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>17 (10%)</td>
<td>36 (22%)</td>
<td>0.0035</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>30 (18%)</td>
<td>90 (55%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>UF Heparin</td>
<td>0</td>
<td>2 (1.3%)</td>
<td>0.15</td>
</tr>
<tr>
<td>No Heparin*</td>
<td>136 (82%)</td>
<td>71 (44%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>0</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>0</td>
<td>1 (0.6%)</td>
<td>0.31</td>
</tr>
<tr>
<td>Abciximab</td>
<td>0</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td><strong>Investigations in hospital</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>153 (92%)</td>
<td>137 (84%)</td>
<td>0.023</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>10 (6.1%)</td>
<td>15 (9.2%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Exercise test</td>
<td>63 (38%)</td>
<td>68 (42%)</td>
<td>0.49</td>
</tr>
<tr>
<td>Angiogram</td>
<td>21 (13%)</td>
<td>32 (20%)</td>
<td>0.085</td>
</tr>
<tr>
<td>No ETT or Angio</td>
<td>89 (54%)</td>
<td>76 (47%)</td>
<td>0.21</td>
</tr>
<tr>
<td><strong>Revascularisation in hospital</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>5 (3.0%)</td>
<td>19 (12%)</td>
<td>0.0026</td>
</tr>
<tr>
<td>CABG</td>
<td>2 (1.2%)</td>
<td>2 (1.3%)</td>
<td>0.99</td>
</tr>
<tr>
<td><strong>In-hospital deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>–</td>
</tr>
</tbody>
</table>

^ Comparing intervention and non-intervention groups * Neither Enoxaparin or UF heparin; ** In days (median and Interquartile range); PCI: Percutaneous coronary intervention UF: Unfractionated ETT: Exercise treadmill test; CABG: Coronary artery bypass grafting Angio: Cardiac angiogram ACE: Angiotensin converting enzyme.

Table 7. Investigations and treatments of unstable angina pectoris (UAP) (n=329) patients according to hospital type
Discharge medications

<table>
<thead>
<tr>
<th></th>
<th>Non-intervention centres</th>
<th>Intervention centres</th>
<th>Total delay (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>115 (69%)</td>
<td>121 (74%)</td>
<td>0.32</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>17 (10%)</td>
<td>35 (21%)</td>
<td>0.0052</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>74 (45%)</td>
<td>96 (59%)</td>
<td>0.0094</td>
</tr>
<tr>
<td>ACE-inhibitors</td>
<td>61 (37%)</td>
<td>62 (38%)</td>
<td>0.81</td>
</tr>
<tr>
<td>ARB</td>
<td>9 (5.5%)</td>
<td>9 (5.5%)</td>
<td>0.99</td>
</tr>
<tr>
<td>ARB or ACE-I</td>
<td>70 (42)</td>
<td>71 (44%)</td>
<td>0.83</td>
</tr>
<tr>
<td>Statins</td>
<td>86 (52%)</td>
<td>107 (66%)</td>
<td>0.011</td>
</tr>
<tr>
<td>Fibrates</td>
<td>4 (2.4)</td>
<td>5 (3.1%)</td>
<td>0.71</td>
</tr>
<tr>
<td>Warfarin</td>
<td>8 (4.9%)</td>
<td>11 (6.8%)</td>
<td>0.45</td>
</tr>
</tbody>
</table>

Length of stay**

1 (1–2) 2 (1–4) 0.0006

Table 8. Total delay in patients not receiving a cardiac angiogram within 2 days of admission, by region, for non-intervention and intervention centres

Discussion

We have shown that in New Zealand, ACS patients receive similar medical management when admitted to either an interventional or non-interventional centre. However, whilst similar numbers of patients admitted with a suspected or definite ACS access angiography, time delays to invasive assessments and treatments exist nationally, and are significantly worse for patients admitted to a non-interventional centre.

Reperfusion therapy for STEMI patients—STEMI patients require reperfusion therapy, which results in better clinical outcomes if delivered by primary PCI rather than by thrombolytic therapy.15, 16 Primary PCI is only available in larger intervention centres, and therefore, not surprisingly, intervention centre patients were more often able to access this treatment (32% vs 3.9%, p<0.05). However, significant opportunities exist to improve access to primary angioplasty in interventional units with two-thirds of STEMI patients presenting to an interventional centre not receiving primary PCI.

At present, only two of these intervention centres plan to treat all eligible STEMI patients with primary PCI over 24 hours, 7 days per week (Auckland City and Waikato hospitals), with the other three major public centres being constrained to...
only offering this service during daytime hours, and for selected patients during the full 24 hours. A coordinated service with close liaison with the emergency ambulance service, in all five major metropolitan centres in New Zealand, would have the potential to facilitate the effectiveness of this primary PCI service. Models of care, using currently available technology for tele-transmission of ECGs of ACS patients are available, and could produce an important improvement to the treatment of these patients.17,18

‘Rescue’ PCI—Despite the benefits of primary PCI for STEMI patients which could be accomplished at the 5 major interventional centres, for logistical reasons, across New Zealand fibrinolytic therapy is likely to remain the standard method of reperfusion. Fibrinolytic therapy is still an effective method of reperfusion for STEMI patients, particularly when ‘rescue PCI’ is actively sought for the approximately 25% of patients who fail to reperfuse their culprit artery.18-20

Advanced planning for this eventuality is already in place at some rural and interventional centres, but an improvement in the plans to complete the ‘hub and spoke’ approach21 to rapid transfer by land or air transport is generally needed, to allow all suitable patients to access ‘rescue’ PCI. In our audit, five patients underwent ‘rescue’ PCI. Further, the transfer of STEMI patients for a cardiac angiogram within 6 hours of hospital presentation, after the use of fibrinolytic therapy at a rural hospital, has been recently shown to significantly reduce ischaemic complications.22,23 Planning for this transfer of patients could readily be undertaken at all New Zealand centres.

Time delays for invasive investigation—Overall there was generally an important delay for patients awaiting cardiac angiography, with a median time of 4.0 (IQR 2.0-6.1) days. The median delay of 2.5 days for patients admitted to an interventional centre is sub-optimal, with only 44% of patients receiving this important investigation within a 48 hour window (Tables 5, 6, Figure 1).

Catheter laboratories have expensive equipment laying ‘dormant’ overnight, unless used occasionally for ‘emergency’ patients. It would seem to be more efficient to inject resource to this area, to allow ‘shifts’ of nursing staff and Cardiologists to undertake ‘routine’ cases, perhaps between 08.00 and 22.00.

Fewer patients admitted to hospital on a Friday or Saturday underwent angiography within 48 hours (Figure 3). Regional centres generally only perform cardiac angiography for ‘emergency’ patients at the weekend. Hence arriving to hospital on a Friday or Saturday will inevitably cause a delay for angiography as patients are scheduled for the following week. The potential improvement in service provision for patients, should all 5 regional centres routinely perform angiography and PCI lists at the weekend, is also clear.

In comparison to patients admitted to an intervention centre, it would be expected that there would be some delay for patients admitted to a non-interventional centre, as a transfer to the interventional centre would be needed. However, a median time delay of 5.1 days (Figure 1), with only 8% of patients receiving this important procedure within 48 hours would suggest both a lack of cohesive planning and a ‘log-jam’ to this service within the interventional centres.

If a time limit of 48 hours is taken as what would be accepted as a reasonable time for all patients to receive a cardiac angiogram, then over the 2 weeks, there was a
cumulative delay of 721 days (Table 8), or (26×721) 18,746 days over a year. Using ‘Diagnosis Related Groups’ corresponding to ACS patient admissions and the ‘National Pricing Programme’ for the Auckland District Health Board (ADHB), the approximate cost of having an ACS patient admitted to a hospital for 24 hours without invasive management is $900 (Patrick Horan, Manager of clinical costing, ADHB; personal communication). Hence, with national extrapolation of this amount over 1 year, the cost of these patient delays would be approximately ($900×18,746) $17 million, for a service which is needed and is eventually supplied, but currently not in a timely manner.

Shorter hospital stays would inevitably produce more efficient care and allow redistribution of this healthcare resource. In addition, patients are at risk of re-infarction, heart failure and death whilst waiting for cardiac angiography. Furthermore, the benefits of revascularisation treatment of ACS patients are abundantly clear and has helped to improve the long-term outcome of heart attack patients in Australia. Regional planning to efficiently deliver these benefits in New Zealand would seem appropriate.

CABG Surgery—For patients awaiting a CABG operation, there have been persistent problems over decades. These delays have resulted in poor health and death for many patients. For patients who are critically unwell and unable to be discharged from hospital until undergoing a CABG operation, time delays are not only clinically sub-optimal, but again leave patients at risk of re-infarction, heart failure and death whilst waiting, as well as the delay being a significant waste of hospital resource. Over the 2 week audit, there were 25 ACS patients awaiting CABG surgery with a median time delay from cardiac angiography to CABG surgery of 6.9 days. If a time limit of 3 days is taken as what would be accepted as a reasonable time for all patients to receive an in-hospital CABG, then over the 2 weeks, there was a cumulative delay of 173 days, or (26×173) 4,498 days over a year. The cost of having a patient waiting for CABG at approximately $900/day, over 1 year would be approximately ($900×4498) $4 million. The provision of CABG surgery appears to need attention and a long-term solution.

Medical Management of ACS Patients—ACS patients benefit from various medications both during their hospital stay and also after hospital discharge. The benefits from such drugs, which are generally well proven in robust clinical trials, are large but they have often not been readily available to New Zealanders. The limited availability of statins and clopidogrel, in particular, has seriously impacted on New Zealand ACS patients with resultant unnecessary death and major morbidity. PHARMAC’s focus on expenditure restricts access to some evidence-based medicines which may not be a good idea if the cost in patients’ well-being and longer term patient burden on the health system is too great.

In the area of discharge medication, the use of ‘key indicators’ and ‘process tools’ have been shown to improve the uptake of proven, prognostically advantageous, medications and programmes overseas have demonstrated a better management of patients if these systems are employed. A similar New Zealand-wide strategy may
increase the prescribing of these medicines: aspirin, clopidogrel, beta-blockers, ACE-inhibitors/angiotensin receptor blockers, and statins.

**National planning of an ACS service**—New Zealand has a unique opportunity to develop a comprehensive cardiovascular health service, with an efficient management team. Solutions and ideas to develop this national service have been previously made, with a ‘hub and spoke’ approach advocated.

Based around the five Regional interventional centres (Auckland, Hamilton, Wellington, Christchurch, Dunedin) advanced planning and a smooth delivery of service should be achievable, with the ability to efficiently manage the health care of 21,500 ACS patients in New Zealand, each year. This should not result in ‘centralisation’ of service, but a structure with real ability to direct funding out to the regional centres where it is needed, and also to provide key central services (e.g. CABG operations), when needed.

**Study limitations**—There are a number of limitations to our audit, including the fact that we did not collect data for investigations and treatment following hospitalisation.

**Conclusion**

We have demonstrated that ACS patients admitted to a New Zealand hospital experience delays in accessing investigations and subsequent revascularisation. Furthermore, inequity exists with delays being significantly longer for patients admitted to a non-intervention centre, who number more than half of the total ACS group.

The current data suggest that there are significant flaws in the management of ACS patients across New Zealand. Improvements in the delivery of health care resource to ACS patients in all centres in New Zealand would improve the efficiency of the service for all. We suggest that a comprehensive service should be based upon the five regional interventional centres, with a ‘hub and spoke’ approach to the management of these high-risk patients.

**Competing interests:** None known.

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by the Steering Committee with total independence from the companies above, and endorsed by the Cardiac Society of New Zealand, which itself made a small contribution to costs. Collection of data was unfunded at local centres, although three centres received a modest donation for personnel support.

We thank these audit leaders and assistants in the following hospitals—from north to south by region (patient numbers in the study are given inside brackets; #Chairman; *Steering Committee member).

**Auckland/Northland (North Island)**

Kaitaia Hospital: Dr E Jeffrey (9). Dargaville Hospital: Dr D Gibbons (5). Rawene Hospital: Dr K Blattner (0). Kawakawa Hospital: Dr A Murray, Ms S August (3). Whangarei Hospital: Dr N Harrison, Dr B Wong, Ms K O’Keefe (37). North Shore Hospital, Auckland: Dr H Hart, Dr T Scott, Ms E Fairhurst, Ms W Young (66).

Waitakere Hospital: Dr H Hart, Dr T Scott, Ms J Hewlett (24). Auckland City Hospital: Dr C Ellis #*, Prof H White*, Mr G Gamble*, Dr A Chateleine (122).

**Mercy Private Hospital, Auckland**: Dr C Ellis, Ms C McGarrigle (6). **Ascot Private Hospital, Auckland**: Dr A Maslowski (2). **Middlemore Hospital, Auckland**: Dr A Kerr, Dr M Lund, Dr J Goh (35).

**Waikato (Central North Island)**

Thames Hospital: Dr J Lennane, Dr Aftabuzzaman (19). Tauranga Hospital: Dr J Tisch, Dr G Porter, Dr C Young, Ms W Bryson, Ms J Goodson (44). Waikato Hospital, Hamilton: Dr G Devlin*, Ms B Killion, Ms A Silverstone, Ms L Boenders (56).

**Whakatane Hospital**: Dr E Edwards, Dr R Steeper, Ms D Garner (12).

Rotorua Hospital: Dr N Crook, Ms A Morley (28). **Tokoroha Hospital**: Dr N Thornton, Dr F Kanan (2). **Te Kuiti Hospital**: Dr N Thornton, Dr K Buswell, Ms T Te Wano (0). **Taupo Hospital**: Dr K Logan (11). Gisborne Hospital: Dr C Duffy, Ms K Weytmans, Ms T Low (16). Taumarunui Hospital: Dr N Thornton, Dr H Wahid (1). **New Plymouth Hospital**: Dr I Ternouth, Dr T Boswell (17).

**Wellington (Southern North Island, Upper South Island)**

Hawkes Bay Regional Hospital, Hastings: Dr R Luke, Ms J MacKenzie (76). Wanganui Hospital: Dr T Thompson, (26). Palmerston North Hospital: Dr D Tang (25). Masterton Hospital: Dr T Matthews, Ms K Lee (10). Hutt Hospital: Dr T O’Meeghan, Ms J Dewar, Ms M Klientjes (25). Wellington Hospital: Dr P Matsis*, Ms D Middleditch, Ms E Walsh (57). Wakefield Private Hospital, Wellington: Dr M Abernethy (0). Nelson Hospital: Dr A Hamer, Ms R Price (14). **Blenheim Hospital**: Dr M Heynike, Ms M Udy (12).

**Christchurch/Canterbury (Central South Island)**

Greymouth Hospital: Dr U Bopitiya, Ms L Skeats (6). Christchurch Hospital: Assoc Prof J Elliott*, Prof M Richards*, Ms L Skelton, Ms L Frost (141). Ashburton Hospital: Dr A Obafemi, Ms A Smart (3). Timaru Hospital: Dr M Hills, Ms Maria Hammond, Ms C Barker (31).
Dunedin/Otago (Southern South Island)

Oamaru Hospital: Dr D Phillips, Ms S McCulloch (10). Dunstan Hospital, Clyde: Dr G Nixon, Ms J Coutts (3). Dunedin Hospital: Assoc Prof MJA Williams*, Ms M McLelland (28). Invercargill Hospital: Dr A Maloney, Dr R Anand (21).

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References:


A programme of Enhanced Recovery After Surgery (ERAS) is a cost-effective intervention in elective colonic surgery

Tarik Sammour, Kamran Zargar-Shoshtari, Abhijith Bhat, Arman Kahokehr, Andrew G Hill

Aim There are few published ERAS cost-analyses in colorectal surgery. The aim of this paper is to evaluate whether costs saved by reduced postoperative resource utilisation would offset the financial burden of setting up and maintaining such an ERAS programme.

Methods A cost-effectiveness analysis from a healthcare provider perspective using a case-control model. The study group consisted of patients enrolled in the ERAS program for elective colonic surgery at Manukau Surgical Centre between December 2005 and March 2007. The control group consisted of consecutive patients from September 2004 to September 2005 (before the start of ERAS). Groups were matched with respect to operation, BMI, ASA, and Cr-POSSUM score.

Results Data were available for 50 patients in each group. There was a significant reduction in total hospital stay, intravenous fluid use, and duration of epidural use in the ERAS group. There were significantly fewer complications in the ERAS group. Implementation of ERAS cost approximately $NZ102,000, but this has been more than offset by costs saved in reduced postoperative resource utilisation, with an overall cost-saving of approximately NZ$6900 per patient.

Conclusion Implementing an ERAS program is cost-effective in the medium term, with costs offset by those recovered by reduced resource utilisation in the postoperative period.

