

NEW ZEALAND

TE ARA TIKA O TE HAUORA HAPORI

MEDICAL JOURNAL

PUBLISHED BY:

 **PMA** PASIFIKA MEDICAL ASSOCIATION
Group

Vol. 139 | No. 1628 | 30 January 2026

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EDITORIAL

Rebuilding confidence in New Zealand's health system





Publication information

published by the Pasifika Medical Association Group

The *New Zealand Medical Journal (NZMJ)* is the principal scientific journal for the medical profession in New Zealand. The *Journal* has become a fundamental resource for providing research and written pieces from the health and medical industry.

The *NZMJ*'s first edition was published in 1887.

It was a key asset of the New Zealand Medical Association (NZMA)
up until July 2022.

It is owned by the Pasifika Medical Association Group (PMAG).

The PMAG was formed in 1996 by a group of Pasifika health professionals who identified a need for an association with the purpose of “providing opportunities to enable Pasifika peoples to reach their aspirations”.

ISSN (digital): 1175-8716

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Further information

ISSN (digital): 1175-8716
Publication frequency: bimonthly
Publication medium: digital only

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Subscription rates for 2026

Individual		Institute	
New Zealand	Free	New Zealand	\$680
International	Free	International	\$700

New Zealand rate includes GST. No GST is included in the international rate.

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Summaries

Rebuilding confidence in New Zealand's health system

Frank Frizelle

As New Zealand enters another election year, health system performance—especially access to general practice—will be the defining health issue, because the public increasingly experiences longer waits, rising costs and worsening inequity. It warns that political promises often outpace deliverability, constrained by workforce shortages and fiscal limits. Cancer care is highlighted as a key credibility test, with calls for Comprehensive Cancer Centres and improved screening/medicine access to reduce fragmentation and inequity. The conclusion calls for credible, costed, evidence-based policy, focused on delivery rather than rhetoric, to rebuild public confidence.

A systematic review of ethnic diversity in clinical trial participation in Aotearoa

Selwyn Te Paa, Tanira Kingi, Johanna Nee-Nee, Allie Eathorne, Trisha Falleni, Jackson Smeed-Tauroa, Bianca Crichton, Melemafi Porter, Gabrielle Shortt, Jordan Tewhaiti-Smith, Richard Beasley, Alex Semprini

This research looks at the participation of Māori and Pacific peoples in research carried out here in New Zealand. It shows that they are under-represented and that the quality of the data being gathered about these populations requires better standardisation.

Is ethnicity an independent predictor of health need? Linked cohort logistic regression analysis to predict amenable mortality

Andrea Teng, Melissa McLeod, Sue Crengle

Current primary care funding is based on the age and sex of practice populations. The minister of health has announced plans to also add deprivation, multimorbidity and rurality to the funding calculations—but has excluded ethnicity. This paper assesses the independent association of ethnicity and health need, in groups with the same age, sex, deprivation, personal income, multimorbidity and rurality. After accounting for all other variables (age, sex, deprivation, personal income, health conditions and rurality), Māori and Pacific ethnic groups had significantly higher rates of deaths from conditions responsive to healthcare (amenable mortality). Compared to European ethnicity, Māori had 43–50% higher amenable death rates, and Pacific had 14–23% higher rates. Māori and Pacific ethnicity are independent markers of health need crucial for the distribution of health services. In order to distribute primary care funding fairly according to health need, we must include ethnicity in the funding calculation.

Anastomotic leak rates between powered and non-powered circular staplers in left-sided colorectal resection; a retrospective cohort study

Jonathan Johns, Binura Lekamalage, Benjamin Cribb, Mark Omundsen

Anastomotic leaks are a major complication of bowel surgery. This study compared the rates of anastomotic leak in left-sided colon resections when using a stapler with a manual firing mechanism with the leak rate when using a stapler with a powered, automated firing mechanism. The study found a statistically significant reduction in anastomotic leak rate and length of hospital stay when using the powered automated stapler. As a single-operator, retrospective study, the study had limitations, and the results should be interpreted in conjunction with evidence from larger, prospective, multi-centre studies.

Percutaneous endoscopic gastrostomy in atypical parkinsonian syndromes: survival and aspiration outcomes from a retrospective international cohort

Tim Ruttle, Edward Jones, Cindy Towns

This international study examined rare neurological conditions known as atypical parkinsonian syndromes, which often lead to severely impaired swallowing. Over time, eating enough food to meet basic nutritional goals becomes impossible. Eating may also come at a price, as food or liquid can enter the lungs and cause chest infections—a process known as “aspiration pneumonia”. We found that placing a feeding tube directly into the stomach (a procedure called percutaneous endoscopic gastrostomy, or PEG) was associated with longer survival. However, there was no reduction in the number of hospital admissions for aspiration pneumonia.

Riluzole use and reasons for non-use in people with amyotrophic lateral sclerosis in Aotearoa New Zealand

Natalie Gauld, James Cleland, Sarah Buchanan, Joanna Hikaka, Chris Frampton, Stephen Buetow

Motor neurone disease (MND) causes increasing paralysis and is fatal. The most common form of it is amyotrophic lateral sclerosis and this is the form that riluzole (the only medicine that is funded by Pharmac in New Zealand that extends survival) is funded for.

Childhood blindness prevention in Aotearoa New Zealand

Madelyne Jouart, Elizabeth Conner, Jason Rodier, Graham Wilson

Childhood blindness in New Zealand is rare but has a big impact because children who are blind lose many years of potential vision, which affects their development, schooling and future opportunities. Many of the main causes, such as brain-related vision problems, premature birth complications, underdeveloped optic nerves, eye injuries and some genetic conditions, can be prevented or treated early. Early detection through newborn and school vision checks, timely treatment, and vaccination are essential to protect children’s eyesight. Prevention works best at several levels: before birth by supporting maternal health, during childhood through screening and treatment and by providing support for children with permanent vision loss. Improving outcomes requires teamwork between eye doctors, children’s doctors, optometrists, schools and public health services to make sure all children, especially Māori, have fair access to eye care.

