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This Issue in the Journal

Under-use of secondary prevention medication in acute coronary syndrome patients treated with in-hospital coronary artery bypass graft surgery
Khang L Looi, Kok L Chow, Jen L Looi, Mildred Lee, Sue Halliday, Harvey White, Chris Ellis

We reviewed all patients admitted to Auckland City Hospital coronary care unit with a heart attack (n=901) over a 13-month period in 2006 to 2007. Of various treatments, 129 patients received a coronary artery bypass operation. These patients are at very high risk of future heart problems, but certain medicines can significantly improve their prognosis, if used long-term. We reviewed these patients 3 years later and found a disappointing use of these medicines. The percentage of patients who were *not* taking these medicines were aspirin (17%), statins (28%), beta-blockers (38%), ACE-Inhibitors or angiotensin receptor blockers (57%). Clearly, greater efforts to optimise the use of these 'secondary preventative' medicines are needed in our health system.

The incidence of atrial fibrillation and the use of warfarin in Northland, New Zealand stroke patients
Angela Bang, Nicole M McGrath

Atrial fibrillation (AF) is an abnormal heart rhythm that can predispose to stroke by causing cardiac blood clots that can then travel to the brain. For patients with AF, the risk of stroke can be significantly decreased by anticoagulation with warfarin. In our study, approximately one-quarter of Northland stroke patients admitted between January and September 2010 were in AF but only one-third were prescribed warfarin. Of those stroke patients on warfarin, only 1/12 was on a therapeutic dose of warfarin. The main reason for no warfarin was patients’ wishes.

Surgical radiofrequency ablation for atrial fibrillation: the Christchurch, New Zealand experience
Frith Coolbear, Ian G Crozier

The long-term results following surgery for the common heart rhythm problem atrial fibrillation as an adjunct to other cardiac surgery at Christchurch Hospital are reported. A total of 44 patients underwent this operation between 2 July 2001 and 28 January 2009. In the short-term the surgery was successful in most patients. However with time the rhythm problem recurred in many patients.
Pulmonary vein ablation for atrial fibrillation: the Christchurch, New Zealand experience
Matthew Daly, Iain Melton, Ian G Crozier

Atrial fibrillation is New Zealand’s most common heart rhythm disorder. The upper chambers of the heart, or atria, are subjected to rapid, chaotic electrical currents and can not pump in a coordinated fashion. This may produce intolerable symptoms in some people. Research has shown that these abnormal currents originate from the pulmonary veins. Since 2001 we have endeavoured to cure patients who have failed other approaches by cauterising these veins, thus electrically disconnecting or isolating them from the heart. We found that although this is an invasive and technically challenging approach it can be effective, provided potential candidates are carefully selected.

Prediction of cardiac rhythm 1 year following cardioversion for atrial fibrillation
Amjad K Hamid, A Mark Richards, Ian G Crozier, John G Lainchbury, Iain Melton, Paul G Bridgman, Suetonia C Palmer, Chris M Frampton, M Gary Nicholls

Atrial fibrillation is the most common cardiac arrhythmia. There are two different strategies when treating this condition: restoring normal rhythm or controlling the heart’s ventricular rate. Cardioversion, a procedure done to restore normal rhythm, has an initial success rate of 80-90%. Most of the patients with initial successful outcome will revert back to atrial fibrillation (only one third of those who had successful cardioversion initially will remain in normal sinus rhythm a year later). A combination of clinical characteristics will help accurately identify those patients most likely to enjoy sustained return to normal rhythm after cardioversion.

Early cardiac morbidity of rheumatic fever in children in New Zealand
Olwen Gilbert, Nigel Wilson, Kirsten Finucane

This study reviewed children who were admitted to the heart ward of Starship Children’s Hospital for rheumatic fever over 2 years. There were 36 children and 49 separate admissions to hospital. 25 children required cardiac surgery. The cost was $1.9 million dollars. In other countries, rheumatic fever has been prevented.

Are we meeting cardiovascular risk targets 3 years after acute coronary syndrome? An evaluation in West Auckland, New Zealand
John A Ford, Jocelyn Bell, Colin Edwards

Our study looked at over 100 patients who were admitted to hospital 3 years ago with a heart attack or severe angina. We wanted to see if the patients were reaching target to prevent another heart attack. We have found that blood pressure, cholesterol and medication prescribing was generally good after 3 years. However there is room for improvement in terms of weight, diabetic control and prescription of a drug called an ACE inhibitor.
Non-adherence to medication and cardiovascular risk

Ralph A H Stewart, Andrew Kerr

In this issue of the *New Zealand Medical Journal*, Khang Looi and colleagues report on adherence to preventive medications during long term follow-up after coronary artery bypass surgery. These patients have severe multi-vessel atherosclerotic disease and a much higher risk of future cardiovascular events compared to individuals with risk factors alone and therefore a greater potential to benefit from interventions which reduce cardiovascular risk.

Smoking cessation, a healthy diet and regular exercise are important, but reliance on lifestyle interventions alone is not sufficient to stop disease progression. Preventive medications which include statins, blood pressure lowering medication, and anti-platelet drugs have been clearly demonstrated to reduce cardiovascular events in large clinical trials and are usually well tolerated.

In combination these preventive medications are estimated to reduce cardiovascular risk by 60 to 80%. For this reason clinical practice guidelines recommend these preventive medications are prescribed to all patients with established atherosclerotic disease. However Looi and colleagues reported that after an average of nearly three years following coronary artery bypass surgery only 83% of patients were still taking aspirin, 82% a statin, 47% an angiotensin converting enzyme inhibitor (ACE inhibitor) or angiotensin II receptor blocker (ARB) and 62% a beta-blocker. Adherence may have been even lower in the 14% of patients with no follow-up.

The problem of poor adherence to preventive medications is widespread, and adherence rates in Looi’s study are comparable or better than others. In another recent New Zealand study 40% of patients were not taking statins one year after hospital admission for acute myocardial infarction. The Prospective Urban Rural Epidemiological (PURE) study, evaluated adherence to preventive medications in patients with a history of myocardial infarction or stroke from 628 communities in high, middle and low income countries. In the high income countries most similar to NZ only 62% who had a history of myocardial infarction or stroke were taking an anti-platelet, 40% a beta-blocker, 50% an ACE inhibitor or ARB and 66% a statin. About half of subjects were taking the evidence based recommended combination of a statin, an anti-platelet and a blood pressure medication.

In middle and lower income countries use of these preventive medications was much less. The economic resources of the country and health system explained ~60% of differences in medication use, while ~40% was explained by individual factors.

In persons with cardiovascular disease poor medication adherence has been associated with about twice the risk of death. Interestingly in clinical trials poor adherence to placebo is also associated with adverse outcomes. This is probably because non-adherence to one medication predicts non-adherence to others and possibly because
medication non-adherence is associated with other unfavourable health related behaviours such as smoking, a poor diet, lack of physical exercise, and failure to seek medical advice for cardiac symptoms. However even after considering these possible confounders, large clinical trials strongly suggest that failure to take proven cardiovascular preventive medications will substantially increase the risk of cardiovascular death, myocardial infarction and stroke.

The decision to take medications is ultimately the patients, but the relationship between patient, doctor and health system has a significant impact. In a Spanish study of anti-platelet treatment after coronary artery stenting, which is critical for preventing stent thrombosis, adherence was better for patients treated in university compared to private hospitals, and better when written information was provided to patients.\textsuperscript{11}

Failure to prescribe preventive medications at hospital discharge could be addressed by simple systems of care and continuous quality improvement programmes.\textsuperscript{12} The transition from secondary to primary care is a time when medications are often stopped, particularly if communication is poor. Inconsistent advice between health providers is likely to undermine the patient’s confidence in the value of treatment.

Better two-way communication such as with the proposed universal medical record may reduce this risk. The recent ability to access pharmacy dispensing information through the electronic medical record may alert clinicians early to medication non-adherence.\textsuperscript{13}

Adherence is worse when drug regimens are complex and require multiple daily dosing.\textsuperscript{14} Statins are often given in the evening and other medications in the morning, but modest theoretical advantages related to timing of medications may not outweigh the benefits of a simpler once daily regimen.

For both statins and blood pressure medications the benefits of treatment are related more to the overall risk than the level of LDL cholesterol or blood pressure.\textsuperscript{15,16} In clinical trials relative risk reductions with ACE inhibitors, ARB’s, beta-blockers and statins were similar whether blood pressure or LDL cholesterol respectively were increased or in the normal range before treatment.

Patients need to understand these treatments reduce risk even when cholesterol and blood pressure are not elevated. Higher doses of medication reduce risk more, although side effects may be more likely. Statins decrease cardiovascular risk in proportion to the reduction in LDL cholesterol, so atorvastatin 40 or 80 mg daily is usually a better choice than lower doses or less potent statins.\textsuperscript{15}

The need to take preventive medication may be difficult to grasp for many patients, because these drugs do not generally improve symptoms and benefits are based on statistical concepts of risk. As with many health related behaviours more immediate ‘rewards’ often have a greater impact on choices. Less certain future benefits are not clearly evident to an individual who may remain well, or have a vascular event, whether taking medication or not. Suspected side effects, even if minor or not definitely related to medication may be a reason to stop.

Education to ensure the patient has a good understanding of each medication and the reason it is recommended, and reinforcement over time may improve adherence.\textsuperscript{17} In general cardiovascular preventive medications are cheap and serious side effects
infrequent, but the ‘hassle’ of medication or related consultations may be an issue for some patients. ‘Health beliefs’ are likely to have a large influence on behaviours. More research is needed to understand the reasons many patients stop taking medication and the most effective strategies for improving long term adherence in different circumstances.\textsuperscript{18}

The ‘polypill’, which combines several medications in a single capsule is a promising strategy designed to both reduce costs and improve adherence, and is currently under evaluation in clinical trials.\textsuperscript{19,20} The ‘polypill’ has the advantage that the patient needs to take only one capsule each day. Individualising medications is more difficult, but it is also be less likely the patient or doctor will decide that one or more of the key preventive medications are not needed. Blister packs have been shown to improve adherence in elderly patients with complex regimens,\textsuperscript{21} and could potentially be more widely used.

New Zealand data on medication adherence post coronary artery bypass grafting was last published in 2003\textsuperscript{22} when performance was similar to that documented by Looi et al. For effective quality improvement more regular and less time intensive access to adherence data is needed. In New Zealand it is now possible to anonymously link the records of all patients who have cardiac procedures in the public sector to medication dispensing records.\textsuperscript{5}

The Northern Cardiac Network, which covers Auckland and Northland, has developed automated 3 monthly reporting of secondary prevention medication dispensing for all patients with a hospital diagnosis of cardiovascular disease, including specific diagnostic subgroups such as coronary artery bypass grafting. Dispensing data can be reported by age, gender, ethnicity, socioeconomic status, District Health Board and general practice group.

These reports can be used to inform quality improvement initiatives in both secondary and primary care. Research which links comprehensive national level data on morbidity and mortality to individual general practice patient management systems and secondary care registries will provide further insights on this challenging problem.

In conclusion, Looi and colleagues have highlighted that non-adherence to proven evidence-based preventive medication is common in patients with severe atherosclerotic disease. Increasing adherence to medication has the potential to both improve the future health of many New Zealanders, and to reduce the costs of medical care.

\textbf{Competing interests:} None.

\textbf{Author information:} Ralph A H Stewart, Andrew Kerr, Cardiologists, Green Lane Cardiovascular Service, Auckland City Hospital, and Cardiology Department, Middlemore Hospital, Auckland

\textbf{Correspondence:} Professor Ralph Stewart, Green Lane Cardiovascular Service, Auckland City Hospital, Private Bag 92024, Auckland 1030, New Zealand. Fax: +64 (0)9 6236422; email: RStewart@adhb.govt.nz
References:


Managing atrial fibrillation: the growing challenge

Nigel A Lever, Peter D Larsen

Atrial fibrillation (AF) is the most common sustained cardiac rhythm abnormality. Affecting 1–2% of the adult population, it is projected that the prevalence of AF will more than double in the next 50 years. While some patients with AF have no problems and are asymptomatic, many experience a significant reduction in quality of life.

Associated with AF is a fivefold increase in stroke risk, together with an increased risk of heart failure and death. It remains unproven that restoration and maintenance of sinus rhythm will make a significant difference to mortality. Symptom control and prevention of complications remain current goals of management.

The search for safe and effective anti-arrhythmic agents has delivered precious little to clinicians recently. Dronaderone held hopes but has failed to keep the promise of an alternate to amiodarone. There are no new pharmacologic agents for rhythm control that have survived through the testing phases in clinical trials.

Whilst the mechanisms that start AF are better understood, the processes that drive the progression to permanent AF are more complex. Developing the tools to define and deliver treatment to targets remains a challenge for those working in the field. It is clear that while “AF begets AF”, AF is a spectrum of disease and a manifestation of multiple processes. The interplay between these processes demands treatments from many different perspectives, not just rate or rhythm control.

This issue of NZMJ publishes papers that address particular facets of AF and its treatment from a New Zealand perspective.

Direct current (DC) cardioversion to restore sinus rhythm has been a mainstay of AF therapy, and continues to be recommended. However, it has long been established that there is a very high recurrence rate of AF following DC cardioversion.

In a series from Christchurch Hospital, Hamid et al describe the low rate of long-term success of DC cardioversion, with only 30% remaining in sinus rhythm at 1 year, although a higher success rate was seen in the smaller group undergoing immediate DC cardioversion. In the elective group, failure to maintain sinus rhythm was related to measures of atrial substrate modification (length of time in AF and atrial size), and also to failure of the first shock to terminate AF.

Given the low rate of success of DC cardioversion, the value of this treatment strategy in elective patients remains an open question. It is our view that cardioversion is probably under used in those who are symptomatic and might benefit most. Clearly both selection of appropriate patients and use of anti-arrhythmic agents are essential when contemplating cardioversion.

Ablation to isolate triggers that constantly irritate the atrium is the cornerstone of current interventional treatment be it surgical or catheter based. The principle is to create an electrical barrier or “fire break” to contain ectopic triggers and exclude them...
from exciting the atrium. This usually involves electrically isolating the pulmonary veins. Less well understood is where and how to treat substrate for those whose disease is more advanced and long standing. In this scenario there is considerable remodelling and fibrosis that involves the body of the atrium not just the trigger sites.1,9

Surgical ablation for AF during valve repair or replacement is anecdotally an increasingly frequently employed treatment. All New Zealand cardiac centres perform this work, but results and volumes have remained unclear in part because there is limited published data. Confounding any results will be issues of defining AF burden, the impact of comorbid disease (such of mitral regurgitation severity) and definitions of success.

The paper by Coolbear and Crozier5 provides useful information about patients who undergo such procedures. This is a complex, heterogeneous group of patients, and overall the level of success, defined as persistent survival free from AF, was disappointing. The study suggests that patients with paroxysmal AF may be the most appropriate candidates for this approach, but the clear message is that careful discussions should be held prior to surgery about suitability for AF ablation work in this patient group.

One of the problems for surgical ablation is the inability to test the electrical barriers made by the ablation lines whilst the heart is not beating. Freezing or burning areas may induce acute changes but do not necessarily provide permanent electrical block, an essential requirement for effective treatment. The “cut and sew” Maze procedures seem to have the best chance of maintaining sinus rhythm, but the various challenges of this approach rightly prevent many surgeons from undertaking this procedure.

Catheter ablation is an attractive method to provide isolation since it is less invasive and has a proven method for testing electrical disconnection. However, it still carries important risks, has a certain failure rate and may require multiple procedures particularly for more chronic or long standing cases. Nonetheless, the technology and approach has evolved considerably over the past decade.

The report from Daly et al is the first on outcomes of this treatment in the New Zealand context.6 The 5-year AF-free survival rates of 74% for paroxysmal AF, and 56% for persistent AF achieved by this group compare well with international figures9. Like overseas centers, a significant number required repeat procedures to achieve these results.

While the ability to achieve long-term success with catheter ablation strategies is encouraging, the authors stress that this therapy was only offered to a select group of patients with a significant symptom burden and no other structural heart disease or significant comorbidity. Careful patient selection again remains a key feature.

Anticoagulation remains an important component of AF management. In the past the limited options of antiplatelet therapy or anticoagulation was problematic, particularly keeping warfarin treated patients within a narrow therapeutic range. In this context, work from Bang and McGrath, in a retrospective review of stroke/TIA presentations to Whangarei Hospital, is of note.4 They reported that AF was a potential cause in 24% of cases. However, only 31% of patients with an AF history were on anticoagulant therapy, and only one patient was in the therapeutic range. Whilst this
may not be of surprise to many, it is an important reminder to us to consider more carefully who and how to provide thromboembolic prophylaxis.

There has been considerable interest in the introduction of the direct thrombin inhibitors dabigatran, \(^{10}\) apixaban\(^ {11}\) and rivaroxaban\(^ {12}\) as potential replacements for warfarin. Of these dabigatran has recently been released in New Zealand, although quite where dabigatran fits in is yet to be seen. Whilst an attractive feature is avoiding INR testing, the side effect profile and difficulties reversing anticoagulation with bleeding problems means that this agent needs to be treated with some caution if we are to translate a positive clinical trial result into mainstream clinical practice.

Where are we going with AF treatment in New Zealand? Clearly more attention needs to be paid to this important condition, and we congratulate the editors and authors of the papers published in this edition of the *Journal* for addressing important clinical issues. Improved awareness of AF is one factor but the ability to offer appropriate treatments equally important.

Support for interventional procedures is required, with data prospectively collected about outcomes. Improved access to new anticoagulant agents is welcomed but better use of warfarin also needs to be encouraged. Local guidelines for AF management are long overdue for revision, which will hopefully be addressed in the near future. These need to provide clear, practical and current guidance in a New Zealand context. For now, we have to exercise caution about the efficacy and safety of treatments that are part of the management of AF, certainly not a cure!

**Competing interests:** None.

**Author information:** Nigel A Lever, Senior Lecturer, Department of Medicine, University of Auckland and Green Lane Cardiovascular Service, Auckland City Hospital, Auckland; Peter D Larsen, Associate Professor, Department of Surgery and Anaesthesia, University of Otago, Wellington

**Correspondence:** Nigel A Lever, , Department of Medicine, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand. Email: NLever@adhb.govt.nz

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Rheumatic fever in New Zealand: from perennial failure to successful eradication

Norman Sharpe

This Journal has published numerous papers on the topic of rheumatic fever in New Zealand over decades. Despite considerable interest and the efforts of many champions, this “Third World disease” persists, now almost exclusively in Pacific and Māori children.

The past decade has shown no suggestion of a decline, but rather an increasing trend in clusters of both acute rheumatic fever registrations and hospital admissions.\(^1,2\) Rheumatic fever is highly associated with other close contact infectious diseases in children which are related to social deprivation, child poverty and overcrowding. The costs of rheumatic fever for individuals, families and whānau, communities and the health system are very high.

In this issue of the NZMJ, Gilbert and colleagues report on a retrospective 2-year review between 2007 and 2009 of children with acute rheumatic fever and rheumatic heart disease admitted to the Starship Hospital.\(^3\) Of 36 children who had 49 admissions, all but 1 was of Māori or Pacific Island ethnicity. The average length of hospital stay was 23 days with the subset of children having surgery requiring 54 days in hospital on average. Total hospital costs over the period were estimated at $1.918 million.

The actual costs are of course very much higher than this and the cumulative human costs over the life-course are immeasurable. Broadly, rheumatic fever should be regarded as a key indicator of child health and how we value our children.\(^4\) Its persistence in New Zealand represents one aspect of our failure to achieve a fair society and health equity for Māori and Pacific Peoples. Last week we were reminded again of the reality of child poverty in New Zealand by the Child Poverty Action Group.\(^5\) This week as New Zealand participates in the UN High-Level Summit on Non-Communicable Diseases in New York, our transition as a “developed” nation can be questioned and certainly our dual disease burden must be acknowledged.

Recently we enjoyed the presence of Sir Michael Marmot in New Zealand. Sir Michael chaired the WHO Commission on the social determinants of health and also the UK review of social inequalities entitled “Fair Society, Healthy Lives”.\(^6\) The Marmot UK review provided a six-point action plan to achieve equity and spoke of “proportionate universalism”—ensuring improvement for all groups and classes but ensuring maximum improvement for the most disadvantaged. In New Zealand in the past decades we have seen disproportionate universalism with increasing relative inequalities—the rich becoming richer and the poor relatively poorer.

Rheumatic fever should be viewed in this context and success with eradication a marker for achievement of health equity and a fair start for all of our children. This aim is consistent with several of the most important actions to reduce health inequities.
in New Zealand which were agreed by participants following the recent Marmot Symposia. Most pertinent are those related to equitable and fair fiscal and social welfare policy, ill-health prevention that addresses risk factors contributing to health inequities, enhancing investment in early childhood and ensuring health services are equitable.

This year has seen a convergence of interests which allows guarded optimism that rheumatic fever can indeed be eradicated in the foreseeable future and this shameful and intolerable situation brought to an end. Following a recent budget allocation, the profile of rheumatic fever has been raised and a new Ministry of Health national rheumatic fever work programme has been commenced across targeted populations.

Key elements include development and delivery of clinical support tools, communications and health promotion to communities at increased risk; provision of support services (clinical facilitator, kaiāwhina and sore throat services in very high-risk communities); rheumatic fever monitoring and surveillance; interagency work to support the health sector in reducing rheumatic fever. This programme will build upon and help sustain the excellent progress already being made in some high risk areas championed effectively by local health professionals in primary care. Work in Northland, South Auckland, Waikato, Bay of Plenty, Tairawhiti, Flaxmere and Porirua provide outstanding local examples of these highly creditable efforts.

It is critical that we now take this opportunity to plan for the long haul and set quality standards that will ensure sustainability and success beyond the life of the programme; also to span the vagaries of political cycles. Whilst the final primordial cure will rest with improvements in the upstream determinants of health (e.g. eradication of child poverty, improvements in housing and overcrowding), the immediate priority is effective treatment of Group A streptococcal pharyngitis in high-risk settings and elimination of the “biofilm” of GAS carriage, the prevalence of which is 10% or higher in some communities.