Background

There has been a rise in popularity in Enhanced Recovery After Surgery (ERAS) programmes in developed countries, but uptake has been varied, and inter-protocol consistency sketchy at best. A major challenge in the implementation of a multimodal care pathway is adequate resourcing, particularly in context of the current healthcare environment which dictates the provision of financial justification prior to the adoption of any new intervention.

Cost-analyses of ERAS protocols in colorectal surgery have been limited to early clinical pathway studies, one study focussing solely on ileal-pouch anal anastomoses, and a study incorporating a very heterogeneous group of patients, some of whom were part of an unrelated international trial. None of these studies addressed the set-up costs of an ERAS protocol nor provided a detailed breakdown of where cost savings were achieved in the postoperative recovery phase.

In December 2005, an ERAS programme was implemented for elective colonic resections at the Manukau Surgical Centre in Auckland, New Zealand. This programme emphasises structured nursing care pathways within an environment...
focusing on early recovery, and incorporates a number of perioperative strategies within the ERAS framework. We have previously published data outlining a significant reduction in intravenous fluid requirement, total day-stay and postoperative complications, as well as improved patient functional recovery as a direct result of instituting this programme.

A considerable investment was required in order to setup this programme and ensure its success. The aim of this paper is to evaluate whether the costs saved by reduced postoperative resource utilisation would offset the financial burden of setting up and maintaining an ERAS programme in elective colonic surgery.

Methods

ERAS Protocol—The ERAS programme was developed in a multidisciplinary fashion and received appropriate institutional approval for implementation. A consultant surgeon, a ward charge nurse, and a colorectal nurse specialist visited an institution in Denmark with an established ERAS programme, and an equivalent programme tailored to the Manukau Surgical Centre was developed. A full-time ward-based junior doctor was then employed as a research fellow in enhanced recovery, to be responsible for the overall running of the programme as well as prospective auditing of safety and effectiveness. The ERAS protocol used in our institution is outlined in Table 1.

All elective colonic resections in patients >15 years old were included in the ERAS programme. Exclusion criteria were: patients requiring a stoma, ASA (American Society of Anaesthesiologists) score ≥IV, significant cognitive impairment, inability to communicate in English, and patients declining consent.

Cost analysis—A cost-effectiveness analysis from a healthcare provider perspective was performed comparing a study group of ERAS patients with a historical group of case-matched controls. Total cost of protocol development, as well as the cost of ward stay at the Manukau Surgical Centre, outpatient clinic time, and patient booklet production was obtained from hospital management budget records. The research fellow yearly salary was obtained from the University of Auckland (Auckland, New Zealand). Costs of oral supplements, non-steroidal anti-inflammatory medications, and intravenous fluids were obtained from the hospital pharmacy, and epidural costs from the hospital anaesthetic department. Costs of readmission and estimates of specific costs associated with postoperative complications were supplied by a hospital clinical analyst (complication costs were determined by calculating the cost of index hospital stay with and without a given complication, excluding cost of day stay and readmission).

Patient groups—The study (ERAS) group consisted of consecutive patients enrolled in the ERAS programme for elective colonic surgery at Manukau Surgical Centre between December 2005 and March 2007. Data for this group were collected prospectively.

The control group consisted of a comparable, consecutive series of patients identified through a hospital electronic database search from September 2004 to September 2005 (before the start of the ERAS programme). Control patients were individually matched with those in the study group with respect to the operation performed, BMI (Body Mass Index), ASA score, and Cr-POSSUM score (Colorectal Physiological and Operative Severity Score for the enumeration of Mortality). Furthermore, these patients all met the inclusion criteria used for the ERAS group and their operations were performed by the same specialist surgeons. Patients in the control group received conventional, non-structured perioperative care. Discharge was left to the discretion of the senior members of the surgical team with no specified discharge criteria in place. Data for this group were collected retrospectively.
Table 1. Enhanced Recovery After Surgery (ERAS) protocol

<table>
<thead>
<tr>
<th>Timing</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preadmission</strong></td>
<td>Preoperative assessment in a dedicated outpatient session.</td>
</tr>
<tr>
<td></td>
<td>Programme information given, including specific daily milestones.</td>
</tr>
<tr>
<td></td>
<td>Social issues are identified and addressed.</td>
</tr>
<tr>
<td></td>
<td>Preoperative ward visit and orientation.</td>
</tr>
<tr>
<td><strong>Preop</strong></td>
<td>Preoperative carbohydrate loading (PreOP®, Nutricia; Numico, Zoetermeer, Netherlands). 4 drinks day before surgery, and 2 drinks 2 hours before surgery.</td>
</tr>
<tr>
<td></td>
<td>Patients admitted to hospital on the morning of their surgery.</td>
</tr>
<tr>
<td></td>
<td>Left-sided operations receive a phosphate enema on arrival at the hospital.</td>
</tr>
<tr>
<td></td>
<td>Mechanical bowel preparation is avoided.</td>
</tr>
<tr>
<td></td>
<td>Limited intraop intravenous fluids (1–2L crystalloids / colloids).</td>
</tr>
<tr>
<td></td>
<td>Transverse incisions for right-sided open surgery if appropriate.</td>
</tr>
<tr>
<td></td>
<td>Prophylactic nasogastric tubes not used.</td>
</tr>
<tr>
<td></td>
<td>Intra-abdominal drains not used.</td>
</tr>
<tr>
<td></td>
<td>Calf stockings applied at the end of surgery.</td>
</tr>
<tr>
<td><strong>Recovery room</strong></td>
<td>Vasopressor agents in preference to intravenous fluids to treat epidural-related hypotension.</td>
</tr>
<tr>
<td></td>
<td>Intravenous morphine / fentanyl PCA initiated.</td>
</tr>
<tr>
<td><strong>Day of surgery</strong></td>
<td>Patients are mobilised to a chair.</td>
</tr>
<tr>
<td></td>
<td>Oral intake of fluids is started, aiming for &gt; 800 ml of oral intake on the day of surgery.</td>
</tr>
<tr>
<td></td>
<td>Pre-emptive regular antiemetics (5-HT3 antagonists as first line).</td>
</tr>
<tr>
<td></td>
<td>Subcutaneous low molecular weight heparin started for thrombo-prophylaxis (Clexane® 20mg once daily until discharge, Sanofi-aventis Ltd, Auckland, NZ).</td>
</tr>
<tr>
<td><strong>Day 1</strong></td>
<td>Urinary catheter removed.</td>
</tr>
<tr>
<td></td>
<td>Full solid oral diet.</td>
</tr>
<tr>
<td></td>
<td>Resource supplement drinks (2–3 per day until discharge).</td>
</tr>
<tr>
<td></td>
<td>Active mobilisation with nursing and physiotherapy input.</td>
</tr>
<tr>
<td><strong>Day 2</strong></td>
<td>Epidural infusion is stopped, and epidural catheter removed.</td>
</tr>
<tr>
<td></td>
<td>Regular oral non-steroidal anti-inflammatory drugs (Tenoxicam 20mg orally twice daily until discharge, Tilcotil tabs®, Roche, Auckland, NZ).</td>
</tr>
<tr>
<td></td>
<td>Oral opiates for break-through pain only.</td>
</tr>
<tr>
<td><strong>Day 3</strong></td>
<td>Discharged home if fulfill following criteria:</td>
</tr>
<tr>
<td></td>
<td>Tolerating full oral diet</td>
</tr>
<tr>
<td></td>
<td>Passing flatus</td>
</tr>
<tr>
<td></td>
<td>Adequate analgesia on oral medication</td>
</tr>
<tr>
<td></td>
<td>Ambulating independently</td>
</tr>
<tr>
<td></td>
<td>Satisfactory support at home</td>
</tr>
<tr>
<td><strong>After discharge</strong></td>
<td>Patient given a phone number for contacting the ward if required.</td>
</tr>
<tr>
<td></td>
<td>Nursing staff contact the patients three days after discharge for a phone interview.</td>
</tr>
<tr>
<td></td>
<td>Follow up outpatient clinic appointment within 7 days of discharge.</td>
</tr>
</tbody>
</table>

Preop: Preoperative; Intraop: Intraoperative

**Data collection**—Data were collected from patient records including physical and electronic clinical, radiology, and laboratory records. Data included patient demographics, ASA score, Cr-POSSUM score, surgical indication, operating surgeon, operation performed, epidural use, intravenous fluid use, cancer staging, postoperative day stay, total day stay, complications and readmission. To ensure that recorded complications were comparable in both groups, specific complications were documented according to previously defined and published criteria. All patients were followed for 30 days after surgery.
Results

Data were available for 50 patients in each group. During the recruitment period, ten patients had been excluded from the ERAS programme; two had significant renal impairment, two had significant cardiac comorbidity, two were cognitively impaired, two could not speak sufficient English, and two declined consent. Eight patients treated from September 2004 to September 2005 were excluded from the conventional treatment control group; two patients had significant renal impairment, two had dementia, one had Addison’s disease, and three had hematologic disorders.

Baseline characteristics—The ERAS and conventional groups were comparable with respect to sex, BMI, ASA score, Cr-POSSUM score, operation performed, and indication for surgery (Table 2). The ERAS group was marginally younger than the conventional group (65.6 vs 70.7 years, p=0.021).

Table 2. Baseline characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>ERAS group (n=50)</th>
<th>Control group (n=50)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean, range)</td>
<td>65.6 (39–92)</td>
<td>70.7 (40–85)</td>
<td>0.021</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>26</td>
<td>28</td>
<td>0.688‡</td>
</tr>
<tr>
<td>Female</td>
<td>24</td>
<td>22</td>
<td>0.688‡</td>
</tr>
<tr>
<td>ASA score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>8</td>
<td>8</td>
<td>1.00‡</td>
</tr>
<tr>
<td>II</td>
<td>29</td>
<td>31</td>
<td>0.683‡</td>
</tr>
<tr>
<td>III</td>
<td>13</td>
<td>11</td>
<td>0.640‡</td>
</tr>
<tr>
<td>BMI</td>
<td>28.6</td>
<td>27.4</td>
<td>0.588†</td>
</tr>
<tr>
<td>CR-POSSUM</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physiologic</td>
<td>10.3</td>
<td>9.7</td>
<td>0.524†</td>
</tr>
<tr>
<td>Operative</td>
<td>9.2</td>
<td>8.3</td>
<td>0.061†</td>
</tr>
<tr>
<td>Operation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Open R hemicolectomy</td>
<td>26</td>
<td>29</td>
<td>0.546‡</td>
</tr>
<tr>
<td>Open L hemicolectomy</td>
<td>19</td>
<td>14</td>
<td>0.288‡</td>
</tr>
<tr>
<td>Lap L hemicolectomy</td>
<td>4</td>
<td>7</td>
<td>0.525‡</td>
</tr>
<tr>
<td>Open Total colectomy</td>
<td>1</td>
<td>0</td>
<td>1.000‡</td>
</tr>
<tr>
<td>Diagnosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diverticulosis</td>
<td>2</td>
<td>4</td>
<td>0.674§</td>
</tr>
<tr>
<td>IBD</td>
<td>1</td>
<td>1</td>
<td>1.000§</td>
</tr>
<tr>
<td>Adenoma</td>
<td>4</td>
<td>2</td>
<td>0.674§</td>
</tr>
<tr>
<td>Dukes A</td>
<td>6</td>
<td>5</td>
<td>0.749‡</td>
</tr>
<tr>
<td>Dukes B</td>
<td>15</td>
<td>8</td>
<td>0.096‡</td>
</tr>
<tr>
<td>Dukes C</td>
<td>19</td>
<td>21</td>
<td>0.683‡</td>
</tr>
<tr>
<td>Dukes D</td>
<td>3</td>
<td>9</td>
<td>0.124‡</td>
</tr>
</tbody>
</table>


†Mann–Whitney U test, §Chi-squared test.

Postoperative recovery—As we have previously shown⁷ there was a significant reduction in postoperative hospital stay, total hospital stay, intravenous fluid use (both
intraoperative and day 1 to day 3 postoperative), and duration of epidural use in the ERAS group compared to the control group (Table 3). There was also a one day reduction in the median time to first full solid meal and passage of flatus, and patients mobilised a median of 2 days earlier.

### Table 3. Postoperative recovery data.

<table>
<thead>
<tr>
<th>Variables</th>
<th>ERAS Group (n=50)</th>
<th>Control Group (n=50)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intravenous fluids</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intraoperative</td>
<td>2 (1–8)</td>
<td>3 (1–7.5)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>First 3 days</td>
<td>2 (1–10)</td>
<td>6.5 (1–12)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td><strong>Epidural analgesia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>44 (89%)</td>
<td>38 (76%)</td>
<td>0.223‡</td>
</tr>
<tr>
<td>Duration of use (days)</td>
<td>2 (0–3)</td>
<td>3 (0–4)</td>
<td></td>
</tr>
<tr>
<td><strong>Recovery</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days to 1st full meal</td>
<td>1 (1–3)</td>
<td>2 (1–15)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>Days to passage of flatus</td>
<td>2 (0–8)</td>
<td>3 (0–18)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>Days to independent mobilisation</td>
<td>1 (1–3)</td>
<td>3 (1–7)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients with &gt; 1 complication</td>
<td>27</td>
<td>33</td>
<td>0.221‡</td>
</tr>
<tr>
<td>Breakdown of complication events</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
<td>2</td>
<td>0.495†</td>
</tr>
<tr>
<td>Reoperation</td>
<td>4</td>
<td>4</td>
<td>1.000‡</td>
</tr>
<tr>
<td>Anastomotic leak</td>
<td>4</td>
<td>3</td>
<td>1.000‡</td>
</tr>
<tr>
<td>Intra-abdominal collection</td>
<td>1</td>
<td>1</td>
<td>1.000‡</td>
</tr>
<tr>
<td>Ileus</td>
<td>5</td>
<td>18</td>
<td>0.005‡</td>
</tr>
<tr>
<td>Wound complication</td>
<td>6</td>
<td>10</td>
<td>0.275‡</td>
</tr>
<tr>
<td>Urinary tract infection</td>
<td>2</td>
<td>12</td>
<td>0.008‡</td>
</tr>
<tr>
<td>Urinary retention</td>
<td>5</td>
<td>3</td>
<td>0.715‡</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>11</td>
<td>21</td>
<td>0.032‡</td>
</tr>
<tr>
<td><strong>Day stay</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. admitted &gt; 1 day before surgery</td>
<td>12 (24%)</td>
<td>29 (58%)</td>
<td>&lt;0.0001‡</td>
</tr>
<tr>
<td>Postoperative stay (days)</td>
<td>4 (3–34)</td>
<td>6.5 (3–18)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td>Total hospital stay (days)</td>
<td>4 (3–34)</td>
<td>8 (4–29)</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td><strong>Readmissions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. patients readmitted</td>
<td>6</td>
<td>7</td>
<td>0.766‡</td>
</tr>
<tr>
<td>Total day stay added (days)</td>
<td>73</td>
<td>44</td>
<td>0.772‡</td>
</tr>
</tbody>
</table>

ERAS: enhanced recovery after surgery; No.: number; % percentage.

Data are medians with ranges in parentheses, unless otherwise dated. †Mann–Whitney U test, ‡Chi-squared est.

Complications are also presented in Table 3. Overall 54% of patients in the ERAS group had at least one complication recorded versus 66% of patients in the control group. There were significantly fewer urinary tract infections, cardiopulmonary complications, and episodes of postoperative ileus in the ERAS group. There was no difference in re-operation rate, with 4 patients in each group requiring an unplanned return to the theatre. Anastomotic leak resulted in three emergency laparotomies in the ERAS group and two in the conventional group, and wound dehiscence led to one re-operation in the ERAS group and two in the conventional group.
Cost analysis—A breakdown of ERAS protocol implementation and maintenance costs, offset against differential cost savings in the postoperative period is shown in Table 4. As can be seen, implementation of the ERAS programme cost approximately NZ$102,000 for the first 50 patients when one-off setup costs are taken into account. However, this has been more than matched by costs saved in reduced resource utilisation in the postoperative period with an overall cost-saving of approximately NZ$6900 per patient in the ERAS group compared to the control group.