Putting communities at the centre for a more effective and equitable health system in Aotearoa New Zealand

Anna Matheson, Johanna Reidy, Lis Ellison-Loschmann

Aotearoa New Zealand will not achieve a fair or effective health system unless communities are genuinely empowered to shape decisions, services and priorities. In this article, we argue that decades of centrally driven reforms have struggled to achieve their aims because they overlook the vital roles that local relationships, knowledge and trust play in shaping health outcomes. Drawing on complexity science, we explain why community “agency” is essential for improving overall system performance—particularly through stronger investment in community-led prevention and responsive primary care. While the COVID-19 response briefly showed how enabling communities could unlock more adaptable, integrated and effective ways of working, the system quickly reverted to established patterns, revealing how much potential for wider change remains untapped. We conclude that lasting progress depends on shifting resources, authority and leaning closer to communities, where health and wellbeing are actually created.

Are you sure it's Crohn's?

Winston Zheng, Zaal Meher-Homji, Minnie Au

Chronic inflammatory changes of the gastrointestinal tract with granulomas does not necessarily automatically reflect a diagnosis of Crohn's disease. Careful consideration to differentials such as intestinal tuberculosis must be considered in order to avoid a misdiagnosis or delayed diagnosis, which, given the different management strategies (immunosuppression vs antimicrobial therapy) can lead to poorer patient outcomes. Intestinal tuberculosis is rare in Australia, but a high clinical index of suspicion is required to ensure that this is not missed. This case report emphasises this teaching point to all.

Chronic oscillopsia and neck dystonia: atlanto-occipital origin

Leonardo Furtado Freitas, Márcio Luís Duarte, Kevin J Abrams

This report describes a 61-year-old woman with severe involuntary neck movements and visual instability, known as oscillopsia, which greatly affected her quality of life. Magnetic resonance imaging scans showed inflammation and damage to the atlanto-occipital joint, a key joint at the top of the spine that controls head movement. These findings explained both her abnormal neck movements and her visual symptoms. This case shows how important it is to check this joint in patients with unexplained neck dystonia or dizziness, as imaging can reveal a treatable cause.

Rebuilding confidence in New Zealand's health system

Frank Frizelle

As New Zealand enters another election year, I suspect the economy will dominate the political debate; however, health will remain a major secondary issue. Health has rarely been out of the media spotlight over recent years, reflecting people's lived experiences: longer waits, difficulty enrolling in general practice, rising out-of-pocket costs and uncertainty about whether improvements will materialise. These realities place the performance of Health New Zealand – Te Whatu Ora and access to primary care at the centre of the health debate. Around them orbit other high-salience issues: cancer treatment and screening, mental health and substance abuse, waiting times, workforce shortages and health funding within fiscal constraint.

Elections inevitably generate promises. The central challenge for voters is distinguishing between broad aspirations and policies that are credible, deliverable and fiscally sustainable. There are plenty of examples where election promises have not been deliverable. A memorable recent example of an undeliverable commitment was the previous Labour Government's KiwiBuild programme, which pledged 100,000 affordable homes over 10 years.¹ The target was abandoned in 2019 when it became evident it would not be achieved and only a small fraction of promised homes had been completed.¹ Aspirational commitments have value, as they signal intent and may give the electorate hope. Yet they also carry risk when they outpace workforce capacity, infrastructure and/or funding. This election will be judged not only by what parties propose, but by whether the public—and the profession—believe those commitments can realistically be implemented.

Performance of Health New Zealand – Te Whatu Ora and access to general practice

A defining health issue in this election is likely system performance—particularly timely access and equity of care. Health New Zealand – Te Whatu Ora was established to unify a fragmented system, improve national consistency and enable

co-ordinated planning. Reform on this scale inevitably takes time. Yet the public experience is shaped less by structural design than by persistent symptoms of strain: crowded emergency departments, long waiting lists and substantial regional and ethnic inequity.

For most people, however, the health system is experienced closer to home—through their general practitioner (GP). General practice is the critical pressure point. Many patients report difficulty enrolling, long waits for routine appointments and rising co-payments. Clinicians experience the same problem from the other side: rising patient complexity, an ageing practice workforce (especially in rural areas), increasing administrative burden and difficulty recruiting and retaining colleagues. Unmet need accumulates and manifests downstream in late emergency presentations, poor chronic disease control, avoidable hospitalisations and worse equity.

System performance cannot improve without a strong primary care foundation. A high-performing Health New Zealand – Te Whatu Ora will be measured less by yet another cycle of restructuring and more by whether timely, affordable access to general practice is restored.² Solutions will require sustainable funding models, multidisciplinary team-based care, better digital integration, robust rural support and strengthened links between primary and secondary services. Without this, downstream hospital pressures will remain structurally entrenched.

Cancer: medicines, screening and public confidence

Cancer related issues will also be an important election issue. Almost every New Zealander has lost family and/or friends to cancer. It is common, feared and uniquely sensitive to timely diagnosis and access to effective treatment. Incidence will rise as the population ages. One modelling study estimated annual diagnoses could increase from around 25,700 per year (2015–2019 baseline) to more than 45,000 per year by 2040–2044: an increase of 76%.³ This projected burden will test

a system already under strain.

New Zealand's cancer services remain distributed across multiple smaller units, with variable access to subspecialist expertise. Fragmentation contributes to inconsistent quality, weakens multidisciplinary decision-making and limits the scale needed to sustain research programmes and clinical trials. Patients requiring complex care may be forced to navigate multiple institutions, amplifying geographic, socio-economic and ethnic inequities.

International experience suggests that Comprehensive Cancer Centres (CCCs)—integrating surgical, medical and radiation oncology with pathology, imaging, supportive care, psycho-oncology, palliative services, data infrastructure and research—are associated with improved outcomes, fewer complications, greater access to clinical trials and stronger training and retention of specialists.^{4,5} CCCs can address current weaknesses by concentrating expertise and volume: embedding consistent multidisciplinary case management, creating high-quality training environments, expanding access to trials and novel therapies, supporting hub-and-spoke models that connect regional providers to tertiary centres and enabling outcome-driven improvement through audit and registry systems. In this sense, CCCs are both a clinical strategy and a workforce strategy. Without them, New Zealand risks continued loss of highly trained clinicians to overseas centres offering stronger integrated clinical-academic careers. Establishing CCCs would require explicit policy decisions: formal designation, sustained investment, partnership with Māori in governance and integrated national and regional network design.