Preventive efforts through community and school-based clinics need underpinning with heightened community awareness. Sore throats do matter! A consistent feature of success with eradication of rheumatic fever even in developing countries has been access to primary care without financial barriers. Models of care in the primary care arena need to be flexible with open access at all hours. An aggressive approach to sore throat management is needed in high-risk communities. Health professionals need to “get with the guidelines”.

Eradiation of rheumatic fever is a priority and a responsibility to be shared amongst health professionals, various agencies and providers. It will require leadership, cohesion, collaboration and a sustained quality effort. It is a challenging test for us and failure is not an option.

**Competing interests:** None.

**Author information:** Norman Sharpe, Medical Director, Heart Foundation, Auckland

**Correspondence:** Norman Sharpe, Medical Director, Heart Foundation, Auckland, PO Box 17-160, Greenlane, Auckland, New Zealand. Fax: +64 (0)9 5719190; email: NormanS@heartfoundation.org.nz
References:


Under-use of secondary prevention medication in acute coronary syndrome patients treated with in-hospital coronary artery bypass graft surgery

Khang L Looi, Kok L Chow, Jen L Looi, Mildred Lee, Sue Halliday, Harvey White, Chris Ellis

Abstract

Background Acute coronary syndrome (ACS) patients treated with inpatient coronary artery bypass graft (CABG) surgery are at significant risk for future Major Adverse Cardiovascular events (MACE). The use of evidence-based medications (aspirin, statins, beta-blockers and angiotensin converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBS)) can reduce MACE in these patients.

Methods We used a prospective database of all patients admitted to the Green Lane Cardiovascular Service, Coronary Care Unit (CCU) at Auckland City Hospital (ACH). We contacted patients General Practitioners for current patient data including MACE, which was supplemented by using the hospital patient records.

Results From 1/6/2006 to 31/7/2007, 901 patients presented with an ACS; of these 129 received inpatient CABG. 2 patients died before hospital discharge. At a median follow up time of 2.9 [IQR 2.7–3.3] years, 109 (86%) patients were traced and their medication assessed. Only 90 (83%) patients remained on aspirin, 78 (72%) on statins, 67 (62%) on beta-blockers and 47 (43%) on ACE inhibitors/ARBS. From the total of 127 patients discharged from hospital, there were a total of 18 MACE (6.2%/year): 3 unstable angina, 4 non-ST elevation myocardial infarction (NSTEMI), 6 congestive heart failure (CHF) and 5 deaths.

Conclusion Suboptimal use of secondary prevention drugs in high risk ACS patients treated with urgent CABG surgery may contribute to subsequent adverse events. Greater efforts to optimise the use of these medications are needed to improve outcomes.

Cardiovascular disease remains the commonest cause of death in New Zealand, being responsible for 10,480 (37%) of the 28,601 total deaths in 2007. The prognostic benefit for the use of secondary prevention medication is well documented in coronary artery disease (CAD) patients including those who have undergone revascularisation with percutaneous coronary intervention (PCI) and coronary artery bypass graft (CABG) surgery.

Previous assessment of young patients <40 years of age from Green Lane Hospital, Auckland who received either PCI or CABG have demonstrated poor secondary prevention at follow up in 1994–1996. We examined the modern day use of evidence-based medications (aspirin, statins, beta-blockers and angiotensin converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs)) in acute coronary syndrome (ACS) patients treated with inpatient CABG surgery. We assessed patients both at discharge and again at 3 years of follow-up in 2010.
Methods

We used a prospective database of all patients admitted to the Coronary Care Unit (CCU) at Auckland City Hospital (ACH) from 1 June 2006 until 31 May 2007. Being a tertiary referral centre, patients were transferred from Northland, Waitemata and South Auckland District Health Boards (DHB) and we have included these patients as part of our analysis. We determined the patient ACS presentation as unstable angina pectoris (UAP), ST elevation myocardial infarction (STEMI) or non ST elevation myocardial infarction (NSTEMI) based on their clinical presentation.

Patients’ preoperative coronary angiograms were classified either as two-vessel disease, three-vessel disease, left main stem disease or left main stem with one-vessel, two-vessel or three-vessel disease. No patients with single vessel disease underwent CABG surgery. Left ventricular systolic function for these patients was assessed either angiographically or with transthoracic echocardiography. The ejection fraction (EF) was classified into EF>50% (normal), EF 35–50% (mild-moderate impairment), EF 20–34% (moderate-severe impairment) or EF<20% (very severe impairment).

Patients’ demographic details, past history, risk factors and medication use were based on clinical staff entry on the admission clerking and discharge documents. At review from 7th December 2009 to 10th March 2010; we contacted patients’ General Practitioners (GP) for patients’ current medications and any major adverse cardiovascular events (MACE). MACE was defined as cardiovascular deaths, myocardial infarction or congestive heart failure. We supplemented this information with data from the patient hospital records for all patients, including for patients who could not be contacted.

Statistical analysis—Descriptive statistics of continuous variables were expressed as mean ± standard deviation (SD), and median with inter quartile range for not normally distributed variables. Categorical variables were expressed as frequency and percentage. The Chi-squared test was used to test significance between categorical variables. The unadjusted survival was estimated for cardiac death and MACE using the Kaplan-Meier method. Survival was measured from the date of admission and censored at the time of death or MACE. Data analysis was performed using SAS statistical package, version 9.1.3 (SAS Institute, Cary, NC).

Results

There were 1580 CCU admissions from 1474 patients of whom 901 patients presented with an ACS. 129 patients admitted with an ACS required inpatient CABG (Table 1). Twenty-eight (22%) of patients presented with a STEMI, 64 (50%) a NSTEMI and 37 (29%) with UAP. Patients requiring inpatient CABG surgery had severe coronary disease, with 52 (40%) having left ventricular systolic impairment (Table 2).

At hospital admission with an ACS, 22 (17%) patients previously known to have coronary artery disease had suboptimal use of secondary prevention medications (Table 3). CABG patients at discharge had better use of medications, although these were not ideal: aspirin (98%), statin (91%), beta-blockers (70%) and ACE inhibitors/ARBs (52%).
Table 1. Baseline characteristics of ACS patients who received inpatient CABG surgery (n=129)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n (%)</th>
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</thead>
<tbody>
<tr>
<td>Age (years), mean±SD</td>
<td>63.2±9.8</td>
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<td>Male</td>
<td>93 (72)</td>
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<td>Ethnicity</td>
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<td>Maori</td>
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<tr>
<td>Hypertension†</td>
<td>68 (53)</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>40 (31)</td>
</tr>
<tr>
<td>Dyslipidaemia†</td>
<td>65 (50)</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>23 (18)</td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>41 (32)</td>
</tr>
<tr>
<td>Non-smoker</td>
<td>65 (50)</td>
</tr>
<tr>
<td>Family history of premature IHD*</td>
<td>29 (23)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>21 (16)</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>9 (7)</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>Nil</td>
</tr>
</tbody>
</table>

† On treatment; * 1st degree relatives (Male < 55, female < 65 years).

Abbreviations: ACS: Acute coronary syndrome, CABG: Coronary artery bypass grafting.

Table 2. Coronary artery disease severity for ACS patients who received inpatient CABG (n=129)

<table>
<thead>
<tr>
<th>Severity of coronary disease</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-vessel disease</td>
<td>10 (8)</td>
</tr>
<tr>
<td>3-vessel disease</td>
<td>75 (58)</td>
</tr>
<tr>
<td>Left main stem with 1 or 2 vessel disease</td>
<td>22 (17)</td>
</tr>
<tr>
<td>Left main stem with 3 vessel disease</td>
<td>22 (17)</td>
</tr>
<tr>
<td>Left ventricular function*</td>
<td>n (%)</td>
</tr>
<tr>
<td>Normal EF &gt; 50</td>
<td>77 (60)</td>
</tr>
<tr>
<td>Mild-moderate impairment EF 35-50</td>
<td>33 (26)</td>
</tr>
<tr>
<td>Moderate-severe impairment EF 20-34</td>
<td>16 (12)</td>
</tr>
<tr>
<td>Very severe impairment EF &lt;20</td>
<td>3 (2)</td>
</tr>
</tbody>
</table>

Abbreviations: EF: Ejection Fraction; * calculated either by angiogram or echocardiogram; ACS: Acute Coronary Syndrome; CABG: Coronary artery bypass grafting.
Table 3. Secondary prevention medication use: 1) in patients known to have coronary artery disease on admission; 2) in patients post CABG surgery at discharge; 3) in patients at 3 years’ follow-up

<table>
<thead>
<tr>
<th>Medication</th>
<th>Admission (n=22)</th>
<th>Discharge (n=127)</th>
<th>3 years post CABG surgery (n=109)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin, n (%)</td>
<td>17 (77)</td>
<td>125 (98)</td>
<td>90 (83)</td>
</tr>
<tr>
<td>Statin, n (%)</td>
<td>14 (64)</td>
<td>115 (91)</td>
<td>78 (72)</td>
</tr>
<tr>
<td>Beta Blocker, n (%)</td>
<td>14 (64)</td>
<td>89 (70)</td>
<td>67 (62)</td>
</tr>
<tr>
<td>ACE-I/ARBs, n (%)</td>
<td>7 (32)</td>
<td>66 (52)</td>
<td>47 (43)</td>
</tr>
</tbody>
</table>

Abbreviations: CABG: Coronary artery bypass grafting

At a median follow-up time of 2.9 (IQR 2.7-3.3) years, 114 patients (of 127) could be traced (from GP and medical records) and were included in the analysis. There were 5 deaths from the 114 patients. Only 90 (83%) patients remained on aspirin, 78 (72%) on statin, 67 (62%) on beta-blockers and 47 (43%) on ACE inhibitors/ARBs (Table 3) and (Figure 1).

There were a total of 18 MACE at 3 year follow-up (3 UAP, 4 NSTEMI, 6 congestive heart failure (CHF) and 5 deaths (Figure 2).

Figure 1. Medication use in ACS patients receiving inpatient CABG surgery

NB: The denominator for the 3 groups is different.

Abbreviations: ACS: Acute coronary syndrome; CABG: Coronary artery bypass grafting; CAD: Coronary artery disease
Discussion

Patients presenting with an ACS requiring in hospital CABG are at very high risk of future adverse CVS events. We have demonstrated a disappointing use of secondary prevention medication 3 years after CABG surgery. 17% patients were not prescribed aspirin, 28% statins, 38% beta-blockers and 57% ACE inhibitors/ARBs.

It is well established that aspirin use minimises early and late graft occlusion post CABG surgery and improves long term cardiovascular outcomes.\(^3\),\(^7\) The current American Heart Association/American College of Cardiology (AHA/ACC) clinical guidelines recommend that aspirin be administered within 48 hours after CABG and should be continued indefinitely.\(^4\)

The Clopidogrel in Unstable angina to prevent Recurrent ischaemic Events (CURE) trial demonstrated that the combination of clopidogrel and aspirin was superior to aspirin alone for patients hospitalised with NSTEMI ACS.\(^8\) At this time there is

Abbreviation: MACE: Major adverse cardiac events
insufficient evidence to recommend the routine use of postoperative clopidogrel following elective CABG. However, for patients who undergo urgent CABG after hospital admission with ACS, the current AHA/ACC guidelines recommend treatment with clopidogrel 75mg daily for at least 1 month and ideally for up to 1 year. Unfortunately, these guidelines are not always being followed, with reports documenting 7-20% of patients not taking aspirin after CABG. These results are very similar to our study; with 17% not prescribed aspirin and none treated with clopidogrel on discharge or at follow up 3 years later.

Clinical trials have shown that statins are beneficial in patients with IHD including those who have undergone CABG surgery. The post CABG trial showed that aggressive statin treatment led to a reduction both in cardiovascular events and the progression of atherosclerosis in native coronary arteries and the saphenous vein grafts.

Left ventricular end-systolic volume has been shown to be an important factor in predicting the long term survival in patients with impaired left ventricular function undergoing CABG surgery. Both ACE inhibitors/ARBs and beta-blockers are effective in preventing LV remodelling and are considered useful secondary preventative measures for patients who have undergone revascularisation.

International and local guidelines support the use of these medications after an ACS. Unfortunately, evidence suggests that a significant gap exists in implementing secondary prevention medications in this group of patients. The benefits of achieving good secondary prevention for CAD patients have been known for many years. Numerous studies had demonstrated benefit. In 5353 patients assessed at 1 year after a myocardial infarction in Germany, total mortality was reduced by 74% in patients receiving “optimal medical therapy” (adj OR 0.26; 95%CI 0.18 to 0.38) versus patients receiving 1 or no drug.

The utilisation of secondary medical therapy after CABG surgery was evaluated in the PREVENT IV trial which was a multicenter, randomised, double-blind, placebo-controlled trial of 3,014 patients undergoing first CABG. The trial investigated whether the ex vivo treatment of vein grafts with the E2F transcription factor decoy, edifoligide, would prevent vein graft failure and improve clinical outcomes. The rates of use of aspirin, statin and beta-blockers were relatively high after discharge (96%, 84% and 89% respectively) but the use of ACE-inhibitors/ARBs was suboptimal (45%).

The rates reported were similar to our study population at discharge. However at 1 year following discharge, the rates of use of these medications were high in PREVENT IV, particularly for aspirin (95%) and statin (87%). The use for ACE-inhibitors/ARBs actually increased substantially, from 45% to 60%. The use of secondary prevention medications in our population was notably lower. This may reflect the differences between the tight follow-up in a randomised, controlled trial versus the community care in the “real world”, but indicates the medication rates which may be achievable with appropriate programmes.
Our results are similar to the ROSETTA-CABG Registry. This was a prospective, multicentre study examining the use of functional stress testing after CABG surgery in 16 centres across Belgium, Canada, France, Pakistan, the United Kingdom and the United States. As part of the protocol, data on medical therapy were prospectively collected at admission, discharge and at 12 months following CABG surgery. The use of aspirin was 92% and 87% of patients, respective at discharge and at 12 months. The use of statins were 57% at discharge but increased substantially to 75% at 12 months. The use of beta-blockers was 71% at discharge and 64% at 12 months. ACE-inhibitors use remained similar, from 33% at discharge to 38% at 12 months.

Another retrospective registry from England demonstrated disappointing secondary prevention medication use at discharge after coronary revascularisation. Among 98 CABG patients, 92% received aspirin at discharge, 70% beta-blockers, 73% statins and only 26% ACE inhibitors.

In New Zealand, disappointing secondary prevention has been demonstrated over many years. A systemic follow-up of 641 survivors of acute myocardial infarction at Auckland in 1998 for a median of 5.5 years showed that lipid management was suboptimal in this high risk population. The mean total cholesterol level (TC) was 5.7 +/- 1.1 mmol/L, and low density lipoprotein cholesterol (LDL) 3.8 +/- 0.9 mmol/L. Only 31% patients reached the (then) recommended goal for total cholesterol of below 5.2mmol/L, from the 1993 New Zealand Guidelines.

An audit of secondary prevention in coronary artery disease patients in South Auckland in 2001 included 147 patients. Statins were only used by 71% of the whole group. 37% failed to achieve the (then) New Zealand Heart Guidelines Group target TC of 3-5mmol/L at the time the audit was conducted. The study concluded that risk factor management remained suboptimal in a significant percentage of secondary prevention patients.

Our study and these previous studies have highlighted an important clinical issue: the episode of admission for revascularisation clearly represents an ideal opportunity to implement secondary prevention drug therapy in this group of patients who are at very high risk of further events. Neither the discharge prescription rates for aspirin, statins, beta-blockers and ACE inhibitors/ARBs were satisfactory nor the rates at follow up. There is unequivocal evidence that these patients derive prognostic benefit from these medications. It is clear that even in 2011 we need to try harder to have our patients taking the appropriate pills.

There are several limitations to our study. The number of patients admitted with a prior ACS (n=22) is small, and the estimates of this group are therefore less reliable. The follow up period was short at 3 years. General practitioners’ prescription of medications might not reflect the medications actually being taken by the patients. Those patients who have moved or did not attend their general practitioners clinic were counted as lost to follow-up; their medication use is unknown.

We also did not assess other variables such as smoking, dietary and lifestyle changes that may influence future cardiac events in the population. Further, we have no information on reasons for non-adherence to medication. Finally, our study may have patterns of care that are not representative of those in other centres in New Zealand.
In conclusion, we have assessed the use of secondary prevention medication in ACS patients undergoing inpatient CABG surgery. The prescription rate was disappointingly low among these very high risk patients at discharge and worse at follow up. Greater effort and strategies must be implemented to ensure greater adherence to appropriate secondary prevention medication to improve the long term outcome in this group of patients.

Competing interests: None.

Author information: Khang L Looi, Cardiology Registrar; Kok L Chow, Cardiology Registrar; Jen L Looi, Cardiology Registrar; Mildred Lee, Statistician; Sue Halliday, Senior Nurse, Coronary Care Unit; Harvey White, Clinical Directory of Coronary Care Unit; Chris Ellis, Cardiologist

Green Lane Cardiovascular Service, Auckland City Hospital, Auckland

Correspondence: Dr Chris Ellis, Cardiologist, Green Lane Cardiovascular Services, Level3, Auckland City Hospital, Grafton, Auckland 1023, New Zealand. Email: chrise@adhb.govt.nz

References:


The incidence of atrial fibrillation and the use of warfarin in Northland, New Zealand stroke patients

Angela Bang, Nicole M McGrath

Abstract

Aim To identify the number of Northland stroke patients with atrial fibrillation (AF) and to assess the effective use of warfarin anticoagulation in this group

Method A retrospective study of patients admitted with stroke or transient ischaemic attack (TIA) to Whangarei Hospital between 1 Jan 2010 and 1 Sept 2010.

Results Of 198 stroke/TIA patients identified, 47 (24%) had confirmed persistent or paroxysmal AF (PAF) or flutter. Only 12 (31%) patients with pre-existing PAF or AF were on warfarin and only 1 patient had an ischaemic stroke while in the therapeutic INR range of 2.0–3.0. The commonest reason cited for no anticoagulation was patients’ wishes.

Conclusion In our region, effective warfarin use for stroke prevention in AF patients is lower than recommended. This may be improved with increased awareness of efficacy and safety of warfarin and more thorough monitoring of INR.

Atrial fibrillation (AF) is a common arrhythmia and is a well established risk factor of ischaemic stroke. The recently released European Society of Cardiology Guidelines promote even broader use of anticoagulation. However recent studies show that warfarin is underutilised and patients are under anticoagulated. A study done at South Auckland’s Middlemore Hospital showed that only 47.4% of eligible patients were given warfarin. This study was undertaken to assess the appropriate use of warfarin in Northland patients with AF who presented to Whangarei Hospital with stroke or Transient Ischaemic Attack (TIA), to increase the awareness of warfarin use to prevent thromboembolic events.

Methods

This was a retrospective descriptive study involving all patients that were admitted with stroke or TIA between 1 Jan 2010 and 1 Sept 2010 to Whangarei Hospital. All eligible patients were identified by the Specialist Stroke Nurse and confirmed by the Stroke Physician. Those patients with atrial fibrillation or flutter were identified and their clinical data was collected using clinical notes. The arrhythmia was confirmed by review of electrocardiograms.

The data collected included age and sex of patients, type of stroke, whether the diagnosis of AF was pre-existing or not, CHA2DS2VASc score, whether or not they were on warfarin, INR on presentation and the reason for not being on warfarin i.e. contraindications. If the clinical notes did not provide the explanation for not being anticoagulated, the patient’s General Practitioner (GP) was contacted, if known.
Results

There were 198 patients that presented to Whangarei Hospital with stroke or TIA between 1 Jan 2010 and 1 Sept 2010. 47 of the patients (24%) had confirmed persistent or paroxysmal atrial fibrillation or flutter. The mean age of the AF patients was 74 years (44-95). There were 26 females and 21 males.

There were 2 patients with haemorrhagic strokes. Of the 45 ischaemic events, 30 were large strokes involving the anterior circulation, 5 were posterior circulation strokes, 2 were small vessel strokes and 8 were TIA. There were 8 newly diagnosed AF patients, 7 pre-existing paroxysmal AF (PAF) and 32 pre-existing AF.

Only 12 patients with pre-existing PAF or AF were on warfarin and only 1 patient had an ischaemic stroke while in the therapeutic INR range of 2.0-3.0 (2.3). The 2 patients who had haemorrhagic strokes were on warfarin with an INR of 2.0 and 1.1 respectively. The remaining 9 patients had subtherapeutic INR levels (1.1-1.6). All patients with pre-existing AF had CHA2DS2VASc of 2 or higher. 11 patients had potential contraindications for warfarin; patients wish, non-compliance, falls and previous significant bleeds.

The reasons for not being on warfarin for 39 patients with pre-existing PAF or AF are listed in Table 1.

Table 1. Reasons for no anticoagulation

<table>
<thead>
<tr>
<th>Reasons for no anticoagulation</th>
<th>Numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s wish</td>
<td>5</td>
</tr>
<tr>
<td>Infrequent episodes of atrial fibrillation</td>
<td>3</td>
</tr>
<tr>
<td>Falls</td>
<td>3</td>
</tr>
<tr>
<td>Not been started / Not been considered</td>
<td>3</td>
</tr>
<tr>
<td>Rhythm controlled</td>
<td>2</td>
</tr>
<tr>
<td>Age</td>
<td>2</td>
</tr>
<tr>
<td>GP unaware of paroxysmal atrial fibrillation</td>
<td>2</td>
</tr>
<tr>
<td>Non-significant bleeding episodes—e.g. epistaxis</td>
<td>2</td>
</tr>
<tr>
<td>Non-compliance</td>
<td>2</td>
</tr>
<tr>
<td>Significant bleed</td>
<td>1</td>
</tr>
<tr>
<td>Advised to stop as low risk</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
</tbody>
</table>

Discussion

This study was able to assess the effective use of anticoagulation in AF patients who presented with stroke or TIA in our region.

