THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

CONTENTS

This Issue in the Journal

Editorials

Original Articles

Case Reports

Viewpoint

100 Years Ago in the NZMJ

Medical Image

Methuselah

Letters

Obituary

Notice

Book Reviews

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

This Issue in the Journal

Benzylpiperizine-based party pills' impact on the Auckland City Hospital Emergency Department Overdose Database (2002–2004) compared with ecstasy (MDMA or methylene dioxymethamphetamine), gamma hydroxybutyrate (GHB), amphetamines, cocaine, and alcohol
L Theron, K Jansen, J Miles

There has been recent interest in possible harms resulting from 'party pills.' One way to assess this is by examining the impact on hospital emergency departments. This study showed that rising use of 'party pills' had relatively little impact on the Auckland Hospital Emergency Department between 2002 and 2004. Amongst the controlled drugs in the study, GHB ('Fantasy') was by far the most serious problem, accounting for 11%, in 2002, of the total adverse reaction/overdoses for these substances. After GHB became illegal, the level fell significantly (to 6% by 2004). Over the same period, the adverse reactions due to party pills rose significantly to 1.6%, still a low figure. There is often much debate about whether prohibition works or not. These results suggest the possibility that prohibition may well have made a positive contribution to public health in the case of GHB, although there may be other explanations. Alcohol was always responsible for the lion's share of adverse reactions (54% in 2002 and 61% in 2004.)

Screening and intervention for alcohol problems among patients admitted following unintentional injury: a missed opportunity?
J Hosking, S Ameratunga, C Bullen, I Civil, A Ng, A Rodgers

Alcohol misuse is a potent risk factor for injury, and injured patients admitted to hospital who have alcohol problems are at high risk of future injury. However, there is currently no formal screening programme in New Zealand hospitals for detecting alcohol problems or their harmful consequences. This review of a trauma registry database found that while the records of 68% of patients admitted following an unintentional injury had a comment on alcohol use, only 7% recorded information that suggested a possible drinking problem, and few documented implementing an alcohol intervention. Effective approaches to screen and manage alcohol-related problems in the hospital setting require greater attention.

Vincristine for the treatment of Kasabach-Merritt syndrome: recent New Zealand case experience
K Thomson, R Pinnock, L Teague, R Johnson, N Manikkam, R Drake

A discussion of four cases where a chemotherapy medication is used successfully to treat the rare but life-threatening childhood disorder, Kasabach-Merritt Syndrome. The article discusses the benefits of this treatment when compared to steroid medications, which have been used traditionally.

Concussion clinic referral demographics and recommendations: a retrospective analysis
H Alexander, N Shelton, J Fairhall, H McNaughton

This study reviewed patients who were thought to have received head injuries and who were admitted to a Wellington area concussion clinic during a 2-year period. Data was collected via an electronic database and written clinical records. 42 (26%) patients were diagnosed as not having a mild traumatic brain injury (TBI). Of the remainder, 72 (47%) had a mild TBI and 36 (22%) had moderate or severe TBI. 21% of attendees were injured in sporting accidents with 19% injured in motor vehicle accidents and 17% in falls. Occupational therapy was the most commonly recommended treatment. Further research is required into return to work, emotional, and cognitive outcomes.

General practitioner diagnosis and management of acute knee injuries: summary of an evidence-based guideline
G Robb, D Reid, B Arroll, R Jackson, F Goodyear-Smith

This guideline summary summarises the evidence and key recommendations of the New Zealand guideline "The diagnosis and management of soft tissue knee injuries: Internal Derangements" Although there is a lack of robust evidence, it does offer primary healthcare providers clear guidance for diagnosing and managing these injuries and for appropriate and timely referral to secondary care providers.

Consumers' knowledge, perceptions, and responsiveness to direct-to-consumer advertising of prescription medicines
J Hoek, N Maubach

Supporters of prescription medicine advertising (DTCA) argue this increases the health knowledge of individuals who consider themselves are less knowledgeable about their health and who have a lower self-reported health status. This study examined this argument. The results suggest that consumers most likely to benefit from increased knowledge find DTCA more confusing and are less likely to seek further information. This finding questions whether DTCA enhances awareness of health issues or helps individuals to overcome barriers to better health.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Party on? BZP party pills in New Zealand

Paul Gee, John Fountain

The need to become intoxicated with mind-altering substances is probably older than civilisation. The history of organised drug control is more recent, and follows general patterns: if a particular psychoactive substance is (ab)used and causes sufficient adverse health or social consequences, a public outcry will result in regulation. Alternately, concern regarding recognised ill-effects of a substance produces immediate or pre-emptive regulation. Indeed, such laws can appear quite confusing: we see legal use of tobacco (responsible for almost 5000 deaths annually in New Zealand), and all are aware of the personal and societal woes of ethanol abuse. It would seem that having escaped 'Pandora's box' some psychoactive substances are then very difficult to return.

Due to their stimulant and euphoric properties, piperazine party pills (PPPs), also known as "herbal highs" or "social tonics", have become increasingly popular in New Zealand since 1999. The main ingredient is 1-benzylpiperazine (BZP), a synthetic chemical originally developed as an antihelmintic. This compound was investigated as a potential antidepressant medication, but rejected when research reported that BZP had amphetamine-like effects and was liable to abuse.[1] This same research concluded that BZP "should be placed under statutory control similar to those regulating the use of amphetamine." Indeed, BZP is banned in the USA, Japan, Australia, Denmark, and Sweden.

In New Zealand, however, PPPs initially enjoyed a complete lack of regulation; and when speciously promoted as "herbal" or "natural", quickly became popular. The industry projects than an estimated 5 million "servings" will be sold in 2007.[2]

In March 2004, the Expert Advisory Committee on Drugs (EACD) met to consider BZP and related substances and to formulate advice for the Associate Minister of Health (The Hon Jim Anderton). After review of the available evidence, the Committee was unable to find sufficient evidence to justify a complete ban. Their report makes interesting reading and included the recommendation that a new category of classification be added to the Misuse of Drugs Act 1975 for "restricted substances":[3] those compounds not considered a high or moderate health risk, but in need of regulation. They were advised that PPPs were being marketed as a 'legal' alternative to illicit substances and that substitution was occurring allowing users to "exit the illicit market". There was, however, no reference identifying the source of this comment, nor one to substantiate the claim.

The Government moved quickly and created a new schedule limiting the sale of PPPs to those 18 years and older; restricted advertising, packaging, and labelling; and required package health warnings.[4] At the same time, research projects were commissioned to provide evidence of safety, or harm, in humans.

In this issue of the *Journal*, Theron et al (http://www.nzma.org.nz/journal/120-1249/2416) have placed the impact of PPPs in context by comparing the number of presentations to Auckland City Hospital Emergency Department (ED) resulting from PPP use during 2002–2004 with alcohol and various illicit drugs. The results show a

significant increase in presentations due to PPPs over the time period, but little impact on illicit drug presentations to the emergency department overall. In general terms, the harm (only one admission) appeared low, however the prevalence of BZP use in the community during the study period is unknown and predates the boom in PPP sales. Their results did not indicate any widespread substitution of ecstasy and/or amphetamine by PPPs.

A previously published review of emergency department attendances due to BZP exposures raised initial safety concerns. Patients presenting to Christchurch ED had a 15% rate of seizure, with two patients requiring ventilatory support and intensive care. Patients with less serious reactions had collapse, confusion, hyponatraemia, headache, vomiting, and psychological symptoms. A 50% rate of admission was reported.[5] This ED's experience may have been influenced by a different drug taking culture or reflected different retail marketing. These factors are impossible to quantify, however serious harm was suffered by many recreational PPP users.

In November 2006, the EACD met to again consider the regulatory status of BZP, and reviewed further relevant information including: research reports, preliminary research reports, email correspondence, and a Coroner's findings.[6] The minutes noted that: PPP use had risen quickly, with one in five adults aged 13 to 45 years having tried PPPs, and almost twice this incidence for those aged 20 to 24 years;[7] dose and warning labelling were ineffective; and that BZP was now being used in intravenous form. Also highlighted was a high rate of adverse effects, with severe effects occurring unpredictably and at relatively low dose. Of note was a statement by the National Drug Intelligence Bureau and Customs Service that there was no evidence of a levelling or decline in use of methamphetamine, despite party pill industry claims to the contrary.

The Committee subsequently recommended to the Associate Health Minister that BZP be re-classified under schedule 3, Part 1 (Class C1) of the Misuse of Drugs Act 1975: illegal substances that are deemed to pose a moderate risk of harm and have no therapeutic purpose. This recommendation is now subject to a consultation process scheduled for completion by 31 March 2007, prior to submission to the Health Select Committee.

The EACD also raised for the Associate Minister's consideration the question of "whether New Zealand wishes to have a legal market for psychoactive drugs". This was regarded as "a key policy issue that needs an explicit decision".

Indeed, such a decision is profound: are we to adopt enlightened legislation, or establish 'laboratory New Zealand'. BZP is but one of many new psychoactive substances that have been introduced to New Zealand through 'grey market globalisation'.

Psychoactive plant use exists in many indigenous cultures in the world, and retailers are importing and selling these plant materials from Asia and South America into an unregulated New Zealand market. Many novel designer drugs exist that are not covered by existing drug legislation, and very little formal work has been done on their pharmacology. Indeed, a legal market for psychoactive substances could release an unknown number of the burgeoning range of little understood psychoactive compounds, and the impact on public health would be difficult to predict.

Interestingly, in the EACD's initial 2004 review, the safety of BZP is not established by the Committee; rather, a seeming lack of toxicity. This is the reverse of standards applied to pharmaceuticals where safety must be established. Further, there has been no formal national monitoring for adverse reactions to BZP similar to that carried out by the New Zealand Pharmacovigilance Centre for medicines. However, a range of research studies (a number supported by Government) have been undertaken.

The results from those studies so far published,[8,9] including Theron et al's, are interesting. If such high levels of adverse reactions were observed with a prescription medicine, it is likely the drug would be withdrawn. Are we to accept a higher level of adverse health effects related to restricted substances? Is it necessary to await an outcome such as severe illness, neurological damage, or death before risk of harm is established? And if so, why, given the lack of therapeutic benefit of restricted substances?

For a medicine, exhaustive trials are required to establish safety prior to clinical use. Indeed, as we have seen with drugs from thalidomide to the COX-2 inhibitors, even after pre-marketing testing, safety issues may not be elicited for some extended time post-release. Yet restricted substances are currently not subject to the same rigorous 'pre-clinical' testing as prescription medicines—quality control of production is not currently formally monitored, and there is no established post-marketing surveillance regimen despite the millions of doses of BZP sold each year.

This would seem a fraught situation, given that compounds classified as restricted substances are likely to act upon the central nervous system, with attendant potential for a range of harms including: abuse; dependence; subtle, gross or delayed neurological damage; interactions with prescription drugs; and adverse effects upon the unborn.

If a legal market for psychoactive drugs is established in New Zealand, and such compounds are to be let out of 'Pandora's box', a responsible approach must be applied prior to their release. In particular, *safety* (rather than apparent lack of toxicity) must be proven; with the onus to provide scientifically robust evidence placed on the suppliers of these substances. And vigorous standards must be applied and enforced regarding the manufacture, packaging and sale of these substances; and effective ongoing national post-marketing surveillance must be established.

Conflict of interest statement: Paul Gee is the recipient of a National Drug Policy Unit Grant to undertake research further into the effects BZP. John S Fountain has no conflict of interest to declare.

Author information: Paul Gee, Emergency Physician, Christchurch Hospital. John S Fountain, Medical Toxicologist, National Poisons Centre, University of Otago, Dunedin

Correspondence: John S Fountain, National Poisons Centre, University of Otago, PO Box 913, Dunedin 9054, New Zealand. Email: john.fountain@stonebow.otago.ac.nz

References:

1. Campbell H, Cline W, Evans J, et al. Comparison of the effects of dexamphetamine and 1-benzylpiperazine in former addicts. Eur J Clin Pharmacol. 1973;6:170–6.
2. Bowden M. Submission to the Health Select Committee, Misuse of Drugs Amendment Bill (No 3). Social Tonics Association of New Zealand. October 2004.

3. Expert Advisory Committee, National Drug Policy Team. The Expert Advisory Committee on Drugs (EACD) advice to the Minister on: Benzylpiperazine (BZP). Ref. No.: 20045663. April, 2004. URL: http://www.ndp.govt.nz/committees/eacd/papers.html#BZP
4. Misuse of Drugs Amendment Act 2005. URL: http://www.ndp.govt.nz/legalpartypills/documents/misuse-drugs-act-2005.pdf
5. Gee P, Richardson S, Woltersdorf W, Moore G. Toxic effects of BZP-based herbal party pills in humans: a prospective study in Christchurch, New Zealand. N Z Med J. 2005;118(1227). URL: http://www.nzma.org.nz/journal/118-1227/1784
6. Expert Advisory Committee on Drugs, National Drug Policy Team. Minutes: Expert Advisory Committee on Drugs meeting. Ministry of Health, New Zealand. 29 November 2006. URL: http://www.ndp.govt.nz/committees/eacd/minutes/eacdminutes291106.pdf
7. Wilkins C, Girling M, Sweetsur P, et al. Legal party pill use in New Zealand: prevalence of use, availability, health harms and 'gateway effects' of benzylpiperazine (BZP) and trifluoromethylphenylpiperazine (TFMPP). Centre for Social and Health Outcomes Research (SHORE) & Te Ropu Whariki, Massey University; 2006. URL: http://www.ndp.govt.nz/legalpartypills/documents/legal-party-pill-use-nz.pdf
8. Alansari M, Hamilton D. Nephrotoxicity of BZP-based herbal party pills: a New Zealand case report. N Z Med J. 2006;119(1233). URL: http://www.nzma.org.nz/journal/119-1233/1959/
9. Austin H, Monasterio E. Acute psychosis following ingestion of 'Rapture'. Australas Psychiatry 2004;12:406–8.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Through the looking glass: toward a brighter future for long-term care in a greying New Zealand

Mark Booth, Edward Alan Miller, Vincent Mor

Abstract

In the USA, a recent report produced to inform the work of the National Commission for Quality Long-Term Care—*Out of the Shadows: Envisioning a Brighter Future for Long-Term Care in America*—should be of interest not only to US observers, but to policymakers, providers, and users of long-term care within New Zealand.

Despite differences in financing and organisation, both the US and New Zealand face similar challenges in meeting the long-term care needs of an ageing population. Information technology systems in long-term care need to be adopted to better enable improvements in quality and efficiency; increased attention needs to be given to recruiting and retaining a well-trained, stable workforce; and continued development of home- and community-based alternatives to residential care must be pursued. The quickly developing culture change movement, which aims to improve the way chronically frail and disabled people live and are treated, must also be encouraged and supported.

New Zealand has many advantages over the USA in its policy context for long-term care. It is critical that New Zealand build upon these advantages in the short term to ensure that the longer term implications of the ageing population can be met.

Financing and improving the quality of long-term care for increasing numbers of elderly citizens is an international concern. In the USA, a recent Brown University report, *Out of the Shadows: Envisioning a Brighter Future for Long-Term Care in America* (available at http://www.chcr.brown.edu/), has been produced to inform the work of the National Commission for Quality Long-Term Care co-chaired by former Senator Bob Kerrey and former Speaker of the House Newt Gingrich.

The purpose of the Commission is to gather evidence and make recommendations on how to improve the quality of long-term care nationally. Given a rapidly ageing population, the present report should be of interest, not only to US observers but to policymakers, providers, and users of long-term care within New Zealand.

The Brown report examines six areas of concern that must be addressed by policy makers and providers to establish a higher quality, more efficient long-term care system: developing adequate sources of financing and insurance; supporting individuals and family caregivers; promoting physical and organisational change; recruiting and retaining a qualified workforce; designing a more effective regulatory control system; and leveraging health information technology.

These concerns are just as applicable to New Zealand as to the US. Indeed, solutions in each of these areas are necessary, not only if New Zealand is to overcome impediments to providing high quality long-term care to its most vulnerable citizens

but also if it is to meet the increased demand for services and high expectations posed by the ageing baby boom generation.

The percentage of the New Zealand population aged 65 years or over is expected to rise from 13.0% today to 25.0% by 2050 and, of those, 25.0% will be over 85 years. Furthermore, together with ongoing improvements in life expectancy, we can expect further growth in Alzheimer's disease and in functional disability going hand-in-hand with the ageing of New Zealand.

Despite differences in financing and organisation, both the US and New Zealand face similar challenges, including an over-reliance on institutional services and difficulties in recruiting and retaining direct care staff. A recent OECD study found that in 2000, home care spending as a proportion of total long-term care expenditure was 17.7% in New Zealand and 25.0% in the USA—both well short of the OECD average of 30.4%.[1] This indicates that efforts in the USA, and especially in New Zealand, to 'rebalance' long-term care away from nursing homes and towards home- and community-based services, lags behind other countries.

Both countries also face a growing shortage of workers at all levels, including nurses, nurse aides, therapists, and geriatricians. The US government estimates that an additional 1.9 million direct care workers will be needed in long-term care settings by 2010.[2] Given that New Zealand's population is ageing even faster than that of the US, and there has been increased demand for health care workers more generally,[3] workforce shortages for long-term care are also likely to become especially acute in New Zealand.

To improve the current system and to better meet the challenges ahead, *Out of the Shadows* highlights areas for change and action. Those of particular relevance to New Zealand are discussed below.

Enhancing the long-term care workforce

It is well documented that nursing homes and other long-term care providers have difficulty recruiting and retaining direct care staff.[4] This is especially true of lower skilled workers for whom the combination of low wages, insufficient benefits, heavy caseloads, inadequate training, and limited prospects for career advancement make recruitment and retention a particular challenge.

Research has consistently demonstrated a relationship between staffing and quality of care in nursing homes,[5] and shown that nursing homes with greater staff turnover have higher costs associated with vacancy, recruitment, and replacement (e.g., overtime pay, temporary staffing), as well as costs associated with lost productivity, low employee morale, and lower service quality.[6]

New Zealand must continue efforts to improve recruitment and retention. Responsibility for this lies at both the provider and funder level and includes competitive wages, more comprehensive benefits, more extensive training and career programmes. One issue within New Zealand has been ensuring that long-term care providers pass on increased public subsidies to direct care staff in the form of increased employee compensation. Twenty-six states in the USA have addressed this issue through the adoption of 'wage pass-through programmes' which mandate use of increased funding for improved salaries and benefits for direct care workers, although the jury is still out as to which approach is most viable for achieving this objective.

Transforming the culture of long-term care

The "culture change" movement consists of those who would like to change the context within which frail and disabled individuals live and are treated.[7] Rather than treating clients as clinical entities, downplaying their psychosocial and spiritual needs, advocates for culture change believe that systems of care should be adopted that accommodate individuals' choices rather than forcing them to adhere to the routines of the provider. Patient participation, client autonomy, and shared decision-making are emphasised.[8]

Though the physical environment is important, deep and long-lasting transformation requires changes in how the caregiving process is organised. This includes de-emphasising top-down authority by placing as much decision making responsibility as possible into the hands of patients and their caregivers.

Especially salient is replacing the practice of rotating staff with 'primary assignments,' in which staff work consistently with the same clients, a practice associated with many documented benefits.[9] While providers in the US are beginning to transform long-term care in ways commensurate with the culture change perspective,[7] there remain significant barriers (regulatory and otherwise) to adopting the changes necessary to make this vision a reality.

Building on lessons from the US, the New Zealand Government should examine how regulation might better enable both physical and organisational innovation to take place. Providers should commit to adopting "home-like" living environments that respect individuals' privacy and autonomy, honour clients' preferences about activities and lifestyle choices, and empower direct care workers through primary assignments, self-managed work teams, and other organisational adjustments.

Leveraging health information technology (HIT)

Most individuals entering nursing homes or paid home care in the US are referred for care following hospitalisation. Consequently, the transmission of clinical information to enable medical and nursing care to proceed uninterrupted is critical. Unfortunately, it is rare for such transitions to occur smoothly. Research in the US reveals high rates of inaccurate or missing information, ranging from diagnoses to a complete listing of a patient's current medications.[10]

As a result, many patients are re-hospitalised a short time later.[11] It is difficult to find comparative details for New Zealand but it is likely that such issues occur here as well. This is because despite New Zealand having a strong HIT tradition within the primary and acute care sectors, use of integrated HIT in the long-term care sector lags behind. Although HIT has yet to penetrate far into US long-term care, nursing homes and home health agencies have well-established common clinical assessment and outcome measurement instruments.

This uniformity should constitute a major advantage since, like hospital diagnoses, the same information can be applied to payment, outcome measurement, and clinical care planning. However, the absence of the requisite electronic information sharing bridges, in addition to a common clinical nomenclature for describing patient functioning, means that interoperability between the acute and long-term care sectors continues to be lacking.

New Zealand is currently examining use of a possible data collection tool to enable the collection of uniform patient-level data that could be compared nationally, or even internationally. Such a system is crucial if information to improve quality in long-term care is to be provided and if interoperable electronic information sharing is to occur.

One step the government could take is to incorporate the needs of long-term care patients into electronic health record designs. Provider investment in HIT, and partnering with hospitals to develop and implement electronic information sharing that promotes smoother patient transitions from one care setting to another should be encouraged.

Conclusion

It is imperative that all relevant groups re-evaluate how long-term care is provided in New Zealand. Information technology systems need to be adopted to better enable improvements in quality and efficiency; increased attention needs to be given to recruiting and retaining a well-trained, stable workforce; and continued development of home- and community-based alternatives to residential care must be pursued. The quickly developing culture change movement, which aims to improve the way chronically frail and disabled people live and are treated, must also be encouraged and supported.

New Zealand has many advantages over the USA in its policy context for long-term care. It has a unitary system of government that is not subject to the boundary issues, competitive stresses, and gridlock that a federal/state system suffers. It also has a strongly articulated plan for meeting the healthcare needs of older people.[12] It is critical that New Zealand build upon these advantages in the short term to ensure that the longer term implications of the ageing population can be met.

Conflict of interest statement: There are no conflicts of interest.

Disclaimer: The views expressed here are those of the authors and not necessarily those of the National Commission for Quality Long-Term Care, nor the Commonwealth Fund, its directors, officers, or staff.

Author information: Mark Booth, Harkness Fellow; Edward Alan Miller, Assistant Professor of Public Policy, Political Science, and Community Health, and Faculty, Center for Gerontology and Health Care Research; Vincent Mor, Professor and Chair, Department of Community Health, and Faculty, Center for Gerontology and Health Care Research; Brown University, Providence, Rhode Island, USA

Acknowledgements: The report on which this essay is based was written while Edward Miller and Vincent Mor were supported by the National Commission for Quality Long-Term Care, whereas Mark Booth is currently a Harkness Fellow from New Zealand and visiting fellow at the Brown University Center for Gerontology and Health Care Research. The Harkness Fellowship is supported by the Commonwealth Fund, a private independent foundation based in New York City. In addition to acknowledging the invaluable support from the Commonwealth Fund and National Commission, the authors also thank Beth Sundstrom, Laura Smith, Zhanlian Feng, Brenna Sullivan, and Nicole Palin for their assistance.

Correspondence: Mark Booth, Harkness Fellow, The Center for Gerontology and Health Care Research, Brown University, 2 Stimson Avenue, Box G, Providence, Rhode Island 02912, USA. Phone: (401) 863 9957. Fax: (401) 863-3489; email: Mark_booth@brown.edu

References:

1. Organization for Economic Cooperation and Development (OECD). Long-term care for older people. Paris, France: OECD; 2005.
2. U.S. Department of Health and Human Services (USDHHS). The future supply of long-term care workers in relation to the aging baby boom generation: Report to Congress. Washington, D.C. USDHHS; May 14, 2003.
3. Cornwall J, Davey J. Impact of population ageing in New Zealand on the demand for health and disability support services and workforce implications. Wellington, New Zealand: Ministry of Health; 2004.
4. Decker FH, Gruhn P, Matthews-Martin L, et al. Results of the 2002 AHCA survey of nursing staff vacancy and turnover in nursing homes. Washington, D.C.: American Health Care Association; February 12, 2003.
5. Centers for Medicare and Medicaid Services. Appropriateness of minimum nurse staffing ratios in nursing homes Phase II final report. Baltimore, MD: Centers for Medicare and Medicaid Services; 2002.
6. Seavey D. The cost of frontline turnover in long-term care. Washington, D.C.: Institute for the Future of Aging Services, America Association of Homes and Services for the Aging; October 2004.
7. Weiner AS, Ronch JL, eds. Culture change in long-term care. New York, NY: The Haworth Press, Inc.; 2003.
8. World Health Organization and Milbank Memorial Fund. Toward an international consensus on policy for long-term care of the ageing. New York, NY: Ageing and Health Programs, World Health Organization and Milbank Memorial Fund; 2000.
9. Farrell D. Consistent assignment. Providence, RI: Quality Partners of Rhode Island; 2005.
10. Coleman EA, Smith JD, Raha D, Min SJ. Posthospital medication discrepancies: prevalence and contributing factors. Arch Intern Med. 2005;165:1842–7.
11. Coleman EA. Falling through the cracks: Challenges and opportunities for improving transitional care for persons with continuous complex care needs. J Am Geriatr Soc. 2003;51:549–55.
12. Ministry of Health. Health of older people strategy. Wellington, New Zealand: Ministry of Health; 2002. URL: http://www.moh.govt.nz/publications/hops

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Benzylpiperizine-based party pills' impact on the Auckland City Hospital Emergency Department Overdose Database (2002–2004) compared with ecstasy (MDMA or methylene dioxymethamphetamine), gamma hydroxybutyrate (GHB), amphetamines, cocaine, and alcohol

Lynn Theron, Karl Jansen, Jennifer Miles

Abstract

Aim To examine the impact of 'party pills' (PP; herbal highs) on the Auckland City Hospital Emergency Department Overdose Database 2002–2004, and to present figures for five other substances in that database.

Method Auckland City Hospital's Emergency Department's overdose database was reviewed for 2002, 2003, and 2004 for 'herbal ingestions' and 'party pills' (PP), ecstasy, methamphetamine, GHB, cocaine, and alcohol. Adverse effects attributed to PP were examined.

Results In 2002, 1 patient presented with PP ingestion; 4 presented in 2003 and 21 in 2004 respectively ($p<0.001$). Of these 21 patients in 2004, 5 had allegedly ingested PP only and none required medical admission. PP only contributed to 1.58% of the overdose database for 2004.

Conclusion 'Party pills' appeared to have a minor impact on the overdose database at Auckland City Hospital between 2002 and 2004. There was a significant decrease in GHB presentations from 2003 to 2004 ($p<0.001$), but no significant fall in stimulant overdose presentations.

'Rapture', 'Charge', and 'Frenzy' are some of the many 'party pills' (PP; 'herbal highs') that are currently available over-the-counter in New Zealand. They are promoted as 'rave drugs' and 'energy drugs' due to their reported ability to produce feelings of euphoria, increased energy, and a desire to socialise. Their manufacturers sometimes promote them as a safe alternative to the use of amphetamines. One supplier claims that their market research indicates that amphetamine users consume less amphetamines when PP are available, and that casual users are more likely to stop using amphetamines entirely and replace them with PP.[1]

Are claims of safety accurate? Presentations to the emergency department requiring treatment, hospital, or ICU admission can be used as indicators of drug adverse effects. Hence the Auckland Hospital Emergency Department Overdose Database was reviewed over the period 2002–2004 to establish presentations of PP and their outcomes in this context.

The main ingredients of these substances are the piperizine derivatives of which the best known compound is N-benzylpiperizine (BZP). The piperazine class also includes trifluromethylphenylpiperizine (TFMPP), which is found in some pills.

Piperazines are readily absorbed from the gastrointestinal tract. The pharmacokinetics in humans has been investigated to a limited extent.[2,3] The main route of metabolism is via cytochrome p450; the remainder is excreted in urine. BZP was synthesised in 1944 and it was found to produce amphetamine-like effects.[4]

These studies suggested an approximate ratio of 10:1 (BZP: amphetamine) in the effect potency. BZP has been shown in animals to act as an indirect sympathomimetic amine, causing release of neurotransmitters in serotonergic, dopaminergic and noradrenergic nerve terminals.[4] It also inhibits serotonin reuptake.

Typical doses of BZP ingested by users range from less than 50 mg to 500 mg and more. The commonest adverse effects from high doses are headaches and severe hangovers that discourage high-dose use. The side-effects in overdose are similar to amphetamine. These include anxiety, panic attacks, tachycardia, hypertension, hyperthermia, restlessness, hallucinations, rash, seizures, nausea and vomiting, insomnia, and psychosis.[1,3–5,9,12] One fatality has been reported[6] in the literature to date: BZP was ingested with ecstasy resulting in hyponatraemia and cerebral oedema. As ecstasy can cause death in this manner without BZP,[7] the role of BZP in the fatality is unclear.

On the grounds that they have a high potential for abuse and no current medical use, the United States Drug Enforcement Administration scheduled these substances as controlled drugs. In New Zealand, they are not listed as controlled drugs, although some restrictions on their sale have been introduced.

Auckland Hospital Emergency Department classifies an ‘overdose’ as any consumption of a substance that has an adverse effect resulting in presentation to the department. Thus taking one pill with a major adverse effect is an ‘overdose’. The database does include suicidal/self harm overdoses.

Method

Auckland City Hospital Adult Emergency Department (ED) had attendants of 49,000 in 2004 and an admission rate of 35% in 2004. It is the hospital closest to the Auckland night-club district. All patients presenting to the department due to an overdose are entered into a database. Information recorded includes age, sex, ethnicity, times in ED, and drugs ingested. The database was reviewed for the period 2002 to 2004 inclusive. Prior to 2002, there were no recorded overdose presentations for PP.

The database was searched for ‘party pills’, ‘herbal highs’, ‘Charge’, ‘Rapture’, ‘Frenzy’, ‘ESP’, ‘Jump’, and ‘Euphoria’; and the cases identified. Herbal overdose ingestions that were not of the party pill nature, e.g. Valerian overdose, were excluded. The following data were recorded: age/sex; PP ingested; co-ingestions; presenting complaints; vital signs; special investigations; past medical history; medications; treatment in the emergency department; disposal. The database was then also reviewed for overdose of ecstasy, methamphetamine, GHB, cocaine, and alcohol.

Differences in the proportion of overdoses resulting from PP and GHB between 2002 and 2004 were analysed for statistical significance using a Chi-squared test. Yates correction was used for the PP due to frequency count(s) of less than 5. All comparisons were conducted at a 5% level of statistical significance and conducted using GraphPad Software.

Results

The results show a significant increase in overdose presentations of PP to the emergency department with 1 (0.07% of total overdoses) presentation in 2002 to 21 presentations in 2004 (1.58% of total overdoses) ($p<0.001$; Table 1).