Against this broader context, public debate has focussed heavily on cancer medicines. Clinicians recognise the complexity of Pharmac decision-making, which must balance evidence of benefit, safety, equity and opportunity cost. Public frustration has arisen less from this complexity than from its political over-simplification. Pre-election signalling of expanded access to cancer drugs created strong expectations, yet delivery has fallen short of what many believed was promised.⁶ This gap between rhetoric and reality increasingly shapes clinical consultations, as clinicians must explain why treatments discussed publicly, available overseas or funded in comparable systems remain inaccessible locally or obtainable only through self-funding.

Bowel cancer screening is a related—and

urgent—issue. International comparisons (particularly with Australia) and the rising incidence of early-onset colorectal cancer have strengthened calls to lower the age of eligibility. Commitments have been made, but implementation has been partial and progress has not matched earlier signalling.⁷ This delay represents a missed preventive opportunity in a disease where stage at diagnosis remains the strongest prognostic factor. For Māori and Pacific peoples—who already experience delays to diagnosis and poorer outcomes—deferred screening expansion risks widening inequity.⁸ Any move to expand eligibility must, however, be matched by investment in colonoscopy capacity and workforce; otherwise, once again, promise will exceed deliverability.

Election-year discussion should, however, move beyond single interventions towards system-level redesign. If New Zealand is serious about improving cancer survival and equity, it must address not only what treatments are funded, but how cancer care is organised and delivered.

Mental health and substance abuse: demand beyond capacity

Mental health and substance abuse remain areas where community need exceeds available capacity. Long waits for psychological therapies, limited access to culturally appropriate services and pressure on crisis care persist despite previous waves of investment. Workforce shortages across psychology, psychiatry, mental health nursing and addiction services remain central constraints.

The burden is not evenly distributed. Māori and Pacific peoples experience higher levels of need linked to structural determinants, colonisation, racism and socio-economic disadvantage. For young people, distress increasingly intersects with educational performance, employment insecurity, substance harm and family vulnerability.

Meaningful improvement requires long-term service design rather than episodic funding boosts. Priorities include early intervention, integration with primary care and schools, expansion of addiction treatment, digital support for remote communities and sustained workforce development.

Waiting lists, targets and real-world delivery

Waiting times—for outpatient appointments, diagnostics or elective surgery—remain acutely

visible to the public, with media accounts of delayed care common. While targets can improve transparency and sharpen accountability, without corresponding capacity they risk becoming symbolic metrics or creating perverse incentives. Real reductions in waiting times require real increases in capacity: additional theatre sessions supported by an expanded peri-operative workforce; greater diagnostic imaging, endoscopy and pathology capability; planned post-operative rehabilitation and community care pathways; and administrative and digital systems that enable efficient triage, booking and scheduling.

Ultimately, assessment of Health New Zealand – Te Whatu Ora's performance will rest less on whether targets exist on paper than on whether people experience shorter waits in practice.

Workforce: the fundamental limiting factor

Behind nearly every health system challenge lies the same determinant: workforce.⁹ New Zealand does not currently have enough doctors, nurses, midwives and allied health professionals to meet rising demand, particularly outside major centres. International competition and migration trends continue to complicate recruitment. Retention is equally urgent, with burnout, moral distress and fatigue increasingly reported.

Workforce constraint cannot be solved by slogans or short-term incentives. It demands sustained investment in local training pipelines, immigration settings that reflect both ethical recruitment and domestic need and working environments that are safe, supportive and professionally sustainable. This includes improved rostering, access to leave, clear career pathways that retain senior clinicians in clinical care, teaching, leadership and research. Without solving workforce constraint, even well-designed reforms will struggle to translate from policy into practice.

Fiscal reality: government debt, health budgets and the limits of the tax base

Any credible election-year discussion of health must confront economic reality. New Zealand faces rising government debt and increasing debt-servicing costs, which directly compete with other public expenditure. At the same time, health demand continues to grow—driven by population ageing, chronic disease burden, technological

innovation and legitimate workforce claims for fair remuneration in a globally competitive labour market.

Health funding is inseparable from the size and stability of the tax base. When economic growth is limited and tax revenue constrained, governments face unavoidable trade-offs. In this environment, limited real growth in health spending relative to demand means many initiatives can proceed only through reprioritisation rather than genuine expansion.

The implications are straightforward. Earlier bowel cancer screening, expanded access to cancer medicines, establishment of CCCs, strengthened mental health services and improved access to primary care all require additional capacity and resource—sometimes after a long lead time. There are only three fundamental pathways: increase revenue, reallocate from other sectors, or achieve genuine productivity gains within health. Ignoring this while making confident promises fuels scepticism and undermines trust. Transparent acknowledgement of fiscal limits and trade-offs is therefore essential—while still leaving room for ambition and hope.

The challenge of broken or delayed promises

We all recognise that making health promises is easier than delivering on them. When delivery falls short of expectations, public confidence erodes and clinicians are left to manage the consequences—explaining to patients and families why anticipated changes have not occurred.

A significant contributor to this dynamic has been ministerial churn. New Zealand has had six ministers of health in the past 6 years: David Clark (26 October 2017–2 July 2020), Chris Hipkins (2 July 2020–6 November 2020, interim), Andrew Little (6 November 2020–1 February 2023), Ayesha Verrall (1 February 2023–27 November 2023), Shane Reti (27 November 2023–24 January 2025) and Simeon Brown (24 January 2025–present).¹⁰ The portfolio has increasingly resembled a political “hot potato”. Each new appointment brings an inevitable learning curve, while turnover undermines continuity, weakens relationships with the sector and fragments strategic direction.

Accountability does not imply rigidity—circumstances change and some plans will not proceed. But accountability requires honesty, early communication and evidence-based explanation when commitments cannot be met. Deliverable,

costed, evidence-informed promises are ultimately more ethical than symbolic announcements that may never be realised.