AF is a well known risk factor for ischaemic stroke. In the United States, AF has been shown to increase annual stroke risk by fivefold and accounts for 15% of all strokes.\(^2\) Of 198 events recorded at our centre, 47 (24%) had atrial fibrillation. We had 30 thromboembolic strokes involving large anterior circulation in 45 ischaemic events (67%). This is similar to previous observations that AF is associated with more severe stroke as a result of more proximal obstruction from embolism of large clots.\(^4,5\)
Warfarin has been associated with a 67% relative risk reduction and absolute annual risk reduction in all strokes of 2.7%.\textsuperscript{6} Compared to aspirin, warfarin was shown to reduce fatal or disabling stroke (ischaemic or haemorrhagic), intra cranial haemorrhage or clinically significant arterial embolism by 52% in The Birmingham Atrial Fibrillation Treatment of the Aged (BAFTA) study.\textsuperscript{7} There was no difference in the risk of major haemorrhage between warfarin and aspirin. Rate of bleeding has been recorded as low as 0.1-0.6% with use of anticoagulation for AF.

Despite the effectiveness of warfarin, only 18-55% of eligible patients have been reported to be anticoagulated.\textsuperscript{2,3,8–11} In this study, all of the 39 patients with pre-existing PAF or AF had a CHA\textsuperscript{2}DS\textsuperscript{2}-VASc score (Cardiac failure or dysfunction, Hypertension, Age >75(Doubled), Diabetes, Stroke(Doubled)-Vascular disease, Age 65–74 and Sex category (Female)) of 2 or higher, that is highly predictive of stroke risk in AF patients.\textsuperscript{1} 11 patients had potential contraindications for warfarin: patients wish, non compliance, falls and previous significant bleeds.

Excluding the patients with contraindications, 28 patients were eligible for warfarin. There were only 12 on warfarin (42%). 2 patients had haemorrhagic strokes while on warfarin. But of 10 patients with ischaemic strokes, only 1 patient had an INR in the therapeutic range. This is consistent with international data that most strokes occur when INR is sub therapeutic or when anticoagulation has been stopped.\textsuperscript{12,13}

The commonest reason for not being on warfarin in this study was patients’ wish not to be anticoagulated. In recent studies that investigate patients’ perception of warfarin and stroke,\textsuperscript{14–16} many preferred to accept the consequences of being prone to severe bleeding than the consequences of debilitating stroke.

Hing et al\textsuperscript{14} showed that 52% of patients would take warfarin for an absolute decrease in stroke risk of 1% over 2 years. Devereaux et al\textsuperscript{15} demonstrated that patients at high risk for AF placed more importance on avoidance of stroke and less importance on the avoidance of bleeding than did physicians who treat patients with AF.

In a study by Solomon et al,\textsuperscript{16} the majority of patients expressed the view that severe stroke is worse than death. With these results we may speculate that utilization of warfarin can be increased with improved patient education and patient-doctor communication.

Other common reasons for not being on warfarin were infrequent episodes of AF and rhythm controlled AF. Patients with paroxysmal AF have similar risk of ischaemic stroke as persistent AF.\textsuperscript{17} Interestingly up to 90% of episodes were found to be asymptomatic.\textsuperscript{18} Van Gelder et al\textsuperscript{12} demonstrated that at the end of 24 months of follow up with careful rhythm control treatment, only 39% had sinus rhythm. Furthermore Boos et al\textsuperscript{19} found only 25% of patients had sinus rhythm at 5years and there were similar thromboembolic events in those with rate and rhythm control.

There were a few limitations in our study. It was a small patient group and demographic and geographic data for this group may be different to the rest of New Zealand population. We were unable to obtain details of reasons against being on warfarin from patients themselves or how frequently patients were having falls. Some of our patients reside in rural areas where they have difficulty travelling to laboratories for frequent INR monitoring, therefore declining to receive warfarin.
However many of these reasons may have to be considered by a physician for each individual patient.

In summary, effective warfarin use for stroke prevention in Northland AF patients is lower than recommended. Underutilisation and undercoagulation of warfarin may be improved with increased awareness of efficacy and safety of warfarin and more thorough monitoring of INR.

Competing interests: None.

Author information: Nicole M McGrath, Physician; Angela Bang, Medical Registrar; Whangarei Hospital, Whangarei, Northland

Acknowledgement: We thank Liz Williams (Clinical Nurse Specialist – Stroke, Whangarei Hospital) for providing basic data.

Correspondence: Nicole M McGrath, Department of Medicine, Whangarei Hospital, Private Bag 9742, Whangarei, Northland, New Zealand. Fax: +64 (0)9 4304117; email: Nicole.mcgrath@northlanddhb.org.nz

References:
Surgical radiofrequency ablation for atrial fibrillation: the Christchurch, New Zealand experience

Frith Coolbear, Ian Crozier

Abstract

Aims To report the long-term results following surgical radiofrequency ablation (RFA) for atrial fibrillation as an adjunct to other cardiac surgery at Christchurch Hospital.

Methods A retrospective observational audit review of outcomes. The sample population included all patients identified as having undergone surgical RFA for atrial fibrillation at Christchurch Hospital, between the first procedure performed on 2 July 2001 and 28 January 2009.

Results A total of 44 patients underwent surgical RFA between 2 July 2001 and 28 January 2009. Postoperatively there were three deaths prior to discharge (7%). Pacemakers were required in four patients (9%), and two patients subsequently underwent catheter ablation for atrial arrhythmias. In the immediate postoperative period only three patients remained in atrial fibrillation. At last follow-up up to 102 months from surgery (45±29 months), 27 patients had developed persistent atrial fibrillation and four persistent atrial flutter. Persisting long-term benefit was seen in seven patients (18%, 7/38); five patients were in stable sinus rhythm, one had paroxysmal atrial fibrillation and one paroxysmal atrial flutter.

Conclusions Whilst the procedure was effectively acutely, the recurrence of atrial fibrillation was high and development of new atrial flutter common over long-term follow-up.

Treatment for atrial fibrillation and atrial flutter has traditionally focused on either rate control or rhythm control. Surgical ablation was first described by Cox in 1993 (the Maze procedure). This was performed in conjunction with other cardiac surgery with a successful outcome between 74% and 90% at 2–3 year follow-up. However, procedure times were long, and 6% of patients required postoperative pacing.

More recently, the creation of a similar line set using radiofrequency ablation (RFA) has been advocated for surgical ablation of atrial fibrillation during cardiac surgery for coronary disease or cardiac valve surgery. A number of studies have shown radiofrequency surgical ablation to have good success rates 6–15 months following surgery, with only one study reporting a longer term efficacy of 73% at 40 months.

This observational audit retrospectively reviews outcomes for patients who underwent surgical RFA for atrial fibrillation at Christchurch Hospital between 2001 and 2009, assessing in particular the long-term efficacy.
Methods

The sample population included all patients identified as having undergone surgical RFA for atrial fibrillation at Christchurch Hospital between the first procedure performed on 2 July 2001 and 28 January 2009. Potential patients were identified using the hospital clinical coding system; their respective clinical records were then accessed to confirm the nature of surgery and to extract relevant data.

Both electronic and hardcopy clinical records were reviewed for each patient in the sample. Notes were screened for information regarding the original operation; all electrocardiograms prior and subsequent to operation; and postoperative clinic and admission notes for comments regarding current heart rhythm. Operation date and cardiac surgery performed were noted for each patient. Patient gender, age at surgery, and relevant documented medical diagnoses, were also extracted. Preoperative left atrial dimension by echocardiography was also documented when available.

All available information pertaining to heart rhythm, from the time of operation to the present, was recorded. Electrocardiograms were interpreted by FC in the first instance, and over read by IC. Rhythm data for each patient were then grouped according to time since surgery. Results are primarily derived from patient electrocardiograms; where no electrocardiograms was available for a given timeframe, current rhythm as documented in clinical notes has been used.

All patients underwent RFA combined with surgery for coronary disease and or valvular heart disease. Initially operations were performed as described by Raman. Radiofrequency lesions were created using a multielectrode, temperature-controlled probe. Initially the flexible, 7-electrode, temperature-controlled unipolar Cobra probe (EP Technologies, Boston Scientific Corp, San Jose, Calif.) was used. Lesion creation parameters were set at 80°C to 85°C for 2 minutes to achieve transmural ablation.

RFA was performed endocardially in the left atrium in patients undergoing mitral valve surgery, and in some of the other patients. The RFA consisted of a linear line set along the roof of the left atrium, an encircling line set around right-sided pulmonary veins, an encircling line set around left-sided pulmonary veins with connecting line set to mitral valve annulus and a connecting line set to mitral annulus from lower right pulmonary vein orifice. In addition the left atrial appendage was over sewn in a linear fashion from within.

In some of the patients undergoing cardiac surgery other than mitral valve surgery, a comparable epicardial line set was performed consisting of a linear line set along the roof of the left atrium, an encircling line set around left-sided pulmonary veins, an encircling line set around right-sided pulmonary veins, and a connecting line set toward the atrioventricular groove. In addition the base of the left atrial appendage was ligated from the outside.

Right atrial epicardial line sets were also placed consisting of a linear line set from behind the sinoatrial node approximately along the direction of the crista terminalis and curving up to the atrioventricular groove, and a connecting line set from the inferior vena caval orifice to the right atrioventricular groove low on the body of the right atrium.

In 2005 the procedure was modified and the line pattern was adjusted to left atrial line sets combined with right and left atrial appendage ablation/ligation only, without other right atrial ablation line sets. Also at this time the ablation system was changed to the Medtronic cooled tip bipolar cardioblate pen.

Results

A total of 44 patients underwent RFA between 2 July 2001 and 28 January 2009. The mean age was 66 (range 30 to 82); 18 (41%) were female and 26 (59%) male. Preoperative atrial fibrillation was persistent in 33 and paroxysmal in 10 patients. Additional preoperative atrial flutter was present in four patients, this being the sole arrhythmia in one patient.

The surgery was mitral valve surgery in 17, mitral valve surgery combined with coronary artery grafting in six, aortic valve surgery in nine, aortic valve surgery combined with coronary grafting in one, and coronary artery grafting only in nine patients. One patient each had aortic and mitral valve combined surgery and atrial
septal defect closure. Patients were routinely given amiodarone postoperatively for 6 weeks.

Postoperatively there were three deaths prior to discharge (7%), and pacemakers were required in four patients (9%). Long-term follow-up data was obtained in all surviving patients, except three patients in whom data was limited to 4–6 weeks, but were subsequently lost to follow-up. Overall patients were followed up for 0 to 102 months (45±29 months, mean±SD). Two patients later underwent transcatheter ablation, one for right atrial typical and atypical atrial flutter, and one with repeat pulmonary vein isolation for atrial fibrillation.

**Atrial rhythm following surgical RFA**—Immediately following surgery three patients remained in atrial fibrillation. The remaining patients were in sinus rhythm, or were in paced rhythm without evidence of atrial fibrillation.

In the 38 patients with extended follow-up, 37 had one or more episodes of atrial arrhythmia; atrial flutter and fibrillation in 20 patients, atrial fibrillation only in 15 patients, and atrial flutter only in two patients. At 6 months 23 had developed persistent atrial fibrillation, six persistent atrial flutter, five were in sinus rhythm, and in four patients the rhythm could not be determined with certainty.

At last follow-up 27 patients had developed persistent atrial fibrillation and four persistent atrial flutter. Persisting long-term benefit was seen in seven patients (18%, 7/38); five patients were in stable sinus rhythm (including the patient who underwent catheter ablation for atrial flutter), one had infrequent paroxysmal atrial fibrillation, and one patient had infrequent paroxysmal atrial flutter (the patient who had subsequent catheter ablation for atrial fibrillation). None of the patients with persisting long-term benefit were on antiarrhythmic medications, and in three the preoperative atrial fibrillation was paroxysmal.

Preoperative left atrial dimension was not available for all patients. However it was available in the seven patients with long-term benefit. These patients were characterized by not having marked left atrial dilation preoperatively with a maximal left atrial dimension of 46 mm.

**Discussion**

In this report we document the long-term efficacy of adjuvant surgical RFA for atrial fibrillation during cardiac surgery, in a single centre with long-term follow-up. Like other series**5–10** we observed good initial efficacy. However with long-term follow-up the majority of patients did not maintain sinus rhythm, with only 18% of patients considered to have long-term benefit; five patients with stable sinus rhythm, and one patient each with infrequent paroxysmal atrial fibrillation and flutter.

Our results are in contrast to the series of Sie et al which reported an efficacy of 73% at 40 months.**10** We can only speculate as to the reasons for the poorer long-term efficacy in our series; possible causes include a longer follow-up period, and different patient populations and surgical techniques. Also in the Sie report success beyond 6 months was determined from clinic reports, rather than electrocardiograms, which may have overestimated the long-term maintenance of sinus rhythm.
The poorer efficacy in this series than with the Cox-Maze procedure,\textsuperscript{2,3} suggests that RFA does not replicate the effect of the cut and sew procedure, with likely recovery of tissue conduction. This was certainly observed in the two patients that subsequently underwent catheter ablation where recovery of conduction across the surgical RFA lines was observed.

Interestingly, a large proportion of patients, many of whom had no prior history of atrial flutter, were found to have at least one episode of atrial flutter (with or without atrial fibrillation also) postoperatively, suggesting the atrial flutter may have been a result of the operation. In a previous report of surgical RFA using a different line set, postoperative atrial flutter was observed in 96% of patients, due to incomplete ablation of atrial tissue and proarrhythmic gaps in the linesets.\textsuperscript{11} Therefore it is probable at least some of the postoperative atrial flutter in this series represent newly generated arrhythmia secondary to incomplete line formation from surgical RFA.

We also observed that long-term maintenance of sinus rhythm was more frequent in patients that had paroxysmal as opposed to persistent atrial fibrillation. Also patients that had long-term maintenance of sinus rhythm did not have marked left atrial dilation, but the absence of data on left atrial size in the whole group limits conclusions. The previous surgical studies did not address the predictive role of left atrial size, and only two studies included patients with paroxysmal atrial fibrillation.\textsuperscript{5,12} However in patients undergoing catheter ablation of atrial fibrillation these factors predict success.\textsuperscript{13,14}

On the other hand, the majority of patients developed recurrent persistent atrial fibrillation with long-term follow-up. This is likely due in part to the severity of their underlying cardiac disease. In addition, the poorer results that we observed with surgical RFA than previously seen with the cut and sew Cox-Maze procedure\textsuperscript{2,3} suggest that RFA does not reproduce its effects.

This is probably related to incomplete tissue ablation in the line sets as evidenced by the observation of new postoperative atrial flutter. An improvement in ablation technology that resulted in complete line sets would be likely to improve the long-term results of surgical RFA for atrial fibrillation.

\textbf{Study limitations}—This report is a retrospective chart-based review with all the inherent limitations of this approach. The duration of atrial fibrillation prior to surgery was not known. Left atrial dimension was not available on all patients. It would have been useful to determine if there was any association between duration of preoperative atrial arrhythmia and incidence of postoperative arrhythmia.

The sample size is small, and the available rhythm data incomplete with the possibility patients may have had undocumented arrhythmia. Similarly, potential confounding factors have not been addressed in the study; for example, the evolving surgical technique during the study period, other illness or cardiac events that may have precipitated atrial fibrillation or flutter.

The lack of patient perspective is also a limiting factor. Patients may have experienced atrial fibrillation or flutter that is unaccounted for in the clinical notes, and therefore not counted in the study.
More importantly, outcomes from the patient’s perspective are more likely to be centred on perceived frequency and severity of arrhythmia episodes pre- and postoperatively. If episodes of arrhythmia were felt to be less disruptive after the RFA procedure, than the procedure would likely be considered a success, regardless of how many episodes actually occurred, and vice versa.

To elucidate the potential relationships between patient and operative factors, further studies would be required. As always, the most compelling information would come from a prospective study, with a control group, and with follow-up rhythm recordings at regular intervals, over a period of years.

**Conclusions**

We report the long-term results following surgical RFA for atrial fibrillation as an adjunct to other cardiac surgery. Whilst the procedure was effective acutely, the recurrence of atrial fibrillation was high with long-term follow-up. Atrial flutter was commonly seen presumably in part due to the surgical RFA. At a mean of 45 months follow-up, 18% of patients had long-term benefit and were in either stable sinus rhythm, or had infrequent paroxysmal atrial flutter or fibrillation. Long-term benefit was associated with absence of marked left atrial dilation and persistent atrial fibrillation preoperatively.

**Competing interests:** None.

**Author information:** Frith Coolbear, Ian Crozier, Department of Cardiology, Christchurch Hospital, PO Box 4345, Christchurch

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**Correspondence:** Ian Crozier, Department of Cardiology, Christchurch Hospital, PO Box 4345, Christchurch, New Zealand. Email: Ian.Crozier@cdhb.govt.nz

**References:**


Pulmonary vein ablation for atrial fibrillation: the Christchurch, New Zealand experience

Matthew Daly, Iain Melton, Ian Crozier

Abstract
Aims To report the long-term results following percutaneous pulmonary vein ablation (PVA) for atrial fibrillation (AF) at Christchurch Hospital.

Methods A retrospective observational audit review of outcomes. The sample population included all patients identified as having undergone percutaneous radiofrequency ablation of multiple pulmonary veins at Christchurch Hospital, from the first procedure performed on 29 September 2001 until 15 December 2009.

Results A total of 187 patients underwent pulmonary vein ablation. The patient population was predominantly younger (mean age 51) and male (83%) with no important comorbidity. Following a single procedure only, the chance of remaining free of AF at 12 months was 0.74 for patients with paroxysmal AF (PAF) and 0.60 for patients with persistent AF (PsAF). 52 patients (28%) underwent a repeat procedure within 12 months of their index ablation owing to early recurrence of AF. 5-year survival free of clinical AF when analysed following these early repeat procedures, if required, was 0.74 and 0.56 for PAF and PsAF patients respectively. Complications occurred following 6% of procedures and were serious in 2.5%. New atrial flutter developed in 6% of patients.

Conclusions PVA is an effective treatment for AF, with better outcomes in patients with paroxysmal atrial fibrillation. However, as it carries a significant risk, we recommend that its application be reserved for patients with highly symptomatic, medication-refractory disease.

Atrial fibrillation (AF) is the most common cardiac arrhythmia and its incidence and prevalence have been increasing steadily.1–3 Rate control and anticoagulation is the appropriate treatment strategy for the majority of patients4. A number of rhythm-controlling medications are available for patients with intolerable arrhythmia-related symptoms.4–7 Unfortunately, some patients remain highly symptomatic despite such therapy.

The pulmonary veins (PV) are the main source of electrical ectopy that triggers and perpetuates paroxysmal AF.8–10 Endocardial ablation to electrically isolate the pulmonary veins is an effective treatment for AF but is invasive and technically challenging.

This observational audit retrospectively reviews outcomes for patients who underwent percutaneous multiple pulmonary vein ablation (PVA) for AF at Christchurch between 2001 and 2009, assessing long-term efficacy and complications.
Methods

The sample population includes all patients who underwent percutaneous PVA at Christchurch Hospital from our first procedure performed on 29 September 2001 until 15 December 2009. They were identified from the catheter laboratory log. The procedure report, electronic and hard copy of clinical records, correspondence from referring clinicians and patients were accessed for each patient. Relevant demographic data were collected including previous medical diagnoses and treatment. Electrocardiograms (ECG), echocardiograms and other cardiac imaging were reviewed. Operative details were noted.

All available information pertaining to heart rhythm from time of intervention to the present was recorded. Results are derived from a combination of patient ECGs and Holter monitor results, and rhythm as documented in clinical notes.

Procedure—All procedures were performed by a consultant electrophysiologist (IM or IC) with or without the assistance of an electrophysiology fellow.

Almost all patients received oral anticoagulation with warfarin for a minimum of 1 month prior to ablation, aiming for an international normalised ratio between 2.0 and 2.5. This was discontinued 3 to 5 days before their procedure. Transoesophageal echocardiography was only performed in selected patients with persistent AF and suboptimal anticoagulation to exclude the presence of left atrial thrombi.

Two patients required a general anaesthetic. In the remainder, PVA was performed under local anaesthesia and light sedation. Access was obtained through vascular sheaths in the femoral vein. Surface ECG and intracardiac electrograms were recorded continuously. A decapolar catheter was advanced into the coronary sinus for monitoring and pacing. The left atrium was entered through a patent foramen ovale if present; otherwise, a single trans-septal puncture was performed in the manner described by Brockenbrough.11

Heparin boluses were given intravenously, maintaining an activated coagulation time greater than 300 seconds. Initially, venous anatomy was documented by direct balloon pulmonary venography, but this has since been superseded by magnetic resonance imaging and currently multi-slice computerised tomography performed before the procedure. A circular mapping catheter was advanced into the PVs for monitoring and timing purposes and an ablating catheter advanced into the left atrium.

Extensive radiofrequency lesions were placed proximal to the ostia of the PVs. Initially, each PV was isolated individually. However, it subsequently became standard for ipsilateral veins to be isolated together with a wide circumferential approach in mid-2006.12 Fluoroscopy and electrophysiological signals were used for guidance, although these were later supplemented by the use of three dimensional (3D) mapping.

The Local Lisa (Medtronic, Minneapolis, MN, USA) platform was first employed in September 2004, and subsequently the Carto (Biosense Webster, Diamond Bar, CA, USA) and NavX (St Jude Medical, St Paul, MN, USA) systems became available. The radiofrequency current was delivered with safety cut-offs set by local temperature, power and delta impedance. Saline-irrigated-tip ablation catheters were routinely used from April 2005.

The end-point for each lesion was loss of local electrical signal, and the end-point for each line set was loss of PV signals and PV bidirectional electrical isolation.

Where appropriate, additional ablations were performed along a cavo-tricuspid isthmus line, left atrial roof line, or mitral isthmus line to treat other atrial tachycardias. Substrate ablation was routinely performed in patients with longstanding persistent atrial fibrillation.