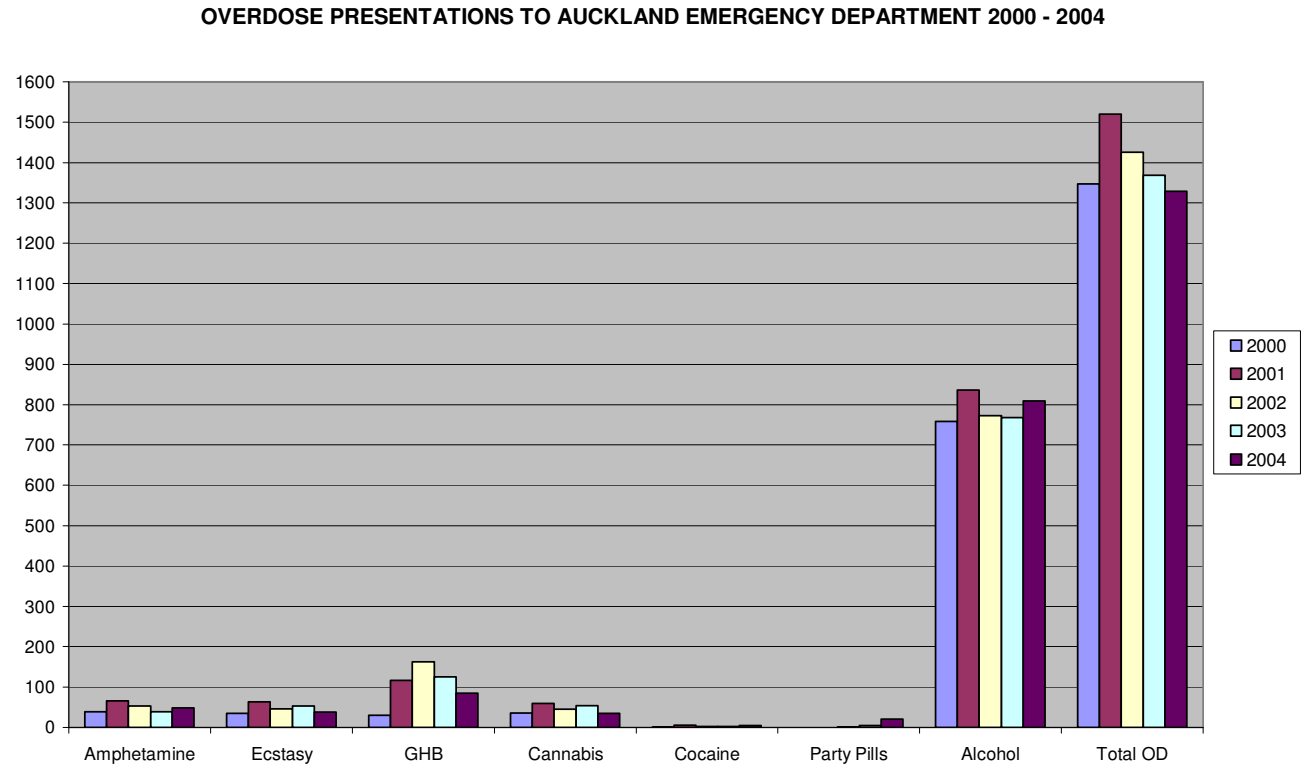
Table 1: Overdose (OD) presentations to Auckland Hospital Adult Emergency Department: 2002–2004 inclusive

Drug	2002		2003		2004	
	n	Total ODs	n	Total ODs	n	%Total ODs
Amphetamine	53	3.72%	39	2.85%	49	3.69%
Ecstasy	46	3.23%	53	3.87%	38	2.86%
GHB	163	11.44%	125	9.14%	85	6.40%
Cocaine	2	0.14%	2	0.15%	4	0.30%
Alcohol	772	54.18%	768	56.14%	809	60.87%
Party pills	1	0.07%	4	0.29%	21	1.58%
Other	388	27.23%	337	24.63%	323	24.30%
Total overdoses	**1425**		**1368**		**1329**	

GHB=gamma hydroxybutyrate.

With a consumption of 200,000 tablets/month, a presentation of 21 patients to the emergency department in a year is relatively small (Figure 1).

Figure 1



Of the 26 overdose charts (the total number) reviewed and included in this study, only 5 ingested PP only (19%) (Table 2).

Table 2: Patient chart search results (age, sex, party pill brand ingested, alleged co-ingestants)

Case no.	Year	Age/Sex	Party pill brand	No. ingested	Alleged co-ingestant
Case 1	2002	?/F	Exodus		ecstasy
Case 2	2003	19/F	Rapture	6 tablets	nil
Case 3	2003	22/M	Herbal high		alcohol, methamphetamine ('P'), nitrous oxide
Case 4	2003	25/F	Herbal high		alcohol
Case 5	2003	18/F	Rapture	1 tablet	cannabis
Case 6	2004	25/M	ESP	2 tablets	nil
Case 7	2004	27/M	Rapture	3 tablets	nil
Case 8	2004	26/M	Party pill*		nil
Case 9	2004	30/M	Party pill*		nil
Case 10	2004		Rapture	5 tablets	alcohol
Case 11	2004		Rapture	2 tablets	one ecstasy tablet
Case 12	2004	29/M	ESP	3 tablets	alcohol
Case 13	2004		Euphoria	9 tablets	nitrous oxide, cannabis
Case 14	2004		Charge		ecstasy, alcohol
Case 15	2004	23/F	Charge	1 tablet	Two ecstasy pills, alcohol
Case 16	2004	30/M	Party pill*		alcohol
Case 17	2004	22/M	Party pill*	6 tablets	three ecstasy pills, alcohol
Case 18	2004	32/M	Party pill*	20 tablets	alcohol
Case 19	2004	17/F	Party pill*		alcohol
Case 20	2004	18/F	Jump	1 tablet	alcohol
Case 21	2004	17/F	Charge	1 tablet	alcohol, paracetamol
Case 22	2004	17/F	Party pill*		alcohol
Case 23	2004	30/F	Charge	4 tablets	alcohol
Case 24	2004	18/M	Party pill*		alcohol
Case 25	2004	25/F	Charge		5HT-P, alcohol
Case 26	2004	32/M	Frenzy, Exodus		alcohol

*Brand unspecified.

One person in 2003 who presented with jerking movements and nausea was treated with intravenous (IV) fluids and diazepam and discharged home with advice. The four patients who presented in 2004 ingested 'ESP', 'Rapture', and two unknown PPs. All presented with anxiety, palpitations, dizziness, and nausea. Two were treated with IV fluids and diazepam, the other two self-discharged without being seen by the emergency physician.

Abnormal vital signs were recorded in this group of PP ingestion only, namely dilated pupils, HR >120 beats/min (125), and elevated BP (Table 3) (168/94; 160/90 mmHg). One patient had a pre-existing condition (asthma) and was prescribed inhalers.

Special investigations performed in this group were unremarkable. The most common presenting complaints of this overdose group were anxiety, palpitations, nausea, and vomiting which occurred in 13 patients (50%). The next most common symptom complex was decreased level of consciousness and confusion. This occurred in eight patients (30%).

Table 3. Patient chart search results (presenting symptoms, vital signs, special investigations)

Case no.	Presenting complaint	Vital signs	Special investigations
Case 1	Anxiety	T 36.6; HR 90; BP 110/80	U/E NAD
Case 2	Involuntary jerking movements, slurred speech	T 36.4; HR 98; BP 104/26; Dilated pupils	FBC; U/E; alcohol NAD
Case 3	Headache, vomiting	T 36; HR 85; BP 150/70; GCS 15	FBC; U/E; CT head NAD
Case 4	Collapse bradycardia, HR 35 atropine by ambulance	T 36; HR 40; BP 140/110; GCS 14	FBC NAD; K 3.2 Alcohol 45
Case 5	Confusion, memory loss	T 37.3; HR78; BP 120/70; GCS 15	FBC; U/E; LFT; ETOH; ECG NAD
Case 6	Dizzy, nausea, vomiting, palpitations	HR 85; BP 160/80	nil
Case 7	Palpitations, anxiety, chest pain	HR 112; BP 168/94	ECG; sinus tachycardia
Case 8	Short of breath, anxious	HR 125; BP 115/70	nil
Case 9	Anxious, hyperventilating	T 36.4 HR 118; BP 160/90	nil
Case 10	Light headed, dizzy, vomiting	HR 110; BP 121/81	Alcohol NAD
Case 11	Anxiety, palpitations, mouth twitching	HR 138; BP 135/70	ECG; sinus tachycardia; WCC 13
Case 12	Abdominal pain, nausea coffee ground vomiting	T 35.9; HR 70; BP 140/90	K 3.2; Hb 172; WCC 11.7
Case 13	Decreased level of consciousness	T 37; HR 90; BP 121/70; GCS 14	ETOH 60; WCC 12
Case 14	Panic, tremor	HR 133; BP 147/78	ECG; sinus tachycardia
Case 15	Agitation, dizziness, insomnia,	T 37.6; HR 110; BP 110/80; Dilated pupils	nil
Case 16	Palpitations, anxiety, nausea	T 37; HR 127; BP 140/80	ETOH 24; FBC; U/E NAD
Case 17	Decreased level of consciousness with ambulance, amnesia	T 35.7 HR 130 145/70 GCS 15; Dilated pupils	ECG; sinus tachycardia
Case 18	Feeling “weird”	T 36.8; HR 94; BP 120/80	nil
Case 19	Drowsy, vomiting	T 36.6; HR 80; BP106/72; GCS 14, Dilated pupils	ETOH 49; U/E: K 3.2
Case 20	Decreased level of consciousness, vomiting	T 36; HR 116; BP 110/60; GCS 14	ETOH 46; K 3.2; FBC NAD
Case 21	Suicidal attempt	T 36.6; HR 110; BP 117/79	nil
Case 22	Loss of consciousness, epigastric pain, vomiting	T 36; HR 90; BP 130/80; GCS 14	ETOH 28; U/E NAD
Case 23	Insomnia, palpitations, paranoia	T 36.5; HR 100; BP 104/50	nil
Case 24	Palpitations, anxiety	T 36.9; HR 115; BP 140/80	nil
Case 25	Combative, agitated	T 36.6; HR 90; BP 120/70; GCS 14	ETOH 40; FBC / U/E: NAD
Case 26	Tremor 24 hrs before decreasing alcohol, nausea, vomiting	T 36.2; HR 90; BP 126/84; GCS 15	FBC NAD; K 3.4

T=Temperature (°C); HR=Heart rate (bpm); BP=Blood pressure (mmHg); GCS=Glasgow Coma Score. NAD=no abnormality detected.

One patient presented with suicidal ideation and attempt, one with a severe headache, and one with upper gastrointestinal bleed (this patient [3%] required admission for recurrent vomiting and abdominal pain). See Table 4. Gastroscopy revealed reflux gastritis that was treated with omeprazole. The patient had not only ingested three PP

but also nine units of alcohol that may have contributed to (or been the cause of) this gastritis. This was the only patient to be admitted.

Table 4. Patient chart search results (past medical history, medication, treatment, disposal)

Case no.	Past medical history / medications	ED treatment	Disposal
Case 1	Nil	IV fluids	Discharge home
Case 2	Nil	IV fluids, diazepam	Discharge home
Case 3	Nil	IV fluids	Discharge home
Case 4	Nil	IV fluids	Discharge home
Case 5	Asthma / diet pills	Psychiatric review	Discharge home
Case 6	Nil	IV fluids	Discharge home
Case 7	Asthma	Diazepam	Discharge home
Case 8	Nil	Nil	Self discharge, not seen by physician
Case 9	Asthma / Ventolin, Flixotide, Serevent	Nil	Self discharge, not seen by physician
Case 10	Nil	Reassurance	Discharge home
Case 11	Hayfever / St Johns Wort	Reassurance	Discharge home
Case 12	Panic attacks	IV fluids, analgesia	*Admitted
Case 13	Nil	IV fluids	Discharge home
Case 14	Depression / amitriptyline	IV Fluids, diazepam	Discharge home
Case 15	Nil	Diazepam	Discharge home
Case 16	Nil	IV fluids, diazepam	Discharge home
Case 17	Minor surgery	IV fluids	Discharge home
Case 18	Alcohol and drug rehabilitation	Reassurance	Self discharge, didn't wait for review
Case 19	Asthma / Ventolin	Reassurance	Discharge home
Case 20	Asthma	IV fluids	Discharge home
Case 21	Depression / Aropax	Psychiatric review	Discharge home, Psychiatric follow-up
Case 22	Alcohol intoxication, gastritis	IV fluids, omeprazole, Mylanta	Discharge home
Case 23	Asthma, depression, alcohol and drug abuse / Ventolin, dothiepin	Reassurance	Discharge home
Case 24	Nil	Reassurance	Discharge home
Case 25	Epilepsy, depression / Epilim, Aropax	Midazolam	Discharge home
Case 26	Alcohol and drug rehabilitation	IV fluids, diazepam	Discharge home

*Medical admission, gastroscopy diagnosed reflux oesophagitis secondary to vomiting. Discharged on omeprazole.

The most common co-ingestant was alcohol—involving 18 patients (69%). Others were 5 cases of co-ingesting ecstasy (19%), 2 cannabis (7%), 2 nitrous oxide (7%), and 1 methamphetamine (3%) (Table 2).

Of this study population, 10 patients were reassured only (38%), 12 were treated with IV fluids (46%), and 6 were given diazepam for anxiety (23%) (Table 4). The only abnormal special investigations were elevated alcohol levels in those who had co-ingested alcohol, and low potassium levels in 3 patients (Table 3).

Table 2 demonstrates trends in overdose presentations. There was a significant decrease in GHB presentations from 2003 to 2004 ($p<0.001$), but no significant fall in stimulant overdose presentations.

Discussion

While the frequency of 'party pill'-related presentations to Auckland Hospital Emergency Department increased over the 3 years after 2002, it still contributed only 1.58% to the overall overdose database in 2004. Most patients presented with multidrug ingestions (and with symptoms of anxiety, palpitations, nausea, and vomiting); 96% were discharged home after reassurance, IV fluids, and diazepam. Only one patient was admitted (case #12). Thus these results lead towards a conclusion that the impact on the emergency department over these years was relatively small in Auckland City.

There is no sharp, major reduction in ecstasy and amphetamine presentations versus a halving in the number of GHB presentations in 2004 in comparison with 2002. This is relevant to the previously mentioned suggestion that PP might replace amphetamine.[1] GHB is a powerful sedative, not a stimulant, and is unlikely to have been replaced by 'party pills.'[8] The fall in GHB presentations may be linked to legislative changes. Although changes in drug use trends can have complex determinants, the data presented here tend not to support an argument that there has been widespread substitution of ecstasy and amphetamine use by party pills in Auckland City over the years studied. However, Wilkins et al have a reported a fall in local amphetamine use over a similar period.[11]

A study at Christchurch Hospital Emergency Department focussing on 2005 found far more concerning results, with 15 persons out of 60 (a total of 80 presentations) having toxic seizures.[10] A possible reason for this regional variation is that more persons in Christchurch in 2005 were taking brands of pills containing much higher doses of BZP than persons in Auckland between 2002 and 2004.

According to Matt Bowden[11] (Spokesperson for the Social Tonics Association of New Zealand), the market leaders in Auckland in 2002–2004 were probably companies producing capsules in which the average BZP dose per capsule was believed to be unlikely to exceed 100 mg, and was quoted as such on packet covers inspected by the authors.

In contrast, different companies were claimed to be dominant in Christchurch in 2005, and some of the packet covers of the products made by these companies claim that the capsules contain at least 500 mg of BZP per capsule. Higher dose capsules from a variety of sources are believed to have become more available in Auckland in 2005. Analysis of Auckland Hospital Emergency Department results for 2005 and 2006 are in progress.

We acknowledge some limitations in this study: the overdose database does not include patients who present to the emergency department with presentations such as anxiety, palpitations, and other 'party pill' side effects who do not disclose ingesting these substances. Routine urine toxicology screening is not performed on overdose patients who present to the ED due to costs incurred. Toxicology screening is performed if the drug ingestion is unknown or the patient is comatose with no collateral history. Some of the material used for the discussion is anecdotal; this is currently an unavoidable feature of this particular topic.

Conflict of interest statement: The authors have no conflicts of interest.

Author information: Lynn Theron, Emergency Medicine Specialist, Emergency Department, Auckland City Hospital; Karl L R Jansen, Consultant Psychiatrist, Te Whetu Tawera/Auckland City Acute Mental Health Unit; Auckland City Hospital; Jennifer Miles, Research Assistant, Liggins Institute; Grafton, Auckland

Correspondence: Lynn Theron, Emergency Department, Auckland City Hospital, Park Road, Grafton, Auckland. Fax: (09) 375 4369; email: ltheron@adhb.govt.nz

References:

1. Benzylpiperazine dihydrochloride. Drug Data Sheet. Stargate International. URL: http://www.stargateinternational.org
2. Staack RF, Fritschi G, Maurer HH. Studies on the metabolism and toxicological detection of the new designer drug N- benzylpiperizine in urine using gas chromatography-mass spectrometry. Journal of Chromatography. 2002;B,773:35-46.
3. Maurer H, Kraemer T, Springer D, et al. Chemistry, pharmacology, toxicology and hepatic metabolism of designer drugs of the amphetamine piperazine and pyrrolidinophenone types. Therapeutic Drug Monitoring. 2004;26:127–31.
4. Campbell H, Cline W, Evans M, et al. Comparison of the effects of dexamphetamine and 1-benzylpiperizine in former addicts. European Journal of Clinical Pharmacology. 1973:(6):170–6.
5. Austin H, Monasterio E. Acute psychosis following ingestion of 'Rapture'. Australas Psychiatry. 2004;12:406–8.
6. Balmeli C, Kupferschmidt H, Rentsch K et al. Fatal brain oedema after ingestion of ecstacy and benzylpiperizine. Dtsh Med Wochenschr. 2001;126:809–11.
7. Henry JA, Jeffreys KJ, Dowling S. Toxicity and Deaths From 3,4-Methylene dioxymethamphetamine ('Ecstasy'). Lancet 1992;340:384–7.
8. Theron L, Jansen KLR, Skinner AM. New Zealand's first fatality linked to use of 1,4-Butanediol (1,4-B, Fantasy): no evidence of coingestion or comorbidity. N Z Med J. 2003;116(1184). URL: http://www.nzma.org.nz/journal/116-1184/650
9. Gee P, Richardson S, Woltersdorf W, Moore G. Toxic effects of BZP-based party pills in humans: a prospective study in Christchurch, New Zealand. N Z Med J. 2005;118 (1227). URL: http://www.nzma.org.nz/journal/118-1227/1784
10. Unpublished communication between the authors and Matt Bowden; 2006
11. Wilkins C, Sweetsur P, Casswell S. Recent population trends in amphetamine use in New Zealand: comparisons of findings from national household drug surveying in 1998, 2001, and 2003. N Z Med J. 2006;119(1244). URL: http://www.nzma.org.nz/journal/119-1244/2285
12. Wilkins C, Girling M, Sweetsur P, et al. Legal party pill use in New Zealand: Prevalence of use, availability, health harms and 'gateway effects' of benzylpiperazine (BZP) and triflourophenylmethylpiperazine (TFMPP). Centre for Social and Health Outcomes Research and Evaluation (SHORE); 2006. URL: http://www.ndp.govt.nz/legalpartypills/documents/legal-party-pill-use-nz.pdf

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Screening and intervention for alcohol problems among patients admitted following unintentional injury: a missed opportunity?

Jamie Hosking, Shanthi Ameratunga, Chris Bullen, Ian Civil, Alex Ng, Anthony Rodgers

Abstract

Aim To describe current screening and intervention practice for alcohol problems in a New Zealand trauma centre.

Methods Retrospective analysis of a trauma registry database at a metropolitan hospital in New Zealand, and hospital chart review for documentation of alcohol screening and intervention on a random sample of 120 adults, stratified by ethnicity and blood alcohol status, admitted following unintentional injury for the period January 2003 to December 2004.

Results Among 1970 patients admitted following unintentional injury during the study period, 23% had a blood alcohol test at admission. Approximately half of these tests were positive. While 68% of charts reviewed included a general comment on alcohol use, only 7.3% recorded information that suggested a possible drinking problem. No formal alcohol screening interviews were documented, and in only 1.5% of admissions was an alcohol intervention in the hospital setting recorded.

Conclusion Formal screening and interventions for alcohol problems among this group of inpatients were infrequent, indicating missed opportunities to reduce alcohol-related harm and, potentially, trauma recurrence. Effective approaches for alcohol screening and intervention in the New Zealand trauma inpatient setting require review.

The burden of disease attributable to alcohol is substantial in New Zealand[1] and in other industrialised countries, as well as in many economically developing countries.[2] Over half of alcohol-attributable mortality in New Zealand is due to injury.[1]

Alcohol-attributable injuries and other alcohol-related harm are substantially higher for Māori than non-Māori;[1] while equivalent data for Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin) are not currently available, it is known that those who drink have more harmful drinking patterns than the general population.[3] Social costs associated with alcohol in New Zealand were estimated at between NZ$1 billion and NZ$4 billion in 1991;[4] a more recent study suggested that alcohol-related motor vehicle crashes alone cost NZ$1.2 billion in New Zealand in 1996.[5]

Trauma patients are at high risk of future injury,[6] with the risk further increased among hazardous drinkers.[7] A high prevalence of alcohol problems in the trauma setting is reported in many western countries[8,9], with alcohol intoxication described as the 'leading risk factor for injury'.[10]

Brief alcohol interventions have been shown to be effective in improving alcohol drinking outcomes in several meta-analyses,[11–13] and have been recommended for hazardous drinkers in guidelines in New Zealand[14] and elsewhere.[15]

Alcohol interventions first require the identification of those eligible, usually through screening, using one of several validated alcohol screening tools. For example, the 10-item Alcohol Use Disorders Identification Test (AUDIT) questionnaire is a validated tool used in New Zealand national health surveys to identify the prevalence of hazardous drinking.[16] Screening and brief intervention (SBI) together appear to be cost-effective, with estimated cost-benefit ratios ranging from 3.8 to 4.3 for costs to the health care system.[10,17]

A Cochrane review concluded that interventions among problem drinkers appear to reduce the risk of future injury,[18] although larger trials were advocated given the low precision of estimated effects on injuries.

Given the above evidence, the trauma inpatient population would appear to be an opportune setting for SBI. Previous SBI trials among trauma patients overseas have been effective in reducing problem drinking behaviour and injury recurrence.[19] However, there are no recent published data on screening and intervention rates for trauma inpatients in New Zealand. Neither is there a formal screening programme in New Zealand hospitals, either targeted or universal, for detecting alcohol problems. The acceptability and resource implications for such a programme are also currently unknown. As part of a feasibility assessment of alcohol interventions for patients admitted following unintentional injury, we describe alcohol screening and intervention practice for trauma inpatients at a metropolitan trauma centre in New Zealand.

Methods

Auckland City Hospital has a catchment area of approximately 415,000 people. The hospital also accepts major trauma patients directly from the site of injury anywhere within the greater Auckland area if they require tertiary trauma care services. Additionally, the hospital receives patient transfers from other District Health Boards for tertiary level trauma care. The Auckland City Hospital Trauma Service's registry of trauma patients admitted to the hospital was searched to identify all patients admitted following an unintentional injury over the 2 years from January 2003 to December 2004.

The database documents blood alcohol levels, if these were obtained, but no other information on alcohol screening or intervention is recorded. Therefore, a detailed chart review was undertaken of 120 records selected using a stratified random sampling procedure (Figure 1).

Records of patients with a documented positive blood alcohol test at admission ('blood alcohol positive') were oversampled to comprise half the final sample, thereby including sufficient records with at least one indicator of possible problem drinking. The remaining 60 records ('blood alcohol not positive') were of patients who either had a negative blood alcohol concentration (BAC) test or were not tested at admission. The stratification procedure also ensured similar numbers of Māori, Pacific, and any other ethnicity in each of the two blood alcohol groups; the trauma registry documents the ethnicity of patients based on the information collected on the hospital admission form. This made it possible to derive estimates of equivalent precision for patients of Māori and Pacific ethnicity—populations considered to be at higher risk of some types of alcohol-related harm.[1,3] However, the sample size was not designed to detect significant differences between ethnic groups or adjust for potential confounding variables such as socioeconomic status. Information was extracted from charts using a standardised form.

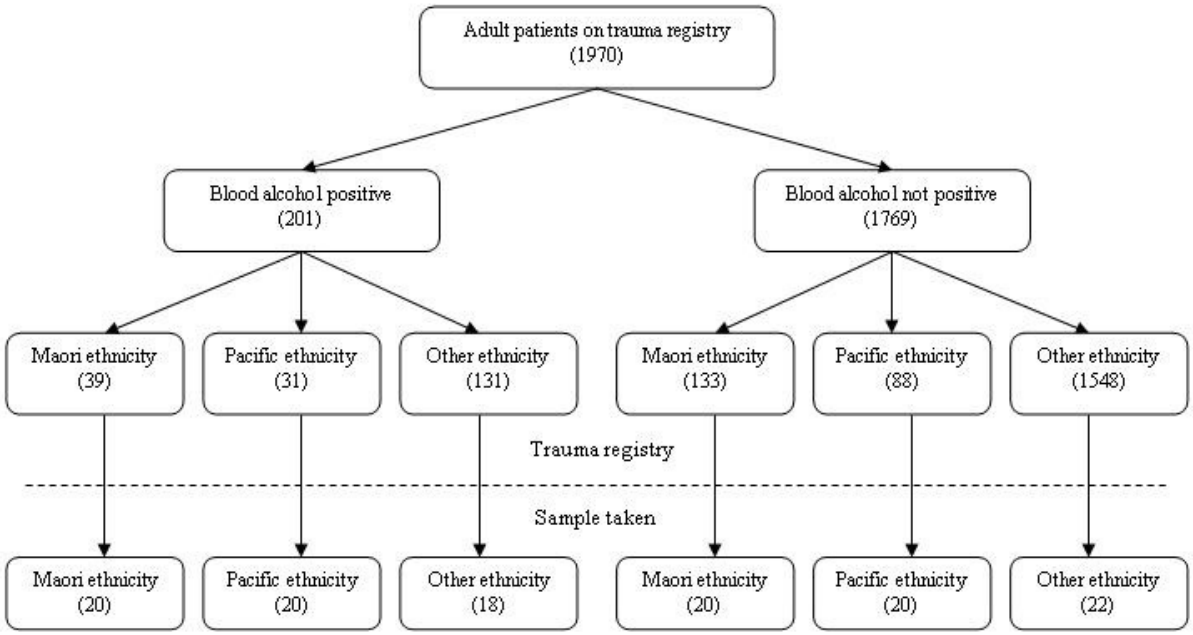
A summary measure denoting a probable drinking problem was created by assessing the presence (or not) of one or more of the following indicators: excessive usual alcohol intake, identified as a problem drinker by hospital staff, suspected alcohol withdrawal during admission, problem drinking recorded in the patient's past medical history, or a positive alcohol screening questionnaire. 'Excessive usual

alcohol intake' was defined as >14 units/week for females and >21 units/week for males, in line with New Zealand guidelines for healthy drinking.[20] A positive BAC test or a history of alcohol consumption prior to injury could also occur in problem drinkers, but these do not necessarily indicate longer-term alcohol problems. The pattern of drinking may also relate to some health effects, but these relationships are less clear. Furthermore, the pattern of drinking was also considered unlikely to be documented in sufficient detail to enable an explicit definition of problem drinking. Injury data obtained from the trauma registry included the Injury Severity Score, a composite score correlated with the threat to life from acute injury.[21]

Analyses adjusted for the stratified sampling in order to generate estimates for the whole population (i.e. all eligible patients on the trauma registry). Statistical analysis was performed using Version 9.1 of the SAS System for Windows, SAS Institute Inc.

Ethical approval for the study was given by the Regional Ethics Committee.

Figure 1. Sampling strategy



Results

Characteristics of the trauma registry population—During the 24-month study period, 1970 admissions of patients aged ≥18 years (median 42; range 18–98) with injury classified as unintentional were recorded in the Trauma Registry. Males comprised 64% of admissions, with 10% and 7% of patients being of Māori and Pacific ethnicity respectively. The median Injury Severity Score was 4 (range 1–75) and the median length of hospital stay 5 days (range 1–92).

A BAC result was recorded in only 23%; 12% tested negative (<3 mmol/L, equivalent to <13.8 mg/100mL[22]), and 11% positive (≥3 mmol/L). Among the latter, 97% and 86% had BAC levels equivalent to ≥30 mg/100mL and ≥80 mg/100mL, respectively (the legal blood alcohol limits in New Zealand for drivers under 20 years of age and for those aged 20 and over are 30 mg/100mL and 80 mg/100mL, respectively[23]).

Characteristics of study participants in the chart review—Demographic and injury data relating to the 120 patients in this review are shown in Table 1. The chart review revealed that two patients in the 'other ethnicity' group who were recorded as blood alcohol positive on the trauma registry were in fact blood alcohol negative, leaving 18 'blood alcohol positive' and 22 'blood alcohol not positive' patients in this ethnicity stratum.

Overall, 24% of those defined as 'blood alcohol not positive' had a negative result on a blood alcohol test, while the remainder had no record of a blood alcohol test being performed.

Table 1. Demographics of sample of trauma patients from Auckland City Hospital Trauma Registry

Variable	Overall	Blood alcohol not positive			Blood alcohol positive		
		Māori	Pacific	Other	Māori	Pacific	Other
n	120	20	20	22	20	20	18
Age – median (range)	28 (18–80)	43 (18–72)	25 (18–66)	33 (19–69)	26 (18–52)	25 (18–61)	28 (19–80)
Gender – male	78%	70%	75%	73%	85%	85%	83%
ISS – median (range)	9 (1–25)	7 (1–43)	9 (1–38)	4 (1–75)	15 (1–45)	9 (1–25)	12 (4–38)
Hospital stay in days – median (range)	6 (1–68)	6 (1–68)	4 (1–56)	6 (1–20)	7 (1–66)	10 (1–52)	7 (1–60)

ISS: Injury Severity Score

Detected prevalence of problem drinking indicators—No patient records were identified that indicated the use of a questionnaire-based alcohol screening tool (Table 2). Two-thirds of records (68%, 95%CI 52–84%) contained some information about usual alcohol intake but this was often not quantified (e.g. 'social drinker').

One or more indicators of problem drinking were documented in 7.3% (0.0–15%) of patient records; 23% (12–35%) had an indicator of problem drinking and/or evidence of alcohol consumption prior to injury. The proportions with one or more documented indicators of problem drinking were: 16% (4.0-28%) for Māori, 7.6% (0.5–15%) for Pacific, and 6.4% (0.0–15%) for patients of other ethnicities. Not surprisingly, the prevalence of indicators of problem drinking—as identified in this chart review—appeared more common in the group that had a positive blood alcohol compared with the group that did not. In the blood alcohol positive group, 24% (11-38%) had one or more other indicators of possible alcohol problems.