Table 4. Cost analysis

<table>
<thead>
<tr>
<th>Item</th>
<th>Cost of one unit</th>
<th>ERAS (units)</th>
<th>Control (units)</th>
<th>ERAS (NZD)</th>
<th>Control (NZD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denmark visit (3 airfare tickets + accommodation)</td>
<td>10561.39</td>
<td>1</td>
<td>0</td>
<td>10561.39</td>
<td>0</td>
</tr>
<tr>
<td>Research Fellow salary for 15 months (1 year salary × 1.25)</td>
<td>84143.75</td>
<td>1</td>
<td>0</td>
<td>84143.75</td>
<td>0</td>
</tr>
<tr>
<td>ERAS patient booklet</td>
<td>4.20</td>
<td>50</td>
<td>0</td>
<td>210.00</td>
<td>0</td>
</tr>
<tr>
<td>Supplements drinks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preop carbohydrate</td>
<td>1.50</td>
<td>300</td>
<td>0</td>
<td>450.00</td>
<td>0</td>
</tr>
<tr>
<td>Resource supplement</td>
<td>1.42</td>
<td>722.5</td>
<td>0</td>
<td>1025.95</td>
<td>0</td>
</tr>
<tr>
<td>Tenoxicam</td>
<td>0.2375</td>
<td>300</td>
<td>0</td>
<td>71.25</td>
<td>0</td>
</tr>
<tr>
<td>Outpatient clinic slot</td>
<td>115.77</td>
<td>50</td>
<td>0</td>
<td>5788.50</td>
<td>0</td>
</tr>
<tr>
<td>Fluids</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intraoperative</td>
<td>34.20</td>
<td>124.5</td>
<td>174.5</td>
<td>4257.90</td>
<td>5967.90</td>
</tr>
<tr>
<td>Postoperative</td>
<td>34.20</td>
<td>131</td>
<td>315.5</td>
<td>4480.20</td>
<td>10790.10</td>
</tr>
<tr>
<td>Epidural</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bupivacaine infusion</td>
<td>48.80</td>
<td>88</td>
<td>114</td>
<td>4294.40</td>
<td>5563.20</td>
</tr>
<tr>
<td>Apparatus and tubing</td>
<td>59.70</td>
<td>44</td>
<td>38</td>
<td>2626.80</td>
<td>2268.60</td>
</tr>
<tr>
<td>Complications</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leak / collection</td>
<td>34853.26</td>
<td>5</td>
<td>4</td>
<td>174266.30</td>
<td>139413.04</td>
</tr>
<tr>
<td>Ileus</td>
<td>6517.37</td>
<td>5</td>
<td>18</td>
<td>32586.85</td>
<td>117312.66</td>
</tr>
<tr>
<td>Wound complication</td>
<td>19703.81</td>
<td>6</td>
<td>10</td>
<td>118222.86</td>
<td>197038.10</td>
</tr>
<tr>
<td>Urinary tract infection</td>
<td>4615.13</td>
<td>2</td>
<td>12</td>
<td>9230.26</td>
<td>55381.56</td>
</tr>
<tr>
<td>Urinary retention</td>
<td>3445.41</td>
<td>5</td>
<td>3</td>
<td>17227.05</td>
<td>10336.23</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>10802.13</td>
<td>11</td>
<td>21</td>
<td>118823.43</td>
<td>226844.73</td>
</tr>
<tr>
<td>Ward stay</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Index admission</td>
<td>881.63</td>
<td>200</td>
<td>400</td>
<td>176326.00</td>
<td>352652.00</td>
</tr>
<tr>
<td>Re-admission</td>
<td>520.885</td>
<td>73</td>
<td>44</td>
<td>38024.61</td>
<td>22918.94</td>
</tr>
<tr>
<td>Total cost</td>
<td></td>
<td></td>
<td></td>
<td>802617.50</td>
<td>1146487.06</td>
</tr>
<tr>
<td>Cost per patient</td>
<td></td>
<td></td>
<td></td>
<td>16052.35</td>
<td>22929.74</td>
</tr>
</tbody>
</table>

NZD: New Zealand Dollars.

Discussion

This cost-effectiveness analysis has shown that an ERAS programme is a very cost-effective intervention in elective colonic surgery in the setting of an elective hospital in New Zealand. While the programme incurred an additional cost of approximately NZ$2000 per patient in the study group to implement, these costs were recouped after
only 15 patients had gone through, with an overall saving per patient of just under NZ$7000.

The majority of the cost was saved by halving the total postoperative day stay, and reducing postoperative complication costs. While the rate of readmission was not significantly different between the groups, day stay cost of readmission for the ERAS group was higher, with patients being readmitted for a longer period of time. This is because 3 patients in the ERAS group were readmitted with a major complication requiring day stay of 10 days or more (2 anastomotic leaks and 1 intra-abdominal abscess), versus 1 in the control group (intra-abdominal abscess).

It should be emphasised that while more patients in the ERAS group were readmitted with intra-abdominal complications, the overall rate of these complications was not significantly different, with patients in the control group manifesting these complications during their relatively longer index admission.

Care pathway cost-analyses have been undertaken for a variety of surgical indications, from vascular access surgery to paediatric urology, and almost invariably demonstrate cost savings. This is largely because these pathways focus on perioperative process of care to prevent or reduce morbidity and mortality, resulting in an improvement of resource utilisation.

In colorectal surgery, cost-analyses of enhanced recovery protocols are limited. Two early clinical pathway programmes proved useful in standardising patient care and reducing costs, and were probably instrumental in the development of modern ERAS protocols. However, there has been a huge paradigm shift in postoperative care principles in colorectal surgery since that time, making the cost-analyses reported in those studies inapplicable to current programmes.

More recently Kariv et al published results of a case-control cost analysis comparing patients undergoing ileal pouch-anal anastomosis in a “Fast Track” postoperative care pathway versus case-matched controls. A significant reduction in median direct hospital costs per patient within 30 days was reported. However, the study was considerably weakened by a significant surgeon confounder with a different group of surgeons performing the surgery in the study group and the control group. This was highlighted in the reduced operating time in the Fast Track arm of the study. Also, the lack of epidural use in the treatment arm may not be consistent with current enhanced recovery recommendations, and the study did not account for costs incurred in protocol development.

Another case-control study by King et al focussing on quality-of-life after colonic and rectal surgery, reported a health-economic analysis estimated on an individual patient level by adding in-hospital costs and postoperative costs derived using a health economics questionnaire.

While this study was successful in demonstrating that implementation of a rigorous and well-designed ERAS programme did not result in transfer of costs to another component of the healthcare service (with an overall trend towards lower costs in the ERAS group), the difference in costs between the groups did not reach statistical significance. However, conclusions were limited by the heterogeneity of the patient cohorts (with significantly higher number of laparoscopic conversions and stomas in the control group) made up of patients enrolled in an unrelated national randomised
control trial which specified a 2:1 randomisation to laparoscopic versus open surgery. This in itself may have introduced significant bias. Also, the health economic questionnaire used was not outlined (this may have not been a validated measure), and the costs of protocol development were not included in the analysis.

A further general criticism of cost analyses in surgery is the gross under-estimation of readmission costs, as patients often represent to other services and hospitals, and this was not easily identified (or necessarily accounted for) in this study or that of Kariv et al.

The value of our study is in the uniformity of the patient cohorts. All patients underwent their treatment in the same facility and were operated on by the same surgeons. Both cohorts were also comparable with respect to sex, BMI, ASA score, Cr-POSSUM score, operation performed, and indication for surgery. The electronic hospital records system which we used allowed complete follow-up for all patients in the study arm for 30 days, and identification of any re-presentation to any service (including the emergency departments) of all three public hospitals in the Auckland region. In addition, we isolated specific costs in the analysis, namely those involved in development and maintenance of the ERAS programme at our unit.

This study has several limitations. Our estimate represents a differential cost-effectiveness analysis based on the identification of areas of difference between the two groups. While this serves our comparison well, it is not an individualised cost-analysis and it is possible that unanticipated cost differences between the groups were un-accounted for.

Secondly, use of historical controls carries an inherent bias, with interval changes in costs or surgical practice (unrelated to the advent of the ERAS programme) potentially occurring during the intervening time. Also, certain development costs which could not be directly measured, such as time invested by the lead surgeon and costs of ward staff training, could not be accounted for in the analysis. Costs of non-hospital medical visits, at after hours emergency care or primary physicians for example, were also unobtainable.

**Conclusion**

Implementing an ERAS programme in the setting of elective colonic surgery is cost-effective in the medium term, with set-up and maintenance costs more than offset by costs recovered by reduced resource utilisation in the postoperative period.

**Competing interests:** None known.

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References:


Audit of cervical screening in women with HIV infection in the Auckland and Northland regions of New Zealand

Jasmin Grewal, Michele Lowe, Hilary Gerrard, Rebecca Henley, Nicky Perkins, Simon Briggs

Abstract

Aim We aimed to review our current cohort of women with HIV infection to document the number of women who had received a yearly cervical smear since their diagnosis of HIV infection and the number of women who were likely to have had undiagnosed HIV infection at the time of their first abnormal cervical smear.

Method This audit was a retrospective review of the cervical smear history of all adult women (≥16 years) with HIV infection who were under active follow-up by the Infectious Diseases and Sexual Health Services at Auckland City Hospital on 31 December 2007.

Results Sixty-nine of the 123 (56%) women in this audit met the definition for yearly cervical smears. The factor associated with not receiving yearly cervical smears was women who had received cervical smears from their general practitioner (GP). Taking into account the women’s CD4 count at the time of the diagnosis of their HIV infection, it is very likely that seven women had undiagnosed HIV infection at the time of their first abnormal cervical smear.

Conclusion The proportion of women with HIV infection in the Auckland and Northland regions who received a yearly cervical smear during the audit period was low. We have put a number of interventions in place that we expect will improve this rate. These interventions include informing GPs of the need for yearly cervical smears for women with HIV infection, informing the National Cervical Screening Unit that these women are immunocompromised which will result in a yearly recall comment and informing these women of options for obtaining a cervical smear at little or no cost.

Cervical smear takers should consider offering an HIV test to all women with an abnormal cervical smear who have resided in areas with high rates of HIV infection.

Women with HIV infection have an increased risk of cervical cytologic abnormalities\(^1\) and cervical cancer\(^2,3\) compared to women without HIV infection. An American study containing almost 20,000 women with HIV infection found a standardised incidence ratio for cervical cancer (observed incidence of cervical cancer in women with HIV infection divided by expected incidence of cervical cancer based on population rates) of 2.9 (95%CI 1.9–4.2).\(^2\)

The current New Zealand and American recommendations are that women with HIV infection receive cervical screening when their HIV infection is diagnosed, 6 months later if the initial screen is normal and then yearly if the second screen is normal.\(^4,5\)
The Infectious Diseases and Sexual Health Services at Auckland City Hospital provide secondary level care for all women with HIV infection in the Auckland and Northland regions.

We aimed to review our current cohort of women with HIV infection in the Auckland and Northland regions to document the number of women who had received a yearly cervical smear since their diagnosis of HIV infection and the number of women who were likely to have had undiagnosed HIV infection at the time of their first abnormal cervical smear.

Method

This audit was a retrospective review of the cervical smear history of all adult women (≥16 years) with HIV infection who were under active follow up by the Infectious Diseases and Sexual Health Services at Auckland City Hospital on 31 December 2007.

We excluded women younger than 20 or older than 69 years of age on 31 December 2007 as routine cervical screening is not recommended for women of these ages by the National Cervical Screening Unit.4

Data including demographics, date of diagnosis of HIV infection, duration of follow-up, and whether the need for a yearly cervical smear was documented in one of the first two clinic letters sent to the woman’s general practitioner (GP) were collected from clinical records at Auckland City Hospital. The cervical smear history, results and the person(s) performing the cervical smear(s) for each woman were obtained from the National Cervical Screening Unit.

Cervical smears were defined as being taken yearly if the following criteria were met. The number of cervical smears performed divided by the number of years since the diagnosis of HIV infection was ≥0.8 and there were no gaps between cervical smears of ≥2 years, i.e. 4 cervical smears performed in a 5-year period without a gap of ≥2 years would meet our definition of a yearly cervical smear. For those women who were diagnosed with HIV infection before their arrival in New Zealand, we used the number of years they had resided in New Zealand instead of the number of years since the diagnosis of HIV infection.

This audit received approval from the Northern X Regional Ethics Committee.

The two-tailed Fisher’s exact test was used to calculate univariate p values. The two-tailed Mann-Whitney test was used to calculate p values for age, time since diagnosis and duration of follow-up.

Results

At 31 December 2007, 128 adult women with HIV infection were under active follow-up by the Infectious Diseases or Sexual Health Services at Auckland City Hospital. Five women were excluded as they were younger than 20 or older than 69 years of age. The remaining 123 women form the basis of this audit.

113 of these women were under the care of the Infectious Diseases Service, 8 were under the care of the Sexual Health Service and 2 had been transferred from the care of the Sexual Health Service to the Infectious Diseases Service. The median age was 38 (range 22 to 59) years.

The self-reported ethnicity was recorded as sub-Saharan African (n=69), Asian/South-East Asian (n=19), New Zealand European (n=18), European (n=9), Pacific Island Person (n=4), Māori (n=3) and other (n=1). Twenty-five women required an interpreter when they were seen in clinic.

These women had been diagnosed with HIV infection for a median of 5 (range 0 to 22) years. They had been under the care of the Infectious Diseases or Sexual Health Services at Auckland City Hospital for a median of 4 (range 0 to 16) years. The
The median CD4 count at diagnosis (available for 112 women) was 310 (range 3 to 877) cells/mm$^3$. The median CD4 count from the most recent test prior to 31 December 2007 (available for all women) was 448 (range 100 to 1341) cells/mm$^3$. Eighty-three (67%) women were receiving antiretroviral treatment on 31 December 2007. These women had been on antiretroviral treatment for a median of 3 (range 0 to 18) years. Sixty-seven of the 83 (81%) women receiving antiretroviral treatment had an undetectable viral load on the most recent test prior to 31 December 2007.

120 (98%) women had had at least one cervical smear since 1991. Sixty-nine (56%) women met the definition for yearly cervical smears. Table 1 shows a number of parameters for women who did or did not meet the definition of a yearly cervical smear. The proportion of women who received a cervical smear for each yearly period from 2003 to 2007 is shown in Table 2.

**Table 1. Parameters for women who did or did not meet the definition of a yearly cervical smear**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Yearly cervical smear</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n=69)</td>
<td>No (n=54)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>Median (range)</td>
<td>38 (22–59)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>Sub-Saharan African</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>Asian/South-East Asian</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>New Zealand European</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>European</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Pacific Island Person</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Māori</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>1</td>
</tr>
<tr>
<td>Required interpreter</td>
<td></td>
<td>15</td>
</tr>
<tr>
<td>Time since diagnosis (years)</td>
<td>Median (range)</td>
<td>4 (0–18)</td>
</tr>
<tr>
<td>Duration of follow up (years)</td>
<td>Median (range)</td>
<td>3 (0–15)</td>
</tr>
<tr>
<td>Those prescribed antiretroviral treatment</td>
<td></td>
<td>46 (67%)</td>
</tr>
<tr>
<td>Those where one of the first two clinic letters discussed the need for a yearly cervical smear</td>
<td></td>
<td>15</td>
</tr>
<tr>
<td>Cervical smears performed by</td>
<td>GP</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>HIV nurse specialist</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Gynaecology Service</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Sexual Health Service</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Mangere Refugee Centre</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Combination including Gynaecology Service</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td>Other combination</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
<td>2</td>
</tr>
<tr>
<td>Those with at least one abnormal cervical smear</td>
<td></td>
<td>41</td>
</tr>
</tbody>
</table>

The need for yearly cervical smears was documented in one of the first two clinic letters of 25 (20%) women.

The cervical smears were performed by a GP (n=33), an HIV nurse specialist (n=24), the Gynaecology Service (n=8), the Sexual Health Service (n=3), the Mangere Refugee Centre (n=1), a combination including the Gynaecology Service (n=31), another combination (n=15) and an unidentified clinician (n=5).
Fifty-three (43%) women had one or more abnormal cervical smears. The most abnormal cervical smear for each woman was abnormal squamous cells of undetermined significance (ASCUS) (n=4), cervical intraepithelial neoplasia (CIN) I (n=27), CIN II (n=6), CIN II/III (n=9) and CIN III (n=7). One patient with a cervical smear that showed CINII/III was found to have a poorly differentiated squamous cell carcinoma requiring chemo/radiotherapy.

Table 2. The proportion of women who received a cervical smear for each yearly period from 2003 to 2007

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of women diagnosed with HIV infection at the beginning of each year</th>
<th>Number of women who received a cervical smear</th>
<th>Proportion of women who received a cervical smear (%)</th>
<th>P value (using 2003 as the comparator)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2003</td>
<td>60</td>
<td>30</td>
<td>50</td>
<td>–</td>
</tr>
<tr>
<td>2004</td>
<td>70</td>
<td>45</td>
<td>64</td>
<td>0.11</td>
</tr>
<tr>
<td>2005</td>
<td>82</td>
<td>58</td>
<td>71</td>
<td>0.01</td>
</tr>
<tr>
<td>2006</td>
<td>98</td>
<td>61</td>
<td>62</td>
<td>0.14</td>
</tr>
<tr>
<td>2007</td>
<td>113</td>
<td>73</td>
<td>65</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Eleven (9%) women had one or more abnormal cervical smears before they were diagnosed with HIV infection. Their details are shown in Table 3. Not all these women would have had HIV infection at the time of their first abnormal cervical smear.