What should matter most in the election debate?

I think several questions deserve prominence:

- Is Health New Zealand – Te Whatu Ora improving timely access and equity of care, particularly through strengthened primary care?
- How will access to general practice be restored and sustained across urban, provincial and rural settings?
- What is the credible pathway to earlier bowel cancer screening and improved colonoscopy capacity?
- How will access to effective cancer medicines be made fair and sustainable?
- Will New Zealand commit to catching up to the rest of the world by establishing CCCs and networked cancer systems?
- What is the long-term workforce strategy across health professions?
- How will prevention, Māori health equity and public health capacity be embedded into policy?
- How will reforms be funded within real fiscal constraints?

These are not partisan questions. They are questions on issues that may have a daily impact

on patients and those who care for them.

Conclusion: performance first, promises second

The defining health issue in this election will be system performance—especially access to GPs and primary care. If people cannot obtain timely primary care, if waiting lists continue to grow and if cancer care remains fragmented, public confidence in the health system will continue to decline regardless of structural reforms.

This election offers an opportunity to reset expectations around delivery rather than rhetoric. Effective health policy must be realistic about workforce and fiscal constraints, equitable in its impact and explicit about implementation. Some goals may be aspirational, but that distinction should be transparent. Promises alone will not deliver earlier diagnosis, improved survival or restored trust.

Success in the years ahead will be judged by whether New Zealanders experience easier access to general practice, earlier cancer detection, equitable treatment irrespective of geography or ethnicity and genuine improvement in outcomes. Clinicians and communities alike will look for seriousness, transparency and courage in the health debate.

The challenge to political leaders is clear: make health not only a centrepiece of the election conversation, but the centrepiece of delivery thereafter.

COMPETING INTERESTS

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<https://nzmj.org.nz/journal/vol-139-no-1628/rebuilding-confidence-in-new-zealand-s-health-system>

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A systematic review of ethnic diversity in clinical trial participation in Aotearoa

Selwyn Te Paa, Tanira Kingi, Johanna Nee Nee, Allie Eathorne, Trisha Falleni, Jackson Smeed-Tauroa, Bianca Crichton, Melemafi Porter, Gabby Shortt, Jordan Tewhaiti-Smith, Richard Beasley, Alex Semprini

ABSTRACT

AIM: Diverse ethnic representation in clinical trials is critical to ensuring research priorities align with patient need and uphold commitments to health equity. In Aotearoa New Zealand, this is crucial given the persistent health inequities faced by Māori despite obligations of the government to Te Tiriti o Waitangi/the Treaty of Waitangi. We report the findings of a systematic review of ethnic representation, with a focus on Māori and Pacific peoples, in randomised controlled trials (RCTs) undertaken in New Zealand between 2010 and 2020.

METHODS: A search was undertaken for RCTs undertaken in New Zealand between 2010 and 2020, registered in the Australia New Zealand Clinical Trials Registry (ANZCTR) and published in a peer-reviewed journal. Ethnicity data were categorised to Stats NZ Tatauranga Aotearoa (Stats NZ) level one or two codes. The Preferred Reporting Items for Systematic reviews and Meta-Analyses guideline was followed. The primary outcome was the proportion of each Stats NZ level one ethnicity code, for all participants recruited to RCTs conducted in New Zealand which reported ethnicity.

RESULTS: One thousand and forty trials were identified, 342 met the inclusion criteria, of which 103 reported no ethnicity data. For 295,254 participants across all 239 included studies, 6.1% of participants were European, 2.9% Māori, 1.4% Pacific peoples, 7.5% Asian, 2.5% Middle Eastern/Latin American/African (MELAA) and 9.0% Other ethnicity, with 70.6% Residual (unable to be categorised).

CONCLUSION: Ethnicity reporting in New Zealand-based clinical trials is inadequate and not standardised. Mandatory ethnicity reporting per Stats NZ codes to the New Zealand Health and Disability Ethics Committees, ANZCTR and peer-reviewed journals, should be considered mandatory for RCTs undertaken in New Zealand.

The lasting effects of colonisation continue to negatively affect the health status of Indigenous populations worldwide.¹ In Aotearoa New Zealand, Te Tiriti o Waitangi/the Treaty of Waitangi establishes the Crown's obligation to ensure equity in health outcomes for Māori (the Indigenous people of New Zealand), yet systemic inequities persist in access to healthcare, workforce representation and exposure to the determinants of health. This continues to be a significant cause of ongoing injustice, with the economic costs of Indigenous health inequities estimated at NZ\$863.3 million annually, while the Waitangi Tribunal's *Hauora* report found that underfunding for Māori primary healthcare alone exceeds NZ\$1 billion per year when unmet need is considered.²⁻⁴

A randomised controlled trial (RCT) is the gold-standard method to determine the efficacy of an intervention. Ensuring diversity in participants recruited to RCTs is essential in ensuring the external validity of data and to best represent

the characteristics of those with the disease being researched. Despite recognition of this, adequate representation of gender, demographics, minority and vulnerable populations is rarely achieved, impacting the external validity of clinical trial results, and propagating a barrier to optimal translation of research findings.⁵⁻⁷

Two commonly used, demographic terms to categorise clinical trial participants are "race" and "ethnicity", defined by the Oxford English Dictionary as "a group of people belonging to the same family and descended from a common ancestor" and "status in respect of a group regarded as ultimately of common descent, or having a common national or cultural tradition" respectively.^{8,9}

Standards for the reporting of race and ethnicity in published research varies by region and journal, with many reports omitting this information altogether.^{6,10}

In New Zealand, an ethnicity standard classification is published by Stats NZ Tatauranga

Aotearoa (Stats NZ).^{11,12} This is a hierarchical system comprising four levels, each of which involves more detailed ethnic categorisation. In health-related data presentation, level one is most commonly used and comprises six categories: 1) European, 2) Māori, 3) Pacific peoples, 4) Asian, 5) Middle Eastern/Latin American/African (MELAA) and 6) Other ethnicity. Users self-identify and can report multiple categories to best reflect their cultural, ancestral and geographic origins. Self-identified categories are then categorised into one of these six categories.