Intervention—Only one patient was documented to have received a brief alcohol intervention during the hospital admission. However, a few other patients were either advised of the negative impacts of their alcohol intake, given educational material on alcohol, or had documented follow-up for their alcohol intake planned after discharge. In total, 1.5% (0.3–2.7%) received some form of intervention for their alcohol intake (Table 3). An intervention was received by 1.1% of Māori patients, 5% of Pacific patients, and 1.3% of patients of other ethnicities. Overall, 13% (1.5–24%) of blood alcohol positive patients received an alcohol intervention, compared with 0.2% (0.0–0.7%) of those patients who were not blood alcohol positive.

Table 2. Indicators of problem drinking in a sample of trauma inpatients at Auckland City Hospital, 2003–04

Variable	Overall*	Blood alcohol not positive			Blood alcohol positive		
		Māori	Pacific	Other	Māori	Pacific	Other
n		20	20	22	20	20	18
1. History of alcohol use prior to injury	16% (6.2–26%)	10% (0.0–23%)	10% (0.0–23%)	9.1% (0.0–22%)	85% (73–97%)	85% (75–95%)	72% (51–94%)
2. Excessive weekly alcohol intake†	1.5% (0.3–2.7%)	5.0% (0.0–15%)	5.0% (0.0–14%)	0%‡	5.0% (0.0–12%)	5.0% (0.0–11%)	11% (0.0–26%)
3. Identified as problem drinker by hospital staff	3.2% (1.6–4.8%)	10% (0.0–23%)	0%‡	0%‡	20% (6.6–33%)	15% (4.8–25%)	28% (6.5–49%)
4. Suspected alcohol withdrawal during admission	1.0% (0.1–2.0%)	0%‡	0%‡	0%‡	10% (0.0–20%)	5.0% (0.0–11%)	11% (0.0–26%)
5. History of past problem drinking	5.1% (0.0–12%)	5.0% (0.0–15%)	0%‡	4.5% (0.0–14%)	20% (6.6–33%)	5.0% (0.0–11%)	11% (0.0–26%)
6. Screened positive for problem drinking this admission	0%‡	0%‡	0%‡	0%‡	0%‡	0%‡	0%‡
At least one indicator of problem drinking (items 2–6)	7.3% (0.0–15%)	15% (0.0–31%)	5.0% (0.0–14%)	4.5% (0.0–14%)	20% (6.6–33%)	15% (4.8–25%)	28% (6.5–49%)
At least one indicator of problem drinking and/or evidence alcohol consumed (items 1–6)	23% (12–35%)	25% (5.8–44%)	15% (0.0–30%)	14% (0.0–29%)	All	All	All

Figures in parentheses denote 95% confidence intervals; *Overall: estimate for all patients on trauma registry; †Excessive weekly alcohol intake: over 21 units per week for males or over 14 units per week for females; ‡No patients in the sample had this documented

Table 3. Documented information regarding alcohol interventions in a sample of trauma inpatients at Auckland City Hospital, 2003–04

Variable	Overall*	Blood alcohol not positive			Blood alcohol positive		
		Māori	Pacific	Other	Māori	Pacific	Other
n		20	20	22	20	20	18
Risks of alcohol discussed with patient	1.3% (0.2–2.5%)	0%†	5.0% (0.0–14%)	0%†	0%†	0%†	17% (0.0–34%)
Educational material provided	0.4% (0.0–1.0%)	0%†	0%†	0%†	0%†	0%†	5.6% (0.0–16%)
Brief intervention given (by general staff)	0.4% (0.0–1.0%)	0%†	0%†	0%†	0%†	0%†	5.6% (0.0–16%)
Intervention given by specialist staff, e.g. psychiatry	0%†	0%†	0%†	0%†	0%†	0%†	0%†
Alcohol follow-up discussed, e.g. CADS	0.8% (0.0–1.6%)	0%†	5.0% (0.0–14%)	0%†	5.0% (0.0–12%)	5.0% (0.0–11%)	5.6% (0.0–16%)
Any of the above	1.5% (0.3–2.7%)	0%†	5.0% (0.0–14%)	0%†	5.0% (0.0–12%)	5.0% (0.0–11%)	17% (0.0–34%)

CADS: Community Alcohol and Drug Services, a local alcohol treatment provider; Figures in parentheses denote 95% confidence intervals; *Overall: estimate for all patients on trauma registry; †No patients had this documented.

Discussion

This exploratory study was designed to assess the extent to which formal screening and interventions for alcohol problems are undertaken during hospitalisations at a New Zealand metropolitan trauma centre. Our findings indicate that rates of screening and intervention for alcohol problems in this setting are low. Overall, 23% of trauma patients admitted to hospital during the study period had a blood alcohol test performed at the initial assessment, half of which were noted to be positive (≥13.8 mg/100 ml).

A high proportion (86%) of the latter had a BAC greater than 80 mg/100 ml. Of the 120 patients whose hospital charts were reviewed in detail, none had formal documented questionnaire-based alcohol screening, but 7.3% of trauma registry patients had indicators of problem drinking recorded. A further 16% had evidence of alcohol consumption prior to their injury (a history of alcohol consumption prior to injury or a positive blood alcohol test) without having other indicators of problem drinking.

Documented information regarding implemented interventions was even lower, with only 1.5% of patients having some form of alcohol intervention recorded. Only one patient in this sample was recorded as having received a brief alcohol intervention in the hospital setting.

By undertaking an analysis of a well-established regional trauma registry and a systematic and standardised review of hospital records in a representative sample of patients, we were able to establish a profile of current practice in relation to patients who were and were not identified as blood alcohol positive. Our sampling strategy permitted us to derive estimates for Māori, Pacific, and other ethnicities, but the study was not designed to identify significant differences between these groups.

Our results have limited generalisability, as these only reflect practice for inpatients admitted following unintentional injury at one major trauma centre in New Zealand. No attempt was made in this study to identify patients in whom screening and intervention were not feasible, such as those patients who died in hospital (death occurred in seven patients in this sample).

A disadvantage of retrospective chart review is that any screening or interventions that occurred (but were not documented) will not have been detected. However, this method is useful in assessing alcohol screening and intervention practice, as it avoids the social desirability bias to which survey-based methods (such as surveys of health professionals regarding their usual practice relating to alcohol screening and intervention) may be susceptible.

Previous studies have found different rates of alcohol problems, screening and intervention, although the hospital setting, patient population, methodology and variable definitions were not always comparable. A chart review of orthopaedic admissions at Dunedin Hospital in 1992 found that ‘alcohol misuse’ was identified in 14% of records, with 36% of these records documenting some action taken as a result of the identified problem (ranging from observation for alcohol withdrawal to referral to alcohol services).[24]

An emergency department-based study at Auckland City Hospital in 2000 found that 35% of injured attendees reported drinking prior to their injury;[25] a figure higher than estimates from other countries using the same methodology.[26]

In the USA, both a survey of consecutive trauma inpatients[27] and a clinical trial that screened trauma admissions for inclusion[19] found the prevalence of problem drinking in trauma inpatients to be around 45%. A 1996 survey of trauma surgeons in the USA found that only 29% of trauma surgeons reported screening most of their patients for alcohol,[28] though this had increased to 56% by 2001.[29] However, use of formal screening questionnaires (such as CAGE, the Short Michigan Alcohol Screening Test or AUDIT) in that population remained at only 25% in 2001,[29] suggesting that much of the screening involved other potentially less valid tools. In one previous North American study, trauma patients were found to be less likely to have their problem drinking documented than other hospital patients.[30]

This study did not investigate the factors (injury, patient-related, or other characteristics) that may have influenced who did and did not get formally assessed for a potential drinking problem. However, all trauma patients are potentially eligible for screening. If the actual prevalence of problem drinking among patients admitted to trauma centres in New Zealand is similar to that in similar settings in the USA (as noted above), then only a small proportion of problem drinkers are being recognised, and even fewer are receiving a formal intervention in the hospital setting. While the management of these patients following discharge is unknown, the findings suggest missed opportunities to address a major public health problem.

Recommendations for an alcohol strategy for hospitals have been made in the United Kingdom.[31] The feasibility, acceptability, and resource implications for such a formal hospital-based alcohol strategy in New Zealand require serious consideration. Options for the provision of SBI in New Zealand hospitals include training general staff or funding specific services such as alcohol clinical nurse specialists. Given the likely prevalence of alcohol problems in hospitals, the provision of dedicated staff may be justified.

Alcohol counsellors are a standard part of the trauma team in some trauma centres in the USA,[19] and the effectiveness of such a service may warrant further exploration. The effectiveness of intervention in the hospital setting may also depend on the availability of resources for support in the community following discharge. The recommendations of the Royal Australasian College of Physicians *Alcohol Policy*[32]—including the training of all health care workers in the recognition and management of alcohol use disorders —require greater attention.

It is important that the most appropriate screening pathway and interventions in the New Zealand setting are carefully appraised prior to implementation. Future research should examine the effectiveness of SBI and other interventions in the New Zealand hospital context, and assess changes in alcohol screening and intervention rates if new services are introduced.

Conflict of interest statement: The authors have no conflicts of interest.

Author information: Jamie Hosking, Research Fellow, Clinical Trials Research Unit, University of Auckland, Auckland; Shanthi Ameratunga, Associate Professor in Epidemiology, Section of Epidemiology & Biostatistics, University of Auckland, Auckland; Chris Bullen, Associate Director, Clinical Trials Research Unit, University

of Auckland, Auckland; Ian Civil, Co-director, Trauma Services, Auckland City Hospital, Auckland; Alex Ng, Co-director, Trauma Services, Auckland City Hospital, Auckland; Anthony Rodgers, Director, Clinical Trials Research Unit, University of Auckland, Auckland

Acknowledgements: The New Zealand Population Health Charitable Trust provided a training endowment for Jamie Hosking.

The following people provided advisory input: Rhondda Paice, Rangi Dansey, and Lynn Tucker of the Trauma Service, Auckland City Hospital; Jennie Connor, Section of Epidemiology and Biostatistics, University of Auckland; John Langley, Injury Prevention Research Unit, University of Otago; and Doug Sellman, Director, National Addiction Centre, Christchurch School of Medicine and Health Sciences, University of Otago. We are also grateful to Ray Lin, Clinical Trials Research Unit, University of Auckland for statistical advice and Gay Richards at the Injury Prevention Research Centre, University of Auckland for information support.

Correspondence: Associate Professor Shanthi Ameratunga, Section of Epidemiology & Biostatistics, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7503; email: s.ameratunga@auckland.ac.nz

References:

1. Connor J, Broad J, Jackson R, et al. The burden of death, disease, and disability due to alcohol in New Zealand. Wellington: Alcohol Advisory Council of New Zealand; 2005. URL: http://www.alac.org.nz/DBTextworks/PDF/BurdenFull.pdf
2. Ezzati M, Lopez AD, Rodgers A, et al. Selected major risk factors and global and regional burden of disease. Lancet. 2002;360:1347–60.
3. Huakau J, Asiasiga L, Ford M, et al. New Zealand Pacific peoples' drinking style: too much or nothing at all? N Z Med J. 2005;118(1216). URL: http://www.nzma.org.nz/journal/118-1216/1491
4. Devlin NJ, Scuffham PA, Bunt LJ. The social costs of alcohol abuse in New Zealand. Addiction. 1997;92:1491–505.
5. Miller TR, Blewden M. Costs of alcohol-related crashes: New Zealand estimates and suggested measures for use internationally. Accid Anal Prev. 2001;33:783–91.
6. Poole GV, Griswold JA, Thaggard VK, et al. Trauma is a recurrent disease. Surgery. 1993;113:608–11.
7. Rivara FP, Koepsell TD, Jurkovich GJ, et al. The effects of alcohol abuse on readmission for trauma. JAMA. 1993;270:1962–4.
8. Deutch SR, Christian C, Hoyer S, et al. Drug and alcohol use among patients admitted to a Danish trauma centre: a prospective study from a regional trauma centre in Scandinavia. Eur J Emerg Med. 2004;11:318–22.
9. Carrigan TD, Field H, Illingworth RN, et al. Toxicological screening in trauma. J Accid Emerg Med. 2000;17:33–7.
10. Gentilello LM, Ebel BE, Wickizer TM, et al. Alcohol interventions for trauma patients treated in emergency departments and hospitals: a cost benefit analysis. Ann Surg. 2005;241:541–50.
11. Bien TH, Miller WR, Tonigan JS. Brief interventions for alcohol problems: a review. Addiction. 1993;88:315–35.
12. Cuijpers P, Riper H, Lemmers L. The effects on mortality of brief interventions for problem drinking: a meta-analysis. Addiction. 2004;99:839–45.

13. Moyer A, Finney JW, Swearingen CE, et al. Brief interventions for alcohol problems: a meta-analytic review of controlled investigations in treatment-seeking and non-treatment-seeking populations. Addiction. 2002;97:279–92.

14. Bushnell J, Martin G, National Advisory Committee on Health and Disability. Guidelines for recognising, assessing and treating alcohol and cannabis abuse in primary care. Wellington, N.Z.: National Health Committee; 1999.

15. Room R, Babor T, Rehm J. Alcohol and public health. Lancet. 2005;365:519–30.

16. Ministry of Health. A portrait of health: key results of the 2002/03 New Zealand health survey. Wellington, N.Z.: Ministry of Health; 2004.

17. Fleming MF, Mundt MP, French MT, et al. Brief physician advice for problem drinkers: long-term efficacy and benefit-cost analysis. Alcohol Clin Exp Res. 2002;26:36–43.

18. Dinh-Zarr T, Goss C, Heitman E, et al. Interventions for preventing injuries in problem drinkers. Cochrane Database of Systematic Reviews. 2005;2.

19. Gentilello LM, Rivara FP, Donovan DM, et al. Alcohol interventions in a trauma center as a means of reducing the risk of injury recurrence. Ann Surg. 1999;230:473–80; discussion 480–3.

20. Alcohol Advisory Council of New Zealand, Ministry of Health. National alcohol strategy 2000-2003. Wellington, N.Z.: Alcohol Advisory Council of New Zealand and the Ministry of Health; 2001.

21. Baker SP, O'Neill B. The injury severity score: an update. J Trauma. 1976;16:882–5.

22. Barker AN. Laboratory Handbook. LabPlus, Auckland District Health Board; 2005 [cited 29 November 2005]. Available from: http://www.adhb.govt.nz/LabPlusHandbook/handbook/manual.hlp

23. Land Transport Act 1998. URL: http://www.ltsa.govt.nz/legislation/lta1998.html

24. Hamilton MR, Menkes DB. How alert are hospital doctors to alcohol misuse among acute orthopaedic patients? N Z Med J. 1992;105:167–9.

25. Humphrey G, Casswell S, Han DY. Alcohol and injury among attendees at a New Zealand emergency department. N Z Med J. 2003;116(1168). URL: http://www.nzma.org.nz/journal/116-1168/298

26. Borges G, Cherpitel C, Orozco R, et al. Multicentre study of acute alcohol use and non-fatal injuries: data from the WHO collaborative study on alcohol and injuries. Bull World Health Organ. 2006;84:453–60.

27. Schermer CR, Bloomfield LA, Lu SW, et al. Trauma patient willingness to participate in alcohol screening and intervention. J Trauma. 2003;54:701–6.

28. Danielsson PE, Rivara FP, Gentilello LM, et al. Reasons why trauma surgeons fail to screen for alcohol problems. Arch Surg. 1999;134:564–8.

29. Schermer CR, Gentilello LM, Hoyt DB, et al. National survey of trauma surgeons' use of alcohol screening and brief intervention. J Trauma. 2003;55:849–56.

30. Bostwick JM, Seaman JS. Hospitalized patients and alcohol: who is being missed? Gen Hosp Psychiatry. 2004;26:59–62.

31. Royal College of Physicians of London. Alcohol - can the NHS afford it? Recommendations for a coherent alcohol strategy for hospitals: a report of a working party of the Royal College of Physicians. London: Royal College of Physicians of London; 2001.

32. Royal Australasian College of Physicians. Alcohol Policy: Using evidence for better outcomes. Sydney: Royal Australasian College of Physicians; 2005.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Vincristine for the treatment of Kasabach-Merritt syndrome: recent New Zealand case experience

Kimberley Thomson, Ralph Pinnock, Lochie Teague, Rachel Johnson, Noel Manikkam, Ross Drake

Abstract

Aims To present a case series showing efficacious use of vincristine in treating Kasabach-Merritt syndrome (KMS).

Methods The case notes of four children treated for KMS by the authors with corticosteroids and vincristine were reviewed. Specific attention was paid to the efficacy and adverse effects of each therapeutic agent.

Results The age of presentation ranged from birth to 11 months. Initial treatment with high dose corticosteroids was uniformly ineffective, and in 2 cases, prolonged use caused significant side-effects. Subsequent or concurrent treatment with vincristine was effective and well-tolerated, with no discernable side effects. The only complications were line-related.

Conclusions Kasabach-Merritt syndrome is rare, but it is associated with significant morbidity and mortality. No definitive treatment regime has been established, but the authors suggest that vincristine should be considered a first-line agent, and that the use of systemic corticosteroids should not be routine.

Kasabach-Merritt syndrome (KMS) was first described by Haig Haigouni Kasabach and Katharine Krom Merritt in 1940.[1] The syndrome results in a consumptive coagulopathy[2,3] from platelet trapping and aggregation within a specific type of haemangioma, and can have a high mortality rate.

The haemangioma is often on the skin but can be present anywhere, including retroperitoneal organs, the mediastinum, the pelvis, visceral organs, or the mesentery. For skin lesions, the mortality rate, with treatment, is under 10%, but retroperitoneal tumours have a mortality rate of approximately 60%.[3] The overall mortality rate is between 12 and 50%[2–5] with death occurring from severe haemorrhage related to disseminated intravascular coagulation, local invasion of vital structures, high output cardiac failure, multi-organ failure, or sepsis.[3]

The lesion associated with KMS is distinct from benign haemangioma of infancy.[6] It is an extremely fast-growing, aggressive, and locally invasive lesion with two histological patterns, kaposiform haemangioendothelioma (KHE) or tufted angioma (TA). Occasionally a lesion will have features of both types suggesting that they may be part of a continuum.[3] This altered histology is probably responsible for the platelet activation and consumptive coagulopathy.

Treatment aims to involute the tumour to prevent significant morbidity or mortality, or in response to a life-threatening event. Surgical excision is curative but most lesions are not amenable to this option. Historically, the first-line of treatment has been high-dose systemic corticosteroids. However, up to two-thirds of lesions will not

respond to corticosteroids, or will quickly relapse once treatment is discontinued.[7] Also, this treatment is not without its own troubling adverse effects. A number of alternative therapies have been tried with variable results, including interferon α-2a and 2b,[8] radiation therapy, and chemotherapeutic agents such as vincristine and actinomycin D.

Interferon has largely been abandoned due to a high association with the development of spastic diplegia,[2,3] bone marrow toxicity, and fatal aspiration pneumonias.[8] Radiation therapy, employed by Kasabach and Merritt with their index case,[1,3] poses the risk of local malignancies developing in later childhood, as well as causing the arrest of bony growth.[3] Vincristine and other chemotherapeutic agents have been shown to be efficacious in some children.[1–18]

The case study literature for the successful employment of vincristine is increasing, and our own case series presents further evidence that this medication can be used safely and effectively in the treatment of KMS.

Case experience

Case 1—Case 1 is a boy who was born with a large haemangioma on his right upper arm. He presented shortly after birth with generalised seizures and required ventilatory support for several days. Investigation revealed a large left-sided intracranial haemorrhage, thrombocytopenia and a severely disrupted coagulation profile and lead to the clinical diagnosis of KMS. No biopsy was taken because of his condition.

He was initially supported with red blood cell, platelet, and fresh frozen plasma transfusions and treated with phenobarbitone for the seizures and tranexamic acid to reduce his bleeding risk. He was commenced on hydrocortisone, which was subsequently converted to high dose prednisolone at 2 mg/kg/day for 2 weeks before increasing to 5 mg/kg/day for a period of 12 weeks. Prednisolone was weaned over an 8-week period at 14 weeks of age after he was diagnosed with *Pneumocystis carinii* pneumonia following an admission to hospital for respiratory distress.

Other adverse effects of corticosteroid therapy included hypertension requiring beta-blocker therapy, a marked Cushingoid appearance, and the requirement for stress dose corticosteroid therapy during intercurrent illnesses for several months after discontinuation of this therapy. He had a line sepsis with a coagulase negative *Staphylococcus species* while on steroid therapy.

At 2 weeks of age, vincristine was commenced on a weekly basis and weaned to fortnightly administration after 17 weeks following the restoration of normal platelet levels, the lesion having reduced in size appreciably by this stage. However, Case 1 required a second, shorter course when his haemangioma demonstrated growth on the prolonged interval.

Case 2—Case 2 is a girl who, as an incidental finding during assessment for an acute respiratory illness, was noted to have an area of redness on the anteromedial aspect of her right knee at 5-months of age. The lesion was initially treated as a cellulitis but an X-ray and orthopaedic referral were sought after it rapidly increased in size whilst on antibiotics.

The X-ray suggested a possible vascular malformation and a biopsy was reported as a benign vascular lesion, although a specific diagnosis was not provided. The tumour continued to grow aggressively, extending into the pelvis and caused a flexion contracture at the knee. Diagnosis of KMS was made at 11 months of age, when preparatory blood tests revealed thrombocytopaenia and a consumptive coagulopathy. Formal review of the histology then confirmed a Kaposiform haemangioendothelioma.

Figure 1. Case 2 before treatment

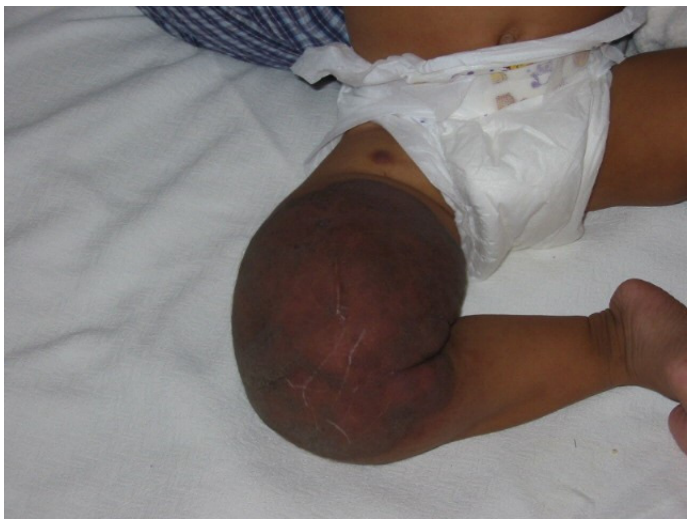
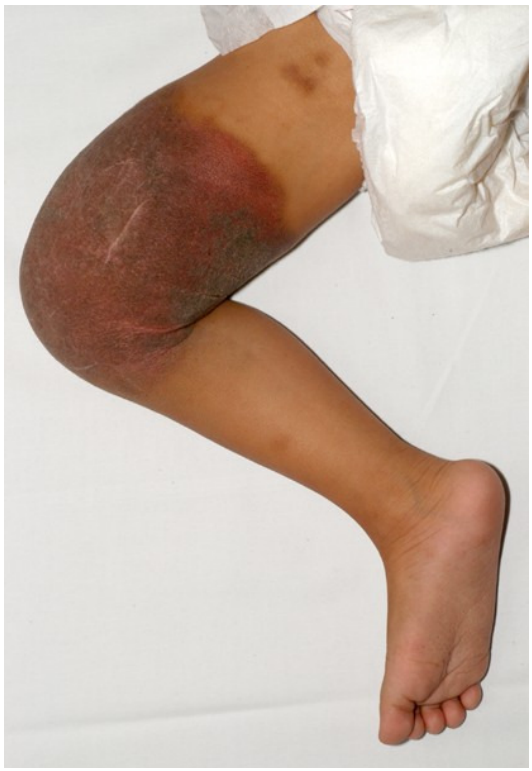


Figure 2. Case 2 end of treatment



Case 2 was commenced on tranexamic acid, promethazine at night for disturbing itch, and vincristine. Prednisone at 2 mg/kg/day and the insertion of a central line were withheld for 13 weeks because of recurrent ear and chest infections. The central line insertion was complicated by a prolonged wound haematoma despite perioperative platelet transfusions and the prednisone dose was weaned after 4 weeks following an admission to hospital for septicaemia and line sepsis with Lancefield group G streptococcus. The weaning phase was prolonged as an intercurrent illness, 9 weeks into weaning, required stress dose corticosteroids. The subsequent wean was completed successfully over a 12-week period.

There was a slow reduction in the size of the lesion but vincristine continued for 16 months before the platelet number returned to normal, at which time her coagulopathy had largely corrected.

Case 3—Case 3 is a boy, who was born with a large vascular malformation of his left arm, extending from the shoulder to the wrist. The diagnosis of KMS was made because of a marked thrombocytopenia, coagulopathy and a blood film consistent with microangiopathic haemolysis. By his second day of life he had developed high output cardiac failure requiring diuretic therapy, and phototherapy was instituted for a raised bilirubin level. He required regular platelet and red blood cell transfusions, and was started on tranexamic acid, Vitamin K, and prednisolone at 2.5 mg/kg/day.

Prednisolone was weaned after 17-days of therapy following a central line infection with *Staphylococcus aureus*. The central line insertion had been complicated by a prolonged wound haematoma with ooze, and had to be removed when it became blocked. He had a further central line sited soon after and this was able to be maintained until completion of his vincristine course despite the development of a *Klebsiella pneumoniae* sepsis, 4 months after insertion. He was not on corticosteroids at this time.

Vincristine was instituted on day 11 of life and was discontinued after 12 weeks when the platelet count had remained consistently above 50 for 3 weeks. Unfortunately, a further course had to be initiated 3 weeks later when the platelet count dropped to 11. The second course was continued for 19 weeks during which time the lesion achieved a considerable size reduction, the platelet count had returned to normal after 3 weeks of this second course.

Case 4—Case 4 is a female, born in Europe, with a blue discolouration to the right side of her neck. Doppler ultrasound indicated a vascular tumour, and at 2 weeks of age she commenced systemic corticosteroids. A follow-up ultrasound 6 weeks into treatment indicated only remnants of the lesion and therapy was discontinued. Case 4 and her family then immigrated to New Zealand and presented to hospital at 10 weeks of age when her parents noticed a painful lump in the region of the previous lesion. On examination, she was found to have a 7×9 cm tender, indurated purpuric mass extending from the side of her neck into the supraclavicular fossa. Routine preoperative blood tests revealed thrombocytopaenia, and an abnormal coagulation profile, and the blood film showed microangiopathic haemolysis. No resection or biopsy was undertaken due to the bleeding risk.

Figure 3. Case 4 before treatment

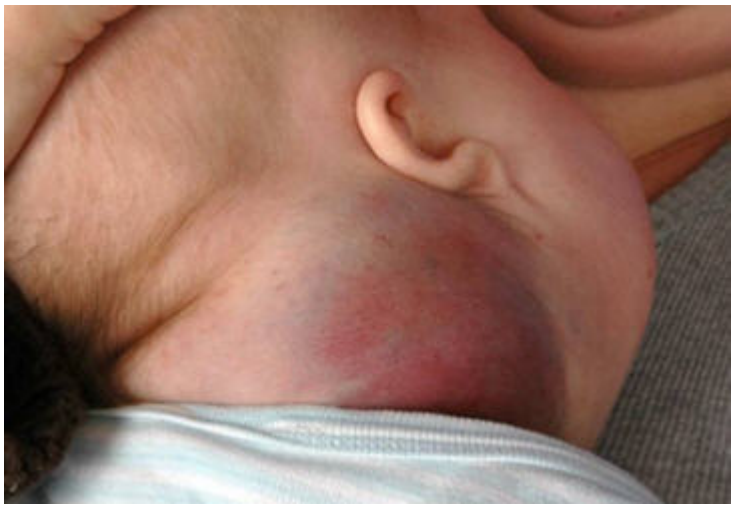
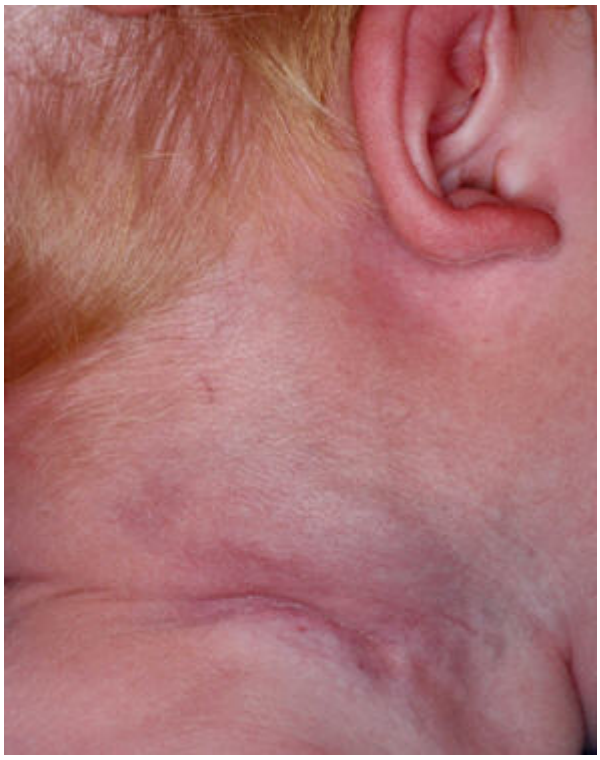


Figure 4. Case 4 end of treatment



She was restarted on prednisolone at 4mg/kg/day but after two weeks of treatment the dose was reduced to 2mg/kg/day because of a marked Cushingoid appearance and improvement of the thrombocytopaenia. However, the tumour steadily increased in size and vincristine was commenced three weeks into corticosteroid therapy. Tumour growth abated on commencing vincristine and after 11 weeks of treatment together with some reduction in tumour size, it was decided to reduce vincristine treatment to

fortnightly. However, simply missing the one dose led to an increase in tumour growth and therefore vincristine was given weekly for a further 7 weeks. The tumour remained static with further reduction in vincristine doses and treatment was eventually stopped after a total of 26 doses. The tumour subsequently involuted.

Table 1. Diagnostic features and effects of disease

Variable	Case 1	Case 2	Case 3	Case 4
Age at diagnosis	1 day	11 months	Birth	10 weeks†
Location of lesion	Right upper arm	Right knee and thigh	Left upper and lower arm	Right side of neck
Platelet numbers at diagnosis	26	16	<10	16
Coagulation profile at diagnosis	APTT 89 INR 2.3 Fibrinogen 0.6	APTT 40 INR 1.1 Fibrinogen 1.8 D-dimer >4000	APTT 31 INR 1.4 Fibrinogen 0.6	APTT 32 INR 1.2 Fibrinogen 0.9
Adverse effects of KMS and/or lesion	Left sided IVH Developmental delay Right upper and lower limb undergrowth	Right knee flexion deformity Right leg overgrowth	Cardiac failure Anaemia Neonatal jaundice Mild fixed flexion deformity of left elbow	Restricted neck movement Right pinna distortion

†Recurrence of lesion first noted and treated at birth.