Patients were hospitalised for 1 or 2 days following ablation. Limited echocardiography was performed in all patients to exclude immediate complications. Oral anticoagulation was reintroduced the evening after the procedure with a loading dose, and low molecular weight heparin was administered subcutaneously for 1 to 3 days.

All patients continued warfarin for a minimum of one month; subsequent use was tailored to each patient. In general, anticoagulation was discontinued in patients who appeared free of AF recurrence, had no other indication for anticoagulation, and had a low CHADS2 score.13

Repeat procedure—In patients requiring a repeat procedure, lesions and isolation were evaluated. Lasso signals and data from electro-anatomic mapping were used to identify gaps, deliver ablation and
assess ablation efficacy. If any other atrial tachycardia occurred, its mechanism was identified and targeted ablation treatment was delivered.

**Data analysis**—Kaplan-Meier analysis was used to determine the proportion of patients free from clinical AF after the initial procedure and after repeat procedures performed within the first 12 months, if necessary, for patients with PAF and PsAF. For the purposes of this analysis we considered repeat procedures performed beyond 12 months as an indication of late disease recurrence.

**Results**

A total of 187 patients underwent PVA between 29 September 2001 and 15 December 2009. By December 2010, 110 patients (58.8%) had undergone one procedure, 60 patients (32.1%) two procedures and 17 (9.1%) patients three or more procedures, giving a total of 284 PVA procedures. (Figure 1).

**Figure 1. Patient flow chart**

![Patient flow chart](image)

**Patient characteristics**—The mean age was 51 (range 24–71) and 83% were male. No patient was older than 75 years of age at the time of their first procedure. AF was paroxysmal in 134 (72%) and persistent (i.e. episodes lasting longer than 7 days or not
self-terminating) in 53 (28%). Average body mass index was 27kg.m$^{-2}$ (range 16.6–41.2).

Hypertension was present in 19%, past history of coronary disease in 2%, previous stroke in 2%, diabetes in 3%, and chronic obstructive airways disease in 6%. CHADS$_2$ index was $\leq$1 in 95%.

All patients had severely symptomatic AF and had failed at least one anti-arrhythmic medication. At the time of their procedure 28% remained on amiodarone, 39% on flecainide, 14% on sotalol, 13% on digoxin, 35% on a $\beta$-receptor blocker, 29% on a calcium channel blocker, 29% on an angiotensin converting enzyme (ACE) inhibitor, and 2% on a diuretic.

Echocardiography was performed on all patients. Left ventricular hypertrophy was noted in 15% of patients, mitral valve disease of moderate severity in 8%, and any regional left ventricular wall motion abnormality was present in 2%. The average left atrial diameter was 44mm (range 24–64). Left ventricular end diastolic diameter was greater than 55mm in 20% and ejection fraction was less than 40% in only 3% of patients.

Outcomes—The average procedure time per patient was 405 minutes (range 140–1170) for patients with paroxysmal AF (PAF) and 484 minutes (range 180–1070) with persistent AF (PsAF) for all procedures combined. The average radiation dose was 116Gy.cm$^2$ (14–510) and 174Gy.cm$^2$ (20–651) for PAF and PsAF respectively.

All four veins were successfully isolated by the end of the first procedure in 140 patients (75%). In the remaining 47, one or more veins were left untreated as they did not appear to be arrhythmogenic. Of these, 22 had long-term success but 25 had clinical recurrence of which 18 underwent repeat ablation.

Kaplan-Meier survival curves for freedom from clinical AF after the initial procedure are shown in Figure 2. The chance of remaining free of AF at 12 months after a single PVA was 0.74 and 0.60 for PAF and PsAF respectively, and 0.70 overall. At 5 years this fell to 0.50 and 0.41 for PAF and PsAF.

At least one repeat procedure was performed in 41% of patients. 52 patients (28%) underwent a repeat procedure within 12 months of their index ablation owing to early recurrence of AF.

As repeat ablation is frequently required in order to achieve long-term pulmonary vein isolation, separate survival curves were produced to determine the outcome for patients who had either had a single procedure or had undergone repeat ablation within 12 months of the index procedure (Figure 3). Following repeat ablation, if required, the probability of remaining free of clinical AF was 0.93 and 0.81 for PAF and PsAF respectively at one year, and at 5 years 0.74 and 0.56.

33 (18%) patients experienced late AF recurrence. Of these, 27 patients (82%) underwent a total of 33 repeat ablations, on average 28 months after their first procedure (range 13–85). Of those 27 patients, 22 (81%) remain free of AF with a mean follow up of 33 months (range 6–72). The chance of remaining free of AF at 5 years after the completion all procedures, irrespective of timing, was 0.95 and 0.73 for PAF and PsAF respectively.
There was a significant difference in log-rank test between the PAF and PsAF survival curves (p<0.001). At last follow up, 18% of patients remained on some form of rhythm-controlling medication to maintain freedom from AF.

Despite considerable changes in technique, there was no significant difference in log-rank test between the survival curves of the first 67 patients with PAF who underwent PVA compared with the last 67 patients.

Complications—There were no procedure-related deaths. Four patients required drainage of symptomatic pericardial effusion. Pulmonary vein stenosis was noted in four patients and led to symptoms in one. Air embolism leading to transient cardiac ischaemia occurred in two patients.

Complications occurring once include a stroke with full resolution, a seizure occurring in an individual with a prior history of seizure, an episode of flash pulmonary oedema, a femoral arteriovenous fistula requiring surgical repair, and a femoral haematoma requiring blood transfusion.

During an attempted transseptal puncture, a sheath was advanced through the posterior wall of the left atrium. This was removed in a cardiothoracic operating theatre with transoesophageal echocardiogram guidance, but self-sealed with no sequelae. During one PVA, a circular catheter became tangled in the mitral valve. It
was eventually freed, but the patient developed severe symptomatic mitral regurgitation. He underwent successful surgical repair. Symptomatic phrenic nerve palsy occurred in one patient, which resolved spontaneously after 12 months.

**Figure 3. Multiple procedure success – Kaplan-Meier event-free survival curve after the last catheter ablation attempt**

One patient who underwent apparently successful PVA but remained on flecainide long-term suffered a community ventricular fibrillation cardiac arrest and was unable to be resuscitated.

Overall, a minor or self-limiting complication was seen after approximately 3.5% of all procedures and a serious complication occurred in 2.5%, for a total complication rate of 6%. In addition, symptomatic atrial flutter developed after PVA in 6% of patients. Generally this required treatment by way of radiofrequency ablation, performed either at the time of redo PVA or as a separate procedure.

**Discussion**

Atrial fibrillation is the most common arrhythmia requiring treatment. Current treatments aimed at either rhythm- or rate-control have drawbacks including poor efficacy and inadequate symptom control. Pulmonary vein ablation offers the potential of “curative” treatment; however international experience documents only moderate success and a significant complication rate.
We report the outcomes of multiple PVA for AF in a single centre in New Zealand with long-term follow-up. Our long-term efficacy and complication rates are comparable to other series. However, the proportion of patients requiring repeat procedures is higher. At all repeat procedures, we observed recovery of tissue conduction across lines which previously had bidirectional block.

Tissue oedema from nearby ablation may lead to transient conduction block; this can give the appearance of a complete line set but leave a gap once the swelling resolves. Achieving a continuous set of permanent lesions remains a challenge despite steady improvements in catheter technology. The significant improvement in Kaplan-Meier survival curves after repeat procedures is likely to reflect achieving permanent electrical isolation of the pulmonary veins. Although most recurrences were observed in the months immediately after the first procedure, there was a slow but steady decline in the arrhythmia-free survival curve.

It is unclear whether there is progressive arrhythmia recurrence with time, or whether there is an eventual plateau. A recent series\textsuperscript{14} suggested an annual recurrence rate of 8.9\% following the last ablation attempt. This suggests a need for long-term surveillance and is relevant in the decision to discontinue anticoagulation after apparently successful ablation.

We noted a significant difference in the maintenance of sinus rhythm in patients with PAF compared with those with persistent or permanent disease. Emerging evidence points to the increasing proportional importance of ectopy and arrhythmogenic substrate outside the pulmonary veins in perpetuating AF in non-paroxysmal cases.\textsuperscript{15,16} This has led to advocacy of increasingly aggressive left atrial ablation line sets.\textsuperscript{17,18}

Our patient selection was mainly limited to those with highly symptomatic AF despite aggressive medical treatment, who, in general, had no structural heart disease and no other comorbidity. Overall, we found good long-term outcomes, particularly in patients with paroxysmal AF. The risk of complication, as well as the length and cost of these procedures means that PVA remains best reserved for patients with highly symptomatic AF who have failed medical treatment.

Study limitations—This report carries all the usual limitations associated with a chart-based retrospective review. As indicated above, this procedure has been evolving and the techniques and patient population changed significantly. For instance, the proportion of patients with PsAF has increased with time, and our ablation sets have become more extensive. Furthermore, there has been a steady stream of new tools and technologies, most notably 3D mapping packages.

Patients were not subjected to routine ambulatory ECG monitoring. This may lead to an underestimate of AF recurrence. However, as we only performed ablation on highly symptomatic individuals with a long history of AF we would expect them to be reliable in reporting ongoing symptoms. Furthermore, as this procedure is currently solely indicated for symptomatic relief, it is appropriate to consider treatment as having been successful in those with only intermittent asymptomatic AF recurrence. International consensus suggests it is reasonable to consider infrequent, well tolerated recurrence of AF as a successful outcome.
Conclusions

We report the outcome of percutaneous multiple pulmonary vein ablation for atrial fibrillation in a single centre in New Zealand with long-term follow-up. Although multiple procedures were required in a significant number of patients, PVA proved to be an effective treatment for highly symptomatic, medication-refractory atrial fibrillation.

Competing interests: None.

Author information: Matthew Daly, Iain Melton, Ian Crozier, Cardiologists, Department of Cardiology, Christchurch Hospital, Christchurch

Correspondence: Ian Crozier, Department of Cardiology, Christchurch Hospital, PO Box 4345, Christchurch, New Zealand. Email: Ian.Crozier@cdhb.govt.nz

References:

Prediction of cardiac rhythm 1 year following cardioversion for atrial fibrillation

Amjad K Hamid, A Mark Richards, Ian G Crozier, John G Lainchbury, Iain Melton, Paul G Bridgman, Suetonia C Palmer, Chris M Frampton, M Gary Nicholls

Abstract

Background There is little recent information regarding outcome and its determinants following cardioversion (CV) for atrial fibrillation (AF) or flutter. This study aims to help improve prediction of cardiac rhythm outcome following CV for AF.

Methods Cardiac rhythm at 6 weeks and 12 months was documented following elective (EC; n = 496) or immediate (IC; n = 52) cardioversion for AF or atrial flutter in a single referral centre.

Results: 65 and 58% of IC patients remained in sinus rhythm (SR) 6 weeks and 1 year after CV (respectively) compared with 43% and 30% in EC patients (P < 0.001). Independent positive predictors of SR 6 weeks after cardioversion included amiodarone therapy (OR 2.04 [1.28-3.33], P < 0.01) and atrial flutter (OR 1.85 [1.09-3.13], P < 0.05). Negative predictors included the need for >1 shock to achieve SR (OR 1.61 [1.12-2.37], P = 0.011) and arrhythmia duration, (OR 0.96 [0.95-0.97], P < 0.001). At 1 year, amiodarone, duration of arrhythmia and the need for >1 shock remained independent predictors of rhythm.

Conclusions The number of shocks required to achieve SR is a newly demonstrated independent predictor of rhythm outcome after elective CV.

Atrial fibrillation (AF) affects 0.4-1% of the general population. Prevalence increases with age to 8% in those over 80 years.1-3 AF is associated with increased risk of stroke, heart failure and all-cause mortality. The significant costs of management are driven largely by hospitalisation.1,4 Treatment consists of either ventricular rate control combined with anticoagulation, or attempts at restoring sinus rhythm (SR).

Robust randomised controlled trials suggest rate control is not inferior to attempted rhythm control regarding survival and morbidity, and is appropriate for many patients with recurrent or persistent AF.1,5,6 Similar findings were observed in patients with congestive heart failure.7 Accurate case selection for cardioversion (CV) is required to offer the best chance of sustained SR.

Whilst CV for AF has been used for over 40 years, follow-up periods have often been brief with reported rates of sustained SR of 44 to 69% at 4 weeks and 23 to 79% at 1 year.8-14 Factors predicting atrial fibrillation recurrence include duration of arrhythmia,9,10,13,14,16-19 type of arrhythmia (AF or flutter),14 echocardiographic indices,9,10,18,20 age,1,13,14,17 gender,1,12 underlying cardiac disease,5,11,12,17 frequency of paroxysmal AF and previous CV attempts,1,16,17 the use of rate-controlling or anti-arrhythmic drugs,11,17,18 functional status (NYHA Class),13,14,19 hypertension or
pulmonary disease, the initial energy used to achieve CV and restoration of SR with drug therapy. We report findings from a large consecutive cohort of patients with AF/flutter presenting over a 2-year period regarding maintenance of SR over 1 year, predictors of rhythm outcomes, and mortality and cardiovascular morbidity.

Methods

The study, approved by the Regional Ethics Committee, was carried out in Burwood and Christchurch Hospitals which serve a population of 481,431 (2001 population census). All patients with AF/flutter undergoing CV between December 2000 and December 2002 were included. Patients were categorised into those who underwent immediate CV within 72 hours from AF/flutter onset (IC) and elective CV after receiving warfarin for at least 4 weeks with an INR at 2 to 4 for 2 weeks prior to CV (EC). A transoesophageal echocardiogram was first performed on patients with AF duration of more than 48 hours, IC group, or they had not been on Warfarin long enough; EC group. Warfarin was continued for at least 6 weeks post CV. Demographics, known duration of arrhythmia, presenting symptoms, comorbid conditions, medications and the number of DC shocks and Joules delivered were recorded. In preparation for CV, all patients had a 12 lead electrocardiograph (ECG), routine blood tests and echocardiography according to American Society of Echocardiography Guidelines. Using a monophasic defibrillator, CV was performed following the intravenous administration of propofol and remifentanil or midazolam with the defibrillator paddles placed on the chest in the anterior and posterior positions. The standard energy selection was 200 Joules for the first shock followed, if required, by 2 shocks each at 360 Joules. CV was considered successful if the patient maintained SR for more than 20 minutes. Cardiac rhythm and medical status were recorded at 6 weeks and 1 year following CV.

Apart from 38 patients, all CV in the EC group were performed by one of the authors (AH). The primary end point of the study was cardiac rhythm at 6 weeks and 1 year. Secondary outcomes included total and cardiovascular mortality and cardiovascular morbidity as determined from the hospital electronic Patient Management System records.

Statistics—Data are shown as mean (± SD or SEM), median (interquartile range) when non-parametric, or frequency (%). Comparisons of categorical and continuous variables between the IC and EC groups were conducted using Chi-square and independent t-tests or Mann-Whitney U tests respectively. Univariable regression analysis was used to determine predictors of SR at 6 weeks and 1 year after cardioversion. Within the EC group only, in view of adequate sample size for robust results, the independent predictive power of atrial flutter, hypertension, medications, requirement for >1 shock, duration of AF/atrial flutter and LA diameter>40 mm to predict SR at 6 weeks and 1 year after EC was tested using multivariable logistic regression. P<0.05 indicated statistical significance. Analyses were conducted using SPSS version 13.

Results

Over 2 years, 53 patients underwent IC and 508 patients EC. One IC patient and 12 in the EC group were lost to follow up leaving 52 and 496 respectively. Patients in the IC group underwent CV while receiving low molecular weight heparin. None of the IC group had undergone prior CV. Seventy EC patients (15%) had one and 17 (4%) had two or more previous cardioversions.

Patient characteristics are shown in Table 1. Males presented with AF/atrial flutter at a younger age than females (statistically significant within EC group, P<0.001).
Table 1. Clinical, treatment & echocardiography data (mean ± SEM, median [interquartile range] or number [%])

<table>
<thead>
<tr>
<th>Variables</th>
<th>Immediate cardioversion (IC) n=52</th>
<th>Elective cardioversion (EC) n=496</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (male) *</td>
<td>56.78±14.7</td>
<td>66.3±11.7</td>
</tr>
<tr>
<td>Age (female)†</td>
<td>61.4±14.7</td>
<td>71.6±9.5</td>
</tr>
<tr>
<td>Male: Female</td>
<td>39:13</td>
<td>343:153</td>
</tr>
<tr>
<td><strong>Presentation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic*</td>
<td>0</td>
<td>111 (22%)</td>
</tr>
<tr>
<td>Palpitations *</td>
<td>38 (86%)</td>
<td>97 (20%)</td>
</tr>
<tr>
<td>Dyspnoea *</td>
<td>9 (17%)</td>
<td>180 (36%)</td>
</tr>
<tr>
<td>Dizziness</td>
<td>2 (4%)</td>
<td>7 (1%)</td>
</tr>
<tr>
<td>Fatigue‡</td>
<td>0</td>
<td>47 (10%)</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>0</td>
<td>23 (5%)</td>
</tr>
<tr>
<td>Myocardial Ischemia (EKG ± ↑TnT)</td>
<td>0</td>
<td>13 (3%)</td>
</tr>
<tr>
<td>Chest Pain †</td>
<td>11 (21%)</td>
<td>4 (1%)</td>
</tr>
<tr>
<td>Syncope</td>
<td>1 (2%)</td>
<td>1 (0.2%)</td>
</tr>
<tr>
<td>CVA/TIA</td>
<td>0</td>
<td>11 (2%)</td>
</tr>
<tr>
<td><strong>Arrhythmia Duration</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>2 hours-72 hours</td>
<td>4 days-3 years</td>
</tr>
<tr>
<td>Median *</td>
<td>14 hours</td>
<td>87 days</td>
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<tr>
<td><strong>Initial Rhythm‡</strong></td>
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<tr>
<td>Atrial Fibrillation</td>
<td>36 (69%)</td>
<td>409 (83%)</td>
</tr>
<tr>
<td>Atrial Flutter</td>
<td>16 (31%)</td>
<td>87 (18%)</td>
</tr>
<tr>
<td><strong>Comorbidities</strong></td>
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<tr>
<td>Diabetes Mellitus</td>
<td>2(4%)</td>
<td>56 (11%)</td>
</tr>
<tr>
<td>Hypertension *</td>
<td>13 (25%)</td>
<td>244 (49%)</td>
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<tr>
<td>Previous MI</td>
<td>7 (14%)</td>
<td>73 (15%)</td>
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<tr>
<td>Coronary Disease</td>
<td>23 (44%)</td>
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<tr>
<td>Airways Disease</td>
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<tr>
<td>Previous CVA/TIA</td>
<td>1 (2%)</td>
<td>16 (3%)</td>
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<tr>
<td>Valvular Disease</td>
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<td>107 (21%)</td>
</tr>
<tr>
<td>Thyroid Disease</td>
<td>2 (4%)</td>
<td>42 (9%)</td>
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<tr>
<td>**CHADS2 score ***</td>
<td></td>
<td></td>
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<tr>
<td>0</td>
<td>30 (58%)</td>
<td>105 (21%)</td>
</tr>
<tr>
<td>≥1</td>
<td>22 (42%)</td>
<td>391 (79%)</td>
</tr>
<tr>
<td><strong>Drug Therapy</strong></td>
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<tr>
<td>Beta Blockers</td>
<td>27(52%)</td>
<td>275 (55%)</td>
</tr>
<tr>
<td>CCBs</td>
<td>10 (19%)</td>
<td>135 (27%)</td>
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<tr>
<td>Digoxin *</td>
<td>3 (6%)</td>
<td>213 (43%)</td>
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<tr>
<td>Amiodarone</td>
<td>15 (29%)</td>
<td>122 (25%)</td>
</tr>
<tr>
<td>ACEI/ARB†</td>
<td>17 (33%)</td>
<td>248 (50%)</td>
</tr>
<tr>
<td><strong>Echocardiography</strong></td>
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<td></td>
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<tr>
<td>Normal †</td>
<td>14 (27%)</td>
<td>55 (11%)</td>
</tr>
<tr>
<td>Dilated LA‡</td>
<td>27 (52%)</td>
<td>333 (67%)</td>
</tr>
<tr>
<td>Dilated LV‡</td>
<td>11 (21%)</td>
<td>191 (39%)</td>
</tr>
<tr>
<td>LVH</td>
<td>18 (35%)</td>
<td>166 (34%)</td>
</tr>
<tr>
<td>LVEF≤45% *</td>
<td>2 (4%)</td>
<td>129 (12%)</td>
</tr>
</tbody>
</table>

P<0.001, † P<0.01, ‡ P<0.05 for comparisons among groups using independent t-test, Mann-Whitney U tests and Chi-square tests as appropriate. All percentages are given to the nearest whole percent.
### Table 2. Univariate & multivariate predictors of SR at 6 weeks and 1 year

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Follow-up at</th>
<th>UNIVARIATE</th>
<th>95% Confidence Interval</th>
<th>P Value</th>
<th>MULTIVARIATE</th>
<th>95% Confidence Interval</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Immediate Cardioversion (IC)</td>
<td></td>
<td></td>
<td>Elective Cardioversion (EC)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Group (n=52)</td>
<td>Risk Ratio</td>
<td>95%</td>
<td>Group (n=496)</td>
<td>Risk Ratio</td>
<td>95%</td>
</tr>
<tr>
<td>Digoxin use</td>
<td>6W</td>
<td>4.13</td>
<td>0.35-48.94</td>
<td>0.26</td>
<td>1.40</td>
<td>0.95-1.97</td>
<td>0.088</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>2.90</td>
<td>0.25-34.19</td>
<td>0.40</td>
<td>1.49</td>
<td>1.00-2.21</td>
<td>0.049</td>
</tr>
<tr>
<td>Amiodarone use</td>
<td>6W</td>
<td>4.67</td>
<td>1.30-16.73</td>
<td>0.018</td>
<td>2.13</td>
<td>1.4-3.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>6.50</td>
<td>1.70-24.93</td>
<td>0.006</td>
<td>2.33</td>
<td>1.57-3.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LA &gt; 40mm</td>
<td>6W</td>
<td>0.64</td>
<td>0.20-2.03</td>
<td>0.40</td>
<td>0.58</td>
<td>0.39-0.86</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>0.5</td>
<td>0.16-1.54</td>
<td>0.23</td>
<td>0.62</td>
<td>0.41-0.94</td>
<td>0.02</td>
</tr>
<tr>
<td>Hypertension</td>
<td>6W</td>
<td>0.52</td>
<td>0.14-1.87</td>
<td>0.32</td>
<td>1.60</td>
<td>1.12-2.28</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>0.81</td>
<td>0.23-2.87</td>
<td>0.75</td>
<td>1.25</td>
<td>0.85-1.83</td>
<td>0.26</td>
</tr>
<tr>
<td>Atrial Flutter</td>
<td>6W</td>
<td>3.86</td>
<td>1.11-13.37</td>
<td>0.033</td>
<td>2.11</td>
<td>1.32-3.39</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>2.27</td>
<td>0.69-7.54</td>
<td>0.18</td>
<td>1.64</td>
<td>1.01-2.66</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>AF/flutter duration (per week increase)</td>
<td>6W</td>
<td>0.96</td>
<td>0.95-0.97</td>
<td>&lt;0.001</td>
<td>0.96</td>
<td>0.95-0.98</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AF/flutter duration (per week increase)</td>
<td>1Y</td>
<td>0.97</td>
<td>0.95-0.98</td>
<td>&lt;0.001</td>
<td>0.97</td>
<td>0.95-0.98</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&gt;1 Shock</td>
<td>6W</td>
<td>0.73</td>
<td>0.13-4.17</td>
<td>0.73</td>
<td>1.59</td>
<td>1.1-2.36</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>1Y</td>
<td>0.50</td>
<td>0.09-2.86</td>
<td>0.44</td>
<td>1.56</td>
<td>1.07-2.26</td>
<td>0.025</td>
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<td></td>
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Initial success rates were similar in both groups regardless of the presenting rhythm. In the EC group, 86.3% were successfully cardioverted (87.4% in AF and 86.1% in atrial flutter). In the IC group 94.2% were cardioverted (94.4% in AF and 93.8% in atrial flutter patients). A significantly higher percentage of IC patients were in SR at both 6 weeks and 1 year than in the EC group (65.4% versus 43.3 % and 57.7% versus 30% respectively—Figure 1).

