THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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In this Issue of the Journal

ATV (quad bike) injuries in New Zealand children: their extent and severity
Kate Anson, Elizabeth Segedin, Peter Jones

This is the first published paper that specifically investigates quad bike injuries in NZ children (under 16 years old). Overseas studies tell us that children are more likely to be killed or injured on a quad bike than an adult. Despite New Zealand safety guidelines that state a child under 12 should never ride an adult-sized quad bike (>90cc), over half the injured children in this study were less than 12 years old; 32 were 5 years old or less. A quarter were injured as passengers. This study highlights 16 deaths, 16 intensive care admissions, and over 200 children hospitalised over a 7-year period (2000–2006). Very few children were wearing a helmet or driving a quad bike of an appropriate size for their age. We conclude that quad bikes have the potential to cause significant injury and death and that public debate is needed to determine whether legislation is needed to protect children.

Emergency Department utilisation: a natural experiment
Cecelia Rademeyer, Peter Jones, Stuart Dalziel, Garry Clearwater, Bernard Foley, Mazin Ghafel

We studied the number of presentations to Emergency Departments (EDs) in all hospitals in the Auckland region over a 5 year period, before, and after the opening of Waitakere Hospital Emergency Department. We found that opening the ED at Waitakere Hospital resulted in a large increase in presentations to the Waitemata District Health Board without reducing presentations to other Auckland Emergency Departments, which has implications for Emergency Services development and resource allocation in the future.

Time to definitive care for patients with moderate and severe traumatic brain injury—does a trauma system matter?
Ritwik Kejriwal, Ian Civil

Time to surgery is important in determining outcome for patients with severe head injuries. We analysed the timelines associated with the sequence of care for patients with head injuries and deficiencies in our trauma system. We found that a significant proportion of patients were taken to a hospital without neurosurgical service and then transferred to Auckland City Hospital, thus delaying surgery. Our recommendation includes better triaging of patients at the scene of injury.

Paramedic-administered prehospital thrombolysis is safe and reduces time to treatment
Anil M Ranchord, Sandhir Prasad, Phillip Matsis, Scott A Harding

The Kapiti Thrombolysis Programme allowed the treatment of heart attacks to be administered safely by paramedics in the ambulance on route to hospital. This lead to a 90-minute reduction in the time to the initiation of treatment that resolves the heart attack and has been shown to reduce heart failure and death. This is particularly relevant in New Zealand where many communities are located a considerable distance from hospital care

Contemporary New Zealand coefficients for the trauma injury severity score: TRISS(NZ)
Philip J Schluter, Cate M Cameron, Tamzyn M Davey, Ian Civil, Jodie Orchard, Rangi Dansey, James Hamill, Helen Naylor, Carolyn James, Jenny Dorrian, Grant Christey, Cliff Pollard, Rod J McClure

Currently, New Zealand Trauma Registries use historical and non-New Zealand information to ascertain a patient's probability of survival when admitted to hospital suffering from a traumatic injury. This study collected and used contemporary New Zealand information to determine whether these probabilities could be improved upon. For both the original and the new New Zealand data, the probability of survival was accurately predicted. However, contemporary New Zealand coefficients were statistically superior to original coefficients. A strong case exists for replacing these original coefficients in the New Zealand Registries with these updated estimates.

Using trauma injury severity score (TRISS) variables to predict length of hospital stay following trauma in New Zealand
Philip J Schluter, Cate M Cameron, Tamzyn M Davey, Ian Civil, Jodie Orchard, Rangi Dansey, James Hamill, Helen Naylor, Carolyn James, Jenny Dorrian, Grant Christey, Cliff Pollard, Rod J McClure

Improvements in trauma care over the last few decades have been responsible for substantial improvements in survival rates following major trauma, but little is known about the extent that improvements in trauma care have lead to decreased injury-related morbidity and improved long-term outcomes. Attempts to develop a "threat to morbidity" injury severity score have focused on modelling length of stay (LOS) in hospital, a commonly adopted proxy measure of morbidity obtainable from routine hospital data. Using the trauma injury severity score (TRISS), a score globally applied to predict injury survival, this study aimed to develop and assess the capabilities of sophisticated statistical model that predicts LOS in survivors of traumatic injury. None of the TRISS models considered had sufficient ability to accurately or reliably predict LOS. Additional predictor variables for LOS and other indicators for morbidity need to be considered.

Sodium fluoroacetate (1080): assessment of occupational exposures and selection of a provisional biological exposure index

Michael Beasley, Penny Fisher, Cheryl O'Connor, Charles Eason

In animal studies, longer-term daily administration (of certain doses) of 1080 (a poison commonly used for poisoning pest possums in New Zealand) has been linked to adverse effects on the heart, the fetus, and fertility in males. Bodily uptake of 1080 has been demonstrated in some formulators and distributors of 1080 baits, using blood and especially urine testing. "Biological monitoring" (employing urine testing protocols) has been used as a tool to help identify some specific workplace roles and activities contributing to measurable 1080 exposures, and is also useful for assessing the effectiveness of introduced control (hazard minimisation) measures. However despite derivation of a provisional "biological exposure index" (BEI); (that is, a probably safe and hence provisionally "acceptable" level of 1080 in urine samples), there remains uncertainty about the degree of human risk as a function of urinary levels (indeed the provisional BEI had to be derived from animal studies; there being as yet no relevant human studies to shed light on risks from low level chronic exposure). These uncertainties provide reason for investigation of the health status of these workers along with ongoing review of exposure control measures backed up by periodic urine testing to help assess their effectiveness.

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Children and ATVs (quad bikes) don't mix

Michael Shepherd

In the article by Anson and colleagues[1] in this issue of the *NZMJ* we are further reminded of the risk of injury and death associated with children using all-terrain vehicles (ATVs, quad bikes, 4-wheel motor bikes, farm bikes). ATV injury in New Zealand has been described previously, but Anson and colleagues have completed a comprehensive review of these injuries in children. Their findings are consistent with international literature on paediatric ATV injuries: serious head, chest, and limb injuries result from children using ATVs as drivers or passengers. International studies clearly show that children on ATVs have a much greater risk of injury and death than adults on ATVs.

There are some simple interventions available that can significantly reduce the risk of ATVs to children. These include restricting the use of adult-sized ATVs to adults, not allowing passengers on ATVs, and the use of helmets on ATVs. The next challenge is to translate this clearly demonstrated risk into a change in ATV use, therefore reducing childhood ATV injury.

Many of these injuries occur in the rural community where independence is highly valued, where childcare is difficult to obtain and where families are involved in farm work together. Basham and colleagues have demonstrated that awareness of this injury risk is unlikely to be sufficient to alter behaviour among many rural families.[2] They propose a series of interventions that may result in a change in ATV use among rural families including; incentives to complete ATV training, promotion of ATV helmets, mechanical changes to ATVs, exploration of rollover protection systems, improving access to childcare, and development of alternative transport options for children on farms.

It seems likely that legislation will be required as part of a strategy to restrict the use of adult-sized ATVs to adults and to prohibit the carrying of passengers. International consensus statements support such legislation.[3–5] While legislation may initially be regarded as a restriction on essential rural activities, it should be viewed in the context of other legislation that guides children's activities. Children less than 14 years of age cannot be left unsupervised and children less than 16 years of age cannot acquire a gun licence. While there may be individual children who are capable of carrying out these activities safely at younger ages, this type of legislation is aimed at the safety of all children (based on the abilities of the average child at a given age).

This study also demonstrates the poor data quality available to researchers in the injury prevention field in New Zealand. An evidence-based approach to NZ's high rate of unintentional injury and death requires the development of a national trauma database and access to an improved quality of data through ACC.

Anson and colleagues have completed a well designed and informative study. The appropriate response to this study should be to ensure that this study does not need to be repeated. Firstly we should aim for the collection of prospective data relating to significant injury. Secondly, in partnership with the rural community, we should aim for a robust programme of information, inducement, innovation, and legislation that makes child ATV injury uncommon.

Competing interests: None known.

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What do we do about increasing demand for Emergency Department care?

Mike Ardagh

In the movie *Field of Dreams* Kevin Costner heard a voice which told him; 'if you build it, they will come.' He did, and they did. If the good folk at Waitemata District Health Board (DHB) had suffered the same hallucination they might have reconsidered building a new Emergency Department (ED) at Waitakere Hospital.

In this issue of the *New Zealand Medical Journal*, Rademeyer and colleagues describe the effect of a new ED on the utilisation of other ED services in the area.[1] Part of the rationale for a new ED at Waitakere Hospital was to reduce the demand on other EDs, particularly the ED at North Shore Hospital (both North Shore Hospital and Waitakere Hospital are part of Waitemata DHB).

Rademeyer and colleagues show that attendance at other EDs did not decrease, but instead the increment of new patients increased the total ED presentations for Waitemata DHB by 74%. While more dramatic, this is consistent with the national trend of demand for ED services growing faster than the population.[2]

Why?

The authors suggest that the 'new' ED patients may be due to redistribution from Primary Care, realisation of a genuine unmet need for secondary care, or some combination of these two explanations. Increasing demand for ED care generally is likely to have contributions from an increased burden of illness and injury, greater expectation that illness and injury will be addressed, greater opportunity for modern medicine to address illness and injury, and a reduced willingness to tolerate financial and other inconveniences when seeking care for illness and injury. However, although we might speculate, and we might support speculation by turning data until it catches an appealing light, the exact contributions to climbing acute demand are unclear.

Does it matter?

Since the start of this financial year New Zealand DHBs have been required to aim for a new heath target; Shorter Stays in EDs (the 6 hour target), defined as; '95% of patients will be admitted, discharged or transferred from an ED within 6 hours.' The target focuses attention on pathways for acute care, deficiencies of which manifest as ED overcrowding. ED overcrowding does matter, with good evidence that it causes a number of harms, the most concerning is an association with increased mortality.[3,4] Patients who travel through an ED when it is overcrowded have approximately one third greater 10 day mortality than those who travel through an ED when it is not overcrowded.[3]

The contributors to ED overcrowding can be considered across three areas; pre-load (the number and complexity of patients attending); contractility (the ability of the ED to manage patients) and; after-load (the ability to move patients to the next phase of care—particularly hospital in-patient beds). Although there will be contributors in all

three areas, the DHBs with the greatest overcrowding, and the poorest performance against the new health target, are mostly crowded with patients on stretchers waiting for admission to hospital beds. Of course increasing 'pre-load' matters, but the most pressing problems causing ED overcrowding (and prolonged ED stays) are in the hospital. While attending to increasing acute care demand is important, particularly for long term improvements in acute care, we must ensure efforts in this area do not distract us from more important problems further along the patient journey.

What can we do about it?

King Cnut made a regal stand against an advancing tide, to prove that even the mightiest of men cannot match the forces of nature. It is possible that attempting to turn the tide of increasing acute demand will achieve no more, and perhaps instead we should prepare for it. This fatalism has two flaws. First, the way acute demand is increasing, if left unchecked it is likely to overwhelm us. Second, our efforts to date haven't been enough to conclude they won't work.

Efforts to progress towards the 6-hour target will be tailored to the issues of most relevance to each DHB, but must include a concerted, coordinated, range of activities prioritised according to their potential to improve acute care. Solutions aimed at improving patient flow in hospital are likely to be prioritised initially because of the potential for greater and quicker gains. However, efforts to reduce acute demand should be concurrent or, at least, follow close behind.

The components of these efforts are likely to include a closer working relationship between Primary and Secondary Care, better chronic disease management and alternative pathways for acute care. However, they are unlikely to include obstructing access to ED care. King Cnut attempted to be a barrier to the advancing tide. Instead, he might have demonstrated that the tide could be redirected with some simple engineering. Similarly, part of the solution to increasing demand for ED care is the lowering of barriers to preventative and alternative care, rather than raising barriers to ED care.

Competing interests: None known.

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ATV (quad bike) injuries in New Zealand children: their extent and severity

Kate Anson, Elizabeth Segedin, Peter Jones

Abstract

Aims *Primary*: To ascertain how many New Zealand (NZ) children are being injured or killed as the result of all-terrain vehicle (ATV) injuries and to define the nature and severity of their injuries. *Secondary*: to examine the effect of age, weight, helmet use, and ATV size on injury severity and to compare the demographics of injury in NZ to other countries.

Methods A retrospective review was undertaken of 643 cases of children less than 16 years old hospitalised between 2000–2006 due to possible ATV-related injury. New Zealand Health Information Statistics (NZHIS) identified the cases through discharge information, supplemented by a search of Auckland's Paediatric Intensive Care trauma database. Only confirmed ATV injuries were included.

Results: Records were unavailable for 150 cases (26%). There were 218 confirmed cases of ATV injury. Mechanisms of injury were: a fall from the ATV, 105 cases (48%), a collision, 59 cases (31%), rolling 31 cases (14%). Mean age was 9.9 years (SD 3.9) with 133 (61%) under 12 years, and 32 (15%) 5 years and under. The child was the driver in 116 cases (53%) and the passenger in 61 cases (28%). Male to female ratio was 2:1. Mean injury severity score was 7.9 (SD 5.2). Median (IQR) length of stay was 2 days (1–4). Helmet use not stated in 62%, with only 30 cases (14%) identified as wearing helmets. The majority of injuries were orthopaedic, soft tissue injuries and head injuries. Multiple injuries occurred in 74 cases (34%). One hundred and eleven children (51%) required a general anaesthetic. Seventeen (7.8%) children required admission to intensive care. Six (2.8%) children were left with a permanent disability. Sixteen children died. There was no correlation between ISS and age or weight (Rho=-0.089, p=0.08 and Rho=0.49, p=0.79 respectively). The observed differences in ISS between helmet users and non-users, ATV drivers and passengers and size of ATV were not statistically significant. There was a trend towards reduced risk of head injury with helmet use RR =0.63 (95%CI 0.36-1.1), Chi-squared=3.09, p=0.09. The mean age of injured NZ children was lower than other countries and length of hospital stay was shorter. Gender distribution, injury type, and severity were similar to elsewhere.

Conclusions ATVs are potentially lethal and have the capacity to inflict significant harm. It is clear that it is not appropriate for a young child to ride an adult sized ATV due to the risk of serious injury and death. Public debate is needed as to whether education or legislation is the answer.

Several high profile child deaths in New Zealand (NZ) were caused by all-terrain vehicle (ATV) accidents but there is little data on the use of ATVs by NZ children or the nature and extent of the injuries caused. The few studies that do exist are

hampered by inability to identify data separate from general farm bikes and off-road motorbikes. They do suggest however that children are riding adult- sized ATVs. For instance, a 1993 survey of Southland farmers found over half the ATVs in use were operated by children with the permission of the owner.[1] In 2004 a safety trainer informally polled students at safety days that he held at rural schools. He found from 12 rural schools with an average roll of 50 children at each, only 5 or 6 children had not ridden an ATV.[2]

The primary objective of this study is to determine the nature and severity of ATV injuries in NZ children and secondarily, the compare injury severity scores in: helmet users and non-users, ATV drivers and passengers, riders of adult and child-sized ATVs. The effect of age and weight on injury severity will be examined, as will injury demographics and patterns in other countries.

Death by injury is the leading cause of death of children in developed countries. NZ children find themselves close to the bottom of a league table of rich countries for child death by injury—22nd out of 26 countries. Indeed, NZ children have an annual mortality rate of 13.7 per 100,000 compared to 9.5 per 100,000 in Australia and 6.1 per 100,000 in the UK. [3] It is not known to what extent ATVs contribute to death and injury in NZ children.

Overseas data show that children are more likely to be killed and injured than adults riding ATVs[4,5] and are also likely to be more severely injured.[6,7] As more ATVs are sold and ATVs become larger and more , the number of children and adults being injured or killed in the US continues to rise.[4] Drivers under 16 years old are 2.5 times more likely to be injured on ATVs than drivers 16–34 and 4.5 times more likely to be injured than drivers aged 35–54.[5] Thirty percent of deaths associated with ATV injuries occurred in those under 16 years old.[4]

Injuries sustained riding an ATV are often multiple and serious.[6,8–16] Injuries sustained riding an ATV are similar to or more severe than those due to motorbike accidents (on or off road) and car crashes and are more severe than those sustained as a result of bicycle accidents.[17–21] A child injured while riding an ATV is 6 times more likely to require hospitalisation and 12 times more likely to die than a child injured riding a bicycle.[22]

Head injuries are the most common cause of death.[6,10,15,16,23–25] The majority of studies suggest helmet use is protective against head injuries or in at least reducing the severity of the head injury.[6,16,25-27]

What is an ATV?

ATVs—also known as quad bikes or farm bikes—are motorised vehicles with 4 large, low-pressure tyres designed for off-road use. ATVs vary in size and power with engine sizes ranging from 50cc–700cc, and weigh up to 500kg with most 4-wheel ATVs being in the 240–280kg range.[28–30] Most ATVs have a solid rear axle (i.e. no rear differential), a motorbike style seat that is straddled, and motorbike style controls on the handlebars. An adult-sized ATV is one with an engine size of greater than 90cc. Three-wheeled ATVs are no longer sold due to safety concerns but it is not known how many remain in use in NZ.

Why is it hard for children under 16 to safely control an ATV?

Although they appear easy to ride and handle, riding an ATV requires the same or greater skill judgement and experience as for driving a car. ATVs are heavy, powerful machines and many accidents occur through loss of driver control.[31]

ATVs also appear to be stable vehicles but in fact have a high centre of gravity, a short wheel base and a narrow track width which make the vehicle inherently unstable, though the 4-wheel ATV is more stable than the 3-wheel ATV.

Several size and developmental factors prevent a child from driving an ATV with the same control as an adult. ATVs demand an active riding technique, where rider movement, strength and weight shifting are required to ensure stability and control. The active riding technique is needed to be able to effectively turn the vehicle round corners due to the lack of rear differential and to prevent vehicle rollover on uneven terrain and slopes. [28,31]

Children often lack the strength or weight to effectively handle an ATV and do not have sufficient mass to act as a counterweight especially on slopes. Some have trouble reaching the controls. Most children have not yet developed the skills and judgement to safely operate or ride as a passenger on an ATV.[28]

Current New Zealand Guidelines

There is no legislation governing the off-road use of ATVs. Current guidelines produced by ACC, Occupational Health and Safety, and the Land Transport Safety Authority recommend that children under 16 years of age never operate an adult-sized ATV. [28, 31, 32]

The guidelines suggest that 12–15 year olds may operate an adult-sized ATV under strict conditions such as the following: the young person must have the size and strength to safely operate the ATV, they must be trained in the use of the ATV, they must wear a helmet and sturdy boots at all times, they never carry passengers, implements or loads, they have speed limits and "no go" areas for difficult terrain, and they are supervised to make sure they stay within their limits.

Children under 12 years should never operate an adult-sized ATV. If a child under 12 is to operate an ATV the engine size should match the child's age, as follows: 6–11 years under 70cc and 12–15 years 70–90cc. There are no vehicles considered suitable for a child under the age of 6. Nor is there any conclusive evidence to suggest that children riding smaller-sized ATVs have fewer accidents or less severe injuries than those on adult-sized ATVs.

Passengers should never be carried on an ATV. Most ATVs are not designed to carry passengers. Many victims of ATV accidents are passengers, often young children riding behind or in front of a parent. ATVs are equipped with large seats to allow the driver to shift weight to control the vehicle, not to carry other people. Passengers restrict the driver's mobility and add weight to an ATV, raising the centre of gravity, making it harder to control and more prone to tipping over.

There have been numerous calls overseas for legislation to prohibit the use of adult sized (>90cc) ATVs by those under 16 years of age.[26,29,33–35] US and Canadian-based

research shows that though ATV legislation such as restricting the use of adult-sized ATVs to those older than 16 years old or mandatory helmet use is widely ignored, states which had some form of legislation had lower rates of death and injury.[13,16,23,25,36]

Methods

Data collection—Data collected by the New Zealand Health Information Service (NZHIS) from 2000–2006 identified 643 cases of children under 16 years who were hospitalised as the result of an injury coded under V86—ATV or other motorised vehicle designed primarily for off-road use. Having obtained multiregional ethics committee approval, information was released from NZHIS, providing the national health identifier (NHI), dates of admission and discharge, ethnicity, and the district health board (DHB) of the hospital.

Copies of the relevant admissions from DHBs around the country were then requested through the clinical records department of Auckland Hospital. Only those cases that were positively identified as an injury sustained while riding a 3 or 4-wheeled ATV were included.

This was supplemented by data from Starship Hospital's Paediatric Intensive Care Unit's (PICU) database (Appendix 1). NZHIS also provided limited data on deaths due to ATV injury from 2000 to 2006.

Data was collected on demographics, weight of patient, site of accident, accident mechanism and helmet use, whether the injured child was the driver or the passenger, presence of adult supervision, ATV size and type, length of hospital stay, the need for admission to intensive care, the need for surgical intervention, and the nature of the injuries sustained. Injury severity score was calculated in the standard manner using the Abbreviated Injury Scale from the Association for the Advancement of Automotive Medicine (1998 Update).

Statistics—Descriptive statistics and charts were used to display demographic data. Means (SD), medians (IQR), and proportions (95%CI) were calculated where appropriate. The frequency distributions of quantitative variables were examined to determine Normality. Non-parametric methods (Spearman's Rank Order Correlation and Mann-Whitney U Tests) were used to analyse non-Normally distributed variables. Chi-squared or Fisher's exact tests were used where appropriate to analyse categorical data. Proportions (95%CI) were calculated using GraphPad Software California, USA and OpenEpi version 2.3. All other analysis was done using StatView© version 5.0 (SAS Institute Inc., North Carolina, USA). All analyses were 2-tailed and the significance level was <0.05.

Results

From a total of 643 discharges with a code of V86, 218 were positively identified as injuries sustained while riding a 3 or 4-wheeled ATV. Fifty-seven were multiple admissions or duplicated admissions for the same patient. Only the first admission was entered in the database. Confirmed ATV injuries made up 50% of available notes.

643 discharges coded V86

- 57 multiple admissions or duplicated admissions.
- 150 sets of notes unavailable (26%).
- 436 sets of notes reviewed.
- 218 excluded as non-ATV related.

218 confirmed ATV-related injuries

Injury demographics—Demographic details of injured children are set out in Table 1.

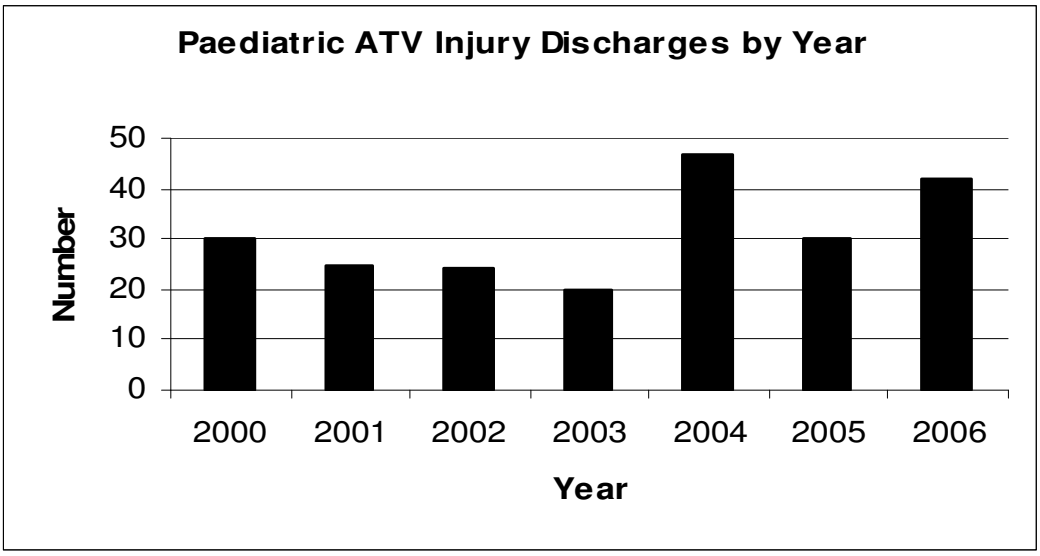
Table 1. Child, Injury and ATV demographics

Variable		Value	%	95 % CI
Age	Mean (SD)	9.8 (3.9)		
	12–15 yr	85	39.0	32.8–45.6
	5–12 yr	101	46.3	39.8–53
	≤5 yr	32	14.7	10.6–20
Weight (n=137)	Mean (SD)	35.9 (17.3)		
	≤50 kg	113	82.5	75.2-88
Gender	Boys	149	68.4	61.9–74.2
	Girls	69	31.7	25.8–38.1
Ethnicity	NZ European	142	65.4	58.6–71.2
	Māori	58	26.7	21.2–32.9
	Other European	5	2.3	0.8–5.41
	Pacific Islander	3	1.4	0–4.2
	Indian	1	0.5	0–2.8
	Not recorded	7	3.2	1.4–6.6
Length of Hospital Stay	Mean (SD)	3.1 (5.4)	Median (IQR)	2 (1–3)
Injury Severity Score	Mean (SD)	7.9 (5.2)	Median (IQR)	9 (4–9)
	< 10	170	78	72.4–83.3
	10–15	31	14.2	10.2–19.6
	>15	17	7.8	4.9–12.3
ICU admissions	PICU	6	2.8	1.1–6
	Adult ICU	11	5.1	2.7–8.9
General anaesthetic		111	50.9	44.3–57.5
Disposition	Home	216	99.1	96.5–100
	Rehabilitation centre	2	0.9%	0–3.5
	Permanent disability	7	3.2	1.4–6.6
Deaths*		16	6.8	4.2–10.9
ATV Type	4 wheels	205	94	88.9–95.9
	3 wheels	13	6	3.4–10
ATV Size **	Adult	23	54.8	39.9–68.8
(recorded n=42, 19%)	Child	19	45.2	31.2–60
ATV Use **	Driver	116	65.5	58.3–72.2
(recorded n=177, 81%)	Passenger	61	34.5	27.9–41.4
Helmet Worn **	Yes	30	36.1	26.6–46.9
(recorded n=83, 38%)	No	53	63.9	53.1–73.4
Adult supervising **	Yes	28	50.9	36.6–61.7
(recorded n=55, 25%)	No	27	49.1	35–60

ATV = All Terrain Vehicle, PICU = Paediatric Intensive Care Unit, ICU = mixed or adult Intensive Care Unit, NZ = New Zealand; SD = standard deviation, IQR = Interquartile range, kg = kilogram, * deaths expressed as % of 234 injured children, ** expressed as % of number of cases where this was documented.

There is an increase over time in the number of children discharged with confirmed ATV injuries, however, the number of unobtainable notes and the short time period make it difficult to draw any firm conclusions (Figure 1).

Figure 1. Children discharged with ATV injury by year



ATV=All Terrain Vehicle

Figure 2 shows the mechanism of injuries and figure 3 shows where the injuries occurred.

Figure 2. Mechanism of injury

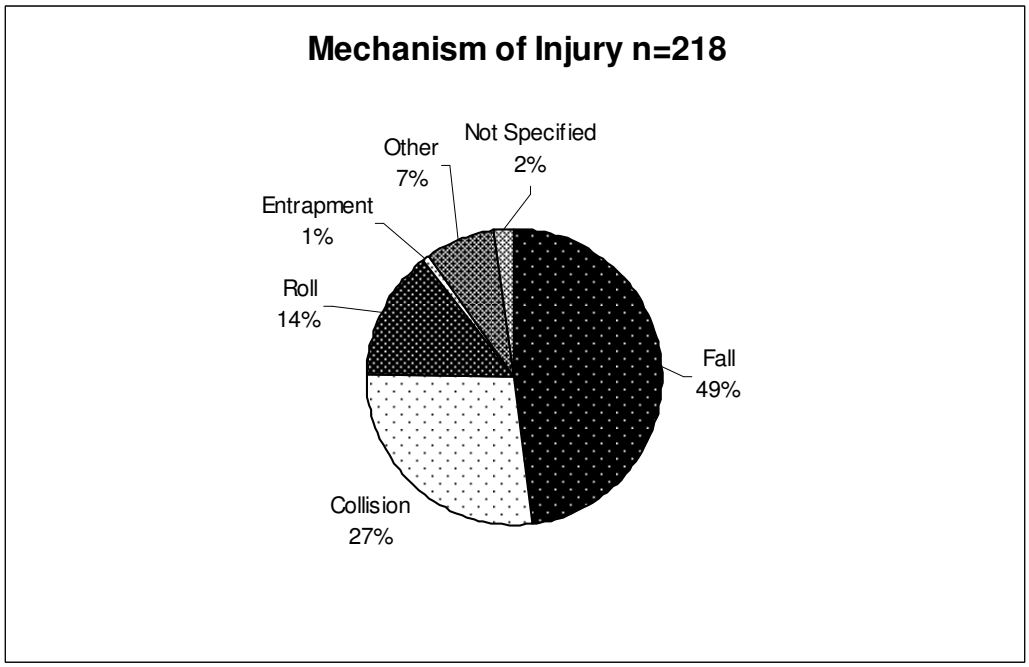
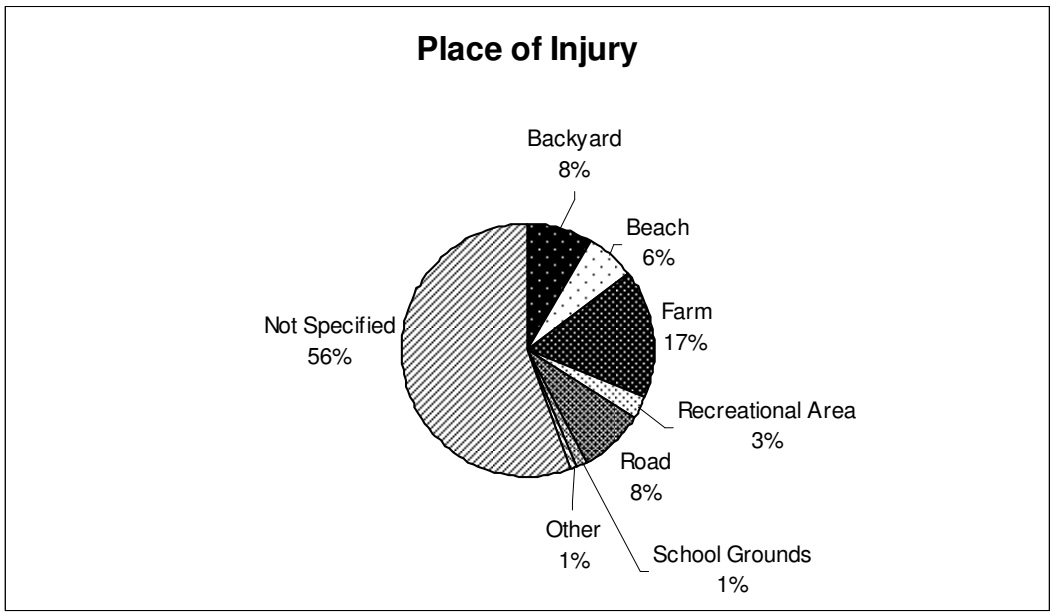


Figure 3. Places where ATV injuries occurred

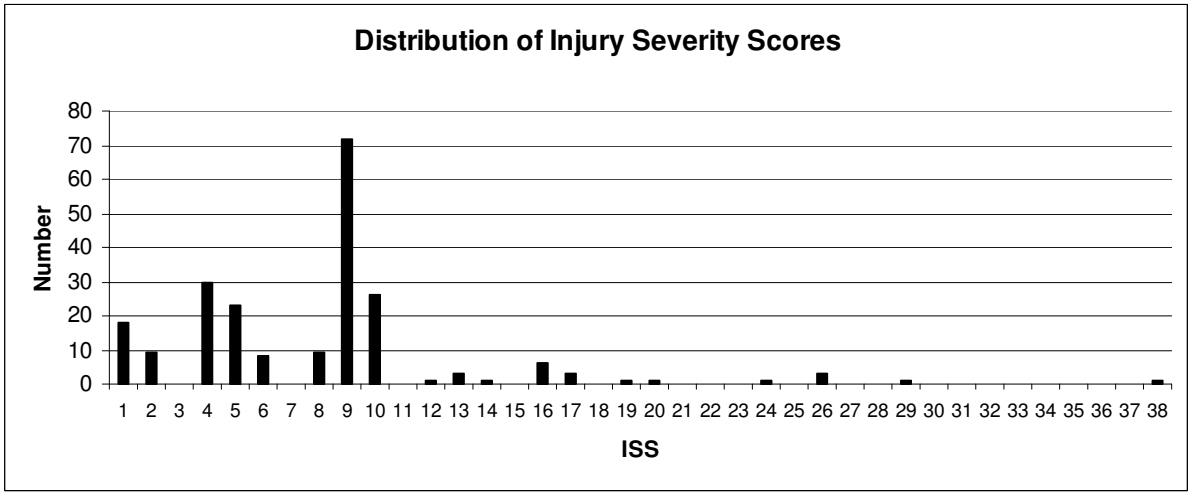


ATV=All Terrain Vehicle.