New Zealand has a vibrant clinical trials presence, contributing research findings of international importance through strong academic and commercial networks. However limited data exist to describe ethnic representation in clinical studies undertaken in New Zealand, despite being critical to ensure research priorities align with the health needs of the community and that the study conduct meets Te Tiriti obligations of partnership, protection and participation for Māori communities.

Here we report the findings of a systematic review of ethnic representation, with a particular focus on Māori and Pacific peoples, in all published RCTs undertaken in New Zealand between 2010 and 2020 and registered on the Australia New Zealand Clinical Trials Registry (ANZCTR). The hypothesis was that structured reporting of ethnicity and representation in clinical trials was inadequate.

Methodology

We conducted a systematic review using the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines, the protocol for which is registered in the international prospective register of systematic reviews (PROSPERO) database (Ref: CRD42020210764).¹³

Search strategy

We searched the ANZCTR for interventional, randomised controlled trials undertaken in New Zealand, registered between 12 October 2010 and 12 October 2020. Additional parameters were studies marked as complete and with ethical approval.

Eligibility criteria

Results were screened and included for analysis if a linked publication was available in the PubMed database. Where multiple publications for a single study were present, the one reporting ethnicity

data was included, or in the event of no ethnicity data reporting the main publication was used. Published protocols were excluded. This screening process was undertaken by two reviewers in duplicate, with conflicts arbitrated by a third.

Data extraction

All data were extracted using standardised forms created in REDCap electronic data capture tools hosted at the Medical Research Institute of New Zealand.¹⁴ Publication details recorded included title, first author, digital object identifier (DOI), study phase, country(ies) of recruitment, single- or multi-centre, funding source and principal investigator host-organisation type. Given the disparity in use of the terms “race” and “ethnicity” across the reported studies, the number of participants were recorded by ethnicity using Stats NZ level one—1) European, 2) Māori, 3) Pacific peoples, 4) Asian, 5) Middle Eastern/Latin American/African (MELAA) and 6) Other ethnicity—and level two codes where available or defined in free-text fields. The classification “residual” is used where responses do not fit into these specified categories.

The primary outcome was the proportion of each Stats NZ level one ethnicity code, for all participants recruited in RCTs conducted in New Zealand, in which ethnicity data were provided. Secondary outcomes included the mean proportion of each Stats NZ level one ethnicity code, per study; study characteristics reported for phase, funding source and sponsor type; study categorisation to “race”, “ethnicity”, “race and ethnicity” or “missing”.

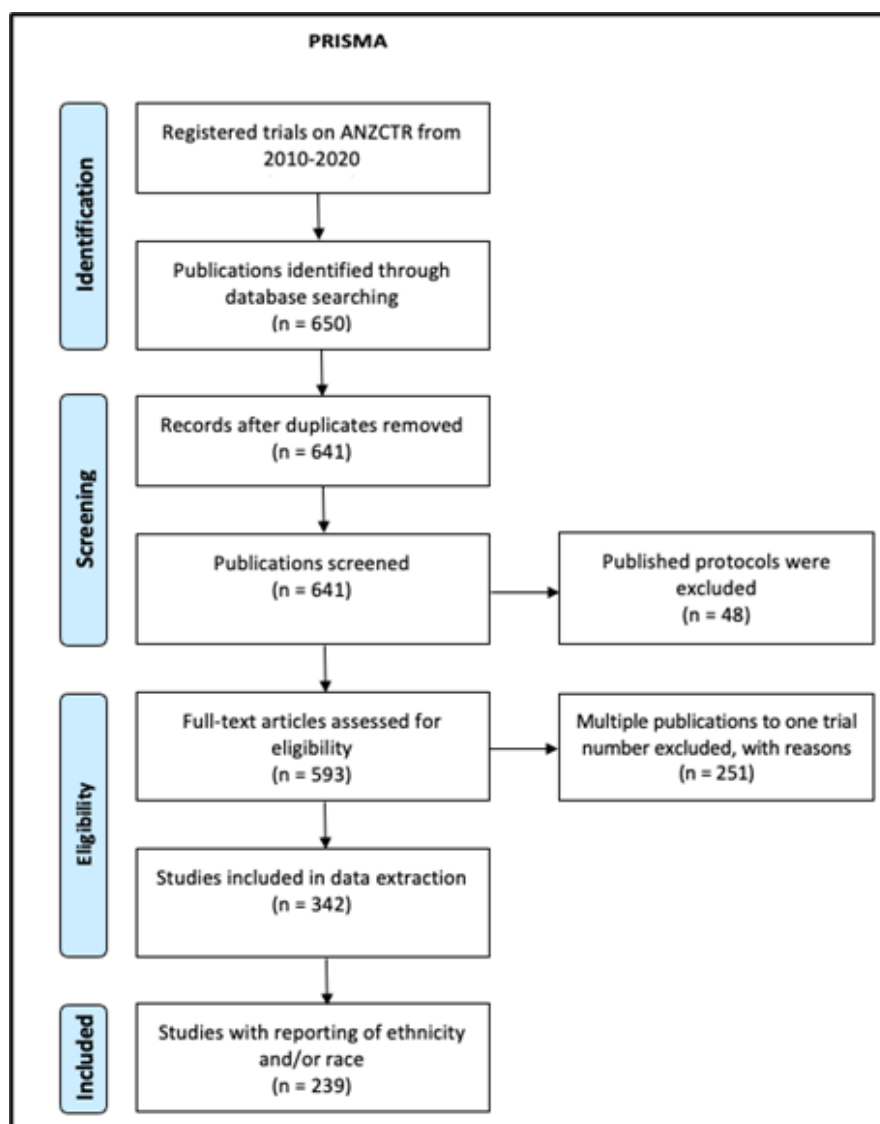
Results

One thousand and forty trials were identified from the ANZCTR search, of which 342 met our inclusion criteria (Figure 1). Of these 342 studies, 103 (38%) published no ethnicity data at all, with the remaining 239 included in the analyses. General descriptors of the extracted studies are described in Table 1.

Study phases, their funding source and type of sponsor for the 239 RCTs are described in Table 1.