Table 2 illustrates the significant predictors of SR maintenance at 6 weeks and 1 year. Combining known duration of AF/flutter with the requirement for >1 shock to achieve SR provided a graded prediction regarding outcome at 6 weeks and 1 year (Figure 2) which was superior to the prediction using either factor alone. Neither previous CV nor CHADS2 score predicted rhythm outcome at 6 weeks or 1 year on either univariable or multivariable analysis.

Figure 1. The percentage of patients in SR at 6 weeks and 1 year according to the number of shocks required to achieve SR in both Immediate and Elective Cardioversion groups combined, n=477

Morbidity and mortality—At 1 year, there was no difference in mortality between groups (IC one death; 1.9%, EC 11 patients; 2.2%) or new-onset heart failure (2 patients; 3.8% compared with 14 patients; 2.8%). Combined cardiovascular events including fatal and non-fatal acute coronary syndromes, stroke (1 patient in the IC; 1.9%, 7 patients in the EC; 1.4%), hospitalisation due to recurrence of AF/flutter and
heart failure affected a higher percentage of patients in the IC group (24.7%) than in the EC group (14.8%; P=0.025).

Figure 2. Maintenance of SR at 6 weeks and 1 year according to a combination of known duration of AF/atrial flutter and number of shocks required for successful cardioversion

Discussion

Only 43.3% and 30% of the EC group remained in SR at 6 weeks and 1 year respectively. The success rate was significantly higher in the IC cohort (65.4% and 57.7% respectively). We have documented, for the first time, that initial resistance to electrical CV, reflected in the number of shocks required to restore SR, is one indicator of the likelihood of reverting to AF.

Along with others,9,13,14,16,18-20 we identified arrhythmia duration as an independent predictor of rhythm outcome. With long-established AF/flutter, electrical remodelling becomes established rendering the atria resistant to resynchronisation and vulnerable to early return of AF or flutter. Our results, along with earlier reports,9,10,13,14,16-20 suggest that every effort should be made to ensure patients are referred early for CV.
The ability of echocardiographic indices to predict outcome following CV is controversial. In our patients, as reported elsewhere,\textsuperscript{9,20} left atrial dilatation was predictive of return to AF/flutter – but on univariate analysis only.

Higher rates of sustained SR in IC compared with EC patients presumably reflect younger age, a higher proportion in atrial flutter, shorter duration of arrhythmia, less prior cardiovascular disease and more frequently normal cardiac structure.

The role of amiodarone in preventing atrial fibrillation post cardiac surgery and post CV is well established.\textsuperscript{23-26} Accordingly, we noted pre-treatment with amiodarone appeared protective against recurrence of AF/atrial flutter. IC and EC patients on beta-blockers were more likely to remain in SR, although statistically not significant, similar to earlier findings suggesting that at least some beta-blockers improve rates of sustained SR after CV\textsuperscript{25-27} and for patients with heart failure beta-blockers reduce the incidence of new AF onset.\textsuperscript{28}

Despite evidence that the renin-angiotensin system promotes arrhythmogenesis\textsuperscript{15} whereas blockade of the system (with ACE inhibitors or angiotensin II receptor blockers) can be inhibitory, we found ACE inhibitors had no impact on post CV outcome. Those on ACE inhibitors, however, were more likely to have impaired cardiac function. Accordingly, it is likely that any protective effect of ACE inhibitors against return of AF was masked by the underlying cardiac disorders for which they were prescribed. Similarly in our group of patients, being on statins did not seem to influence SR maintenance.

Hypertensive cardiovascular disease is the most common antecedent of AF.\textsuperscript{29,30} Accordingly, one might have anticipated hypertension to be a negative, rather than positive predictor of outcome. However, hypertension did not predict outcome at 1 year, and did not retain independent predictive power on multivariate analysis. Similar to earlier findings,\textsuperscript{14} the presence of atrial flutter rather than AF was a positive predictor of sustained SR.

**Limitations**—The effect of medications on cardiac rhythm cannot be ascertained with confidence from our data. Beyond the factors we have evaluated, a variety of medications and chromosomal variants may have played a role in determining success or failure in maintaining SR.

A monophasic defibrillator was utilised in all patients undergoing cardioversion in this study. Current guidelines and recent robust papers recommend the use of biphasic shocks and the extrapolation of our findings to biphasic defibrillators may not be appropriate.

In summary, in a large consecutive cohort from a single referral centre, 57.7% of patients requiring immediate cardioversion for AF/atrial flutter remained in SR after CV at 1 year whereas the figure was only 30% in patients undergoing elective cardioversion. The number of shocks required to achieve SR, and the known duration of AF/atrial flutter are independent additive predictors of rhythm at 6 weeks and 1 year after elective CV.
Competing interests: None.

Author information: Amjad K Hamid, SMO, PhD Student*; A Mark Richards, Professor*; Ian G Crozier, Cardiologist†; John G Lainchbury, Cardiologist†; Iain Melton, Cardiologist†; Paul G Bridgman, Cardiologist†; Suetonia C Palmer, Nephrologist*; Chris M Frampton, Biostatistician*; M Gary Nicholls, Professor*

*Department of Medicine, Otago University – Christchurch
†Department of Cardiology, Christchurch Hospital, Christchurch

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Correspondence: Amjad K Hamid, Cardiology Department, Christchurch Hospital, Private Bag 4710, Riccarton Avenue, Christchurch, New Zealand. Fax: +64 (0)3 3648303; email: amjadh@cdhb.govt.nz

References:


Early cardiac morbidity of rheumatic fever in children in New Zealand

Olwen Gilbert, Nigel Wilson, Kirsten Finucane

Abstract

Aim The aim of this study was to review the severity and morbidity of acute rheumatic fever (ARF) and rheumatic heart disease (RHD) for children with the most significant cardiac disease in the current era in New Zealand.

Method Retrospective 2-year review of children with ARF and RHD admitted to Starship Children’s cardiology ward. Medical and surgical admissions were classified. Echocardiographic severity of cardiac disease and cardiac surgical data were analyzed. Using length of stay data and 2009 District Health Board costings, admission costs were calculated.

Results 36 children had 49 admissions. Mean age 11.8 ± 2.4 years. All but one child was of Māori or Pacific Island ethnicity. 10 children had symptoms and signs of congestive cardiac failure on admission. The average length of stay was 23 days, but the subset of children with ARF requiring cardiac surgery at the same admission had an average of 54 days (range 36–78 days) in hospital. The total hospital costs over the 2-year period was $1,918,600.

Conclusion Failure to prevent rheumatic fever in New Zealand means that there is significant cardiac sequelae for those children who develop severe RHD. The early morbidity includes heart failure, need for cardiac surgery, and prolonged hospital stay.

Acute rheumatic fever (ARF) continues unabated in New Zealand in Māori and Pacific Island populations.1,2 The cardiac sequelae of ARF includes rheumatic heart disease (RHD) which can lead to impairment of exercise, need for cardiac operations and premature mortality. There are still over 200 deaths per year attributed to RHD in New Zealand, mainly in adults.3

In New Zealand, children with severe cardiac disease from ARF or RHD that may require operation are referred to the sole cardiac surgical unit at Starship Hospital, Auckland. The aim of this study was to review the severity and morbidity of ARF and RHD for children with the most significant cardiac disease in the current era.

Method

This study was a retrospective chart review of those children with ARF or RHD admitted to the Paediatric and Congenital Cardiology Ward between July 2007 and June 2009 inclusive. Demographic analysis was by patient. Length of stay and cost analysis was by each admission. Definitions of groups:

- ‘ARF-medical’ was defined as children fulfilling the diagnosis of ARF by the NZ rheumatic fever guidelines criteria 2 which have greater sensitivity for ARF than the Jones criteria. These patients had carditis as the major criterion of ARF, raised inflammatory markers and elevated streptococcal titres.
‘ARF-surgical’ was defined as those with ARF who proceeded to cardiac surgery during the same admission.

‘RHD-surgical’ was defined as those who had elective cardiac valve surgery beyond the acute phase of ARF.

‘RHD-medical’ was defined as any patient who was admitted with known RHD but not for the purpose of cardiac surgery.

Echocardiographic results analyzed were severity of valve regurgitation and assessment of ventricular function. Cardiac dilatation was assessed by relating the left ventricular end systolic and diastolic diameters and volumes to body surface area, expressed as a Z score, which represents the number of standard deviations from the normal population mean value. Left ventricular ejection fraction less than 55% was regarded as abnormal.

Admission costs were calculated by using the 2009 Auckland District Health Board (ADHB) costing, used for inter-DHB charging. (S. Adams, personal communication) Cardiac ward cost is $1200/night, paediatric intensive care unit $4700/night, theatre costs $2700/hour.

The average time for single valve replacement was assigned 4 hours, aortic root replacement 5 hours, single valve repair 5 hours, double valve repair 5 hours and triple valve repair 6 hours.

The cost of valve replacement was $3600 per prosthetic valve, $2000 per homograft and $6000 per porcine valve. Analysis of the continuous variables hospital costs and length of stay was expressed as mean and standard deviation.

The study received ethical approval from the New Zealand Northern Y Regional Ethics committee.

Patients referred from the Pacific Islands for RHD cardiac surgery were excluded from the analysis.

Results

Patient demographics—Over the 2-year period 36 children had a total of 49 admissions. Twenty-four children had a single admission, 11 children had a second admission, and one child had three admissions in the time period studied. There were 21 male and 15 female patients.

Recorded ethnicity: Māori (13), Samoan (11), Cook Island Maori (7), Tongan (2), other Pacific not specified (1), Niuean (1) and Indian (1). All but one child were domiciled in the North Island. 22 of the 49 admissions were from the Auckland region with most of the disease burden in South Auckland (Figure1).

Diagnostic Admission Groups—There were 23 admissions with ARF of which 16 were ARF-medical and seven ARF-surgical. There were 26 admissions with RHD, 18 were RHD-surgical and eight RHD-medical. Reasons for admission for the RHD-medical group were readmission after cardiac surgery with wound infection (n=2) or pleurisy (1). Two children with RHD and sepsis were admitted to exclude bacterial endocarditis. One had a final diagnosis of Salmonella typhi septicaemia and the second had reactivation of Hepatitis B. One child was admitted for non-cardiac surgery, two were admitted for diagnostic work up of valve disease aetiology or RHD disease severity requiring transoesophageal echocardiography under anaesthetic.
The mean age of admission was 11.8±2.4 years with an age range 5 to 15 years. The mean body mass index was 25.4±6.1 with a range 12.4–38.5.

**Cardiac disease severity**—Ten children had symptoms and signs of congestive cardiac failure on admission (Six of the 16 children in the ARF-medical group had cardiac failure on admission but this was controlled without the need for early cardiac surgery).

Four of the seven children in the ARF–surgical group had fulminant ARF requiring early cardiac surgery during their initial presentation. One of these was the youngest child of 5.4 years who was cachectic with a BMI of 12.4. Her heart failure symptoms were recent, but judging by the degree of cachexia and chronic rheumatic changes of the valve by echocardiography, the length of illness was likely several months duration. Two of the 18 in the RHD-surgical group had signs of cardiac failure. No child showed mitral stenosis.

One child was admitted with ARF had complete heart block which resolved spontaneously. She was discharged with mild residual pathological mitral regurgitation.

Left ventricular size and function was recorded on 46 admissions. The left ventricular size related to body surface area was significantly increased for the cohort: left ventricular end diastolic dimension Z score+4.8±2.8, left ventricular end systolic dimension Z score+3.6±3. Left ventricular function was depressed in 10 patients and normal in 36. The type of cardiac valve with regurgitation is shown in Figure 2.
Figure 2. Type of cardiac valve involvement

Cardiac surgery—Twenty-five patients underwent cardiac surgery, mean age 11.6 ± 2.6 years. Ten had single valve surgery, 14 had double valve surgery and one had triple valve surgery. Of those who underwent mitral valve surgery, 17 had a mitral valve repair and two received a mitral valve replacement as the valve changes were too severe to achieve a repair. Four of 15 patients who underwent aortic valve surgery had a repair and 11 had aortic valve replacement, usually an aortic homograft for which warfarin anticoagulation is not required.

Complications in hospital—There was no mortality for this cohort during the study period. One child developed acute cerebral cortical ischaemia perioperatively leading to hemiplegia and seizures following cardiac surgery. One child developed Stevens Johnson Syndrome as an allergic response to penicillin. Erythromycin was started for on going antibiotic prophylaxis to prevent ARF recurrence. Other complications included *Klebsiella pneumonia* (n = 1), children who had transient heart block (2), pericarditis (1) and epistaxis (3).

Complications for those undergoing cardiac surgery included left lower lobe collapse (n = 4), arrhythmia requiring either pacing or anti-arrhythmic medication (4) post operative pulmonary oedema (3), and pericardial effusion (4). All of these had resolved by discharge.

All children continued to receive 28 day intramuscular benzathine penicillin as secondary prophylaxis to prevent recurrences of ARF except the one child who was continued on erythromycin prophylaxis.

The mean length of stay for all admissions was 22.7 ± 20.3 days with a range of 2–78 days. Length of stay by admission type is shown in Figure 3. The ARF-surgical group had the longest length of stay with a mean of 54 days ± 16, range 36–78 days. The shortest stay group was RHD medical with a mean of 5.4 days.
Figure 3. Length of stay for each admission group.

![Mean Length of Stay](image)

Figure 4. Cost of admission for each patient group

![Approximate Cost of Admission](image)
Six children with ARF who did not require early operation were transferred back to their local paediatrician for further inpatient management. Four of the children with RHD who had surgery were transferred back to their own hospital for management of moderate pericardial effusions. The two children who had wound infections were transferred back for ongoing antibiotic treatment.

**Cost of admissions**—The average costs of admissions by group are shown in Figure 4:

The most expensive category was ARF-Surgical patient group, with an average cost of $90,157±$6,388, range $66,500-113,300. The total hospital costs over the 2 year period was $1,918,600 or nearly $1,000,000 per year.

**Discussion**

This study has examined the subset of children with ARF or RHD who had the most significant cardiac sequelae of the disease. It reveals considerable early morbidity with heart failure, need for cardiac surgery, and prolonged length of hospital stay. Rheumatic fever continues to affect almost exclusively Māori and Pacific populations in New Zealand, confirmed in this study.

There was a need for cardiac surgery in this group of children due to cardiac symptoms or cardiac dilatation that resulted from cardiac valve regurgitation, with the risk of long term myocardial damage and premature mortality. Mitral valve repair is preferred to valve replacement in children as the morbidity of prosthetic valves and need for warfarin anticoagulation is significant. There has been extensive use of aortic valve homografts for aortic valve replacement in children, as these do not require warfarin. Tricuspid valve surgery can usually be achieved by an annuloplasty ring and valve repair, again without the need for warfarin.

Prolonged hospitalisation is part of the morbidity of ARF. Traditionally, those with ARF were managed with strict bed rest, due to the known ongoing inflammation which can last months, judged by acute inflammatory markers, active inflammation found at the time of cardiac surgery or at post-mortem.

At KidsFirst Hospital, South Auckland, Nicholson and colleagues showed that those with ARF without carditis could be safely mobilized within 2 weeks and this has lead to earlier mobilization of ARF patients nationally. However, currently at KidzFirst hospital, the average length of stay for those with severe valve regurgitation is 45 days (R Nicholson, personal communication) similar to the average 29 days in the current study when allowance is made that some children returned for inpatient care to their referral hospital.

The calculated costs were direct in-patient care costs, not the total medical costs of this disease. North and colleagues calculated that costs for rheumatic fever in Auckland in 1992 to be $3.6m per year. As Auckland has approximately half the disease burden of ARF and RHD in New Zealand, a simple doubling of these costs, with an inflation factor allowance to 2010, gives current national medical costs to be $9.5–10.5m per year.
There is considerable recent endeavour to reduce the disease burden of ARF and RHD, outlined in New Zealand guidelines \(^1,^2,^10,^11\) with renewed leadership by the New Zealand Rheumatic Fever Steering group. The aetiology of ARF in New Zealand shows striking ethnic differences, which reflects health and social inequality.\(^12\) Primordial and primary prevention is possible.\(^13,^14\)

The development of severe RHD may be reduced by detecting mild disease by echocardiography, then treating with penicillin.\(^15,^16\) Until all these measures take effect, there will continue to be children and young adults with severe cardiac consequences of ARF and RHD in New Zealand.

**Competing interests:** None.

**Author information:** Olwen Gilbert, Paediatric Registrar; Nigel J Wilson, Paediatric Cardiologist; Kirsten Finucane, Cardiothoracic Surgeon; Green Lane Paediatric and Congenital Cardiac Services, Starship Children's Hospital, Auckland

**Correspondence:** Nigel Wilson, Paediatric Cardiologist, Green Lane Paediatric and Congenital Cardiac Services, Starship Children's Hospital, Private Bag 92024, Auckland 1142, New Zealand. Fax: +64 (0)9 3757026; email: nigelw@adhb.govt.nz

**References:**


Are we meeting cardiovascular risk targets 3 years after acute coronary syndrome? An evaluation in West Auckland, New Zealand

John A Ford, Jocelyn Bell, Colin Edwards

Abstract

Aim Several studies have shown poor achievement of cardiovascular targets in high risk patients. We measured these targets in patients with Acute Coronary Syndrome, three years after discharge from Waitakere Coronary Care Unit.

Method A retrospective observational study was performed. All patients discharged in 2006 were included. Admission data was extracted from computerised records and patients were subsequently invited for appointment. Data collected included: blood pressure, lipid profile, BMI, smoking status, HbA1c, medications and contraindications, and lifestyle factors. Results were analysed and compared with national targets.

Results Data was collected on 112 patients (22 patients died, 18 excluded and 18 lost to follow up). There was good compliance with blood pressure (mean 120/70 mmHg), smoking cessation and medication targets. However 22% of patients were not prescribed an ACE inhibitor at follow-up. Lipid profile improved, although only 52% of patients met LDL targets. There was no difference between admission and follow-up BMI. HbA1c had increased slightly, however this was not statistically significant. Eight diabetic patients (n=27) had an HbA1c of less than 7% at follow-up.

Conclusion Although a small sample population, results showed mixed compliance but not as poor as previously reported. More effort is needed to attain LDL, HbA1c and BMI targets, and ensure ACE inhibitor initiation.

Cardiovascular disease remains the leading cause of mortality in New Zealand. The modification of cardiovascular risk factors has been proven to reduce mortality. Subsequently national guidelines have been devised to improve cardiovascular risk factors. The New Zealand Guideline Group (NZGG) published extensive guidelines in 2003 which were updated in 2009. This guideline outlines the most important targets with respect to: smoking, weight, diabetic control, diet and exercise, blood pressure, cholesterol and pharmacological treatment. The targets for patients with established cardiovascular disease and high risk patients are:

- Blood pressure less than or equal to 130/80 mmHg
- BMI less than 25, but in patients with a BMI less than 35 an initial goal of 10% weight loss is more realistic
- Lipids
  - Total Cholesterol less than 4 mmol/L
o HDL more than 1 mmol/L
o LDL less than 2.0 mmol/L
o Cholesterol ratio less than 4.0
o Triglycerides less than 1.7 mmol/L

- All patients should be prescribed the combination of aspirin, beta-blocker, ACE Inhibitor and statin unless contraindicated
- Minimum of 30 minutes of moderate intensity physical activity on most days of the week
- Smoking cessation should be strongly and repeatedly recommended
- HbA1c less than 7%
- At least 3-4 portions of fruit and 3-4 servings of vegetables per day

In New Zealand, several studies have suggested that compliance with targets is poor, especially among certain population groups. El-Jack et al, audited patients in South Auckland with established coronary artery disease that had recently been discharged from hospital. They reported that 37% of patients failed to reach total cholesterol of less than 5 mmol/l and 55% had a LDL of more than 2.6 mmol/l. In addition, over 40% of patients had a blood pressure greater than 140/90 mmHg. Thirty-four of patients were prescribed ACE inhibitors, and 45%, prescribed beta-blockers. Selak et al reported that only 28%, increasing to 32% in 2003, of people with established vascular disease were on blood pressure and cholesterol lowering medications. Peris et al found better compliance, 78.1% of high risk patients were prescribed anti-hypertensive medication, 71.9% lipid lowering and 65.3% anti-platelet agents. With increasing numbers of patients throughout New Zealand being admitted with ACS, compliance is of growing importance.