Injury pattern and severity

Figure 4 shows the range and distribution of injury severity scores.

Figure 4. Injury severity score



ISS = Injury Severity Score (sum of squares of three most severely injured body regions)

Orthopaedic, soft tissue, head, and facial injuries were the most frequent injuries. Seventy-four children (33.9%, 95%CI 28–40%) had multiple injuries involving more than one body part (Table 2).

Table 2. Injury pattern*

Injury type	Number	%	95% CI
Fractures/dislocations	126	57.7	50.7–63.7
Soft tissue	121	55.5	48.9–62
Head	61	27.9	23.3–35.3
Face	51	23.6	18.4-29.8
Thorax	7	3.2	1.4–6.6
Abdomen	17	7.8	4.9–12.2
Spine	10	4.6	2.1–7.9
Pelvic fractures	3	1.4	0–4.3

*Total > 100% due to multiple injuries

The most common orthopaedic injuries were closed fracture of the radius ± ulna and fracture of the tibia ± fibula. There were 11 compound fractures (6 of these were compound fractures of the tibia and/or fibula), 19 fractured femurs (in 2 cases bilateral fractured femurs) and a dislocated hip associated with acetabular fractures.

There was one case of a closed Lisfranc injury and another of compound fractures of the mid-foot ultimately requiring a below knee amputation. Two patients had tendon injuries of the hand and forearm requiring surgical repair.

Soft tissue injuries were frequent, comprising lacerations, contusions, abrasions and limb sprains. Fifty-six (26%, 95%CI 20–32%) sustained lacerations which required wound closure and 41 of these (73%, 95%CI 60–83%) had their wound closed under a general anaesthetic.

Some of the soft tissue injuries were severe; including 2 de-gloving injuries of the hand with partial amputation of digits, 6 deep thigh lacerations from handlebars injuries, an open pneumothorax, 11 compound fractures, and a burn from an exhaust which required skin-grafting.

Sixty-one patients sustained head injuries (28%, 95%CI 23–39%), of which 52 (85%, 95%CI 19–31%) were minor head injuries. Concussion was diagnosed in 49 (78%, 95% CI 66–86.4%). Thirty–three children were admitted primarily for neurological observation. Three had moderate and 6 had severe head injuries. There were 10 children with skull fractures. Amongst the moderate to severe head injuries were 2 extradural haemorrhages, 4 children with intracerebral contusions, 2 with intracerebral haemorrhages, 3 with deep white matter or basal ganglia petechiae, 2 with diffuse axonal injury, and one who suffered a hypoxic encephalopathy when trapped beneath the ATV for 20 minutes.

All head injuries were managed conservatively. Only one child required neurosurgical intervention to elevate and wash out a compound, depressed skull fracture. All 6 patients admitted to PICU had a head injury.

There were 51 patients with facial injuries (23%, 95%CI 18–29%). Ten patients had facial fractures, 5 of whom had other soft tissue facial injuries. 26 patients sustained facial lacerations (excluding scalp) and another 19 sustained facial contusions or abrasions. Four children required surgery for facial fractures—3 maxillary and 1

mandibular. There were 7 children with chest injuries, 5 cases with pulmonary contusions and 4 with pneumothoraces. One child had multiple rib fractures.

Abdominal injuries were predominately contusions of the abdominal wall with a small number being admitted for observation due to abdominal pain but with normal imaging with ultrasound or CT scan. There were two cases of a liver laceration and two of splenic laceration, all were managed conservatively. There were two cases of microscopic haematuria with flank pain but no renal abnormalities on imaging.

Spinal injuries were infrequent. There were eight children with cervical sprains two of which resulted in the child wearing a Philadelphia collar for 2 weeks. Two children had stable fractures of the thoracic or lumbar spines.

Secondary outcomes—There was no correlation between ISS and age or weight (Rho = -0.089, P=0.08 and Rho = 0.49, p=0.79 respectively). The observed differences in ISS between helmet users and non-users, ATV drivers and passengers and size of ATV (Table 3) were not statistically significant. There was a non-significant trend towards reduced risk of head injury with helmet use RR=0.63 (95%CI 0.36–1.1), chi-squared=3.09, p=0.09. The large proportion of missing data for helmet use and ATV Size mean these results should be interpreted with caution.

Table 3. Effect of helmet use, position on ATV, and ATV size on injury severity

Variable n=212		n	Injury Severity Score		P**
			Mean(SD)*	Median (IQR)	
Helmet Use	Yes	30	7.6 (5.0)	8 (4–10)	0.72
n=83	No	53	8.6 (6.1)	9 (5–10)	
ATV Use	Driver	116	7.8 (4.7)	9 (4–9)	0.44
n=177	Passenger	61	8.3 (6.3)	9 (5–10)	
ATV Size	Child	19	6 (3.2)	5 (4–9)	0.16
n=42	Adult	23	9.8 (7.2)	9 (4–10)	

*Although Injury Severity Score is not Normally distributed and strictly speaking an ordinal variable, means (SD) are also presented by to facilitate comparisons with other studies where it is treated as a continuous variable.
**Mann Whitney U Test. ATV = All Terrain Vehicle, SD = standard deviation, IQR = interquartile range

International comparisons—New Zealand's experience with children and ATVs is broadly similar to that in the US and Canada, where most studies have been based, with similar ISS, helmet use, proportion of males injured and injury pattern. Our study appeared to show a slightly younger age of injured children and shorter length of stay than international studies which may be due to the fact that the current study looked at all hospital admissions whereas overseas studies looked predominantly at tertiary paediatric referral centre admissions (Appendix 2).

Deaths—There was limited information regarding the deaths of 16 children (<16 years old) killed in an accident involving an ATV between 2000 and 2006. At least 3 of these children died in hospital. The cause of death is not recorded in NZHIS data, however the mechanism of the accident is known for most. Twelve of the children were NZ Europeans, two were Māori, one was "other European" and one had no ethnicity recorded (Table 4).

Table 4. Paediatric ATV-related deaths 2000–2006

Year	Age	Mechanism
2000	2	Passenger on 4W ATV with 3 adults, flipped going up steep hill
	4	Passenger on 4W ATV with loaded trailer, overturned & rolled off steep bank, trapping child
	8	Riding 4W ATV, collided with barrier around tree, head & chest injuries
2001	12	Driving 4W ATV
	14	Driving 4W ATV, overturned, trapped child under ATV, sustained cervical spine, chest, liver & splenic injuries
	11	Poorly maintained 4W ATV with overloaded trailer, rolled, trapped child under ATV
	11	Attempted to jump over top of a ridge, fell from 2m, sustained head injuries
	7	No details given
2002	14	4W ATV flipped and trapped child under it
	12	4W ATV flipped on farm track
	9	4W ATV flipped and trapped child under it
	14	4W ATV flipped and trapped child under it
	14	Drowned after falling from 4W ATV trying to cross a flooded ford
2004	12	Riding 4W ATV on farm, ATV rolled and pinned him by the neck
2005	4	Child was driving the ATV
2006	9	No details given

Discussion

This study represents the first attempt to ascertain the number and severity of childhood injuries caused by ATVs in New Zealand. A clear pattern of injury has emerged, which is similar to that reported in the international literature, with orthopaedic, soft tissue and head and facial injuries predominating.

This study highlights a number of areas of concern. The current NZ guidelines recommend that children under 12 years never ride an adult-sized ATV. In this study over half of children injured were under 12 years old with a significant number aged 5 and under.

Few children under 12 were confirmed to be riding a child-sized ATV. Guidelines also state that ATVs should never carry passengers; however one quarter of the patients in this study were injured as passengers and most of the passengers were under 12 years old. If current safety guidelines had been followed two thirds of the injuries documented in this study may not have happened.

It is noteworthy that there was a potentially clinically important difference in injury severity for child sized versus adult sized ATVs which did not reach statistical significance due to the small number of cases where ATV size was recorded. It is logical to assume that child sized ATVs would be safer as the mass of the child will therefore better match that of the vehicle. It is, however, also possible that this effect becomes less important than developmental limitations with reducing age and demands further study.

The quality of the written notes was often poor and sometimes gave no indication of mechanism of injury. Helmet use was poorly documented, even in patients presenting with head and facial injuries.

US and Canadian attempts to legislate or educate regarding safe ATV use have proved to have limited, though some, effect. In NZ, rural children in particular are encouraged to take part in life on the farm or are carried as passengers while their parents go about their work. There is an attitude of "she'll be right" and of fostering independence and self reliance in farm kids. In addition ATVs are increasingly used for recreational activities outside the farm setting when they appear no less dangerous.

Limitations of this study—This is a retrospective review that relies on accurate coding to correctly identify all ATV related injuries. It is possible that injuries due to ATVs have been missed due to incorrect discharge coding. Accurate coding of course relies on there being an adequate description of the incident in the clinical notes. This study may underestimate the number of ATV related injuries in children due to difficulties obtaining all potentially relevant clinical notes.

Extracting data from clinical notes similarly depends on their detail and accuracy. There is also the possibility of human error in the extraction and data entry process. This study only reviewed ATV injuries that resulted in hospitalisation so is likely to be an underestimate of the true burden of ATV-related injury in New Zealand's children.

Recommendations—Public debate is needed. Is the current high level of child death and injury in this country acceptable to New Zealanders? Use of ATVs by children is manifestly risky. While children and adolescents need to learn how to assess risk and gain strength and coordination by partaking in activities that provide such challenges, there are significant size and developmental realities that children face that adult operators do not. It is clear that it is not appropriate to allow a young child to ride an adult sized ATV due to the potential for serious injury and death.

Organisations such as ACC, Occupational Health and Safety and the Land Transport Authority produce widely available guidelines and instructions on safe use of an ATV. These guidelines appear a reasonable compromise on the US and Canadian positions that children under 16 years should never ride an adult sized ATV, but are clearly widely ignored. It is possible that many parents are unaware of the dangers and at the very least guidelines should be made available at the point of purchase. Is further public awareness and education the answer or is legislation required?

Enforcement particularly in a rural setting might be a challenge and may have to rely instead on prosecution after a child is injured. Legislation at least would inform and set a standard both to the point of sale and to parents and may be required to protect children from life threatening injuries.

Health care professionals that encounter children injured as a result of an ATV accident should be mindful of the potential for severe injury and assess the child thoroughly. Much improvement in the detail of clinical notes particularly injury mechanism and helmet use is required. Health care professionals are in a unique position to advocate for child safety with regard to ATV injured children and need to embrace this responsibility.

International experience shows a marked reduction in head injury with helmet use. In NZ a specific helmet has been designed for ATV use by adults. We recommend all children on an ATV wear a full face motorbike style helmet, given the high rate of head and facial injuries.

In conclusion, ATVs are potentially lethal and have the capacity to inflict significant harm. It is clear that it is not appropriate for a young child to ride an adult-sized ATV due to the risk of serious injury and death.

Public debate is needed as to whether education or legislation is the answer.

Competing interests: None known.

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See following Appendices

Appendix 1. Children admitted to PICU with an ATV injury: 1997–2008

Year	Age & gender	Mechanism	Helmet	Injuries	Intervention	Outcome
1997	10 male	Passenger on 4W ATV driven by cousin into a ditch, fell off, hitting head on roll bar	no	Initial GCS 14, skull # with CSF leak, orbital and nasal #, traumatic maculopathy left eye, eyelid laceration, facial contusions	? sutures, observation, later left eye vitrectomy, MUA nasal #	3 days in hospital, < 24 hrs in PI Discharged home, uncertain if permanently decreased acuity le
1998	12 male	Driver of 4W ATV, hit fence in school grounds	no	Initial GCS 8, skull#, large subdural haemorrhage, with smaller extradural component + midline shift, bifrontal cerebral contusions, forehead and scalp contusions	Ventilated, craniectomy for evacuation of haematoma & ICP monitor insertion, later second craniectomy for repeat evacuation haematoma & resection contused temporal lobe	15 days in hospital, 6 in PICU, discharged to rehab unit, decreas higher functions, partial left homonymous hemianopia
2000	9 male	Driver 4W ATV, hit pole, found unconscious with ATV on top of him	no	Initial GCS 6, right intracerebral haemorrhage, bilateral closed femur #s, hip contusions	Ventilated, head injury managed medically, ORIF left femur, EXFIX right femur	10 days in hospital, 4 days in PI discharged to rehab in regional hospital, higher cognitive functi defects, left sided weakness, sen changes & neglect
2002	3 male	Unspecified if driver or passenger, fell off back 4W ATV, back wheels ran over his legs, head hit road, low speed	no	Initial GCS 6, diffuse axonal injury, petechial haemorrhages in basal ganglia and left temporal lobe, 3rd nerve palsy	Ventilated, head injury managed medically, tracheostomy, later required laryngeal reconstruction for severe sub-glottic stenosis, later had eye surgery to correct squint	35 days in hospital, 13 days in P discharged to rehab centre, gradu resolving bilateral motor deficits dystonia
2004	11 male	Driver 4W ATV on gravel road, 30kph, served to avoid car, ATV went into ditch, trapped under ATV for 20 mins	yes	Initial GCS 4, small frontoparietal contusions, hypoxic encephalopathy, unilateral pulmonary contusions, multiple abrasions & contusions, scalp laceration	Ventilated, head injury managed medically, scalp laceration sutured	5 days in hospital, 2 days in PIC discharged home with mild left s weakness and higher cognitive function deficits which resolved the next 12 months
2004	14	Passenger on 4W ATV, crossed road and hit by	no	Initial GCS 10, left base of skull #, small cerebral contusions, pneumocephalus, scalp	Ventilated, head injury managed medically	5 days in Auckland hospital, 1 d PICU, Discharged to regional

	Male	car at approx 80kph		contusions, microscopic haematuria but normal CT kidneys, closed # R 2nd metacarpal		hospital, unknown if any perman deficit
2004	6 Male	Passenger on 4W ATV, hit by 4WD vehicle from behind	no	Initial GCS 3, left frontal contusion & intracerebral haemorrhage, lacerations to forehead and scalp, left femur #	Ventilated, head injury managed medically, ORIF & hip spika L femur, lacerations sutured	18 days in Auckland Hospital, 1 in PICU, discharged to re hospital, no permanent deficits
2006	2 Male	Driver on child-sized (50cc) 4W ATV, briefly unsupervised, found unconscious under ATV in backyard	yes	Initial GCS 6, CT head normal, abrasions to chest & abdomen	Ventilated, head injury managed medically	6 days in Auckland Hospital, < in PICU, discharged to re hospital, no reports of any long sequalae
2007	4 Male	Driver of 4W ATV, unwitnessed fall from ATV	yes	GCS 15, presented in respiratory distress, ruptured right main bronchus, unilateral pulmonary contusions, stable #s C6-T3, multiple abrasions and contusions	RSI & right chest drain in ED, continuous air leak, taken to theatre for thoracotomy & repair ruptured right main bronchus, spinal #s managed conservatively	6 days in Auckland Hospital, < in PICU, full recovery
2007	14 Male	Driver of 4W ATV, rolled 6m down embankment	N/S	Initial GCS 14, #/dislocation T9 on T10 with complete paraplegia from level T8, unilateral pulmonary contusion, pleural effusion	Ventilated <24hrs, spinal cord decompression, spinal fusion T7-12, chest drain	26 days in Auckland Hospital, 1 in PICU, permanent T8 parap discharged to spinal unit
2008	6 Male	Driver of child-sized 3W ATV, rode into tree	no	GCS 7, depressed skull # extending into L orbit, haemorrhagic contusions L frontal lobe, intraventricular haemorrhage, traumatic neuropathy L optic nerve, massive scalp laceration with avulsion temporalis muscle	Ventilated, craniectomy, elevation & debridement skull#, ICP monitor, medical management head injury	? stay in Auckland Hospital, 10 in PICU, developed di insipidus, discharged to rehab c R sided hemiparesis, dec cognitive function

Appendix 2. International comparisons

Year published & author	Years studied	Group studied	Mean age	Mean ISS	Mean length of stay	Male	Wearing helmet	ICU	Deaths	Injury pattern
2007 Kirkpatrick10	2001-2007	73 children < 16 yrs, Level I trauma centre, US	9.9	10.3	n/s	n/s	n/s	n/s	4 due to head injuries	45% head 29% upper limb 21% lower limb 27% face 15% abdomen 14% chest 8% pelvis 5% spine
2007 Kute27	1995-2005	238 children <16 yrs, Level I Paediatric Trauma Centre, US	11.4 ± 3.6	7.3 ±5.6	4.3 ± 4.0	70%	16%	18%	none	32% lower limb 25% upper limb 25% skull or face # 27% soft tissue 18% closed head injury 13% abdomen
2006 Prigozen12	2001-2004	26 children with craniofacial injuries (children not defined)	13.1	n/s	4.6 ± 5	65%	8%	36%	1	77% skull or face # 65% soft tissue facial injuries 35% closed head injuri
2006 Yanchar20	1993-2002	130 children <16 yrs at tertiary paediatric centre, Canada	n/s	n/s	8.3 (SD 2.5-14.2)	n/s	n/s	31%		48% fractures/dislocati 40% cuts & bruises 18% deep soft tissue in 10% head injury 4% internal organ injur 1% facial injury
2006 Gittleman36	1995-2001	285 children <16 yrs from 7 US paediatric trauma centres	11.1	9.2	n/s	76%	28%	n/s	2	57% multiple injuries 31% fractures 23% head injury 22% soft tissue injury
2006	2001-2004	50 children, aged 3-	Median 13	n/s	6 (range 1-47)	50%	16%	14%		54% head injury

Su13		17 years, Children's Hospital, Canada								28% orthopaedic injury 14% facial # 4% abdomen
2006 Alawi37	1998-2003	17 children aged 8-17 yrs **with ISS>12**,children's hospital, Canada	13.7	22.8	9.7	82%	65%	n/s	none	76% fractures 47% splenic laceration 35% head injury
2005 Kelleher38	1993-2002	184 children (not defined) at paediatric hospital, US	11.5 ± 3.9	10 ± 8.7	5 ± 12 Median 2	71%	35%	19%	3	68% multiple injuries 50% required surgery 51% orthopaedic injury 50% soft tissue 37% craniofacial 23% thorax/abdomen
2004 Murphy16	1990-2002	92 children < 16 yrs, tertiary paediatric trauma centre, Canada	12.1	7 ± 6.6 (range 1-35)	7.5 ± 14.8 Median 3	79%	40%	18%	2	45% multiple injuries 72% orthopaedic 29% soft tissue 24% head & face 13% chest & abdomen
2001 Cvijanovich14	1992-1996	130 children < 16 yrs, US	11.2 ± 3.6	8.0 ± 6.0	Median 2 Range 0-43	73%	n/s	n/s	1	60% orthopaedic 60% soft tissue 22% head 5% abdomen 3% chest
1998 Lister39	1991-1995	218 children aged 2-16 yrs	12.4	8.76 ± 6.0	4.3 ± 5.2 Range 0-29	75%	12%	19%	4	38% required surgery 53% orthopaedic 40% head 35% face 25% abdomen 20% chest 8% spine

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Emergency Department utilisation: a natural experiment

Cecelia Rademeyer, Peter Jones, Stuart Dalziel, Garry Clearwater, Bernard Foley, Mazin Ghafel

Abstract

Aim In February 2005 a new Emergency Department (ED) was opened at Waitakere Hospital in West Auckland, New Zealand. Part of the rationale for this was the expectation that it would reduce attendances to the four established EDs in the Auckland region. This study was undertaken to determine whether this happened.

Method A retrospective analysis of ED presentations to Auckland City, Starship and North Shore hospitals for the 2 years prior to the opening of Waitakere ED (February 2005) was conducted. This was compared with the attendances to all hospitals in the 2 years following the opening of the new ED. The effect of the opening of Waitakere ED on ED presentations to other hospitals was assessed using control charts. Presentations to Middlemore Hospital during the same time period were used as a control.

Results ED attendance to hospitals in the Auckland District Health Board (DHB) area increased by 9% over the study period (Auckland Hospital = 13%, Starship Children's Hospital = 2%), similarly ED attendance to Middlemore Hospital increased by 6%, consistent with population growth. However ED attendance to hospitals in the Waitemata DHB area (North Shore and Waitakere Hospitals) increased by 74%, disproportionate to population growth (8%).

Conclusion The opening of a new ED may have contributed to an increase in total ED presentations seen within the region overall, with no corresponding reduction in attendances at neighbouring hospitals.

Overcrowding in Emergency Departments (EDs) has become an international phenomenon[1–5] and has been hotly debated in the medical literature. Overcrowding has been associated with increased morbidity and mortality, raising serious concerns about quality of care.[6–9] This problem has also attracted media attention in New Zealand, especially for North Shore Hospital, with newspaper reports of long waits for patients in corridors.[10–14]

Causes identified internationally include a reduction in the number of EDs and available ED beds, as well as an increase in patient visits.[15,16] Attempts to reduce overcrowding have included a number of strategies from central government funding incentives, addressing assessment and treatment times, to increasing ED beds. Despite increase ED bed numbers being suggested as an answer to this problem, little research has focused on the effect of increasing the number of available ED beds within a geographic region on ED utilisation.

In February 2005 a new hospital-based ED was commissioned at Waitakere Hospital (WH) in Henderson, West Auckland. Part of the rationale for opening this new

facility was the expectation that it would reduce attendances, and thus overcrowding, in neighbouring EDs.

As the regional population is geographically well defined, the opening of the new facility presented an opportunity to study the impact that a new ED had on patient presentations to neighboring EDs.

Background

Auckland City is located in the top third of the North Island of New Zealand and is bordered by two natural harbours, the Waitemata and the Manukau. These harbours divide the city into northern and southern parts, with only two highways and one rail link connecting them across a narrow isthmus, 800m wide (Figure 1).

Figure 1. Auckland geography: the Auckland isthmus and hospital locations

NSH=North Shore Hospital, ACH=Auckland City Hospital, SSH=Starship Children's Hospital, WH=Waitemata Hospital, MMH=Middlemore Hospital. Black Arrow=Isthmus.

The area had a population of 1,231,500 in the 2001 Census, and 1,387,780 during the 2006 Census.[17] At the northern and southern boundaries of the Greater Auckland area the population density is low and divided from the neighbouring areas (Northland and Waikato respectively) by natural geographic barriers (Figure 2).

Figure 2. Auckland geography: District Health Boards (DHBs)

Source: Sector Accountability & Funding Directorate, Ministry of Health.

Health services within Auckland are provided by three District Health Boards (DHBs): Auckland District Health Board (ADHB) and Waitemata District Health Board (WDHB) which respectively serve the central and northern part of the city, and Counties Manukau District Health Board (CMDHB) which serves the southern part (Table 1).

Table 1. Hospital-based services within the Greater Auckland region

DHB	Hospital (location)	Services	Presentations 2007
ADHB	Auckland City Starship Children's (Auckland City)	Tertiary teaching hospitals Designated trauma centres All except Plastic and Maxillo-facial surgery	85,000
CMDHB	Middlemore Hospital (South Auckland)	Tertiary teaching hospital Designated trauma centre All except Neurosurgery, Cardiothoracic surgery	76,500
WDHB	North Shore Hospital (North East of the city) Waitakere Hospital (West Auckland)	District General Hospitals Adults and children, no paediatric inpatient service	75,400

Prior to February 2005, the population north of Auckland City was served by one ED at North Shore Hospital (NSH). In response to projected population growth and overcrowding at NSH ED a new ED was commissioned at Waitakere Hospital (WH). It was postulated prior to the opening of WH ED that this new ED would result in a decrease in presentations at NSH and ADHB hospitals (Auckland City Hospital (ACH) and Starship Children's Hospital (SSH)).

Ambulance patients from West Auckland would now be taken directly to WH. Self-presenting patients were expected to present to WH rather than NSH or ACH/SSH as this was closer. This study was designed to test that hypothesis.

As Middlemore Hospital (MMH) in the south of the region is geographically separated from the other hospitals (Figure 2) the opening of the new ED was not expected to affect presentations to MMH ED. Furthermore, because of its location at the northern boundary of its catchment area, CMDHB patients usually present to the MMH ED rather than others north of the narrow isthmus. MMH therefore served as a natural control for the study.

Methods

Study design—A retrospective analysis of all ED attendances obtained from the electronic databases of the EDs within the Auckland region was undertaken over a 49-month period from February 2003 to February 2007. Clerical staff record patient attendance electronically at the time of first presentation, to be used for funding and audit of hospital performance. The data is believed to be accurate.

The study is an observation of an intervention (at time point N), the opening of the new WH ED. Data obtained included all ED attendances to the four existing EDs for two 12-month periods (Year N-1 and N-2) prior to the opening of WH ED (N), and to all five hospitals for two 12-month periods (N+1 and N+2) after its opening. Data are presented by hospital and by DHB. See below.

Year N-2	Feb 2003 – Jan 2004
Year N-1	Feb 2004 – Jan 2005
N	Feb 2005 – (excluded)
Year N+1	Mar 2005 – Feb 2006
Year N+2	Mar 2006 – Feb 2007

The month of February 2005 was excluded from analysis *a priori* as this was the month the new WH ED opened.

Statistics—The effect of the opening of WH ED on ED presentations was explored using control charts (also known as Shewhart charts or 'process-behaviour charts').[18] The control charts were generated using SPSS v14 software (SPPS Inc, Chicago, USA). A control chart is a tool used to study how a process changes over time. It helps distinguish between variation in a process resulting from common causes (i.e. natural/non-significant variation) and variation resulting from special causes (significant variation).

A special cause is anything which leads to an observation beyond a control limit. A control chart presents a graphic display of process stability or instability over time. Data are plotted in time order. A control chart has a central line for the mean, an upper line for the upper control limit and a lower line for the lower control limit. The control limits are commonly set at three standard deviations (SD) from the mean (which corresponds to a false alarm rate of 0.27%). By comparing the plotted data to the mean and control lines conclusions can be drawn about whether the variation in data, and hence the process, is within predictable limits or is unpredictable, in other words affected by special causes of variation.

Special causes can be identified by the following signs:

- Eight or more points in a row on one side of the mean line.
- One or more data points falling outside the control limits.
- Six or more points in a row steadily increasing or decreasing.

Control charts can also be used to see if an event occurring at a given time point has a significant impact on the process. For our study data ED attendance (the process) was analysed as monthly data points with control lines determined at three standard

deviations from the mean. Eight or more data points in a row on one side of the mean line were considered to be significant.

Population estimates for each DHB (as expected in June of each year) for the years 1996 to 2010 were obtained from Statistics New Zealand[19] to see if there were any major population shifts in individual DHB populations that might explain changes in ED presentation numbers to the DHBs.

Results

The population in the region increased a total of 9.4% over the study period (Table 2).

Table 2. Population per District Health Board

Year	ADHB	WDHB	CMDHB	Total
2003*	413,000	474,700	416,500	1,304,200
2004*	420,500 (+1.7%)	484,700 (+1.7%)	426,800 (+1.9%)	1,332,000 (+1.8%)
2005*	427,800 (+0.1%)	492,700 (+2.4%)	435,000 (+4.6%)	1,355,500 (+2.4%)
2006	428,280 (+1.7%)	504,710 (+1.6%)	454,790 (+2.0%)	1,387,780 (+1.7%)
2007*	435,420 (+1.8%)	512,670 (+1.6%)	463,760 (+2.1%)	1,411,850 (+1.8%)

Source: Statistics New Zealand[19]. *Data for 2003, 2004, 2005 and 2007 is estimated. ADHB=Auckland District Health Board, WDHB=Waitemata District Health Board, CMDHB=Counties Manukau District Health Board.

Yearly presentations to each hospital and DHB over the study period are shown in Tables 3 and 4. There is a 74% increase in presentations to WDHB, with only minor increases in the other DHBs. This increase is mainly due to presentations to the new ED at WH.