Race and ethnicity categorisation

Of the publications that categorised participants, 112/259 (46.9%) categorised participants under a heading of “race”, 95 (39.7%) under a heading of “ethnicity”, 31 (13.0%) under both race and ethnicity and 1 (0.4%) study had no definition (Table 2).

Figure 1: PRISMA flow diagram of studies included in the review.**Table 1:** Study phase, funding source and sponsor type.

Study phase N (%)	1	2	3	4	n/a	
	14 (5.9)	53 (22.2)	114 (47.2)	10 (4.2)	48 (20.1)	
Funding source N (%)	Government body	Industry	Hospital	University	Charities	Other
	16 (6.7)	178 (74.5)	4 (1.7)	24 (10.0)	10 (4.2)	7 (2.9)
Sponsor N (%)	University	Hospital	Pharmaceutical	Other		
	116 (48.5)	50 (20.9)	32 (13.4)	52 (21.8)		

Participant representation by ethnicity

There were 295,254 participants enrolled across all 239 RCTs analysed. The proportion of each Stats NZ level one ethnicity code for all participants are presented in Table 3.

Ethnicity reporting per trial

The mean proportions of participations per

RCT, by ethnicity, are presented in Table 4.

Discussion

This analysis of RCTs recruiting participants in New Zealand between 2010 and 2020, highlights inconsistent and inadequate demographic reporting, with 38% of studies publishing no ethnicity/

Table 2: Use of “race” and “ethnicity” in study reporting.

	N/239 (%)
Ethnicity	95 (39.7)
Race	112 (46.9)
Ethnicity and race	31 (13.0)
Missing	1 (0.4)

Table 3: Overall participant ethnicity representation.

Level 1 ethnicity	Participants in studies reporting ethnicity/race N/295,254 (%)
European	18,031 (6.1)
Māori	8,428 (2.9)
Pacific peoples	4,088 (1.4)
Asian	22,068 (7.5)
MELAA	7,516 (2.5)
Other	26,534 (9.0)
Residual*	208,589 (70.6)

*Unable to be classified

Table 4: Ethnicity representation per study reporting ethnicity and/or race data.

Level 1 ethnicity	Mean (SD)
European	23.2 (36.7)
Māori	2.7 (7.4)
Pacific peoples	1.8 (6.1)
Asian	9.2 (18.2)
MELAA	2.3 (8.3)
Other	4.8 (11.1)
Residual*	55.9 (42.9)

*Unable to be classified

Figure 2: New Zealand HDEC ethnicity, optional reporting.

Ethnicity Data	
FR S.	Please provide ethnicity data for participants enrolled in the study in New Zealand. State the number of participants identifying as:
	<input type="text" value="New Zealand European"/>
	<input type="text" value="Māori"/>
	<input type="text" value="Samoa"/>
	<input type="text" value="Cook Island Māori"/>
	<input type="text" value="Tongan"/>
	<input type="text" value="Niuean"/>
	<input type="text" value="Chinese"/>
	<input type="text" value="Indian"/>
	<input type="text" value="Other (e.g. Dutch, Japanese, Tokoloan)"/>

race data at all. Of the studies that reported ethnicity or race data in some form, 70.6% of all participants could not be classified according to Stats NZ level one codes.

When combining all 295,254 participants across these RCTs, the reported ethnic representation was 6.1% European, 2.9% Māori, 7.5% Asian, 1.4% Pacific peoples, 2.5% MELAA, 9% Other and 70.6% residual in which responses did not fit into these specified ethnic categories. The mean study enrollment proportion, across all 239 studies that reported ethnicity or race data, was 23.2% European, 2.7% Māori, 9.2% Asian, 1.8% Pacific peoples and 2.3% MELAA. Differences between these figures are explained by the influence of very large RCTs and targeted ethnicity studies on the pooled data description.

The 2023 Census reports figures of 67.8% European, 17.8% Māori, 17.3% Asian, 8.9% Pacific peoples and 1.9% MELAA respectively.¹⁵ This significant discrepancy between reported proportions of all ethnic groups and the census data is driven by a majority of participants placed in the Stats NZ residual category, comprising those data points unable to be defined within the official level one or two coding. For example those reported as “non-hispanic”, “white”, “don’t know”, “unable or willing to answer” or placed in combined groupings such as “Māori/Pacific” were classified as “residual”.

This gross disparity between ethnic propor-

tions reported in RCTs and those derived from the census reinforces our major finding that reporting of ethnicity in RCTs is inadequate and mandates cautious interpretation of these data. However, potential under-representation of Māori in research is a concern, given the many health conditions that disproportionately affect Māori and obligations to Te Tiriti o Waitangi to ensure equitable participation in New Zealand-based RCTs. Several factors may contribute to this, including historical trauma and mistrust from health and research interactions, lack of cultural responsiveness in trial design, and anxieties about data sovereignty. Ensuring clarity around who controls research data, how they are used, and whether they serve Māori aspirations for Hauora (health and wellbeing) are fundamental to increasing representation in New Zealand research.

International efforts to increase equitable access to clinical trials are ongoing.⁷ Assessing progress toward equitable representation in New Zealand-based clinical trials requires a standardised, mandatory reporting mechanism.

Currently, every research team undertaking an RCT in New Zealand is required to submit their study for ethical review by the Health and Disability Ethics Committees (HDEC) and formally register their studies in the ANZCTR. HDEC requires that all research reports require ethnicity breakdown; however, as this is not explicitly stipulated at the

review and approvals stage, it may not be prospectively implemented. Furthermore, the optional ethnicity classification fields do not align with any of the Stats NZ levels, making interpretation within an approved framework difficult (Figure 2).

The data presented reinforce the importance of structured, prospective collection of ethnicity data in clinical trials in order to gauge progress toward a goal of equitable participation. For New Zealand trials, consideration should be given to mandatory recording of ethnicity to HDEC at the final study report stage, aligning with Stats NZ level one codes as a minimum. For international multi-centre trials that require protocolised ethnicity or race classifications locally relevant to the sponsor, this can be achieved as an additional, country specific, data collection. Extending this to the ANZCTR would further promote transparency in reporting, allowing open analysis of trial related ethnicity data to better track representation in specific specialist fields, trial phases or novel methodologies such as decentralised designs. Given the dominance of industry sponsored clinical trials (Table 1), including requirements at the regulatory stage will establish clear expectations for all research conducted in New Zealand.