Taking into account the women’s CD4 count at the time of the diagnosis of their HIV infection it is very likely that seven women (patients 4 to 9 and 11) had undiagnosed HIV infection at the time of their first abnormal cervical smear. It is likely that two women (patients 1 and 2) had HIV infection at the time of their first abnormal cervical smear.

It is unlikely that one woman (patient 3) had HIV infection at the time of her first abnormal cervical smear and one woman (patient 10) is known to have contracted HIV infection just after her first abnormal cervical smear.

The median time between the first abnormal cervical smear and the diagnosis of HIV infection for the women who were very likely to have had undiagnosed HIV infection at the time of their first abnormal cervical smear was 24 (range 3 to 44) months. The self-reported ethnicity of these women was recorded as sub-Saharan African (n=5), New Zealand European (n=1) and South-East Asian (n=1).
Table 3. Women who had one or more abnormal cervical smears before they were diagnosed with HIV infection.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Ethnicity</th>
<th>Age at time of abnormal cervical smear (years)</th>
<th>Date of abnormal cervical smear (abnormality)</th>
<th>Date of HIV diagnosis</th>
<th>CD4 count at time of HIV diagnosis (cells/mm$^3$)</th>
<th>Time between abnormal cervical smear and diagnosis of HIV infection (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Māori</td>
<td>19</td>
<td>1997 (CIN I)</td>
<td>2006</td>
<td>22</td>
<td>108</td>
</tr>
<tr>
<td>2</td>
<td>NZE</td>
<td>42</td>
<td>1998 (ASCUS)</td>
<td>2006</td>
<td>4</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2005 (CIN I)</td>
<td></td>
<td></td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>NZE</td>
<td>28</td>
<td>1988 (CIN II)</td>
<td>1993</td>
<td>818</td>
<td>60</td>
</tr>
<tr>
<td>4</td>
<td>South-East Asian</td>
<td>38</td>
<td>2002 (ASCUS)</td>
<td>2006</td>
<td>203</td>
<td>44</td>
</tr>
<tr>
<td>5</td>
<td>Sub-Saharan African</td>
<td>30</td>
<td>2004 (ASCUS)</td>
<td>2007</td>
<td>113</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>NZE</td>
<td>34</td>
<td>1993 (ASCUS)</td>
<td>1995</td>
<td>251</td>
<td>30</td>
</tr>
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<td>7</td>
<td>Sub-Saharan African</td>
<td>24</td>
<td>2004 (ASCUS)</td>
<td>2006</td>
<td>221</td>
<td>24</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Sub-Saharan African</td>
<td>37</td>
<td>2004 (ASCUS)</td>
<td>2006</td>
<td>477</td>
<td>18</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Sub-Saharan African</td>
<td>34</td>
<td>2004 (CIN I)</td>
<td>2004</td>
<td>102</td>
<td>7</td>
</tr>
<tr>
<td>10</td>
<td>Sub-Saharan African</td>
<td>47</td>
<td>2005 (ASCUS)</td>
<td>2005</td>
<td>722</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>Sub-Saharan African</td>
<td>24</td>
<td>2005 (CIN I)</td>
<td>2005</td>
<td>155</td>
<td>3</td>
</tr>
</tbody>
</table>


Discussion

In this audit of 123 women with HIV infection followed by our services for a median duration of 4 years, 120 women had at least one cervical smear but only 69 (56%) met our definition for yearly cervical smears.

Sixty-five percent of the women in this audit had a cervical smear in 2007. This is somewhat lower than two cohorts of women with HIV infection from the UK and USA that found that 73 and 77% of women respectively had a cervical smear in the previous year although the American cohort relied on self-reported cervical smear uptake.6,7 In 2002, it was estimated that 73% of all eligible New Zealand women had a cervical smear in the previous 3 years.8

The factors associated with receiving yearly cervical smears were women who had a shorter duration of follow up, women who had received their cervical smears from the Gynaecology Service and women who had at least one abnormal cervical smear. We would expect women with a shorter duration of follow up to have a higher rate of yearly cervical smears compared to those with a longer duration of follow up, as the
The importance of yearly cervical smears in women with HIV infection has become more widely recognised in recent years.

Women with a shorter duration of follow up will also have had less opportunity to miss one or more scheduled cervical smears. We would also expect that women receiving their cervical smears from the Gynaecology Service would have a high rate of yearly cervical smears given this service’s awareness of the need for yearly cervical smears in women with HIV infection and their effective recall system. This reason could also explain why women who had at least one abnormal cervical smear were more likely to have yearly cervical smears as the majority of women with an abnormal cervical smear were referred to the Gynaecology Service.

The only factor we found that was associated with not receiving yearly cervical smears was women who had received cervical smears from their GP. Not all GPs may have been aware of the need for yearly cervical smears in women with HIV infection. This potential lack of awareness was not helped by two factors. Firstly, clinic letters from our services to the woman’s GP often did not document the need for yearly cervical smears; only 20% of the first two clinic letters for women in this audit documented this issue. Secondly, the comment at the bottom of a normal cervical smear report from the National Cervical Screening Unit states that “the next smear should be taken at the usual screening interval”. It is only if the National Cervical Screening Unit knows that a woman is immunocompromised that the comment “please repeat this smear in 12 months” is included; we expect that most GPs would not have documented that women in this audit had HIV infection on the cervical smear request form.

We have instituted a number of changes in an attempt to increase GPs awareness of the need for yearly cervical smears in women with HIV infection. These include sending a letter documenting this issue to all GPs caring for women with HIV infection seen by our services, documenting this issue in the first clinic letter for women with HIV infection who are newly referred to our services and sending a list of all women with HIV infection seen by our services to the National Cervical Screening Unit stating that these women are immunocompromised. We will continue to send an updated list to the National Cervical Screening Unit once a year.

Another reason why women with HIV infection may not obtain a yearly cervical smear is the potential cost of this examination. While women seen by the Infectious Diseases Service could previously obtain cervical smears free of charge from our HIV nurse specialists, this service is no longer provided due to the increasing workload of our nurse specialists and the lack of an effective recall system. Women seen by the Sexual Health Service continue to be able to obtain cervical smears free of charge from this service.

Women seen by the Infectious Diseases Service may not be aware that a cervical smear can be obtained from Family Planning Clinics at a cost of 5 dollars for women with a community services card or from WONS (previously known as Well Women’s Nursing Services) in Auckland free of charge for Māori and Pacific Island women, women with a community services card and women who have not had a cervical smear within the last 5 years.
We have sent a letter to all women seen by the Infectious Diseases Service informing them of these options for obtaining a cervical smear. Despite the availability of a cervical smear at little or no cost, some women may be reluctant to access these options as this will require them to inform a new healthcare professional of their HIV infection.

Given the retrospective nature of this audit we were unable to explore other reasons why some women with HIV infection did not obtain a yearly cervical smear. We need to ensure that we carefully discuss the need for yearly cervical smears with all women with HIV infection. This is especially important for women who require an interpreter.

It is very likely that seven women in this audit had undiagnosed HIV infection at the time of their first abnormal cervical smear. Assuming that this was the case, the median delay in the diagnosis of these women’s HIV infection was 24 months. Six of these seven women had immigrated to New Zealand from sub-Saharan Africa or South-East Asia; areas with high rates of HIV infection.

The World Health Organization estimates rates of HIV infection in women aged 15 to 49 years of up to 20% in sub-Saharan Africa and 2% in South-East Asia. Cervical smear takers should consider offering an HIV test to all women with an abnormal cervical smear who have resided in areas with high rates of HIV infection. It should be acknowledged however that a similar risk based approach was not successful when used for antenatal HIV screening in New Zealand.

The strengths of this audit include the use of the National Cervical Screening Unit’s data and that our services see most women with known HIV infection in the Auckland and Northland regions. Use of the National Cervical Screening Unit’s data enabled us to obtain accurate cervical smear histories for all women. These data will be significantly more robust than studies that use self reported rates of cervical smear uptake.

While it can be argued that our definition of what constituted a yearly cervical smear was somewhat lax, we felt that this definition reflected the real world where there may be a delay between notification of the need for a cervical smear and having this test performed. This audit is limited by its retrospective design and its relatively small size.

The proportion of women with HIV infection in the Auckland and Northland regions who received a yearly cervical smear during the audit period was low. We have put a number of interventions in place that we expect will improve this rate. We have recently audited the proportion of women under active follow up by our services on 31 December 2007 who received a cervical smear in 2008. This showed that the number of women who had received a cervical smear in 2008 had increased to 73% (p=0.003, using 2003 data as the comparator).

We intend to repeat audits of the proportion of women with HIV infection who receive a yearly cervical smear at regular intervals to ensure that this rate continues to increase.
Competing interests: None known.

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Acknowledgement: We thank Hadir Elkerdani (Programme Principal, National Cervical Screening Programme, Auckland Region) for her assistance.

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References:


Homozygous familial hypercholesterolaemia and treatment by LDL apheresis

Richard I King, Russell S Scott, Christopher M Florkowski, Andrew D Laurie, Nicola Reid, Peter M George

Case report

We report the first known case of true homozygous familial hypercholesterolaemia (FH) in New Zealand and her treatment by LDL-apheresis.

This 13-year-old girl was referred to the lipid clinic by her general practitioner. A recent migrant, she attended requesting statin/ezetimibe combination therapy, as previously prescribed in her native South Africa.

She gave a family history of premature cardiovascular disease and elevated serum lipids. On physical examination marked tendon thickening of both Achilles and the extensor tendons of the hands were noted. Xanthomata were present on the elbows, Figure 1. No arcus corneae were visible. Echocardiography and CT coronary artery scoring indicate a moderately narrowed and calcified proximal aortic root but no coronary calcification.

Figure 1. Xanthomata affecting the extensor tendons of the hands, elbow and Achilles tendons

Biochemical analysis showed an off-treatment total cholesterol of 17.8 mmol/L, triglycerides 1.3 mmol/L and high-density lipoprotein 1.09 mmol/L, giving a calculated low-density lipoprotein (LDL) of 16.1 mmol/L. Lipoprotein(a) was 668 mg/L and apolipoprotein B 2.86 g/L (0.49–1.03).

She was found to be homozygous for the c.681C>G (D206E) mutation on analysis of the LDL-receptor gene, Figure 2.
Figure 2. DNA sequence electropherograms depicting part of exon 4 of the LDLR gene, showing the proband is clearly homozygous c.681C>G, and her parents are heterozygous for this variant.

Rosuvastatin treatment was commenced, and the dose titrated to 60 mg daily in combination with ezetimibe 10 mg daily. This resulted in a reduction in total cholesterol to 10.5 mmol/L and LDL to 9.0 mmol/L. Cholestyramine was added but her cholesterol level remained largely unchanged.

Despite the 37% reduction in LDL achieved with medication, she remained at significant risk of premature cardiovascular disease and hence LDL-apheresis was commenced.

Following her first treatment, the interval mean total cholesterol was 8.3 mmol/L, further treatments are scheduled at fortnightly intervals.

Discussion

Familial hypercholesterolaemia is an autosomal codominantly inherited condition characterised by high serum LDL concentrations leading to Fredrickson’s type II hypercholesterolaemia. The raised LDL results in deposition of cholesterol in peripheral tissues and accelerates atherosclerosis.
The monogenic inheritance of FH was first proposed in the mid-1960s by Khachadurian, Brown and Goldstein\(^2\) elucidated the LDL-receptor pathway, followed shortly afterwards by the cloning of the LDL-receptor gene and identification of the first mutation. Over 800 mutations have since been identified.

Five classes of mutation are recognised: class I are null alleles, class II affect protein transport from the ER to the Golgi, class III interfere with LDL binding, class IV hinder clustering in the coated pits and class V impede receptor recycling.

The ‘founder effect’ accounts for the increased frequency of the disease in the Afrikaner, French Canadian, Jewish, Lebanese and Icelandic populations.\(^3\) Three common point mutations, including the c.681C>G (D206E), so called Afrikaner-I mutation, account for approximately 90 % of inherited hypercholesterolaemia in Afrikaners.\(^4\) This class II mutation in exon four leads to the amino acid substitution Asp227Glu and is consistent with severe FH phenotype.

A genetic lipid disorder should be considered in patients with total cholesterol ≥8 mmol/L with or without a family history of premature coronary heart disease; the current New Zealand guidelines\(^5\) recommend referral to a lipid specialist for mutation analysis and cascade screening in these patients.

In our laboratory a combination of direct DNA sequencing and high-resolution melting analysis after PCR amplification are used to screen exons one to 17 for mutations.\(^6\) Multiplex ligation-dependant probe amplification (MLPA) is performed in selected patients to detect large deletions. The APOB gene is also screened for the phenotypically similar Familial Defective Apolipoprotein-B disease (FDB).

Treatment for homozygotes typically involves multiple medications in addition to diet and lifestyle changes. Statins, ezetimibe, resins and niacin may all be employed, and liver transplantation has been advocated. LDL-apheresis, involving extracorporeal filtration and selective removal of LDL, undertaken weekly or biweekly is accepted treatment for homozygous and compound heterozygous FH patients in Europe and the US.

Guidelines for patient selection and treatment goals have been published.\(^7\)\(^–\)\(^10\) Patients not achieving a 50 % reduction in plasma LDL on drug therapy are appropriate for LDL-apheresis and an interval mean total cholesterol of <7 mmol/L, or an acute reduction in total cholesterol of ≥65 % have been proposed as treatment targets.

In Christchurch, LDL-apheresis is run in conjunction with the NZ Blood Service and as such could become more widely available. Given the frequency of FH there are likely to be four to five undiagnosed homozygous or compound heterozygous patients in New Zealand who may benefit from this treatment modality.

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Acknowledgement: The authors thank Dr K Badami and staff at the NZ Blood Service for support in setting up the apheresis service.

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References:
Angiomatosis: a case report

Hamesh Jina, Philippa Mercer, Malcolm Gordon, Harsh Singh, Martin Whitehead

A 19-year-old male was involved in a motor vehicle accident where he sustained a closed acetabular fracture and a left posterior hip dislocation. The preoperative CT abdomen showed an incidental finding of a right 8cm adrenal mass with extensive retroperitoneal and mediastinal lymphadenopathy (Figures 1, 2).

Figures 1 and 2. CT Abdomen arterial phase in coronal plane and transverse plane. Findings: Large adrenal mass which shows renal vessel invasion and IVC displacement anteriorly. Tumour is adherent to right crus and posterior diaphragm. Extensive ipsilateral, retrocural, retrocaval and contralateral para-aortic lymphadenopathy (not shown)

Following orthopaedic fixation at Southland Hospital, the patient was referred to the endocrine surgeons at Christchurch Hospital for further investigations. Preoperative laboratory findings included normal urine and serum catecholamines, aldosterone, cortisol, αFP, βHCG and sex hormones. The provisional diagnosis was a right adrenal adenocortical carcinoma.

The operation involved endocrine, cardiothoracic and vascular surgeons. A subcostal thoraco-abdominal approach was undertaken with aortocaval and mediastinal lymph node resection, ligation of the renal and adrenal vessels and the tumour and kidney dissected en bloc. The diaphragmatic repair was performed using a dual mesh patch. There was further mediastinal lymph node involvement superior to the carina although this was inaccessible to resection with approach used. The lymphadenopathy was considerably more extensive than what was demonstrated on the preoperative imaging.

Histology from the right adrenal gland, right kidney, diaphragm, right crus and mediastinal lymph nodes gave a provisional diagnosis of a kaposiform
haemangioendothelioma. There were 56 lymph nodes in the abdominal dissection although none were implicated in the disease. The tumour was present on the superior margin of the main specimen encompassing diaphragmatic skeletal muscle fibres. The histological specimen was sent internationally for further opinions and after several months and much controversy, a final diagnosis of angiomatosis was made.

Postoperative recovery was smooth with an uneventful overnight admission to ICU. He was an inpatient for a week and discharged stable. The patient was followed up two weeks later and had recovered well. He was not offered adjuvant therapy and further surgery is not currently considered.

Discussion

Angiomatosis is a rare benign vascular lesion which presents during childhood or adolescence. The lesion will slowly and typically develop in a contiguous fashion in single or multiple tissue types (e.g. subcuticular, muscle or bone). The incidence and prevalence of the condition is unknown. Over 50% of the lesions are usually confined to the lower extremities and 72% of cases present within the first 2 decades of life.\textsuperscript{1} Familial cases are rare.\textsuperscript{2} The clinical presentation is typically with persistent swelling often associated with pain and skin pigmentation. There are characteristic histological features.