Table 3. Presentations per study hospital over the study period

Year	ACH	SSH	NSH	WH	MMH
N-2	50,661	27,595	43,274		72,426
N-1	55,251 (+9.1%)	28,898 (+4.7%)	45,068 (+4.2%)		72,982 (+0.8%)
N+1	56,199 (+1.7%)	28,153 (-2.6%)	44,150 (-2.0%)	21,324	74,740 (+2.4%)
N+2	57,338 (+2.0%)	28,244 (+0.3%)	47,277 (+7.1%)	28,121 (+31.9%)	76,572 (+2.5%)

ACH=Auckland City Hospital, SSH=Starship Hospital, NSH=North Shore Hospital, WH=Waitakere Hospital, MMH=Middlemore Hospital.

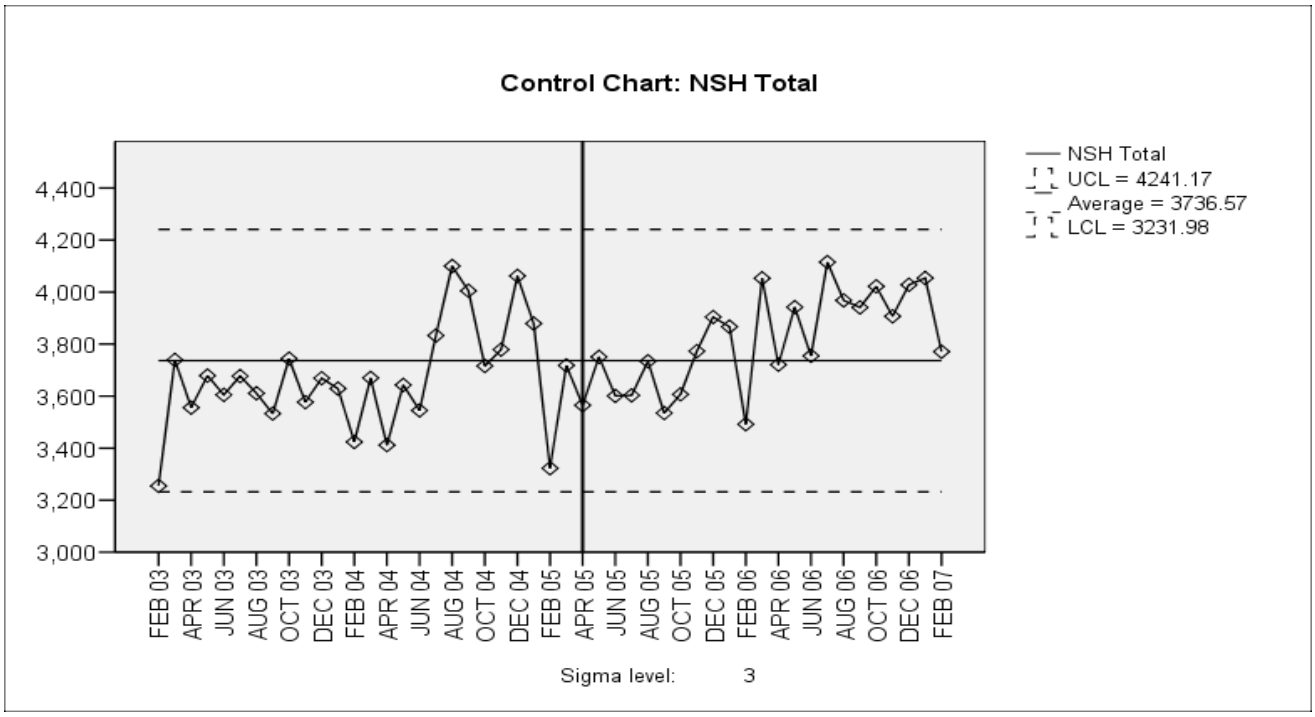
Table 4. Presentations per DHB over study period

Year	ADHB	WDHB	CMDHB	Total
N-2	78,256	43,274	72,426	193,956
N-1	84,149(+7.5%)	45,068(+4.1%)	72,982 (+0.8%)	202,199(+4.2%)
N+1	84,352(+0.2%)	65,474(+45.3%)	74,740(+2.4%)	224,566(+11.1%)
N+2	85,582(+1.4%)	75,398(+15.2%)	76,572(+2.5%)	237,552(+5.8%)

ADHB=Auckland District Health Board, WDHB=Waitemata District Health Board, CMDHB=Counties Manukau District Health Board.

The total population of the three DHBs is similar across the region (Table 2). Although WDHB served the largest population, it received only 22% of regional ED presentations in the first two study years (Years N-2 and N-1; (per capita ED attendance rate=0.09). However by the end of the study period ED presentations were more evenly distributed and a closer match to the population distribution, with per capita ED attendance rates of 0.20 at ADHB, 0.15 at WDHB and 0.17 at CMDHB.

Figure 3 is the control chart for ED presentations to NSH over time

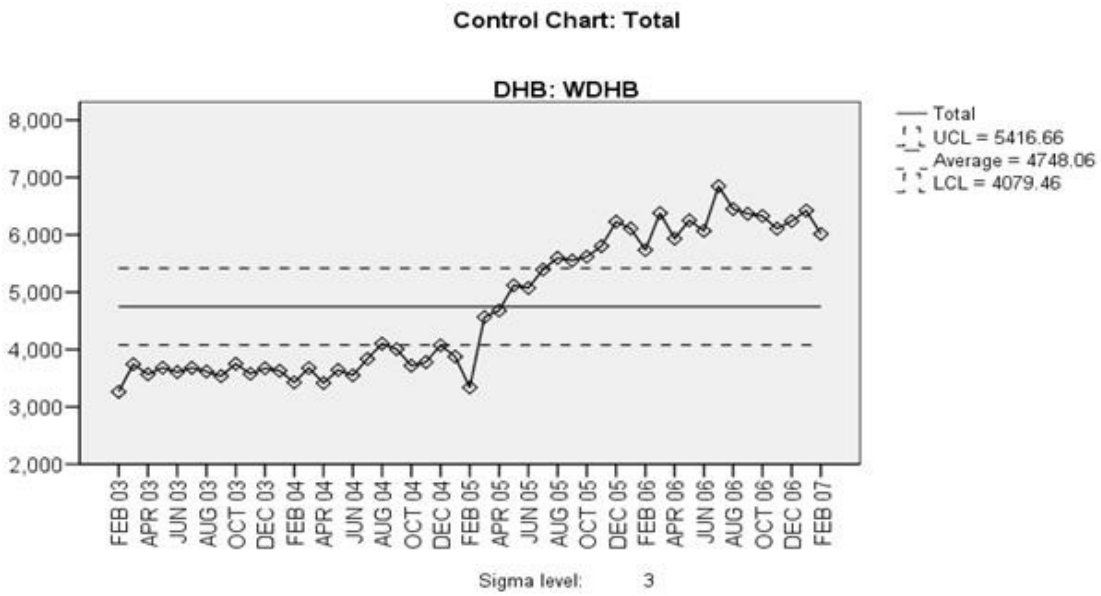


NSH=North Shore Hospital

There is a grouping of points below the centre line for the period before June 2004. The increase after this point is explained by the fact that in June 2004 NSH started accepting Orthopaedic patients, coupled with a severe winter. There is no evidence of special cause variation following February 2005. Figure 4 shows the control chart for ED presentations to WDHB over time.

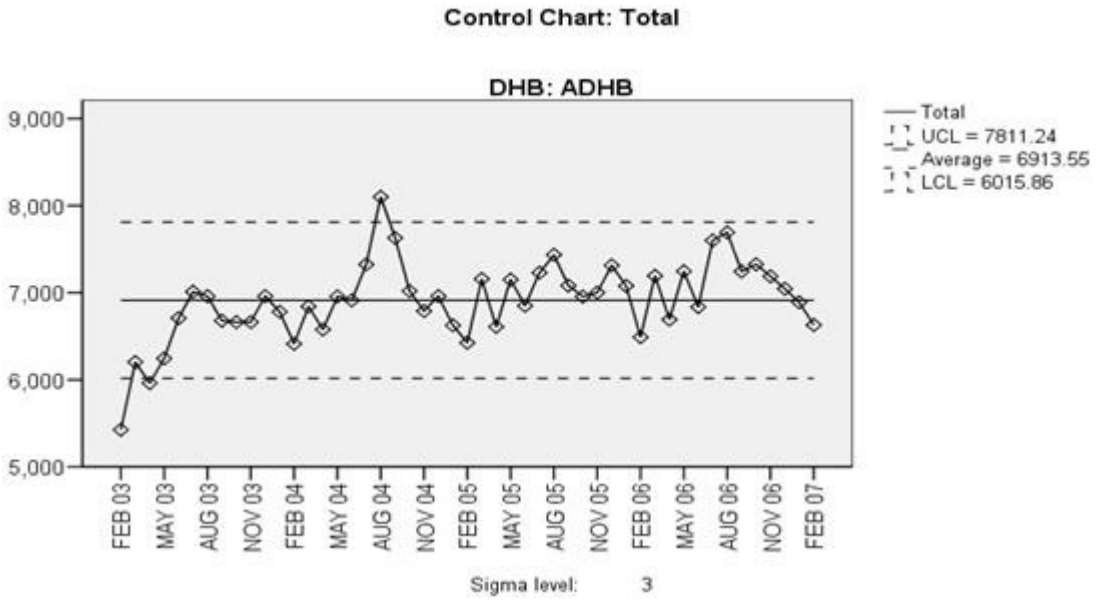
There is evidence of special cause variation after February 2005, with an increase in attendances. The number of presentations to WDHB increased 45% in the year after WH ED opened. Figure 5 shows the change in ED presentations to ADHB over time.

Figure 4. Change in Emergency Department presentations over time to Waitemata District Health Board



WDHB = Waitemata District Health Board (North Shore and Waitakere Hospitals).

Figure 5. Change in Emergency Department presentations over time to Auckland District Health Board

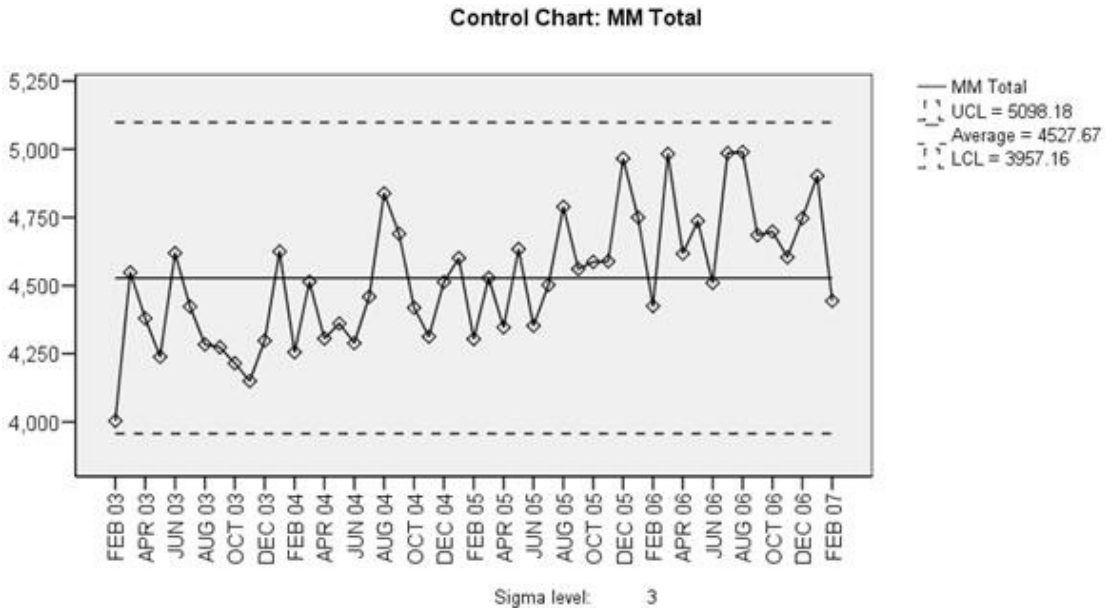


ADHB = Auckland District Health Board (Auckland City and Starship Hospitals)

Two points below the lower control limit in February 2003 reflects a time prior to merger of Obstetric and Cardiothoracic services onto a single hospital site in 2003.

There is one point outside the upper control limit in August 2004, reflecting a severe winter. This is a high point on all control charts, reflecting a regional phenomenon. There is no evidence of special cause variation following February 2005. Figure 6 shows the change in ED presentations to CMDHB (MMH) over time (control group).

Figure 6. Change in ED presentations to the control hospital over time



CMDHB = Counties Manukau District Health Board, MMH=Middlemore hospital

The chart displays a clear stable trend of increasing ED presentations over the whole study period.

Discussion

Recently Han[20] reported that ED expansion resulted in increased ED attendance rate and length of stay. It was thought that opening a new hospital ED would help ease the load on neighbouring hospitals in Auckland. However, the opening of the WH ED did not result in reduced patient presentations to other EDs in the region. Instead, the numbers increased in line with population growth (Figures 2 and 4) for both nearby and distant hospitals (Figure 5). In contrast to this there was a marked increase in ED presentations to WDHB, the area in which WH is situated, disproportionate to population growth (45% in the first year and 74% over the first 2 years after the opening of the new ED).

The effect of increasing hospital beds has been known for sometime. In 1961 Roemer et al published a landmark study[21] which found that increasing hospital bed availability was responsible for an increase in bed utilisation and length of stay in a community previously thought to be adequately served. Roemer suggested that doctors were the main drivers for the increased use of hospital beds as more beds

became available. Our study is the first to report the effect of building a new ED on the patient attendances to neighbouring EDs. The results of this study suggest that patients may directly increase utilisation of a service, independent of the influence of doctors: the increased ED utilisation at WDHB was mainly from lower-acuity self-presentations. (This aspect is subject of a separate paper in publication).

A number of possibilities may explain our study findings. Firstly, the increase in numbers of patients seen at WDHB following the opening of the WH ED may reflect redistribution from the primary health care sector in that area. This behaviour may be motivated by cost: in New Zealand ED care is free while there is a variable part charge for primary care; or the perception that presentation to a hospital ED allows greater access to specialist services.

We sought, but were unable to obtain information concerning after-hours primary care presentations to the after-hours primary care centre near the new facility. It is relevant to note that EDs see only a small proportion of all primary (self-presenting) attendances in a community. The attendance rate in general practice averages 4 visits per capita per annum,[22] whereas the average ED attendance rate (in this study) is less than 0.2 visits per capita per annum.

A relatively small redistribution of primary care visits to ED would barely register in all General Practice attendances but could have a significant impact on ED attendances. In this study, the per capita ED attendance rate at WDHB increased from a baseline of 0.09 visits per annum in 2003, to 0.15 visits per annum in 2007: this would barely register within the margin of error for all primary care attendances over the same period.

An alternative explanation is that the need for secondary emergency medical care for the population that lived closest to WH was not being addressed by the available EDs prior to the opening of WH ED. There is some evidence that this may have occurred in that the proportion of total ED presentations that have occurred in the regional DHBs following the opening of the new ED now more accurately reflects the distribution of the population in the region.

It is likely that the increase in total presentations within the WDHB, without a reduction in presentations to the nearby DHBs, reflects a combination of these possibilities. This finding has considerable implications for funding and resource allocation in the future.

As a retrospective observational study, our data may be subject to information bias. However as all EDs in the region use a national electronic database to establish a given individual's unique hospital number, this is unlikely. The data extracted did not include patient demographics, address, presenting complaint, length of stay or final diagnosis and disposition, which may have informed the debate regarding the appropriateness of given presentations for primary care services.

Ideally, control charts would have been created for more than 2 years before the opening of WH ED in order to identify long-term trends in attendance rate. However in 2001 NSH underwent a major change in their recordkeeping system and it is not possible to accurately compare ED data before and after this change. No change in record keeping occurred in the three DHBs during the study timeframe. Therefore we do not believe the results can be explained by changes in data collection.

Given the large numbers involved in this study, any differences in numbers attending would be likely to reach statistical significance if subjected to traditional statistical tests of significance. We believe that in this situation, use of control charts is more appropriate to detect clinically important differences in the process of patient attendance across the region.

The control chart method determines whether there has been change at a given time-point not explained by natural variation or chance alone. However, it does not determine why that change has occurred. It is possible that the increase in ED presentations to the WDHB area at February 2005 is due to a factor other than the opening of the WH ED. As there were no similar sharp increases in presentations to either the adjacent Auckland, or geographically distinct CMDHBs, we believe that there was no change in regional disease morbidity to account for the increase in presentations.

During the study period there was also no change in provision of primary care facilities in the area. Information about attendances to General Practitioners may help explain where the additional patient visits were created from, however we were unable to access this information.

Conclusions

The opening of WH ED had no effect on the number of presentations to the other hospitals in the region. However it appears to have resulted in a marked increase of presentations to the new facility, thus increasing the total numbers of ED patients seen within the Auckland region.

Conflict of interest: None known.

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Time to definitive care for patients with moderate and severe traumatic brain injury—does a trauma system matter?

Ritwik Kejriwal, Ian Civil

Abstract

Aim The presence of a trauma system has been associated with improved outcomes in patients with traumatic brain injury (TBI) by speeding up transfers to a neurosurgical centre. Improved outcomes are associated with time to neurosurgical intervention for those with significant extradural and subdural haemorrhages of less than 4 hours. To compare the outcomes for patients with TBI transferred directly from the scene of injury to Auckland City Hospital (ACH) with those transferred from other hospitals, transfer times and outcomes were evaluated in a consecutive cohort of patients recorded on the ACH trauma registry.

Method Patients admitted to ACH in 2004 and recorded on the trauma registry with a moderate or severe head injury (Abbreviated Injury Scale (AIS) score of 3 or greater) were included. The primary outcomes assessed were median time from injury to arrival and surgery, patient mortality, length of ICU stay and length of hospital stay.

Results 198 patients were admitted at ACH in 2004 with moderate and severe TBI. 95 patients (48%) were transferred from another hospital. Patients transported to ACH from the scene of injury arrived to ACH and underwent neurosurgery within a mean of 3 hours 50 minutes, whereas patients transferred from another hospital took significantly longer than 4 hours to arrive at ACH. Patients transferred from another hospital had similar mortality rate, length of ICU stay and length of hospital stay to those admitted directly.

Conclusion TBI patients who were transferred from another hospital arrived well outside the recommended guidelines. While no significant difference in outcome was noted in this small cohort of patients further studies are warranted. The development of a national trauma registry would allow accumulation of data on larger numbers of patients and determine the true relevance of international best practice guidelines in New Zealand.

The presence of a trauma system has been associated with decreased mortality and improved outcomes by potentially speeding up transfer of trauma patients to a major trauma centre.[1–3] This is particularly relevant in patients with head injuries where time to neurosurgical intervention from the time of significant traumatic brain injury (TBI) is important in determining the outcome.[4] North American guidelines recommend a maximum of four hours from the time of injury to neurosurgical attention for patients requiring evacuation of an intracranial haematoma.[4–7]

Trauma care in New Zealand is delivered in an ad hoc trauma system as opposed to a regional trauma system.[8] Auckland City Hospital (ACH) provides adult brain trauma care for a population of approximately two million people in the upper North Island of New Zealand with the most distant referring hospital 346 km from ACH by air.[9]

Patients with TBI are transported to the closest regional hospital for airway, breathing and circulation assessment and stabilisation. Once haemodynamically stable, an urgent computed tomography brain scan is performed that is then reviewed electronically by the neurosurgeon and intensivist in ACH. Transfer is recommended on the basis of a need for neurosurgical intervention or brain-oriented intensive care.

The objectives of this study were to determine the timelines associated with the sequence of care for TBI patients and identify any correlations between time to definitive care and outcome. We aimed to do so by comparing patients that were transported directly to ACH with those transferred from another hospital, as well as comparing the data with the current literature.

Patients and Methods

The study was carried out at ACH, New Zealand. The ACH Trauma Registry was interrogated for all patients admitted in 2004 with an Abbreviated Injury Scale (AIS) of 3 or greater for head injury. The registry used AIS-90 (98 update) version software.[10] This extraction method was selected as Glasgow Coma Scale (GCS) on arrival is often confounded by intoxication or other injuries. Therefore we sought to specifically identify that cohort of TBI patients who had a clinically significant TBI.

The ACH Trauma Registry includes all patients presenting to the hospital following injury who are admitted. 1137 patients were recorded in the ACH registry in 2004 calendar year. Patients who presented with an injury due to an underlying chronic subdural haematoma were not included in the Registry.

The following data were extracted from the registry: patient demographics, AIS, Injury Severity Score (ISS), details of injury, entrapment time, GCS, presence of other trauma, mode of transport, time and type of surgery, intracranial pathology, in-hospital survival, length of Intensive Care Unit (ICU) stay, and length of stay in the hospital. Patients transferred to ACH after 24 hours and patients without time of injury or time of arrival were excluded.

The primary outcomes of the study were median time from injury to arrival at ACH, time to neurosurgical intervention, mortality, length of intensive care, and length of hospital stay. The secondary outcome was effect of age, sex, mode of transport, ISS, GCS, presence of multiple trauma and road crashes on time from injury to arrival at ACH.

Statistical analysis—All analysis was performed using SAS version 8.2 (SAS Institute Inc., Cary, NC, USA). Comparison between primary and secondary groups was made using chi-square tests for equal proportion and student t-tests, and validated using Wilcoxon rank sum tests. Time from injury to arrival was found to be well approximated by a lognormal distribution and was consequently log-transformed prior to analysis. The univariate relationships between log (time from injury to arrival) and all other variables were assessed using linear regression, whilst multivariate analysis was performed using multiple linear regressions.

Multivariate models were constructed using a stepwise selection procedure and validated using a backwards elimination procedure. Results are presented as parameter estimates with a standard error. A two-sided p-value of 0.05 was considered to be statistically significant.

Results

198 patients were admitted at ACH in 2004 with moderate and severe TBI (defined as an AIS score of 3 or greater for head injury) in this study. Baseline data is outlined in Table 1; 48% of patients were transferred from another hospital.

Table 1. Baseline data

Patients	198
Male	153 (77%)
Female	45 (23%)
Median age (IQR)	35.5 (15–94)
Median ISS (IQR)	17 (9–50)
Cause of injury	
Road crashes	74 (37%)
Falls	54 (28%)
Assault	37 (19%)
Other	33 (17%)
GCS at the scene of injury	(173)
≤8	58 (34%)
≥9, <14	52 (30%)
14–15	63 (36%)
Mode of transport (386)	
Road ambulance	166 (84%)
Rotary wing (helicopter)	10 (5%)
Private	16 (8%)
Other / Unknown	6 (3%)
Multiple trauma	38 (19%)
Trapped patients (392)	17 (9%)
In-hospital survival	165 (83%)
Patients transferred from another hospital	95 (48%)

Fifteen percent of the patients were excluded due to time of injury or arrival time not being available, or due to patients presenting to the trauma hospital more than 24 hours after injury. After excluding these patients, the data was divided in two groups—patients transported to ACH directly from the scene of injury (PRIMARY group) and patients taken to another hospital before they were transferred to ACH (SECONDARY group). There were 97 patients (57%) in the PRIMARY group and 73 (43%) patients in the SECONDARY group. 43 (59%) patients in the SECONDARY group were transferred from two hospitals within Auckland region. Baseline comparison between the two groups at ACH is outlined in Table 2.

Neurosurgical procedures performed within 24 hours of injury were included in the analysis. 24 neurosurgical procedures were performed on 13 patients in the PRIMARY group and 18 procedures were performed on 16 patients in the SECONDARY group. 20 procedures were excluded due to missing data or surgery performed later than 24 hours after injury.

Primary outcome of median time from injury to arrival at ACH was adjusted for entrapment (Table 3). Overall the median time from injury to arrival was 1 hour 43 minutes. The median time from injury to arrival at ACH as well as to neurosurgery for the SECONDARY group was significantly greater than the PRIMARY group ($p<0.0001$). There were no statistically significant differences between the two groups for in-hospital survival, length of ICU stay, and length of hospital stay.

Table 2. PRIMARY group vs SECONDARY Group

Variables	Primary	Secondary	P values
Patients (injury time available)	97	73	
Median age (IQR)	40 (15-94)	33 (20-49)	0.04
Median ISS (IQR)	17 (9-50)	16 (6-25)	0.27
Road crash	45 (47%)	22 (57%)	0.06
Auckland region transfers		43 (59%)	
Other transfers		30 (41%)	
Mode of transport Road ambulance Rotary wing (helicopter)	 86 (89%) 6 (6%)	 64 (88%) 3 (4%)	 0.84
Multitrauma	24 (25%)	9 (12%)	0.04
Patients admitted to ICU	44 (45%)	33 (45%)	
Neurosurgical procedures	23	18	
EVD/ICP (burr hole)	6 (26%)	3 (17%)	
Evacuation of mass lesion - craniotomy - craniectomy - burr hole	 7 (30%) 6 (26%) 0 (0%)	 13 (72%) 1 (6%)	
Other procedures	4 (17%)	1 (6%)	

Table 3. Primary outcome

Variables	PRIMARY	SECONDARY	P values
Median time Injury to arrival Injury to surgery Arrival to surgery	 0:50 3:50 3:10	 7:03 7:33 2:17	 <0.0001 <0.0001 0.24
In-hospital survival Median length of ICU stay Median length of hospital stay	0.82 1 day 7 days	0.90 3 days 7 days	0.10 0.74 0.10

Time to definitive care was analysed for patients transferred to ACH from other hospitals in Auckland region as well (Table 4). Median time from injury to arrival for Auckland region transfers (6 hours 16 minutes) was significantly greater than the PRIMARY group ($p<0.0001$).

Table 4. Analysis of Auckland region transfers

Median time	PRIMARY	Auckland region transfers	Other transfers
Injury to arrival	0:50	6:16	7:16
Injury to surgery	3:50	6:46	8:33
Arrival to surgery	3:10	1:20	1:21

Transport by road ambulance (p=0.04) and road crashes (p=0.05) were predictors of a reduction in time from injury to arrival in univariate regression analysis as well as the multivariate model.

Discussion

It is well established that time to neurosurgery from time of injury is critical in determining neurological outcome. In the United States, the Brain Trauma Foundation recommends a time limit of four hours to surgical intervention for acute subdural haematomas based on 1981 study by Seelig et al.[4]

European guidelines do not set a time frame, but there is a consensus that the speed of referral and transfer to neurosurgical care may critically influence the outcome.[3,11] Even as Wilberger et al found no improvement in mortality rate in patients treated within four hours,[12] other studies show that time from point of neurological deterioration to surgery is related to improved outcome.[13,14]

From the time of injury, the PRIMARY group median time to arrival (50 minutes) and median time to surgery (3 hours 50 minutes) at ACH were within four hours. On the other hand, median time from injury to arrival at ACH for the SECONDARY group was well outside the recommended guidelines (7 hours 3 minutes). Therefore patients transported directly to ACH are likely to undergo neurosurgery within international guidelines whereas those transferred are not.

These times are comparable to the ones reported in the literature. Our SECONDARY group times were similar to a Liverpool Hospital study that reported a median time to definitive care of 6 hours 39 minutes for patients transferred from another hospital.[15] Another study reported a median delay of 4 hours and 22 minutes in patients who were transferred from another hospital.[16]

ACH had significantly larger proportion of patients transported from another hospital (48%) compared to the trauma systems in the literature[16], which means that the effect of undue delay and its potential effect on outcome even more important than in systems where there are fewer transfers. It is particularly relevant that even from hospitals within the metropolitan region there was a significant delay to from injury to surgery compared with those transported directly to ACH.

While there were no differences between the two groups in hospital stay ICU stay, and mortality in this study, the numbers were very small. A similar study in western Virginia found that patients transferred from another hospital had worse outcomes in the above-mentioned parameters compared to patients directly transferred to a major trauma centre.[17]

The linear regression analyses of ACH data suggests that transport by road is associated with shorter transport times than when transport is by helicopter. This is due to rotary wing transfers being used sparingly in the region serviced by ACH and being limited to patient transfers from places with difficult road access.

Benefits of a state trauma system have been well documented. Cooper et al assessed management of road traffic fatalities and suggested that having a trauma system in Victoria, Australia is likely to decrease preventable death rates.[18] Similarly, Mullins et al attributed improved outcomes among patients with head injuries in Oregon to the

institution of a state trauma system.[2] It compared the state trauma system of Oregon to an ad-hoc trauma system of Washington and found a significant difference in patient mortality. Another study carried out in Rhode Island concluded that the presence of a major trauma centre that is not part of a state trauma system results in delays in time to definitive care for injured patients.[1]

State trauma systems have triage criteria that allow patients with major trauma injury to be directly transported to a designated major trauma centre. This is related to improved outcomes according to Poon et al,[19] who concluded that direct admission of head injury patients to the primary care of the neurosurgeons is the best policy in the reduction of mortality and morbidity. Patel et al and Cooper et al provided strong evidence as well in his observational study that patients who have neurosurgical trauma are better managed in a neurosurgical centre as part of a Level 1 Trauma Centre.[14,20]

While the ACH SECONDARY group were not worse off in outcomes measured, based on the literature ACH trauma care may be improved by an introduction of a triage criteria or an ambulance bypass protocol. Lind et al stated that triaging is not an option for patients outside Auckland,[9] but triaging for patients within Auckland may be beneficial as 43 out of 73 patients in the secondary group were transferred from hospitals within the Auckland metropolitan area. This will potentially enable these patients to undergo neurosurgical intervention within recommended guidelines.

This retrospective study is only a snapshot of the performance of ACH ad-hoc trauma system in one calendar year. A limitation of the study is incomplete data for some of the patients at ACH. Nonetheless, approximately 85% of the patients with AIS of three or greater were still included for primary outcome analysis making it a fair representation of the baseline population.

Another limitation was the small proportion of patients that underwent neurosurgery within 24 hours, which made it difficult to compare time to surgery between the two groups. This study was also limited by lack of available data from the hospitals of primary presentation. Were NZ to have a national trauma registry, such as the state registry in Victoria, Australia, a much more comprehensive dataset could have been assembled to allow more comprehensive analysis of this topic.