Table 2. Features of Vincristine therapy

Variable	Case 1	Case 2	Case 3	Case 4
Dose (mg/kg)	0.025 (2 weeks) then 0.05	0.05	0.05	0.05
No. of weekly treatments	Course 1 – 17 Course 2* – 6	27	Course 1 – 12 Course 2† – 19	Course 1 – 11 Course 2* – 7
No. of fortnightly treatments	Course 1 – 2 Course 2* – 3	6	Nil	Course 2* – 4
No. of monthly treatments	Nil	6	Nil	Course 2* – 4
Side effects	Nil	Nil	Nil	Nil
Maximum circumference before treatment (cm)	20.5 (neonate)	31.5	22 (neonate)	7x9
Maximum circumference end of treatment (cm)	25 (8 months)	25.5	20.5 (8 months)	5
Duration until platelets >50 (weeks)	9	59	Course 1 – 10 Course 2† – 20	3‡
Duration until resolution of coagulation abnormalities (weeks)	12	68	20	4¥

*Second course instituted after lesion showed evidence of growth; †Second course instituted after platelet number dropped below 50; ‡Steroid therapy only; ¥Steroid therapy and 1 dose of vincristine.

Discussion

This case series reflects the difficulties associated with managing KMS, the morbidity associated with traditional high dose corticosteroid therapy and the effectiveness of vincristine therapy. The lesion often provides a diagnostic dilemma, as only around 50% are present at birth and these are often mistaken for benign haemangiomas of infancy. The result is a false reassurance to health professionals and parents alike that the lesions are harmless and will spontaneously involute.

KMS requires the haematological picture of thrombocytopenia; and potential abnormalities of microangiopathic haemolytic anaemia, deranged coagulation profile such as prolonged APTT or INR, hypofibrinoginaemia, and elevated fibrin degradation products, including elevated D-dimer levels.[3,8] MRI of the lesion shows multiple tissue planes with surrounding oedema, signal voids which represent haemosiderin deposits, and, often, dilated feeding and draining vessels.[3]

Histology is a useful aid in diagnosis. Kaposiform haemangiendotheliomas are locally aggressive tumours with sheets and lobules of round and spindle-shaped endothelial cells, which infiltrate the surrounding tissue layers. Tufted haemangiomas consist of groups of dermal capillary tufts, which are often much larger than those seen in infantile haemangiomas. The differential diagnosis includes cancer, but the tumours associated with KMS have not been shown to metastasise.[9]

First-line treatment has (traditionally) been systemic corticosteroids at a dose of 2 to 4 mg/kg/day.[3,4] However, up to two-thirds of lesions will not respond, or will quickly relapse once treatment is discontinued.[7] The morbidity of prolonged use of high-dose systemic corticosteroids are well-known, and include Cushingoid facies, hypertension (up to 100% in one series),[8] adrenal suppression, immunosuppression, growth retardation, bone demineralization, thinning of skin, irritability, and gastritis. Two of our four patients showed some of these features and one patient suffered significant morbidity.

Vincristine, cyclophosphamide and other chemotherapeutic agents have been shown to be efficacious in children and the use of vincristine has been described in many case series.[1–18] Vincristine is found in the leaves of the plant *Catharanthus roseus,* more commonly known as the periwinkle.[10] It is a vinca alkaloid, which inhibits angiogenesis through the prevention of cell mitoses in metaphase by preventing tubulin polymerisation, thus preventing microtubule formation, and induction of microtubule depolymerisation.[10,11] Potential side effects of vincristine include gastrointestinal upset, constipation, fever, headache, and peripheral and autonomic neuropathies.

Vincristine can be used to treat life-threatening vascular malformations. It was administered to a 3-month-old girl with a tumour that compromised the airway, after she failed to respond to 3 weeks of corticosteroid therapy.[7] The tumour regressed dramatically with six doses of vincristine. However, the child developed a bowel obstruction during treatment, possibly secondary to an autonomic neuropathy. This case was not one where KMS had developed, but treatment delays were considered unacceptable.

Payarols et al[4] described the case of a 9-month-old baby with a large tumour in the left supraclavicular fossa, complicated by KMS. After 55 days of systemic

corticosteroid treatment, vincristine was added for 5 weeks. The tumour decreased in size, haematological parameters returned to normal and the tumour was undetectable after 1 year of follow-up.

In a multicentre, retrospective study of 15 patients with KMS[10] who had previously been treated with another therapeutic agent, 14 had received systemic corticosteroids, vincristine was used. Four weeks was the average response time to improve the platelet count and 4 of the 15 cases relapsed; however they responded well to a second course of vincristine. Three children developed minor side effects of abdominal pain, transient loss of the deep tendon reflexes, and irritability. The reviewers concluded that vincristine was safe, effective, and should be considered as a first-line treatment in cases of KMS.

Yang and colleagues[12] describe a case where vincristine was not effective in treating KMS after the failure of several other treatment modalities. However, the length of vincristine treatment was not stated before the curative option of absolute ethanol embolectomy was administered. Ethanol embolisation is a curative technique where the vascular malformation is embolised under fluoroscopic angiographic guidance. It is indicated if medical treatment is unsuccessful, [13] and if the characteristics of the lesion are suitable.

It carries the usual risks associated with angiography (deterministic skin effects of radiation, perforation, bleeding, infection[13,14]) and with embolisation (post embolisation oedema and haemorrhage, multistage procedures, treatment failure, and embolisation of other important end arteries[13,14]). Angiography also requires relatively stable coagulation tests prior to arterial puncture, so may not be suitable in most cases of KMS.

Conclusion

It is the clinical impression of the authors that this case series adds to the previous literature, that vincrisitne should be considered early in the management of Kasabach Merritt syndrome. In addition, there is nothing to suggest that it could not be used as a first line agent as the low dose recommended has evidence showing it to be effective, safe, and well-tolerated..

High-dose corticosteroids are often ineffective in the treatment of KMS and the adverse effects well-documented and, as seen in this case series, can be significant. Moreover, clinicians should not underestimate the financial and psychological benefits for the child and family of weekly vincristine treatments in an outpatient setting over the possible adverse effects, cosmetic implications, and hospitalisations associated with high-dose steroid therapy.

Conflict of interest statement: There are no conflicts of interest.

Author information: Kimberley J Thomson, Radiology Registrar[1]; Ralph Pinnock, Paediatrician[2]; Lochie Teague, Paediatric Oncologist[3]; Rachel Johnson, Paediatric Registrar[4]; Noel Manikkam, Paediatrician[5]; Ross Drake, Paediatrician[2]

1. Department of Radiology, Christchurch Hospital, Christchurch
2. Department of Paediatrics, Starship Hospital, Auckland
3. Auckland Paediatric Oncology, Starship Hospital, Auckland
4. Department of Paediatrics, Middlemore Hospital, Auckland
5. Paediatrics, Waikato Hospital, Hamilton

Correspondence: Kimberley Thomson, Radiology Registrar, Radiology Department, Christchurch Public Hospital, Private Bag 4710, Christchurch.; Email: Kimberley.Thomson@cdhb.govt.nz

References:

1. Kasabach HH, Merritt KK. Capillary hemangioma with extensive purpura: report of a case. American Journal of Diseases of Children. 1940;59:1063–70.
2. Hall GW. Kasabach-Merritt syndrome: Pathogenesis and management. British Journal of Haematology. 2001;112:851–62.
3. Maguiness S, Guenther L. Kasabach-Merritt syndrome. Journal of Cutaneous Medicine & Surgery. 2002;6:335–9.
4. Perez Payarols J, Pardo Masferrer J, Gomez Bellvert C. Treatment of life-threatening infantile hemangiomas with vincristine. New England Journal of Medicine. 1995;333:69.
5. Vin-Christian K, McCalmont TH. Frieden IJ. Kaposiform hemangioendothelioma: An aggressive, locally invasive vascular tumor that can mimic hemangioma of infancy. Archives of Dermatology. 1997;133:1573–8.
6. Sarkar M, Mulliken JB, Kozakewich HPW, et al. Thrombocytopenic coagulopathy (Kasabach-Merritt Phenomenon) is associated with Kaposiform hemangioendothelioma and not with common infantile hemangioma. Plastic & Reconstructive Surgery. 1997;100:1377–86.
7. Moore J, Lee M, Garzon M, et al. Effective therapy of a vascular tumor of infancy with vincristine. Journal of Pediatric Surgery. 2001;36:1273–6.
8. Wananukul S, Nuchprayoon I, Seksarn P. Treatment of Kasabach-Merritt syndrome: A stepwise regimen of prednisolone, dipyridamole, and interferon. International Journal of Dermatology. 2003;42:741–8.
9. Hu B, Lachman R, Phillips J, et al. Kasabach-Merritt syndrome-associated kaposiform hemangioendothelioma successfully treated with cyclophosphamide, vincristine, and actinomycin D. Journal of Pediatric Hematology/Oncology. 1998;20:567–9.
10. Haisley-Royster C, Enjolras O, et al. Kasabach-Merritt phenomenon: a retrospective study of treatment with vincristine.[erratum appears in J Pediatr Hematol Oncol 2002;24:794. Journal of Pediatric Hematology/Oncology. 2002;24:459–62.
11. Perez J, Pardo J, Gomez C. Vincristine—an effective treatment of corticoid-resistant life-threatening infantile hemangiomas. Acta Oncologica. 2002;41:197–9.
12. Yang Y-H, Lee P-I, Lin K-H, Tsang Y-M. Absolute ethanol embolotherapy for hemangioma with Kasabach-Merritt syndrome. Chung-Hua Min Kuo Hsiao Erh Ko i Hsueh Hui Tsa Chih. 1998;39:51–4.
13. Stith JA. Current diagnostics in office practice evaluation and non-operative management of vascular malformations. Current Opinion in Otolaryngology & Head and Neck Surgery. 2000;8:448–53.
14. Yakes WF, Haas DK, Parker SH, et al. Sympathetic vascular malformations: Ethanol embolectomy. Radiology. 1989;170:1059–66.
15. Ghadially FN. Ultrastructural pathology on the cell and matrix. Cristalline inclusions. London: Butterworth; 1988, p978.
16. Buckmiller LM. Update on hemangiomas and vascular malformations. Current Opinion in Otolaryngology & Head & Neck Surgery. 2004;12:476–87.
17. Blei F, Karp N, Rofsky N, et al. Successful multimodal therapy for kaposiform hemagioendothelioma complicated by Kasabach-Merritt. Pediatric Hematology & Oncology. 1998;15:295–305.

18. Enjolras O. Vascular tumors and vascular malformations: Are we at the dawn of a better knowledge? Pediatric Dermatology. 1999;16:238–41.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Concussion clinic referral demographics and recommendations: a retrospective analysis

Hamish Alexander, Nicola Shelton, Jacob Fairhall, Harry McNaughton

Abstract

Objective To review the demographic factors, mechanism of injury and treatment recommendations for patients attending a concussion clinic in New Zealand.

Methods Retrospective analysis of data for all patients attending a concussion clinic in a single centre over a 2-year period. Data was collected via an electronic database and written clinical records.

Results Data from a total of 161 patients was collected; 8 patients did not attend clinic appointments, yet their mechanism of injury was available from referral notes. 42 (26%) patients were diagnosed as not having a mild traumatic brain injury (TBI). Of the remainder, 72 (47%) had a mild TBI and 36 (22%) had moderate or severe TBI; 21% of attendees were injured in sporting accidents with 19% injured in motor vehicle accidents and 17% in falls. More treatment recommendations were made in those patients diagnosed with TBI than those with no TBI (p=0.038). Occupational therapy was the most commonly recommended treatment.

Conclusions Considering the high number of injuries with mild TBI that occur every year, there was a relatively small number seen in the Wellington area concussion clinic. Only half of clinic attendees had had a mild TBI. Treatment recommendations were similar throughout patient diagnostic groups; occupational therapy input was probably offered because it was resourced by the clinic funder. Further research is required into return to work, emotional and cognitive outcomes.

'Concussion' is often used as a synonym for mild traumatic brain injury (MTBI), a common problem in primary and secondary healthcare.

The World Health Organization defines MTBI as an acute brain injury resulting from mechanical energy transmission to the head from external physical forces. It usually has one or more of the following: confusion or disorientation; loss of consciousness for 30 minutes or less; a Glasgow Coma Scale Score of 13–15, 30 minutes or later after injury;[8] post-traumatic amnesia for less than 24 hours; or other transient neurological abnormalities such as focal neurological signs, seizure, or cerebral contusion not requiring surgery.

These manifestations must not be due to other conditions, penetrating brain injury or intoxication. Common causes include assault, sport, and accidents involving motor vehicles and bicycles.

Post-concussion symptoms such as headache, dizziness, poor sleep, depression, and emotional lability may occur in many trauma patients even without diagnosis of MTBI.[2] Attentive deficits in visual and cognitive domains are common. Ability to withstand distraction and multi-task is also impaired.

Concussion is associated with a significant socioeconomic cost because it affects a predominantly productive, young age group.[3] Although most patients recover within 6 to 12 weeks, longer recovery is associated with increased age, previous head injury, and pre-existing medical conditions.[1]

Management of persisting post-concussion symptoms involves careful diagnosis and consideration of the physical, neuropsychological, social, and emotional factors contributing to the patient's impairment.[4]

Randomised controlled trials have shown that intervention from a specialised service reduces the social disability and minor post-concussive symptoms after head injury compared with standard services.[7] Often this intervention is delivered in the setting of a specialised concussion clinic, such as that operating at Capital and Coast District Health Board (C&CDHB), offering a multidisciplinary approach.

The C&CDHB Adult Concussion Clinic operates from the Capital Coast Rehabilitation department. Paediatric referrals (0-15 years) are seen by a child development team.

Referrals for assessment and treatment of the ongoing effects of a mild TBI are received from general practitioners, Accident Compensation Corporation (ACC) case managers, and ward & emergency department staff. Referrals from the ward are usually made via medical, physiotherapy or occupational therapy (OT) staff. Funding is via ACC under the Mild Traumatic Brain Injury contract.

The service provides assessment by a rehabilitation physician, screening assessment by a neuropsychologist, and may also include functional assessment by an Occupational Therapist (OT). Following approval from the ACC Case manger, up to 12 OT sessions can be provided.

The aim of this analysis is to identify the demographic trends and mechanisms of injury of patients presenting to the concussion clinic, and to identify recommendations for ongoing management, in relation to diagnosis.

Method

A retrospective analysis of concussion clinic data was performed for 1 January 2003 to 31 December 2004. Data was collected via electronic records. In the event that electronic records were unavailable, paper clinical records were accessed.

The following data were collected: age, sex, mechanism of injury, medical diagnosis, and recommendations following medical and neuropsychological assessment. Patient demographics were collated.

Medical diagnosis was grouped into one of several categories: external force to the head with no traumatic brain injury, mild traumatic brain injury, and greater than mild traumatic brain injury (i.e. moderate to severe TBI), based to the medical report. Categorical variables were analysed using the Chi-squared (χ^2) statistical measure.

Results

A total of 161 (101 male, 62 female) patients were included in the study; their mean age was 38 years (SD 14).

The mechanism of injury is shown in Table 1. In six cases, the mechanism of injury was unclear due to post-traumatic amnesia or alcohol, and was recorded as unknown. Falls were divided into 'fall', defined as fall from standing (e.g. trip over curb) and

'fall from height' greater than standing (eg. fall from ladder). Involvement of alcohol and sub-classifications of sport are also displayed.

Table 1. Mechanism of injury

Variable	n	(% of total, to nearest whole number)
Mechanism of injury		
Fall	**28**	**(17%)**
Fall from height	**14**	**(9%)**
Motor vehicle accident	**31**	**(19%)**
Sport	**35**	**(22%)**
- Cycling	15	(9%)
- Contact sport	12	(7%)
- Water sport	3	(2%)
- Other	5	(3%)
Assault	**28**	**(17%)**
Workplace trauma	**12**	**(7%)**
Trauma not otherwise specified	**7**	**(4%)**
Unknown	**6**	**(4%)**
Alcohol present	**23**	**(14%)**

The diagnosis documented by the physician was divided into three categories. Medical considerations included loss of consciousness, duration of post-traumatic amnesia, seizure activity, and post-concussion symptoms. Table 2 shows the diagnoses in absolute and proportional terms respectively.

Table 2. Medical diagnoses

Variable	n	(% of total)
External force to the head not meeting definition for TBI	42	(26%)
Mild traumatic brain injury (MTBI)	75	(47%)
Moderate or severe traumatic brain injury	36	(22%)
Did not attend	8	(5%)

The recommendations following each consultation are recorded in Table 3. Multiple recommendations were made for many patients. The return to work program was administered by the clinic's occupational therapist (OT), and is accounted for under both OT and return to work headings.

The recommendations made following medical and neuropsychological consultations are shown against diagnosis in Table 4.

Overall there were significantly more treatment recommendations (OT and other vs nil) made for those patients who had diagnosis of TBI (of any severity) compared to no TBI (χ^2=6.555, p=0.038).

Table 3. Medical and neuropsychological recommendations

Variable	Medical recommendations		Neuropsychological recommendations	
	n	(% of total)	n	(% of total)
Nil	38	(18%)	24	(14%)
Occupational therapy (OT)	39	(19%)	82	(46%)
Return to work programme	30	(14%)	22	(12%)
Onward referral	12	(6%)	3	(2%)
Counselling/Social work	10	(5%)	20	(12%)
Further investigations	7	(3%)	0	(0%)
Medication changes	35	(17%)	0	(0%)
Physiotherapy	7	(3%)	1	(0.5%)
Further neuropsychological assessment	2	(1%)	13	(7%)
Driving assessment	7	(3%)	0	(0%)
Driving restriction	23	(11%)	0	(0%)
Clinical psychologist	0	(0%)	9	(5%)
Mental health team	0	(0%)	2	(1%)

Table 4. Recommendations by medical diagnosis (n, % of total)

Variable	Nil	(% of total)	OT	(% of total)	Other	(% of total)
No TBI	18	(28%)	21	(33%)	25	(39%)
Mild TBI	23	(15%)	72	(48%)	54	(36%)
> Mild TBI	9	(13%)	42	(59%)	20	(28%)

There was no significant difference in recommendations (nil, OT, other) by diagnosis (no TBI, mild TBI, moderate/severe TBI) for medical recommendations (χ^2=2.773, p=0.60) but there was a significant difference (χ^2=13.795, p= 0.008) for neuropsychological recommendations.

Similarly, the relationship was non-significant for medical recommendations (χ^2=0.838, p=0.36) and significant for neuropsychology recommendations (χ^2=4.458, p=0.035) when the groupings were simplified: TBI/no TBI vs no treatment/any treatment.

These groups were further stratified to TBI/no TBI vs no treatment/OT (thus excluding other treatments). Analysis of these groups showed again no significant difference between these groups in medical recommendations (χ^2=1.266, p=0.261) and a significant difference for neuropychology recommendations (χ^2=6.476, p=0.011). These analyses were considered because a proportion of the medical recommendations concerned symptomatic management of headache and dizziness which might apply whatever the diagnosis.

Discussion

Our patients were predominantly male, with sport injuries and assaults (with alcohol involvement) being common modes of presentations. This is similar to figures in the neurotrauma and rehabilitation literature.[2] A previous local study showed comparable mechanisms of injury and mean age.[5] It was unfortunately not always clear if alcohol was directly involved in the injury from the patient notes.

It is interesting to compare sporting mechanisms of injury. Cycling was the dominant sport responsible for MTBI referral. Rugby and soccer presentations were less frequent. This contrasts with most of the world literature where contact sports are responsible for a greater number of presentations.

The relatively small number of rugby presentations is surprising, considering the prevalence of rugby as a sport in the Wellington region—there may be a bias in presentation with players not being appropriately referred because of a lack of perceived need or accessing alternative services, particularly sports medicine doctors.

Recommendation for further investigations was low (11% of patients). This likely reflects that the majority of patients were referred by services that had investigated appropriately at the time of injury and/or investigations are not usually indicated outside the acute phase for mild TBI.

It was anticipated by the researchers that diagnosis would impact on recommendations made. However, we identified no significant difference in recommendations arising from medical consultations between the three diagnostic groups, but a significant difference from the neuropsychological consultations.

One factor that may contribute to these results is that OT sessions were contractually funded (pending approval) for clinic attendees. OT was by far the most common recommendation, made for 43% of patients on medical assessment, and 65% on neuropsychological assessment. Because the OT sessions were funded, it is likely that there was a low threshold for recommending OT, even in the absence of diagnosed TBI.

Another factor contributing to these findings is that because the concussion clinic is a secondary referral service with a proportion of attendees being referred due to ongoing symptoms (rather than a representative sample from all head injuries), then regardless of diagnosis the patients' ongoing symptoms may require treatment.

Patients are referred to the concussion clinic from the emergency department, ACC case managers, inpatient services, and GP clinics. In the latter group, referrals are made due to ongoing issues related to the injury, necessitating further treatment or advice regardless of the diagnosis. These patients are already in a subclass of those with persistent post-concussive symptoms. In these cases, Concussion Clinic recommendations are important in symptom management and return to societal, occupational, and family roles.

During consultations, advice was given regarding vestibular rehabilitation, modification of activity levels, and avoidance of 'at risk' activities and behaviours. Data pertaining to such advice was not collected however.

The concussion clinic's funding is based on the premise that MTBI is a syndrome with societal and economic impact. The clinic is potentially a valuable tool.

These results suggest at least four separate functions for such a clinic:

- Clear diagnosis to separate those with TBI from those not meeting diagnostic criteria for TBI,
- Identification of residual cognitive and emotional impairments for those with TBI,
- Clear diagnosis and adequate management plan for those with moderate and severe TBI who may have been previously mislabelled as having only mild TBI,
- A primary focus on improving functional performance (rather than simply symptom management) while acknowledging that adequate symptom management may be important for some people in allowing them to achieve functional goals.

The numbers seen in the clinic suggest that it currently does not serve much of a screening role for people with more than very mild TBI, for example seeing all patients for one-off review that have been admitted to hospital with TBI of any severity. Unfortunately this is probably the only role of such a clinic that is supported by strong evidence from randomised controlled trials.[6,7]

So that the precise functions of mild TBI clinics, appropriate volumes, and timing of assessments can be determined, more comprehensive research measuring appropriate end points for the subjects along with costs is necessary.

Conflict of interest statement: There are no conflicts of interest.

Author information: Hamish Alexander, House Surgeon; Nicola Shelton Physiotherapist; Jacob Fairhall, Neurosurgical Registrar; Harry McNaughton, Rehabilitation Physician; Capital and Coast DHB, Wellington

Correspondence: Dr H Alexander, Hutt Valley DHB, PO Box 31-907 , Lower Hutt. Fax: (04) 566 6999, email: Hamish.Alexander@huttvalleydhb.org.nz

References:

1. Alexander MP. Mild traumatic brain injury: pathophysiology, natural history, and clinical management. Neurology. 1995;45:1253–60.
2. Chambers J, Cohen SS, Hemminger L, et al. Mild traumatic brain injuries in low-risk trauma patients. J Trauma. 1996;41:976–80.
3. Maull KI. Head injury and multisystem trauma. Philadelphia: WB Saunders; 1989.
4. Kushner D. Mild traumatic brain injury: Toward understanding manifestations and treatment. Archives of Internal Medicine. 1998;158:1617–24.
5. McNaughton H, Wadsworth K. Assessing the accuracy of hospital admission and discharge diagnosis of traumatic brain injury in a New Zealand hospital. N Z Med J. 2000;113:184–6. URL: http://www.nzma.org.nz/journal/113-1110/2182/content.pdf
6. Wade DT, King NS, Wenden F J, et al. Routine follow up after head injury: a second randomised controlled trial. J Neurol Neurosurg Psychiatry. 1998;65:177–83.
7. Von Holst H, Cassidy J D. Mandate of the WHO collaborating centre task force on mild traumatic brain injury. J Rehabil Med. 2004;Suppl.43:8–10.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

General practitioner diagnosis and management of acute knee injuries: summary of an evidence-based guideline

Gillian Robb, Duncan Reid, Bruce Arroll, Rod Jackson, Felicity Goodyear-Smith

Abstract

Aims To summarise evidence and key recommendations for general practitioner diagnosis and management of acute soft-tissue knee injuries, based on the New Zealand guideline.

Methods A multidisciplinary team developed the guideline by critically appraising and grading retrieved literature using the Graphic Appraisal Tools for Epidemiology, Clinical decision rules and the Scottish Intercollegiate Guideline Network. Recommendations were derived from resulting evidence tables.

Results For both diagnosis and management there is a paucity of good evidence to support diagnosis and treatment of internal derangements of the knee, hence some aspects of the guideline are guideline team consensus. Good evidence supports the use of the Ottawa Knee rules to guide decisions about the use of X-ray, and the Lachman test in diagnosing anterior cruciate ligament (ACL) tears. Evidence supports inclusion of proprioceptive training in rehabilitation programmes following ACL reconstruction and in people with ACL-deficient knees. There is good evidence that ultrasound is of little benefit, and there is no evidence that physiotherapy be routinely advocated following meniscectomy.

Conclusion This guideline provides an evidence-based framework for diagnosis and management of internal derangements of the knee following acute injury. Moreover, its development highlights significant gaps in the evidence base and identifies priorities for new research.

In 2001, the Accident Compensation Corporation (ACC) of New Zealand (NZ) identified nationwide variation in practice in the diagnosis and management of acute soft-tissue knee injuries. At that time, the American Academy of Orthopaedic Surgeons' "Clinical Guideline on Knee Injury" was the only available published guideline. This guideline had a broad scope, including patella dislocation/subluxation, contusion, and quadriceps tendon rupture in addition to ligament and meniscal injuries. However it was published as an algorithm with only a brief supporting document stating that it was developed by a multi-professional team of physicians and based on both evidence from the literature and consensus.[1] This guideline was limited to internal derangements of the knee and was developed following explicit guideline methodology.

The ACC scheme provides personal injury cover for all NZ citizens, residents, and temporary visitors to NZ on a no-fault basis. Knee injuries ranked second for new claims and on-going claims after low back injuries in their injury statistics for the 2000/2001 period, and the ACC Injury Statistics 2004 (Third Edition) for "sport claims" shows that the knee joint ranks within the top five injury sites for nearly all major sport and recreational activities.

Since these injuries represent a significant cost, ACC commissioned a specific acute soft-tissue knee injury guideline. This was developed by a multidisciplinary team, led

by the EPIQ Group (Effective Practice, Informatics and Quality Improvement) University of Auckland, under the auspices of the New Zealand Guidelines Group (NZGG).

The objective of the guideline is to provide evidence-based recommendations for the diagnosis and management of internal derangements of the knee in adults involving acute injuries to the menisci, collateral, and cruciate ligaments. It is primarily aimed at general practitioners and other primary contact practitioners such as physiotherapists.

Methods

A broad-based multidisciplinary team (orthopaedic surgery, general practice, physiotherapy, musculoskeletal radiology, musculoskeletal medicine, sports medicine) was convened in 2002 including nominated professionals and representatives for Māori, Pacific people (mostly of Samoan, Tongan, Niuean, or Cook Islands origin), and consumers. The team met twice over 12 months, with a teleconference to concur on the final draft of the guideline. There were also numerous consultations between members of the group during the guideline development process.

The Royal New Zealand College of General Practitioners (RNZCGP) endorsed the guideline, and the full document is available on the NZGG website (http://www.nzgg.org.nz).

The following diagnostic questions were considered by the team:

- The accuracy of the history and physical examination in diagnosing internal derangements in primary and secondary care settings.
- Indications for the use of X-ray in acute knee injuries.
- The accuracy and role of MRI in the diagnosis of internal derangements of the knee.

The following treatment questions involving effectiveness were considered by the team:

- "RICE" (rest, ice, compression, elevation).
- Aspiration in the first 24 hours for haemarthrosis.
- Medication: nonsteroidal anti-inflammatory medication (NSAID) or paracetamol.
- Bracing in the early non-operative management of acute knee injury.
- Physiotherapy modalities (aspects of rehabilitation and electrotherapy).
- Complementary therapies (acupuncture, chiropractic and osteopathy).
- Operative versus non-operative management of internal derangements.
- Postoperative management including bracing and aspects of rehabilitation (proprioceptive training, open versus closed kinetic chain exercise, and home-based programmes versus supervised physiotherapy)

Chronic and recurrent knee injuries, overuse injuries, arthritic conditions, injuries to the patella ligament and patellofemoral joint, bone bruises, fat pad impingement and entrapment, the iliotibial band syndrome, and surgical methods for managing meniscal and ligament injuries were excluded from the guideline.

For each clinical question, a comprehensive literature search was undertaken in the major electronic databases (Medline, CINAHL. EMBASE, AMED SPORT Discus and Current Contents). Searching also was undertaken using the Cochrane Database of Systematic Reviews, the Controlled Trials Register, Database of Abstracts of Reviews of Effectiveness (DARE), and the Cochrane Complementary Medicine Field Trials register.

Relevant internet sites were searched including PEDro (Physiotherapy Evidence Database), NHS clinical trials, Health Technology Assessments for NHS, and the National Guideline Clearing House. Reference lists of included studies were checked for additional studies.

For diagnostic questions, studies had to have a minimum of 35 participants;[2] blind assessment of the new test and the reference standard; a comparison of a reference test with the new test in >90% of people; and an appropriate spectrum of participants.

For management questions, only systematic reviews, meta-analyses, randomised trials, or quasi-randomised trials of interventions were included.

The evidence from the relevant studies was summarised into evidence tables (available on the NZGG website). Each study was critically appraised and graded using the Graphic Appraisal Tools for Epidemiology (GATE) (http://www.epiq.co.nz). Evidence statements relating to interventions were graded according to the NZGG "Grading system for guidelines". Clinical decision rules were graded according to the criteria described by McGinn et al.[3]

For each question, recommendations were developed based on all included studies using a "considered judgment" process (SIGN Guideline development process: http://www.sign.ac.uk/guidelines/fulltext/50/compjudgement.html).

Grading of the recommendations is based on the strength of the evidence and does not indicate the relative importance of the recommendations.