The diagnostic workup involves clinical assessment, imaging and histology. CT is the imaging modality of choice and the lesion appears as a non homogeneous mass which has similar features to a sarcoma except dense areas due to thick-walled tortuous vessels.\textsuperscript{1} There is often a lot of fat which can confuse the diagnosis. Histology is the diagnostic gold standard.

Typical histological features can assume two patterns.\textsuperscript{3} Most commonly, there is an irregular collage of veins, cavernous vascular spaces and capillaries diffusely positioned. The venous vessels have irregular thick walls and arteries are occasionally identified with a variable incomplete muscle layer (Figures 3, 4). The less common histological presentation is characterised with smaller vessels arranged in nodules around a larger vessel which diffusely infiltrates soft tissue, unlike a capillary haemangioma.
Spontaneous regression of the tumour is rare. Treatment options are limited due to rarity of the condition. Conservative therapy, immune modulating therapies (e.g. interferon, steroids and cyclophosphamide) or surgery are acceptable treatment options and have been suggested in the literature. Ideal follow up time is not known and in the series by Rao and Weiss,1 patients were followed up from 1-24 years. They found that nearly 90% of patients had recurrences with 40% having recurrences within 5 years and this varies in the literature from 60–90%.3,4 The high recurrence rate means that these lesions are difficult to treat.

Conclusion

This case led to the incidental finding of a rare vascular tumour. Angiomatosis is predominantly a childhood benign lesion and variable in its presentation. The diagnosis is confirmed on histology. There is no gold standard in treatment and conservative and surgical modalities are both supported in the literature.

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References:

A case of intestinal-type gastric adenocarcinoma metastatic to a caecal tubulovillous polyp

James McKay

Case report

Mr M, a 70-year-old man, was referred for surgical review for heartburn, reflux and early satiety. Past surgical history included a pyloroplasty and gastrojejunostomy (GJJ) in 1965 for a pyloric peptic ulcer.

Gastroscopy revealed a large incarcerated hiatus hernia, severe oesophagitis, Helicobacter pylori gastritis and a normal gastrojejunostomy.

CT confirmed the hernia, which was laparoscopically repaired with a 180-degree posterior wrap, resulting in marked symptom improvement. Six months later, symptoms had returned in addition to weight loss and post-prandial vomiting.

Barium swallow and endoscopy confirmed complete gastric outlet obstruction and CT showed marked dilatation of GJJ limbs from the previous anastomoses.

Laparotomy revealed a distended, thick-walled stomach, palpable tumour at the GJJ, serosal deposits and an incidental caecal mass. Distal gastrectomy, resection of previous GJJ and en-bloc extended R) hemicolectomy was performed.

Histology confirmed intestinal-type gastric adenocarcinoma with synchronous metastasis to a caecal tubulovillous adenoma.

Discussion

Metastatic spread of gastric cancer is not uncommon, but colonic metastases are rare. Niimi et al reported two cases of large bowel metastases of gastric cancer; one localised to sigmoid colon, the other with rectal and transverse colon lesions but none sited in a polyp.

Ogiwara et al reported a case with multiple colonic polyps shown as metastatic deposits of poorly differentiated adenocarcinoma; the primary being gastric cancer resected 11 years previously. Lee et al reported a case of colonic metastases from gastric cancer in the form of 5 or 6 flat slightly elevated lesions throughout the colon with a signet-ring pathology similar to the gastric tumour.

The closest case we could find to Mr M was published by Tiszlavicz of a 69-year-old man with diffuse type gastric cancer, where post-mortem found widely disseminated disease with a metastasis in an adenomatous polyp of the caecum.

As to why a polyp would be an isolated site for a metastatic deposit is unknown, and even more unusual about Mr M is it being present on the mucosal and not serosal surface as may be expected with transcoelomic spread. The pathogenesis of such a lesion could not be adequately explained other than hypothesising that the spread is likely haemotogenous/lymphatic in nature with certain cell expression.
factors/adhesion molecules in the adenomas that allow the tumour cells to settle and grow there. This may be more so with the intestinal-type gastric tumours given their histological morphology is described as being like that of intestinal mucosa?

Mr M is, to our knowledge, the first known report of intestinal-type gastric cancer with metastatic spread to a tubulovillous caecal adenoma.

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**Acknowledgement:** The author thanks Mr Alf Deacon for his verification of conclusions and for his helpful advice.

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**References:**

Refractory ascites due to ascites praecox

Pazhanivel Mohan, Jayanthi Venkataraman

A 14-year-old girl presented with gradually increasing abdominal distension for 6 months. She recently noticed mild breathlessness on exertion. Examination revealed a tense ascites with mild oedema of the lower limbs. Her jugular venous pulse (JVP) was not seen to be elevated in the supine or 45° position but was raised up to the ear lobes in sitting up position. A pericardial knock was heard on auscultation.

Lateral X-ray of the chest showed calcification of the pericardium (Figure 1). CT scan showed a circumferential calcification of the pericardium (Figure 2) along with massive ascites (Figure 3).

Figure 1. X-ray chest (lateral view)

Figure 2. CT scan of the chest

Figure 3. CT scan of the abdomen
Echocardiography findings were consistent with constrictive pericarditis. She underwent a pericardiectomy with complete resolution of symptoms. Tuberculosis was confirmed as the cause of chronic calcific constrictive pericarditis on the histologic sections of the pericardium postoperatively.

Ascites secondary to constrictive pericarditis typically occurs before the oedema of the lower limbs, unlike other cardiac causes. Hence it is referred to as ‘ascites praecox’. Abdominal signs such as hepatomegaly and ascites frequently over shadow the cardiac signs causing difficulty in diagnoses.

The most important physical sign in constrictive pericarditis is a raised JVP which is often missed as it is grossly elevated above the angle of the jaw. A sitting up position is preferred in such cases. The importance of making an accurate diagnosis lies in the fact that surgical intervention can provide complete relief of symptoms in these patients.

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Report of St Helen’s Hospital, Dunedin

Published in NZMJ 1910 August; 8(35):45–48 and compiled by Emily H. Sideberg, Physician and Lecturer at St Helen’s Hospital.


((Libraries, print out the PDF from the link above and replace this page))
Equipment of Yore: a contribution from the Cotter Medical History Trust

Max H Abernethy

Can you identify this instrument? Inside the brass box (4×4×3 cm) there are two axles, each holding a rack of six thin sharp steel plates. At the base are two parallel rows of six narrow slits. The trigger-shaped handle on the top is a cocking device that tensions strong springs attached to each axle. Pressing the button on the side produces an audible click but no visible action.

These instruments date from the 1600s and appear in our earliest medical catalogue, that of S Maw and Sons of 1866, and as late as the Aesculap catalogue of 1973 in their vaccination section.

Figure 1. An instrument thought to date from mid 19th Century engraved ‘H&H Hilliard Edinburgh’
Those thin plates on the two shafts are sharp rounded blades. The shiny metal pieces under the axles are strong springs, cocked by the trigger. When pressed, the button at the side releases these springs to make the opposing rows of blades spring out through the slits, far too quickly to see. Their arcs are up to 15 mm long, adjustable to between 3 and 8 mm deep. This design was a great advance on its predecessors as it eliminated the recoil of a single axle.

**Answer**—The instrument is a *scarificator*, originally to puncture the skin for bloodletting. The associated ‘cupping’ involves a partially evacuated glass bottle quickly placed over the wound. The cup’s low pressure promoted bleeding.

Typically, a skilled bloodletter (one slick enough never to spill a drop) took 8 to 16 fluid ounces of blood from virtually anywhere on the skin surface. Bleeding’s heyday as a panacea was the early 19th Century. Perhaps an American aptly named Physick who, at 90 ounces at one go,\(^1\) may hold the record! Whichever bloodletting atlas was at hand prescribed the site for the patient’s condition. Even the temples!

The patient’s approaching unconsciousness was a common end-point of the procedure.
Springs and gears need greasing. This and its general complexity obviated sterilization. However, (hygiene conscious?) makers sold blade-cleaning mats of suede leather or other soft material.  

Bloodletting slowly became outmoded in the 19th Century as evidence to its dangers grew.  

An early trial, described in Alexander Hamilton’s MD thesis, occurred in the Peninsula wars in 1809. Hamilton and two British Army surgeon colleagues each cared for similar batches of over 100 casualties each. The non-bleeders, Hamilton and Anderson, lost 4 and 2 patients compared with 35 deaths under their regularly bleeding third colleague.

Later, in 1873, Dr Shann cautioned not to lightly dismiss such a well-established practice as bleeding in his case report of an oesophageal puncture.

By 1900 therapeutic bleeding virtually stopped, except for conditions like haemochromatosis. However cupping remains alive and well among alternative practitioners, but thankfully, deliberate bleeding seems restricted to a few token drops.

More recently, the scarificator sold solely as a vaccination instrument. This is the purpose of the Aesculap example. But what vaccine requires such drastic application?

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References:


2. Davis A, Appel T. Bloodletting instruments in the National Museum of History and Technology. Smithsonian studies in history and technology. 1979, no.41. This authoritative account of scarificators and bloodletting is online at  
   http://www.sil.si.edu/smithsoniancontributions/HistoryTechnology/pdf_lo/SSHT-0041.pdf  
   (large file).

   http://jech.bmj.com/content/56/1/1.2.full

Perceptual learning and intelligibility gains in dysarthria associated with Parkinson’s disease. S. A. Borrie¹, M. J. McAuliffe¹, J. M. Liss², C. Kirk³, G. A. O’Beirne¹, & T. Anderson⁴. ¹New Zealand Institute of Language, Brain and Behaviour and Department of Communication Disorders, University of Canterbury, ²The Department of Speech and Hearing Science, Arizona State University, ³Department of Special Education and Clinical Services, University of Oregon, ⁴Van der Veer Institute for Parkinson’s and Brain Research, Christchurch.

Perceptual learning describes the effect whereby experience with a specific signal alters a listener’s perceptual processes during subsequent encounters with that same signal. Research investigating perceptual learning with foreign-accented speech (e.g.2) and artificially degraded acoustic signals (e.g. 3) provides substantial evidence regarding the perceptual benefit of prior exposure. While such findings may have significant implications for the management of neurological speech disorders, existing research with such populations is minimal and study findings have been equivocal.

Sixty healthy listener participants were randomly assigned to one of three perceptual learning conditions: (1) control; (2) passive familiarisation; and (3) explicit familiarisation. Word accuracy of listener transcriptions of phrase level stimuli produced by speakers with a moderate hypokinetic dysarthria secondary to Parkinson’s disease were used to document the magnitude and stability of effects of a familiarisation experience on speech intelligibility. In addition, listener transcripts were analysed for lexical boundary errors (suprasegmental level) and syllable perception (segmental level) to investigate the cognitive-perceptual processes which may underlie any benefit associated with familiarisation.

Statistically significant improvements in intelligibility were observed for familiarised listeners relative to the control group, with explicitly familiarised listeners demonstrating greatest intelligibility gains following exposure to the degraded signal. At follow-up, intelligibility for listeners who received explicit familiarisation remained significantly higher than controls, however no lasting intelligibility gains were observed for listeners who received passive familiarisation. Statistically significant differences were also observed for all groups on syllable level measures and type and distribution of lexical boundary error patterns.

Findings provide compelling evidence that listeners can “learn” to better understand neurologically degraded speech, and furthermore, that explicit familiarisation facilitates largest and lasting intelligibility gains. Changes to both segmental and suprasegmental levels of perceptual processing advance the current understanding of learning mechanisms associated with perception of neurologically-disordered speech, and provide direction for future research.


Does albumin overload albuminuria increase urinary Cystatin C excretion? M Nejat, JV Hill, JW Pickering, ZH Endre. Christchurch Kidney Research Group, Department of Medicine, University of Otago, Christchurch, New Zealand

Urinary cystatin C (uCysC) has been proposed as a marker of tubular kidney injury. Albuminuria may interfere with tubular reabsorption of uCysC. We evaluated whether albuminuria induced in rats via intraperitoneal (IP) bovine serum albumin (BSA) would increase uCysC.

Seven male and female (6-8 week old) Sprague-Dawley rats received 5 mg.day$^{-1}$.g body wt$^{-1}$ IP BSA in saline. Four rats of each sex received saline only. Injections were given twice daily for 2 consecutive days. Rats were placed in metabolic cages with free access to food and water on the day before injection (Time 1), the day following the last injection (Time 2) and, 96 hours later (Time 3). Timed collections of spontaneously voided urine was obtained to measure albumin, total protein, and uCysC. Rate of uCysC, protein, and albumin excretion were calculated. Repeated measures two-way ANOVA was used to find the effects of sex, BSA injection, and time of sampling. Post-hoc, Fisher’s LSD and Pearson’s correlation were used to explore the primary effects and interactions.

Male rats excreted more protein, albumin, and uCysC than female rats (p<0.0001, <0.0001, and <0.01, respectively). In both sexes, BSA treatment caused an increase in the rates of excretion of protein (p<0.01), albumin (p<0.05), and uCysC (p<0.05). The rates of protein and albumin excretion were significantly correlated with the rate of uCysC excretion ($r^2=0.54$ and $r^2=0.29$ respectively; p<0.01). In BSA treated rats, urine analytes were similar between Time 1 and Time 3, which show a transient effect of BSA overload. The control groups showed no significant changes in urine analytes over time.

There was a significant increase of uCysC excretion in rats with induced albuminuria. The degree of cystatinuria and albuminuria were correlated. Males had higher albuminuria and cystatinuria than females.

**Hunger hormones and binge eating: an interaction between biology and behaviour. KJ Taylor, VVW McIntosh, J Jordan, P Joyce. Department of Psychological Medicine, University of Otago, Christchurch.**

Eating disorders are often chronic illnesses which significantly impact on the lives of those affected. In those with eating disorders, appetite is ignored, and individuals learn to ignore hunger signals or are unable to stop eating in response to satiety signals. Disregard of these signals has a deleterious impact on wellbeing, including
weight status. As such, studying satiety hormones such as ghrelin may contribute to the development of new treatments. Ghrelin, a 28-peptide neurohormone, is the only known peripheral orexigenic hormone. Laboratory studies show fasting and energy restriction increase circulating ghrelin whereas normal food intake and over-eating reduce ghrelin levels. It seems likely that insulin may modulate this response.

This study investigated ghrelin and insulin in 44 women with binge eating (bulimia nervosa (BN) and binge eating disorder (BED)) compared to 44 women without binge eating matched for age and body mass index (BMI). The relationship between ghrelin and the frequency of bingeing, purging and restricting was also investigated. Participants presented after fasting overnight, and blood samples were taken at regular intervals pre- and post- an oral glucose load.

The control group showed the expected pattern of an increase in insulin and a decrease in ghrelin after ingestion of the glucose. There was a similar pattern overall in the BN group, however, the increase in insulin was smaller and ghrelin secretion was lower. Women with BED showed a different pattern with a trend towards higher insulin levels but reduced ghrelin secretion than controls. The BMI of the BED group was higher than the BN group and it is likely that this contributed to the higher insulin levels. Frequency of bingeing and purging was positively correlated with ghrelin in the BN group. These results suggest that eating disorder pathology may influence ghrelin secretion.

High resolution multi-energy CT imaging of atherosclerotic plaque. R Zainon¹, N Cook², AP Butler³⁻⁵, SP Gieseg⁶, NG Anderson⁴, TM Buckenham⁴, G Shelkov⁵, L Tlustos⁵, JA Roake⁷, P Butler¹⁻⁵. ¹Department of Physics and Astronomy, University of Canterbury, Christchurch, ²Department of Medical Physics and Bioengineering, Canterbury District Health Board, Christchurch, ³Department of Electrical and Computer Engineering, University of Canterbury, Christchurch, ⁴Department of Radiology, University of Otago, Christchurch, ⁵European Organisation for Nuclear Research (CERN), Geneva, Switzerland, ⁶Free Radical Biochemistry Laboratory, School of Biological Sciences, University of Canterbury, Christchurch, ⁷Department of Vascular, Endovascular and Transplant Surgery, Christchurch Hospital.