In view of this reported poor compliance in high risk patients we have evaluated patients 3 years following discharge from Waitakere Hospital after admission with Acute Coronary Syndrome (ACS). Waitakere Hospital provides secondary healthcare for over 180,000 people. Patients admitted with acute coronary syndrome receive education from doctors and nurses while in hospital. After discharge patients attend an outpatient appointment for review by a cardiologist or cardiology registrar. They are invited to attend a cardiac rehabilitation program. This comprehensive programme consisting of six sessions, during which patients are educated regarding their condition, new medications, secondary prevention and the importance of risk factor modification. The aim of our study was to measure attainment of NZGG targets and highlight any areas of weakness.

**Methods**

Local ethics committee and Nga Kai Tataki Maori Research Committee approval was obtained. Each patient gave written consent before participation. Patient details were obtained from the Coronary Care Unit (CCU) admission book. All patients discharged from Waitakere CCU in 2006 with acute coronary syndrome were included. All discharge summaries from 2006 were analysed and a study population was identified. Acute coronary syndrome was defined as unstable angina, non-ST elevation myocardial infarction or ST elevation myocardial infarction. Patients were excluded if the troponin rise was attributed to a cause other than myocardial infarction, if they had relocated or could not attend clinic.
The following admission variables were recorded from the computerised records (Concerto) or cardiac rehab database; coronary angiography report, age, sex, diabetic status, admission and discharge dates, diagnosis, lipids, HbA1c, discharge medications and contraindications, ejection fraction, past medical history of depression or hypertension and attendance at cardiac rehab. No admission blood pressure was collected. The authors felt measurements would not have been a true reflection of long term hypertension due to treatment effects and the acute phase response during ACS.

Patients were then invited to attend a research appointment (April to May 2010) with a follow-up range of 41-53 months. The following variables were collected; blood pressure (complying with NZGG guidelines), BMI (complying with NZGG guidance), smoking status, family history (first degree relative younger than 55 years for male or 65 years old for female), current medication and known contraindications, lifestyle habits and employment. Contraindications for beta-blockers were obstructive airway disease, peripheral vascular disease, bradycardia, hypotension, heart block, decompensated heart failure and allergy.

For aspirin contraindications were previous gastrointestinal bleed, hypercoagulability and allergy. Liver disease or allergy were contraindications for statins. And finally, contraindications for ACE inhibitors were bilateral renal artery stenosis, electrolyte disturbance, symptomatic hypotension and allergy. If patients did not tolerate a medication because of side effects they were included in the contraindicated group. Exercise was classified as more than a total of 30 minutes of activity per day; enough to cause the patient to be slightly short of breath. Follow-up lipids levels and HbA1c were recorded from Concerto. If levels had not been recorded in the previous six months blood tests were rechecked. Data was analysed using paired t-test in SPSS (version 17.0, 2007).

**Results**

**Patients**—A total of 369 patients were discharged from CCU in 2006 (see Figure 1). 170 patients were diagnosed with ACS. Twenty-two patients had died at follow-up. Eighteen patients were excluded and 18 patients were lost to follow-up, leaving 112 patients. Cardiac rehabilitation was defined as attending at least two of six rehab session. Sixty-seven patients met this definition.

Patient demographics are shown in Table 1. Most patients were male (n=86) and the mean age was 66.73 years. NZ Europeans comprised the majority of the participants. Over 20% of patients suffered from diabetes and a considerable number had a family history of cardiovascular disease (49.1%) or hypertension (59.8%).

Only one patient did not have lipids checked on admission and this was due to a laboratory strike. Thirteen patients had not received lipid levels measurements in the six months prior to follow up. Twelve of these patients attended a blood test upon request. Four patients did not have HbA1c checked on admission. Only one patient had not had their HbA1c checked in the six months prior to follow-up. Admission BMI was unavailable for two patients.
Figure 1. Flow chart showing included and excluded patient

![Flow chart](image)

<table>
<thead>
<tr>
<th>Table 1. Patient demographics</th>
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<table>
<thead>
<tr>
<th>Mean age (SD)</th>
<th>66.73 years (11.92)</th>
</tr>
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<tbody>
<tr>
<td><strong>Sex (%)</strong></td>
<td>Male 86 pts (76.8)</td>
</tr>
<tr>
<td></td>
<td>Female 26 pts (23.2)</td>
</tr>
<tr>
<td><strong>Diagnosis (%)</strong></td>
<td>NSTEMI 73 pts (59.8)</td>
</tr>
<tr>
<td></td>
<td>STEMI 31 pts (27.7)</td>
</tr>
<tr>
<td></td>
<td>Unstable Angina 8 pts (7.1)</td>
</tr>
<tr>
<td><strong>Ethnicity (%)</strong></td>
<td>NZ Europeans 69 pts (61.1)</td>
</tr>
<tr>
<td></td>
<td>Indian 10 pts (8.9)</td>
</tr>
<tr>
<td></td>
<td>Pacific Island 6 pts (5.4)</td>
</tr>
<tr>
<td></td>
<td>Asian 3 pts (2.7)</td>
</tr>
<tr>
<td></td>
<td>Maori 5 pts (19.6)</td>
</tr>
<tr>
<td></td>
<td>Other 19 pts (16.9)</td>
</tr>
<tr>
<td><strong>Diabetes (%)</strong></td>
<td>Non-insulin requiring DM 22 pts (19.6)</td>
</tr>
<tr>
<td></td>
<td>Insulin requiring DM 6 pts (5.4)</td>
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<tr>
<td><strong>Family history (%)</strong></td>
<td>Cardiovascular disease 55 pts (49.1)</td>
</tr>
<tr>
<td></td>
<td>Hypertension 67 pts (59.8)</td>
</tr>
<tr>
<td></td>
<td>Depression 7 pts (6.3)</td>
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</table>

**Blood pressure, smoking status, BMI, HbA1c and lipid targets**—Median systolic blood pressure was 120 mmHg (range 78-180 mmHg, SD 15.08) and median diastolic blood pressure was 70 mmHg (range 50-90 mmHg, SD 8.02). Eighty-five patients (75.9%) had a blood pressure less than 130/80 mmHg.

On admission 24.1% (n=27) of patients were current smokers, 41.1% (n=46) ex-smokers and 34.8% (n=39) had never smoked (see Figure 2). At follow-up 51.9% (n=14) of the smokers had stopped. Three patients who were ex-smokers on admission re-started smoking by follow-up.
Figure 2. Smoking, BMI and HbA1c results on admission and follow-up

<table>
<thead>
<tr>
<th>Admission Smoking (n=112)</th>
<th>Follow-up Smoking (n=112)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never (27)</td>
<td>Never (16)</td>
</tr>
<tr>
<td>Ex-smoker (39)</td>
<td>Ex-smoker (39)</td>
</tr>
<tr>
<td>Current (46)</td>
<td>Current (57)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Admission BMI (n=110)</th>
<th>Follow-up BMI (n=112)</th>
</tr>
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<tbody>
<tr>
<td>&lt; or = 25 (40)</td>
<td>&lt; or = 25 (38)</td>
</tr>
<tr>
<td>&lt; or = 30 (52)</td>
<td>&lt; or = 30 (27)</td>
</tr>
<tr>
<td>&gt; 30 (18)</td>
<td>&gt; 30 (47)</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Admission HbA1c (n=24)</th>
<th>Follow-up HbA1c (n=27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; or = 7 % (16)</td>
<td>&lt; or = 7 % (19)</td>
</tr>
<tr>
<td>&gt;7 % (8)</td>
<td>&gt;7 % (8)</td>
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</table>

Median BMI on admission was 28.4 (range 18.8-44.9, SD 4.6). At follow-up median BMI was 27.8 (range 20.5-48.9, SD 5.1). There was no statistical difference between admission and follow-up (p value 0.58, 95% CI -0.02, 0.90 ). At follow-up 27 patients (24.1%) had a BMI of less than 25 and 74 patients (66.1%) had a BMI less than 30, as seen in Figure 2. Twenty-two patients (20.0%) lost more than 5% body weight, eight patients (7.3%) lost more than 10% body weight and 16 patients (14.5%) gained more than 5% bodyweight.

The median HbA1c in patients with diabetes was 6.8% (range 5.7-13.4, SD 1.8) on admission and 7.6% (range 5.8-12.3, SD 1.6) at follow-up. Figures 2 show that on admission 16 patients (total =24) had a HbA1c of less than 7% and at follow-up only eight (total =27) had a HbA1c of less than 7%. There was no significant statistical difference between admission and follow-up (p value 0.79, 95% CI -1.14,0.07).
Table 2. Admission and follow-up lipids

<table>
<thead>
<tr>
<th>Lipids</th>
<th>Admission (SD)</th>
<th>Follow-up (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.7 (1.2)</td>
<td>3.9 (1.1)</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>1.1 (0.3)</td>
<td>1.1 (0.3)</td>
</tr>
<tr>
<td>LDL (mmol/L)</td>
<td>2.8 (1.1)</td>
<td>2.0 (0.8)</td>
</tr>
<tr>
<td>Ratio</td>
<td>4.2 (1.2)</td>
<td>3.4 (1.3)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.6 (0.9)</td>
<td>1.4 (1.0)</td>
</tr>
</tbody>
</table>

Lipid results are shown in Table 2 and Figure 3 show the number of patients who met targets at follow-up. Eighty-two patients (73.9%) met HDL targets but only 58 pts (52.3%) met LDL targets at follow-up. Over 60% of patients met targets for total cholesterol (62.2%), ratio (64.9%) and triglycerides (64.0%).

There was a significant statistical difference in total cholesterol levels (p value <0.001, 95% CI 0.38, 0.87), LDL (p value <0.001, 95% CI 0.34, 0.77), ratio (p value <0.001, 95% CI 0.42, 0.90) and borderline significant difference in triglycerides (p value 0.05, CI 0.0002,0.39). There was no significant statistical difference between admission and follow-up HDL (p value 0.82, CI -0.90,0.01).

Figure 3. Patients who met lipid targets at follow-up

Medications—One patient on discharge and one at follow-up were not prescribed aspirin without contraindication (see Figure 4). Only one patient on discharge and three patients at follow-up were not prescribed a statin without contraindication. Three patients (2.7%) were not prescribed a beta-blocker on discharge and seven (6.3%) at follow-up without contraindication. Thirty-one patients (27.7%) were not prescribed an ACE inhibitor or ARB on discharge and 25 (22.3%) were not prescribed an ACE inhibitor or ARB without contraindication at follow-up. On discharge 74
patients (66.1%) were not prescribed GTN spray; however at follow-up 30 patients (26.8%) were not prescribed GTN.

Figure 4. Discharge and follow-up medications

![Figure 4](image)

Figure 5: Follow-up exercise and diet responses

![Figure 5](image)
Exercise and diet—Nineteen patients (17.0%) at follow-up were performing 30 minutes of exercise more than five times per week. Twenty patients (17.9%) claimed not to be performing any exercise (see Figure 5). Most patients either consumed 1-2 or 3-4 portions of fruit and vegetables (36.6% and 39.3% respectively). Only four patients had seven or more portions. Approximately half of patients (47.3%) consistently reduce their salt intake and 8.9% never attempted to reduce salt consumption (see Figure 5). Seventy-two patients (64.3%) consistently reduced dietary fat and there were no patients who never attempted to reduce their intake of fat.

Discussion

Our results show mixed achievement of NZGG targets, but this was not as poor as previous reported studies. Our results show considerable improvement, despite higher targets, compared with El-Jack et al.10 Hopefully this improvement reflects better clinical management, reflected in other previous studies.11 However the differences in health inequalities cannot be excluded.

The most comparable study by El Jack et al,10 was performed in South Auckland; a population with a higher percentage of Pacific Island and Maori peoples14. Whereas there is a higher percentage of New Zealand European peoples in the west Auckland region surrounding Waitakere Hospital.14 Unsurprisingly our results show improved lipid management compared to Ellis et al in 1998.15 In a study of 641 patients they found a mean total cholesterol of 5.7 mmol/l, although only 32% of patients were prescribed lipid lowering medications.

Collecting data directly from patients, in combination with computerised records system, provides high quality data; although inaccuracies of patient reported smoking status, exercise and diet are well known.16 Guidelines were followed when measuring blood pressure and BMI at follow-up, providing consistently accurate results. However admission BMI was collected from a range of sources, such as clinic letters and angiogram reports. Sixteen percent of patients were lost to follow up which may result in a small selection bias. Twenty-two patients died over the three year follow-up period.

The resulting survivor bias may have lead to an over estimation of compliance. There were eighteen patients who were lost to follow-up, this may have led to further bias if these patients were less compliant. It should also be noted that a considerable proportion of patients may have previous cardiovascular disease. Therefore many of them may have already been prescribed blood pressure or lipid lowering medications. If all patients with previous established cardiovascular disease had been excluded there may have been a greater improvement in target compliance. Admission medication histories were taken from discharge summaries and therefore should be accurate. However we suspect that many patients were prescribed GTN spray on discharge and this was omitted from the discharge summary.

We compared results with current guidelines issued in 2009. However patients’ primary care provider, usually a general practitioner, may still be using 2003 guidance. Although a detailed lifestyle questionnaire was not completed, we believe our results show the need for further efforts to improve diet and exercise.
Although only a small number of patients were included, our data is more consistently accurate compared with large population studies. Validity was further improved by including all patients discharged from 2006. Only patients admitted to CCU were included. Although it is hospital policy to admit all patients with ACS to CCU there may have been patients in non-CCU wards who had ACS and were not included in our study. We did not face the same problems of missing data as reported in other studies measuring compliance. In our study we recorded patients who had a documented contraindication or were able to provide a contraindication or intolerance at clinic appointment. This may reflect our better compliance with medication targets. Undoubtedly our population is composed of patients who have had considerable interaction with secondary care. It is therefore unsurprising, that compliance in our population is better.

Blood pressure control was surprisingly good, with nearly 76% of patients achieving target blood pressures. Despite the potential “white coat effect” median blood pressure was 120/70 mmHg. Similarly lipid levels show good compliance with targets. Ninety-seven percent of patients were taking statins. Only 58% achieved LDL target suggesting inadequate uptitration after discharge. At follow-up over 70% of patients met targets for HDL, over 60% targets for total cholesterol and triglycerides and over 80% target for cholesterol ratio. Only HDL did not show a statistically significant improvement. The attainment of blood pressure and lipid targets is due to patient education. This requires contribution from cardiac specialists, nurse specialists, cardiac rehabilitation team (specialist, nurse specialist, pharmacist, dietician and physiotherapist) and general practitioners post discharge.

Compliance in aspirin, statin and beta-blockers prescription are excellent. Prescriptions of ACE inhibitors require improvement. ACE Inhibitors have been shown to benefit patients post ACS but perhaps more so the subgroups with left ventricular impairment and/or hypertension. Normotensive subjects may get postural hypotension potentially making it difficult for the clinician to establish the patient on this therapy. Prescription of beta-blockers and statin decreased marginally over the follow up period. This may reflect contraindications which were not documented. Alternatively, three years following an event, medication compliance may decrease as patients no longer appreciate the importance of secondary prevention.

High rates of smoking cessation were achieved (51% of smokers stopping). This demonstrates the effective use of smoking cessation advice. Improvements could still be made. Slightly concerning was the finding that three patients had restarted smoking during follow-up. This highlights the need for continual smoking cessation support for months and years post ACS.

Undoubtedly weight reduction continues to be challenging. It was pleasing to see that most patients were making a concerted effort with dietary and exercise regimens. Despite this, the majority of patients remained overweight or obese. One in five patients had lost 10% body weight. Current advice is that in the immediate period after a cardiac event patients should aim to maintain the same weight. We have found that even after three years patients still struggle to lose weight.

Over three years HbA1c increased. Although this increase is not statistically significant it is clinically significant. Only eight patients had an HbA1c of less than
7%. This increase probably reflects disease progression rather than poor management. The important role of diabetic control in cardiovascular risk is well known. Policy makers and clinicians need to increase efforts at tighter glycaemic control within a well structured multidisciplinary team.

Our study shows that compliance within West Auckland is better than reported, for patients who remain alive at 3 years follow-up and are able to attend follow-up clinic. However there is still a large treatment gap, especially in relation to BMI, HbA1c and lifestyle. Compliance with blood pressure and lipid targets are very good, although there is room for improvement in relation to ACE inhibitors. Further study is needed to conclude if these differences reflect differences in data quality, health inequalities or improved public health measures.

Competing interests: None.

Author information: John A Ford, Research Assistant, HTA Group, University of Aberdeen, Scotland; Jocelyn Bell, Cardiology Nurse Specialist, Waitakere Hospital, Auckland; Colin Edwards, Cardiologist, Waitakere Hospital, Auckland

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Correspondence: Dr John A Ford, Department of Public Health, University of Aberdeen, Polworth Building, Foresterhill, Aberdeen, AB25 2ZD, Scotland, United Kingdom. Email: johnalexanderford@gmail.com

References:


Anti-tumour-necrosis-factor-alpha agents and the heart: beyond left ventricular systolic dysfunction?

Vassilis Vassiliou, Eleana Ntatsaki

Anti-tumour-necrosis-factor-alpha (anti-TNF-a) agents are increasingly used in many specialities including rheumatology, dermatology and gastroenterology. By modulating immune response, infection risk increases.

We present, what is to our knowledge, the first patient diagnosed with infective endocarditis whilst receiving adalimumab treatment.

Case report

A 25-year-old man presented to his general practitioner with a 4-week history of polyarthropathy, lethargy, weight loss, pyrexia and drenching night sweats. A referral was made to the hospital outpatient department where he was reviewed some 10 days later. Clinical examination revealed mild pyrexia, bilateral painful knees with effusions, significant weight loss, splinter haemorrhages and Osler's nodes.

Cardiovascular examination revealed a loud ejection systolic murmur which had been noted previously. Apart from plaque psoriasis treated with adalimumab 40 mg subcutaneously every other week for the last 4 months, his past medical history included balloon dilation for aortic coarctation at the age of 3, for which he was not under active follow-up.

In view of the deterioration in his functional status and the uncertainty of the diagnosis at that time, he was admitted for further investigations. The differential diagnosis on admission included vasculitis, tuberculosis, infective endocarditis and lymphoma. He denied excessive alcohol, smoking or recreational drugs.

Blood tests on admission revealed new normocytic anaemia (Hb 11.3 g/dL), leukocytosis (12.1×10^9/L) with neutrophilia, elevated erythrocyte sedimentation rate (ESR) of 113mm/hr, and C-reactive protein (CRP) of 117 mg/L. Urine dipstick was positive for blood. A vasculitis screen and three sets of blood cultures were collected. Knee aspiration suggested acute synovitis with no evidence of infection and computed tomography of chest, abdomen and pelvis was normal.

As anti-TNF-a agents might lead to left ventricular (LV) systolic dysfunction and are contraindicated in patients with severe heart failure (NYHA class III and IV) echocardiography can be performed around the time of therapy initiation as a baseline. Our patient had a transthoracic echocardiogram 3 months previously for systolic function assessment. Although LV systolic function was normal, the report suggested moderate aortic stenosis.

In order to make a definite diagnosis of infective endocarditis, either two major criteria, one major and three out of five minor criteria, or all five minor criteria have to be met. As our patient already fulfilled four minor criteria of the revised classification for infective endocarditis (Table 1), he was started on broad spectrum
antibiotics—intravenous benzyl penicillin and gentamicin—whilst awaiting blood culture results and further assessment of the aortic valve.

Table 1. Major and minor criteria for infective endocarditis

<table>
<thead>
<tr>
<th>MAJOR CRITERIA</th>
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<tbody>
<tr>
<td>1. Typical microorganism consistent with infective endocarditis isolated from two separate blood cultures: <em>Staphylococcus aureus</em>, viridans group streptococci, <em>Streptococcus bovis</em>, HACEK group, or community-acquired enterococci in absence of a primary focus or single positive blood culture or positive antibody titre for <em>Coxiella burnetii</em></td>
<td></td>
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<tr>
<td>2. Echocardiography showing vegetations, abscess or new partial dehiscence of a prosthetic valve or new valvular regurgitations</td>
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<table>
<thead>
<tr>
<th>MINOR CRITERIA</th>
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<tbody>
<tr>
<td>1. Predisposition: e.g. prosthetic valves, heart condition, intravenous drug use</td>
<td></td>
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<tr>
<td>2. Fever: temperature &gt;38°C</td>
<td></td>
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<tr>
<td>3. Vascular phenomena: arterial emboli, septic pulmonary infarcts, mycotic aneurysms, intracranial haemorrhages, conjunctival haemorrhages, janeway lesions</td>
<td></td>
</tr>
<tr>
<td>4. Immunological phenomena: glomerulonephritis, Osler’s nodes, Roth spots, rheumatoid factor</td>
<td></td>
</tr>
<tr>
<td>5. Microbiological evidence: positive blood cultures but does not meet major criterion or serological evidence of organism that is not consistent with infective endocarditis</td>
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</table>

Definite diagnosis of infective endocarditis in presence of two major criteria or one major and three minor criteria or all five minor criteria

Possible diagnosis of infective endocarditis in presence of one major and one minor criteria or three minor criteria


Blood cultures isolated a fully sensitive *Streptococcus viridans* in all six bottles; an organism suggestive of endocarditis. Transoesophageal echocardiography was suspicious of a vegetation on the aortic valve therefore the diagnosis of infective endocarditis was now definite fulfilling two major and four minor criteria. He was therefore treated with intravenous benzyl penicillin monotherapy for 4 weeks according to the European Society of Cardiology Guidelines. Following this he was referred to our regional cardiothoracic centre where he underwent successful aortic valve replacement.