Results

Results are presented according to the grade of recommendation placed under these headings:

- Diagnosis,
- Referral to a specialist,
- Acute and postoperative management recommendations for internal derangements of the knee due to injury,

Recommendations

Diagnosis recommendations

"A" recommendations (supported by good evidence)

1. The Ottawa knee rule is a valid tool to guide the use of x-rays for excluding fractures in people with acute knee injuries in an emergency department setting.[4-7] The Ottawa rule is that knee x-ray series to exclude fracture are only required for acute knee injury in people with one or more of the following:
    - Aged 55 or older
    - Tenderness at the head of the fibula
    - Isolated tenderness of the patella
    - Inability to flex the knee to 90 degrees
    - Inability to walk four weight-bearing steps at time of injury and at examination.[4]
2. Lachman test in a secondary care setting is reasonably accurate in the diagnosis of anterior cruciate ligament tears and more accurate when acute pain and swelling have subsided at about 10 days.[8-12] The accuracy of the Lachman test in a primary care setting has not yet been established. The Lachman test is performed with the patient supine and the knee flexed to 20˚-30˚. The femur is stabilised by grasping just above the knee with one hand, while the other hand grasps the proximal tibia and gives a brisk forward tug. A positive test is when there is an increased anterior translation of the tibia on the femur and/or absence of a discrete end-point.[13]

"C" recommendations (supported by expert opinion only)

1. In patients with a haemarthrosis, X-ray is appropriate to check for fractures, otherwise the routine use of X-rays is not recommended.
2. There is insufficient evidence that any aspect of the history or clinical test other than the Lachman test for ACL tears is valid. However, familiarity with the typical mechanisms of injury and presenting symptoms for each diagnosis may assist clinicians in differentiating between common internal derangements.
3. Some clinical tests are likely to be useful in confirming a diagnosis in the context of an appropriate history.
4. The diagnosis of meniscal and cruciate ligament injuries to the knee can be made by MRI with a reasonable level of accuracy.[8, 14-19] However, many patients can be diagnosed without the need for this expensive investigation.
    a) Where there is an equivocal diagnosis specialists may consider MRI to clarify the diagnosis and inform treatment decisions.
    b) MRI should generally be used ahead of diagnostic arthroscopy.

Table 1. History and physical examination

History	Test	Diagnosis
Valgus injury Medial pain Tackling injury	Valgus laxity at 30˚ flexion Tenderness along course of MCL	Suspect MCL tear
Pivot or leap Sense of disruption Audible pop Instability Early swelling (1–2 hours)	+ve Lachman +ve anterior drawer +ve pivot shift* loss of hyperextension	Suspect ACL tear
Squatting, cutting, twisting injury Giving way Trivial twisting injury in older persons Locking and catching	Joint line tenderness McMurray +ve effusion Loss of extension (locked knee)	Suspect meniscal tear / reconsider ACL
Direct injury to anterior tibia (e.g. MVA) Forced hyperflexion / hyperextension injury Posterior pain Pain with kneeling	+ve sag test +ve posterior drawer mild swelling / slow onset posterior swelling painful limitation flexion 10˚–20˚	Suspect PCL tear
Significant athletic trauma or MVA Forced hyperflexion / hyperextension injury Posterior pain	Varus laxity at 30˚ and in extension Increased external rotation tibia	Suspect posterolateral complex

*The pivot shift test is best performed and interpreted by an experienced clinician.

Referral to specialists recommendations

There is no evidence relating to the appropriateness and timeliness of referral to specialists for people with knee injuries, however the nature and extent of injury as well as the practitioner's training and experience influence the need for referral.

1. Injuries to the anterior cruciate ligament (ACL), combined ACL and collateral ligament injuries, posterior cruciate ligament (PCL) and posterolateral complex are frequently missed in primary care. Early referral for these injuries is therefore recommended to clarify the diagnosis and discuss treatment options.
2. Early referral is also recommended for people with a locked knee due to a meniscal tear, and where the diagnosis is equivocal.

Acute management recommendations

"A" recommendations (supported by good evidence)

1. In the initial non-operative management of an acute internal derangement of the knee, there is good evidence that topical NSAIDs are effective and safe for relieving pain.[20]

"B" recommendations (supported by fair evidence)

1. Proprioceptive training can enhance functional stability and should be included in rehabilitation programmes for people with ACL deficient knees.[21]
2. Ultrasound is of little additional benefit and should not be used.[22-29]

"C" recommendations (supported by expert opinion only)

1. There is no evidence that "RICE" (rest, ice compression elevation) is effective for acute musculoskeletal injuries. However it is widely accepted as standard management.
2. Paracetamol is probably the most cost-effective and potentially least harmful choice of analgesic for soft tissue knee injuries.
3. Bracing is not generally required for the conservative management of most soft tissue knee injuries. [30-34]
4. Rehabilitation based on functional activity is important for some sub-groups of people with identifiable impairments due to a knee injury.
5. All grades of isolated medial ligament injuries can be successfully managed without surgery. [35-40]
    a) Grade I and II injuries best managed with early functional rehabilitation without the need for bracing. Return to sport can be expected in 6-8 weeks.
    b) Management of Grade III MCL injuries is similar but bracing is recommended for the first 4-6 weeks to stabilise the knee and facilitate initiation of rehabilitation and early return to activity.

6. Operative management has the most to offer those people with recurrent instability who must perform multidirectional activity as part of their occupation or sport. Age should not be considered a barrier for the older athlete who wishes to pursue a more active lifestyle. [41-44]
7. Non-operative management is preferred for the treatment of clinical stable meniscal tear where there is potential for healing and symptoms are mild, however this decision may need to be modified for people whose occupations demand a stable knee and the time frame required for repair is not appropriate. People with a suspected meniscal tear should be referred for a trial of rehabilitation for 6-8 weeks, and if symptoms persist, referred to a specialist.
8. Non-operative management is generally indicated for Grade I and II posterior cruciate ligament (PCL) tears.
9. For isolated Grade III PCL tears, there is insufficient evidence to establish the relative benefits of operative versus non-operative management. Early specialist referral for further evaluation is recommended.

"I" recommendation (no recommendation can be made because of insufficient evidence)

1. There is insufficient evidence of effectiveness for any physiotherapy intervention,[45] including electrotherapy modalities such as NMES (neuromuscular electrical stimulation),[46–49] Laser (light amplification by stimulated emission of radiation),[50] TENS (transcutaneous electric nerve stimulation)[49] and EMG (electromyography or biofeedback).[51-54]
2. No recommendations can be made about the use of acupuncture chiropractic or osteopathy due to lack of good quality evidence. There is no evidence for the efficacy of osteopathy or chiropractic. The effectiveness of acupuncture has not been consistently demonstrated.[55]

Postoperative management recommendations

The postoperative management following knee surgery varies according to the specific protocols of the operating surgeon. However some evidence can guide treatment.

"A" recommendation (supported by good evidence)

Physiotherapy should not be routinely advocated following menisectomy. [56]

"B" recommendations (supported by fair evidence)

1. Bracing is not effective in the early management following ACL reconstructive surgery.[57-65]
2. Proprioceptive training should be included in the post-operative rehabilitation programmes for people with ACL deficient knees.[21]
3. Open kinetic chain exercises should be included from about 4-6 weeks, in a restricted range of knee flexion from 45-90 degrees.[66-70]

"C" recommendations (supported by expert opinion only)

1. Rehabilitation following ACL reconstruction should be based on Shelbourne's accelerated rehabilitation programme.[71, 72]
2. For meniscal repairs and repairs or reconstruction of the PCL or posterolateral complex, the advice of the operating surgeon should be followed for any post-operative rehabilitation programme.

Discussion

Summary of main findings

There is a lack of good quality evidence to support accurate diagnosis and treatment decisions in the management of internal derangements of the knee, therefore most recommendations are based on consensus rather than more robust evidence. These results are similar to the guideline for acute knee injuries developed by the American Academy of Orthopaedic Surgeons (AAOS), although there is insufficient information in the support document to evaluate their guideline methodology.[73]

Our guideline offers guidance for some aspects of the diagnosis and management of internal derangements of the knee based on good evidence (A recommendations): the use of the Ottawa Knee rules to guide decisions about the use of X-ray; the Lachman test in the diagnosis of ACL tears; topical NSAIDs for acute sprains and soft tissue injuries and that physiotherapy not be routinely advocated following meniscectomy.

All these recommendations are supported by more recent evidence published since the development of the guideline.[13,74–76] Other relevant evidence includes a systematic review on the use of ice the treatment of acute soft tissue injury, which found little evidence to suggest a significant effect.[77]

While our guideline did not specifically focus on the post-operative management following ACL reconstruction, we did review some aspects of management. A comprehensive systematic review on the evidence for the post-operative rehabilitation following anterior cruciate ligament reconstructions has consistent conclusions to ours except that they support the use of NMES.[78]

Further high quality research is needed to guide the diagnosis and treatment of internal derangements of the knee, in particular the optimal use of physiotherapy in the non-operative and post-operative management of knee injuries.

Dissemination and implementation

A summary document containing the key messages and the diagnostic and management algorithm was sent to all NZ GPs and ACC registered physiotherapists. The complete version of the guideline and its supporting documents was made available on the NZGG website: http://www.nzgg.org.nz/guidelines/0009/ACC_Soft_Tissue_Knee_Injury_Fulltext.pdf

Further dissemination included two ACC reviews (one on diagnosis, the other on management of soft tissue knee injuries) distributed to all of ACC's treatment providers; a DVD on management of knee Injuries produced for all health providers; a case study on a knee injury for GPS plus a health provider-mediated information sheet

('Caring for your knee injury') for patients. There has been no evaluation conducted of the guideline implementation.

Priorities for future research are validity of the knee examination in primary care and physiotherapy settings, and a trial of RICE versus control.

View diagnostic and management algorithms at:
http://www.nzgg.org.nz/guidelines/0009/Diagnostic_Management_Algorithm.pdf

Appendix 1. Working party for guideline for diagnosis and management of acute soft tissue knee injuries

Bruce Arroll, Associate Professor General Practice University of Auckland (Chair); Gillian Robb, University of Auckland (Project Manager); Emma Sutich, NZGG Project Manager; Sunia Foliaki Public Health Consultant, Pacific Representative; Peter Gendall, Musculoskeletal Radiologist; Chris Milne, Sports Medicine Physician Hamilton; Graeme Moginie, Physiotherapist Dunedin; Jolene Phillips, Consumer Representative Wellington; Rocco Pitto, Orthopaedic Surgeon Auckland; Rachel Thomson, General Medical Practitioner Maori representative Hamilton; Russell Tregonning, Orthopaedic Surgeon Wellington; James Watt, Musculoskeletal Physician, Auckland; Russell Blakelock, Paediatric Surgeon, Pacific representative Christchurch; John Matheson, Orthopaedic Surgeon Dunedin; Neil Matson, General Medical Practitioner Tauranga; Peter McNair, Professor, Physiotherapy AUT Auckland; Ian Murphy, Sports Medicine Registrar Auckland; Peter Pfitzinger, Consumer Representative Auckland; Paul Quin, Musculoskeletal Physician Auckland; Keri Ratima, GP Maori representative; Duncan Reid, Physiotherapist and Head of Division of Rehabilitation and Occupation Studies; AUT Auckland; Barry Tietjens, Orthopaedic Surgeon, Auckland.

Conflict of interest statement: There are no conflicts of interest.

Author information: Gillian Robb, Project Manager, University of Auckland; Duncan Reid, Physiotherapist and Head of Division of Rehabilitation and Occupation Studies, Auckland University of Technology (AUT); Bruce Arroll, Professor, General Practice; University of Auckland; Rod T Jackson, Professor of Epidemiology and Biostatistics, Department of Epidemiology and Biostatistics, University of Auckland; Felicity Goodyear-Smith, Associate Professor, Department of General Practice and Primary Health Care, School of Population Health, University of Auckland

Acknowledgement: We thank Accident Compensation Corporation (ACC) for funding this guideline.

Correspondence: Felicity Goodyear-Smith, Department of General Practice and Primary Health Care, School of Population Health Faculty of Medical and Health Science, University of Auckland Private Bag 92019 Auckland. Fax: (09) 373 7624; email: f.goodyear-smith@auckland.ac.nz

References:

1. AAOS Clinical Algorithm on acute knee injury; 1997.
2. Kent DL, Larson EB. Disease, level of impact and quality of research methods: three dimensions of clinical efficacy assessment applied to magnetic resonance imaging. Investigative Radiology. 1992;27:245–54.
3. McGinn TG, Guyatt GH, Wyer PC, et al. Users' guides to the medical literature: XXII How to use articles about clinical decision rules. JAMA. 2000;284:79–84.
4. Stiell IG, Greenberg GH, Wells GA, et al. Derivation of a decision rule for the use of radiography in acute knee injuries. Annals of Emergency Medicine. 1995;26:405–13.
5. Stiell IG, Greenberg GH, Wells GA, et al. Prospective validation of a decision rule for the use of radiography in acute knee injuries. [see comments]. JAMA 1996;275:611–15.
6. Stiell IG, McKnight RD, Greenberg GH, et al. Implementation of the Ottawa ankle rules. [comment]. JAMA. 1994;271:827–32.
7. Stiell I, Wells G, Laupacis A, et al. Multicentre trial to introduce the Ottawa ankle rules for use of radiography in acute ankle injuries. Multicentre Ankle Rule Study Group. BMJ. 1995;311:594–7.
8. Spiers AS, Meagher T, Ostlere SJ, et al. Can MRI of the knee affect arthroscopic practice? A prospective study of 58 patients. Journal of Bone & Joint Surgery - British Volume. 1993;75:49–52.
9. Munk B, Madsen F, Lundorf E, et al. Clinical magnetic resonance imaging and arthroscopic findings in knees: a comparative prospective study of meniscus anterior cruciate ligament and cartilage lesions. Arthroscopy. 1998;14:171–5.
10. Curtin W, O'Farrell, McGoldrick F, et al. The correlation between clinical diagnosis of knee pathology and findings at arthroscopy. Irish Journal of Medical Science. 1992;161:135–6.
11. Adalberth T, Roos H, Lauren M, et al. Magnetic resonance imaging, scintigraphy, and arthroscopic evaluation of traumatic hemarthrosis of the knee. American Journal of Sports Medicine. 1997;25:231–7.
12. Wong JWK, Chien EP, Yip DKH, Tang WM, Chin ACW, Peh WCG. Is MRI necessary to confirm an acute ACL rupture? Hong Kong Journal of Sports Medicine & Sports Science. 1999;8:1–5.
13. Scholten RJ, Opstelten W, Van der Plas C, et al. Accuracy of physical diagnostic tests for assessing ruptures of the anterior cruciate ligament: a meta-analysis. The Journal of Family Practice. 2003;52:689–94.
14. Grevitt M, Pool CJF, Bodley RN, Savage PE. Magnetic resonance imaging of the knee: initial experience in a district hospital. Injury. 1992;23:410–12.
15. Boeree NR, Watkinson AF, Ackroyd CE, Johnson C. Magnetic resonance imaging of meniscal and cruciate injuries of the knee. Journal of Bone & Joint Surgery - British Volume. 1991;73-B:452–7.
16. Muellner T, Weinstabl R, Schabus R, et al. The diagnosis of meniscal tears in athletes. A comparison of clinical and magnetic resonance imaging investigations. [see comments]. American Journal of Sports Medicine. 1997;25:7–12.
17. Rappeport ED, Wieslander SB, Stephensen S, et al. MRI preferable to diagnostic arthroscopy in knee joint injuries. A double-blind comparison of 47 patients. Acta Orthopaedica Scandinavica. 1997;68:277–81.
18. Raunest J, Oberle K, Loehnert J, Hoetzinger H. The clinical value of magnetic resonance imaging in the evaluation of meniscal disorders. Journal of Bone & Joint Surgery - American Volume. 1991;73:11–16.

19. Vincken. Effectiveness of MR imaging in selection of patients for arthroscopy. Radiology. 2002;223:739–46.

20. Moore RA, Tramer MR, Carroll D, et al. Quantitative systematic review of topically applied non-steroidal anti-inflammatory drugs. BMJ. 1998;316:333–8.

21. Beard DJ, Dodd CA, Trundle HR, Simpson AH. Proprioception enhancement for anterior cruciate ligament deficiency. A prospective randomised trial of two physiotherapy regimes. Journal of Bone & Joint Surgery - British Volume. 1994;76:654–9.

22. Ogilvie-Harris DJ, Gilbart M. Treatment modalities for soft tissue injuries of the ankle: a critical review. Clinical Journal of Sport Medicine. 1995;5:175–86.

23. Beckerman H, Bouter LM, van der Heijden GJ, et al. Efficacy of physiotherapy for musculoskeletal disorders: what can we learn from research? British Journal of General Practice. 1993;43:73–7.

24. van der Heijden GJ, van der Windt DA, de Winter AF. Systematic review of randomised clinical trials of physiotherapy for soft tissue shoulder disorders. BMJ. 1997;315:25–30.

25. van der Windt DA, van der Heijden GJ, van den Berg SG, et al. Ultrasound therapy for musculoskeletal disorders: a systematic review. Pain. 1999;81:257–71.

26. van der Windt DA, van der Heijden GJ, van den Berg SG, et al. Ultrasound therapy for acute ankle sprains. Cochrane Review: Cochrane Library. 2001;Issue 1.

27. Falconer J, Hayes KW, Chang RW. Therapeutic ultrasound in the treatment of musculoskeletal conditions. Arthritis Care Research. 1990;3:85–91.

28. Gam AN, Johannson F. Ultrasound therapy in musculoskeletal disorders: a meta-analysis. Pain. 1995;63:85–91.

29. Baker KG, Robertson VJ, RV, Duck FA. A review of therapeutic ultrasound: Biophysical effects. Physical Therapy. 2001;81:1351–8.

30. Liu SH, Mirzayan R. Current review: Functional knee bracing. Clinical Orthopaedics & Related Research. 1995;317:273–81.

31. Cawley PW, France EP, Paulos LE. The current state of functional knee bracing research: a review of the literature. American Journal of Sports Medicine; 1991;19:226–33.

32. Sitler MR. Role of prophylactic knee and ankle bracing in injury reduction. Journal of Sport Rehabilitation. 1992;1:223–36.

33. Requa RK, Garrick JG. A review of the use of prophylactic knee braces in football. Pediatric Clinics of North America. 1990;37:1165–73.

34. Kramer JF, Dubowitz T, Fowler P, et al. Functional knee braces and dynamic performance: a review. [Review] [17 refs]. Clinical Journal of Sport Medicine. 1997;7:32–9.

35. Ellsasser JC, Reynolds FC, Omohundro KR. The non-operative treatment of collateral liagment injuries of the knee in professinoal football players. Journal of Bone & Joint Surgery - American Volume. 1974;56-a:1185.

36. Indelicato PA. Nonoperative management of complete tears of the medial collateral ligament of the knee. Journal of Bone & Joint Surgery - American Volume. 1983;65-A:323.

37. Indelicato PA, Hermansdorfer J, Huegal M. Nonoperative management of complete tears of the medial collateral ligament of the knee in intercollegiate football players. Clinical Orthopaedics & Related Research. 1990;256:174.

38. Hastings DE. The non-operative management of collateral ligament injuries of the knee joint. Clinical Orthopaedics & Related Research. 1980;147:22–8.

39. Jokl P, Kaplan N, Stovell P. Non-operative treatment of severe injuries to the medial and anterior cruciate ligaments of the knee. Journal of Bone & Joint Surgery - American Volume. 1984;66-A:741.

40. Sandberg R, Balkfors B, Nilsson B, Westlin N. Operative versus non-operative treatment of recent injuries to the ligaments of the knee: A prospective randomized study. Journal of Bone and Joint Surgery-American Volume. 1987; 69-A:1120–6.

41. Barber FA, Elrod BF, McGuire DA, Paulos LE. Is an anterior cruciate ligament reconstruction outcome age dependent? Arthroscopy. 1996;12:720–5.

42. Plancher KD, Steadman JR, Briggs K, Hutton KS. Reconstruction of the anterior cruciate ligament in patients who are at least 40 years old. A long-term follow up and outcome study. Journal of Bone & Joint Surgery - American Volume. 1998;80-A:184–97.

43. Novak PJ, Bach BR Jr, Hager CA. Clinical and functional outcome of anterior cruciate ligament reconstruction in the recreational athlete over the age of 35. American Journal of Knee Surgery. 1996;9:111–16.

44. Adams MA, Moore KD. Intra-articular ACL reconstruction in the over 40-year old patient. Arthroscopy. 1995;11:374.

45. Thomson LC, Handoll HHG, Cunningham A, et al. Physiotherapist-led programmes and interventions for rehabilitation of anterior cruciate ligament, medial collateral ligament and meniscal injuries of the knee in adults (Cochrane Review Issue 2 2002). In: The Cochrane Library, Issue 2, 2001. Oxford: Update Software; 2001.

46. Callaghan MJ, Oldham JA. A critical review of electrical stimulation of the quadriceps muscles. Critical Reviews Physical Rehabilitation Medicine. 1997;9:301–14.

47. Snyder-Mackler L, Delitto A, Stralka SW, Bailey SL. Use of electrical stimulation to enhance recovery of quadriceps femoris muscle force production in patients following anterior cruciate ligament reconstruction [see comments]. Physical Therapy. 1994;74:901–7.

48. Snyder-Mackler L, Delitto A, Bailey SL, Stralka SW. Strength of the quadriceps femoris muscle and functional recovery after reconstruction of the anterior cruciate ligament. A prospective, randomized clinical trial of electrical stimulation. Journal of Bone & Joint Surgery - American Volume. 1995;77:1166–73.

49. Paternostro-Sluga T, Fialka C, Alacamliogliu Y, et al. Neuromuscular electrical stimulation after anterior cruciate ligament surgery. Clinical Orthopaedics & Related Research. 1999;368:166–75.

50. Gam AN, Thorsen H, Lonnberg F. The effect of low-level laser therapy on musculoskeletal pain: a meta-analysis. Pain. 1993;52:63–6.

51. Draper V, Ballard L. Electrical stimulation versus electromyographic biofeedback in the recovery of quadriceps femoris muscle function following anterior cruciate ligament surgery [see comments]. Physical Therapy. 1991;71:45–61.

52. Draper V, Lyle L, Seymour T. EMG biofeedback versus electrical stimulation in the recovery of quadriceps surface EMG. Clinical Kinesiology: Journal of the American Kinesiotherapy Association. 1997;51:28–32.

53. Draper V. Electromyographic biofeedback and the recovery of quadriceps femoris muscle function following anterior cruciate ligament reconstruction. Physical Therapy. 1990;70:11–17.

54. Krebs DE. Clinical electromyographic feedback following menisectomy: a multiple regression experimental analysis. Physical Therapy. 1981;61:1017–21.

55. Hodges I, Maskill C. Effectiveness of acupuncture for he treatment and rehabilitation of accident-related musculoskeletal disorders. NZHTA Report. 2002;5:1–106.

56. Goodyear-Smith F, Arroll B. Rehabilitation after arthroscopic meniscectomy: A critical review of the clinical trials. International Orthopaedics. 2001;24:350–3.

57. Risberg MA, Holm I, Steen H, et al. The effect of knee bracing after anterior cruciate ligament reconstruction. A prospective, randomized study with two years' follow-up. American Journal of Sports Medicine. 1999;27:76–83.

58. Muellner T, Alacamlioglu Y, Nikolic A, Schabus R. No benefit of bracing on the early outcome after anterior cruciate ligament reconstruction. Knee Surgery, Sports Traumatology, Arthroscopy. 1998;6:88–92.

59. Pippolo E, Ostelo RWJ. [Braces and elastic bandages: systematic review of effectiveness of braces and elastic bandages after anterior cruciate ligament reconstruction] [Dutch]. Nederlands Tijdschrift voor Fysiotherapie. 2002;112:2–8.

60. Martinek V, Friederich NF. [To brace or not to brace? How effective are knee braces in rehabilitation?]. [Review] [66 refs] [German]. Orthopade. 1999;28:565–70.

61. Moller E, Forssblad M, Hanson L, et al. Bracing vs non-bracing in rehabilitation: A randomised prospective study with 2 year follow up. Knee Surgery, Sports Traumatology, Arthroscopy. 2001;9:102–8.

62. Risberg MA, Holm I, Eriksson J, et al. A prospective randomized study on the effect of knee bracing after ACL reconstruction - preliminary results with 1-year follow- up [Abstract]. Acta Orthopaedica Scandinavica. 1998;69:9.

63. Brandsson S, Faxen E, Kartus J, Karlson JA. Is a knee brace advantageous after anterior cruciate ligament surgery? A prospective randomised study with a 2 year follow up. Scandinavian Journal of Medicine and Science in Sports. 2001;11:110–14.

64. Harilainen A, Sandelin J, Vanhanen I, Kivinen A. Knee brace after bone-tendon-bone anterior cruciate ligament reconstruction. Randomized, prospective study with 2-year follow- up. Knee Surgery, Sports Traumatology, Arthroscopy. 1997;5:10–13.

65. Kartus J, Stener S, Kohler K, et al. Is bracing after anterior cruciate ligament reconstruction necessary? A 2-year follow-up of 78 consecutive patients rehabilitated with or without a brace. Knee Surgery, Sports Traumatology, Arthroscopy. 1997;5:157–61.

66. Hooper DM, Morrissey MC, Drechsler W, et al. Open and closed kinetic chain exercises in the early period after anterior cruciate ligament reconstruction: Improvements in level walking, stair ascent, and stair descent. American Journal of Sports Medicine. 2001;29:167–74.

67. Bynum EB, Barrack RL, Alexander AH. Open versus closed chain kinetic exercises after anterior cruciate ligament reconstruction. A prospective randomized study. [see comments]. American Journal of Sports Medicine. 1995;23:401–6.

68. Mikkelsen C, Werner S, Eriksson E. Closed kinetic chain alone compared to combined open and closed kinetic chain exercises for quadriceps strengthening after anterior cruciate ligament reconstruction with respect to return to sports: a prospective matched follow-up study. Knee Surgery, Sports Traumatology, Arthroscopy. 2000;8:337–42.

69. Morrissey MC, Hudson ZL, Drechsler WI, et al. Effects of open versus closed kinetic chain training on knee laxity in the early period after anterior cruciate ligament reconstruction. Knee Surgery, Sports Traumatology, Arthroscopy. 2000;8:343–8.

70. Morrissey MC, Drechsler W, Morrissey D, et al. Effects of distally fixated versus nondistally fixated leg extensor resistance training on knee pain in the early period after anterior cruciate ligament reconstruction. Physical Therapy. 2002;1:35–43.

71. Shelbourne KD, Nitz P. Accelerated rehabilitation after anterior cruciate reconstruction. American Journal of Sports Medicine. 1990;18:292–9.

72. Shelbourne KD, Klootwyk TE, De Carlo MS. Update on accelerated rehabilitation after anterior cruciate ligament reconstruction. Journal of Orthopaedic & Sports Physical Therapy. 1992;15.

73. AAOS. AAOS Clinical Guideline on Knee Injury:Support Document. In; 2001.

74. Bachmann LM, Haberzeth S, Steurer J, Ter Riet G. The accuracy of the Ottawa Knee Rule to rule out knee fractures: A systematic review. Annals of Internal Medicine. 2004;140:121–4.

75. Mason L, Moore RA, Edwards JE, et al. Topical NSAIDs for acute pain: a meta-analysis. BMC Family Practice. 2004;5:10.

76. Goodwin PC, Morrisey MC, Omar RZ, et al. Effectiveness of supervised physical therapy in the early period after arthroscopic partial menisectomy. Physical Therapy. 2003;83:520–35.

77. Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft tissue injury: a systematic review. American Journal of Sports Medicine. 2004; 32:251–61.

78. Risberg MA, Lewek M, Snyder-Mackler L. A systematic review of evidence for anterior cruciate ligament rehabilitation : how much and what type? Physical Therapy in Sport. 2004; 5:125–45.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Consumers' knowledge, perceptions, and responsiveness to direct-to-consumer advertising of prescription medicines

Janet Hoek, Ninya Maubach

Abstract

Aim This research explored whether direct-to-consumer-advertising of prescription medicines (DTCA) increased disadvantaged consumers' knowledge of important health issues and encouraged those with lower health knowledge to consult their doctor (as has been argued by supporters of DTCA).

Method A mail survey of 1042 New Zealanders was undertaken between October and December 2002 using a stratified random sample drawn from the electoral roll. After two reminders were sent, 632 completed questionnaires were returned (64% response rate). We examined the relationship between respondents' self-assessed knowledge of health-related issues, their perceived health status, and their response to DTCA (using self-efficacy theory to aid interpretation of the results).

Results Respondents with greater health knowledge found DTCA easier to understand and were more likely to have sought further information about an advertised medicine than those with less knowledge.

Conclusions These results suggest DTCA may reinforce existing knowledge rather than educate or provide new knowledge. The results also cast doubt upon claims that DTCA enhances awareness of health issues among groups with lower health knowledge thus helping them overcome barriers to better health. Although changes to DTCA regulation could increase the information conveyed by this advertising, the advertising and pharmaceutical industries' failure to respond to well-documented concerns about DTCA raises serious questions about the power of policy refinements to control advertisers' conduct.