Recent advances in computed tomography (CT) scanner technologies have generated a great deal of interest in dual-energy CT. With dual energy computed tomography quickly becoming the clinical standard, spectral X-ray imaging is the next step in clinical imaging. In this study, we assess the ability of high resolution multi-energy MARS-CT in imaging excised atherosclerotic plaque components. The MARS research group based at Canterbury and Otago Universities in New Zealand have constructed a multi energy CT scanner using the Medipix detectors. This scanner provides high resolution imaging of tissue components without the use of contrast agents. This technology is being developed to generate high resolution spectral images of atherosclerotic plaque showing the location of calcium, lipid and soft tissues. The fine structure and composition of the atherosclerotic lesion, rather than the degree of stenosis, are currently considered to be the important determinants for acute clinical events.
Human atherosclerotic plaque specimens, which had been imaged in vivo, were excised during surgery and set in plastic to stabilise the tissue for imaging in the MARS-CT scanner. The scanner uses a microfocus X-ray tube operating at 75 kV with a target current of 0.15 mA. For the scan 303 projects were taken, each been recorded at eight separate energies levels ranging from 14.45 keV to 39.2 keV. This allowed for high resolution CT images at a resolution of 43 µm to be obtained with spectral information. Colour X-ray CT images of atherosclerotic plaques were obtained using Principal Component Analysis (PCA).

The plaque with calcium is clearly distinguishable from the non-calcified plaque by both the magnitude of the attenuation values and also spectroscopically. Images obtained from spectral CT provide advantages in imaging of human plaques giving novel characterisation of complex atherosclerotic lesions in vivo and offering new insight into plaque development.


Pets or pests in airplane cabins?

The Canadian Transportation Agency is reviewing its policy on allowing cats, dogs and birds in airplane cabins following complaints from passengers with allergy to pet dander. Obviously significant reactions, such as anaphylaxis in mid air, would pose major problems. How significant is the problem—could it be solved by seating allergic subjects away from the pets? Probably not.

A study by IR Martin and colleagues in our journal (NZMJ 1998;111:356–8) is cited, as it identified clinically relevant concentrations of cat allergen on 100% of sampled airplane seats on domestic flights and 16% of seats on international flights. The authors of this editorial are firmly in favour of banishing pets to the cargo holds (with the exception of service animals—presumably blind aid dogs).

CMAJ 2010;182:421.

Low dose aspirin for primary prevention of cardiovascular disease?

Daily low dose aspirin is established in the secondary prevention of cardiovascular disease. This benefit has been extrapolated and some believe that it should be used as a primary prevention agent in those without proven cardiovascular disease who have risk factors such as diabetes and hypertension. Indeed the British Hypertension Society have recommended this for hypertensive subjects this year. This review refutes this and points out that although such treatment does reduce serious vascular events this benefit is offset by a significant increase in major gastrointestinal or other extracranial bleeding. The reviewers, two of whom are associated with the “Drug & Therapeutics Bulletin” in the UK, point out that low dose aspirin is not licensed for primary prevention in the UK. We can expect a riposte from the Hypertension Society.


Myocardial infarction and heart failure—is there an association with the use of non-steroidal anti-inflammatory drugs (NSAID)?

Current evidence suggests the use of cyclo-oxygenase-2 (selective) NSAIDs and the non-selective NSAIDs are associated with an increased risk of myocardial infarction and heart failure. However, debate continues about this association. This paper from South Australia reports on a retrospective nested case control study on Australian veterans. The numbers involved are large—83,623 NSAID users and 1,662,099 matched non-users. And they concluded that use of NSAIDs within the last 2 years did not increase the risks of myocardial infarction or heart failure. There was a modest reduction in all-cause mortality in the users of NSAIDs or selective NSAIDs.

Chronic obstructive pulmonary disease (COPD)—antibiotic therapy for acute exacerbations?

This large retrospective study involves 84,621 patients hospitalized with an acute exacerbation of their COPD in 413 hospitals throughout the USA. The issue is whether early antibiotic treatment produced better outcomes compared with late or no antibiotic treatment. Apparently 79% received early antibiotics. These patients fared better than the untreated cohort—viz less mortality, less need to mechanically ventilate and less need for readmission within 30 days. One downside was that the antibiotic treated patients had a slightly higher rate of readmission for Clostridium difficile problems (0.19% vs 0.09%). Their conclusion is that early antibiotic treatment is appropriate for those hospitalized with acute exacerbation of their COPD.

JAMA 2010;303(20):2035-42.

Chronic nasal sinusitis—does therapeutic ultrasound help?

Antibiotics often do not cure nasal sinusitis. One of the reasons may be that the bacteria may be inaccessible because of biofilm formation. We recall that a biofilm is a community of micro-organisms that are associated with a surface and typically enveloped in an extracellular matrix. The authors of this paper (from Auckland) note ultrasound may disturb such biofilms and allow antibiotic penetration. They treated 22 subjects who had not responded to antibiotics with therapeutic pulsed ultrasound at 1MHz—6 sessions of 20 minutes over 2–3 weeks. 2 patients did not complete the treatment because of inability to attend. 18 patients had improvement in symptoms and 2 had worse symptoms. Interestingly these 2 had Type 2 diabetes.

They conclude that therapeutic ultrasound is useful. Antibiotics were not used in these patients—presumably ultrasound and antibiotics might be better still. The authors note that greater numbers of patients and a control group would have been desirable.

The 1987 National Women’s Hospital (NWH) ‘Unfortunate Experiment’. Accusations of unethical experiments and undertreatment, resulting in excess deaths from cervical cancer. Facts and fables

Was there an ‘Unfortunate Experiment’?—From the early 1960s NWH clinicians became increasingly aware that eradication of grade 3 cervical dysplasia CIN3, (abnormal cells but not cancer) was unpredictable and often had little relationship to the initial management or the completeness of excision of the lesion—thus the standard management of immediate, major, sterilizing surgery of hysterectomy on young women began to be questioned.

In 1966 Professor Green and other senior NWH clinicians endorsed policy changes in dysplasia management. Younger women were to be continuously monitored, by repeat smears, colposcopy, lesser biopsies and appropriate more major surgery if evidence of early cancer. This policy was later described in Metro 1987 as the Unfortunate Experiment. In this era, diagnosis of CIN3 rapidly escalated worldwide. Opinions in grading of cytology, histology and microinvasion were contentious and often acrimonious.

The McIndoe Review—In 1984, three NWH staff, a pathologist, and a colposcytologist (both now deceased), and an obstetrics and gynaecology (O+G) clinician Dr Ron W Jones, published a retrospective review (known as the McIndoe Paper) of the management of 948 women presenting at NWH 1955–76 with CIN3. The objective of the review was to clarify whether post-treatment continuing CIN3, was related to the type of initial treatment or the completeness of excision of the lesion. Treatment had been by three separate clinical teams at NWH.

The McIndoe authors in 1984 divided the 948 women reviewed, into two groups, based on cervical cytology two years after initial treatment. In the 817 group 1 women with post-treatment normal smears, the initial treatment had been hysterectomy in 217 (26%), cone excision 579 (70%), amputation of cervix 6, punch or wedge biopsy 15. In the 131 group 2 women with ‘post-treatment’ continuing abnormal smears (CIN3), the initial treatment had been hysterectomy in 33 (25%), cone excision in 88 (67%) and punch or wedge biopsies in 10 (p454 para 4). Note the percentage of initial major
treatments is similar in groups 1 + 2. Because of post initial treatment continuing CIN3, the 131 group 2 women received a subsequent 107 additional major treatments of hysterectomy in 29 and further cone excisions in 78. The total major treatments in these 131 group 2 women, were now a total of 228.

The genesis of the NWH 1987 Inquiry was a 1983 liaison between two groups disaffected with the two senior NWH Professors. The first group was the authors of the 1984 McIndoe Paper who believed that conservative management of CIN3 was an increased risk of invasive cancer—but could not convince the Professors or most of the senior NWH clinical staff. The second group comprised of members of a women’s reform group (Fertility Action) who had antipathy to male-dominated issues of women’s health and one of the Professor’s negative attitude towards ‘abortion on demand’, rather than than for medical reasons.

Unexpectedly, the 1984 McIndoe Paper statistics listed below showed no relationship between the type of initial treatment or the completeness of excision of the lesion—to persisting CIN3.

- ‘Thus whether or not the lesion is completely excised does not appear to influence the possibility of invasion occurring subsequently’.1 (p457 para 4)

- ‘The 817 patients in group 1 remained clinically and cytologically normal for the first 4 years after the initial biopsy irrespective as to whether or not there was evidence of complete excision of CIS’.1 (p453 para 6)

- ‘The 131 patients in group 2 continued to produce abnormal cytology—irrespective of the initial management or completeness of excision of the lesion’.1 (p454 para 4) This failed to confirm the McIndoe authors’ belief of dangers in conservative management. What to do now? The solution—create a major scandal.

**Genesis of a major medical scandal**—The division of the 948 women reviewed 1955–76 into groups 1 and 2 was by the McIndoe Paper in 1984, and was based on positive or negative smears 2 years after initial treatment. However the McIndoe Paper text paradoxically implies groups 1 and 2 were two separate groups treated differently in an unethical prospective study (1955–76). Page 458 para 3 states ‘the conservative management of group 2 patients in whom complete excision was not considered a necessity’.1 There were no groups 1 and 2 in the 1955–76 era reviewed.

The McIndoe authors further enhanced the above damaging inferences by stating—that continuing abnormal cytology after initial treatment had a high risk of developing cervical cancer—again inferring inadequate initial treatment. They failed to inform that after their 121 initial major treatments, the 131 group 2 women received a subsequent 107 major treatments of hysterectomy in 29 and cone excision in 78. A total of 228 major treatments in 131 group 2 women.

The McIndoe authors ‘concealed’ the subsequent 107 major treatments in the 131 group 2 women, uncured by their initial 121 major treatments. They stated they were further biopsies. ‘Final diagnosis in this group was established by further biopsy’—‘by cone biopsy in 78 and hysterectomy in 29’.1 (p455 para 2) Stating these 107 additional, major treatments were ‘further biopsies’, later gave validity to the 1987 Fertility
Action, _Metro_ accusations of ‘limited or no treatment’ of the 131 women of McIndoe group 2.

**Perfidious manipulation**—In _Metro_ 1987, members of Fertility Action compounded the McIndoe Paper’s damaging inferences of two separate groups treated differently. They stated that the 1984 ‘McIndoe group 2’ of 131 ‘uncured’ women, was a group 2 of 131 ‘limited or no treatment’ women. Ref. _Metro_ June 1987 p60 para 5, ‘12 of the total number had died of invasive cancer, 4 or 0.5% of group 1 women, and 8 or 6% of the group 2 women who had limited or no treatment’. This fiction was accepted. The Unfortunate Experiment was now a proven reality and remains so.

**The McIndoe text is duplicitous**—Are the 1984 McIndoe cancer deaths’ statistics correct? They showed 41 cancers and 12 cancer deaths (1.25%) in 948 women treated for CIN3 at NWH 1955–76. In 1988 a larger review, 1955–86, was completed by two senior NWH cancer unit clinicians. It included the 948 women from the 1955–76 reviewed in the McIndoe Paper. This later review showed 32 cancers and 8 cancer deaths (0.25%) in 3037 women treated for CIN3 in the 30 years 1955–86. These discrepancies require clarification.

In a book _The Unfortunate Experiment_ (Penguin 1988, p17) a 1987 _Metro_ author confirms that in 1985 (2 years before their 1987 _Metro_ article) they knew that groups 1 and 2 were not treatment-based but were cytology-based and designated as such in 1984. They were thus aware that the McIndoe 131 group 2 women had received 228 major treatments. This _Metro_ unchallenged accusation of a mythical ‘limited or no treatment’ group 2, precipitated a major medical scandal—The Unfortunate Experiment.³

In the above book, _The Unfortunate Experiment_, page 6 refers to the final submission of the Ministry of Women’s Affairs to the 1987 Inquiry, ‘Ultimately the issues are about who controls medicine’ and ‘the central issue above all others is power’. This suggests that the Ministry’s agenda was wider than an investigation into alleged patient mismanagement. There was rapid endorsement of the 1987 _Metro_ revelations, by the Ministry of Health, the Ministry of Women’s Affairs, Fertility Action and sensation-seeking media.

A Judicial Inquiry, and its terms of reference, was announced by the Minister of Health, just 6 days after the _Metro_ accusations. This announcement was prior to any dialogue with the accused NWH clinicians or their employers, the Auckland Hospital Board. Was the Inquiry waiting in the wings for the _Metro_ exposé?⁴

**The 1987 Judicial Inquiry**—Pivotal was the McIndoe authors (including Dr Jones’) continued silence throughout the Inquiry in regard to the correct statistics of 228 major treatments in 131 women they designated as group 2 in 1984. This allowed the Fertility Action accusations of ‘limited or no treatment’ in the 131 group 2 women to remain unchallenged.

This incorrect information presented to the Inquiry by two disaffected groups, was accepted.

Inquiry Report p95: ‘The _Metro_ article and its emphasis are correct’. Inquiry Report p150: ‘The McIndoe Paper distinguishes between two groups and 22% of those whose abnormalities were ‘untreated’ developed invasive cancer.’ Following the acceptance
of these extreme accusations, the acceptance of others, involving ‘patient’s rights and informed consent’, were little more than a formality. For over two decades this fallacy of an undertreated group 2, has been promoted and zealously guarded by a coterie of agenda driven groups—some not medically qualified, and others ‘non’ clinicians. They have powerful political connections, essentially unlimited resources, favoured media access and show degrees of demeaning paranoia to opposing opinions.

Following the ‘scandal’ of the ‘Unfortunate Experiment’ there were demands for major changes in control and direction of New Zealand Health Services. These were very successful. The Medical Profession was essentially disenfranchised and mainly excluded from the Medical Council. Expensive, escalating bureaucracy, sympathetic to specific agendas, became the new order.

The Post Graduate School of O+G was closed. The respected NWH and the Greenlane Cardiac Unit were downgraded. St Helen’s Obstetric Hospital was closed and midwives replaced doctors as lead caregivers in maternity services. General practitioners were strictly controlled. Availability of state-funded first class medical services has rapidly declined. Many New Zealand doctors have moved overseas to better salaries, less control by bureaucrats and greater respect for their contributions.

The learning curve—Optimal management of CIN3, in the 1960s–70s was controversial and sometimes acrimonious. Contrary opinions were rife and exploited by Fertility Action in their 1987 fictitious Metro accusations. The astute observations of clinicians, such as Professor Green and others worldwide, were well ahead of the ‘later clarification’ of the role of the human papilloma virus. Countless young women were thus spared unnecessary, major sterilizing surgery. Who did what, how, where and when, in this 40–50 years dysplasia treatment debate shows an excess of variable subjective opinions. The only non-contestable denominator is ‘patient’ deaths. To repeat—8 cancer deaths in 3037 women presenting at NWH with CIN3, over 30 years, 1955–86, confirms excellent management. NWH deserves praise and gratitude. Vindication of the wrongly accused and disgraced NWH clinicians is of paramount importance.

Postscript—The value and strengths of a democratic process are that credible and verifiable opposing opinions should be able to be expressed without prejudice—and be open to public debate. However in revisiting the Unfortunate Experiment this is not possible. Contrary opinions are not welcome, are seldom printed and invoke demeaning criticism rather than discussion. In New Zealand, revisiting the Unfortunate Experiment is a minefield inviting self-destruction. This is a dangerous precedent and merits urgent discussion involving the public, politicians and unbiased media.

Graeme H Overton
Senior Consultant (FRCOG, FRCS)
Associated with National Women’s Hospital 1960–99

Reference and endnotes
2. A memo from Statistician of 1984 McIndoe Paper to the lawyer Mr Kevin Ryan, 15-6-90, 
“The implication that the abnormalities were untreated is, on the information presented in our 
1984 paper, quite false: the group was defined as “continuing to produce abnormal cytology”, 
not as having been untreated. Again the 1984 Paper was in terms of a second group of patients 
who “continued to produce abnormal cytology”, not a group that was “conservatively treated”.

3. Confirmation that the 1987 Metro authors were aware group 2 was not untreated. Book “The 
concerned how the authors had divided the women into two groups. “We thought this had 
been done on the basis of the treatment they had received, whether conservative or otherwise. 
But the key fact in establishing the two groups, had actually been whether the women had 
positive or negative cytology” (post treatment).

4. Confirmation by Superintendent in Chief of Auckland Hospital Board (AHB) of 
announcement of Judicial Inquiry prior to discussion of Metro accusations with NWH 
clinicians or AHB. On 5-6-87, AHB received a letter from Minister of Health requesting a 
report on the Metro article, re cancer treatment at NWH. “On or about 8-6-87 I prepared a 
preliminary report to the Board and on the 10-6-87 a letter was forwarded to the Minister of 
Health”, i.e. on the same day the Minister announced the establishment of a committee of 
Inquiry and its terms of reference, i.e. just 6 days after the Metro Magazine accusations.
Why did so many women develop cancer? Part 2

Professor Bryder’s response to my letter (30 April 2010) confirmed previous expressions of concern about her ability to understand and communicate the medical science of the “unfortunate experiment”. In my letter I asked Professor Bryder to explain “why so many women with carcinoma in-situ (CIS) of the cervix at the National Women’s Hospital developed cancer”. Either she failed to understand the question or she deliberately avoided it, responding to a totally different question of her own construction, namely the outcome of women treated with invasive cervical cancer. The management of CIS and invasive cervical cancer are two totally different subjects.