Beyond this, consideration must be given to how whakapapa, or ancestral lineage, shapes a participant's identity, including interaction with

healthcare services and clinical research opportunities. This underlines the importance of adequately incorporating other cultural worldviews and context when designing and implementing clinical trials in New Zealand. For Māori, systemic efforts have been made to ensure responsiveness in research grant applications, study protocols and workforce representation, toward improving participation and health related outcomes.^{16,17} To our knowledge, no previous overall reporting of Māori participation in New Zealand-based clinical trials has been undertaken, undermining these efforts toward equity and limiting opportunity for continuous improvement. We therefore recommend the mandatory reporting of trial participation according to Stats NZ level one ethnicity codes by regulatory bodies and registries such as HDEC and ANZCTR.

Conclusion

Ethnicity reporting in New Zealand-based clinical trials is inadequate and not standardised. Mandatory recording of ethnicity to Stats NZ codes should be considered during trial protocol development and reporting to the New Zealand HDEC, ANZCTR and peer reviewed journals. The lack of accurate data on Māori participation in clinical trials is undermining health equity goals and obligations under Te Tiriti o Waitangi.

COMPETING INTERESTS

None. This review was funded with the assistance of Health Research Council Independent Research Organisation funding.

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URL

<https://nzmj.org.nz/journal/vol-139-no-1628/a-systematic-review-of-ethnic-diversity-in-clinical-trial-participation-in-aotearoa>

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Is ethnicity an independent predictor of health need? Linked cohort logistic regression analysis to predict amenable mortality

Andrea Teng, Melissa McLeod, Sue Crengle

ABSTRACT

AIM: This study examines whether ethnicity is an independent marker of health or if ethnic disparities are fully explained by age, sex, rurality, socio-economic position and morbidity.

METHOD: Using the Stats NZ Tatauranga Aotearoa Integrated Data Infrastructure, we identified the resident population of Aotearoa New Zealand each year from 2009 to 2018, establishing 10 cohorts that were followed up with at 12 months for amenable mortality, i.e., deaths from conditions responsive to healthcare in under-75-year-olds. Age, sex, ethnicity, rurality, small area deprivation, personal income and morbidity of cohort members were described. Logistic regression analyses and likelihood ratio tests were used to assess the independent association of these variables with amenable mortality.

RESULTS: Ethnicity, socio-economic position and morbidity, along with age, sex and rurality, made significant independent contributions to predicting amenable mortality. Ethnicity predicted amenable mortality after adjusting for other variables. Compared with Europeans, the odds of amenable mortality were 1.46 (95% confidence interval [CI] 1.43–1.50) times greater in Māori and 1.18 (95% CI 1.14–1.23) times greater in Pacific and half as likely in Asian (0.54, 95% CI 0.52–0.57).

CONCLUSION: Māori and Pacific ethnicity, and also socio-economic position and morbidity, are independent markers of health need relevant to the distribution of health resources and targeting of health services.

The inclusion of ethnicity alongside deprivation, morbidity and rurality in the design and delivery of health services and programmes, health resourcing and targeting has been politically contested. The Aotearoa New Zealand Cabinet issued a directive in 2024 restricting the use of ethnicity as a measure of need for targeting services, requiring strong rationale, efficacy, consideration of other markers of need and evaluation.¹ Public health experts, however, have cited extensive evidence for ethnicity as a marker of health need and its importance for targeting services.²

Current and intergenerational racism has been cited as the “basic” cause of ethnic inequities in health.³ Ethnic inequities are created and maintained through the differential distribution of the determinants of health (such as socio-economic position, education), and differential access to and the quality of healthcare.⁴ Including ethnicity in analyses of health is important to capture differential health need as well as the wide-ranging effects of racism on health⁵ that may be otherwise poorly measured within administrative datasets—for

example, differential access and quality of care,⁶ and differential exposure to risk factors⁷ (air pollution, housing quality, occupational exposures, tobacco). For Māori, the ongoing existence of inequities in health and government inaction to address these are a breach of Te Tiriti o Waitangi/the Treaty of Waitangi⁸ and Indigenous rights.⁹

Controversy around use of ethnicity as a marker of health need has implications for resourcing and targeting of health services. For example, primary care and community care has been under review. The WAI 2575 Waitangi Tribunal claim¹⁰ found that the current approach to the funding of primary care embeds historical inequity and systematically underfunds services for Māori by not recognising higher needs and historical under-utilisation. The formula to determine how money is distributed among primary care services has been based on population age and sex distributions and on historical fees for service subsidies, and thus embeds and perpetuates historical patterns of differential access and entirely ignores those with past (and future) unmet need for healthcare.¹¹ A report was commissioned in

2022 by the Health Transition Unit within the Department of Prime Minister and Cabinet.¹¹ It advises on the development of a new capitation funding formula for general practice services, highlighting that the major problem with the current formula is that it does not appropriately capture health need, e.g., as the population ages and patient complexity increases. The report recommends funding be based upon a combination of age, sex, ethnicity, deprivation and morbidity. In 2025, the minister of health announced that deprivation and multimorbidity will be added to a revised primary care funding formula from July 2026. However, this revision has failed to include ethnicity, as recommended.¹¹ The weighting on each factor remains unknown.

In another example, the use of universal age criteria to start screening programmes ignores the epidemiological evidence that the incidence of long-term conditions and most cancers increase at earlier ages for Māori. The bowel screening age was introduced at 60–74 years. However, there is a higher proportion of bowel cancer in Māori before they reach 60 years (58% Māori females vs 27% non-Māori females, and 52% Māori males vs 29% non-Māori males).¹² As a result of the universal starting age (60 years), Māori have less opportunity to benefit from bowel cancer screening than non-Māori.