Discussion

Anti-TNF-a agents are increasingly used by rheumatologists for the treatment of rheumatoid arthritis, ankylosing spondylitis and psoriatic arthropathy whilst dermatologists and gastroenterologists also use them in plaque psoriasis and inflammatory bowel disease respectively. It is recognised that by modulating immune response patients on anti-TNF-a become more susceptible to infection.

In a series of 5562 patients receiving anti-TNF-a therapy in Australia and New Zealand from 1999–2009,4 14 cases of serious infections were identified, half of them occurring within the first 6 months of anti-TNF-a initiation.

There is further evidence from European registries that opportunistic infections are more common within the first six months of therapy initiation.5 Appreciating this risk,
patients and physicians alike should be more vigilant for signs of infection during this initial period. Increased awareness should enable early diagnosis and appropriate treatment.

This is the first report of confirmed infective endocarditis in a patient receiving adalimumab. It is likely that the combination of a structurally abnormal valve and immunosuppression made him more susceptible to valvular infection. We would therefore recommend full cardiovascular examination in all patients prior to initiation of anti-TNF-α treatment and referral for echocardiography if any abnormalities are detected or if patients had previous valvular cardiac history. This will enable risk-stratification for infective endocarditis and prompt advice for improvement in dental hygiene- the only policy known to reduce endocarditis risk.⁶

In addition, where echocardiography is performed to assess LV systolic function one should look for any valvular abnormalities as well. Furthermore, the diagnosis of infective endocarditis should be considered both in primary and secondary care in patients receiving anti-TNF-α treatment presenting with signs of infection if no clear source is identified, particularly within the first six months of therapy initiation.

Author information: Vassilis Vassiliou, Clinical Supervisor, Department of Medicine, University of Cambridge, Cambridge, UK; Eleana Ntatsaki, Specialist Registrar in Rheumatology, Department of Rheumatology, Norfolk and Norwich University Hospital, Norwich, UK

Correspondence: Dr Vassilis Vassiliou, 3 Ventress Farm Court, Cambridge, CB1 8HD, United Kingdom. Email: vassiliou@doctors.org.uk

References:

How safe is the ‘safe triangle’?

Sisira Jayathissa, Stephen Dee

Flexible small-bore chest tubes are commonly used to drain large pleural effusions, as they are easy to insert by the Seldinger technique and comfortable for the patient. The commonly recommended site for insertion of a chest drain is the ‘safe triangle’, formed anteriorly by the lateral border of the pectoralis major, laterally by the lateral border of the latismus dorsi, inferiorly by the line of the 5th intercostal space and superiorly by the base of the axilla (as shown in Figure 1). This position minimises the risk to blood vessels, muscle and breast tissue.

Figure 1. ‘Safe Triangle’ (reproduced with permission from the BTS guidelines on pleural procedures 2010)\textsuperscript{11}

Perforation of internal organs is a rare but recognised complication after chest drain insertion. We present a report of a patient who developed myocardial perforation after insertion of a small-bore, flexible chest drain through the ‘safe triangle’ by the Seldinger technique, and we suggest improvements for safety.
Case report

An 82-year-old man was admitted with congestive heart failure and bacterial endocarditis. He had dilated cardiomyopathy, mild renal impairment, chronic leg ulcers and *Staphylococcus* bacteraemia. A CT scan showed a large left sided pleural effusion with partial left lower lobe collapse and consolidation. The patient’s chest X-ray is shown in Figure 2.

**Figure 2. Chest X-ray prior to the procedure showing evidence of pleural effusion and cardiomegaly**

The patient was breathless at rest, even after treatment with intravenous frusemide, spironolactone and bendrofluazide. The results of the diagnostic pleural tap were consistent with a para-pneumonic effusion. A chest drain was offered to the patient in an attempt to alleviate his breathlessness.

The ‘safe triangle’ was identified, and a 14F Rocket chest drain was inserted. Soon after insertion, blood was seen pulsing into the draining system. Cardiac perforation was suspected, and the tube was clamped. A CT chest scan revealed the chest drain in the left ventricle (Figure 3). The patient was transferred to the thoracic surgery unit and the drain was successfully removed by a mini-thoracotomy. The patient was discharged home 2 months later.
Figure 3. Showing chest drain in the left ventricle and pleural effusion and partial collapse of the lung

Discussion

Chest tube insertion is a relatively common procedure, often carried out by junior medical staff, but it is associated with significant complications.\(^1\)\(^-\)\(^9\) During a 3-year period, 12 deaths and 15 cases of serious harm were reported to the National Patient Safety Agency,\(^1\) and the true incidence of significant complications is thought to be substantially higher.

The main complications associated with chest drain insertion include bleeding and haemothorax due to intercostal artery perforation, perforation of visceral organs and major vascular structures, intercostal neuralgia, subcutaneous emphysema, re-expansion pulmonary oedema, infection of drainage site, pneumonia and empyema.\(^2\)

Only a few cases of cardiac perforation have been reported after insertion of small-bore chest drains; the insertion technique was not described in detail, and various types of drain tubes were used.\(^3\)\(^-\)\(^5\) The drains were successfully removed by mini thoracotomy, and patients made a good clinical recovery. Contributing factors in the cardiac perforations were distorted anatomy of the chest, insertion below the ‘safe triangle’ and not using Doppler ultrasound. Kerger et al reported a case of left atrial perforation after insertion of a Matthy catheter through a right-sided chest drain.\(^3\) In that case, neither ultrasound nor CXR picked up the extent of cardiomegaly.

Griffiths et al. conducted an audit to determine the ability of 55 junior doctors to insert a chest drain safely.\(^6\) The doctors were asked to mark on a photograph where they would insert a chest drain for a pneumothorax in a non-emergency situation. An area outside the ‘triangle of safety’ was marked by 45% of the junior doctors surveyed. Ball et al. conducted a retrospective audit of complications from chest drains among trauma patients presenting to an Auckland hospital.\(^7\) The overall complication rate was 40%; surgical residents had a lower complication rate than medical residents.

Two registrars who were involved in the procedure had inserted more than 10 chest drains each, which was higher than the average experience reported in a previous New Zealand study.\(^8\) The registrar who performed the procedure had been unable to attend
a chest drain insertion tutorial. The tube was inserted in the fifth intercostal space over the anterior axillary line, within the boundaries of the “safe triangle” and the drain position was subsequently confirmed by 3D reconstruction. It is likely that the massive cardiomegaly and flabby ventricular wall contributed to the cardiac perforation. The extent of the cardiomegally was not appreciated by the registrars performing the procedure.

An independent physician reviewed the case and identified some shortcomings, including failure to perform radiological imaging immediately before the procedure and lack of a system for monitoring attendance at a practical simulation session of chest drain insertion for medical registrars at the beginning of the rotation. It also advocated greater use of ultrasound and highlighted the trend toward routine use of pleural ultrasound internationally. The resulting recommendations were approved by the hospital patient safety group and implemented. The case was reported to the Ministry of Health as a sentinel event.

Cardiac perforation is a very rare complication of chest drain insertion, but the incidence may be higher with blind insertion than with blunt dissection. The Seldinger technique is not better than traditional methods, even though complications appear to be uncommon.

The most recent British Thoracic Society guideline advocates routine use of Doppler ultrasound before insertion of chest drains, and the National Patient Safety Agency has recommended ultrasound before inserting a drain for fluid.9 The current Australasian guidelines make no recommendations for pre-procedural Doppler ultrasound. Bedside Doppler ultrasound can accurately identify fluid collection and blood vessels, resulting in a higher success rate and fewer complications.10

The best site for inserting a chest drain should be determined by real-time Doppler ultrasound rather than by adhering to the ’safe triangle’. Each hospital should develop protocols and teaching for safe chest drain insertion. In our hospital, a lead chest physician is responsible for providing chest drain tutorials, and, since this case, we have made it mandatory for registrars to attend these tutorials. A national or regional strategy is needed to teach the skills for Doppler ultrasound for chest drain placement if this is to be the new standard of care.

In cases of severe cardiomegaly and distorted thoracic anatomy, blind insertion of a chest tube through the ‘safe triangle’ is not safe. Proper training and routine use of Doppler ultrasound for chest drain placement are the only effective means for reducing the risk for internal organ perforation.

Author information: Sisira Jayathissa, Stephen Dee, Consultant Physicians, Hutt Hospital, Hutt Valley District health Board, High Street, Lower Hutt

Correspondence: Dr Sisira Jayathissa, Consultant Physician, Hutt Hospital, Hutt Valley District health Board, High Street, Lower Hutt, 5010, New Zealand. Email: Sisira.Jayathissa@huttvalleydhb.org.nz

References:

Post mortem diagnosis of severe sepsis

Piet Krijtenburg, Harvey B M Fijn, Petra Heutink, Roel Schellaars, Marco Knook, Dries Mulder, Dave H T Tjan

A 69-year-old woman was admitted to our Emergency Room with somnolence, hypotension, dyspnoea and central cyanosis. Vital signs: temperature 38.4°C, blood pressure 70/50 mmHg and heart rate 120/minute. Remaining physical examination was uneventful. Laboratory examination showed a severe metabolic acidosis and high infection parameters. Radiographic investigations revealed no abnormalities. Patient worsened rapidly in hours and developed all signs of severe sepsis with multi-organ dysfunction with septic shock, oliguric renal failure and lactic acidosis.

The patient was intubated and mechanically ventilated. Broad-spectrum antibiotics were started after cultures were taken. Despite adequate fluid resuscitation, correction of electrolyte abnormalities and acidosis, administration of steroids and high doses vasopressors the patient developed progressive refractory circulatory shock with multi-organ failure.

The patient died within 2 hours after admission to the ICU before a clear clinical diagnosis was established. Macroscopically no sepsis focus could be established during the autopsy, however microscopic examination revealed the diagnosis of severe sepsis (Figures 1 and 2) due to bacteraemia and intravascular coagulation.

Figure 1. Gram stain of liver (obj. 100×)

Note: Many Gram-positive coccoid bacteria in the sinusoids. Note that the much larger erythrocytes are also stained.
Figure 2. H&E stain of kidney (obj. 63×)

Note: High magnification of a glomerulus showing intravascular coagulation: thrombotic microangiopathy with amorphous dark pink material in the capillaries (arrows). Inside the capillaries also many Gram-positive coccoid bacteria are present, which most probably caused the intravascular coagulation.

Gram stain of the liver showed Gram-positive coccoid bacteria in the sinusoids and in the kidney many bacteria were present inside the capillaries. Blood cultures taken on admission grew *Staphylococcus aureus*.

Discussion

Infection is a common problem in intensive care medicine and its course can be severe and fulminant. Sometimes the diagnosis is not clear during the ICU stay. Post mortem examination may be beneficial.

Author information: Piet Krijtenburg, Resident Intensive Care¹; Harvey B M Fijn, Resident Intensive Care¹; Petra Heutink, Resident Intensive Care¹; Roel Schellaars, Consultant Anesthesiologist-Intensivist¹; Marco Knook, Consultant Intensivist¹; Dries Mulder, Consultant Clinical Pathologist²; Dave H T Tjan, Consultant Anesthesiologist-Intensivist¹

1. Department of Intensive Care Medicine, Hospital Gelderse Vallei, Ede, the Netherlands

2. Department of Clinical Pathology, Alysio Hospital Arnhem, the Netherlands

Correspondence: Dave H T Tjan MD, Department of Intensive Care Medicine, Hospital Gelderse Vallei, Ede, the Netherlands. Email: TjanD@zgv.nl
Pre-hospital antibiotics for meningococcal disease remains low

The recent media attention following a number of fatal cases of meningococcal disease has raised the issue of pre-hospital care. Early antibiotic administration remains a goal in the care of these seriously ill patients.

Between 1 June 2008 (when the MeNZB vaccination programme was discontinued) and 31 August 2011, 395 notifications for *N. meningitidis* disease were received nationally. Of those cases, 193 were recorded as having seen a doctor before hospital. Of these, 30% were noted to have subsequently received antibiotics in the community; this is about the same as noted during the serogroup B epidemic.¹

There were 28 deaths during the period, of which 13 were seen by a doctor in the community, and only 2 received antibiotics (15%). While the numbers are low this would suggest the delivery of antibiotics prior to admission remains worthwhile.

While acknowledging the limitations of this data set, particularly with regard to severity at presentation, the disease manifestation does appear to influence the likelihood of receiving pre-hospital antibiotics. In the presence of signs of both meningitis and septicaemia, 37% received antibiotics. This was lower for presentations with either meningitis or septicaemia alone (26% and 23% respectively).

Despite the increased education during the epidemic, overall still less than a third of cases receive antibiotics prior to admission. This rapidly progressing disease continues to present a diagnostic challenge and clearly that is one of the challenges to administering antibiotics prior to admission.

More focus on primary care attention to early administration of antibiotics on suspicion of meningococcal disease remains a worthwhile recommendation with such a potentially life-threatening illness.

**Acknowledgements:** We thank Nikki Turner of the School of Population Health, University of Auckland, for her suggestions.

**Bronwyn Morris**
Public Health Registrar

**Don Bandaranayake**
Public Health Physician

Institute of Environmental Science & Research Ltd
Porirua, New Zealand

**Reference:**
Response to letter ‘New Zealand’s shocking diabetes rates can be reduced—9 urgently needed actions’


As professionals in human development, nursing, pathology, health, nutrition, social sciences, and physical and health education, we call for a more nuanced approach to dealing with this complex issue.

We argue that the authors of the letter need to be cautious about where they lay blame and be more modest about their claims in the literature. We note that the actions proposed are focused on reducing healthcare costs and on self-managing citizens, and fail to take into account the social and cultural implications of their proposed actions.

We would argue that health initiatives (particularly those implemented in schools) need to be co-constructed and negotiated within local communities to allow context specific implementation and that all key stakeholders need to be involved in its implementation.¹

We are concerned about the assumptions² being made in the letter. These assumptions include, but are not limited to, the following:

- Obesity is the cause of Type II diabetes;
- Fat people do not exercise nor eat a nutritious diet;
- Non-fat people do eat nutritious diets and exercise;
- Obese people suffer from poor health and earlier death;
- A once fat person has the same health as a never fat person;
- The action points presented will reduce obesity rates in New Zealand.

These assumptions bolster the incorrect belief that weight is a suitable proxy for projecting disease incidence.³

We are also concerned by the limited picture presented in the letter. We believe that the pieces of information the authors chose to include do not present an accurate representation of the situation. For example, the authors note that between 1989 and 1997, the average New Zealander gained 3.2 kg. They failed to note, however, that obesity rates in New Zealand levelled off in 2002 (1997 for Māori adults)⁴.

Furthermore, the authors claim that the estimated healthcare costs associated with obesity are between 2–7% of the annual budget. This statement is drawn from the WHO report⁵, in which 2-7% cost is suggested as an estimated generalisation for all developed countries.

The document also includes country specific information, including Australia, where obesity related healthcare costs are estimated to be less than 2% of the annual budget.
The estimated generalisation is calculated by considering direct costs (diseases associated with obesity; which works only if you ignore that non-obese people develop these diseases as well), intangible costs, and indirect costs. The report also notes that, ‘the highest direct cost category is most likely to be the personal expenditure on weight-loss programmes incurred by overweight and obese individuals’ (p. 81).

Our population's weight-anxiety, caused by the fatphobic discourse, is the driver behind the take-up of weight-loss programmes. Thus combating weight-anxiety will likely reduce expenditure on these ineffective weight-loss programmes and subsequently reduce the so-called 'obesity related healthcare costs'.

Lastly, the authors fail to address the costs associated with creating a hostile environment for fat people. A consideration of costs associated with obesity must take into account the mental health, physical health, social, and economic costs that result from fat people living in an anti-fat environment.

The suggestions provided by the authors provide little evidence that they will reduce obesity rates in New Zealand. In fact, past projects in schools in New Zealand have not necessarily contributed to children’s health and well-being, but rather suggest an acceptance of discourses that are associated with guilt and the self-monitoring of the body.

We argue that the messages of eating well and exercising regularly at first glance seem a relatively “common sense” approach to impacting positively on children’s health and well-being. However, the evidence to date would suggest that many children are marginalised by the introduction of new health imperatives in schools that seem obsessed with promoting healthy food and thin bodies at the expense of other learning that could take place in health and physical education.

Furthermore, there is an ever-increasing demand for schools and other government institutions to implement public health initiatives. If these health initiatives are to be successful then they need to include key stakeholders in the community and ensure that those on the ground implementing these policies have access to professional development and all the resources that they require.

We agree that the issue of having high rates of diabetes in New Zealand needs to be addressed, but the problem with the suggested action points is that they are presented within an obesity pandemic discourse. We would encourage, instead, a discourse of health that is directed at all individuals, instead of a discourse of obesity panic that is directed at an already marginalised group.

There is so much more to be gained by the creation of a culture and health service in which people of all sizes feel safe and our energies are directed towards ensuring that people regardless of size, ethnicity, education and poverty have full access and the knowledge to support a healthy lifestyle.

Cat Pausé  
School of Arts, Development, and Health Education, Massey University, Palmerston North

Seth Brown  
School of Arts, Development and Health Education, Massey University, Palmerston North
Jenny Carryer  
School of Health & Social Services, Massey University, Palmerston North  

Fran Wolber  
Human Nutrition and Physiology, Massey University, Palmerston North  

Lynda Finn  
Author of: Largely Happy - changing your mind about your body 
and Healthy Kids, Happy Kids – better health for larger kids in New Zealand  

Robyn Longhurst  
Geography, School of Social Sciences, University of Waikato, Hamilton  

Lisa Hunter  
Department of Sport and Leisure, University of Waikato, Hamilton  

Katie Fitzpatrick  
School of Critical Studies in Education, University of Auckland, Auckland  

Trudie Cain  
College of Humanities and Social Sciences, Massey University, Auckland  

Lisette Burrows  
School of Physical Education, University of Otago, Dunedin  

Wil Hoverd  
School of Art History, Classics and Religious Studies, Victoria University, Wellington  

Andrew Dickson  
College of Business, Massey University, Wellington  

References:  

Portrayal of tobacco in televised music videos: content analysis and trends

Despite there being a law against tobacco marketing and sponsorship, smoking is frequently (and legally) shown in a range of mass media in New Zealand: children’s television, prime-time television, and popular films. Media exposure of smoking is less amenable to control compared to overt marketing.

Multiple international and New Zealand studies have found evidence that such exposure is likely to be hazardous. For example, a Dunedin birth cohort study found that watching television for more than 2 hours a day during childhood and adolescence was associated with subsequent increased smoking prevalence. Various studies of adolescent attitudes by Auckland researchers also suggest smoking in films is problematic (e.g. McCool et al.). Internationally, smoking portrayal on television and film has also been studied, including one youth focused genre that has not been studied in New Zealand: music videos. We therefore aimed to study smoking portrayals in music videos aimed at a New Zealand youth audience.

**Methods**—As part of a larger study on the portrayal of alcohol in televised music videos (yet to be published), we added in the study of the portrayal of tobacco and smoking. This involved replicating a previous study conducted in 2005, in which one of us was involved in a supervisory role (NW). Key details of the two studies are shown in Table 1.

In the 2010 study, one of us (KS) observed all the recorded television and performed the coding. Where there was uncertainty over the portrayal of alcohol or tobacco content, another team member (NW) also viewed the video and a collective decision was made. We did not repeat an assessment of inter-observer agreement given this was so high with the 2005 study (Kappa = 0.9 for tobacco references and 1.0 for alcohol references). To best reflect level of exposure to viewers we counted every music video, even if the same video had been repeated elsewhere in the sampling.

**Results & Discussion**—The proportion of tobacco content in the music videos was significantly lower in 2010 in the subscriber TV channel than for the same channel in the 2005 study (4.4% vs 8.2%; Table 2). However, this difference was not statistically significant when considering data from all channels. Other significant results were the decline in tobacco content in videos where the main artist was not from New Zealand; and for the decline in tobacco brands shown (Table 2). In the 2010 study there was an average of 0.6 music videos with tobacco content per hour of television viewing time [38 / (4.5h × 14 days)]. This level of exposure is still of concern from a public health perspective given the potential size of the youth audience for televised music videos and when considering multiple other exposure sources (e.g. other television and movies).
Table 1. Channels and timing for this 2010 study compared to the former 2005 study

<table>
<thead>
<tr>
<th>TV channel</th>
<th>Type of channel</th>
<th>Day / time of data collection in the 2005 study (September/October)</th>
<th>2010 study (November/December)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C4 (channel 12 on Sky)</td>
<td>Free-to-air</td>
<td>Monday–Friday: commencing between 1600h to 2030h for a continuous 3-hour period (for 10 days). Weekend: commencing between 0900h and 1400h for a 3-hour period (for 2 days).</td>
<td>Not studied as this channel had undergone substantive changes in content.</td>
</tr>
<tr>
<td>Juice (channel 62 on Sky)</td>
<td>Subscriber TV*</td>
<td>As above</td>
<td>Daily for 14 days (22 November to 5 December) between 4pm and 8.30pm. This was the channel with the highest music video content per hour and with a youth focus.</td>
</tr>
<tr>
<td>TV2</td>
<td>Free-to-air</td>
<td>Weekend: from 1000h to 1200h (for 2 days)</td>
<td>Not studied due to changes in programming (no music videos).</td>
</tr>
</tbody>
</table>

* Except in Auckland where at the time of this study the “Juice” channel was available for free to up to 1.1 million viewers via UHF aerials.