Professor Bryder has raised a number of issues in her response to my earlier letter which I address below.

1. The 1974 natural history paper by Green was “seriously flawed” because Green retrospectively removed cases of women originally diagnosed with CIS from his study and who later developed invasive cancer.

2. The 1975 “Whitewash Committee” arose because of concerns regarding the welfare of the women with CIS who were now developing invasive cancer. The Committee failed to stop the experiment, focusing on the interrelationships between the doctors involved.

3. Professor Bryder states that Dr Overton had no intention to publish his revisionist perspective, yet Mrs Overton states (NZ Herald 8/05/10) that “they wouldn’t publish anything”.

4. While Bryder states she is not an advocate for any group, she has chosen to communicate with the Doctors’ Overton camp who have produced the revisionist unpublished material she has so heavily relied upon.

5. Contrary to her assertion, Bryder has never approached me nor have I discussed the “unfortunate experiment” with her. At one of her lectures I asked her to explain why so many women with CIS developed cancer and she did not provide me with a satisfactory response.

Bryder’s credibility is undermined by her failure to accept there was an experiment, study—call it what you like—that many women with CIS of the cervix were untreated or inadequately treated, that many developed cancer some of whom died, and her inability to sustain her argument with scientific and historical rigour.

Professor Bryder is clearly unable to “assess the evidence” as she suggests. There can only be two possible answers – either she does not understand her subject or she is guilty of deliberate obfuscation.

In the bitterness and rancour shown by those bent on supporting Bryder’s view, the welfare of the women has been forgotten. My role as a doctor is to be an advocate for women (not the medical profession), to protect their welfare and prevent such a tragedy happening again.
Professor Ronald W Jones  
Clinical Professor of Obstetrics and Gynaecology, National Women’s Hospital  
Auckland

Linda Bryder response

To deal with Dr Jones’s points in the order he raised them:

1. A ‘retrospective’ analysis was exactly what Green was doing, and not just of his own patients but of all the patients in the hospital who had been diagnosed with CIS. The fact that histopathological diagnoses were highly subjective was constantly discussed in the medical press. One could argue that it was the 1984 paper which was flawed. For instance Figure 2 shows that Group 2 (those with a positive smear at 24 months) included a woman with a negative smear at 24 months who advanced to cancer – she should have been in Group 1. This clearly affected the percentages, and if this mistake is found in the small sample displayed in Figure 2, how many other mistakes were there in the analysis? Moreover, of the 29 placed into Group 2, 14 were diagnosed as ‘occult invasive’ (FIGO Stage 1b occult) which was a histopathological diagnosis and the clinical significance of this was still subject to debate.

2. The 1975 Committee was first suggested by Dr Bruce Faris because of disputes among senior staff relating to the management of women with CIS. He did not suggest any mismanagement at the time he made the suggestion, nor in the conclusion of the Inquiry.

3. Mrs Overton referred to the period following the 2008 ‘Cartwright Celebrations’ and more specifically following the publication of my book. The Seber and Mullins paper was written in 1990 and Mullins has confirmed that they had no intention to publish it. Mrs Overton was right to suggest, ‘They wouldn’t publish anything’ if she is referring to the New Zealand Herald. The Herald did not publish my response to journalist Chris Barton’s article on my book on 19 September 2009; I understand that other letters supporting my book were not published.

4. I did not rely on any ‘revisionist unpublished material’ but rather the extensive archives generated by the Cartwright Inquiry itself and the medical literature of the time. I had no communication with Dr Overton until after I had written the full manuscript of my book.

5. I discussed the general history of National Women’s Hospital with Dr Jones in his office in 2004. At that time I had not done any research on, or developed an interest in, the Cartwright Inquiry. However he sent me his article on the history of cytology and colposcopy following that meeting.

Like other staff members of the hospital he was interviewed by Jenny Carlyon. The interview took place on 2 December 2004, and I sent him the tapes and transcripts on 18 October 2006. Seven pages of the transcript are devoted to the ‘Unfortunate Experiment’. I subsequently returned the manuscripts and tapes to him at his request. Others at the seminar he refers to did consider my answers to Dr Jones satisfactory.
Dr Jones is concerned for the welfare of women, but which women is he referring to? Does he mean the eight women who had persistent positive smears and died of cancer in the period 1955 to 1976, or the hundreds of women who would have been given unnecessary treatment with concomitant side-effects when there was no certainty that the lives of those eight women could have been saved in any case?

Linda Bryder
Professor of History, The University of Auckland
Auckland

References:


3. National Women’s Hospital Medical Committee minutes, 23 June 1974, BAGC A638 21a, 16 October 1975, BAGC A638 41a, Archives New Zealand Auckland.

Why won’t defenders of the Cartwright Inquiry provide evidence to justify their use of the term ‘conventional treatment’ for carcinoma in situ?

Professor Ron Jones chides me for not having read the report of the Cartwright Inquiry.¹ The reason that I had not done so is straightforward: I have challenged Professor Paul in private and in public² to define and justify her use of the term ‘conventional treatment’ of carcinoma in situ. I felt sure that she would have pointed me to the relevant parts of the Cartwright Report if it contained the evidence I sought from her. The claim by Paul and others associated with the Cartwright Inquiry that a ‘conventional treatment’ existed in the late 1960s and early 1970s is of fundamental importance to judgements about the treatment of carcinoma in situ in Auckland during that era.

Prompted by Professor Jones’ criticism, I have now consulted the Cartwright Report in search of the evidence needed. To justify a claim that ‘conventional treatment’ was withheld from women with carcinoma in situ of the cervix, the first requirement is to show that there was international agreement on management of the condition. This requires research to find out whether such international consensus existed.

The relevant sections of the Cartwright Report are entitled 'Management of CIS before 1966: Outside New Zealand' (p 24–25) and ‘The International Debate’ (p 88–91). These short passages do not cite scientific articles, but simply quote the opinions of four witnesses (and one interviewee). In brief, there is no evidence in the Report, or in any of its Appendices, that any attempt was made to conduct the international review of management that might have justified use of the term ‘conventional management’.

Twenty years ago Professor Paul and the other medical advisers to the Inquiry could have sought systematically, analysed and published evidence of (i) international consistency of gynaecological practice in this respect; and (ii) empirical research evidence justifying such consistency. At the very least, they could have referred to Göran Larsson’s detailed review of treatment of pre-invasive and early invasive carcinoma of the cervix.³ This was published as a 40-page supplement in Acta Obstetricia et Gynecologica Scandinavica and contained a bibliography of over 400 articles.

The section on treatment opens with the following sentence: “There are marked differences in the treatment of preinvasive and early invasive carcinoma of the uterine cervix between different countries.” (p 114). The section on the treatment of preinvasive carcinoma refers to 216 publications from authors in the United States, Canada, New Zealand, Australia, England, the European mainland, and Scandinavia. Larsson’s review reveals dramatic differences of opinion about how the condition should be managed—ranging from extended radical hysterectomy to local electrocoagulation—and it refers to a debate about “whether conisation was overtreatment”.

¹ The Cartwright Inquiry was a New Zealand government inquiry into gynaecological practice in Auckland during the late 1960s and early 1970s.
Professor Paul began over two decades ago suggesting that ‘conventional treatment’
had been withheld from women with carcinoma in situ in Auckland. On 24 July 2008
she wrote to me in an email (with ‘New Zealand study of untreated cervical neoplasia’
in the subject line of the message), because she wanted to know about the nature of
my connection with Linda Bryder, who has recently published a book challenging the
conclusions reached by the Cartwright Inquiry. Professor Paul invited me to send her
my ‘views about what went on’.

In response I mentioned that I had held junior posts in gynaecology in the late 1960s
and early 1970s, and that I was not made aware of any generally accepted
‘conventional treatment’ for ‘carcinoma in situ’. Far from it: my specialist mentors
exhibited a range of views about what should be done for which abnormalities. I
concluded my email to Professor Paul with a challenge:

“What would you, as an experienced scientist with an obvious concern to involve women in
decisions about their care, have written about screening for and treatment of cervical
carcinoma in situ if you had been asked in the late 1960s and early 1970s to draft an evidence-
based, honest information sheet inviting women to attend for cervical screening?”

Professor Paul did not respond to this invitation. I ended my correspondence with her
when she suggested that I had an “understandable bias towards defending researchers
who are attacked”.

In my 12 September 2009 letter published in The Listener I challenged Professors
Charlotte Paul and Jo Manning to detail the elements of what they had referred to in
their articles as ‘conventional treatment’ in the 1960s and 1970s. I asked them to
cite the evidence upon which their definition of ‘conventional treatment’ was based,
and the extent of international concordance with their definition. Paul’s and
Manning’s 19 September response ignored my challenge.

Why do defenders of the Cartwright Inquiry like Professors Paul and Manning
continue to refer to ‘conventional treatment’ for carcinoma in situ without defining it?
Professor Paul fails again to define ‘conventional treatment’ in her 23-page chapter
‘Medicine in context’ in the recently published collection of Cartwright papers edited
by Professor Manning. Instead, she compounds her earlier failures to address this
challenge by repeating her references to ‘conventional treatment’ (p 124, 133 ) and
‘current best practice’ (p 138). Furthermore, she alludes to ‘international guidelines’
(p 120) without referencing them, and fails to draw attention to WHO guidelines that
refer to continuing uncertainties.

Aspects of Paul’s chapter that are perhaps even more disturbing are her intemperate
attacks (there and elsewhere) on the analysis published in Linda Bryder’s recently
published book. Because I had read a pre-publication draft of the book (albeit not for
the publisher), the publisher invited me to draft some text for the back cover. This is
what I wrote:

‘Professor Bryder has addressed a question that has remained inadequately investigated for
over a quarter of a century. What was the ‘generally accepted', ‘conventional' treatment for
abnormal cervical cytology which women in Auckland were allegedly denied in the late 1960s
and 1970s? Her thorough review of international practice at that time makes clear that there
was no generally accepted treatment, a fact that reflected the haphazard way in which
screening for cancer of the cervix had been introduced and evaluated.’
Professor Bryder’s detractors have failed to acknowledge that she has done research that should have been fundamental to the Cartwright Inquiry. By contrast with the medical advisers to the Cartwright Inquiry, Bryder devotes 24 pages (Chapter 3) of her book to placing practice in Auckland in an international context, including a paragraph summarising Larsson’s findings.

The chaotic way in which screening for cervical cancer had been introduced was one of the reasons that Archie Cochrane awarded ‘the wooden spoon’ to obstetrics and gynaecology for being the medical speciality that had been most negligent in obtaining reliable research evidence to inform its policies and clinical practice - among other things, for its “determined refusal to allow ‘Pap smears’ to be randomised, with disastrous results for the whole world”.

Bryder puts it very well in the opening paragraph of the conclusion of her Chapter 3 (p 55):

“What then was the conventional treatment’ that the patients at National Women’s Hospital were apparently denied by Herb Green? According to Cartwright it was not hysterectomy, which had already been rejected throughout the world as a routine response to CIS in favour of cone biopsy or local excision by the 1960s. Yet many gynaecologists still believed that hysterectomy was the appropriate response to the problem, including star witness to the Inquiry Ralph Richart. A significant minority of gynaecologists was questioning the appropriateness of hysterectomy and cone biopsy, both of which were far from benign procedures. Kolstad might have queried Green's clinical decisions, but he was the first to admit that there were no clearcut answers. Jordan might also have been critical of Green's approach, but he did acknowledge the ‘dilemmas’ in deciding appropriate treatment for asymptomatic women when the treatment options themselves carried a ‘high morbidity’. Jeffcoate recommended cone biopsy only when smears repeatedly contained cells indicative of malignancy.”

I find it extraordinary that the world has had to wait for a historian to expose the sloppy job done in the past by epidemiologists in New Zealand. Bluster and unreferenced, ex cathedra references to ‘conventional treatment’ are simply not good enough. However hard they and others with vested interests in defending the methods used in the Cartwright Inquiry may try to brush aside this issue, an evidence-based defence of the use by Professor Paul and others of the term ‘conventional practice’ is fundamental to judgements about whether or not what happened at the National Women’s Hospital was a scandal.

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Acknowledgement: I am grateful to Linda Bryder for commenting on earlier drafts of this communication.

References:


A response to Ron Jones’ letter of 30 April 2010

I cannot leave Dr Jones’ commentary in the NZMJ (30 April 2010) to go unchallenged, as he was responding to my letter in the NZMJ (19 March 2010). In that letter I explained why the allegations that there was an ‘experiment’ at National Women’s Hospital (NWH) were false and I now wish to elaborate on my argument.

For 20 years, New Zealanders have been led to believe there were two groups of women at NWH managed for CIN, one treated and one untreated, the latter having much higher rates of cancer.

I reiterate my position; there never was an ‘Unfortunate Experiment’, in which ‘Frankenstein Doctors’, ‘withholding treatment’, led women ‘like lambs to the slaughter’ as NWH treatment has been described.

I quote again the Cartwright Inquiry:

“...the 1984 McIndoe Paper distinguishes between two groups, and 22% of those whose abnormalities were untreated developed invasive cancer.”

The 131 women who were retrospectively allocated to group two by McIndoe/Jones in 1984 had had 228 major treatments (initial cone biopsy 88, hysterectomy 33, or later management with 78 cone biopsies or 29 hysterectomies).

How can this be misconstrued as no treatment?

Dr Jones appears now to be attempting to move the discussion away from ‘treated vs untreated patients’ to ‘adequate vs inadequate treatment’.

Dr Jones refers to ‘inadequacy of treatment’ yet even the McIndoe Paper categorically states:

“The 131 patients continued to produce abnormal cytology, irrespective of initial management.”

Does Dr Jones now acknowledge that New Zealand was misled: that Cartwright ‘got it wrong’...it was not ‘untreated patients’ but ‘treated patients with different outcomes’?

That is not how it was portrayed and that is a very different story.

To quote Jones’ own reference, Soutter:

“After the first year following treatment for CIN, the rate of invasive disease remained about 56 per 100,000 women years, until at least 20 years after treatment.”

and;

“ There was no statistically significant difference in the incidence of invasive recurrence between those series in which women were treated with hysterectomy and those in which one of the local, conservative methods of treatment was used.”

In other words, despite ‘adequate treatment’, including hysterectomy, there is still a failure rate, that appears to continue for years.
Dr Jones then asks why so many women got cancer, quoting 1:20 for his paper, but 1:200 for the rest of the World. Was treatment therefore ‘inadequate’, as judged by the high rates for invasion in the McIndoe/Jones Paper?

When results differ markedly from other comparable studies, by a factor of 10 in this case, one should confirm if the figures quoted and calculations are correct, and not a source of error.

Were the McIndoe/Jones figures correct? I believe they are not reliable.

A review of all cases of CIS seen at NWH from 1955–1986 was undertaken by M. Jamieson and A. Macintosh, (senior cancer specialists at NWH) and accepted by the Cartwright Inquiry (Source: Dr G Collison, NWH, Auckland, NZ, A Position Paper, July 1988, referenced in Cartwright Report).

This review found 1222 cases from 1955–1976 and 1815 from 1977–1986. Thirty-two patients were identified as having invasive genital tract disease following persistent CIS of cervix and eight of those patients had died of genital tract cancer by the end of 1986.

Astonishingly, these results were simply ignored by the Inquiry...Yet they were not rejected as incorrect.

The results do however give a very different set of statistics. In fact, only 32 cancers over 30 years in 3037 patients. This gives cancer rates of 1% which are comparable to other studies. For example, Kolstad treated 1121 women with CIN of whom 12 developed invasive cancer. (about 1%).

Soutter, also noted that the groups containing a high proportion of women with CIN 3 had a higher rate of invasive recurrence, than those with lesser grades. Jamieson and Macintosh, like McIndoe/Jones, were analysing data from women who had been diagnosed with CIN 3, as did Kolstad, but many other studies included all CIN cases, so one would expect better figures in those groups.

In the 1960s and 70s there was a change in the management of CIN, and at NWH and other places there were heated debates as to how ‘conservative’ one could be. Learning curves of clinical management involve positive and negative outcomes. In this case unnecessary hysterectomy vs invasive disease, as treatment changed from obligatory hysterectomy in the 1950s to the more conservative approach now used.

I do not hold with the Cartwright conclusion that the doctors at NWH withheld treatments as an experiment, which as recently as 2009 was described in the NZ Health Research Council’s Ethical Notes...as ‘similar to Nazi experiments’, and which resulted in excessive cancers.