Advertising of prescription medicines directly to potential end-users (DTCA) has become pervasive in both New Zealand and the United States, the only two countries where these promotions are permitted. Not surprisingly, the effects and ethics of this advertising have attracted detailed scrutiny in both countries.[1,2]

Opponents argue that prescription medicines differ from fast-moving-consumer-goods and should not be promoted using the same techniques or via similar media.[3] They note that medicines have potentially serious consequences that lay consumers may not understand and suggest only those trained to assess these risks should receive promotional material.[2,4,5] Others suggest DTCA erodes the trust on which a healthy doctor-patient relationship depends,[6,7] and makes doctors more likely to accede to patients' requests even if they do not fully agree with these.[8]

As a result, patients may adopt pharmaceutical solutions to health problems instead of implementing lifestyle changes, such as losing weight.[9, 10] This alleged increase in the "medicalisation" of well populations has been compounded by the confusion resulting from incomplete or omitted risk, side effect, and cost details.[11,12]

More seriously, critics claim DTCA's profit-driven goal is at odds with health practitioners' aim to improve their patients' quality of life.[2,3,13] They argue that since DTCA increases demand for promoted drugs (thereby reducing funding available for other medicines), it may also result in a sub-optimal allocation of scarce health resources.[14]

Supporters of DTCA have responded by arguing that consumers can no longer rely on doctors to provide them with details of all treatment options for a condition within a standard 15-minute consultation.[15] DTCA is thus said to fill an information void by providing facts consumers may be unlikely to obtain from other sources.[16–20]

Provision of information that contains details of both symptoms and treatment options is also claimed to promote earlier diagnosis of chronic disorders and improve patients' quality of life while reducing the need for more expensive interventions in the future.[19,21] Moreover, disadvantaged individuals with less access to health information are said to be most likely to benefit from DTCA,[22] as it will help them overcome "lack of awareness and understanding, misinformation and low health literacy".[17]

Overall, supporters of DTCA claim it fulfils a valuable function by increasing patients' knowledge; more generally, they believe it reflects a social environment where patients see themselves as active participants in their health care management.[17,23]

Although consumer survey evidence suggests many individuals have welcomed the opportunity to access information disseminated via DTCA;[24–26] surveys of the New Zealand public have consistently revealed serious limitations in the comprehensiveness and balance of information provided via DTCA.[26,27] Indeed, several researchers have called for details in DTCA promotions to be better formatted, made available aurally as well as visually, and re-defined to emphasise key risk elements.[5,27–29] However, in New Zealand at least, these concerns have persisted, despite revisions to the self-regulatory process developed to manage DTCA.[27]

There has been considerable debate over the robustness of the self-regulatory model currently used to oversee DTCA promotions. While tighter regulation—including strengthening the provisions of the self-regulatory code or instituting a more defined role for government regulators—could improve the quality of information provided via DTCA, others question whether restrictions would protect consumers. They argue that stricter regulatory frameworks increase consumers' trust in advertising and thus create greater incentives for advertisers to utilise misleading promotions.[15]

The existence of these market-driven incentives has led others to conclude that DTCA is most unlikely to assist the improvement of public health and that, on balance, the negative effects of medicine advertising greatly outweigh any benefits it could be expected to bring.[2,9]

At the same time as researchers have debated whether DTCA can be adequately regulated, consistent concerns over the lack of balance provided in DTCA promotions have been identified, as has a core group of consumers that sees DTCA as unbalanced, potentially misleading, and ultimately unhelpful.[27,30]

There are several possible explanations for the existence of this group. Research into consumers' responses to advertising has noted that some find advertising distasteful and irritating, and resent its intrusiveness; opposition to DTCA may thus stem from a

more generic dislike of advertising.[31] However, an alternative explanation is that some respondents find DTCA difficult to access and understand; this latter view implies that consumers may react against the content and format of DTCA if they find this incomplete or poorly structured. This latter explanation implicitly challenges the claim that DTCA provides information consumers may not otherwise be able to access and raises particular questions about whether it benefits people less likely to obtain health information from other sources.

Because DTCA includes complex information that lay consumers may not always understand, individuals' response to DTCA may be mediated by their perceived knowledge of health related issues. Consumers who are confident in their ability to access and utilise the information provided might be more likely to have positive perceptions of DTCA, and to be more responsive to it. However, few studies have explored how consumers' knowledge of health issues might mediate their responses to DTCA.

This paper briefly reviews the psychological construct of self-efficacy, which we propose may mediate consumers' response to pharmaceutical advertisements, before examining the relationship between respondents' perceived health knowledge and status, and their perceptions of and responses to DTCA advertising.

Self-efficacy is an important psychological construct that has been used successfully to explain and predict a wide variety of health related behaviours.[32] Originally outlined by Bandura,[33] self-efficacy describes individuals' beliefs regarding their ability to perform a specific behaviour, and has two dimensions: individuals' assessment of the likely outcome of a particular behaviour, and their belief in their own competence to achieve that outcome.

Self-efficacy is conceptualised as highly domain specific, rather than as a stable trait, so it varies across behaviours within individuals.[34,35] When individuals have low self-efficacy beliefs, they only feel capable for performing the most simple behaviours, while those with high self-efficacy are assured in their ability to undertake more complex tasks.[32] Those with high self-efficacy for particular behaviour patterns are thought more likely to adopt these where they offer clear and accessible benefits.[36,37]

Variance in self-efficacy levels may help explain consumers' reactions to DTCA. Respondents' perceptions of this advertising—particularly their ability to access and understand the information it contains, their trust in this, and their use of it—may reflect self-efficacy for 'health management'; a term used here to reflect patients' confidence or willingness to actively participate in health care decisions, including discussions with professionals about diagnosis and treatment of conditions. This framework recognises that DTCA contains technical information that not all consumers will process or understand sufficiently to act upon.

Because medicines cannot be purchased directly, behavioural indications of self-efficacy may include attempts to obtain additional information from websites or 0800 telephone numbers and requests for promoted brands from doctors. These actions depend on consumers' belief that they will locate and comprehend more detailed information, and then be able to use this in a discussion about the promoted drug with their doctor.

DTCA advertisements might also shape consumers' personal agency beliefs: if DTCA improves overall knowledge of health issues, then it could promote self-efficacy for health management by increasing respondents' perceived control over their well-being by providing them with a greater understanding of symptoms, possible conditions and some treatment options.

If DTCA does provide valuable information to those with limited health knowledge or access to information from other sources, then we would expect respondents with lower reported levels of health knowledge to view these messages positively. Similarly, those who report higher levels of ill health might also be more appreciative of this advertising if, as claimed by supporters, it increased their knowledge of treatments they could discuss with their doctor.

This paper tested the assertion that DTCA benefits disadvantaged consumers. Firstly, we examined factors that may influence consumers' response to DTCA by exploring the relationship between their self-assessed knowledge of health issues and their reactions to pharmaceutical advertising. More specifically, we explored whether respondents with lower self-assessed health knowledge and health status were more responsive to DTCA and held more favourable views of this advertising.

Methods

A mail survey of 1042 New Zealanders was undertaken between October and December 2002. A random sample was drawn from the 2002 electoral roll and was stratified by electorate, with over-sampling in the Māori electorates, to ensure adequate representation of Māori in the final sample.

After an initial mail out and two reminders, the survey achieved a 64% response rate, representing 632 completed responses. The sample was weighted to ensure it corresponded to the age and gender profile of the population based on 2001 census data.

The questionnaire began by defining DTCA (to ensure respondents shared a common understanding of this advertising).

It then gauged respondents':

- Awareness of different prescription medicines and media (through which they saw or heard about these medicines);
- Recall of information in DTCA promotions as well as their ability to read and understand this information;
- Attitudes to DTCA and its wider implications, and
- Behavioural response to DTCA promotions;

Finally, it collected demographic information and details of respondents' perceived health status and knowledge.

Respondents used a single-item 4-point scale to estimate their personal health knowledge; responses were subsequently dichotomised. Respondents rated their current health status on a single-item 5-point scale anchored by 'poor' and 'excellent'. (A copy of the full questionnaire is available from the first author.)

The questionnaire was pre-tested using depth interviews on a small sample of respondents and the penultimate version was circulated to several interest groups known to hold varied views on the desirability and effects of DTCA. Improvements and clarifications resulting from both procedures were incorporated before the questionnaire was finalised.

The data were analysed using a series of statistical procedures. Chi-squared (χ^2) tests were used to examine the relationship between respondents' health status and their self-assessed health knowledge. Next, a series of ANOVAs were undertaken to test whether health knowledge affected respondents' perceptions of DTCA.

Results

Initial analyses explored the relationship between respondents' knowledge of health-related issues and medicines and their own perceived health status. As Table 1 shows, respondents' perceived health knowledge was significantly related to their perceived health status; respondents who rated themselves less knowledgeable about health issues were more likely to describe themselves as having less robust health, and vice versa. Respondents' education level was also significantly related to their perceived health knowledge, and followed the same pattern (χ^2=10.75, p<0.01).

Table 1. Perceived health knowledge and perceived health status

Perceived health status	Perceived health knowledge	
	Less knowledgeable (n=243) %	More knowledgeable (n=373) %
Poor–Fair	19	15
Good	43	32
Very Good	26	33
Excellent	13	20

χ^2=12.74, p<0.01

The relationship reported in Table 1 is important, since it highlights the potential role of DTCA as an educational tool that could augment respondents' knowledge of health issues, thereby assisting them to improve their actual health.

Self-efficacy theory suggests that if DTCA supported knowledge development, it would be viewed more positively by respondents who felt it extended their knowledge. Thus, if DTCA consolidated existing knowledge, it would be viewed more positively by those with higher levels of knowledge; conversely, if it developed new knowledge, we would expect those with lower levels of knowledge to be more positive about this advertising.

A series of ANOVAs were undertaken to test the relationship between respondents' knowledge status and their attitudes to DTCA. The attributes tested were examined using a 5-point Likert scale; the higher the mean score, the higher the level of agreement. Mean scores for the two groups are reported in Table 2.

Respondents first indicated the ease with which they could ascertain whether a medicine required a prescription. Although respondents who considered themselves knowledgeable about health issues found it significantly easier to establish the status of advertised medicines, the overall mean scores were low, thus suggesting that neither group found this information particularly easy to access.

Scores across other attributes were very similar, although the more knowledgeable group was less likely to agree that DTCA contained too much risk information or was difficult to understand. This group was also more cynical about the content of DTCA; members were more likely to agree that DTCA overstated the benefits of the promoted drug and more likely to disagree that only the safest drugs were advertised.

Although respondents considered that DTCA provided them with information they could use in discussions with their GPs, they were less positive about the quality of information provided and the extent to which they could trust this information.

Table 2. Perceived health knowledge and perceptions of DTCA (direct-to-consumer advertising)

Attribute	Perceived level of health knowledge	
	Less knowledgeable (n=223)	More knowledgeable (n=361)
Knowledge of Rx (treatment) status		
Easy to know prescription required in TV DTCA[α]	2.8	3.2
Easy to know Rx required in print DTCA[α]	1.9	2.5*
Information provision [β]		
DTCA increases awareness of medicines	4.1	4.1
DTCA should have more risk information	4.1	4.1
DTCA provides enough information	3.6	3.6
DTCA should have more benefit information	3.5	3.5
DTCA contains too much risk information	2.3	2.1†
Easy to read TV DTCA[α]	1.8	2.0
Usefulness of and trust in information [β]		
DTCA promotes better decisions about health	3.4	3.3
I find DTCA helpful	3.3	3.4
DTCA make medicines seem better than they are	3.1	3.3†
DTCA confuses people	3.0	3.0
I trust the information in DTCA	2.9	2.9
DTCA is difficult to understand	2.7	2.6†
Only the safest drugs are advertised	2.7	2.5*
DTCA should be banned	2.3	2.2
Effect on GP relationship [β]		
DTCA fosters better discussions with doctors	3.6	3.7
DTCA implies a GP is not required	3.0	3.0

[α] 1 (very difficult) – 5 (very easy);

[β] 1 (strongly disagree) – 5 (strongly agree);

[*] $p<0.01$; †$p<0.05$; TV=Television.

Respondents who considered themselves knowledgeable about health issues were significantly more likely to have sought more information about a medicine they had seen advertised than members of the less knowledgeable group (52% cf. 37%; $p<0.001$).

To explore this relationship further, and to examine the potential effect of other variables (such as respondents' demographic characteristics), a logistic regression model was developed. The dependent variable was whether respondents had sought additional information following exposure to DTCA, while the predictor variables included respondents' use of prescription medicines; their perceived health knowledge and status; and demographic traits. Table 3 contains details of the model developed.

Table 3. Factors influencing respondents' search for further information

Variables entered	Coefficient (B)	Statistical significance	Estimated odds ratio
Perceived knowledge	0.575	0.002	1.777
Number of medicines taken	0.342	0.000	1.408
Age	-0.001	0.920	0.999
Perceived health status	-0.032	0.746	0.968
Gender	-0.160	0.367	0.852
Constant	-1.167	0.025	0.311

Nagelkerke R Square=0.08

Two variables were highly significant predictors of the likelihood that respondents would seek more information about a medicine they had seen advertised: their perceived health knowledge ($p<0.01$), and the number of medicines they take ($p<0.001$).

The estimated odds ratio shows the magnitude of influence that greater perceived knowledge and number of current medications increases likelihood of seeking further information. This result suggests respondents' perceived knowledge may be a function of their health condition, which in turn increases the salience of drugs designed to treat that condition and prompts the search for further information.

Respondents' perceived health status was negatively associated with the search for information on advertised medicines. Logically, this is consistent with the expectation that people who consider themselves to be in good health are less likely to seek out information on medicines they have seen advertised. However, this variable failed to reach significance in the model, thus there was no evidence that DTCA encourages people who evaluate their health status as less robust to obtain more information about conditions they may have or treatments that could potentially assist these. Neither of the demographic variables (age and gender) included in the model approached significance.

Overall, the results in Table 3 do not support the claim that DTCA increases the likelihood that individuals with undiagnosed conditions will become more aware of symptoms that could foster diagnosis; nor does it appear to be encouraging people to seek further information about potential health conditions they may have.

Conclusions and policy implications

Supporters of DTCA argue it improves consumers' knowledge of treatment options that may enhance their quality of living. However, our results suggest consumers who are most likely to benefit from increased knowledge are also those who find DTCA most difficult to understand, and who are less likely to seek further information after exposure to DTCA promotions.

Respondents who viewed themselves as less knowledgeable about health issues were more likely to report difficulty in identifying whether a medicine was prescription only and more likely to trust the content of advertisements for prescription medicines than those who considered themselves more knowledgeable.

The greater the number of medicines respondents took, the more likely they were to have sought more information about a medicine they had seen advertised. This

suggests that DTCA reinforces consumers' existing knowledge, thus it may promote greater self-efficacy among those who already consider themselves knowledgeable about health issues. However, we found no evidence that DTCA promotes a greater awareness of health-related topics among those who consider their health knowledge or status to be low.

Those most likely to respond to DTCA were also those who were more cynical about the quality of information provided in this advertising, which may explain their desire to seek further details from sources seen as more disinterested.

Consumers with lower perceived health knowledge had lower self-efficacy for health management and lacked the confidence to seek new information or discuss treatment options with medical professionals. These differences in consumers' self-efficacy could explain the varying reactions responses to DTCA observed in more general consumer surveys.

While a similar proportion of both the knowledgeable and less knowledgeable groups had requested a drug they had seen advertised from their doctor, those from the former group were more likely to understand the implications of their request. Doctors report having to dispel patient misconceptions attributed to DTCA;[38] our findings in the current study suggest this confusion may be more likely to affect those with poorer health status and knowledge of health issues.

Although preliminary, our findings question claimed benefits of DTCA, and we found no evidence to support the claim that DTCA assists individuals to overcome barriers to better health. Instead, it seems more plausible that DTCA reinforces knowledge among those who already have high levels of interaction with medical professionals and better knowledge as a consequence of this engagement. As a result, DTCA may only increase self-efficacy for consumers who already have moderate levels of belief in their ability to manage their health.

Our findings have important regulatory implications for Australian regulators, who are considering whether to introduce DTCA; and for New Zealand regulators, who are reviewing whether and in what form they should retain this advertising.

While replication research should be undertaken these results raise questions about the value of DTCA as an information source. Evidence that respondents with higher health knowledge and self-efficacy were less trusting of DTCA, while those with lower health knowledge and self-efficacy found it difficult to understand, suggests the information DTCA is claimed to provide could be more effectively and appropriately imparted via another source.

In summary, research is needed into alternative methods of communicating health information to those individuals whose health status and knowledge suggests they have the greatest potential to benefit from greater access to such information .These individuals are currently less likely to comprehend or respond to the information provided via DTCA.

Conflict of interest statement: This project was funded by a grant from the Academy of Business Research of the College of Business, Massey University. Professor Hoek has acted as an independent consultant to the Association of New Zealand Advertisers; she has also been an expert consultant to the Ministry of Health.

Author information: Janet A Hoek, Professor; Ninya Maubach, PhD Candidate; Department of Marketing, Massey University, Palmerston North.

Correspondence: Janet Hoek, Department of Marketing, Massey University, Private Bag 11 222, Palmerston North. Fax: (06) 350 2260; email J.A.Hoek@massey.ac.nz

References:

1. Rosenthal M, Berndt J, Donohue M, et al. Promotion of prescription drugs to consumers. New Engl J Med. 2002;345:498–505.
2. Toop L, Richards D, Dowell T, et al. Direct to consumer advertising of prescription drugs in New Zealand: For health or for profit? Report to the Minister of Health supporting the case for a ban on DTCA; 2003. URL: http://www.chmeds.ac.nz/report.pdf
3. Lexchin J, Mintzes B. Direct-to-consumer advertising of prescription drugs: The evidence says no. J Public Policy Marketing. 2002;21:194–202.
4. Moynihan R, Heath I, Henry D. Selling sickness: The pharmaceutical industry and disease mongering. BMJ. 2002;324:886–91.
5. Davis J. Riskier than we think? The relationship between risk statement completeness and perceptions of direct to consumer advertised prescription drugs. J Health Commun. 2001;5:349–69.
6. Cockburn J, Pit S. Prescribing behaviour in clinical practice: Patients' expectations and doctors' perceptions of patients' expectations. BMJ. 1997;315:520–3.
7. Wolfe SM. Direct-to-consumer advertising: Education or emotion promotion? New Engl J Med. 2002;346:524–6.
8. Lipsky MS, Taylor CA. The opinions and experiences of family physicians regarding direct-to-consumer advertising. J Fam Pract. 1997;45:495–9.
9. Coney S. Direct to consumer advertising of prescription pharmaceuticals: A consumer perspective from New Zealand. J Public Policy Marketing. 2002;21:213–23.
10. Mansfield PR. Report on DTC pharmaceutical promotion for PHARMAC; 1999. URL http://www.healthyskepticism.org/other/dtca/Report%20on%20DTCA%20for%20PHARMAC.doc
11. Wilkes MS, Bell RA, Kravitz RL. Direct-to-consumer prescription drug advertising: Trends, impact and implications. Health Aff. 2000;19:110–28.
12. Bell RA, Kravitz RL, Wilkes MS. Direct-to-consumer prescription drug advertising and the public. J Gen Intern Med. 1999;14:651–7.
13. Hollon M. Direct-to-consumer marketing of prescription drugs. JAMA. 1999;281:382–4.
14. Pharmac. Pharmac submission for Ministry of Health review of direct to consumer advertising. Wellington: Pharmac; 2000.
15. Vaithianathan R. Better the devil you know to the doctor you don't: Is advertising drugs to doctors more harmful than DTC?; 2004, 28 September. URL: http://homes.eco.auckland.ac.nz/rvai001/DTCA6.doc
16. Pinto M, Pinto J, Barber J. The impact of pharmaceutical direct advertising: Opportunities and obstructions. Health Marketing Quart. 1998;15:89–101.
17. Kelly P. Advancing public health through effective consumer health communication. Presentation to Food and Drug Administration DTC public hearing; 2005, 1 November. URL: http://www.fda.gov/cder/ddmac/dtc2005/Kelly.ppt
18. Holmer A. Direct-to-consumer prescription drug advertising builds bridges between patients and physicians. JAMA. 1999;281:380–2.

19. Calfee J. Public policy issues in direct-to-consumer advertising of prescription drugs. J Public Policy Marketing. 2002;21:174–93.

20. Calfee J. What do we know about direct-to-consumer advertising of prescription drugs? Health Aff. 2003;Web Exclusive(February):W3:116-W3:9.

21. Bradford WD, Kleit AN. Evaluating the welfare effects of drug advertising. Regulation. 2006;29:58–62.

22. Saunders B. Direct to consumer advertising: Where does the public interest lie? N Z Med J. 2003;116(1180). URL: http://www.nzma.org.nz/journal/116-1180/557/

23. Researched Medicines Industry Association of New Zealand. DTC advertising can enhance public health: The case for direct-to-consumer prescription medicine advertising. 2000, June. URL: http://www.rmianz.co.nz/pdfs/fulldtc.pdf

24. Center for Drug Evaluation and Research. Attitudes and behaviors associated with direct-to-consumer (DTC) promotion of prescription drugs: Main survey results; 2006, 13 January. URL: http://www.fda.gov/cder/ddmac/dtcindex.htm

25. Food and Drug Administration. Patient and physician attitudes and behaviors associated with DTC promotion of prescription drugs: Summary of FDA survey research results. 2004, 19 November. URL: http://www.fda.gov/cder/ddmac/Final%20Report/FRfinal111904.pdf

26. Hoek JA, Gendall P. Direct to consumer advertising of prescription medicines: A consumer survey. Palmerston North: Department of Marketing, Massey University; 2003.

27. Hoek JA, Gendall P, Calfee J. Direct-to-consumer-advertising of prescription medicine in the United States and New Zealand: An analysis of regulatory approaches and consumer responses. Int J Advertising. 2004;23:197–227.

28. Kopp S, Bang HK. Benefit and risk information in prescription drug advertising: A review of empirical studies and marketing implications. Health Mgmt Quart. 2000;17:399–56.

29. Anthony JJ, Hoek JA, Holdershaw J. Communication of risks and side effects in prescription medicine advertising. In: Geursen G, Kennedy R, Tolo M, editors. Australia New Zealand Marketing Academy Conference. Adelaide: ANZMAC, 2003:83–9.

30. Slaughter E. 5th annual survey: Consumer reaction to DTC advertising of prescription medicines: Honoring requests for prescription medicines. Emmaus, Pa: Prevention Magazine, Rodale Publications; 2002.

31. Hoek JA, Gendall P. Wishful thinking or hardcore hostility? Viewers' responses to television advertising. Int J Advertising. 1994;13:461–72.

32. Conner M, Norman P. Health behavior. In: Johnston, DW, Johnston, M, editors. Comprehensive clinical psychology volume 8: Health psychology. Amsterdam: Elsevier; 1998, p1–37.

33. Bandura A. Self-efficacy: Toward a unifying theory of behavioral change. Psych Rev. 1977;84:191–215.

34. Lennings C. An evaluation of a generalised self-efficacy scale. Personality and Individual Differences. 1994;16(5):745-50.

35. Bandura A. Self-efficacy: The exercise of control. New York: WH Freeman; 1997.

36. Rothschild ML. Carrots, sticks and promises: A conceptual framework for the management of public health and social issue behaviors. J Marketing. 1999;63:24–37.

37. Berry TR, Howe BL. The effects of exercise advertising on self-efficacy and decisional balance. Am J Health Behav. 2005;29:117-26.

38. Maubach, NB, Hoek, JA. New Zealand general practitioners' views on direct-to-consumer advertising of prescription medicines: a qualitative analysis. NZMJ. 2005;118(1215). URL: http://www.nzma.org.nz/journal/118-1215/461/content.pdf

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

A huge abdominal lump with multiple bony bumps

Ujjwal Bansal, Dipesh Duttaroy, Jitendra Jagtap, Gunjan Patel

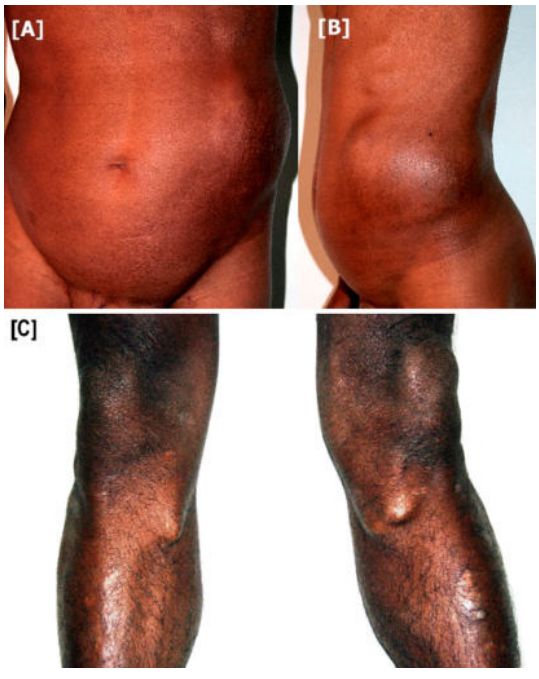
Hereditary multiple exostoses (HME) or osteochondromatosis is a familial autosomal dominant disorder associated with multiple bony protrusions, with cartilage caps usually arising from the lower femur, upper tibia, upper humerus, or the pelvis. Osteochondromas are usually asymptomatic, but may be associated with cosmetic deformities, impairment of neurovascular or musculo-tendinous functions, and malignant transformation into chondrosarcoma.

We report on a patient with multiple exostoses: well-differentiated secondary chondrosarcoma arising from an iliac osteochondroma.

Case report:

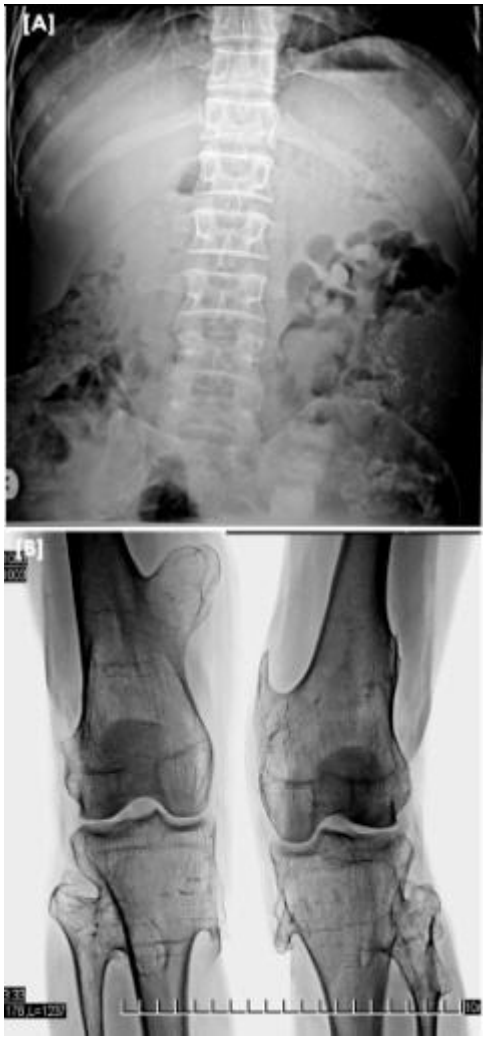
A 45-year Asian Indian male presented with a huge lump on the left side of abdomen of 5 months duration with dull aching continuous pain over it for 1 month. Examination revealed bony hard projections on the medial aspect of both the lower end of femur and upper end of tibia (Figure 1C). There was a lobulated, hard, immobile retroperitoneal lump (25×20 cm) occupying the left hypochondrium, lumbar, iliac, hypogastric, and umbilical regions (Figure 1A,B) whose lower border merged with the ilium.

Figures 1A: Anterior abdomen showing the lump; 1B: Left lateral abdomen showing the lump; 1C: Anterior aspect of both knee joints showing exostosis arising from the tibia



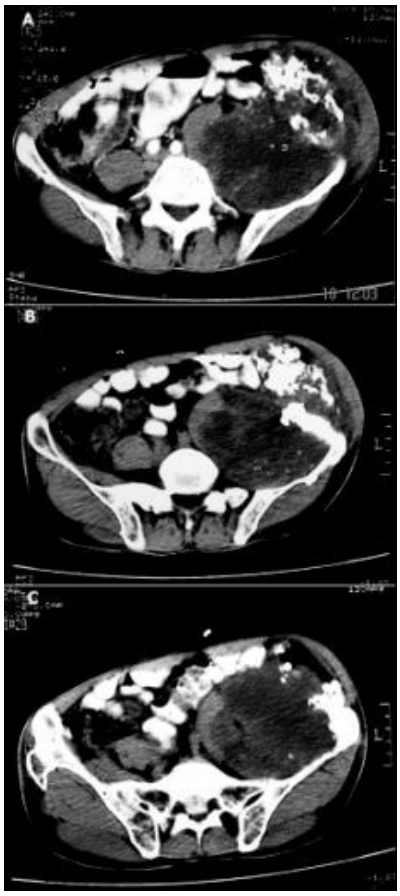
Ultrasonography demonstrated a retroperitoneal mass with mixed echogenicity extending into the pelvis. Radiography revealed a large soft tissue shadow with multiple calcific foci in left abdomen (Figure 2A) as well as pedunculated exostoses of tibia and left femur, and sessile exostoses of the right femur and the left fibula (Figure 2B).

Figures 2A: Radiograph of abdomen showing calcification with soft tissue shadow in the left lower abdomen; 2B: Radiograph both knees showing multiple exostoses

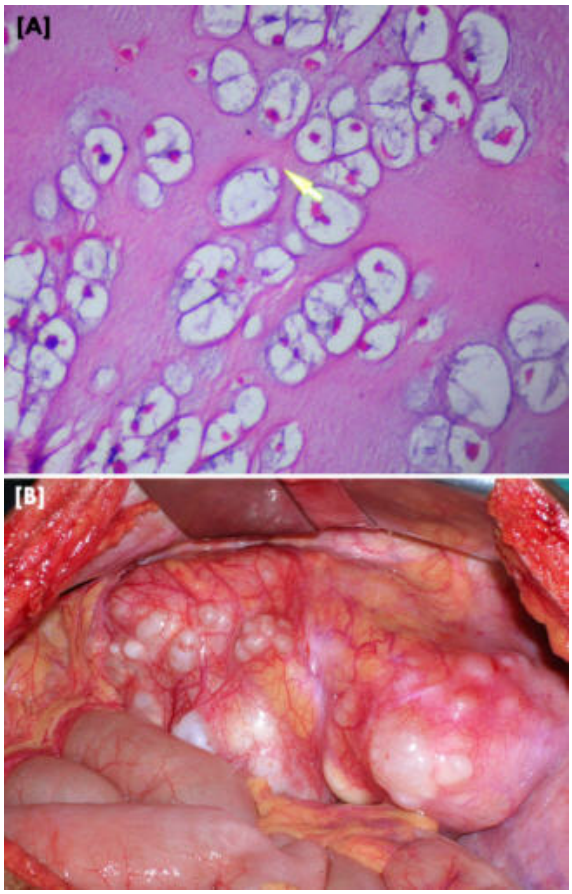


Abdominal CT (Figure3 A,B,C) showed a large heterogeneously enhancing soft tissue mass lesion in the left flank. There was evidence of splotchy calcification and central non-enhancing areas in the lesion. It was displacing the ilio-psoas muscle anteriorly, bowel medially, and left external iliac artery to right. There was pressure erosion of the left iliac wing with exostoses involving both the iliac blades.

Figures 3A,B,C: Axial CT scan abdomen through different levels showing heterogeneously enhancing soft tissue mass with calcification, pressure erosion, and bony exostoses



Figures 4A: Microphotograph 45 × (haematoxylin-eosin) showing nuclear atypia consistent with well differentiated chondrosarcoma. 3B: Intraoperative view showing a nodular white neoplastic mass



Histopathology after a trucut needle biopsy revealed multiple clustered chondrocytes with vacuolated cytoplasm, a few multinucleated, in lacunae within a chondroid matrix, consistent with a well-differentiated chondrosarcoma (Figure 4A). The patient underwent an exploratory laparotomy with wide excision of the tumour (Figure 4B).