Table 2. Portrayal of tobacco in music videos in 2005 and 2010 on New Zealand television

<table>
<thead>
<tr>
<th>Variables</th>
<th>2005 study</th>
<th>2010 study</th>
<th>Rate ratio (RR) for 2010 vs 2005 result (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeat videos in the sample</td>
<td>47.3%</td>
<td>59.0%</td>
<td>1.25 (1.12–1.38)</td>
</tr>
<tr>
<td>Any visual reference to tobacco*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of channel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free-to-air</td>
<td>3.9%</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Subscriber TV “Juice channel”</td>
<td>8.2%</td>
<td>4.4%</td>
<td>0.54 (0.33–0.89)</td>
</tr>
<tr>
<td>Total</td>
<td>6.0%</td>
<td>4.4%</td>
<td>0.73 (0.47–1.15)</td>
</tr>
<tr>
<td>Genre</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip-hop</td>
<td>10.9%</td>
<td>5.3%</td>
<td>0.49 (0.21–1.18)</td>
</tr>
<tr>
<td>Rock</td>
<td>4.8%</td>
<td>4.0%</td>
<td>0.82 (0.36–1.93)</td>
</tr>
<tr>
<td>Pop</td>
<td>1.4%</td>
<td>3.9%</td>
<td>2.89 (0.62–13.4)</td>
</tr>
<tr>
<td>Rhythm &amp; Blues (R&amp;B)</td>
<td>10.5%</td>
<td>1.4%</td>
<td>0.14 (0.02–1.11)</td>
</tr>
<tr>
<td>Other (including electronic)</td>
<td>7.0%</td>
<td>6.5%</td>
<td>0.92 (0.27–3.21)</td>
</tr>
<tr>
<td>Main artist nationality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td>1.8%</td>
<td>6.5%</td>
<td>3.61 (0.84–15.43)</td>
</tr>
<tr>
<td>Other</td>
<td>7.1%</td>
<td>3.6%</td>
<td>0.51 (0.30–0.86)</td>
</tr>
<tr>
<td>Other specific aspects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarettes/packs etc shownc</td>
<td>Not detailed but it was found that 1.6% had tobacco “present only” and 4.4% had “present and used”</td>
<td>3.1%</td>
<td>27/861</td>
</tr>
<tr>
<td>Probably or definitely tobacco smoke visibled</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco brand / logos present</td>
<td>5.9%</td>
<td>0.0%</td>
<td>0/861</td>
</tr>
</tbody>
</table>

Notes: * Any visual reference to smoking, to tobacco products or visible tobacco smoke; | | |
| Classification of music genre was based on the first-listed genre on the artist’s Wikipedia page (http://www.wikipedia.org/). If the artist/genre was not available on Wikipedia, a Google search was conducted. For collaborative artists, the genre of the first named artist was used; c Included cigarettes, pipes or cigarette holders, & cigarette packs/tobacco pouches, rolling papers, chewing tobacco; d Any smoke that is not from another obvious source eg, candle, fire, vehicle exhaust, fog machine “smoke” etc.
Of note is that there were various differences between the two studies that may limit comparability of the results: the study in 2010 was larger, had a significantly higher proportion of “repeat videos” (Table 2), there were some differences in channels (and timing), and different researchers were involved in the coding. Also future studies would probably need to be larger if differences between music genre were to be explored in more detail.

There are a range of policy options to reduce the tobacco content of mass media in New Zealand e.g:

- The government agency “NZ On Air” could restrict or ban funding for New Zealand-made programmes that have tobacco content.
- The government could require TV programmes broadcast in New Zealand to be rated by presence/absence of tobacco content.\(^{11}\)\(^{12}\)
- The government could restrict any programming with tobacco content to late evening hours (as per current restrictions on alcohol advertising on NZ television).
- The government could disregard the above options but could compensate by strengthening other forms of tobacco control—particularly around further increases in tobacco taxes and restricting the availability of tobacco products.

**Conclusion**—In both these studies, there was tobacco content at a level of public health concern—particularly given the government’s goal of a “Smokefree Nation 2025”. However, policy makers do have a range of plausible options to address the problem of tobacco displays in the mass media.

**Competing interests:** Nil.

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Nick Wilson, Kate Sloane, Fiona Imlach Gunasekara, George Thomson
Department of Public Health, University of Otago, Wellington, New Zealand
nick.wilson@otago.ac.nz

**References:**

Response to review article by Ernst and Posadzki on spinal manipulation

The recent ambitious effort by Professor Edzard Ernst [retired] and Paul Posadzki of the Peninsula Medical School, Universities of Exeter & Plymouth, Exeter, UK, to critique Spinal Manipulation (SM)\textsuperscript{1} is fraught with avoidable weaknesses and is therefore more fragile and limited than might appear at first glance.

For example there are significant methodological errors in this particular study, including:

- No attempt made to ensure that the definition of "spinal manipulation" (SM) between the studies was compatible.
- No attempt to demonstrate, from existing literature, that SM provided by different providers using different specific physical techniques is comparable.
- No attempt to demonstrate the validity of the quite unusual systematic review approach, where a heterogeneous treatment modality (SM) is assessed against multiple different conditions each with different aetiologies.

Inherent weakness in this study also comes from the fact that 13 of the systematic reviews (approximately one-third of those cited) that have been reviewed come from their own research unit. The authors do admit that this may affect the independence of the study and, as a result, this data is skewed by their own publications.

In addition there is contradiction within the article:

"Collectively these data fail to demonstrate convincingly that spinal manipulation is an effective intervention for any condition."

Yet in the abstract it is stated that there were positive conclusions for psychological outcomes (n=1) and whiplash (n=1).


There are numerous articles that support the use of SM in a range of conditions\textsuperscript{3–6} [by way of example]. The most recent being by Senna, Mohammed K. MD; Machaly, Shereen A. MD, and published in Spine.\textsuperscript{5}

This article concludes:

"SMT is effective for the treatment of chronic non-specific LBP. To obtain long-term benefit, this study suggests maintenance SM after the initial intensive manipulative therapy."
It appears facile to attempt to discredit a modality when there is an expanding body of evidence that provides support.

Patients often choose practitioners who utilise SM approaches because other standard medical approaches have failed, or they are unwilling to undergo a surgical option. Therefore one rationale for the use of SM is that for those for whom standard medical treatments are not satisfactory; SM provides an excellent backstop option.

Prof Ernst has been repeatedly criticised for using references inaccurately, with the apparent intent to mislead. In doing so he compromised the integrity of the scientific reporting and the validity of his own research. This article is just another example of the misuse of scientific data.

Professor Ernst has long demonstrated extreme bias against spinal manual therapy and chiropractic. His writings often fall well short of good peer-review standards.

Writing in the BMJ in 1999, Dr Gordon Waddell, a leading UK orthopaedic surgeon and back pain authority, described Ernst as offering “inter-professional confrontation under the guise of scientific objectivity.”

It would appear that Professor Ernst’s perspective hasn’t changed despite the growing body of evidence to refute it.

Corrian Poelsma
President
New Zealand Chiropractors Association

References:
Is colorectal cancer preventable?

Back in 1975, Englishman Richard Doll, who earlier had found the link between smoking and lung cancer, published a paper in the *International Journal of Cancer*\(^1\) which included the following graph.

This graph shows the astonishing variation in prevalence of colorectal cancer (CRC) between nations, a variation which cannot easily be attributed to genetic variations between nations. It also shows the striking link between meat consumption and CRC.

Since 1975 there has been an increasing volume of research on the role of meat with cancer. While many studies have linked meat intake to increased mortality from cancer, the nature of the association wasn’t well defined. To explore this association, two landmark studies were initiated, one in Europe, the other in the United States. So as to maximise the validity of the ensuing statistics, unprecedented numbers of people were recruited.

In Europe, starting in 1992, 520,000 people were recruited from 10 nations: Denmark, France, Germany, Greece, Italy, the Netherlands, Norway, Spain, Sweden and the
UK. The study became the *European Prospective Investigation into Cancer and Nutrition (EPIC).*

Detailed information on diet and lifestyle was obtained by questionnaires, by physical measurements and by blood samples. After studying people in different countries, with widely differing diets, EPIC has been able to produce much more specific information about the effect of diet on long-term health than any previous study.

In the US, starting in 1995, 550,000 people were recruited by the National Cancer Institute (NCI) from members of the American Association of Retired Persons (AARP) into the *NCI-AARP Diet and Health Study.* The NCI is one of the prestigious US National Institutes of Health. The AARP is an organisation dedicated to enhancing quality of life for all as we age. Questionnaires similar to those used in Europe were used by the NCI.

The EPIC and NCI-AARP trials illustrate the pre-eminent role of epidemiology in modern medical research. (Epidemiology being the study of linkages between lifestyle factors, with differing patterns of disease in differing populations or groups.) Just as the cigarette was found by epidemiology to be the cause of lung cancer, and just as the three primary risk factors of coronary disease—cholesterol, smoking and hypertension—were found by epidemiology, EPIC and NCI-AARP have identified two primary risk factors for colorectal cancer: excessive meat consumption and fibre deficiency. EPIC and the NCI-AARP are ongoing studies, and both produce periodic reports which are made available to the people of the world, online.

**Key results:**

- The hypothesis that a diet high in fibre reduces CRC risk has been corroborated by both studies.
- The hypothesis that consumption of red and processed meat (sausages, bacon, salami, ham etc) increases CRC risk, while eating fish decreases risk, is strongly supported by the studies.
- The dietary combination of too little fibre and fish, but excessive red and processed meats, plays a major role in causing colorectal cancer.
- Elevated risks, ranging from 20% to 60%, were evident for oesophageal, colorectal, liver, and lung cancer, comparing individuals in the highest group of meat intake, with those in the lowest group.

These key results beg the key question. Why meat? The lead author of the NCI-AARP study, Dr Rashmi Sinha, addressed that question as follows:

“There are various mechanisms by which meat may be related to mortality. Meat is a source of several carcinogens, including chemicals which are formed when meat is cooked at high temperatures. Iron in red meat may also increase the formation of carcinogenic compounds. Furthermore, meat is a major source of saturated fat, which has been shown to increase the risk of breast and colorectal cancer.”

The implications for the New Zealand public are very important. The implications are of particular importance for those with a family history of bowel cancer. Most of these families have been led to believe that they have a genetic predisposition to this
cancer. The wide international variation in prevalence revealed by the graph, however, complimented by the EPIC and NCI-AARP data, strongly suggests that excessive meat consumption and dietary fibre deficiency are the dominant causes of this cancer; and that inheritance, in general, plays a minor role.

The recipe to avoid this cancer is simple. In practice, however, the recipe—less meat, and less meat products, together with more fibre and more fish—is anything but simple for many New Zealanders. From the time when the earliest European settlers arrived, meat has been regarded as the healthiest of food. Some view it, quite wrongly, as an essential food. Reducing meat consumption is no great hardship. Chicken and fish are acceptable alternatives, as is eating meat on fewer days each week.

Increasing fibre, on the other hand, would be a very high hurdle for some. While many women know about fibre, and believe they eat lots of it, the reality is that on average, they probably eat less than 50% of what their bodies need for optimum health (30–40 g/day). Worse, many of the products sold in supermarkets contain woefully minute amounts of fibre. (One cup of the popular Nutri-Grain cereal contains just 1, pitiful, gram [g], of fibre).

Lettuce-based salads are seen by many as a valuable source of fibre. But they’re not. A 500 g lettuce contains only 3 g of fibre. And that’s not all. While some earlier studies suggested that fruit and vegetables give a degree of protection against CRC, recent results fail to confirm this. In February 2010, an analysis of the EPIC data involving 142,000 men, and 335,000 women, revealed the protection from fruit and vegetables to be so minimal as to be almost irrelevant.

The essential point in this, relates not only to the amount of fibre in foods, but to the type of fibre. The fibre in wholemeal bread, fibre-rich cereals, beans, lentils, and peas, root vegetables including potatoes and parsnips, is likely to provide protection against CRC, whereas that in fruit and leafy vegetables may not. Unfortunately, the public has been persuaded by quack ‘nutritionists’, that wholemeal breads and root vegetables will make people put on weight, and should be avoided. The consequence of this woefully ignorant advice, is that many people are instead, eating and snacking on fibre-depleted carbohydrates in their purest (and most fattening) forms—anything made with white flour, sugar, and fat.

Checking the fibre content on food packaging, because of the tiny print used, can be very tedious. Far better is to get a brochure listing fibre and fat details. And now, to return to my original question, Is colorectal cancer preventable. I believe the EPIC and NCI-AARP evidence shows that for the vast majority of people, it is.

But will the data emerging from these momentous, authoritative studies reduce our dreadful CRC stats? In my opinion, probably not, at least not in the short-term. Following the publication in 1954 of the cause of lung cancer, few doctors took note. It wasn’t until the late 1970s—a quarter of a century later—that the medical profession (including me) woke up to the enormity of the danger posed by the cigarette.

Later, when the evidence began to show the causal role of cholesterol in coronary disease, it became fashionable, in some medical circles, to dismiss cholesterol as “controversial and unproven”. The result was that the aggressive management of
raised cholesterol levels was delayed by two decades, during which probably many hundreds, if not thousands, of Kiwis died each year from heart attacks, which today would be prevented.

Astonishingly, the EPIC and the NCI-AARP trials have yet to register in this country. I’ve seen no mention of them in any New Zealand medical journal, or in conference programs, or in the website of the Cancer Society. Colonoscopy is widely advocated, but where is the intellectual rigour of diagnosing and treating a disease, without at the same time, attempting to remove its cause?

What then, should a responsible medical community do? Two things:

1. State emphatically that, just like the link between cigarettes and lung cancer, and just like the link between elevated blood cholesterol and coronary heart disease, there is a strong scientific link between eating too much meat and not enough dietary fibre and fish, and the subsequent risk for getting bowel cancer.

2. Thus bowel cancer should be regarded as substantially a preventable disease, and measures should be used to publicise this, and to guide New Zealanders on how to prevent this cancer.

Finally, along with a colonoscopy screening policy to detect bowel cancer, there should be active govt involvement in prevention of this cancer, similar to the programs which have been embraced for smoking cessation and for prevention of coronary artery disease.

Dr Michael Cooper
St Heliers
Auckland, New Zealand

References:


A case of fracture of the patella by indirect violence without separation of the fragments

Written by A. Clark, R.R.C.S., Cd., Hon. Radiologist, Auckland Hospital, and published in NZMJ May 1912;11(42):134.

The patient H. T. walked into my consulting room on October 27th, 1911, limping slightly and complaining that he had slipped at his work on October 20th and had sprained his knee in trying to save himself from falling.

The knee joint was considerably swollen, and on examining the Patella a transverse ridge could be felt and soft crepitus elicited. The patient told me that he had been getting about without a stick, but with a good deal of pain, since the accident.

A radiogram showed the Patella to be transversely fractured without any separation of the fragments. The patient was sent to bed with the leg on a back splint until December 12th, when I put a removable plaster of Paris splint on and allowed him to get about on crutches, and to have massage.

Early in January he discarded the crutches for a stick and returned to work on January 26th 1912.

The case is interesting in, the aspects:—(a.) That the fragments were not separated although the lesion was due to indirect violence. (b.) That the patient was able to walk home after the accident and a week later to my room.
Advancing women’s heart health through improved research, diagnosis and treatment

Recently two United States (US) organisations presented a report which draws attention to the burden of cardiovascular disease in women and the disappointing research into this predicament.

The report points out that in the US women are twice as likely as men to have heart failure, 1.5 times more likely to die with a year of a heart attack, and twice as likely to have a poor outcome after a coronary artery bypass graft. And there are the gender-specific pregnancy-related complications (e.g. gestational diabetes, hypertension, pre-eclampsia) which significantly increase cardiovascular disease later.

The report also makes the point that women are under-represented in clinical trials. We hope that the report has the desired effect—more attention to women’s cardiovascular problems.


Rivaroxaban versus warfarin in subjects with nonvalvular atrial fibrillation

The use of warfarin reduces the rate of ischaemic stroke in patients with atrial fibrillation (AF) by two-thirds. However, its usage is impeded by the need for frequent monitoring and dose adjustment and the risks of unwanted haemorrhage.

Recently PHARMAC has approved the use of dabigatran as an alternative to warfarin (see abstract NZMJ 8 July 2011). This report concerns the use of rivaroxaban, an oral factor Xa inhibitor, which may provide more consistent and predictable anticoagulation than warfarin. Over 14,000 patients at risk were randomised to receive either rivaroxaban (at a daily dose of 20 mg) or dose-adjusted warfarin. Rivaroxaban was shown to be non-inferior to warfarin as a stroke preventer. There was no significant between-group difference in the risk of major bleeding, although intracranial and fatal bleeding occurred less frequently in the rivaroxaban.

So both dabigatran and rivaroxaban provide an alternative to warfarin without the need for monitoring and dose adjustment. However, as an editorial points out, there is no antidote to either of these new drugs if bleeding occurs.


Dystextia—a very modern neurological problem

The authors of this case report point out that formulating and sending a text message requires complex motor, visual and language skills and involves coordinated function of several areas of the brain and could be affected by stroke or transient neurological disturbance.
They report on a patient who presented with a headache, dressing apraxia and expressive dysphasia, the latter not only verbal but also manifest as an inability to send a text message on his phone. Investigations were negative, the patient recovered and a diagnosis of complex migraine was made.

A literature search produced one relevant paper in the Irish Med J (2006;99:157). The authors of that paper reported on the symptom in a patient who suffered an internal capsule infarct and they coined the expression—dystextia.

Int Med J 2011;41:646.

Podiatry intervention to prevent falls in community dwelling older people with disabling foot pain

Older people with foot pain are known to fall more frequently than healthier folk. The report concerns a randomised study on 305 elderly men and women with foot pain. Half of them received a podiatry intervention consisting of foot orthoses, footwear advice, a footwear subsidy, a home based exercise foot and ankle exercise programme, a falls prevention education booklet, and routine podiatry care for 12 months. The primary outcomes were falls and injuries and at 1 year the intervention group had 36% less falls. One of the intervention group had a fall with a fracture compared with 7 in the control group.

As expected the intervention was very useful. The researchers speculate whether the intervention would be effective in residential care settings or in older people without foot pain. Very likely I think.

BMJ 2011;342:d3411.

Does comprehensive geriatric assessment improve outcomes in frail older people being discharged from acute hospital care?

The authors of this study note that many frail older people who attend acute hospital settings and who are discharged home within short periods (up to 72 h) have poor outcomes. We would expect that a comprehensive geriatric assessment (CGA) of such patients would be beneficial in many respects such as lowered mortality rates and need for hospital readmission.

Hence this systematic review of relevant randomised trial evidence. They found only five trials of sufficient quality. They report that there was no clear evidence of benefit for CGA interventions in this population in terms of mortality or readmissions or for subsequent institutionalisation, functional ability, quality-of-life or cognition.

Disappointing. They recommend more higher quality studies. Should their hypothesis be proved, the need for geriatric physicians would escalate significantly.

Peter John Little

MB ChB, FRACP, FRCP; Head of the Department of Nephrology, Christchurch Hospital 1967–1979; President of the Australasian Society of Nephrology 1976–1978

Peter Little was born in Hastings and attended St John’s High School, Hastings, where he played in the Rugby First XV and was Dux of School in 1947.

He developed a sense of justice at a young age influenced by his Catholic upbringing and education, family experiences during the Depression, and work in the freezing works during university holidays.

After graduation from the University of Otago in 1954, Peter worked in Napier and Wairau Hospitals before trying his hand at general practice in Napier.

He married Cynthia, an English-trained nurse, in 1957 and they subsequently had 5 children. Peter found he was unsuited for general practice and, with Cynthia’s encouragement, went to the UK for further medical experience in 1958.

He was fortunate to be working as a locum medical registrar for Professor Hugh de Wardener, a pioneer of nephrology, at the Fulham Hospital, London in 1960 and decided to become a nephrologist at a time when the subspecialty was just emerging following the establishment of the feasibility of maintenance dialysis.

Peter worked first as a research fellow at the old Charing Cross Hospital from 1961 to 1963 and then as a Lecturer in Medicine for the Charing Cross Hospital Medical School at Fulham Hospital from 1963 to 1966. Peter experienced the challenges of the early days of dialysis and through his clinical research made significant contributions to the understanding of the pathophysiology of urinary tract infections. He was elected to membership of the Medical Research Society and Renal Association of Great Britain.

In 1966 he was appointed as Canterbury’s first nephrologist to set up a renal unit at Christchurch Hospital. Peter was the Head of Department from 1967 to 1979. In 1969, he established the first home dialysis programme in New Zealand and, in 1972, the regional kidney transplantation programme. Peter recognised that the hospital service would never have the resources to meet the demands for dialysis treatment. He decided that he would offer only home dialysis and return patients to the care of their general practitioner. Peter was ahead of time in recognising the need to involve the general practitioner in the care of patients with chronic illness.
Peter was active in the Australasian Society of Nephrology (now Australian and New Zealand Society of Nephrology) and its President from 1976 to 1978.

Pete Little’s views on the need to remove barriers to transplantation, particularly from living donors, were developed from his experiences working as a nephrologist in the Middle East. He was outspoken in his criticism of what he thought were unhelpful and unrealistic positions taken by ethics and medical groups such as The Transplantation Society.

Peter’s work in Baghdad, Iraq gave him a unique perspective on the plight of the ordinary Iraqi, including his patients, after the two Gulf Wars. He was critical of the Western stance on the need to remove Saddam Hussein and correctly predicted the civil unrest that followed the end of the military phase of the West’s involvement.

Peter will be remembered as quite a character. Having a great intellect and being a clear analytical thinker, he did not always get on with the establishment but he was held in high esteem by patients and doctors in training. He had strong socialist convictions and was always one to support the underdog. Peter was an excellent cook and gourmet, enjoyed international travel and was well known for his flamboyant dress and trademark bowtie.

Peter left Christchurch in 1979 to establish and head a renal unit in Saudi Arabia and subsequently worked in Baghdad, where he developed a large, successful living donor kidney transplant programme. He returned to New Zealand to live in retirement but continued to travel, particularly to Ireland and Thailand.

He is survived by his second wife, Dolores, and his 5 children.

Kelvin Lynn (Medical Director, Kidney Health New Zealand, Christchurch) wrote this obituary.
Medical Benevolent Fund

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

Applications should be directed through the NZMA:

Central Office
P O Box 156
Wellington
Tel: 0800 656161