Conclusion

Almost half of the TBI patients treated at ACH in 2004 were transferred from another hospital and they arrived well outside the recommended time guidelines. While this made no difference in patient outcomes as measured by LOS and survival in our study, there may be benefit in having a greater proportion of patients directly transferred from the scene of injury. Development of a prospective national trauma registry would allow ongoing analysis of the process of trauma care and outcome in NZ. Future prospective studies are recommended to look at outcomes including neurological outcome of patients with TBI in New Zealand.

Competing interests: None known.

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Paramedic-administered prehospital thrombolysis is safe and reduces time to treatment

Anil M Ranchord, Sandhir Prasad, Phillip Matsis, Scott A Harding

Abstract

Introduction The Kapiti Coast region is remote from Wellington Hospital with an ambulance transport time of 1 hour. To reduce delays in the treatment of myocardial infarction (MI), a prehospital thrombolysis (PHT) programme was initiated in 2003.

Methods This study evaluated outcomes of the Kapiti PHT programme between 2003 and 2007. Paramedics attending patients with suspected MI-transmitted electrocardiograms to our Coronary Care Unit where a physician made the decision whether or not to thrombolyse. Thrombolysis was then administered by a paramedic. Patients from the Kapiti region treated with in-hospital thrombolysis (IHT) between 1999 and 2003 formed the control group.

Results A total of 50 Kapiti patients received PHT. The group receiving IHT were older than those receiving PHT but other baseline characteristics were similar. No patients without MI or with a contraindication received PHT. In the PHT group there was one minor bleed but no major bleeding, stroke or death occurred during transport to hospital. The median scene to thrombolytic time for PHT was 89 minutes faster (44 minutes versus 133, P<0.0001) than in patients transferred for IHT. The median scene to thrombolytic time for PHT was similar to the door to thrombolytic time for IHT (P=0.13). In-hospital mortality in the PHT group (8.0%) was similar to the IHT group (6.0%, P=1.0) but heart failure was reduced (10% vs. 26%, P=0.04)

Conclusions Prehospital thrombolysis administered by paramedics is safe and reduces the time to treatment and was associated with a reduction in heart failure.

Treatment of acute ST segment elevation myocardial infarction (STEMI) with thrombolysis has been shown to reduce both early and late mortality by about 20%.[1,2] Large-scale trials have also demonstrated that the benefit is greatest when thrombolytic therapy is given early.[3,4] The TIMI 2 trial demonstrated that for every hour thrombolytic therapy was delayed mortality increased by 1%.[5] Given these findings the introduction of strategies that reduce the time between onset of symptoms to administration of thrombolysis (symptom to needle time) are essential.

One such strategy is to administer fibrinolytic therapy in a prehospital setting. The randomised trials performed to date have generally shown that prehospital thrombolysis (PHT) substantially reduces symptom to needle times.[6–8] A meta-analysis of these trials by Morrison and colleagues demonstrated a 58 minute reduction in the time to thrombolysis that was associated with a reduction in mortality.[9]

Wellington Hospital provides the secondary and tertiary cardiac services for the Kapiti Coast region. The remote location of this region means that on average an extra

hour is required to transport patients with acute myocardial infarction to Wellington Hospital. To reduce delays in the treatment of myocardial infarction (MI), a PHT programme for the Kapiti region was initiated in 2003. This study aimed to evaluate the safety and efficacy of the Kapiti PHT programme.

Methods

Study population—All patients in the Kapiti region who presented with chest pain consistent with myocardial ischaemia and changes on their electrocardiogram meeting the diagnostic criteria for a STEMI between June 2003 and November 2007 were considered for PHT. Patients were considered ineligible for PHT if they were over the age of 75, had chest pain for more than 12 hours or had a contraindication to thrombolysis. During the study period 50 patients were treated with PHT and included in the study. A further 21 patients from the Kapiti region with STEMI received in-hospital thrombolysis (IHT) during the study period for the following reasons: failed intravenous cannulation (3), age > 75 (2), STEMI not recognised in the ambulance (2), uncontrolled hypertension (1), telemedicine system upgrade (6), and other reasons (7).

A historical cohort of 50 consecutive patients from Kapiti who were transferred to Wellington Hospital for in-hospital thrombolysis from June 1999 to May 2003, prior to the establishment of the PHT programme, were used as a control group. In addition, door to thrombolysis times were also collected on a cohort of 161 patients from the central Wellington region that received IHT during the study period (June 2003 to November 2007). Ethical approval for the study was obtained from the Central Regional Ethics Committee.

Delivery of prehospital thrombolysis—All ambulances attending to emergency calls in Kapiti were initially equipped with Biolog systems (Biolog 3000, Micromedical Industries Limited 1998, Australia) to enable telephonic transmission of 12-lead electrocardiograms (ECGs) to a desktop computer in the coronary care unit (CCU) of Wellington Hospital. This system used the Telecom CDMA cellular data network via an attached cell phone with data cable. There were several problems with this system: paramedics could not confirm the ECG recording, incomplete cell phone coverage of the Kapiti area, latency in the data signal resulting in failed transmission and inability to identify point of transmission failure.

In October 2004 the system was upgraded to a LIFENET STEMI management system. This consisted of a LIFENET receiving station (Physio-Control Inc., a division of Medtronic, USA) for the CCU and activation of the communication package and installation of a Vodafone GSM network SIM card in the LIFEPACK® 12 defibrillators (Physio-Control Inc., a division of Medtronic, USA) carried by the ambulance service. This system has proven to be superior mainly in its ease of use, better area coverage and automaticity in receiving and printing of ECGs in the CCU.

All paramedics staffing these ambulances were trained to record and interpret 12-lead ECGs and in the use of the transmission system. During the study period patients presenting to the ambulance service in the Kapiti region with ischaemic chest pain had an ECG recorded. If ECG changes possibly consistent with a STEMI were detected the ECG was transmitted to the Coronary Care Unit of Wellington Hospital where it was reviewed by the medical staff. For those being considered for fibrinolytic therapy paramedics also completed a checklist recording the presence or absence of contraindications to this.

Following review of the ECG medical staff then contacted the paramedics in the ambulance by cell phone and discussed the ECG findings and clinical scenario. Patients with ischaemic symptoms of less than 12 hours in duration, an ECG consistent with an acute STEMI and no contraindications to thrombolysis were considered eligible for PHT. Informed consent was obtained and 5000 units of intravenous heparin followed by the initial 10 mg bolus of reteplase was given on-site or on-route to Wellington Hospital.

Patients were also given aspirin 300 mg to chew if this had not been previously administered. A second bolus of reteplase was given 30 minutes later. All ambulances attending patients in the Kapiti Coast were equipped with a defibrillator.

Data collection and definitions—We retrospectively collected information on patient demographics and clinical characteristics, time delays to thrombolysis (which form a routine part of paramedic recordings in our region), appropriateness of PHT and adverse events related to PHT occurring in the ambulance. In addition, we determined the in-hospital and 30-day incidence of death, reinfarction and

heart failure. This data was obtained from review of patient medical records, the National Admissions Database and by contacting general practitioners when this information was unavailable.

STEMI was defined as >2 mm ST elevation in two or more contiguous precordial leads, >1 mm in two or more contiguous limb leads or presumed new left bundle branch block. Heart failure was defined as being present if pulmonary vascular congestion or oedema was present on a chest X-ray or if clinical signs of heart failure were present that resulted in a diuretic being prescribed or the dose of a diuretic being increased. Reinfarction was defined according to the joint ESC/ACCF/AHA/WHF task force redefinition of myocardial infarction.[10]

Statistical analysis—Continuous variables are presented as median (25^{th}, 75^{th} percentile) and categorical variables as percentages. Univariate analysis was performed using the Mann-Whitney U test for continuous variables and either chi-square or Fisher's exact test for categorical variables. A P value <0.05 was considered statistically significant. All statistical analyses were performed using GraphPad Prism v4.0 software.

Results

Baseline clinical characteristics are shown in Table 1. Patients that received PHT were younger than those that received IHT (median age 62 years versus 69 years, P=0.0007). The remaining baseline demographics and clinical characteristics were similar between the two groups (Table 1).

Table 1. Patient demographics and clinical characteristics

Variables	Prehospital thrombolysis (n=50)	In-hospital thrombolysis (n=50)	P values
Age (years)*	62 (53–69)	69 (63–79)	0.0007
Male sex	80 %	70 %	0.25
Diabetes mellitus	12 %	12 %	1
Hypertension	38 %	40 %	0.84
Hyperlipidaemia	34 %	28 %	0.52
Smokers	34 %	20 %	0.11
Previous MI	12 %	20 %	0.28
Previous PCI	6 %	6 %	1
Previous CABG	2 %	2 %	1
Anterior MI	50%	34%	0.11

*Median (25^{th}–75^{th} percentile); CABG=coronary artery bypass grafting, MI=myocardial infarction, PCI=percutaneous coronary intervention.

Successful transmission of ECGs from the ambulance to CCU was achieved in 89.2% with use of the Biolog 3000 system. Following upgrade to the Medtronic Lifenet system the rate of successful transmission of ECGs improved to 98.6%. No patients without a STEMI or with a contraindication to treatment received thrombolysis in the PHT or IHT groups. However, 3 patients aged over 75 years were given PHT after consideration of the risks and benefits of early treatment.

All patients that received PHT were administered aspirin and heparin prior to thrombolysis. In the PHT group there was one minor bleed but no major bleeding, stroke or death occurred during transportation to hospital. A cardiac arrest occurred in 8% (4) of patients that received PHT during transportation compared to 2% (1) in the IHT group, however this difference was not statistically significant (P=0.36). All of these patients were successfully defibrillated.

Time delays—Time delays to thrombolysis are shown in Table 2. There was no difference in the median scene to departure time for patients treated with PHT (28 minutes) compared to IHT (25 minutes, P=0.39). The median transport time to hospital was similar for the PHT (59 minutes) and IHT (56 minutes, P=0.19) patients. However, the median scene to thrombolytic time for PHT was 89 minutes faster than patients transferred for IHT (44 versus 133 minutes, p <0.0001). The median scene to thrombolytic time for PHT (44 minutes) was similar to the door to thrombolytic time for Kapiti patients previously transferred for IHT (50 minutes, P=0.13).

Furthermore, the scene to thrombolytic time for PHT was also similar to the median door to thrombolytic time (44 minutes versus 51 minutes, P=0.07) for a contemporaneous cohort of 161 patients from Wellington who received IHT. Thrombolysis was more likely to be administered within 3 hours of symptom onset in patients treated with PHT compared to those transferred for IHT (69% versus 32%, P=0.0008).

Table 2. Time delays (minutes)

Scenario	Prehospital thrombolysis (n=50)	In-hospital thrombolysis (n=50)	P values
Ambulance call to scene	8 (7–12)	7 (5–10)	0.15
Scene to departure	28 (19–38)	25 (17–33)	0.39
Ambulance call to thrombolytic	57 (44–65)	145 (123–165)	<0.0001
Scene to thrombolytic	44 (35–58)	133 (110–160)	<0.0001
Symptom onset to thrombolytic	129 (85–205)	210 (176–312)	<0.0001
Door to thrombolytic	–	50 (40–62)	–
Transport to hospital	59 (51–71)	56 (47–69)	0.19

Data are presented as median (25^{th}–75^{th} percentile).

In-hospital and 30-day outcomes—In-hospital mortality (8% versus 6%, P=1), rescue PCI (24% versus 10%, P=0.06) and reinfarction (8% versus 4%, P=0.68) were similar in the PHT & IHT groups. However, heart failure was less frequent in the PHT group (10% versus 26%, P=0.04). At 30 days mortality (8% versus 10%, P=1) and reinfarction (12% versus 8%, P=0.74) remained similar and heart failure remained less frequent in the PHT (10% versus 28%, P=0.04) group compared to IHT.

Discussion

Our study demonstrates that PHT administered by paramedics safely reduced the time to thrombolysis for Kapiti patients by 89 minutes. This compares favourably with a meta-analysis of randomised trials of IHT versus PHT that showed a 58 minute reduction in the time to thrombolysis.[9] Previous studies have reported a 4 to 16-minute extension of the time from paramedic arrival at the scene to departure in patients who receive PHT.[11-13] This has been attributed to time spent in the assessment and delivery of thrombolytic therapy.

A more recent study by Mclean et al found there was only a 4-minute delay at the scene to deliver intravenous heparin and bolus thrombolytic. In our study the scene to

departure and transport times were similar. This is likely to be due to the performance of the ECG and administration of the first dose of thrombolytic on route in the majority (80%) of cases.

Consistent with previous studies, the time saved by PHT in our study significantly exceeded the time taken to transport patients to hospital. Prehospital thrombolysis not only reduces delays due to transport but also avoids further delays that occur once the hospital is reached due to reassessment in the emergency department.

The reduction in time delay with PHT was not associated with any cases of inappropriate thrombolysis or major bleeding during transport to hospital. This is consistent with previous studies which have also shown no increase in adverse events in patients receiving PHT.[8,14,15]

We observed a lower incidence of heart failure in the PHT group both in-hospital and at 30-day follow-up. Two factors may explain this result. Firstly the substantial reduction in time to thrombolysis in the PHT group, as the benefits of thrombolysis are directly proportional to the time from symptom onset to administration.[5] Secondly the trend towards greater use of rescue PCI in the PHT group may have resulted in a greater rate of coronary artery patency and myocardial salvage.

Our findings are also consistent with those of previous studies which have shown improved left ventricular function[8,16] and/or reduced incidence of heart failure with PHT.[15,17] A meta-analysis of randomised trials performed by Morrison and collegues[9] as well as the findings from the French nationwide USCI registry[18] suggest that PHT also improves survival. Interestingly an increased use of rescue PCI in those treated with PHT has been observed in previous studies.[15,18]

Use of paramedics to deliver PHT is potentially advantageous as they already staff the ambulances that provide first medical contact. Initially concerns were raised about the safety of this approach. However, several studies including our own have now documented the safety and feasibility of paramedic-administered thrombolysis.[8,13–15,19] The meta-analysis of PHT by Morrison et al that included studies with paramedic, general practitioner and intensivist-administered thrombolysis found that there was no difference in clinical outcomes between physician and paramedic delivered PHT.

Furthermore, the ASSENT-3 PLUS trial[20] demonstrated a longer treatment delay with physician compared to paramedic administrated PHT which may have been due to the limited numbers of physician staffed ambulances and the longer time to arrival at the scene that followed.

Over the last decade there has been much debate over the relative advantages of primary PCI compared to thrombolysis. There is now general consensus that primary PCI when provided in a timely manner is superior to thrombolysis.[21] However, it is clear that thrombolysis remains a highly effective treatment. In fact patients with STEMI who receive thrombolysis within 3 hours have been shown to have similar outcomes to those treated with primary PCI. Regardless of the mode of reperfusion, the overarching concept is to minimise time from onset of symptoms to initiation of reperfusion strategy.

Many communities in New Zealand are remote from interventional centres and therefore do not have rapid access to primary PCI. Our study and other studies of

prehospital thrombolysis have consistently demonstrated substantial reductions in symptom to thrombolysis times using this strategy. More widespread use of this strategy needs to be promoted in areas remote from interventional centres. At the very least the data from a number of studies indicate that paramedics should be performing 12-lead electrocardiography in the field.[11,14,15,19]

Our study is retrospective and uses a historical cohort of patients treated with IHT for comparison. As such it is possible that confounding factors may occur. The baseline demographics and clinical characteristics of the PHT and IHT groups were well matched apart form the IHT group being older. This age difference is unlikely to account for the additional delay in the time to thrombolysis in the IHT group as the call to scene, scene to departure, and transport times to hospital were similar. However, it is possible that age may have had an impact on clinical outcomes such as the occurrence of heart failure.

Furthermore, streptokinase was used in 82% of the historical IHT cohort compared with 100% use of retaplase in the PHT which also may have influenced clinical outcomes as the use of retaplase may result in a higher rate of infarct related artery patency at 90 minutes. Finally, there were only 100 patients assessed in this study, therefore it was not powered to detect differences in clinical outcomes.

Conclusion

Our study demonstrates that PHT can be performed safely and appropriately by paramedical personnel with the support of the local coronary care unit. Furthermore, our programme resulted in substantial reductions in the time to thrombolysis and was associated with a reduction in the occurrence of heart failure. More widespread use of this strategy needs to be promoted in regions where rapid access to an interventional centre is not available

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Contemporary New Zealand coefficients for the Trauma Injury Severity Score: TRISS(NZ)

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Abstract

Aims To develop local contemporary coefficients for the Trauma Injury Severity Score in New Zealand, TRISS(NZ), and to evaluate their performance at predicting survival against the original TRISS coefficients.

Methods Retrospective cohort study of adults who sustained a serious traumatic injury, and who survived until presentation at Auckland City, Middlemore, Waikato, or North Shore Hospitals between 2002 and 2006. Coefficients were estimated using ordinary and multilevel mixed-effects logistic regression models.

Results 1735 eligible patients were identified, 1672 (96%) injured from a blunt mechanism and 63 (4%) from a penetrating mechanism. For blunt mechanism trauma, 1250 (75%) were male and average age was 38 years (range: 15–94 years). TRISS information was available for 1565 patients of whom 204 (13%) died. Area under the Receiver Operating Characteristic (ROC) curves was 0.901 (95%CI: 0.879–0.923) for the TRISS(NZ) model and 0.890 (95% CI: 0.866–0.913) for TRISS (P<0.001). Insufficient data were available to determine coefficients for penetrating mechanism TRISS(NZ) models.

Conclusions Both TRISS models accurately predicted survival for blunt mechanism trauma. However, TRISS(NZ) coefficients were statistically superior to TRISS coefficients. A strong case exists for replacing TRISS coefficients in the New Zealand benchmarking software with these updated TRISS(NZ) estimates.

A fundamental component of any trauma system is a defined performance improvement programme centred on the audit capacity provided by a properly functioning trauma registry.[1,2]

Trauma system quality control programmes have traditionally focused on decreasing the number of preventable deaths.[3] Thus while trauma system performance monitoring can be a complex multifaceted activity, in its most simple form it can be reduced to the question *For any trauma system, is the proportion of patients who survive a given injury as high as that achieved by the best systems in the country (and comparable systems internationally)?*

A negative answer to this question does not necessarily imply suboptimal care, but it does prompt a search for explanations that may lead to better outcomes for injured patients. The importance of a continued search for improved trauma system performance is that even after trauma system implementation, 15–20% of trauma-related deaths continue to be preventable.[4–8]

Performance monitoring essentially involves comparing actual survival outcomes with expected norms.[9] The Trauma Injury Severity Score (TRISS), a weighted combination of patient age, Injury Severity Score (ISS) and Revised Trauma Score (RTS), is one score developed to estimate this expected survival.[3]

The TRISS is not without limitations, many of which have been extensively documented elsewhere.[10–12] However, despite these limitations, TRISS continues to be the most commonly used tool for benchmarking trauma outcomes.[10,13,14] Until a superior score is developed and widely adopted, it is likely that the TRISS will continued to be used for this purpose in the coming years. As such, the question of interest is *Is the TRISS a valid predictor of survival in contemporary localized populations?*

The TRISS coefficients used for weighting were derived using ordinary logistic regression models from the Major Trauma Outcome Study (MTOS), a cross-sectional United States of America (USA) study conducted over 20 years ago.[3] At that time it was acknowledged that "as improvements in trauma care over time result in decreased mortality, these [TRISS] coefficients can be expected to change."[3]

Advances in trauma management over the past 20 years coupled with the considerable differences in case mix between the USA and other countries, including New Zealand, suggests that these TRISS coefficients may not reflect current optimal performance benchmarks.[11,15,16] Should important differences be found between the existing and re-estimated contemporary local jurisdiction coefficients, then these re-estimated coefficients might be employed to allow relevant and valid routine benchmarking of the performance of trauma systems within that jurisdiction.

Using data from four New Zealand Trauma Registries, serving approximately half the nation's population, this study aimed to develop local contemporary coefficients for the Trauma Injury Severity Score in New Zealand, TRISS(NZ). Once estimated, we then sought to evaluate the predictive performance of TRISS(NZ) against TRISS.

Methods

Study design—Retrospective cohort study.

Study population and period—All adult New Zealanders (aged ≥15 years) who sustained a serious traumatic injury, defined as having ISS>15, who survived until presentation at Auckland City Hospital, Middlemore Hospital, Waikato Hospital, or North Shore Hospital between 1 January 2002 and 31 December 2006. Cases of poisoning, burns, hangings, and simple fractured neck of femurs were excluded.

Procedure—Unit record data was extracted from each of the participating trauma registries. Three of the four registries used the Collector Trauma Registry software and database, and the fourth has replicated the data collection tool manually. However, not all registries operated for the duration of the data collection period, thus data was obtained for the period of times available at each registry (Auckland and Middlemore Hospitals: full coverage from 1 January 2002–31 December 2006; Waikato Hospital: 1 January 2002–31 December 2003; and North Shore Hospital: 14 June 2004–31 December 2006). Waikato Hospital's Trauma Registry was restarted in June 2006. Data from this period was not available at the time of data extraction.

Data extraction included demographic characteristics (date of birth or age, gender); anatomical and physiological parameters of injury severity, where available before and after arrival in the emergency department; external cause and intent of injury; hospital stay; and survival status. A hospital trauma number was also extracted for data validating purposes. De-identified data were downloaded into separate password protected Microsoft Excel files for cleaning, combining, and subsequent data analysis.

The TRISS model—Derived from ordinary logistic regression models on the MTOS data, the TRISS model has two separate specifications for adults:

- For injuries sustained from a blunt mechanism, and
- For injures sustained from a penetrating mechanism.

TRISS coefficients give the probability of survival (P_S) rather than the probability of death (P_D); naturally $P_D = 1 - P_S$. The probability of survival for any one patient can be estimated from:

$$P_S = 1/(1+e^{-b})$$

where, for blunt mechanism trauma

$$b = -0.4499 + (0.8085 \times RTS) - (0.0835 \times ISS) - (1.7430 \times Age), \textbf{(1)}$$

for penetrating mechanism trauma

$$b = -2.5355 + (0.9934 \times RTS) - (0.0651 \times ISS) - (1.1360 \times Age), \textbf{(2)}$$

Injury Severity Score (ISS) has values from 16 to 75; Age is coded: 0 if patient age is 15–54 years, and 1 if patient age is ≥55 years; and the Revised Trauma Score (RTS) is given by

$$RTS = (0.2908 \times RR) + (0.7326 \times SBP) + (0.9368 \times GCS). \textbf{(3)}$$

Respiratory rate (RR), systolic blood pressure (SBP), and the Glasgow Coma Score (GCS) each have values assigned to them as included in Table 1. If the expression for the RTS (equation 3) is directly substituted into TRISS equations (1) and (2) above, then it is convenient to re-write the equation for blunt mechanism trauma as

$$B = -0.4499 + (0.2351 \times RR) + (0.5923 \times SBP) + (0.7574 \times GCS) - (0.0835 \times ISS) - (1.7430 \times Age) \textbf{(4)}$$

and the equation for penetrating mechanism trauma as

$$b = -2.5355 + (0.2889 \times RR) + (0.7278 \times SBP) + (0.9306 \times GCS) - (0.0651 \times ISS) - (1.1360 \times Age) \textbf{(5)}$$

Table 1. Values associated with respiratory rate (RR), systolic blood pressure (SBP), and the Glasgow Coma Score (GCS) used in the calculation of the Revised Trauma Score (RTS), and when considered separately in logistic regression models

Value	Respiratory rate (RR) per minute	Systolic blood pressure (SBP) mmHG	Glasgow Coma Score (GCS)
0	0	0	3
1	1–5	1–49	4–5
2	6–9	50–75	6–8
3	>29	76–89	9–12
4	10–29	>89	13–15

Statistical analyses—Raw data from each hospital registry was converted into a separate file that used consistent variable names and definitions. Consistency and range checks were performed to identify any discrepant or anomalous data. When identified, checks were made to the raw data file or, where permitted, to the hospital registries for data verification. No data trimming or replacing aberrant unvalidated data with missing values was undertaken. The separate databases were then combined using SAS version 9.1 software (SAS Institute Inc, Cary, NC, USA) and exported into one Microsoft Excel file for subsequent analysis in Stata version 10 software (StataCorp, College Station, TX, USA). Comparisons of categorical variables between groups was made using Fisher's exact test.

Like the TRISS model, it was intended that separate derivations would be undertaken for adults with:

- Injuries sustained from a blunt mechanism; and
- Injuries sustained from a penetrating mechanism.

A progressive model development approach was undertaken to derive TRISS(NZ). However, each considered model incorporated only those variables already used in TRISS, and the variable characterisations and values specified by equations 1–5 and Table 1.

Initially, the TRISS model was run on the New Zealand data and the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) statistics calculated. We label this model 1. Information criteria can be used to determine model superiority between competing nested or non-nested regression models.[17] A nested regression model is one which is completely subsumed within another larger regression model whereas non-nested regression models each have unique terms. Both the AIC and BIC penalise for model complexity and reward for goodness-of-fit; with the preferred model balancing these competing demands and yielding the lowest values.[17]

Next, employing the same statistical techniques used to determine the coefficients for TRISS, an ordinary logistic regression model was conducted on the New Zealand data to estimate local coefficients and calculate the AIC and BIC statistics. We label this model 2. If the AIC and BIC statistics for model 2 were not superior to model 1, then this would suggest that New Zealand coefficients are no better than those of the TRISS and the investigation would cease. Alternatively, if model 2 demonstrated superiority over model 1, then this would suggest that New Zealand coefficients are statistically superior to those of the TRISS.

As the RTS variable in both models 1 and 2 also had its component coefficients estimated from the MTOS (equation 3), it is of interest to determine whether these component variables may also benefit from re-estimation. Thus the ordinary logistic regression was repeated on the New Zealand data except that the RTS variable was replaced by: RR, SBP and GCS. We label this model 3. If model 3 was no better that model 2, then this would suggest that New Zealand coefficients for ISS, age, and RTS are statistically superior to those of the TRISS, but that the New Zealand coefficients for the component variables of the RTS are no better than those derived from the original MTOS. However, if model 3 was superior to model 2, then this would suggest that all variables used in TRISS would be improved with New Zealand coefficients.

Finally, recognising that patients were nested within hospitals, and that there is likely variation in case-mix and survival rates between hospitals, a multilevel mixed-effects logistic regression model was employed. This regression model, labelled model 4, uses the variables defined by either model 2 or 3, depending upon which model was superior, and a higher-level hospital variable (with values indicated by an anonymous code) which is treated as a random effect within the model. Again AIC and BIC statistics were computed and compared to the previous models. Based on the AIC and BIC statistics, we defined TRISS(NZ) to be the best model from candidate models 2-4.

The predictive abilities of the fixed-effects portion of the TRISS(NZ) and TRISS models were then assessed using nonparametric Receiver Operating Characteristic (ROC) curves and analysis. A ROC curve is a graphical plot of the sensitivity vs. (1 – specificity) of survival as its discrimination threshold is varied. The ROC analysis comprised of a test of equality between the fixed-effects portion of the TRISS(NZ) and TRISS model ROC areas, and was undertaken using the χ^2 test.

We used only the fixed-effects portion so that the complexity of the final TRISS(NZ) model would be no different to that of TRISS for end-users, and the only potential changes would be in the specification of the predictor variable coefficients. An α=0.05 was used to defined significance for all statistical tests.

Ethics—Patients were identified by each registry according to the inclusion criteria and all data was made anonymous before study investigator access. The study was conducted in accordance with a protocol approved by the Griffith University Ethics Committee, Australia; Auckland District Health Board and the Northern X Regional Ethics Committee in Auckland, New Zealand. For the remaining participating registries Gatekeeper Approvals were sought as the project was assessed to be a de-identified audit activity and was deemed not to require ethics approval (North Shore Hospital, Middlemore Hospital and Waikato Hospital).

Results

There were 1735 eligible patients identified, 1672 (96%) injured from a blunt mechanism and 63 (4%) injured from a penetrating mechanism.

Blunt mechanism trauma—Of the 1672 patients suffering blunt mechanism trauma, 1250 (75%) were males and the average age was 38 years (range: 15 to 94 years). Complete information to calculate the TRISS was available for 1379 (82%) patients

and prehospital information allowed another 186 (11%) patients to be included in the analyses, yielding a total sample of 1565 (94%) trauma patients. Of these, 204 (13%) died and 1361 (87%) survived their injury.

Of the 107 patients with incomplete information (where a TRISS could not be calculated), 22 (21%) died and 85 (79%) survived their injury, a distribution with significantly more deaths than seen in the sample with complete information (Fisher's exact test P=0.04).

Table 2. Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) statistics for the progressive model development of TRISS(NZ) using various coefficient estimation regression models and variable combinations

Model	Method of coefficient estimation	Fix-effect variables	AIC	BIC
Model 1 [TRISS]	Ordinary logistic regression on MTOS data	ISS, Age, RTS	802.9	829.7
Model 2	Ordinary logistic regression on NZ data	ISS, Age, RTS	784.6	806.0
Model 3	Ordinary logistic regression on NZ data	ISS, Age, RR, SBP, GCS	760.0	792.2
Model 4 [TRISS(NZ)]	Multilevel mixed-effects logistic regression on NZ data	ISS, Age, RR, SBP, GCS	751.7	789.2

Note: The preferred model has the lowest AIC and BIC values.

Deriving TRISS(NZ)—AIC and BIC statistics for the progressive model development appear in Table 2. ISS, age and RTS coefficients estimated from the New Zealand data (model 2) yielded lower AIC and BIC statistics than those of TRISS (model 1). When the component variables of the RTS, namely RR, SBP and GCS, were also re-estimated with the New Zealand data (model 3), the resultant AIC and BIC statistics were lower still.

Finally, the introduction of the higher-level hospital random effect into the regression (model 4) yielded the lowest and best AIC and BIC statistics. The fixed-effect component of this multilevel model thus defined TRISS(NZ). The coefficients and associated 95% confidence intervals (95% CI) for TRISS(NZ) appear in Table 3.