Discussion

Osteochondroma is the most frequent benign bone tumour.[1] Multiple such tumours occur in an autosomal dominant disorder known as osteochondromatosis, hereditary multiple exostoses (HME) or diaphyseal aclasis. They are commonly seen at the metaphyses of the lower femur, upper tibia, upper humerus, scapula, and the pelvis; 1% to 5% of multiple osteochondromas may undergo malignant transformation into chondrosarcomas.[1–3]

Chondrosarcoma is the second most common primary malignant tumor of bone of cartilaginous origin, representing approximately 25% of all primary osseous

neoplasms.[4–7] They usually involve the pelvic bones, femur, humerus, ribs, scapula, sternum, or spine and are classified according their location into central, peripheral and juxtacortical.[1,4,8]

Primary chondrosarcomas arise *de novo*, whereas secondary chondrosarcoma originate in a preexisting lesion such as an enchondroma or osteochondroma.[4,5,8,9] The risk of chondrosarcoma is greater in people with enchondromatosis syndromes (Ollier disease, Maffucci syndrome, metachondromatosis) and in those with HME.[1,4] They are graded into well, moderately and poorly differentiated types based on their histological features such as cellularity, matrix content, character of cells, and replicative activity.[1,3–5,8,10]

Transformation of osteochondromas into chondrosarcomas occurs between the ages of 20 to 40 years.[1,4] Radiographs classically show lucent lesions due to cortical destruction, soft tissue mass, periosteal reaction, destruction or pressure erosion of adjacent bone, and endosteal scalloping with matrix calcification and well-organized calcific rings in well-differentiated tumors.

Poorly differentiated tumors contain scattered, irregular, punctate calcifications and sometimes tumour matrix with no calcification at all.[4,8,9] CT scan provides important knowledge regarding the intraosseous and soft tissue extent of the tumour. Tumours appear as lucent areas containing chondroid matrix calcification.

Differential diagnosis of retroperitoneal tumours with calcification include ganglioneuroma, schwannoma, paraganglioma, hemangioma, mature and malignant teratoma, undifferentiated pleomorphic sarcoma, dedifferentiated liposarcoma, malignant mesenchymoma, and extraskeletal osteosarcoma.[4,11]

MRI is useful in defining the full extent of the tumour in anatomically complex regions and shows lobulated homogeneous or inhomogeneous lesions of high signal intensity in T2-weighted spin echo images.[2,4,8] There is greater focal or diffuse enhancement following intravenous contrast administration in high grade chondrosarcomas.[8]

Surgery is the main treatment modality. Surgical options—which include radical excision; wide local excision; and even marginal, partial, or intralesional excision—depends on the tumour size, tumour grade, and the local infiltration. High-grade tumours require complete surgical excision while low grade chondrosarcomas may be treated with contaminated margins to reduce operative morbidity, without compromising survival rates.[5,6]

Chondrosarcomas respond poorly to radiotherapy and chemotherapy.[4–7,9,10] The prognosis with respect to local control and/or survival depends on the histological grade, the surgical stage, the subtype of chondrosarcoma, adequate margins of resection and, the location of tumour.[7,9,10]

Due to a high incidence of late local recurrence, chondrosarcomas should be histologically graded accurately, treated effectively via surgery, and carefully followed up for at least 5 years.

Author information: Ujjwal Bansal, Junior Resident; Dipesh D Duttaroy, Associate Professor; Jitendra Jagtap, Junior Resident; Gunjan Patel, Junior Resident; Department of Surgery, Government Medical College & Sir Sayajirao General Hospital, Baroda, Gujarat, India

Correspondence: Dipesh D Duttaroy, Associate Professor, Department of Surgery, Government Medical College & Sir Sayajirao General Hospital, Baroda, Gujarat, India 390001. Email: drduttaroy@gmail.com

References:

1. Rosai J. Bone and joints. In: Rosai J ed. Rosai and Ackerman's Surgical Pathology. 9/e (Vol.2). St.Louis, Missouri: Mosby; 2004, p2137–236.
2. Bovee JV, Sakkers RJ, Geirnaerdt MJ, et al. Intermediate grade osteosarcoma and chondrosarcoma arising in an osteochondroma. A case report of a patient with hereditary multiple exostoses. J Clin Pathol. 2002;55:226–9.
3. Kivioja A, Ervasti H, Kinnunen J, et al. Chondrosarcoma in a family with multiple hereditary exostoses. J Bone Joint Surg Br. 2000;82:261–6.
4. Hide G. Chondrosarcoma. [cited 11 August 2005]. URL: http://www.emedicine.com/radio/topic168.htm
5. Etchebehere M, de Camargo OP, Croci AT, et al. Relationship between surgical procedure and outcome for patients with grade I chondrosarcomas. Clinics. 2005;60:121–6.
6. Donati D, El Ghoneimy A, Bertoni F, et al. Treatment and outcome of conventional pelvic chondrosarcoma. J Bone Joint Surg Br. 2005;87:1527–30.
7. Pring ME, Weber KL, Unni KK, Sim FH. Chondrosarcoma of the pelvis. A review of sixty-four cases. J Bone Joint Surg Am. 2001;83-A:1630–42.
8. Resnick D, Kyriakos M, Greenway GD. Tumors and tumor-like lesions of bone: Imaging and pathology of specific lesions. In: Resnick D ed. Diagnosis of bone and joint disorders, Vol 6. 3rd ed. Philadelphia: WB Saunders Company; 1995, p3628–938.
9. Gibbs CP Jr, Weber K, Scarborough MT. Malignant bone tumors. Selected Instructional Course Lectures: The American Academy of Orthopaedic Surgeons. J Bone Joint Surg Am. 2001;83-A:1727–45.
10. Lee FY, Mankin HJ, Fondren G, et al. Chondrosarcoma of bone: an assessment of outcome. J Bone Joint Surg Am. 1999;81:326–38.
11. Secil M, Mungan U, Yorukoglu K, et al. Case 89: Retroperitoneal extraskeletal osteosarcoma. Radiology. 2005;237:880–3.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Multi modality treatment of an extensive pleural thymoma

Elizabeth Clayton, Yasotha Kathiravel, Harsh Singh

Abstract

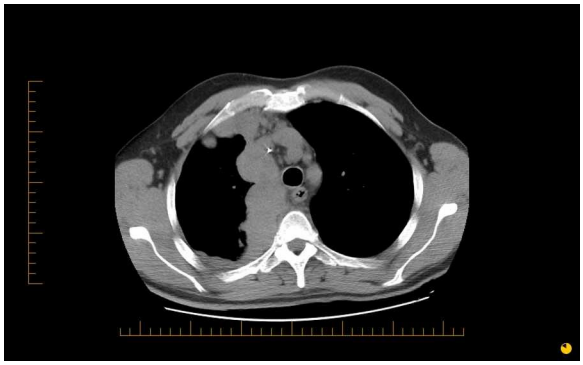
Thymomas are the most common tumours of the anterior mediastinum, with most cases presenting incidentally. We present a case of extensive pleural thymoma initially thought to be unresectable, which was treated with neoadjuvant chemotherapy and pleuro pneumonectomy. We discuss the multimodality approach to management of this tumour, in particular the use of neoadjuvant chemotherapy prior to surgical resection.

A 47-year-old male presented with numbness of his tongue, and altered taste sensation. He subsequently complained of malaise and right-sided pleuritic chest pain, with an associated cough, slight mucoid sputum, and fever. He was a lifelong non-smoker with no significant past medical history. A provisional diagnosis of lower respiratory tract infection was made, and he was initially managed with oral antibiotics. A chest radiograph subsequently revealed pleurally based lesions.

After the patient developed further symptoms of weight loss, anorexia, dysphonia, increasing fatigue, and nasal regurgitation of water, a clinical diagnosis of myasthenia gravis (MG) was made.

An initial computed tomography (CT) scan of the thorax showed extensive multifocal pleural thickening and nodularity of the right lung, with involvement of the fissure and intra parenchymal pulmonary nodules were noted. The right paratracheal and sub carinal lymph nodes were enlarged. The core biopsy (under CT guidance) revealed features of a type B1 thymoma.[1] Based on the Masaoka clinical staging system[2] this was considered as stage IVA disease, defined as pleural or pericardial metastatic spread.

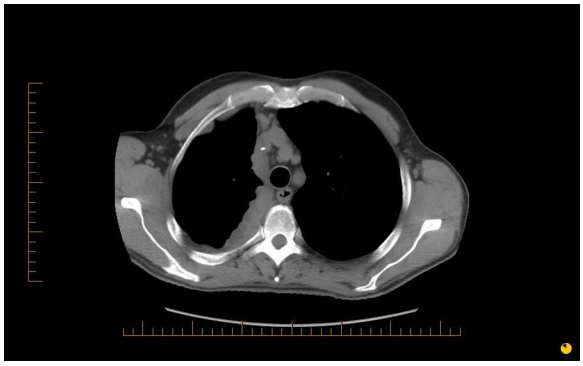
Figure 1. Inoperable extensive right pleural thymoma



However the initial size and extent of the tumour, made it unsuitable for surgical resection (Figure 1). Following a multimodality discussion, it was thought that surgical debulking, and possible complete resection, could be considered, following review of response to neoadjuvant chemotherapy. He underwent a chemotherapy regime of cisplatin, doxorubicin, and cyclophosphamide for four cycles over an 8-week course.

Post chemotherapy CT demonstrated marked interval reduction in the size of the mediastinal and right pleural disease (Figure 2). He underwent an extended right pleuro pneumonectomy with excision of the diaphragm and pericardium. The thymus gland and a large area of pericardium with tumour involvement were excised. The pleural cavity showed no further evidence of tumour seedlings.

Figure 2. Post chemotherapy marked interval reduction of pleural thymoma



Postoperatively he spent less than 24 hours in the intensive care unit prior to transfer to the cardiothoracic ward. On the fifth postoperative day he developed an episode of acute shortness of breath, and tachycardia. He underwent a VQ scan, which showed no evidence of a pulmonary embolus. He returned home 9 days postoperatively.

Histology revealed an extensive thymoma mixed type B2/B3[1] with extensive focal capsular invasion and tumour identified abutting lung parenchyma, involving pleura, and pericardium, with no evidence of lymph node metastases.

Two months postoperatively the patient presented to clinic with a week's history of productive cough associated with pyrexia. Chest radiography was unchanged from previous films. He was commenced on intravenous antibiotics and admitted to hospital for observation. He was found unresponsive the next morning.

Post mortem revealed that the cause of death was respiratory failure in the context of acute bronchitis in his remaining left lung. It was noted that his co-existing myasthenia gravis might have been the cause for his respiratory depression. There was no evidence of tumour reoccurrence at post mortem.

Discussion

Thymomas are derived from epithelial cells of the thymus and are the commonest anterior mediastinal neoplasm; in the adult population, however, the overall incidence is rare.[3] The propensity of the tumour to be malignant is determined by the

invasiveness of the thymoma, locally, via pleural dissemination, or systemic metastases.[4]

Peak incidence of onset is between 40 and 60 years of age, with no sexual predilection.[5] Presentation of thymomas varies, from local symptomatic symptoms such as cough, to incidental detection on chest radiographs, or from screening due to its associations with autoimmune and immunodeficiency disorders.[5,6]

Up to 30 to 65% of patients with thymoma have been reported to suffer from myasthenia gravis. Furthermore, 28% will present with other immune disorders such as lupus erythematous, pure red cell aplasia, Cushing's syndrome, and hypogammaglobulinaemia.[6]

Traditionally, thymomas are classified into three histological types based on the predominant cell type of lymphatic, epithelial and lymphoepithelial.[4] Recently the World Health Organization reached a histological classification based on both morphology and lymphocyte to epithelial cell ratio.[1] In 1981, Masaoka developed an anatomic classification based on the presence or absence of gross or microscopic invasion of the capsule and the presence or absence of metastases.[7] This has since been upgrade in 1994 to the modified Masaoka staging system and is now the most widely accepted staging system on which current management options are based.[2]

We present a case of an extensive pleural malignant thymoma (IVa) with myasthenia gravis. Management involved a multimodality team of cardiothoracic surgeons, oncologists, radiologists, histopathologists, and neurologists. The extensive tumour, thought to be initially unresectable, was subsequently completely resected, following neoadjuvant chemotherapy. His death was thought to be related to infection, possibly complicated by myasthenia gravis.

Surgical intervention is the treatment of choice by wide consensus, as complete resection remains an important factor in the survival of patients with locally advanced malignant thymoma.[8,9] However, radical resection is not always feasible for invasive and metastatic lesions (stages III and IVa).[2]

Thymomas are seen to be both chemo- and radio-sensitive, and recent reports using preoperative (neoadjuvant or induction) chemotherapy to transform a patient with inoperable disease into a patient with operable disease have shown good results.[8,9]

Venuta et al analysed multimodality treatment of thymomas compared to historical controls (those who received surgery alone). They showed that the complete resection rates were improved in the multi modality group relative to the surgery alone group (77% vs 33.3% for stage IV tumours).[9]

Shin et al have shown good response to neoadjuvant chemotherapy of 92% in 12 patients (stage III and IVA), and subsequent complete resection in 82%. Following adjuvant chemotherapy and radiotherapy they have shown disease-free survival at 7 years to be 73%.[8] Ongoing problems with research relate to low incidence, and therefore small numbers of patients with thymoma to produce randomised control trials.

This case demonstrates that thymomas are often associated with paraneoplastic syndromes such as myasthenia gravis and therefore their treatment may affect prognosis.[10] Furthermore, as this case highlights, multimodality treatment with

neoadjuvant chemotherapy, followed by radical surgical resection, is highly effective and may cure locally advanced, unresectable thymomas.

Author information: Elizabeth S J Clayton; Yasotha Kathiravel; Harsh P Singh; Department of Cardiothoracic Surgery, Christchurch Hospital, Christchurch

Acknowledgement: We acknowledge the assistance of Dr Sharyn MacDonald for radiographic images and interpretation.

Correspondence: Harsh P Singh, Department of Cardiothoracic Surgery, Christchurch Hospital, Private Bag 4710, Christchurch. Fax: 03 364 1361; email: HarshS@cdhb.govt.nz

References

1. Okumura M, Ohta M, Tateyama H, et al. The World Health Organization histologic classification system reflects the oncologic behaviour of thymoma: a clinical study of 273 patients. Cancer. 2002;94:624–32.
2. Masaoka A, Yamakawa Y, Niwa H, et al. Thymectomy and malignancy. Eur J Cardiothorac Surg. 1994;8:251–3.
3. Engels EA, Pfeiffer RM. Malignant thymoma in the United States: demographic patterns in incidence and associations with subsequent malignancies. Int J Cancer. 2003;105:546–51.
4. Hasserjian RP, Strobel P, Marx A. Pathology of thymic tumours. Semin Thorac Cardiovasc Surg. 2005;17:2–11.
5. Wright CD, Kessler KA. Surgical treatment of thymic tumours. Semin Thorac Cardiovasc Surg. 2005;17:20–6.
6. Souadjian JV, Enriquez P, Silverstein MN, Pepin JM. The spectrum of diseases associated with thymoma. Coincidence or syndrome? Arch Intern Med. 1974;134:374–9.
7. Masaoka A, Monden Y, Nakahara K, Tanioka T. Follow-up study of thymomas with special reference to their clinical stages. Cancer. 1981;48:2485–92.
8. Shin DM, Walsh GL, Komaki R, et al. A multidisciplinary approach to therapy for unresectable malignant thymoma. Ann Intern Med. 1998;129:100–4.
9. Venuta F, Rendina EA, Pescarmona EO, et al. Multimodality treatment of thymoma: a prospective study. Ann Thorac Surg 1997;64:1585–91; discussion 1591–2.
10. Kondo K, Monden Y. Thymoma and myasthenia gravis: a clinical study of 1,089 patients from Japan. Ann Thorac Surg. 2005;79:219–24.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

What part does a national health call centre play in an integrated primary care service?

Ian St George, Matthew Cullen, Michelle Branney

When the then National government's Health Minister, Wyatt Creech, saw the England telephone triage and advice service, *NHS Direct*, he discerned a need for a similar service in New Zealand (NZ).

McKesson NZ Ltd won the tender and *Healthline* was introduced as a pilot programme in 2000, in association with St John. It was supported by succeeding Labour governments, and after an independent evaluation had judged the pilot a success, with the incorporation of *PlunketLine* for well child calls, it became a national programme in 2005.

It remains a free, 24×7 programme, in which nurses give advice supported by clinical software, and their activities are recorded and analysed by sophisticated information technology.

Aims

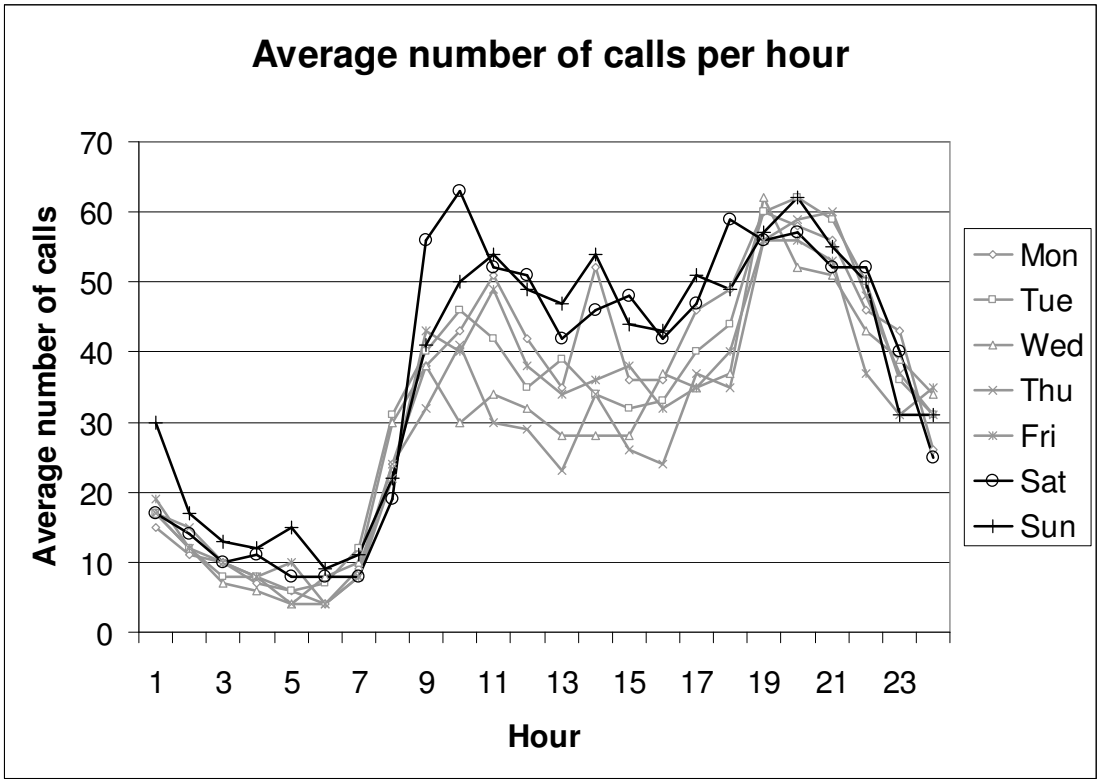
The initial aims in introducing the service were altruistic and economic. A free advice line would be accessible to all, especially to those for whom the cost posed a barrier to existing primary care services. Furthermore, it would manage demand so people who were uncertain what level of care they should seek would be directed to the right place, and at the right time; they would be taught to cope with their minor illnesses at home, freeing emergency departments and general practices for more serious work.[1]

Healthline has succeeded in reaching those in greatest need: use by lower socioeconomic groups is consistently higher than that by higher socioeconomic groups.[2] Use by Maori is consistently higher than use by other ethnic groups,[3] and use by the aged is similar to their patient-initiated consultation rate in general practice.[4]

The effect on other primary care services has been a volume reduction in telephone consultations—in emergency departments,[5] and after hours in rural practices.[6] Furthermore, callers who do need to see a doctor are more often triaged to a lower than to a higher level of acuity and urgency, thus reducing the demand on acute primary care (notably emergency departments and after hours general practice) services.

About a third of *Healthline* calls are at night, a third at weekends, and a third during business hours.[7] Figure 1 shows the number of calls by hour of the day and by day of the week; 9am to 9pm are the busy hours, and that trend is accentuated at weekends, thus suggesting *Healthline* is used as an alternative source of primary care advice when other services are perceived to be less easily available. That choice has been judged to be safe.[8]

Figure 1. The average number of calls to *Healthline* per hour, by per day of the week, over three months.



Current call centre services

McKesson New Zealand has call centres in Wellington and Manukau City. They are linked to form a single virtual health call centre. This health call centre delivers several services:

- *Healthline* provides triage for symptomatic callers, but also health information to asymptomatic callers, and provider referral for those seeking, for instance, a general practitioner, the nearest branch of the Cancer Society, a Plunket nurse, or other providers. The database of providers is updated regularly by local St John officers.
- In 2006 McKesson NZ won the tender to provide the *Well Child Telephone Advice Service*, and that service is also accessed via the *Healthline* 0800 telephone number. As in all calls to the line, the emphasis is initially on triage, to ensure that the problem is not symptomatic of underlying illness, then on well child and parenting counselling or referral to existing services.
- McKesson NZ also operates *Mental Health Line* for a number of District Health Boards; mental health professionals field calls from providers and service users/tangata whaiora, and triage or give advice.[9]
- During the recent Meningococcal Vaccine Strategy, the Ministry contracted McKesson NZ to provide the *National MeNZB Support Line*, to answer

enquiries about immunisation. Overall 44,674 calls were made over 78 weeks, 92% during business hours, and 86% from landline phones. 2233 follow-up calls were made, 692 callers were referred to a local provider, 5163 to a Public Health Unit, 443 to the Immunisation Advisory Centre, and 334 to *Healthline*.

What else can a national health call centre do?

Syndromic surveillance—There is an opportunity to use a triage line for a national public health system of symptom surveillance. In Britain, Canada, and the USA the term "syndromic surveillance" has been used for such activities.

Public health surveillance serves many functions, but one important task is outbreak detection—identifying a rise in frequency of disease above the background pattern. Currently outbreaks are recognised from accumulated reports of notifiable diseases, via voluntary reporting by sentinel practices and laboratories, or by alert clinicians bringing clusters of diseases to attention.

Now the threats of bioterrorism and pandemic influenza, and the advent of a national telephone triage service, with its ready availability of electronic data, have triggered new surveillance systems to detect outbreaks earlier (in the United States data from telephone triage calls were one to five weeks ahead of surveillance data collected by the Center for Disease Control using orthodox reporting methods).[10] These systems detect unusual geographic or temporal clustering of symptoms, and thus provide an early alert that may indicate an outbreak.

Harvard researchers have identified five stages in the detection of an outbreak: *data acquisition, syndrome grouping, modelling, detection,* and *alarm*.[11]

Data acquisition and syndrome grouping requires a sophisticated recording and reporting system linked to telephony (McKesson has that).

Historical data have to be analysed over several years to establish a model (a denominator) for the normal or expected pattern of complaints. McKesson now has that.

Detection of a real outbreak involves comparison of the observed values (for example, daily frequencies of patients presenting with a syndrome) against the expected pattern, to determine if activity is really abnormal, and to decide whether the abnormal pattern warrants raising an alarm. Such a system requires partnerships among public health agencies, clinicians, data providers, emergency response teams, the police, and the wider community.

In England and Wales, call data (site, symptom, age-group, call outcome) on 10 key symptoms are transferred every weekday from 23 *NHS Direct* call centres to the Health Protection Agency at West Midlands. Upper confidence levels (99.5% level) of symptomatic calls are developed, and significant statistical excesses ("exceedances") are automatically highlighted and assessed by a multidisciplinary team. The team considers the proportion of outcomes recommending emergency care, the age distribution, seasonal baselines, levels of similar activity at neighbouring *NHS Direct* sites, previous exceedances at that site and current known community levels of disease. A geocoding system can then be used to map the call addresses to check locality clustering.

If concerns persist, local health protection teams are alerted.

GP consultation with other specialists—Bradstock and her colleagues have described *GP-Psych Support*, a national Australian mental health management advice service that links general practitioners with psychiatrists by phone, fax, or email within 24 hours. The service is federally funded, began in March 2004, and is operated by McKesson Asia-Pacific.

Over the first 6 months of operation of the phone/fax arm, there were 726 case discussions between GPs and psychiatrists. A third of the GPs were rural, and 17% used the service twice or more. Most GPs (94%) accessed the service through the 1800 freecall number, rather than by fax. Three-quarters identified no other suitable, accessible source of urgent psychiatric advice. The most common topic discussed was medication (77%), with lower demand for discussions of general management principles (12%) or diagnosis (7%).

The feedback was very positive:

- 99% of respondents indicating that they would consider using the service again.
- Over 95% were satisfied with the service in terms of ease of use, helpfulness of advice, and ease of interaction with the psychiatrist.
- Over 85% rated *GP-Psych Support* as more accessible, reliable, and the advice more appropriate than other sources.
- Over 70% said contact with the service had increased their knowledge about the management of mental disorders and their confidence in managing mental health problems, and had improved the quality of care they provided to their patients.
- 53% reported greater willingness to manage complex mental health problems.[12]

In the coming world of primary care—with a big increase in chronic disease in an ageing community, and with a worsening undersupply of general practitioners as well as other specialists—the model of easy-access telephone consultation between practitioner and consultant offers sound education, skill enhancement, fewer outpatient referrals, and thus financial advantages.[13]

Chronic disease management—Disease management programs for chronically sick people are proliferating for the same reasons, and because chronic illness accounts for most health expenditure. “The personal and economic burden of a rapidly ageing population with its inherent challenge on how better to manage chronic disease represents the next global crisis,” is the slogan repeated in every issue of *DM World e-Report*.

Conditions such as asthma, chronic obstructive lung disease, heart failure, depression, diabetes, hypertension, and renal disease are being managed by programmes that incorporate emerging technologies, in particular computers with telephone links.

The results are impressive: disease management programmes appear to reduce service use and to create financial savings while enhancing self management by support, education, and involvement. That is true of diabetes,[14]asthma,[15,16] paediatric asthma,[17] and heart failure in the elderly.[18] For instance, the authors of one study noted, “a

commercially delivered heart failure disease-management program significantly reduced hospitalisations, emergency department visits, and skilled nursing facility days. The intervention group had 17% lower costs than the control group; when intervention costs were included, the intervention group had 10% lower costs".

General practitioners think it is their job to manage chronic diseases; they do not always see the need for disease management programmes. Lagging physician acceptance is the biggest obstacle to implementing disease management programmes in the United States. Many doctors retain the model of episodic care, so do not see the value of education focused, prevention focused disease management programmes.

Good programmes support (rather than replace) medical services. By providing regular contact with chronically ill patients, telephone programmes can monitor clinical status between doctor visits, deliver patient education and other counselling, send appointment reminders, and facilitate peer support and referrals for coping with illness. Doctors are freed to practise clinical medicine.

McKesson Asia Pacific is providing pilot chronic disease management programmes with several health insurers in Australia, and results should be available soon.

Telephone-based programmes are clinically effective and cost-effective. They are here to stay, and District Health Boards and Primary Health Organisations will have to decide whether to provide them themselves ("CarePlus" is one model), or to contract with an outside provider, with the advantages of sophisticated technology support.

Other applications—Bentley listed the activities of the McKesson Asia Pacific health call centre in Perth:[19] *HealthDirect* triage and advice; *HealthInfo* information and health policy; *SouthWest24* mental health services; *Residential Care Line*; *Sexual Assault Referral Centre Crisis Line*; *Drug Cautioning Line*; *Health Incident Lines* (public health emergency lines as required, e.g. SARS line); and *PEP* (post-exposure prophylaxis for HIV).

Programmes being piloted were *Secondary triage for the St John Ambulance; Chest pain program* for insured patients; *Outpatient bookings* (an appointment, reminder and tracking system); and *Surgical patient follow up.*

McKesson's Sydney centre runs the mental health service *Greater Murray Access Line*, as well as a gambling line, and chronic disease management.

Nurse-on-call has started in Melbourne. Tenders are to be called for a national service in Australia.

Conclusion

The original aims—easy access to health advice, and demand management—have expanded and multiplied, and the broader capabilities of a national health call centre are now beginning to be utilised.

Conflict of interest statement: There will be a perception of conflict of interest: all of us work for, or in association with, McKesson NZ Ltd, which operates Healthline, the Well Child Advice Line, Mental Health Line, and which operated the MenzB Line.

Author information: Ian M St George, Medical Director McKesson New Zealand; Matthew J Cullen, Co-Director McKesson Asia Pacific; Michelle Branney, General Manager McKesson New Zealand.

Correspondence: Ian St George, McKesson New Zealand, PO Box 10643, Wellington. Fax: (04) 499 2357, email ian.stgeorge@mckesson.co.nz

References:

1. St George IM, Cullen M, Branney M. A primary care demand management pilot in New Zealand: telephone triage using symptom-based algorithms. Asia-Pacific Family Medicine. 2003;2:153–6.
2. St George IM, Cullen MJ, Branney M. The deprivation levels of callers to Healthline. (For future submission.)
3. St George IM, Branney M, Horo-Gregory W, et al. Maori callers to a telephone triage service. N Z Family Physician. 2003;30:261–3.
4. St George IM. Cullen MJ, Branney M. How well does telephone triage meet the needs of older people? N Z Family Physician. 2005;32:94–7.
5. Graber DJ, O'Donovan P, Ardagh MW, St George IM. A telephone advice line does not decrease the number of presentations to Christchurch Emergency Department, but does decrease the number of phone callers seeking advice. N Z Med.J. 2003;116 (1177). URL: http://www.nzma.org.nz/journal/116-1177/495
6. St George IM, Cullen MJ, Branney M, et al. Telephone triage reduces out-of-hours work for country doctors. N Z Family Physician. 2003;30:95–9.
7. St George IM, Cullen MJ. The Healthline pilot: call centre triage in New Zealand. N Z Med.J. 2001;114(1140):429–30.
8. St George IM, Cullen M. Branney M. Healthline: do primary care doctors agree with the advice? N Z Med J. 2005;118 (1224). URL: http://www.nzma.org.nz/journal/118-1224/1693
9. St George IM, Cullen M, Wilson A, Wilson F, Dolan B, McGeorge P. The Mental Health Line. Submitted to NZ Family Physician, 2006.
10. Espino JU, Wagner MM, Tsui FCet al. The RODS Open Source Project: removing a barrier to syndromic surveillance. Medinfo. 2004;11(Pt 2):1192–6.
11. Mandl KD, Reis BY, Cassa C. Measuring outbreak-detection performance by using controlled feature set simulations. MMWR Morb Mortal Wkly Rep. 2004;53 Suppl:130–6.
12. Bradstock SE, Wilson AJ, Cullen MJ, Barwell KM. Telephone-based psychiatry advice service for general practitioners. Medical Journal of Australia 2005;183 (2):90.
13. St George IM. A renaissance of true consultation? N Z Family Physician. 2006;33:64–5.
14. Berg GD, Wadhwa S. Diabetes disease management in a community-based setting. Managed Care. 2002;June:6–11.
15. Johnson AE, Yin M, Berg G. Utilisation and financial outcomes of an asthma disease management program delivered to Medicaid members. Dis Manage Health Outcomes. 2003;11:455–65.
16. Johnson A, Berg G, Fleegler E, Sauerbrun M. A matched-cohort study of selected clinical outcomes for an asthma care support program. Disease management 2005;8:144–54.
17. Berg GD, Johnson A, Fleegler E. Clinical and utilization outcomes for a pediatric and adolescent telephonic asthma care support program. Dis Manage Health Outcomes. 2003;11:737–743.
18. Berg GD, Wadhwa S, Johnson AE. A matched-cohort study of health services utilization and financial outcomes for a heart failure disease-management program in elderly patients. JAGS. 2004;52:1655–61.
19. Bentley PJ, Turner VF, Hodgson SA, et al. A central role for the health call centre. Australian Health Review. 2005;29:435–8.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Annual Meeting in Dunedin: medical inspection of schools

The Annual Meeting of the Branch held this year in Dunedin, proved, as was expected from the high reputation for hospitality enjoyed by that city, a great success. With such hosts as Drs. Barnett, Ferguson, Batchelor, Colquhoun and many others, the success of the social part of the meeting was a foregone conclusion, and the fact of Dunedin being the seat of the medical school, practically assured the success of the scientific part.