I believe Dr Green was determined not to do unnecessary radical surgery. Dr McIndoe believed in more invasive treatment; both were doing their best to avoid women getting invasive cancer—and importantly both were driven by a desire to do the best for their patients.

Dr Helen Overton
Vejle Hospital
Denmark
References:

A particular relationship

Since Professor Linda Bryder published her controversial book *A History of the 'Unfortunate Experiment' at National Women's Hospital* attacks on her have been persistent and personal. Whatever one’s views, this approach has done little to enhance the discourse and has detracted significantly from the tenor of the debate.

She has been accused by the University of Otago vice-chancellor Professor David Skegg of either misunderstanding the science or of deliberate obfuscation (*NZ Herald*, September 2009). One might accept that this is all part of vigorous academic intercourse, but for his close colleague Professor Charlotte Paul to imply (without a whit of solid, specific evidence) in *The Cartwright Papers* that Professor Bryder had developed a “particular relationship” with two senior gynaecologists, such that they may have influenced her in some presumably clandestine way, is entirely different. If in fact this term has some other more obscure interpretation, then perhaps she could enlighten us as to what this is. This of course elevates the debate to another level and clearly implies that Professor Bryder is not only stupid and or a liar, but incapable of independent study.

Whatever their opinions in this debate, the manner in which these two outstanding clinicians (Professor Colin Mantell and Dr Tony Baird) have been portrayed is, in my view, unfortunate. To use such a ploy as a vehicle to attack Professor Bryder is not what one would expect from the leaders of an academic department but reminds one of a chapter from *The Famous Five*. For a vice-chancellor (irrespective of his or her own views) to be associated with or supportive of such an approach would, I think, be unprecedented and I hope would be seen by his peers, at the very least, inappropriate.

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Discussion of Morris and Jewell’s editorial ‘Array of hope for high-resolution genetic screening services in New Zealand’

Further to the editorial titled above, I am disappointed the authors (Morris and Jewell) made no effort to contact diagnostic referral centres directly and accurately determine the current level of service provision of microarray technology within New Zealand prior to publication.

The Cyto-Molecular Genetic Services Laboratory, Wellington Hospital, implemented aCGH in mid 2008, gaining IANZ accreditation in May 2009 and we wish to acknowledge the support of Agilent Technologies and Pacific Laboratory Products Ltd during the validation phase and Capital and Coast District Health Board’s vision in investing in microarray technology.

Service promotion has been widespread and demand is rapidly increasing. Diagnostic testing commenced in May 2009 and is currently available to tertiary specialists including paediatric services in consultation with Clinical Genetics. This technology is increasingly cost effective, and has largely replaced the stepwise targeted approach of multiple genetic tests in patients with unclear aetiologies.

If readers would like to learn more about our aCGH services, please contact genetic.services@ccdhb.org.nz

Clive Felix
Team Leader
Genetic Services Laboratory
Wellington Hospital

Response by Morris and Jewell

We are pleased that the Wellington laboratory believes that it is now offering a full service. This is a significant achievement for the timeframe, and considering assay complexity, necessary assessment of an expanding choice of commercially available array platforms, requirement for independent validation of assay results and not least, proper evaluation of their diagnostic impact. We were led to believe otherwise about the extent of the Wellington service by their array CGH representative who spoke at the Asia Pacific Human Genetics User Summit (May 13, 2010), which unfortunately Mr Felix was unable to attend.

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Managing your student loan

The study *Taking the pulse: medical student workforce intentions and the impact of debt* published in the 16 July 2010 issue of the NZMJ concluded that student loan debt is a major factor in a significant proportion of medical graduates choosing to leave New Zealand and work overseas.

Inland Revenue is aware medical students on average end up with a large student loan. But we also know that medical graduates with an average student loan leaving balance, and who take advantage of the voluntary workforce bonding policy, are forecast to repay their loan in about four years. The majority of borrowers are meeting their obligations.

A student loan doesn’t have to rule a borrower’s life. The key is to make sure you understand all the options available.

If you make extra repayments of $500 or more during the tax year (1 April – 31 March) you will generally qualify for the voluntary repayment bonus. An amount equalling ten percent of your voluntary repayments will be credited to your loan account after the end of the tax year. A large number of borrowers—many based overseas—are taking advantage of the bonus to help them repay their loan. See what difference voluntary repayments could make to your loan by trying out our student loan repayment calculator ([http://www.ird.govt.nz/calculators/tool-name/tools-s/calculator-sl-repayments.html?id=righttabs](http://www.ird.govt.nz/calculators/tool-name/tools-s/calculator-sl-repayments.html?id=righttabs)).

Our website ([http://www.ird.govt.nz](http://www.ird.govt.nz)) has more information, including eligibility criteria, about the voluntary repayment bonus ([http://www.ird.govt.nz/studentloans/payments/voluntary/bonus/](http://www.ird.govt.nz/studentloans/payments/voluntary/bonus/)). While you are there register online so you can view your loan balance and communicate with us by secure email. This is a great tool if you are going overseas as it makes it easy to stay in touch.

If you do go overseas you need to let Inland Revenue know before you go and give us a contact address for while you are away. For the first three years you are away you are entitled to a repayment holiday, although interest still accumulates on your loan. Given the average size of a medical student’s loan we strongly recommend making repayments during this period, otherwise interest will see the loan balance grow quickly. Making repayments is also simple and convenient if you use your credit or debit card ([http://www.ird.govt.nz/news-updates/like-to-know-pay-credit-debit-card.html](http://www.ird.govt.nz/news-updates/like-to-know-pay-credit-debit-card.html)).

We realise student loans may not be a priority when you’re enjoying your time overseas. But please don’t ignore your obligations. Student loan arrears can restrict your lifestyle choices when you return to New Zealand.

If you’re having difficulty repaying your loan, contact us sooner rather than later. Together we can work out a solution that fits your circumstances. For example, you may qualify for an instalment arrangement so you can pay your arrears over a period of time.

We’ll do everything we can to help you get back on track.

Charles Ronaldson
Group Manager Assistance
Inland Revenue

Reference:

Emeritus Professor John Parr, who died aged 87, was a pioneering ophthalmologist, a skilled gardener and a true son of Otago. He was also active in community affairs, in particular through Rotary and the Otago Peninsula Trust.

Born in Roxburgh, where his father was headmaster of the local school, he grew up with a love of Central Otago.

He tramped the hills and worked on farms during his student days. When the family moved to Dunedin, he attended Otago Boys’ High School and then the University of Otago.

He had hoped to be a farmer but family finances made this impossible. His love of the land was not diminished and when he began university studies he was still undecided whether he would become a doctor or an agricultural scientist. Medicine won.

John Parr had it in mind to return to Roxburgh and take over the practice of a family friend, but his academic results were so outstanding that specialisation beckoned. Again he faced a dilemma, unsure whether to embrace neurosurgery or ophthalmology. He decided on the latter discipline and in 1949, armed with the New Zealand Travelling Scholarship in Medicine, set off for London. He worked at Moorfields Eye Hospital and by 1952 had completed a Fellowship in Ophthalmology.

Back in New Zealand, he spent a short time in private practice but in 1961 was appointed senior ophthalmologist at Dunedin Hospital and a senior lecturer at the Medical School. In 1968 he was appointed Associate Professor (his family enjoyed referring to him as ‘Aspro’) and in 1977 was elevated to a personal chair in ophthalmology.

The academic department he set up in the Medical School became renowned for its quality. Prof Parr revisited the undergraduate training programme and created a postgraduate programme. For his revolutionary efforts he needed a textbook. None was available, so he wrote his own. It has moved through several editions and is still in use at medical schools in many countries.

Nothing but the best would satisfy Prof Parr. He was a demanding teacher who expected students to perform to the highest standards of which they were capable. He was also active on various hospital and medical school committees where he was known for his precision and organising ability. He and his friend neurosurgeon Richard Robinson fought so hard for improvements to facilities and teaching standards that one medical dean christened them “the Terrible Twins”.

For almost four decades John Parr was at the forefront of his chosen profession and the scientific foundation he had has inspired ophthalmology in this part of the world.
In 2008 he was given a Distinguished Service Award by the Royal Australian and New Zealand College of Ophthalmologists.

After retirement, he returned for a time to his beloved Central Otago, leaving behind a splendid garden in Macandrew Bay and moving to Ettrick. There he took over (and modified and improved) a distinctive garden designed by Graham Miller, who had worked at Glenfalloch. Prof Parr had a great love of alpine plants and became known to nurserymen throughout New Zealand as he sought rare plants. He also imported seeds from overseas.

Before the move to Ettrick, Prof Parr spent years in the Rotary Club of Dunedin where he was responsible for setting up a committee which focused on city affairs. During his presidential year in 1966–67 the club produced a hardcover book called Dunedin: Friendly City of the South, much of it written by him. When it went on sale 12,000 copies were sold in short order. The Dunedin City Council later took over distribution and used the book for promotional purposes.

As a member of the Roxburgh Rotary Club, Prof Parr was again active in community projects. He was especially interested in the Roxburgh District Medical Trust, giving much time to the challenge of maintaining good health services in the area.

He helped and encouraged Dunedin Jaycees to set up the Otago Peninsula Trust and would have been its first chairman had he not been so heavily involved in Rotary. When eventually he did chair the trust, he brought his enthusiasm and innovative skills to the task. For several years he served as chairman of the Glenfalloch Garden Committee, introducing new species and developing previously neglected areas.

To the end of his life, Prof Parr was a reader, even though a cruel fate reduced the quality of his eyesight. He subscribed to scientific journals and balanced his diet with the Guardian Weekly, a publication which suited his liberal and questing mind.

He was married twice. Diana, his first wife, he met at Moorfields where she was a nursing sister in charge of the private patients’ ward. A devoted partner, she shared his enthusiasm for gardening and walking for more than 40 years until her death. For the last 14 years of his life he was married to Margaret Swann, a former deputy matron at Dunedin Hospital. She was a great companion, who made it possible for him to remain at home even though his health was failing.

Prof Parr was an outstanding doctor and scientist, a plants man and botanist of great ability, a citizen who contributed much, always without fuss, and a loyal friend.

He is survived by Margaret and his daughter Alison, a former broadcaster and now an oral historian.

The obituary was written by Gordon Parry of Dunedin and was originally published in the 14 November 2009 edition of the Otago Daily Times. We thank them for the reprint permission.
Irwin Bruce (Bill) Faris

QSO; 1918 – 2009

Bill Faris was an important figure in the service for women in Auckland during the latter part of the 20th Century.

He was born in Dunedin on 10 December 1918 and, after his secondary education at the New Plymouth Boys High School from 1930 to 1935, he was awarded a Taranaki Travelling Scholarship which he used to go to the University of Otago in 1936.

He graduated MB ChB in 1941 and gained general registration in March 1942. After graduation he joined the New Zealand Army then transferred to the New Zealand Air Force, he went overseas to serve in the New Hebrides (now Vanuatu) and was in an air force plane that went down into the sea near a remote island.

With the others, Bill survived for 4 days before being rescued by a Catalina Flying Boat.

After serving as a registrar in obstetrics and gynaecology in Dunedin Public Hospital he was awarded a Doris Gordon travelling scholarship which took him to Crown Street Women's Hospital in Sydney, followed by postgraduate study in the United Kingdom.

He gained the MRCOG in 1949 and the Edinburgh FRCS in 1950. In the UK he met Gabrielle, another New Zealander, whom he married in Wellington in 1952. They had two daughters and four sons, none of whom went into medicine.

Returning to New Zealand he went into general practice in Takapuna, in partnership with his father and, about 2 years later was appointed to a fulltime position at what was then Cornwall Hospital, later to become National Women's Hospital, participating in clinical work, teaching and research. He was appointed to the part-time staff in 1955 and built up a private practice in central Auckland. He continued to study, gaining the FRACS in 1961, the same year as he was awarded the FRCOG. Bill was a foundation member of the RNZCOG and retired from practice in 1990.

The positions he held included Head of one of the clinical teams at National Women's and from 1963 to 1983 was medical superintendent at St Helen’s Hospital, which provided excellent experience and training in maternity care. He was a member of the New Zealand Council of the RCOG between 1965 and 1969. He was an elected member of the Auckland Hospital Board, serving as deputy chairman during the late 1970s. For his services to medicine Bill was awarded the Queens Service Order (QSO) in 1977.
He enjoyed skiing and spending time on his small farm in South Auckland but his retirement was marred by censure from the Medical Council in the wake of the Report of the Committee of Inquiry into allegations concerning the treatment of cervical cancer and into other related matters.

Bill Faris was a member of the sub-committee that was established in 1975 at National Women’s Hospital to review the care of women with cervical dysplasia and, subsequently, was found guilty of “conduct unbecoming a medical practitioner” in July 1995—20 years later. The relatively minor nature of the breach is demonstrated by the modest penalty of a fine of $500. Despite this, Bill continued to deny that he was in any way responsible, pointing out that the programme had stopped 2 years before he and a colleague were asked to read certain selected cases.

The Medical Council relied heavily on the report of the Committee of Inquiry, itself flawed and inaccurate in many ways in the opinion of this writer; the censure of Bill Faris was unfair and yet could not be challenged. The sub-committee ended the observational study conducted by Associate Professor Herb Green and the recent publication by historian Linda Bryder called “A History of the Unfortunate Experiment at National Women’s Hospital” has contributed reality and a balance. This includes the comment “The Cartwright Inquiry was not just about the research of one individual and his approach to female patients, it was the combination of a clash of ideologies and approaches to medicine, with women’s bodies having become highly politicised.”

It is a shame that Bill Faris did not live long enough to see this book, nor to have his fine contribution to obstetrics and gynaecology fully recognised.

Prepared by MAH (Tony) Baird with assistance from Bill’s sister, Felicity Tompkins.


This book was initiated to gather together the papers presented at a 2008 conference Twenty years after the Cartwright Report: What have we learned? and much of that material is presented here.

However, with the 2009 publication of a book by an Auckland historian, Prof Linda Bryder, putting forth a different version of the science, proposing a feminist conspiracy, and asserting that the Cartwright Report got the wrong answer, The Cartwright Papers took on the additional task of critiquing this work.

The rebuttal of Bryder’s revised history is formidable. The mistakes, misunderstandings and mischief in the scientific material and the failure of process in her research are laid bare.

Many of the assertions have been dealt with before; in the Inquiry, the Report, and the NZMJ in the past. However there is much else of interest in The Cartwright Papers.

The Cartwright Report was released in 1988 and was the result of a six-month judicial inquiry into the research of Dr Herbert Green at National Women’s Hospital. It found that Green had been experimenting on women without their consent by withholding treatment for carcinoma-in-situ of the cervix, and that his research had led to significant harm.

The Report sparked a cascade of change in the health system and debate about the practice of medicine in New Zealand, which was accompanied by widespread discomfort and demoralisation in the profession, and considerable resistance from some quarters.

As well as documentation of the events by those who were present, this book provides thoughtful reflections on the big issues that were raised by the Inquiry and its aftermath. Following the Foreword by Professor Sir David Skegg, Joanna Manning provides an introduction and then a succinct summary of the Cartwright Report.

The first set of essays includes Clare Matheson’s personal story as one of Herbert Green’s patients, Sandra Coney’s description of the initial investigation that prompted the Inquiry and the real role of feminism, and Ron Jones’ account of being a true insider at National Women’s and the inability of his profession to say “I’m sorry”.

Then Professor Charlotte Paul, who was a medical adviser to the Inquiry, discusses the scientific nature of Green’s study and the subsequent research that confirms the substantial harm that was done to the women involved.
The second set of essays comprises three responses to Linda Bryder’s book by Professor Barbara Brookes, a medical historian at the University of Otago, Charlotte Paul, and Sandra Coney.

In the final part, the essays focus on the lessons learned. The Cartwright Report changed the way we conduct medical research and also the way we deliver health care. An intense focus on patients’ rights and informed consent highlighted the central roles of communication and partnership with patients when making health care decisions. The shift in health professionals’ attitudes that has followed may have been largely unspoken but has been far-reaching and has been cemented in place with structural changes.

Ron Paterson, the former Health and Disability Commissioner, and Joanna Manning, both experts in medical law, discuss the New Zealand Code of Patient’s Rights that was developed after the Inquiry. Jan Crosthwaite responds to “Could it happen again?” from the perspective of an ethicist, and Alastair Campbell, Voo Teck Chuan and Jacqueline Chin discuss the international implications of the Cartwright legacy.

This is a fascinating book. Partly because of the light it sheds on the revised version of events that has attracted recent media attention. Mostly because it is a very engaging account of an important period of recent New Zealand history, of complex scientific and ethical issues, and of rapid change in the power politics of medicine in this country. This account is provided by a diverse group of intelligent and thoughtful critical thinkers, most of whom have been involved in this story since its beginnings.

Professor Jennie Connor
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