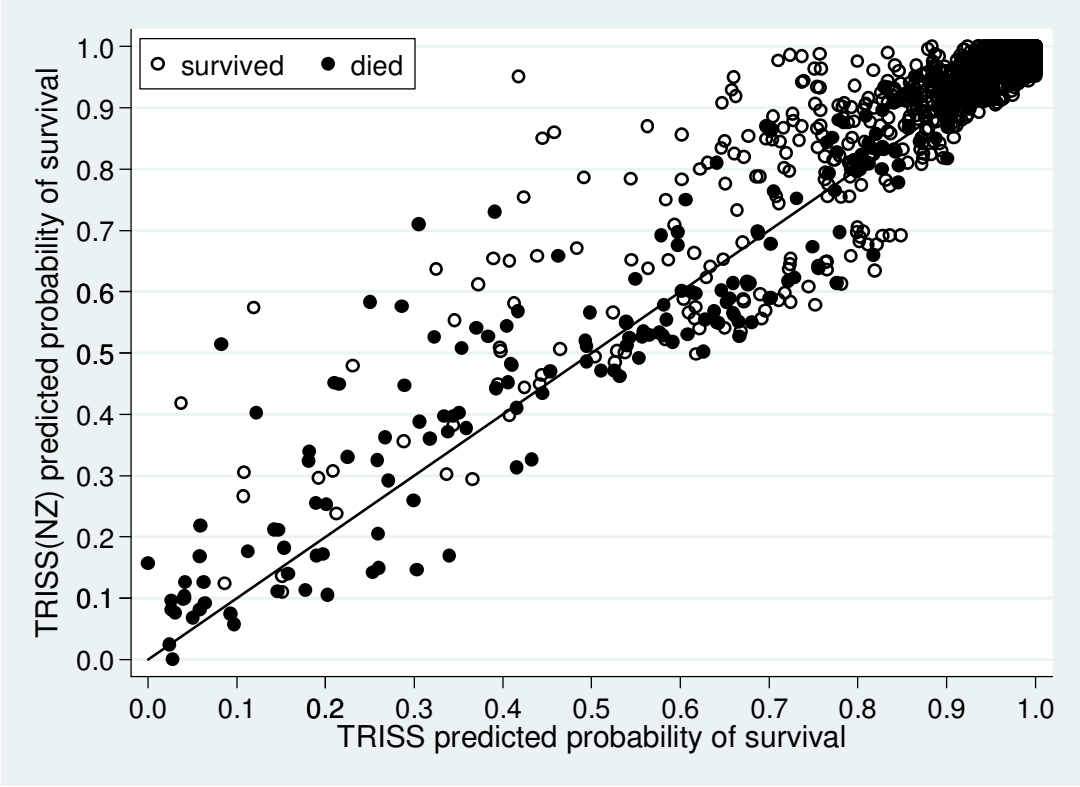
Table 3. Estimated coefficients of blunt mechanism injury for TRISS (equation 4) and the fixed-effects portion of the TRISS(NZ) model, together with the 95% confidence intervals (95%CI)

Variables	TRISS Coefficient	TRISS(NZ) Coefficient (95%CI)
Intercept	-0.4499	-0.8118 (-2.1353 – 0.5117)
ISS	-0.0835	-0.0443 (-0.0639 – -0.0248)
Age	-1.7430	-2.1864 (-2.6867 – -1.6862)
RTS		
RR	0.2351	0.0031 (-0.1958 – 0.2020)
SBP	0.5923	0.5594 (0.2899 – 0.8289)
GCS	0.7574	1.0749 (0.9266 – 1.2232)

A scatter-plot of the predicted values from the fixed-effects portion of the TRISS(NZ) model and those derived from TRISS appears in Figure 1. Departures from the 45° degree line give the magnitude of difference between predicted values from the two models. The mean difference between the predicted values from the two models was 0.019 (standard deviation 0.062) and ranged from -0.202 to 0.510.

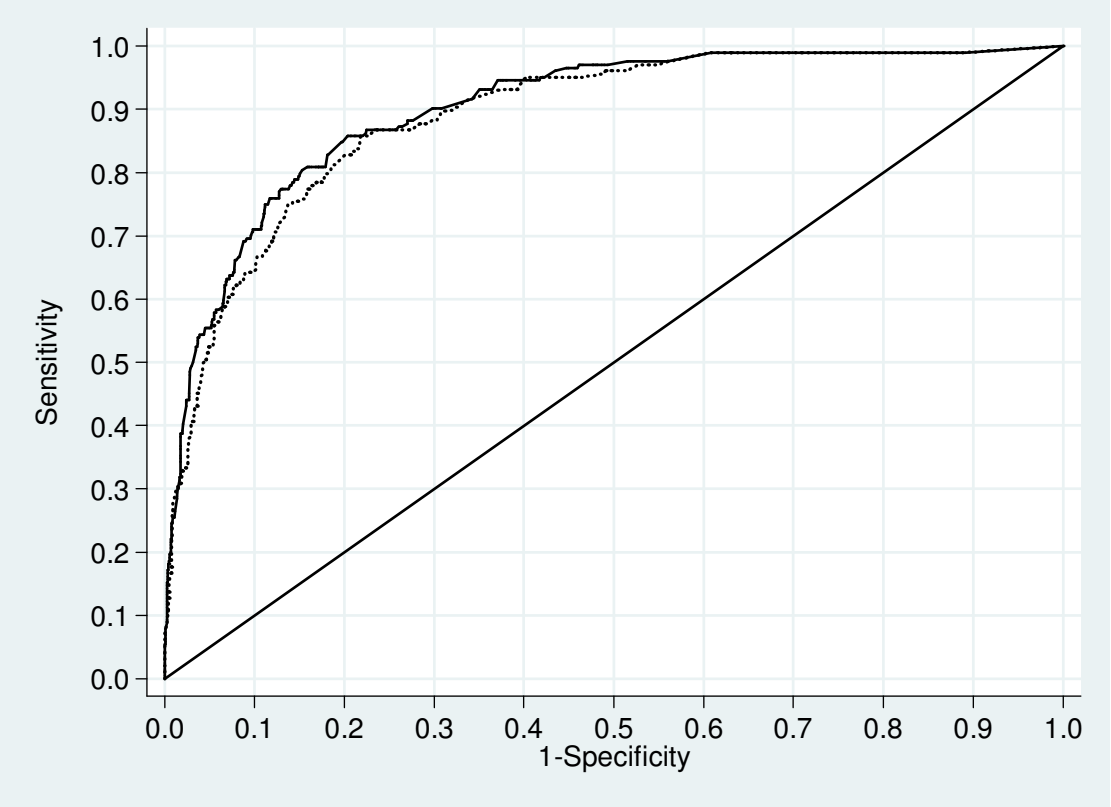
So while the average difference was small, there were substantial positive and negative differences between predicted values for many patients.

Figure 1. Scatter-plot of predicted probability of survival (P_S) from the fixed-effects portion of the TRISS(NZ) model against values derived from TRISS, together with the 45-degree reference line



The ROC curves corresponding to the fixed-effects portion of the TRISS(NZ) and TRISS models, together with the 45-degree reference line, appears in Figure 2. The area under the ROC curves was 0.901 (95%CI: 0.879–0.923) for the TRISS(NZ) model and 0.890 (95%CI: 0.866–0.913) for TRISS; a difference that was statistically significant (P<0.001). This implies that, for these empirical data, the TRISS(NZ) model's predictive capacity is significantly better than that of the TRISS model.

Figure 2. Nonparametric Receiver Operating Characteristic (ROC) curves corresponding to the fixed-effects portion of the TRISS(NZ) model (solid-line) and TRISS (dotted-line), together with the 45-degree reference line



Penetrating mechanism trauma—Of the 63 eligible patients suffering trauma from a penetrating mechanism, 53 (84%) were male and the average age was 35 years (range: 16 to 74 years). Complete information to calculate the TRISS was available for 45 (71%) patients and prehospital information allowed another 10 (16%) patients to be included in the analyses, yielding a total sample of 55 (87%) trauma patients. Of these, 9 (16%) died and 46 (84%) survived their injury.

Of the 8 patients with incomplete information, 2 (25%) died and 6 (75%) survived their injury, a distribution not significantly different to that seen in the sample with complete information (Fisher's exact test P=0.62). Unfortunately, a total sample of 55 is insufficient to produce a valid statistical model for TRISS(NZ).

Discussion

The TRISS methodology for evaluating the performance of trauma systems has been well established, and is widely used. Recent publications however have tended to focus on identifying and highlighting its limitations,[10] and often present the authors' own alternative.[18–22] Most new alternatives perform only marginally better, and usually at the expense of parsimony and often tautologically including outcome (e.g.

complications) as a predictive variable. In this context the first major finding of the current study was to illustrate how robust the simple TRISS model is.

In yet another sample of patients the simple TRISS(NZ) model accurately predicted survival with area under the ROC curves of 0.901 (95%CI: 0.879, 0.923). For a simple algorithm with only five variables easily obtained at time of admission with injury, this is a remarkable feat.

The second main finding of this study was that for trauma caused from a blunt mechanism, contemporary locally specified coefficients of the TRISS variables were statistically superior to the original coefficients. This improvement was seen when employing the same method of estimation as that used to define TRISS (i.e. ordinary logistic regression).

Further improvements were observed when locally specified coefficients for the components variables of the RTS were derived and by extending the model to include hospitals as a higher level random effect. The use of multilevel models in this analysis has theoretic and conceptual advantages, particularly when there is likely variation in case-mix and survival rates between hospitals.[17]

On the basis of this finding, there is a strong case to be made for replacing TRISS coefficients in the New Zealand benchmarking software with these updated TRISS(NZ) estimates. With the development of a New Zealand national registry, or a bi-national trauma registry in Australia and New Zealand, the estimates obtained in this sample could be retested in a second sample to validate the calculations.[2]

A national (or bi-national) sample encompassing a greater time period would increase the case numbers for analysis and improve the precision of the estimates. With a functioning national trauma database, coefficient revisions might be made on a regular basis (say every 3-5 years) thereby accommodating changes in demographics and case-mixing.

Unfortunately, we have insufficient data for penetrating trauma to make a similar assessment in this study. However, it is likely that with greater case numbers that locally specified coefficients would also be significantly superior.

There are several data quality issues mostly relating to consistency of data management between hospitals that may be usefully addressed at the time of establishing a national New Zealand trauma registry. The first of these issues are the discrepancies across registries for case inclusion and recording of individual fields. These were investigated during the data management phase of the current study and for major trauma cases (ISS>15) consistency of variable collection across New Zealand trauma registries was found to be high.

Multiple fields were collected in all registries with similar systems of collection. However, some key discrepancies did exist. A particular problem recognised in trauma registries worldwide is the measurement of the physiological state of the patient using the components of the Revised Trauma Score (RTS).[23, 24] There was variation in whether prehospital scores were recorded, when they were done, and at what point after presentation emergency department scores were measured and recorded.

These issues are accentuated in cases of patient transfer or when the patient was intubated. All but one hospital in the sample used AIS-98 (the exception used AIS-90), although the literature suggests this difference is unlikely to have affected the resulting TRISS calculations.[25]

Conclusion

TRISS has proved to be a valid predictor of survival from major trauma and a simple, robust instrument for benchmarking in the New Zealand trauma system. The use of locally derived contemporary New Zealand coefficients, TRISS(NZ), increases the predictive capabilities of the methodology. There would appear to be little reason to replace TRISS(NZ) with a more complex statistical model that uses variables that may be less universally collected or inconsistently defined.

Future studies might consider testing the sensitivity of the TRISS(NZ) model, and perhaps make improvements by using re-specifications or re-categorizations of the original predictor variables, consider variable interactions, and by investigating power components.[20,26]

Competing interests: None known.

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Using Trauma Injury Severity Score (TRISS) variables to predict length of hospital stay following trauma in New Zealand

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Abstract

Aim To develop and assess the predictive capabilities of a statistical model that relates routinely collected Trauma Injury Severity Score (TRISS) variables to length of hospital stay (LOS) in survivors of traumatic injury.

Method Retrospective cohort study of adults who sustained a serious traumatic injury, and who survived until discharge from Auckland City, Middlemore, Waikato, or North Shore Hospitals between 2002 and 2006. Cubic-root transformed LOS was analysed using two-level mixed-effects regression models.

Results 1498 eligible patients were identified, 1446 (97%) injured from a blunt mechanism and 52 (3%) from a penetrating mechanism. For blunt mechanism trauma, 1096 (76%) were male, average age was 37 years (range: 15–94 years), and LOS and TRISS score information was available for 1362 patients. Spearman's correlation and the median absolute prediction error between LOS and the original TRISS model was ρ=0.31 and 10.8 days, respectively, and between LOS and the final multivariable two-level mixed-effects regression model was ρ=0.38 and 6.0 days, respectively. Insufficient data were available for the analysis of penetrating mechanism models.

Conclusions Neither the original TRISS model nor the refined model has sufficient ability to accurately or reliably predict LOS. Additional predictor variables for LOS and other indicators for morbidity need to be considered.

In New Zealand, injuries account for approximately 1600 deaths and 42,000 hospitalisations per annum; with associated social and economic costs of NZ$6–7 billion per year.[1]

Reducing preventable injuries is one of the main public health challenges for New Zealand in the 21st Century.[2] The national burden of injury can be addressed by the primary prevention of injury occurrence, improving acute care of injury and maximising opportunities for rehabilitation of the injured person.

Improvements in trauma care over the last few decades have been responsible for substantial improvements in survival rates following major trauma,[3–5] although further safety and error reductions are achievable. A recent South Western Sydney trauma death review analysis found that 22.5% of all deaths were avoidable;[5] a result consistent with other preventable death studies conducted in Australia and New Zealand.[6–8] However, little is known about the extent that improvements in trauma

care have lead to decreased injury-related morbidity and improved long-term outcomes.

Quality control programs for existing trauma systems focus on fatality indicators and pay scant attention to improving measures of long-term outcome of injury patients.[5,9] Given that the majority of patients survive their injuries, and a large proportion of these experience disability up to 12 months following injury,[10–13] ignoring non-fatality indicators neglects a fundamental component of trauma system performance measurement.[3,14]

Capturing both fatality and non-fatality indicators would enable trauma management to facilitate improvements that aim to reduce mortality and morbidity. For instance, a 2007 United Kingdom national confidential enquiry into patient outcome and death following trauma showed that almost 60% of the patients received a standard of care that was less than good practice.[15] It is naturally of interest to determine the effect of mortality and morbidity rates by increasing the level of good practice standard care.

The focus on fatality indicators in existing trauma systems is due, in part, to the lack of a valid "threat to morbidity" injury severity score available for use in routine trauma data collection. Further, there is no international consensus on appropriate morbidity scores that can be used to benchmark non-fatal outcomes from injury.[3,10]

The trauma injury severity score (TRISS) methodology is a simply applied parsimonious model with good predictive capabilities for threat to survival and has become the backbone of trauma system quality assurance.[9] Attempts to develop a "threat to morbidity" injury severity score have focused on modelling length of stay (LOS), a commonly adopted proxy measure of morbidity obtainable from routine hospital data.[11,16] Many complicated techniques have been advocated and a multiplicity of variables used to model LOS.[17–19] These techniques are limited by the need for additional information and techniques above those already collected or available in many trauma registries around the world.

Given the universal acceptance of the TRISS methodology, and that a recent paper has confirmed its utility in the New Zealand context for quantifying threat to life,[20] it is important to quantify the extent to which the variables used in the TRISS methodology can be used to predict LOS before embarking upon the development of complex models to predict threat to morbidity. This question has not yet been addressed within the New Zealand trauma care context.

The TRISS variables (age, Injury Severity Score [ISS], Glasgow Coma Scale [GCS], systolic blood pressure [SBP], and respiratory rate [RR]) are universally collected and consistently defined in registries throughout the world. Using data from four New Zealand trauma registries, serving approximately half New Zealand's population, this study aimed to develop and assess the capabilities of a multivariable two-level mixed-effects regression model that predicts LOS in survivors of traumatic injury based on the routinely collected TRISS variables.

Method

Detailed information about the sample and procedure is described elsewhere.[20]

Study design—Retrospective cohort study.

Study population and period—All adult New Zealanders (aged ≥15 years) who sustained a serious traumatic injury, defined as having Injury Severity Score (ISS)>15, and who survived until discharge from Auckland City, Middlemore, Waikato, or North Shore Hospitals between 1 January 2002 and 31 December 2006. The ISS is a sum of the square of the three most injured body regions (head, face, thorax, abdomen, lower and upper extremities), each having severity assessed via the Abbreviated Injury Scale: 1 Minor, 2 Moderate, 3 Serious, 4 Severe, 5 Critical.[21] Cases of poisoning, burns, hangings, and simple fractured necks of femurs were excluded.

Procedure—Unit record data was extracted from each of the participating trauma registries. Not all registries operated for the duration of the data collection period, thus data was obtained for the period of times available at each registry (Auckland City and Middlemore Hospitals: full coverage from 1 January 2002–31 December 2006; Waikato Hospital: 1 January 2002–31 December 2003; and North Shore Hospital: 14 June 2004–31 December 2006). Waikato Hospital's Trauma Registry was restarted in June 2006 but data from this period was unavailable for extraction.

Extracted variables included: date of birth or age, and gender; anatomical and physiological parameters of injury severity before and after arrival in the emergency department; external cause and intent of injury; hospital stay and survival status. A hospital trauma number was also extracted for data validating purposes. De-identified data were downloaded into separate password protected Microsoft Excel files.

TRISS model—The TRISS model, which estimates the probability of survival (P_S), has two separate specifications:

- For injuries sustained from a blunt mechanism, and
- For injures sustained from a penetrating mechanism.

The $P_S = 1/(1 + e\text{-}b)$ where: $b = -0.4499 + (0.2351 \times RR) + (0.5923 \times SBP) + (0.7574 \times GCS) - (0.0835 \times ISS) - (1.7430 \times Age)$ for blunt mechanism trauma, and $b = -2.5355 + (0.2889 \times RR) + (0.7278 \times SBP) + (0.9306 \times GCS) - (0.0651 \times ISS) - (1.1360 \times Age)$ for penetrating mechanism trauma.[22]

In these expressions, ISS has values from 16 to 75; Age is coded: '0' if patient age is 15–54 years, and '1' if patient age is ≥55 years; respiratory rate (RR) is coded '0' if patient has 0 breaths/minute, '1' for 1-5 breaths/minute, '2' for 6–9 breaths/minute, '3' for >29 breaths/minute, and '4' for 10–29 breaths/minute; systolic blood pressure (SBP) is coded '0' if patient has 0 mmHg pressure, '1' for 1–49 mm Hg, '2' for 50–75 mmHg, '3' for 76–89 mmHg, and '4' for >89 mmHg; and the Glasgow Coma Scale (GCS) is coded '0' for a score of 3, '1' for scores 4–5, '2' for scores 6–-8, '3' for scores 9–12, and '4' for scores 13–15.

Statistical analyses—Raw data from each hospital registry was converted into a separate file that used consistent variable names and definitions. Consistency and range checks were performed to identify any discrepant or anomalous data. When identified, checks were made to the raw data file or, where permitted, to the hospital registries for data verification. No data trimming or replacing aberrant unvalidated data with missing values was undertaken. The separate databases were then combined using SAS version 9.1 (SAS Institute Inc, Cary, NC, USA) and exported into one Microsoft Excel file for subsequent analysis in Stata version 10 software (StataCorp, College Station, TX, USA).

LOS was calculated as: discharge date-admission date. As the trauma registries in the sample do not track patients between hospitals, and data were de-identified, it was not possible determine the cumulative LOS of an injury event for a patient who had a hospitals transfer. Instead, these transfers and readmissions were generally counted as separate admissions in each registry, each having a separate non-cumulative LOS value. Recognising that patients were nested within hospitals, two-level mixed-effects regression models were employed to model LOS. Hospitals were treated as random effects within the model.

Given the skewed distribution of LOS, three standard power transformations of the LOS data were considered (logarithmic, square-root and cubic-root), and residuals assessed from two-level mixed-effects regression models using the TRISS variables and their original specifications to determine:

- Which transformation was most appropriate for the data, and
- Whether the assumptions of the regression model would be importantly violated. Shapiro-Wilk's W statistics and a visual assessment of a histogram of the residuals was used to make this assessment.

Various characterisations of all candidate predictive variables were the derived and compared in bivariable two-level mixed-effects regression models using the Bayesian information criterion (BIC).[23] The BIC penalises for model complexity and rewards for goodness-of-fit; with the preferred model balancing these competing demands and yielding the lowest BIC value. Separate regression models were then developed for each potential explanatory measure. Those that were statistically significant were entered into a multivariable model and a manual backward selection process was used to eliminate non-significant variables until the most parsimonious main effects model was determined. Then all two-factor interactions were included and again a backward selection method was employed to eliminate non-significant variables.

Residual checks were undertaken after the derivation of the final multivariable two-level mixed-effects regression model to determine whether important violations of the model's assumptions existed. Finally, the predictive abilities of the final regression model was assessed using Spearman's correlation coefficient and the median absolute prediction error (MAPE), derived by taking the absolute difference between predicted and observed LOS values. For comparison, the MAPE analysis was repeated using predicted values derived from a regression model that included the original TRISS variables and their original specifications. The α=0.05 was used to defined significance for all statistical tests.

Ethics—Patients were identified by each registry according to the inclusion criteria and all data was made anonymous before study investigator access. The study was conducted in accordance with a protocol approved by the Griffith University Ethics Committee, Australia; Auckland District Health Board and the Northern X Regional Ethics Committee in Auckland, New Zealand. For the remaining participating registries Gatekeeper Approvals were sought as the project was assessed to be a de-identified audit activity and was deemed not to require ethics approval (North Shore, Middlemore, and Waikato Hospitals).

Results

There were 1498 eligible patients identified, 1446 (97%) injured from a blunt mechanism and 52 (3%) injured from a penetrating mechanism.

Blunt mechanism trauma

The average age of the 1446 blunt mechanism trauma survivors was 37 years (range: 15–94 years) and 1096 (76%) were male. All 1446 patients had a valid LOS value. Overall, the median LOS was 11 days (range: 0–171 days). For those who were transferred from another hospital, their median LOS was 10 days (range: 0–121 days), while for those who made no hospital transfer, their median LOS was 11 days (range: 0–171 days). LOS of 0 days represents the situation where discharge date and admission date were the same. Figure 1 reveals that the LOS distribution is highly positively skewed; confirmed by the empirical dispersion index (variance/mean ratio)=18.3.

Sufficient information was available to calculate TRISS scores for 1362 (94%) patients. There was no difference in LOS between patients with and without available information to calculate TRISS scores (two-sample Wilcoxon rank-sum test, P=0.77).

Data transformation

Table 1 includes the Shapiro-Wilk's W statistics from the standardised residuals yielded from the two-level mixed-effects regressions using untransformed and three standard power transformations of LOS (logarithmic, square-root, and cubic-root). Note that the W statistic must lie in the range $0<W\leq1$ and small values of W lead to the rejection of the normality assumption. Table 1 reveals that the cubic-root transformation yields residuals most consistent with the normality assumption (having

the largest W statistic). A histogram of these residuals provided little evidence that the assumption of normality was importantly violated.

Figure 1. Histogram of length of stay for traumatic injuries resulting from blunt mechanisms in New Zealand adult (≥15 years)

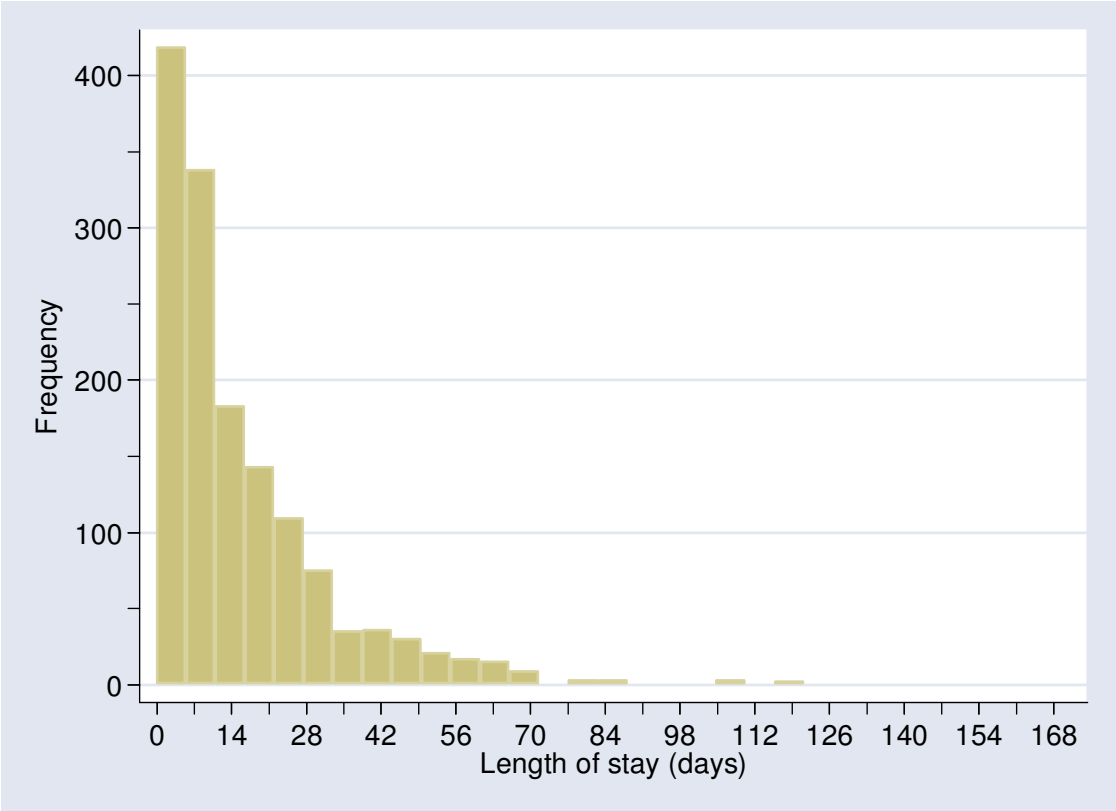


Table 1. Shapiro-Wilk's W statistics for the standardised residuals of various power transformations of the length of stay (LOS) data regressed from two-level mixed-effects regression models using the TRISS variables and their original specifications

Power transformation	Shapiro-Wilk's W
Identity (untransformed)	0.795
Logarithmic [log(LOS)]	0.987
Square-root [$\sqrt{}$LOS]	0.962
Cubic-root [$\sqrt[3]{}$LOS]	0.990

Optimising the components of the predictive model

All variables used in the original TRISS model were investigated. Each variable was investigated separately in the two-level mixed-effects regression model of cubic-root transformed LOS, and characterisations were assessed using the BIC statistic.

Age—Four characterisations of age were considered:

- Age (years) as a continuous variable (mean=36.6 years, SD=18.2 years);
- Log(age) as a continuous variable (mean=3.5, SD=0.5);
- Age partitioned into approximate quartile: 15–20 years (n=356, 25%), 21–35 years (n=458, 32%), 36–50 years (n=309, 21%), and >50 years (n=323, 22%); and
- Age dichotomised according to the original TRISS specifications: 15–54 years (n=1190, 82%), and ≥55 years (n=256, 18%).

The resultant BIC=3611.84, 3603.87, 3623.43, and 3606.34, respectively. Based on this BIC statistic, log(age) as a continuous variable best predicts LOS.

Injury Severity Score (ISS)—Four characterisations of ISS were considered:

- ISS as a continuous variable between 16 and 75, as used in the original TRISS model (mean=22.6, SD=7.5);
- Log(ISS) as a continuous variable (mean=3.1, SD=0.3);
- ISS trichotomised into clinically relevant groups: 16-24 (n=951, 66%), 25-41 (n=456, 32%), and 42-75 (n=39, 3%); and
- ISS partitioned into approximate quartiles: 16 (n=268, 19%); 17-20 (n=447, 31%); 21-25 (n=388, 27%); and 26-75 (n=343, 24%).

The resultant BIC=3503.93, 3484.02, 3532.80 and 3486.48, respectively. Based on this BIC statistic, log(ISS) as a continuous variable best predicts LOS.

Glasgow Coma Scale (GCS)—Two characterisations of GCS were considered:

- Categorising GCS according to the original TRISS specifications: 3 (n=74, 5%), 4–5 (n=46, 3%), 6–8 (n=129, 9%), 9–12 (n=140, 10%), 13–15 (n=1,024, 72%), and then treating these groups as a continuous variable as used in the original TRISS model; and
- Treating the GCS groups as a categorical variable.

The resultant BIC=3450.41 and 3477.64, respectively. Based on this BIC statistic, treating GCS groups as a continuous variable best predicts LOS.

Systolic blood pressure (SBP)—Two characterisations of SBP were considered:

- Categorising SBP according to the original TRISS specifications: 0–49 mmHg (n=14, 1%), 50–75 mmHg (n=22, 2%), 76–89 mmHg (n=50, 4%), and >89 mm Hg (n=1,311, 94%), and then treating these groups as a continuous variable as used in the original TRISS model; and
- Treating the SBP groups as a categorical variable.

The resultant BIC=3418.43 and 3403.55, respectively. Based on this BIC statistic, treating the SBP groups as categories best predicts LOS.

Table 2. Coefficient estimates (Est.) and associated 95% confidence intervals (95%CI) of the 2-level mixed-effects regression models, with patients nested in hospitals, and hospitals treated as random effects, for bivariable comparisons between each candidate predictor variable and the cubic-root transformed length of stay (LOS) variable, for the multivariable main-effects model of all significant candidate predictor variables, and for the full multivariable two-level mixed-effects regression model which includes the main-effects model and all significant two-factor interactions

	Bivariable analysis			Multivariable main effects			Full multivariable model		
Predictor variables	**Est.**	**(95%CI)**	**P-value**	**Est.**	**(95% CI)**	**P-value**	**Est.**	**(95%CI)**	**P value**
log(Age in years)	0.120	(0.032–0.208)	0.008	0.160	(0.076–0.244)	<0.001	0.159	(0.075–0.243)	<0.001
log(Injury Severity Score)	0.855	(0.709–1.001)	<0.001	0.760	(0.608–0.912)	<0.001	0.756	(0.604–0.908)	<0.001
Glasgow Coma Score	-0.105	(-0.143–-0.067)	<0.001	-0.081	(-0.121–-0.041)	<0.001	-0.088	(-0.130–-0.047)	<0.001
Systolic blood pressure mm Hg			<0.001			0.02			0.007
0-49	-0.792	(-1.216–-0.369)		-0.635	(-1.169–-0.100)		-3.002	(-5.059–-0.945)	
50-75	0.370	(0.032–0.709)		0.107	(-0.214–0.429)		-0.734	(-1.666–0.197)	
76-89	0.413	(0.186–0.640)		0.234	(0.003–0.444)		0.442	(-0.267–1.152)	
>89	0.000	reference		0.000	reference		0.000	reference	
Respiratory rate breath/minute			<0.001			0.007			0.005
0-5	0.407	(0.276–0.538)		-0.057	(-0.443–0.330)		-0.112	(-0.500–0.276)	
6-9	0.267	(-0.115–0.649)		-0.066	(-0.449–0.318)		-0.093	(-0.476–0.290)	
10-29	0.000	reference		0.000	reference		0.000	reference	
>29	0.407	(0.276–0.538)		0.225	(0.097–0.353)		0.224	(0.096–0.352)	
Systolic blood pressure (SBP) × Glasgow Coma Score (GCS) interaction									0.02
0-49 × GCS							0.664	(0.111–1.218)	
50-75 × GCS							0.251	(-0.009–0.511)	
76-89 × GCS							-0.063	(-0.258–0.132)	
>89 × GCS							0.000	reference	

Respiratory rate (RR)—Two characterisations of RR were considered:

- Categorising RR according to the original TRISS specifications (with 0 and 1-5 breaths per minute groups combined): 0–5 breaths per minute (n=28, 2%), 6–9 breaths pre minute (n=17, 1%), 10–29 breaths per minute (n=1,157, 85%), and >29 breaths per minute (n=163, 12%) and then treating these groups as a continuous variable as used in the original TRISS model; and
- Treating the RR groups as a categorical variable.