It is to be regretted that with characteristic modesty, so many of the Dunedin doctors abstained from reading papers, preferring to give the visitors every opportunity to do so, the meeting was thus, we think, robbed of some papers which would have lent an added distinction to it, nevertheless, there were many valuable discussions, and many, old friendships were renewed.

The meeting was held in the Council Chambers of the Town Hall and the having the use of this handsome room was of course much appreciated by all.

Undoubtedly the most important business done was the holding of a debate on the question of the medical inspection of schools. Much public interest was taken in the subject and the meeting was largely attended, amongst those present being the leading teachers of the district, most of whom addressed the meeting. The debate was opened by Dr Mason with all his characteristic vigor and command of language, Dr Ferguson, Dr Truby King, Dr Ogston and many others spoke at length on the subject.

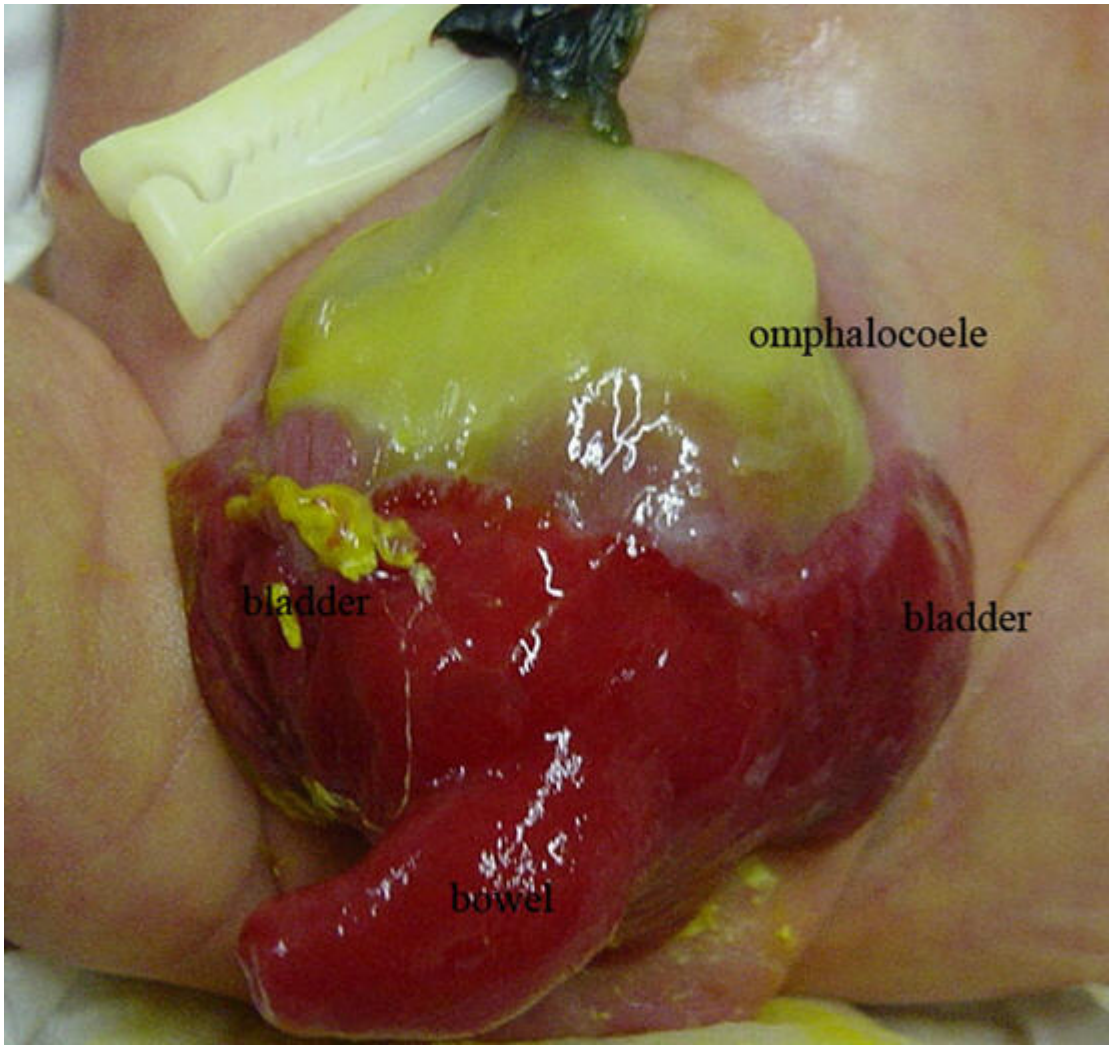
THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Exstrophy of the cloaca

Neetu Kumar, Navroop Johal, Imran Mushtaq

A newborn female presented with exstrophy of the cloaca, a condition in which there is an incomplete coverage of the infra-umbilical wall. This results in the most severe ventral midline abdominal wall defect with exposure of both bowel and bladder elements and duplication of the genitalia (Figure 1).

Figure 1. Exstrophy of the cloaca with the 'elephant-trunk' deformity, a centrally prolapsed ileocaecal segment flanked by bladder hemisections



Discussion

Exstrophy of the cloaca is one of the rare and complex malformations occurring only once in 200,000 births;[1] it is the most extreme form of the exstrophy-epispadias complex. The main features are an exstrophic central bowel field flanked by two hemi bladders.[2] Anomalies of the upper renal tracts and other organ systems are common,[3] and it is often associated with an omphalocoele, imperforate anus, and spina bifida.

The external genitalia are also abnormal and sex assignment may be difficult in the newborn period[4]—boys frequently have a minimal phallic structure. As a result many of these children are reared as females and have genital reconstruction at a later date.

Urinary continence is seldom possible, however surgical reconstruction has transformed the quality of life experienced by children with exstrophy of the cloaca. Repair of the cloacal exstrophy with a colostomy and bladder closure was performed in the neonatal period in this case and further surgery is planned in the future.

Author information: Neetu Kumar, Senior House Officer; Navroop S Johal, Research Fellow; Imran Mushtaq, Consultant Paediatric Urologist; Department of Paediatric Urology, Great Ormond Street Hospital, London, England

Correspondence: Mr N Johal, Department of Paediatric Urology, Great Ormond Street Hospital, London, W1T 8AA, England. Email: navjohal@hotmail.com

References:

1. Austin PF, Homsy YL, Gearhart JP, et al. The prenatal diagnosis of cloacal exstrophy. J Urol. 1998;160:1179–81.
2. Malone P. Bladder exstrophy and epispadias complex. In Atwell JD, editor. Paediatric Surgery. 1st edn. London: Arnold; 1998, p590–602.
3. Warner BW, Ziegler MM. Exstrophy of the cloaca. In: Ashcraft KW, editor. Pediatric Surgery. 3rd edn. Philadelphia: WB Saunders; 2000, p493–501.
4. Groner JI, Zeigler MM. Cloacal exstrophy. In: Puri P, editor. Newborn Surgery. 2nd edn. London: Arnold; 2003, p629–36.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Prostate cancer screening in elderly US men is often inappropriate

A paper and editorial in *JAMA* discuss this topical subject. The paper shows that, within the Veterans Health System in 2003, 56% of men older than 70 years who had no previous history of prostate cancer, elevated PSA level, or prostate cancer symptoms had a PSA test performed.

As most (US) guidelines do not recommend PSA testing in elderly men, the authors and commentator speculate on why practice does not comply with policy. They conclude that physicians order the tests anyway because of the patients' exaggerated fear of prostate cancer mortality and their overestimation of treatment efficacy. Physicians also order PSA tests because the reward for treatment can be significant and the penalty for failing to diagnose can be severe.

A very sad commentary on a health system bearing in mind the very old and those in poor health are unlikely to live long enough to enjoy any potential benefits from screening, whereas the harms are immediate and include anxiety, false positive tests followed by repeated needle biopsies, and lifelong serious side effects from invasive treatments.

JAMA 2006;296:2336–42

Immunising infants—does needle size matter?

Within UK general practices, infants are immunised at 2, 3, and 4 months of age, usually with a combined diphtheria, pertussis, tetanus, and *Haemophilus influenzae* type b vaccine and a meningococcal C vaccine. Much the same as in NZ.

Apparently there is controversy about whether it is better to use 25 mm length (23 gauge) needles or 16 mm length (25 gauge) needles. The point being that longer needles may cause less local reactions. A specialist vaccine group in Oxford (UK) report on a trial involving 696 infants. They conclude that long (25 mm) needles for infant immunisations can significantly reduce reactogenicity at each dose while achieving comparable immunogenicity to that of short (16 mm) needles.

BMJ 2006;333:571–4

End-of-life care decisions

The introduction of cardiopulmonary resuscitation (CPR) has been beneficial to those who have survived. But CPR decisions are often difficult to make in the elderly with multiple comorbidities, bearing in mind that the success rate in these patients is very low. Occasionally the patient will have a view on this topic—usually not for resuscitation (NFR). In most cases, medical staff raise the topic late in the illness with relatives—often with mutual discomfort. How to do it better?

This paper compares CPR decisions made in their geriatric service with those made in general medical wards. And the geriatricians did it better—much better (37% vs 2.5%!). The authors state that "geriatricians make significantly more CPR decisions that general physicians do, but still involve patient and family views in only a minority of cases, and an assessment of capacity is rarely explicitly documented." Their solution is a 3 step plan which basically involves early assessment and documentation.

Q J Med 2006;99:683–90

How to decrease catheter-related bloodstream infections in the ICU (and elsewhere?)

Intravenous catheters facilitate treatment and save lives, but catheter-related bloodstream infections are common, costly, and potentially lethal.

In this paper from Michigan, USA it is stated that each year in the United States central venous catheters may cause an estimated 80,000 catheter-related bloodstream infections and, as a result, up to 28,000 deaths among patients in intensive care units (ICUs)—an alarming statistic.

This study involved 108 ICUs, and patients were randomised to evidence-based interventions versus routine care. The recommended procedures are handwashing, using full-barrier precautions during the insertion of central venous catheters, cleaning the skin with chlorhexidine, avoiding the femoral site if possible, and removing unnecessary catheters. The intervention arm had a large (up to 66%) and sustained reduction in rates of catheter-related bloodstream infection.

Excellent—but your scribe wonders why "interventions" were not routine practice in the first place.

N Engl J Med 2006;355:2725–32

How to reduce the need for blood transfusion in hip surgery

Blood transfusion is commonly needed after hip surgery. This paper reports on a retrospective case control study testing the merit of cell salvage (i.e. returning lost blood to the patient) and the use of tranexamic acid, a fibrinolytic inhibitor. The mean amount of blood transfused was significantly reduced (62.5%) in those managed with cell salvage and tranexamic acid. The authors estimate that this would save over £300,000 (NZ $900,000) in their unit each year. There would also be a benefit from less transfusion-related illness.

J Bone Joint Surg (Br) 2006;88-B:1141–2

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Colchicine: time to rethink

We applaud Jayaprakash et al on their series of cases (in the previous issue of the *NZMJ*) highlighting the intricacies in colchicine therapy and indeed questioning the current knowledge-base on colchicines: a drug with a narrow therapeutic index often used in the treatment of acute gout.[1] Jayaprakash et al report on a majority of patients with accidental overdose,[1] however (interestingly) even the recommended dose is much higher than what is thought to be less toxic, yet effective.[2,3]

A recent review from the United States alluded to the fact that colchicine probably has the smallest therapeutic window of any drug used to treat acute gouty arthritis, but they suggested:

> "In treating acute gouty arthritis, colchicine is typically administered as an oral 0.6 mg dose, followed by 0.6 mg at hourly intervals until gastrointestinal side effects (e.g. nausea, vomiting, or diarrhea) occur or a maximum total of six to eight doses has been administered."[4]

The British National Formulary also recommends a similar high daily dose.[3] This is very similar to the dosage of colchicine suggested more than a decade ago,[2] and indeed comparable to the regimen which was also expressed in grains in Hollander's *Textbook of Rheumatology,* 1960.[3]

Regardless of the fact that there is perhaps only one double-blind placebo-controlled study on colchicine in acute gout where gastrointestinal side effects occurred before the relief of pain,[2–5] and the optimal dose of colchicine still remains elusive,[6] there has not been any significant change to the recommended dosage in acute gout after nearly half a century later.[2,3]

The suggestion to administer colchicine at frequent intervals until the development of gastrointestinal side effects is a matter of significant concern.[7] The morbidity and mortality associated with complications from colchicine therapy should be explored in detail, with the caveats to be exercised in judiciously using colchicine from a practical perspective in routine clinical practice.[8,9]

Of late, a recent systematic review has shown that there is a lack of robust data to inform the debate on the management of a common problem such as gout, and interestingly all of the drugs used to treat gout can have serious side effects.[10] Indeed, Morris et al had suggested an effective yet less toxic alternative regime with colchicine in the setting of acute gout.[3] Hence, anecdotal published case reports should not be underestimated and dismissed too quickly,[1,3,8] as they remain a valid and efficient source for signal generation and are indeed of great value for drug safety.

George I Varughese
Specialist Registrar in Diabetes
Endocrinology & General (Internal) Medicine, Queen's Hospital
Burton-on-Trent, UK
(georgeiv@doctors.org.uk)

Abraham I Varghese
Senior House Officer in Oral & Maxillofacial Surgery
Northampton General Hospital
Northampton, UK

Abd A Tahrani
Clinical Teaching Fellow (GLF Office)
Warwick Medical School, University of Warwick
Coventry, UK

References:

1. Jayaprakash V, Ansell G, Galler D. Colchicine overdose: the devil is in the detail. N Z Med J. 2007;120(1248). URL: http://www.nzma.org.nz/journal/120-1248/2402
2. Varughese GI, Varghese AI. Colchicine in acute gouty arthritis: the optimum dose? Arthritis Res Ther. 2006;8:405.
3. Morris I, Varughese G, Mattingly P. Colchicine in acute gout. BMJ. 2003;327:1275–6.
4. Cronstein BN, Terkeltaub R. The inflammatory process of gout and its treatment. Arthritis Res Ther. 2006,8(Suppl 1):S3.
5. Ahern MJ, Reid C, Gordon TP, et al. Does colchicine work? The results of the first controlled study in acute gout. Aust N Z J Med. 1987;17(3):301–4.
6. Cox AR. Colchicine in acute gout: optimal dose of colchicine is still elusive. BMJ. 2004;328:288.
7. Lilley LL, Guanci R. 'Until diarrhea occurs'? There's a maximum dosage to prevent colchicine toxicity. Am J Nurs. 1999,99:12.
8. Tahrani A, Sharma S, Macleod A, Moulik P. A case of multi-organ failure. Int J Clin Pract. 2007 (In Press).
9. Varughese GI, Varghese AI. Colchicine myoneuropathy: the role of rhabdomyolysis. Nephrology. 2006;11:481–2.
10. Sutaria S, Katbamna R, Underwood M. Effectiveness of interventions for the treatment of acute and prevention of recurrent gout--a systematic review. Rheumatology (Oxford). 2006;45:1422–31.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Pharmac dogma

In the August 1994 New Zealand Pharmaceutical Schedule, long-acting (LA) dipyridamole 150 mg caps were a specialist script fully funded at $31.99/60 caps and soluble aspirin was fully funded at $26.64/1000.

In 1995, Pharmac reference priced the two drugs and reduced the subsidy on dypyridamole to $0.96c/60 caps with full funding available on application for a special authority by a tertiary specialist cardiologist, cardiothoracic surgeons, neurologists , neurosurgeons, and general physicians. Applications for funding were for 1 to 2 years, and renewals had to be by the same group of specialists with patients having demonstrated a return of symptoms (transient ischaemic attacks [TIAs], strokes, etc).

Branded Panadol in 1992 was $41.47/1000 with a patient part charge and the fully subsidised brand of paracetamol was $33.00/1000. Neo-cytamen injections in 1992 were fully subsidised at $2.80 for 3 injections; depot medroxyprogesterone 150 mg and triamcinalone 40 mg were also fully subsidised and available on MPSO to general practitioners, for administration to patients.

By 2002, LA dipyridamole 150 mg caps were still subject to specialist application special authority for full funding of the manufacturer's price which had fallen to $22.39/60. The referenced subsidy had also reduced down to $0.78c/60.

Pharmac had obviously reviewed the drug in the intervening 8 years since 1994, because, by then, approvals were valid indefinitely and didn't have to be renewed every 1 or 2 years by an application from the GP to the specialist to fill in the form to get the patient a subsidy.

Soluble aspirin was no longer fully funded in 2002 and carried a manufacturer's premium (also called an EFP: extra charge for proprietary medicine) and a patient part charge, because the drug was now reference priced to insoluble aspirin at $26.50/1000. The cheapest soluble brand of aspirin (which at the time was a scored tablet) was $26.64/1000.

Patients who demanded a fully subsidised aspirin as prophylaxis against heart attacks and strokes got insoluble aspirin dispensed, rather than the more sensible scored (but only partially subsidised) soluble Solprin brand, which carried a part charge of $0.14c/1000, plus mark-up etc. Patients with more personal funding bought unfunded enteric-coated Cartia from the chemist at about $4.00 per 30.

In the Pharmaceutical Schedule as at 1/2/2007 the supplier's price for 60×150 mg dypiridamole has dropped to $11.52 for 60. Assuming the excellent reasons for the necessity of the specialist application special authority that existed in 1999 to 2003 based on the massive cost to the health service of this drug, and the likely squandering of scarce resources by general practitioners is still the main reason for keeping the bureaucracy intact (if it now no longer exists), surely the Special Authority requirements can be dropped completely and the suggested guidelines added instead.

Alternatively, are GPs being given free* access to modern drugs for their patients, like low molecular weight heparin and clopidogrel?

I note that GPs can now apply for special authority funding for clopidogrel at $168.17 per 28; a cost which hugely exceeds the cost of dipyridamole.

It would thus appear that Pharmac does not have a process of annual reviews of restrictions on prescribers' access to funded medications. Perhaps the justification for all specialist endorsements and special authorities need review every year to eliminate (as much as possible) wastage of patient and doctor time and effort as well as the country's coffers.

With the removal of Vit B12 and kenacort from the medical practitioner supply order (MPSO), every injection carries a dispensing fee and prescription charge for the patient. As well, there is the trip to the GP for the diagnosis, treatment plan, and prescription; then a trip to the pharmacist; then another appointment with the doctor or nurse for the administration of the injection.

Assuming Pharmac's cost efficiency/benefit ratio, experts completely discounted the patient's time and the two appointments necessary under the new system, as well as the individual dispensing fee required to be paid by the tax payer for the injectable [medication], then the dogma associated with removing the items (but not Depot Provera) from the MPSO may be justified.

If the increased dispensing fees plus the patient's time and the two appointments are factored into the cost benefit to the country of removing the items from the MPSO, then the cost of dogma is excessively high, and while neither of the drugs are emergency medications , neither is depot medroxyprogesterone acetate injection, and the overall cost to both patients and Pharmac's/DHB coffers would be considerably reduced.

As with the tender for crumbly, difficult-to-swallow Pacimol brand paracetamol which saved less than 1c per day per patient on 8 per day on Pharmac's drug budget over the previously subsidised Panadol brand, a Pharmac representative said "how could we not save ½ a million dollars?" Given the difficulties so many patients had with Pacimol, fortunately now replaced with slip coated biconvex brand Panadol, should patients have been given the option of paying an extra 24c per month or $3.50 (usually reimbursed on a disability allowance anyway) for something they could actually take.

Sometimes it seems that the doctrinal approach of the drug buying agency in New Zealand forgets there are patients and doctors and their time and values to also consider when making extremely penny-pinching decisions of drug quality versus price.

* Free means unencumbered by petty bureaucracy like writing to a specialist colleague to get a drug with a specific indication funded for a patient or the worse Special Authority bureaucracy which is solely designed to create a bureaucratic impediment to prescription of usually expensive drugs.

Bill Douglas
General Practitioner
Wanganui

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

GP work: what is it worth?

Having read the lengthy dissertation form LECG consultancy on the DHB website[1] (http://www.dhbnz.org.nz/Site/Current-Issues/GP-fee-increases.aspx) on how it arrived at the fair and equitable figure of 4.5% as being a reasonable increase for GPs to put up their fees for 2006, I should be most interested in the rationale and basis where by the Ministry of Health approved a 275% increase in the LMC payments from a very reasonable fee of $90.00 in July 2002 to $337.50 for the first two trimesters of pregnancy care from 1/7/2007.[2,3]

Assuming there is a widespread perceived shortage of GPs around the country—giving one group of health workers such a huge percentage and real increase in income and such a pittance to another group—what is the Ministry's preconceived idea of what a midwife should earn from their work?

Matching up the registered addresses from the Midwifery register in November 2005[4] (with the place of delivery of all 54,581 births for 2003 in the NZHIS Bulletin 2006[5] for most areas against a birthing centre) the ratio of Midwives with an Annual Practicing Certificate to deliveries in each area is about 1:20, with the range being 1:8 to 1:30.

For example, Winton in 2003 had three resident registered midwives in 2005 and 30 deliveries in 2003, giving a ratio of 1:10. Thus it would appear that the Ministry is working towards an income for independent midwives of around $60,000 to $80,000 for being LMC for 20 pregnancies per annum (Caesareans included) The workload generated by 20 pregnancies and deliveries would be unlikely to involve more than 800 hours per annum, thus giving a net profit of around $100/ per hour or $3,000–$4,000 per pregnancy.

Self-employed General Practice is perceived as paying very well according to the 2006 Waikato Management School Survey[6] but requires the GP to see patients and perform revenue generating activities for 2000–2500 hours per annum. Employee GPs are starting to get vocational registration recognised as a specialist pay rate in their awards with West Coast and some Trusts paying the same annual base rates as the equivalent ASMS National Meca.[7]

As yet, the equal work for equal pay doesn't seem to be a catchcry for the public versus private primary sector health workers.

Many managers in PHOs, DHBs, and Ministry see value in themselves but don't perceive that General Practice also needs a fair remuneration for their work.

Bill Douglas
General Practitioner
Wanganui

References:

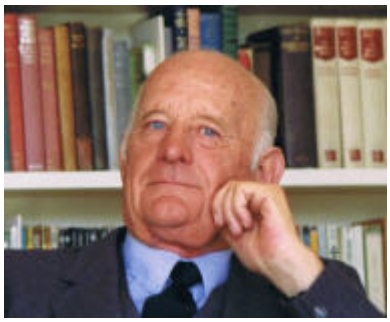
1. Davies P, Hope S, Moore D. LECG report on provision of an annual Statement of reasonable General Practice fees increase; Dec 2006. URLhttp://www.dhbnz.org.nz/Site/Current-Issues/GP-fee-increases.aspx
2. Primary Maternity Services Notice 2007 Schedule 1 Fees P64–66.
3. Fee Comparison 2002/2007 in Guide to the proposed Section 88 Notice for Primary Maternity Services 2006 PP 15–16 Ministry of Health.
4. Midwivery Register. Published electronically by the Midwifery Council of New Zealand. Purchased Nov 2005.
5. Report on Maternity Maternal and New Born Information 2003. Published 2006 by New Zealand Health Information Service.
6. General Practice Overheads and Profit 1998-2005 Waikato Management School Benchmarking Survey 2005. URL: http://wms-soros.mngt.waikato.ac.nz/NR/exeres/4B7EC13B-21EC-4FCF-9422-6550366940E5.htm
7. National DHB Collective Agreement (MECA) 2006. http://www.asms.org.nz/SITE_Default/SITE_Employment_in_NZ/National_DHB_Collective_Agreement_-_MECA/MECA.asp

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Edwin Keith Macleod

24 December 1918—4 December 2006

Keith Macleod was born and raised in Christchurch, his father an academic physicist, his mother a published author.



Named for an uncle killed at the Battle of Passchendaele, he attended Christchurch Boy's High School excelling as a cricketer, a rugby player, and a poet. Graduating in 1943 he soon found himself in Italy with the 27th Battalion. Following the war and entrance into the RACP, he attended at Queen Square, London and realised that neurology combined with literature, philosophy, and theology could sustain his energetic and creative mind.

After brief appointments at Burwood and Timaru Hospitals he was appointed to Horace Smirk's Department of Medicine at the University of Otago in 1951.

He retired in 1983 having served as neurologist at Dunedin Hospital, Ashburn Hall and in part-time private practice over these decades. Though having widely differing interests to Martin Pollock, together they most compatibly provided neurological services to the region over much of this period.

Keith was a busy and humane clinician, intensely curious about his patient, their nervous system and their 'self'. Roberta Highton recalls every one of his patients was of deep concern and that he would be prepared to give his greatest efforts even to the most unpromising.

Unusually for a neurologist he was interested in psychiatry, indeed he was fascinated by all aspects of suffering, sickness, and the human condition. Clinically he attempted to integrate biology, the clinical concepts of Hughlings Jackson and Kurt Goldstein, and psychoanalysis. He was innately inquisitive, his ideas continually evolving.

Ian McDonald, who sadly died just 9 days after Keith, freely acknowledged the profound influence Keith had upon his life and neurological career. Keith had a talent of exciting the intellect of others. Graeme Bydder considered him 'a remarkable mixture of the everyday, coupled with the most radical and original ideas'.

His clinical teaching was often focused on the doctor-patient relationship, though these ideas were not fashionable at the time. Generations of students, though uncertain of some of the content, found that once in practice these ideas became illuminated. Bruce Todd, a Christchurch GP, recalls Keith's as his most influential and memorable teacher in Dunedin, commenting that he still has the notes made of his lectures 'or should I say philosophical discussions'.

Language was his vehicle, and understanding the clinical world and the nervous system, together with everything that affected them, was his daily passion. In retirement in Dunedin and Wanaka he continued to actively work on these immense

intellectual concepts. He published little, his active mind continually pursuing new ideas and new directions. Ian McDonald, Graeme Bydder, Roberta Highton, Ed Kairis, Charlotte Paul, Sue Hallwright, John Bulow, myself, and many others (in many fields) continued, despite our other careers, to assist Keith by listening, learning, and deciphering his rich intellectual life up until his death caused by lymphoma.

He will be remembered for his ability to stimulate original thought by his imaginative and energetic teaching and mentorship. Keith is survived by his wife Barbara and his four children (Roderick, Jan, Sandy, and Jonathan).

Dr A D (Sandy) Macleod at Burwood Hospital, Christchurch wrote this obituary.

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

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

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

Osteoporosis and the osteoporosis of rheumatic diseases

Nancy E Lane and Philip N Sambrook. Published by Mosby (Elsevier), 2006.
ISBN-13: 9780323034371. Contains 289 pages. Price $235.00

There is an emerging and expanding knowledge base in osteoporosis, spread over many journals. In this monograph of 26 chapters, international authors review and discuss the literature, emphasising advances over the past 5 years.

Initial chapters cover epidemiology, pathogenesis, clinical aspects, and investigations. Middle chapters discuss therapy such as exercise, selective oestrogen receptor modulators, calcitonin, and parathyroid hormone. Lastly, osteoporosis associated with systemic inflammatory rheumatic diseases, solid organ transplantation, and glucocorticoid therapy are reviewed.

Repetition is minimal despite multiplicity of authors and chapters—which (when present) serves to remind that identification and management of fracture risk factors involve more than performing bone densitometry and prescribing alendronate. The discussions are generally and necessarily detailed, reflecting study results that are often preliminary or allow no dogmatic conclusions.

Nonspecialist readers may lose their way among the mass of information on offer, which includes reviews of cellular and molecular mechanisms. Nevertheless, busy clinicians can find practical pointers in the chapters on case finding, bone densitometry, and bisphosphonate therapy; a useful discussion on the limitations of follow-up bone densitometry for predicting fracture risk reduction in response to therapy, is located in the chapter on biochemical markers of bone turnover.

Overall, the book provides good in-depth clinical and technical information (there is an interesting chapter on bone biomechanics). The print and layout is of good quality, with many useful figures and tables. It should prove a good reference text for any medical library.

Min Loke Wong
Consultant in Rheumatology and Rehabilitation
Waikato Hospital
Hamilton

THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1249 ISSN 1175 8716

The motoneurone and its muscle fibres

Daniel Kernell. Published by Oxford University Press, 2006. ISBN-13: 978019852655. Contains 1360 pages. Price US$98.50

The main audience for this book is likely to be researchers and postgraduate students working in the field of neuromuscular physiology. It is not directed at clinicians, although some clinically related issues are described. Others who would find considerable value in this book include students and practitioners of exercise science, physiotherapy, and medical sciences, along with individuals wishing to extend their knowledge on neuromuscular function.

The aim of the author is to provide a state-of-the-art survey of the essential information on the motoneurone, motor-unit, and muscle physiology. He indeed gives a critical account of the present understanding in the field by using experimental evidence that ranges from key historical findings to the most recent research.

A notable difference between this book and many others in neuroscience is that it continuously emphasises the interaction between motoneurone and functional muscle physiology, rather than treating them as almost separate entities. The book starts with an overview of basic properties of neurons, neuromuscular transmission, motor-units, and muscle, followed by several detailed chapters on the motoneurone and its regulation of muscle force production.

The remainder of the book deals with specific topics such as neuromuscular fatigue and potentiation; denervation and reinnervation; training, chronic stimulation and disuse adaptations; then developmental and ageing aspects of the neuromuscular system. This structure and the writing style make the book very usable for the target audience.

The author Daniel Kernell has been working on neuromuscular function for over 40 years, and has published over 80 original papers. Several years ago I was fortunate enough to meet Kernell in his laboratory in Groningen and was impressed with his expansive knowledge, passion, and willingness to discuss fatigue and many other ideas from his research. These characteristics are undoubtedly revealed in this book which should inspire many others in the field.

Simeon Cairns
Senior Lecturer
Division of Sport & Recreation, Faculty of Health & Environmental Sciences
Auckland University of Technology