The resultant BIC=3326.87 and 3306.30, respectively. Based on this BIC statistic, treating the RR groups as categories best predicts LOS.

Multivariable model development

Bivariable comparisons between each of the predictor variables listed above and the cubic-root transformed LOS variable were conducted and results appear in Table 2. All candidate variables were significantly associated with LOS. The main effects model then combined all variables significant in the bivariable comparisons and eliminated those variables no longer significant. However, all variables significant in the bivariable comparisons remained statistically significant in the multivariable main effects model.

Once the main effects model was found, all two-factor interactions were investigated separately. Only the interaction between SBP×GCS (P=0.02) was statistically significant. The full multivariable two-level mixed-effects regression model was thus derived by combining this significant interaction with the main effects model (Table 2). The hospitals, treated as random effects, had a significant variance component estimated at 0.079 (95%CI: 0.016–0.386); and this final multivariable two-level mixed-effects regression model was superior to an ordinary regression model (P=0.03).

Residual checks included a scatter-plot of the standardised residuals against the untransformed predicted LOS values (in days) together with a superimposed lowess curve (a nonparametric estimator of the mean function over time[24]), and a histogram of the standardised residuals together with a superimposed normal curve (Figure 2). No evidence was found that demonstrated that the assumptions of the final multivariable regression model had been importantly violated. Henceforth, we refer to this final multivariable two-level mixed-effects regression model as the final or refined model.

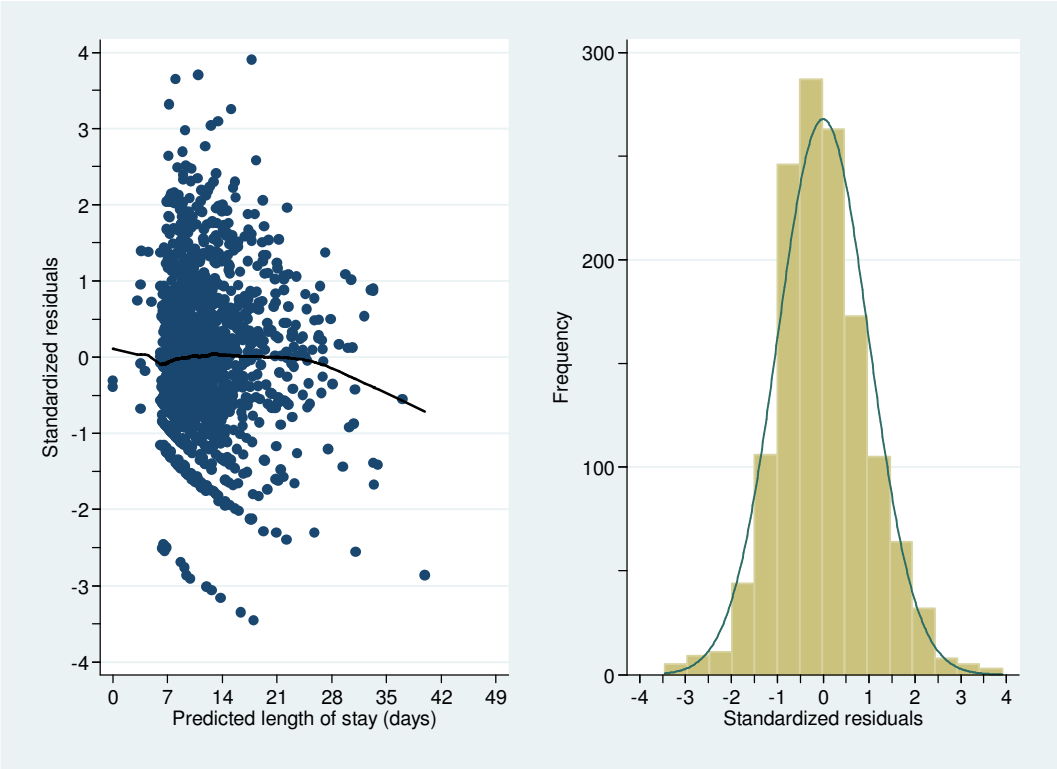
Predictive ability of final multivariable two-level mixed-effects regression model

Spearman's correlation between LOS and the final model's untransformed predicted values was ρ=0.38, a substantial improvement over the model using the TRISS variables and their original characterisations with ρ=0.31. A scatter-plot of the untransformed predicted LOS values from the final model and the observed LOS records appears in Figure 3. The absolute prediction error (APE) between untransformed predicted LOS from the final model and the actual LOS had a 25th

percentile of 3.3 days, median of 6.0 days, 75th percentile of 11.6 days, 90th percentile of 25.1 days, and 95th percentile of 35.9 days. This implies that for 50% of patients, the difference between the predicted LOS and the actual LOS was between 0 and 6.0 days.

The APE between untransformed predicted LOS from a regression model using the TRISS variables and their original characterisations and the actual LOS had a 25th percentile of 5.0 days, median of 10.8 days, 75th percentile of 22.0 days, 90th percentile of 38.0 days, and 95th percentile of 49.8 days.

Figure 2. (i) Scatter-plot of the standardised residuals for the final multivariable two-level mixed-effects regression model against the untransformed predicted length of stay values (in days) together with a superimposed lowess curve, and (ii) a histogram of the standardised residuals together with a superimposed normal curve



Penetrating mechanism trauma—The average age of the 52 blunt mechanism trauma survivors was 33 years (range: 16 to 74 years) and 43 (83%) were male. Unfortunately, a total sample of 52 is insufficient to produce a valid statistical model.

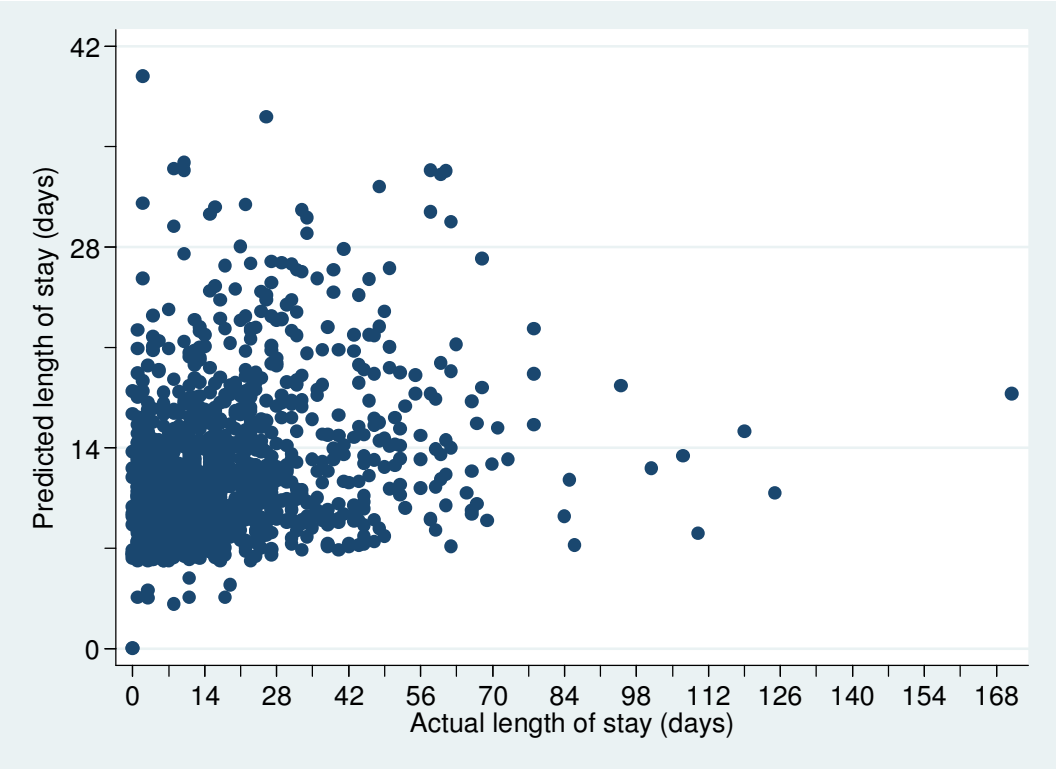
Discussion

All the TRISS variables were significantly associated with LOS for adults with blunt traumatic injuries. The original TRISS model had a moderate correlation with LOS

(ρ=0.31), which was improved when the variables were refined and re-estimated using a multivariable two-level mixed-effects regression model on these New Zealand data (ρ=0.38). However, the MAPE was 10.8 days for the original TRISS model and 6.0 days for the refined model.

So while the TRISS methodology provides a sound benchmarking tool for performance monitoring of trauma systems in terms of expected survival,[20] this study demonstrates that neither the original TRISS model nor a refined model based on the same TRISS variables provided adequate predictive capacity to allow it to be used to benchmark trauma systems in terms length of stay of non-fatal injuries. This finding is consistent with published work in the field.[11,13,14,25,26]

Figure 3. Scatter-plot of the predicted length of stay, untransformed from the final multivariable two-level mixed-effects regression model, against the actual length of stay observed from hospital records



Morbidity, the degree or severity of a health condition associated with the injury event, is also influenced by personal (such as personality, education, resilience), social (such as support, family, networks) and environmental (such as housing, location, employment) factors in which the person is situated.[27]

It may be possible to identify other issues about the personal, social and environmental characteristics of an injured person at the time of injury occurrence to insert into an appropriate statistical model that will better predict LOS. However,

much of this additional information is not routinely available in administrative data sets and would need to be included on the trauma registry data collection form.[12] Elicitation of additional patient information is likely to present a significant barrier to most registries worldwide.

Another important assumption used here and elsewhere is that LOS is a valid measure of injury-attributable health status. While there is face validity in using LOS as a measure of morbidity,[11, 16, 19] there are no studies reported in the literature that have formally explored the extent to which LOS is valid for use in this context (although LOS has been shown to predict functional outcomes 12-months post-injury[12]).

Moreover, as there is no international consensus on appropriate morbidity scores or the use of LOS in benchmarking non-fatal injury outcomes, there is considerable variability in how LOS is analysed and reported. For example, some treat LOS categorically as a binary variable, using a 5-day threshold,[11] a 7-day threshold,[19] or a 10-day threshold[16], whereas we treated the transformed LOS as a continuous variable. These different definitions make comparisons between studies and registries difficult.

While the study had salient strengths, including the large sample and sophisticated methods of statistical analysis, it also suffers from weakness. These include: some data quality issues, as described elsewhere[20]; the fact that data transformation was required, as two-level mixed-effects negative binomial regression models which might yield improved predictions are not currently available in Stata or SAS specialist statistical software packages; and there is potential inaccuracy of some LOS values for patients transferred to another hospital.

Currently, trauma registries in New Zealand do not track patients between hospitals; primarily because these registries operate independently and are hospital-specific. So there is no readily available mechanism to match de-identified patient records between hospital registries. As such, patients with hospital transfers and readmissions will be generally counted as separate admissions in each registry, each having a separate non-cumulative LOS value. However, given the median LOS for those with hospital transfers (10 days) was similar to those without transfers (11 days), the bias associated with this limitation is likely to be negligible compared to the poor overall predictive performance of the TRISS models noted in this paper.

Even if all these limitations were resolved, it is unlikely that the predictive capacity of the final model would improve substantially or sufficiently for its use as a prognostic tool. Another limitation is the lack of data available for the modelling of penetrating injuries.

Conclusion

Neither the original TRISS model nor a refined model based on the same TRISS variables has predictive capacity with sufficient utility for benchmarking LOS and, by proxy, morbidity. This New Zealand analysis adds to the consensus from the few studies conducted in the international literature that TRISS scores currently calculated in trauma registry software need to be supplemented by additional score(s) which enables performance monitoring of trauma systems in terms of expected outcomes of patients with non-fatal injury.[3,11,12]

Given that the vast majority of trauma patients survive and the substantial costs related to long term morbidity arising from non-fatal injury, it is imperative that trauma systems maximise their care to ensure the best possible outcomes are being achieved. There is an urgent need to develop a "threat to morbidity" and a "threat to disability" based injury severity score. It will be important to build these severity scores into the trauma audit and performance management systems of existing trauma centres and into the computer based trauma registries that support these audits. A substantial focus of research in this area is critically needed.

Competing interests: None known.

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Sodium fluoroacetate (1080): assessment of occupational exposures and selection of a provisional biological exposure index

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Abstract:

Aim Sodium fluoroacetate (1080) is used for control of vertebrate pests in New Zealand. Little is known about chronic effects in humans, but animal studies demonstrate potential for adverse fetal, male fertility, and cardiac effects. We aimed to employ analyses of 1080 to help assess the degree of exposure of bait formulators and distributors, and identify specific tasks where exposure reduction appeared most indicated. We also aimed to utilise the (limited) 1080 toxicity data to assess the significance of the analytical results.

Method Exposures during various activities were assessed by monitoring air levels and blood and urine concentrations. To help evaluate the results, a provisional "biological exposure index" (BEI) was later derived, by extrapolating from experimental data.

Results Early monitoring indicated exposures were highest in relation to (cereal) bait manufacturing and aerial carrot baiting procedures. A provisional BEI of 15 µg/L for 1080 in urine was proposed.

Conclusion Further protective measures and ongoing workplace monitoring are required, particularly in the above situations. Compliance with the current BEI cannot guarantee complete safety. Any information regarding chronic adverse effects in humans, along with the associated urine levels, would assist risk assessment. Further investigation of the human kinetics of fluoroacetate would be helpful.

In New Zealand, introduced vertebrate pests such as brushtail possums cause widespread damage to agricultural land and native forests. Possums are also the most significant wildlife reservoir for (bovine) tuberculosis, so control of their populations remains a priority. In some instances, the aerial application of the vertebrate pesticide sodium fluoroacetate (1080) in baits is considered the most cost-effective method for large-scale possum control operations.[1] However increasing concern has arisen over broad-scale application of 1080, particularly its wider impact on non-target species and possible adverse effects on the environment.[2] Concern extends to possible human risks, especially from aerial operations where contamination of waterways might occur.

While 1080 is highly toxic, the risk of acute human poisoning from environmentally distributed bait is considered low, because of the relatively low concentrations used in bait formulations (up to 0.15% w/w), the size and application rates of baits, and the location of baiting operations.

There are no well validated cases of "exploratory" 1080 poisoning in children in NZ—in contrast to the United States (US) decades ago, where 1080 was used in more concentrated form as a household rodenticide, with occasional severe or tragic consequences.[3,4] Intentional ingestion has also been responsible for serious human poisoning in other countries,[5,6] but very few proven cases have been reported in NZ.

Limited applications as a vertebrate pesticide in the US,[7] and elsewhere,[8] have meant that overseas regulatory requirements for toxicological data have been limited. Given the ongoing unique use patterns in NZ (and Australia), the need for an updated regulatory toxicology database was recognised[7] and relevant studies meeting internationally recognised protocols were commissioned. These included an *in vivo* developmental toxicity study, which estimated a "no observed effect level" (NOEL) of 0.1 mg/kg/day for teratogenic effects,[9] and a 90-day oral gavage dose study;[10] both in rats.

In the latter, a "lowest observed effect level" (LOEL) of 0.25 mg/kg/day was reported, where the heart weight was increased, and (in males) effects observed in the epidiymides and testes, with statistically significant adverse changes in some sperm parameters. A daily dose of 0.075 mg/kg/day was the "no observed effect level" (NOEL) for both these types of effects.[10] A similar, unpublished study of subchronic effects produced a NOEL estimate of 0.05 mg/kg/day for cardiotoxicity and male gonadotoxicity.[11]

Oligospermia and/or aspermia were noted in mink ingesting 0.08 mg/kg/day,[12] suggesting a general mammalian effect on testicular and epididymal function. It is clear both the testis and heart are sensitive target organs; indeed adverse cardiac effects were recognised in livestock from plant sources of fluoroacetate prior to its usage as a pesticide,[13,14] and later studies provided further evidence.[15]

The mechanism underlying these chronic effects are not well established. However it is likely its impairment of aerobic metabolism (due to inhibition of the citric acid or Kreb's cycle), largely responsible for acute 1080 poisoning, is a significant factor. Cellular hypoxia is a recognised cause of adverse cardiac, fetal, and testicular effects.[16,17] Some fluoroacetate is converted *in vivo* to fluorocitrate, which plays a major role in disrupting the citric acid cycle,[8] as well as risking hypocalcaemia.

Given clear laboratory evidence of sublethal effects of oral exposure, there is obvious concern regarding risks of similar effects from human exposures. Pest control industry workers engaged in the preparation and distribution of 1080 baits are the group most likely to be repeatedly exposed, and this paper outlines the initiation of protocols for monitoring such workers in NZ. Our aims were to employ analyses of 1080 as a tool to help identify hazardous situations, where exposure control measures were most indicated.

Here we report the earliest analytical findings, and our subsequent efforts to interpret these in terms of their possible risk, in the face of little previous human data. This involved establishment of a provisional "biological exposure index" (BEI) for 1080, in response to a request for a specific "action" criterion by the regulatory authority.

(A BEI generally indicates a concentration in a biological fluid below which it is considered nearly all workers should not experience adverse health effects from a chemical. Historically, it has often been based on a "threshold limit value" (TLV), which is an average air concentration under which it is believed nearly all workers may be repeatedly exposed (e.g. for 8 hours, 5 days a week) without such effects. The term "workplace exposure standard" (WES) has also been applied to an air level similarly designed to protect workers from the adverse effects of long term exposure).

Method

Identification of subjects and sample collection—After providing information to the pest control industry regarding the proposed monitoring programme, and gaining ethics committee approval (Southern Regional Health Authority Ethics Committee Otago, No. 98/11/088), volunteers from various occupational groups within the industry were obtained. They were classified into groups; workers involved in 1080 cereal bait manufacture at either of two sites (n=9), and applicators involved in either of two (separate) aerial carrot bait (n=9) or aerial cereal bait (n=11) operations. This provided a diverse, if relatively small sample. The details are outlined in Table 1.

Table 1. Monitoring of 1080 operators: work activity, sample type, and timing; numbers sampled

Bait manufacture (*n*=9; 6 at Site 1 and 3 at Site 2)						
Day	***Blood***		***Urine***		***Air***	
	am	pm	am	pm	am	pm
1	9	9	–	9	2	6
2	–	–	–	9	–	6
3	9	9	–	9	2	2
4	–	–	–	9	–	–
5	9	9	–	9	–	–
8	9	9	–	9	–	–
Aerial carrot baiting (*n*=9; 5 at site 1 and 4 at site 2)						
1	9	8	–	8	–	6
2	4	3	–	6	–	–
3	4	3	–	–	–	3
4	–	–	–	5	–	3
5	4	4	–	8	–	–
6	–	–	–	4	–	–
Serial cereal baiting (*n*=11; 6 at Site 1 and 5 at Site 2)						
1	10	10	–	10	–	5
2	5	5	–	1	–	4
3	–	–	–	1	–	–
4	5	4	–	5	–	–
5	5	5	–	5	–	–

Measurement of air levels was targeted to clarify specific tasks presenting inhalation hazards. Workplace contamination at sites of bait manufacture and application was monitored by measuring the concentration in respirable airborne particles <10 µm in Stokes diameter. Gilian air pumps, with intake close to the face, were worn on at least 2 separate days. They were set to sample ~ 2 litres per minute volumetric flow rate- and calibrated at the start and end of each sampling.

The average pump flow rate and sampling duration were used to calculate the total volume of air drawn through each cartridge, and the average air concentration (µg/m^3) was calculated from the mass of 1080 collected on the air filter (0.8 µm mixed cellulose ester filter) divided by the above volume.

Monitoring for larger 1080 particulates was also carried out, as these may be deposited on horizontal surfaces, with risk of inadvertent hand to mouth contact. Swabs were taken from representative sites on the stair and platform rails, desk, taps, and toilet door handle on 2 days during each site visit using the air filters moistened with methanol which were wiped over a small area using a gloved hand.

Blood and urine tests for 1080, being biomarkers of absorption from all relevant tasks and exposure routes, were also undertaken. Blood samples (10 ml) were collected at the start and end of working days (as shown), by professional (Medlab) staff. Plasma were collected, held at 20ºC, and (along with urine samples) delivered to the Landcare Research laboratory for analysis. Total urine for the working day (approx. 12 hours) was collected every day for 6 days, or the length of the field operation. Urine was collected in a 1000-ml sample bottle whenever toilet visits were made.

Analysis of samples for 1080 concentrations—Testing was conducted using a gas chromatography method developed by Landcare Research and based on the work of Ozawa and Tsukioka.[18,19] Plasma (after precipitation of the protein with acetonitrile) and urine (or water) samples were added to 2% sodium chloride solution for derivatisation. The 1080 in aqueous extracts or urine samples was acidified with hydrochloric acid and converted to the dichloroaniline derivative with N,N' dicyclohexylcarbodiimide (DCC) and 2,4 dichloroaniline (DCA) using ethyl acetate as solvent.

The derivative was cleaned on a silica solid-phase extraction cartridge to remove excess derivatising agent, eluted with toluene, and quantified by gas chromatography on a BP-5 capillary column with electron capture detection. The method limit of detection (MLD) was 0.006 µg/ml in blood and 0.0005 µg/ml in urine. The air filter and swab samples were eluted with 50 ml 2% saline solution, then prepared as for a water sample, with a MLD of 0.005 µg 1080.

Initial defining of exposure categories—Initially, three levels of personal exposure were defined (Table 2), on the basis of 1080 concentrations found (or not) in blood and urine.

Table 2. Initial classification of exposure levels

Classification	Measured 1080 concentration in urine and blood
Level 1	below detection limits in blood (<0.006 µg/mL) and urine (<0.0005 µg/mL)
Level 2	not detectable in blood (<0.006 µg/mL), and also <(0.02 µg/mL) in urine
Level 3	blood level ≥(0.006 µg/mL), and/or urine level ≥(0.02 µg/mL)

Derivation of a biological exposure index (BEI) for occupational exposure to 1080—It became clear that a formal, "transparent" guideline value was needed, to more critically evaluate the significance of individual biological monitoring results. A value based on urine sampling was preferred, due partly to its easier detectability than in blood, and greater worker acceptance.

However there is little human data shedding light on what an appropriate biological exposure index would be. One report outlines an incident of excessive exposure from misapplication of 1080 as rat poison in a steel mill, with the generated dusts causing relatively high acute exposures, with several workers becoming seriously ill.[20]

There is also a report of salivation, visual disturbance, paresthesiae, convulsions, and coma after a wind gust blew concentrated powder into one worker's face.[21] However no data were found on chronic, low level human exposures which could indicate minimum daily toxic doses, or the corresponding urine concentrations. Therefore, animal data were utilised to derive an estimated acceptable daily exposure to 1080.

Firstly an appropriate NOEL estimate for 1080 (in rats) was identified; while the lowest reported figure was 0.05 mg/kg/day,[11] the value of 0.075 mg/kg/day, from a more recent study[10] meeting current internationally recognised protocols, was chosen. Then a safety or uncertainty factor (SF, UF) of 10 was applied, in effect allowing for the possibility of a 10-fold greater susceptibility of humans relative to rats. Lastly, the need for further UFs was considered.

In public and environmental health contexts, regulatory agencies routinely incorporate extra UFs, including factors to account for limited data sets and relative lack of chronic studies specifically, which circumstances both applied in the case of 1080.Thus the minimum (mammalian) study requirements

are two chronic, one multigeneration reproductive, and two developmental toxicity studies; so the lack of chronic (lifetime) 1080 studies was a major reason for its adjudgedly inadequate database.

On the other hand, a major purpose of lifetime studies is to assess carcinogenicity, but three complementary toxicity studies had previously indicated that 1080 is not mutagenic, and therefore "not anticipated" to be a carcinogen,[10] or at least not a genotoxic one. Therefore an extra safety factor (of 3) was applied to the chosen NOEL, to derive a so-called reference dose (RfD).

Some approximations and assumptions were then made regarding the likely kinetics of 1080 in humans, to determine the approximate urine concentration to be expected with daily intake of the RfD. The relevant calculations are presented in the Results section.

Results

Air, blood and urine monitoring

Personal air samplers generally showed low levels of airborne 1080 during bait application, with maximum concentrations of 5.54 and 5.62 µg/m^3 respectively for the two aerial carrot operations. Maximum concentrations for the aerial cereal bait operations were lower, at 0.8 and just 0.09 µg/m^3 respectively. However much higher levels were found at times at the factories involved in bait manufacture.

At both sites, a brief but high exposure (~1.9 mg/m^3 over 18 minutes, ~3.5 mg/m^3 over 36 minutes) while weighing out 1080 powder (of technical concentration at least 97%), was sufficient to exceed the NZ workplace exposure standard (WES) of 0.05 mg/m^3 as an 8-hour time weighted average (TWA),[22] even without adding the (much lower) exposures over the rest of the day. However, as these workers were wearing a respirator during the weigh-out period, their effective exposure (inhaled amounts) would have been within that permitted by the WES.

Surface swabs from one cereal bait manufacture site showed a clear relationship to when powder was handled. With the bait distribution operations, less detailed data was obtained (air monitoring was intended primarily to compare the two bait types and to "scope" whether any specific tasks were associated with high airborne levels).

Table 3 summarises the blood and urine results (classified as per Table 2); the maximum concentration found in samples from each worker was used to categorise their 'level' of exposure, with no indication of how many of their samples contained no detectable 1080. Of the nine workers involved in bait manufacture, three had a level 3, and six had a level 2 exposure, detected in at least one sample. Of the nine monitored during aerial carrot baiting operations, four had a level 3 exposure and five a level 2 exposure, on at least one occasion. This latter occurred despite the relatively low air levels noted. Workers in aerial cereal baiting had lower exposure profiles; six having level 2 and five level 1 (no detectable) exposure.

Overall, level 2 exposures were the most common (comprising ~60%).

Table 3. Levels of exposure: monitoring results by occupational activity

Occupational activity	Exposure levels (subject numbers, by site)					
	Level 1		Level 2		Level 3	
	(Site 1)	(Site 2)	(Site 1)	(Site 2)	(Site 1)	(Site 2)
Aerial cereal baiting	2/6	3/5	4/6	2/5	–	–
Aerial carrot baiting	–	–	3/5	2/4	2/5	2/4
Bait manufacture	–	–	4/6	2/3	2/6	1/3

Calculation of a biological exposure index

Derivation of reference dose

- Chosen NOEL: 0.075 mg/kg/day[10]
- Selected total UF or SF: 30 (comprising factors of 10 and 3)
- Therefore derived reference dose (RfD) was 0.0025 mg/kg/day

Estimation of urine concentration expected from daily intake of reference dose— A biological exposure index (BEI) was derived by estimating the likely minimum urine concentration (as a 24-hour average) to be expected following daily intake of the reference dose. The urinary level will depend not only on that dose and body weight, but also the percentage of 1080 excreted in urine and the daily urine output. The steps involved in calculating urine concentrations corresponding to the reference dose are outlined below. (This approach is based on complete absorption of the RfD.)

- Acceptable daily ("reference") dose (RfD) = 0.0025 mg/kg/day
- Acceptable daily urine excretion = RfD × Wt (kg) × (fraction or %ge excreted in urine; F_e)
- Acceptable average urine concentration, C_u = (RfD × Wt × F_e) / (24-hour urine volume, V_{24})
- Therefore general calculation for the mean urine concentration to be expected from the RfD is:
- C_u (mg/L) = 0.0025 mg/kg × Wt (kg) × Fe / V_{24}

The result will depend on individual values for Fe, V_{24}, and body weight. Using the fairly conservative figure of 60 kg for an adult weight, Table 4 indicates the derivation of the selected value.

Table 4. Urine levels (mg/L) corresponding to RfD as modified by urinary excretion and flow

Urine output	Fractional urinary excretion of (unchanged) 1080 in 24 hours (F_e)				
(24 hr, V_{24})	20%	30%	40%	50%	60%
1.5	(0.02)	(0.03)	(0.04)	(0.05)	(0.06)
1.7 L	(0.0175)	(0.0265)	(0.035)	(0.044)	(0.053)
2 L	**(0.015)***	(0.0225)	(0.03)	(0.0375)	(0.045)
2.5	(0.012)	(0.018)	(0.024)	(0.03)	(0.036)

Unfortunately, no data was located shedding light on the typical elimination half life of 1080 in humans, nor, more specifically, the fraction likely to be excreted in urine over any given time. However various experimental data (discussed below) led us to believe that at least 20% of an absorbed dose of 1080 will likely be excreted in urine in unchanged form within 24 hours. Employing also an estimate for daily urine output of 2 litres in adults, it was thus calculated that the average urinary concentration in a 60 kg worker with a daily dose of 0.0025 mg/kg would likely be at least 0.015 mg/L, and this value was selected as a provisional BEI.

Discussion

These monitoring programmes were limited by the relatively small numbers of workers involved, and the self-selected nature of the "sample". (The age, ethnicity and gender mix of volunteers was not examined, partly to preserve confidentiality, and their degree of representativeness of the wider pest control workforce cannot currently be assessed.) There was also limited longitudinal monitoring and tracking of individual workers. However there was sufficient data and consistency therein to identify the more hazardous operations (and occasionally, specific tasks).

Early monitoring indicated a need for improvement in aspects of the cereal bait manufacturing environment and in aerial carrot baiting procedures. The latter (at preparation stage) involves dipping carrots into concentrated solutions of 1080, with risks of splashes to the face and other skin areas; it was not uncommon for operators to be wearing contaminated, damp clothing for prolonged periods.

In contrast, cereal baits were prepared at the formulating factories, and products handled by the "downstream" user contained low levels of 1080. Further monitoring indicated aerial carrot baiting operations remained a significant exposure source, despite increasing use of long water proof gloves and face shields, (and masks in the majority). Likely contributory factors included inconsistent use of and/or substandard protective equipment, and individual hygiene practices (including possible inadvertent urine contamination during sample collection).

It was thought level 1 exposures were unlikely to be harmful, given no detectable 1080, and that level 2 were of relatively low risk, as none was detectable in blood, but the significance of the urine levels became a little more clear after derivation of the BEI.

A BEI should ideally be set by consideration of relevant data relating to the human health experience with chronic exposure. Indeed the most usual, if not always ideal, means of establishing a BEI has been from the threshold limit value (TLV) in air, itself usually derived directly from human experience. However, there was insufficient human data to enable this.

The American Conference of Governmental Industrial Hygienists (ACGIH), in the documentation of its TLV (adopted in NZ as a WES) does not outline in (quantitative) detail any underlying rationale based on human exposures, and while it states the selected TLV should minimise the risk of acute systemic toxicity, it does not explicitly extrapolate from any chronic exposure data.[23] Similarly, LaGoy et al (1992) described excessive exposures following a steel mill contamination incident, but this

resulted in several workers becoming seriously ill, and the report does not shed light on possible more subtle effects from lower level longer term exposure.[20]

Thus neither of these sources are highly informative in terms of establishing a BEI. However it is reassuring to note that our "reference dose" of 0.0025 mg/kg/day is just half that utilised by the above authors in their development of suitable cleanup levels following the mill incident.[20] Similarly, it is somewhat more conservative than the TLV (WES) of 0.05 mg/m^3, in that a 70 kg worker under moderate work load inhaling 10 m^3 of air per work day at the WES would have a respiratory intake of 0.5 mg or ~0.007 mg/kg/day. Moreover, unlike the WES, it also "incorporates" potential doses arising from skin absorption or inadvertent "hand to mouth" ingestion, as opposed to just inhalation.

(Dermal absorption though would seem relatively low, given large disparities between experimental median lethal doses (LD_{50s}) when administered orally versus dermally. Thus summary data[24] indicate dermal LD_{50} values 480 and 253 times higher than oral LD50 values for rats and mice respectively. However these come from different studies and may not be completely comparable, and there was only a 5.3-fold difference between cited dermal and oral LD_{50} values for guinea pigs. Prolonged contact with liquid formulations, or solubilisation of dusts by sweat, could increase absorption, particularly through compromised skin).

Basing a BEI on urine rather than blood levels has advantages besides greater worker acceptability. Urine levels are slower to decline, and hence less likely to "miss" significant exposure, so timing is a little less critical. Additionally, for many industrial compounds, a blood level gives little indication of the daily exposure ("dose") giving rise to it, while the amount excreted in urine is potentially useful for roughly estimating daily (absorbed) doses, though this is more so for compounds with substantial, quantified, prompt urinary excretion in their unchanged form.[25]

This is significant, because of our need to utilise data from experimental animals (given lack of human data). The standard approach in such situations is to extrapolate from adjudgedly safe daily doses in animals to estimate likely safe doses in humans. This is more enshrined than extrapolation from an experimentally derived no-effect blood level to a safe human blood concentration.

However there are several potential imprecisions in this process, including the selection of appropriate safety or uncertainty factors. This approach is well established in public health regulatory practice, where a safety factor (of typically 10) is employed for inter-species extrapolation, but also other precautionary factors are applied, generally in conservative fashion. These include factors to take into account intra-species (human) variation in susceptibility (often 10), an incomplete overall database (up to 10), and to extrapolate from a subchronic to a chronic exposure (up to 10).

Indeed in the regulatory context, the US EPA has applied a total uncertainty factor of 3000 to their chosen NOEL (0.05 mg/kg/day) for 1080, comprising factors of 10 for all but one of the above considerations, the incomplete database, where instead a factor of three was used to adjust for lack of reproductive/developmental studies and toxicity studies in a second species.[26] The same general approach has been taken in

New Zealand in setting a drinking water standard,[27] and further lowering has been proposed., based on a total UF of nearly 3000.[28]

However a formal "safety factor" approach is not typically used in occupational health practice, though when the concept has been employed, emphasis has been on a factor of ten for inter-species differences, with less expressed need for a safety factor for intra-species variability, given the presumed exposure of healthy, working age adults only. Therefore a total UF of 3000 was not considered necessary or appropriate. Having said that, it could be argued that the total UF should be more than 30. It was chosen to limit it to this figure for the time being, partly to ensure reasonable achievability of compliance in the medium term, and not too large a "mismatch" with the WES. However, the proposed BEI should be regarded as provisional and subject to ongoing review.

This is doubly so as the estimate of urine levels corresponding to the RfD also has elements of uncertainty and imprecision. Certainly the chosen figure of 20% for the percentage excreted (as unchanged 1080) in urine over the first 24 hours is a rough estimate (or "guestimate") based on extrapolations from experimental animal data.

The kinetics of fluoroacetate, including its half life, are not well established in humans.[6] Blood levels have not been employed in management of acute poisoning cases, as the test is time consuming and of little clinical utility.[5] Two recent reviews discuss aspects of its toxicokinetics, but indicate there is very little quantitative human data.[29,30] Our monitoring programmes focused on the maximum blood and especially urine levels in individual workers as markers of their "worst case" exposure, rather than the details of rate of change of these parameters, such as to estimate elimination half lives. In the event of opportunity for further study, more detailed kinetic analyses involving multiple serial blood levels could be considered, subject to ethical approval.

Neither is there human data on the fractional elimination of (unchanged) fluoroacetate in urine over time. Relating our raw data on an individual's urine volume to their measured 1080 concentrations in 12-hour urine would provide interesting information on the amount excreted, but this would not provide a fractional elimination estimate without knowledge of the (absorbed) dose.

However in most if not all mammalian species thus far tested, estimated mean plasma elimination half lives have been less than 12 hours. Estimates include 10.8 hrs (sheep), 5.4 hrs (goats), ~1.6 hrs (mice), and 1.1 hrs (rabbits).[31,32] Further, in several studies involving various species, 1080 has been found at higher (or similar) concentrations in plasma than in major organs, including kidney, muscle, heart, liver, and spleen.[31,33,34] In sheep, plasma levels were about twice skeletal muscle levels at 2.5 hours, and levels were virtually the same (and very low) at 96 hours.[31]

Therefore it seems plasma concentrations represent a "worst-case persistence profile"[31] or a conservative indicator of changes in whole body load. Hence it would appear that in the species tested (whose 1080 in plasma half lives were all <12 hours), at least 50% of a dose could be eliminated within 12 hours, and 75% by 24 hours. There is no specific reason to believe the situation in humans is different.

However, experimentally, the percentage excreted in unchanged form in urine is less clear. Eason et al[31] found that in (three) sheep, ~7.5% to 14%, was excreted in "pure" urine samples over the first 24 hours, (and the fraction ranged up to ~34% by 72 hours

when urine contaminated faeces was included). In rats, one study found up to 17% unchanged in urine over 2 days.[33] However, while Gal et al collected 30–35% of a radiolabelled dose from urine within 2 days, the "unchanged" percentage was 60% by 4 hours but only 7.3% of the total 24-hour collection.[35]

These studies involved one-off (sometimes high) doses as opposed to chronic dosing. However a comparison between our human data (on urine levels versus blood levels) and that of a 90-day experimental study[10] suggests that the fraction eliminated unchanged in urine could be higher in humans than rats.

In any case, the more recent data on sheep were considered more relevant to humans. Therefore, we considered a figure of 20% for 24 hour urinary (unchanged) excretion is unlikely to be a significant over-estimate for humans. However we recognise that animal studies are not an ideal basis for establishing a BEI.

Regarding an optimum time for urine testing, it is likely (given experimental kinetic data) that the end of the work day would be preferable to the beginning of the next, and certainly the end of a work "week" preferable to after a weekend. We collected total urine for the working day (~12 hours), but any subsequent programme might involve comparing spot levels at the above times.

Air levels during baiting operations were generally low. However those brief periods of handling concentrated powders during manufacturing are cause for concern. This is particularly so as (unfortunately) only respirable levels were measured, not total inhalable levels, which latter may have been substantially higher.

We have not examined in detail the correlation between personal zone air levels and urine (or blood) levels, partly because this could be misleading, given that operators (particularly in the factories) were often wearing respirators at the most hazardous times, so were not effectively exposed to the air levels measured in their breathing zones, which thus would have little or no impact on their urine levels. However with tasks that had not typically been associated with respirator use, any such correlation would be interesting to explore, (though the TLV for systemic toxins is based on inhalable, not just respirable levels).

Conclusions

Our initial findings indicated there remained a need for further improvements in exposure reduction and ongoing monitoring. The derived BEI should be regarded as provisional and subject to ongoing review in the light of further information, particularly any monitoring results relatable to demonstrable adverse health effects in humans from chronic, low level exposures. Further, the aim should always be to reduce toxic exposures (and markers thereof) to as low a level as possible. The kinetics of fluoroacetate in humans also requires more investigation (though kinetic studies in otherwise non-exposed volunteers are not advocated).

Subsequent to the monitoring reported here, further programmes were undertaken, where the provisional BEI was first used as an "action level," with workers exceeding this value being temporarily suspended while work practices were reviewed and urine tests repeated.

Some progress in exposure reduction was achieved. However these programmes did not include detailed clinical appraisal, and (while there is no clear evidence of harm), more extensive health surveillance should be considered in those exposed long term, given the limitations of our proposed BEI as an indicator of risk. This might include questionnaires assessing fertility parameters,[36] though low subject numbers limit the power of such studies, whose methodology is still evolving.[37]

In 2007, in response to health and ecological concerns, the NZ Environmental Risk Management Authority (ERMA) undertook a reassessment of the role of 1080 in pest control management. The ERMA identified a need for further improvements, including a tightening of mandatory controls, closer monitoring, and further research into its adverse effects.[38]

Since then, there has been increased development and availability of "how-to" guidelines for occupational monitoring of 1080,[39] facilitating the increasing adoption of such monitoring as a routine. It is pleasing in 2009 to hear that at least in some sections of the industry, progress has continued to be made in controlling exposures, to such a degree that workers' urine levels are typically below the lower limit of detection of the test (which at 0.0005 ug/mL, is 30 times lower than our provisional BEI). In such cases adverse effects would be extremely unlikely.

Competing interests: None known.

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THE NEW ZEALAND MEDICAL JOURNAL
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A bizarre case of drowning as a result of pandemic influenza (1918 pandemic)

Nick Wilson, Jennifer Summers, Michael Baker, Geoff Rice

We present a historical case of a New Zealand soldier on a troop ship in 1918 who contracted pandemic influenza, became delirious, "jumped overboard", and drowned. Some lessons from this case of current relevance are discussed.

Case report and outbreak context

The case was that of a private in the New Zealand Expeditionary Force whose records are publicly available, as are all World War One participants from New Zealand (http://muse.aucklandmuseum.com/databases/Cenotaph/RecordDetail.aspx?OriginalID=2351&SearchID=7638822&Ordinal=1). He was travelling aboard the troop ship *Tahiti* (Figure 1) which was off the coast of West Africa en route from Wellington to Plymouth, England.

Figure 1. The HMNZT 107 *Tahiti* in Wellington Harbour ca 1914–1919 (National Library of New Zealand Reference number: 1/2-014597-G)

In an archival report[1] it was noted that during an outbreak of pandemic influenza, a soldier with influenza became delirious, "jumped overboard", and was lost at sea (presumed drowned) on 4 September 1918. Another archival document stated that this particular individual was "missing at sea" on this date.[2]

There was no doubt amongst the medical staff onboard the vessel and in the subsequent Official Enquiry, that the outbreak on the *Tahiti* was that of influenza.[1] Indeed, an outbreak of "fever" was reported to be circulating in Freetown, Sierra Leone (where the *Tahiti* stopped to take on coal and to meet other ships to form a convoy). Also other ships in the convoy reported influenza outbreaks around this time with significant mortality.[1]

Furthermore, the first cases of the more virulent second wave of the 1918 influenza pandemic in West Africa occurred in Freetown, Sierra Leone (on 22 August 1918)[3] and this site was one of the three major distribution points for this second wave.[4]

Discussion

From a clinical perspective, altered mental state and other neurological complications are known features of influenza infection. Indeed, for the current (2009) pandemic of a novel Influenza A(H1N1) such features have already been described in the literature (e.g. confusion, disorientation, and even seizures).[5]

In terms of this being a potentially preventable death, there are two aspects which suggest this possibility:

- Firstly it is reasonable to suggest that the spread of infection to the *Tahiti* from either a known outbreak of fever onshore or from another ship in the convoy, could have been prevented. For example, this could have been achieved by ensuring that the provision of coal for the ship by local workers was done with no close contact with those onboard. Also preventable could have been the spread of pandemic influenza from a meeting of officials from the various ships in the convoy on another ship (name unspecified) in Freetown's harbour.[1] This meeting was attended by officials from the *Mantua*, which was the only other ship reported to have influenza cases whilst in Freetown[1], and was probably the ship which had brought the pandemic influenza to Freetown from England.
- Secondly, the soldiers who were sick and delirious could have been better supervised and located further away from the decks, especially after a previous case of a delirious man going overboard (but who was reported to have been rescued).[6]

Another aspect of this case highlights a problem with researching past pandemics. That is in the Official Roll-of-Honour records for World War One, the online Cenotaph record (as per the URL above), and the archival casualty records[2] state that this individual either "drowned" or was "missing at sea", with no mention of influenza. Only when an archival source was consulted,[1] was it possible to identify this case as being an influenza-related fatality.

Additionally, the outbreak of pandemic influenza on this ship highlights a research deficit in that no modern epidemiological analysis to describe this outbreak (and identify risk factors) has been published to date. This is despite very high levels of morbidity and 77 deaths resulting. Indeed, it appears to have been the worst maritime disaster in New Zealand's history since the 1881 shipwreck of the *Tararua* (n=131 deaths).[7]

The inadequate research around past influenza pandemics has been raised as a concern previously.[8,9] Nevertheless, we are currently investigating this outbreak and other (previously unstudied) pandemic-related deaths of many other New Zealand soldiers in 1918.

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THE NEW ZEALAND MEDICAL JOURNAL
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Cutaneous myiasis due to *Dermatobia hominis* (the human botfly) in a New Zealand traveller returned from South America

Simon C Dalton, Stephen T Chambers

Increasing adventure travel can result in the import of unusual illnesses to the traveller's home country. A case of cutaneous myiasis (infestation by fly larvae) is presented, along with an overview of the management of the condition.

Case report

A 15-year-old male returned from an overseas holiday to Peru and Bolivia. One month prior to presentation and shortly after a 2-week stay in the Amazon Basin the patient noted an intensely itchy lesion on his left shoulder. This was presumed to be a mosquito bite, as he had noted innumerable mosquitoes in recent weeks. The lesion repeatedly discharged a bloody serous fluid.

Over the course of a further 2 weeks it became slightly larger and erythematous. The patient self treated with a short course of amoxicillin for presumed bacterial infection of the bite with no effect. He remained systemically well, with no fever or constitutional symptoms. The nonhealing nature and the continual discharge prompted medical attention on return to New Zealand. There was no other history of note. His parents and sibling who undertook the same journey were well. He remained on antimalarial prophylaxis.

Examination of the patient demonstrated a single slightly erythematous lesion on the posterior aspect of the left shoulder with a central pore through which discharged serosanguineous fluid (Figure 1). There was clear movement within the lesion and intermittent protrusion of a small white structure from the central pore. A diagnosis of cutaneous myiasis, most likely due to *Dermatobia hominis* (also known as the human botfly) was made.

With firm sustained pressure over 30 seconds a single live larva was completely expelled (Figure 2) without the need for forceps. It bore the characteristic morphological features of *D. hominis,* and was subsequently formally identified as a second instar (developmental stage) example, measuring 10 mm (Figure 3).

Figure 1. The presenting skin lesion: the central pore is filled with serosanguineous fluid

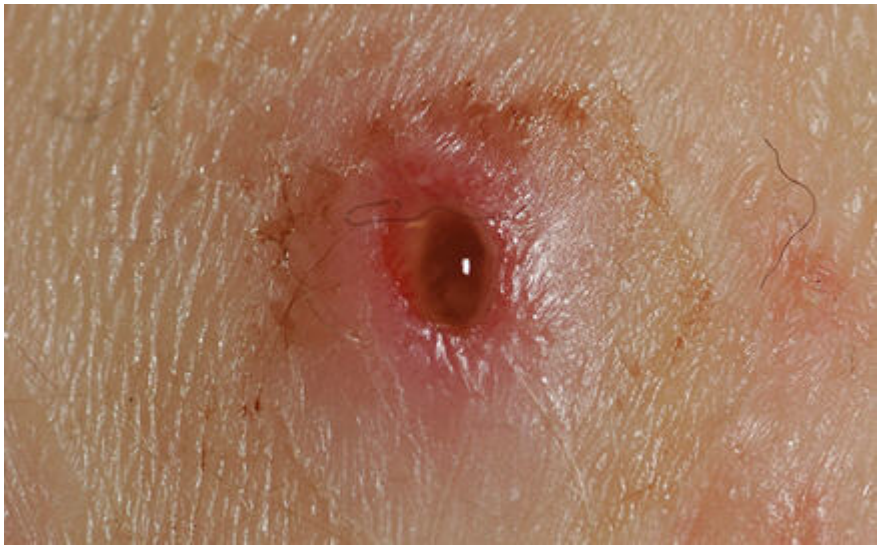


Figure 2. Removal of the larva with firm sustained pressure

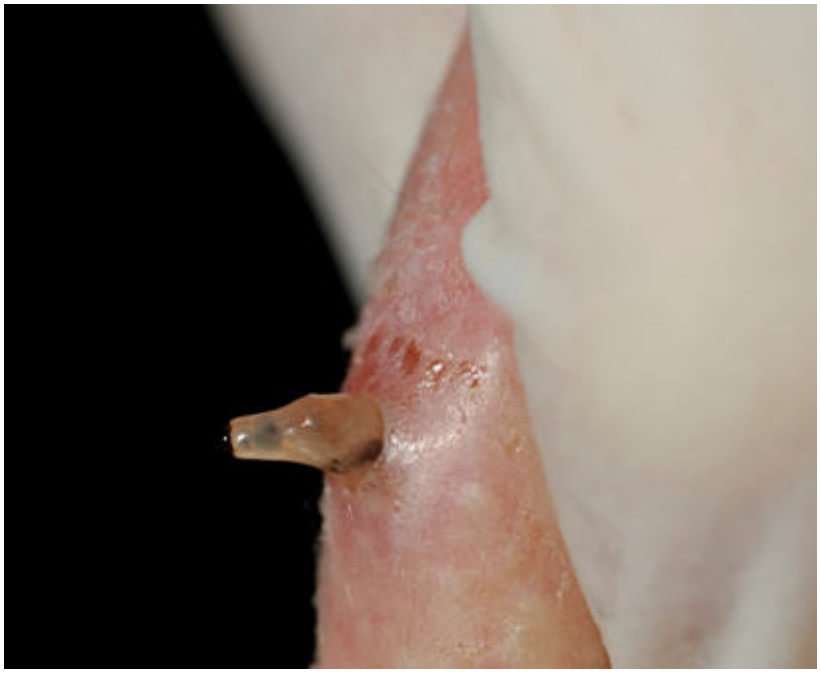
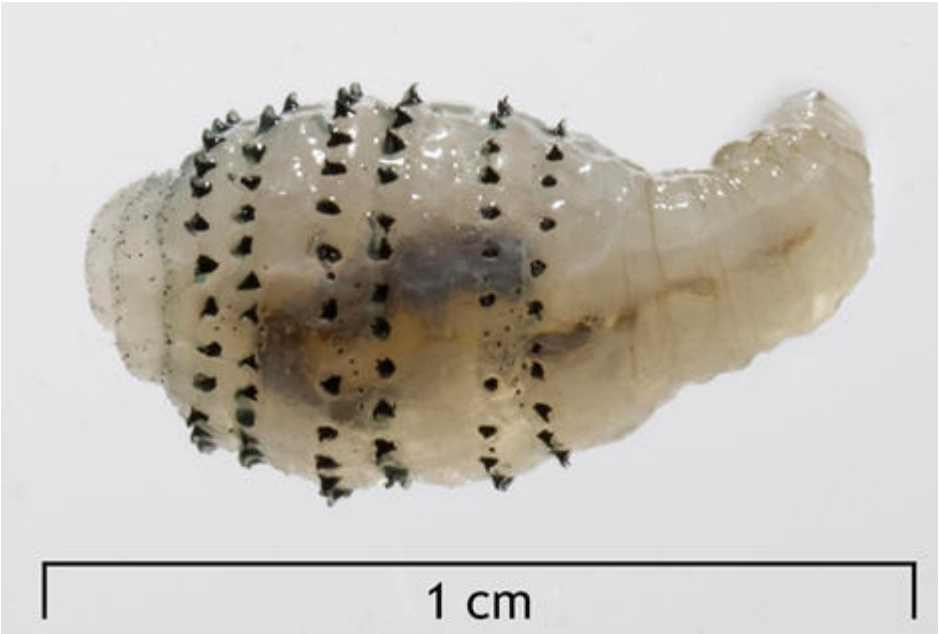


Figure 3. Following complete removal, a 10 mm example of *Dermatobia hominis* (the human botfly) is identified



Discussion

D. hominis is endemic through Central and South America from Mexico (~26° N) southward through all countries except Chile to the northern districts of Argentina (~32° S).[1]

The adult fly is rarely seen due to its unique lifecycle: the adult female attaches her eggs to the body of another insect for carriage to the host. Large mosquitoes are often used but many other species of insects and ticks have been reported to be egg carriers.[2] When the carrier alights on a mammal, the eggs hatch immediately and the larvae enter the skin through the bite of the carrier, some other small trauma or unbroken skin.

The larvae remain in the skin, aided by parallel rows of posterior-directed spines (Figure 3) and continue to develop over 4 to 14 weeks, reaching up to 20 mm in length.[3] Whilst in the skin, each larva breathes through a small opening or pore (Figure 1) which they also use to dispose of their serosanguineous faeces.[4] Eventually the larva emerges from the skin and drop to the ground, where pupation occurs before an adult fly is released.

Important clues to this unusual clinical diagnosis are a detailed travel history, ineffectiveness of antibiotics,[1] and a furuncular-like lesion with a central pore draining serosanguineous fluid.[5] The sensation of movement within the lesion, accompanied by pain but little tenderness or inflammation, also suggests myiasis.

The tip of the larva may protrude from the central pore, and bubbles produced by its respiration may be seen.[2] The majority of lesions are found on exposed skin, although other sites including the eye and external genitalia have been described.

Given its gross appearance, myiasis is commonly misdiagnosed as an infected bite or sting. Other differential diagnoses include infected epidermoid cyst, tungiasis (infestation by the burrowing flea *Tunga penetrans*), and cutaneous larva migrans.[1] It should also be differentiated from cutaneous leishmaniasis and onchocerciasis, which have a different prognosis and treatment.[2]

The most important complications in humans are bacterial superinfection and tetanus.[1] Curative treatment involves removing the entirety of the larva, and many methods have been described. In general they involve a combination of manual removal, occlusion, or surgical exploration.

Firstly, removal can be attempted with firm sustained manual pressure on both sides of the lesion to force the larva up and outward. Toothed forceps can be used to provide gentle traction if required. During this procedure it is important to grasp the larva well down its length as the breathing tube is fragile and breaks easily.[6] A slight enlargement of the pore may sometimes be needed.[1,7]

Occlusion of the central pore—with a variety of substances including petroleum jelly (e.g. Vaseline), nail polish, bacon, tape, wax—has been previously described.[4–7] This creates a hypoxic environment and encourages the larva to move to the surface for respiration. This can take minutes to many hours[5,7,8]. Removal can then be attempted as previously outlined. In some cases occlusion may result in asphyxiation of the larva without inducing it to emerge. Occlusion therapy also becomes less effective in the later stages of infestation.[7]

In situations where conservative methods of larval removal have failed, use of a snake venom extraction device has been described as rapidly effective.[9] In general however, if unsuccessful with the above techniques a surgical exploration under local anaesthesia is indicated.

Whichever method is used, care should be taken not to break or rupture the larva. This may cause a strong inflammatory reaction, often followed by secondary infection.[2] Complete removal is critical for the same reason.[7]

Patients travelling to Central and South America should be advised to use both effective insect repellents and protective clothing to prevent mosquito bites, and thus infestation by *D. hominis* larvae.[5]

Finally, it is worth noting that the larvae of the Tumbu fly (found in tropical Africa) causes a similar form of myiasis, usually by contaminating clothing whilst drying.[4] Methods of removal are essential the same as those described above.[7]

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Acknowledgement: The photographs in Figures 1 and 2 were kindly supplied by the father of the patient described in this case report.

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The fractured mandible—potential pitfall

Patrick Mehanna, David Leinkram, Ian Wilson

A 24-year-old presented Thursday to his local country hospital following an assault involving a punch to his jaw. He was discharged and advised to present to his GP Friday for review which he failed to do. He represented Saturday with pain but self discharged following radiographs. Upon returning Sunday he was diagnosed with a fractured mandible (left angle) and referred to John Hunter Hospital (Newcastle, NSW, Australia). No antibiotics were prescribed.

Upon admission Tuesday he was febrile, WCC 30 with increasing facial swelling. A CT scan showed marked airway deviation with the presence of a large neck collection. Incision and drainage with ICU postoperative admission was performed. It is likely the lower left wisdom tooth with possible pre-existing inflammation contributed to the infective picture.

This case illustrates the need for antibiotics and early diagnosis and referral for fractured mandibles.

Figure 1. OPG of fractured left angle of mandible with associated wisdom tooth

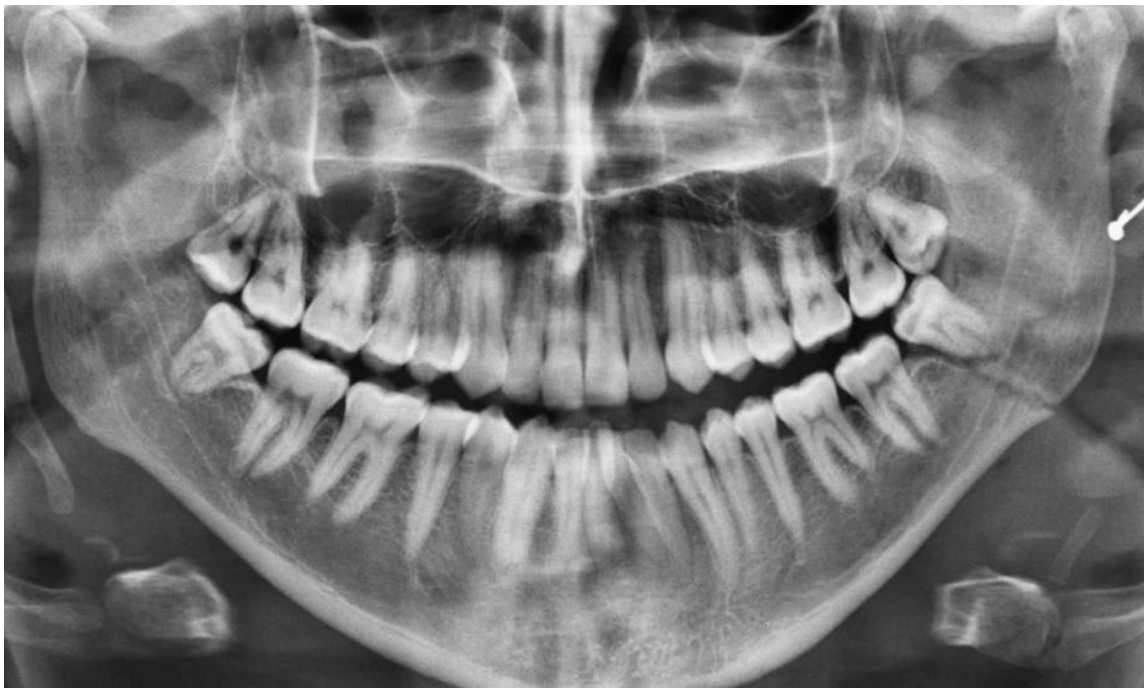


Figure 2. Fractured left angle of mandible

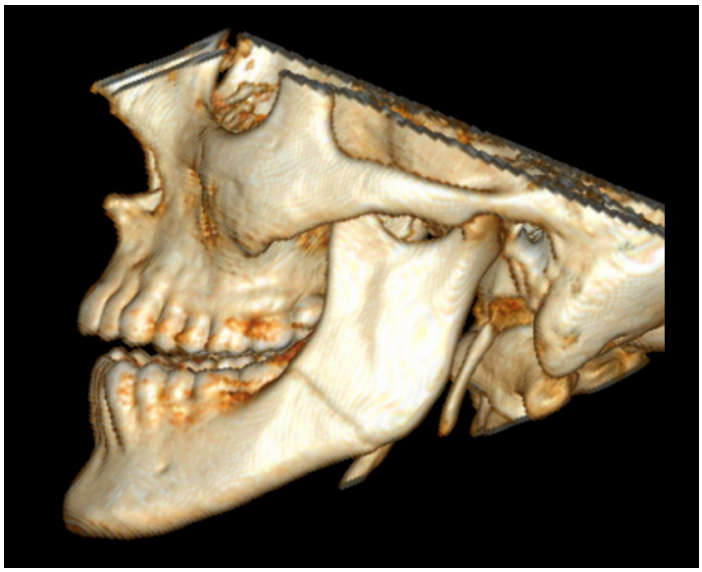
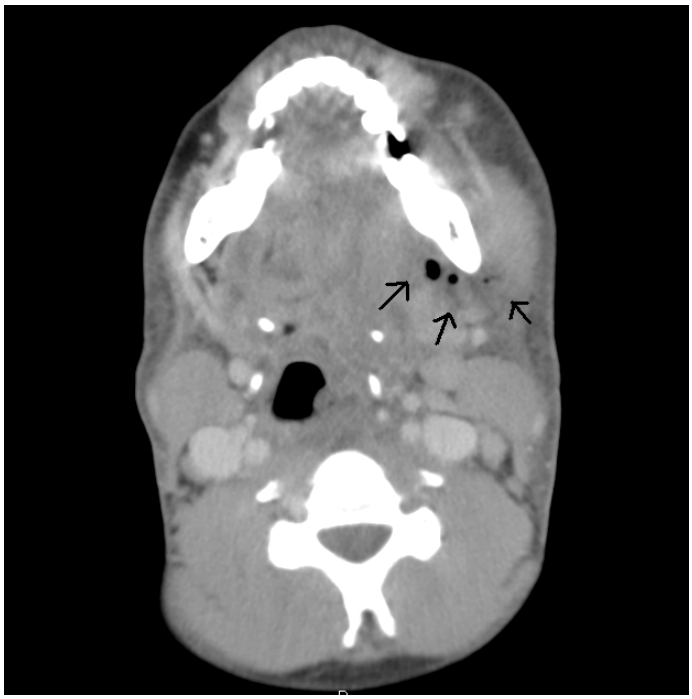


Figure 3. CT scan demonstrating airway deviation and neck collection. Arrows outlining collection with marked inflammatory changes



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THE NEW ZEALAND MEDICAL JOURNAL
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Hemichorea associated with nonketotic hyperglycaemia: MR imaging findings

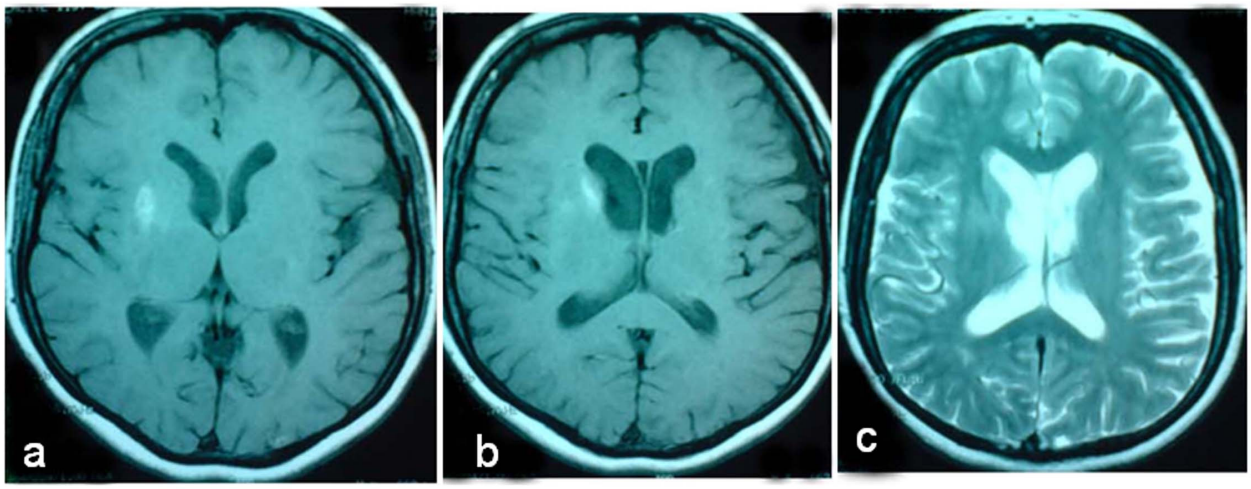
Mustafa Koplay, Demet İlhan Algin, Erim Gulcan

Clinical

A 47-year-old woman presented with involuntary movements of the left arm that had lasted 2 days. Neurological examination revealed that she had choreo-athetoid movements of the left arm and hyperreflexia. In the left arm, muscle tone was hypotonic with no evidence of weakness. Sensation was intact and no cerebellar signs were detected. Blood glucose was 400 mg/dl and urine analysis was negative for ketones.

Brain magnetic resonance (MR) imaging showed that the right putamen and caudat nucleus had high signal intensity in T1- and T2-weighted images (Figure 1). As haloperidol did not treat involuntary movement, depakin was added to the prothocol. The choreiform movements completely disappeared in 2 months.

Figure 1



Discussion

Hemichorea associated with particular neuroradiological abnormalities and nonketotic hyperglycaemia has been reported as an uncommon symptom of diabetes mellitus, usually occurring in women with Type 2 diabetes. Hemichorea may be a complication in patients diabetic.[1,2] The related aetiology and pathogenesis are unclear for developing hemichorea. Many different causes such as cerebral vascular insufficiency, acute dysfunction secondary to hyperglycaemic, hyperviscosity,

petechial haemorrhage, and decreased synthesis of GABA and acetylcholine secondary to metabolic changes, and inflammation have been stated as possible mechanisms of hemicorea [1,3]. The typical MR imaging finding is high signal intensity in the contralateral basal ganglia to the side of hemichorea in T1-weighted images. The findings in T2-weighted images are variable with signal characteristics ranging from hyperintensity and iso- to hypo-intensity.

In conclusion, MR imaging findings should be kept in mind in a patient with diabetes mellutus and hemichorea in terms of different diagnosis.

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THE NEW ZEALAND MEDICAL JOURNAL
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Clinical notes on a few cases of hydatid disease of the lung

A case report taken from a series of six cases written by Dr HM Wilson, Napier in NZMJ Nov 1909;8(32):15–21.

Case 2.—N.D., aged 17, labourer. This boy was one of the most interesting cases that I have ever seen, and he illustrates in many ways the insidious and the acute symptoms of hydatid disease of the chest. For some five to six days before admission he had been suddenly seized with pain in the right axilla, and then seen by a doctor the case had been diagnosed as pleurisy.

He walked up to the Hospital at Napier one afternoon from the town and applied for admission. In spite of the walk of about a mile, partly up hill; he was not distressed but only a little pale, and slightly cyanosed about the lips. On examination it was at once noted that the right side of his chest near the sternum was markedly prominent, and on being questioned he said that it ad been like that since he was 11, when his waistcoats had had to be made to fit it. I have seen such a prominence on one side or other of the sternum before and since, without any evidence of hydatid.

As he was an Australian, this prominence put hydatid of the lung into my mind, but finding no bulging in any of the intercostal spaces I dismissed it, thinking then in ignorance that the history of the bulge was of too long standing to have been caused by a cyst. I have further elicited the fact that he was a farm labourer, and had never been ill or short of breath at his work, nor had he had any cough at all—very remarkable facts when we saw the condition of his chest, P.M.

This insidious nature of hydatid disease will be seen in a case reported in this JOURNAL by Dr. Godfray of a Maori with a large ruptured hydatid cyst of the liver, which had evidently been pressing on the lungs years, but yet he was well enough to play football, and so meet his fate.

The physical signs observed were a dullness over the right chest at roughly a level with the nipple, with some pleural friction in the axilla and near the angle of the scapula. Over the lower part of the bulge noted near the sternum the note was impaired and the air entry poor.

I diagnosed pleural effusion, but as his back was so sore and septic from mustard plasters, deferred tapping him, although his apex beat was pushed out to the left, 3½ fingers breadths. In the next four or five days he had several attacks of dyspnoea although lying on his right side all the time.

His back now being cleaner, I inserted an exploring needle, and at once the condition of hydatid collapse occurred. He became more and more dyspnoeic, his pulse worse and worse, until it disappeared, and at the same time he looked bluer and bluer.

I incorrectly interpreted the symptoms, thinking that they were due to cardiac displacement, and hurriedly made an incision through an intercostal space, and drove a pair of forceps into the fluid cavity letting out a pint of fluid which subsequent examination showed to contain no Hooklets.

This did not relieve the condition, and at this time Dr. Leahy saw him with me and suggested hydatid disease.

He was very ill, so I inserted a tube into the wound and stimulated him, until at the end of au hour or two he was better. The next few days he ran a high temperature, had a quick pulse, and marked dyspnoea. He was dressed daily, but evidently the tube had not ill his extreme condition in the first place found the original cavity. The whole picture of the chest now altered.

There was resonance everywhere with a loud tubular breathing in one part, moist rales in another, sharp fine crepitations in another, in fact there were to be heard with the stethoscope the sounds, of every variety of pathological lung. This collapse and variety and alteration of physical signs are very typical of hydatid disease of the lung, coming on as they do after the insertion of an exploring needle. Unfortunately I have no case in this series, although of course they are legion, of the bursting of a hydatid cyst in the lung, after being explored with a needle.

The boy gradually improved for five days, and his temperature remained down then for four days, but his respirations remained at 30 to the minute. This marks another prominent symptom, that is the marked dyspnoea which this boy had, more especially later on. In eight days from the original exploration with the needle, up went his temperature to 103 again, and with it the pulse and respiratory rhythm.

At a consultation of the staff, some more clear fluid was drawn out of the old wound in the back (no Hooklets present), but owing to the variety of chest symptoms it was thought that there might be a pyopneumothorax, and so it was decided to excise a piece of rib near the nipple so as to be able to explore the prominence in front as well as the condition behind. This was done under local anaesthesia, a lot of fluid was evacuated from the pleura, a large cyst was felt over against the heart, and at the base the adherent lung. The cyst was packed round and opened two days later, when the endocyst was removed.

The boy died two days after the operation. The most fatal symptom was his dyspnoea, which nothing seemed able to relieve. It was not due to cardiac displacement, as the removal of the pleural fluid after the first stage of the operation had brought the apex beat back to the nipple line. At a P.M. another cyst was found at the back of the lung, where the original incision had been made, but in the emergency of the first collapse the sinus had been lost and subsequently obscured through the area dull before becoming resonant.

I fancy it would have been better to have opened the cyst at once, removed the endocyst, and drained through the pleural space where the fluid was let out of at the first stage of the operation. The finding of the cyst in front and the desperate condition at operation of the boy made one do as little as possible, or else the cyst that remained at the base, and which had been opened, could hardly have escaped one.

THE NEW ZEALAND MEDICAL JOURNAL
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Obesity—weight loss with calcium supplementation?

Some believe that increasing calcium intake may help prevent weight gain. The rationale is that calcium can combine with fatty acids in the intestine to form insoluble soaps which are not absorbed. Another theory concerns calcium and adipocyte triglyceride deposition. Anyway, this report concerns a prospective randomised trial in which overweight or obese subjects were randomised to calcium carbonate 1500 mg per day or placebo over a 2-year period.

You will not be surprised to learn the trial result was negative. Calcium supplements do not help the overweight or obese to lose weight.

Ann Intern Med 2009;150:821–9.

Prostate cancer screening—perspective from the United States

The American Urological Association (AUA) has recently provoked a stir by suggesting that all asymptomatic men at age 40 years with an estimated life expectancy of more than 10 years should have a baseline prostate specific antigen (PSA) test. Depending on the baseline, a strategy might involve annual PSA testing and digital rectal examination (DRE) or more infrequent testing.

On the other hand, the American Cancer Society suggest annual screening at age 50 years after discussing risks and benefits with patients. And more conservatively, the US Prevention Services Task Force (USPSTF), the American Academy of Family Physicians, and the American College of Physicians believe that the evidence is insufficient to assess the balance of benefits and harms of screening.

The controversy continues…

JAMA 2009;301:2538–9.

Hand-carried ultrasound machines in the wards?

The editor of the *American Journal of Medicine* speculates in an editorial on this possibility. Clearly they could be useful as he claims but there are certain impediments. One is the cost—up to US$40,000 each and another is training the clinicians to use them. In the same issue is a relevant paper. Ten hospitalists (full-time hospital physicians) performed heart examination and portable ultrasound on 354 general medical patients. When they compared their clinical assessment with the ultrasound they found the ultrasound increases the accuracy of the assessment of left ventricular dysfunction, cardiomegaly, and pericardial effusion, and fails to improve assessment of valvular heart disease.

Whether this helped the patient has not been determined. We think that it will be some time before we have these machines in our wards.

The American Journal of Medicine 2009;122:35–41 & 1–3.

Imaging strategies in patients with acute abdominal pain

Decision making in patients with acute abdominal pain on the basis of clinical and laboratory evaluation alone can result in unnecessary interventions or in delayed treatment of urgent conditions. This is the starting point of this paper from the Netherlands. This prospective study, involving over 1000 patients with acute abdominal pain, evaluates the efficacy of plain radiography, ultrasonography (US), and computed tomography (CT) after clinical and laboratory evaluation. Plain radiology was not useful. Diagnostic sensitivity was 89% for CT and 70% for US.

The conclusion is that although CT is the most sensitive they recommend using ultrasonography first and CT only in those with negative or inconclusive ultrasonography results. This tactic lowers radiation exposure as, in their experience, only 49% of their patients needed to proceed to CT.

We suspect that most surgeons already proceed along these lines but it is always useful to have evidence to back it up.

BMJ 2009;338:b2431.

Vertebroplasty for painful osteoporotic vertebral fractures

Recently (NZMJ 22 May 2009) we reported on the probable benefits of balloon kyphoplasty in the treatment of vertebral compression fractures. Recently two papers have been published on vertebroplasty—similar to kyphoplasty without the balloon.

In these randomised trials, polymethylmethacrylate was injected into the collapsed vertebral body, or a sham procedure was performed. In one trial there were similar significant reductions in overall pain in both study groups at each follow-up assessment (1 week; 1,3, & 6 months). The other trial showed similar results with symptomatic improvements in the vertebroplasty and sham treatment cohorts.

So vertebroplasty appears to be no more beneficial than sham procedures. A pity, but a good demonstration of the value of randomised prospective trials.

NEJM 2009;361:557–9.

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Informed choice—is the student doctor immunisation policy deficient?

Despite maximal medical care, Zachary Brian Harding Gravatt (Zac) died at 7:15pm on 8 July 2009 of severe adrenal haemorrhage and septic shock associated with meningococcal septicaemia, approximately 5 hours after being admitted to hospital.[1] At the time, Zac had been caring for patients as a MBChB IV student doctor in the general surgery wards at Auckland Hospital. Serology confirmed infection with the C strain of meningococcal disease.

Zac's death is a sentinel example of why the old adage "an ounce of prevention is worth a pound of cure" still rings true. No amount of care was able to "cure" Zac and yet "prevention" of Meningococcal C was available in New Zealand through a $40 vaccine that has around 95% long-term efficacy in young adults.[2]

Meningococcal C is the second most common strain in New Zealand with those most at risk being infants less than 1 year old, teenagers 15–19 years of age, and certain healthcare workers exposed to close patient contact. The Ministry of Health in New Zealand recommends vaccination of first-year university hostel students.[3] While the vaccine is not publicly funded in New Zealand due to limited financial resources, both Australia[4] and the United Kingdom[5] undertake publicly funded general population vaccination programs against meningococcal C.

Student doctors like Zac are required to be immunised with the publicly funded vaccines (mumps, measles, polio, hepatitis B, etc),but information and advice are not provided regarding meningococcal C vaccination (or indeed other unfunded options such as pneumococcal or rotavirus vaccines).[6]

Zac's death should have been entirely preventable. The medical school must update the vaccination advice given to student doctors under their guardianship so that others like Zac can make an informed choice whether or not to vaccinate against killer diseases like meningococcal C. Everyone deserves the opportunity to make an informed choice to protect themselves and potentially save their own life regardless of public funding.

Ironically, 8 July (the day Zac died) is the same day that Dr Benjamin Waterhouse administered the first smallpox vaccine to his own son more than 200 years ago in 1800. While New Zealand may not have been able to afford to save Zac through a publicly-funded vaccination program, neither were we afforded the chance to give our own son the $40 meningococcal C vaccine.

Informed Choice as embodied in Rights 6 and 7 of the New Zealand Code of Health and Disability Services is not an ideal but a necessity, particularly in this age of ever increasing rationing of public funds.[7] Updating the student doctor immunisation policy to include advice about unfunded options such as meningococcal C is seriously overdue.

Lance Gravatt [BSc, MSc (Hons) 1st Class, PhD]
(Zac's Father)
Auckland

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Additional impacts of regional nuclear war on New Zealand

I am writing about concerns I have regarding the letter *New software for modelling impacts of regional nuclear war* in the *New Zealand Medical Journal* of 3 July 2009 (Vol 122 No 1298). I am a member of the National Consultative Committee on Disarmament and when I raised this issue at a recent meeting the Chairman, Dr Rod Alley, suggested that I should write to you.

I certainly agree with, and appreciate, the points which are made in the letter regarding the effects of the dust in the atmosphere which would be caused by the explosion of nuclear bombs, and the severe damage which would be caused, and you may well say that describing these effects ought to be sufficient to awaken people to the dangers of nuclear conflict, even to countries which have not taken part in it.

Nevertheless it seems to me that a nuclear conflict would have even more immediate results which ought to be considered. You speak of the atmospheric dust as if it were like the dust from, for example, a volcanic eruption. But the dust from the explosion of nuclear bombs would contain radioactive dust.

It used to be thought that the radioactivity from nuclear explosions was confined to the area of the explosion. But researchers into the effects of the explosion of DU weapons, such as Dr Chris Bushby in a book produced by the United Nations Institute for Disarmament Research—the Disarmament Forum book—have shown that dust from those explosions travels long distances in the air and is inhaled by humans or animals, or else eventually settles in the soil or groundwater, so that it is ingested by people or animals who drink the water or eat plants which have absorbed it. It has been shown by medical tests that it remains in the body tissues for a long time.

If this is so of the explosions of depleted uranium weapons, how much more would it be true of uranium bombs made of full-strength enriched uranium, much more powerful and more numerous. Dr Ian Fairlie in an article *The Health Hazards of Depleted Uranium* published in the *Disarmament Forum* magazine has discussed the health hazards of uranium. He states:

> Like other heavy metals such as chromium, lead, nickel and mercury, uranium is chemically toxic to kidneys, the cardiovascular system, liver, muscle and the nervous system. Also, since all uranium isotopes are radioactive, they emit radiation—a known carcinogenic agent. This was thought to be of concern mainly when uranium was inhaled as aerosols or dusts, because their long residence times in the lung could result in lung cancer.

It seems to me that this information about the immediate and great dangers posed to those who inhale or ingest the radioactive dust produced by the explosion of nuclear bombs is even more important that what is in your article, and should have been included in it.

Kathleen Loncar
National Consultative Committee on Disarmament member
Wellington

Response

In response to the above letter (commenting on our previous letter[1]) we note that our aim was to address an under-recognised impact of regional nuclear war: that of fire-induced atmospheric dust spreading globally. In such a brief letter it was not possible to adequately survey all the adverse impacts of nuclear war which span direct local impacts[2] as well as global impacts relating to ozone destruction, radiation dispersal and social and economic collapse of countries affected by trade disruptions.[3–7]

Nevertheless, we agree that a nuclear war would likely result in substantial stratospheric injection of radionuclides that spread globally. This problem has been quantified previously around a large US-Soviet nuclear war for New Zealand[8] and for the Southern Hemisphere as a whole.[3]

For regional nuclear war between China and India it has also been estimated that radiation could be expected to cause 230 fatal cancers per million people in the Northern Hemisphere and 0.2 per million in the Southern Hemisphere (or more precisely, at latitude 20 to 40 degrees North in the Northern Hemisphere and at latitude 30 to 50 degrees South in the Southern Hemisphere).[9] But these calculations involve many uncertainties—e.g., ground bursts versus airbursts of the nuclear weapons, the explosive size of the weapons (kilotonnes/megatonnes), the size of fires in attacked cities, whether or not nuclear power plants are attacked, and also the estimated hazard of low-level radiation for cancer causation.

The complexity of assessing the impact of radioactive fallout on the New Zealand population from nuclear war is increased when considering how weather patterns (particularly rainfall) determine radionuclide deposition rates. Some individuals and industries may also act in a post-war setting to reduce radionuclide intake via the food pathway. Such measures include using pre-war powdered milk instead of fresh milk (and other pre-war stored food), increased washing of fruit and vegetables, and avoiding certain fresh foods produced in high rainfall areas etc.

Even so, given the estimate above for radiation-induced cancers from a China-India nuclear war, it is unlikely that radiation would be a major health problem for New Zealand compared to the other impacts of a regional nuclear war. That is why we consider that nuclear disarmament activities are far more likely to be stimulated by concerns of nuclear winter-type impacts from fire-related dust in the stratosphere than radiation concerns—especially if these climate impacts could cause a billion deaths from starvation as previously estimated.[7]

As we argued before,[1] the New Zealand Government and its citizens need to do much more to prevent nuclear war for numerous reasons and such activities would build on the strong track record that this country has already established.[10] Even so, such efforts also need to be balanced with the urgent need to address other global problems such as climate change.

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Erratum

Colquhoun D. *Doctor Who? Inappropriate use of titles by some alternative "medicine" practitioners*. N Z Med J. 2009;121(1278). http://www.nzmj.com/journal/121-1278/3158 and http://www.nzmj.com/journal/121-1278/3158/content.pdf

It has come to our attention that some details in reference #6 pertaining to a quote in the above paper were incorrect as follows:

Incorrect—Long PH. Stroke and spinal manipulation. J Quality Health Care. 2004;3:8–10.

Correct—Long PH. Stroke and spinal manipulation; 2004. http://skepticreport.com/sr/?p=88

Please refer to the links above to view the corrected paper.

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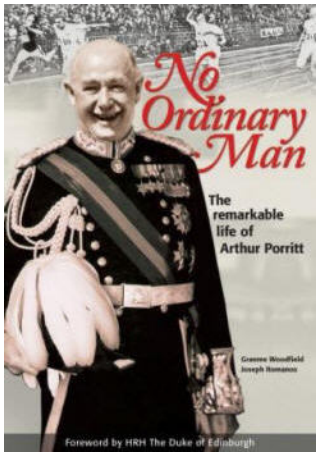
No Ordinary Man: the remarkable life of Arthur Porritt

Graeme Woodfield and Joseph Romanos. Published by Trio Books Ltd (Wellington), 2008. ISBN 9780958283953. Contains 308 pages. Price $59.99

This hard-covered book tells of the life and times of Sir Arthur Porritt.



It's a boys' own type story. The story starts growing up in Wanganui and the adventures with his father, a local GP.The story develops with his subsequent entry into medical school in Dunedin, his Rhodes Scholarship, his time at Oxford, selection for the New Zealand Olympic Team, his Olympic medal success (bronze medal at the 1924 Paris Olympics), surgical training in London and subsequent surgical career, his marriages and war time events, finally leading to being Governor General in New Zealand, and life thereafter.

The forword for the book is written by HRH The Duke of Edinburgh, no doubt because of the relationship that Arthur Parriott had with the Royal Family—as he knew them well being a surgeon to the Royal Family plus due to his involvement with the International Olympic Committee and wartime roles.

The book is full of good quality interesting photographs, which helps the reader understand the events of the time. There are many personal stories of events and people that make the book very readable. The style of the book which covers topics such as "Surgeon to Royalty" and "Serving Sport" means that often the same time period is covered repeatedly from different angles, each time adding more colour to the events of the period. This style is much more interesting than covering certain years without linking the topics.

The book is an engrossing read, and for those with an interest in the history of medicine hard to put down.

Frank Frizelle
Editor
NZMJ

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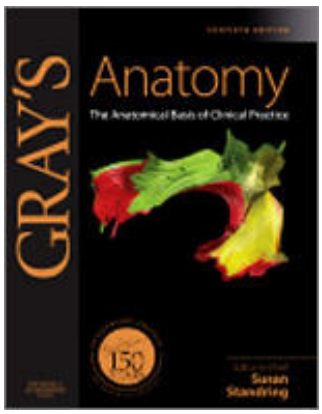
Gray's Anatomy: the anatomical basis of clinic practice (40th edition)

Susan Standring (Editor-in-Chief). Published by Churchill Livingstone Elsevier, 2008. ISBN 9780443066849. Contains 1576 pages. Price A$335.70 (online price—see link http://shop.elsevier.com.au/Medicine/Anatomy-and-Physiology/Grays-Anatomy/9780443066849.html)

Gray's Anatomy has been the Gospel of anatomy. The first edition was written in 1856–8 and published late in 1858. This is the 40th edition and was produced for the 150th anniversary of the original.



For Christmas last year I was given a book on the history of Gray's Anatomy and was fascinated at the origin of the book, so I was delighted when this large tome (over 1500 pages) turned up needing review.

Typical of most large medical books it is multi-authored from an international array of contributors, mostly from the United Kingdom. The are however two New Zealand-based contributors Drs Ming Zing from Dunedin and Dr Louise Moore from Tauranga. Amongst the international reviewers there is a surprising number of New Zealand Reviewers (9 of the 54).

The book itself comes with a unique online access number to allow online access of the book. As one would expect it is a very well produced textbook, and has excellent layout and presentation, with colour and black and white images and quality paper and binding.

The book follows the expected format for anatomy book, bones, muscles, nerves, blood supply, surface anatomy, etc and besides the usual anatomical diagrams it has CT scan and MI images. It also has relevant clinical interpretation of the anatomy, for example in the chapter on the rectum, it contains relevant anatomical information on rectal prolapse, rectocele, mesorectal excision of the rectum, abdominoperineal excision of rectal cancer, and local excision of the rectum. The book, however, does not met the standard of surgical relevance of "Skandalakis Surgical Anatomy".

In all it's a great book, has great pedigree, and looks fantastic. It's exact role, however, I have struggled to define as it is neither a guide for dissection (like "Cunningham's"), a cheap medical student textbook giving a overview, nor is it a surgical anatomy book.

Frank Frizelle
Editor (and Colorectal Surgeon, Christchurch Hospital)
NZMJ

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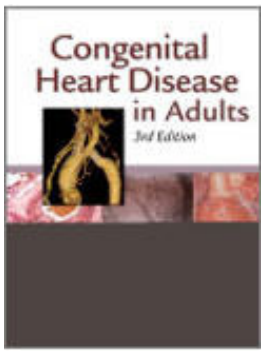
Congenital Heart Disease in Adults (3rd edition)

JK Perloff, JS Child, J Aboulhosn. Published by Saunders Elsevier, 2008. ISBN 9781416058946. Contains 504 pages. Price $286.20 (online) plus large booksellers.

This book addresses a rapidly expanding area of cardiology.



Whereas in previous decades few children with significant congenital heart disease reached adulthood, the situation has now changed as a result of markedly improved medical and surgical treatments of young infants and children.

In this 3rd edition, a thorough revision has resulted in a fascinating and useful review of this large topic. The lead author (Professor Joseph Perloff) has a vast insight into the discipline having lived through the major developments which he succinctly describes in this large book.

The 23 individual chapters, divided into 5 logical sections, are written by a combination of 26 clinical experts, mostly from the Ahmanson/UCLA Congenital Heart Disease Center in Los Angeles, California, whose depth of academic knowledge can only be admired. However, the final draft of each chapter was then drafted by Joseph Perloff in order to maintain a consistency of approach, which is clearly achieved. His understanding of the history and developments in each area are illuminating.

A particular highlight of the book is that with each chapter, there is a comprehensive list of references, from which further reading can be focused. There are also more 'modern' sections relating to psychological issues encountered by these now 'grown up' children, and the plea for more adult congenital cardiologists to care for this expanding patient group.

The potential readers of this book would be adult cardiologists with some patient load which includes adult congenital patients, as well as cardiology trainees, general paediatricians and of course the handful of paediatric cardiologists and surgeons who deliver a high class service to the New Zealand community; many of these colleagues have their own papers credited in the reference sections of each chapter!

I would highly recommend this book to my colleagues in both the adult and paediatric cardiology world, for a general read, as well as being a reference for future enquiry of specific adult congenital heart disease topics.

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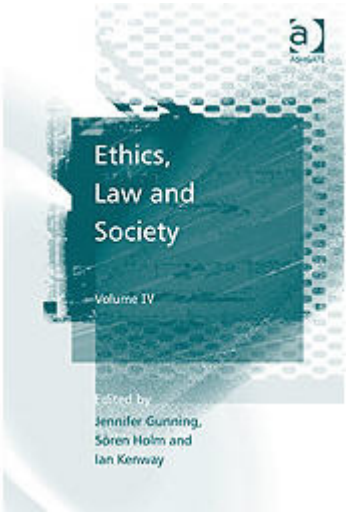
Ethics, Law and Society (Vol 4)

J Gunning, S Holm, and I Kenway (eds), Published by Ashgate (Surrey, UK), 2009. ISBN 9780754676461. Contains 438 pages. Price £67.50 (Ashgate website price)

This book is part of a series on ethics, law and society.



Jennifer Gunning (a former scientist) is a bioethicist at Cardiff Law School; Søren Holm is a medical doctor and philosopher in Cardiff and Professor of Medical Ethics at the University of Oslo; and Ian Kenway is an ethicist and expert in information and communication technologies at Cardiff University. These editors have produced a collection of slightly uneven papers from over 30 contributors, mainly from Wales and Scandinavia.

The book is oriented towards the serious ethicist or philosopher. It is divided into three main sections, covering agriculture and food, bioethics, and ethics and society, and ends with a selection of case commentaries.

I found some of the chapters heavy going, notably those primarily on legislation, regulation and recent reforms, mostly in the European context. I found others more accessible.

Given the recent publicity over pig farming in New Zealand, and the ongoing debate over agricultural efficiency and genetic engineering, I was particularly interested in the detailed and authoritative first section, which includes an overview of modern farming practices and their impact on animal welfare, and chapters on animal integrity, animal cloning, and globalisation and sustainability.

The fascinating chapter *Puzzle-solving for Fun and Profit: The Abusive Potential of Non-Genetic Health Data in Epidemiological Banks* is relevant to any researcher who deals with large databases of de-identified information. Many will be surprised to know how easily privacy can be breached, and anonymous records re-identified. Several chapters deal with genomics and the balance between public and personal interests. There is a discussion of the war against Iraq, and chapters on child soldiers, youth justice, reproductive technologies, and doping in child athletes. The allocation of healthcare resources and the responsibility of healthcare professionals in crises are also considered.

This book is topical and has much that is relevant to New Zealand, and also much of direct or indirect interest to those involved with healthcare.

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