There is plenty of evidence quantifying the strong relationship between comorbidities and health outcomes such as mortality and cancer (e.g., using the M3 index¹³). However, it is more rare for health research to investigate the independent predictive effect of all three—ethnicity, deprivation and comorbidities—simultaneously. This paper will address that gap. Similar papers investigating emergency department (ED) attendances, ED outcomes and ambulatory-sensitive hospitalisations have been identified.^{14,15} Our approach will use national datasets and focus on amenable mortality, which is unlikely to be susceptible to biases from different levels of healthcare access. Amenable deaths are from conditions responsive to healthcare that occur in individuals under the age of 75 years.

The aim of this study was to quantify to what extent ethnicity can predict health need over and above age, sex, rurality, deprivation, income and morbidity. The goal of this study was to inform approaches to distributing health resources and targeting of health services that address differential health needs and support health equity by ethnicity. We were interested in contributing to

what could be improved to ensure people get the right level of services, rather than maintaining current approaches that are not working.

Methods

This retrospective cohort study uses the Stats NZ Tatauranga Aotearoa (Stats NZ) Integrated Data Infrastructure (IDI), a research database of whole-population administrative data, census and sub-population survey datasets. In the IDI, datasets (nodes) such as health and census are probabilistically linked to a central spine dataset. The spine aims to include anyone who has been a resident in New Zealand, identified from linked birth, tax and migration records.

Population and demographic data

The IDI-estimated residential population (IDI-ERP) of New Zealand¹⁶ was identified on 30 June (reference date) in each year from 2009 to 2018, thus allowing for population mobility during the 10-year study period and avoiding COVID-19 effects. The IDI-ERP includes individuals in the IDI spine (ever residents) who had activity in accident or injury insurance, tax, health or education datasets in the 12 months prior, and excludes those who have died or spent several months overseas (in the 6 months either side of the reference date). This gave us 10 national resident cohorts over a 10-year period. On each reference date, the cohort was characterised by age, sex, ethnicity, deprivation quintile, rurality and morbidity; and then they were followed-up over the subsequent 12 months to assess mortality outcome. Individuals 75 years old or more were excluded because no deaths in this age group are classified as amenable.

The IDI personal details table was used to identify age (0–14, 15–24, 25–34, 35–44, 45–54, 55–64, 65–74 years old on the reference date), sex and total response ethnicity. Māori, Pacific, Asian, Middle Eastern/Latin American/African (MELAA), Other and European ethnic groups were identified, based on data availability in the highest-ranked source for ethnicity data quality, i.e., from a census, the Department of Internal Affairs or the Ministry of Health – Manatū Hauora, etc. Total response ethnicity was used for descriptive results (allowing individuals to identify with more than one ethnicity), and prioritised ethnicity was used for regression analyses (with ethnicity assigned as the first one identified in the order of the groups listed above).

The IDI address notification table was used to identify the most recent address, reported to contributing agencies before the study reference date. The meshblock of this address was used to assign the corresponding New Zealand Index of Deprivation 2013 or 2018 quintile and the Geographic Classification for Health.¹⁷ Inland revenue data were used to sum income from the previous 5 years for everyone in the cohort who was 25 years old or more. Quintiles were produced after stratification by year and age group.

Multimorbidity

Multimorbidity is commonly defined as the presence of multiple diseases or conditions, often with the cutoff of two or more conditions.¹⁸ As has been recommended,¹⁸ we selected a multimorbidity index that has been locally validated using mortality outcomes. The M3 index has been developed in New Zealand to predict hospitalisation events and death using a 5-year look-back period of diagnoses during hospitalisation. M3 has outperformed both the Charlson and Elixhauser indices in predicting hospitalisation events and death in New Zealand,¹³ and was selected as the primary multimorbidity measure for this analysis.

We collated public and private hospital discharge datasets in the IDI using a look-back period of 5 years for everyone in each cohort. All hospitalisation events and their corresponding unique primary and secondary diagnoses (International Classification of Diseases 10th revision [ICD-10]) were selected. We applied the X3 index macro¹³ to give each individual an M3 weighting. If no weighting was available for an individual (e.g., they had no hospital admissions) then they received an M3 index of 0. The M3 index was categorised into 0, >0 to 1, >1 to 2, and >2 for ease of interpretation.

Amenable mortality

Measuring health need is fraught because service utilisation is a poor measure of the true health need, i.e., healthcare services may be needed but are not sought due to cost and other barriers. Administrative data rely on health service use, and therefore we focussed on amenable mortality as a measure of health need. Amenable deaths are from conditions responsive to healthcare that occur in individuals under the age of 75 years old,^{19,20} and can be considered to include an element of unmet health need. Amenable mortality does not rely on health service use for its measurement (i.e., mortality datasets include

deaths irrespective of where they occurred), and has been used previously as a measure of health need in New Zealand.^{20,21} The greatest health needs and costs to the health system are in the last year of life,²² supporting the use of a mortality measure.

We followed-up each cohort for 1 year (1 July to 30 June) from the index date to identify cases of amenable mortality. Mortality registrations (with month and year) were identified from the Ministry of Health – Manatū Hauora Mortality Collection in the IDI. We collated the underlying causes of death using the World Health Organization definitions (ICD-10, Australian modification). Deaths were categorised as amenable or non-amenable based on 2016 Ministry of Health – Manatū Hauora definitions.²⁰

Analysis

The analysis investigated the predictive effects of variables on study outcomes and did not seek to report on the causal pathways. Amenable mortality rates were reported for each New Zealand residential population cohort, comprising 10 years of follow-up from July 2009 to June 2019 (Table 1). Rates were reported overall and by sex, age, ethnicity, deprivation, income, morbidity index and rurality.

Logistic regression analysis was used to assess the independent explanatory power of ethnicity, deprivation, morbidity, rurality, age and sex for predicting amenable mortality, reported with odds ratios (OR) and their 95% confidence intervals (CI) in Table 2. Logistic regression was appropriate for a binary outcome and preferred over time-to event analyses given the limited added value of the latter for 12-months of follow-up. Univariate and multivariate results were reported and used to assess whether there are significant independent predictive effects of ethnicity, deprivation, morbidity and rurality, after adjusting for age and sex. Likelihood ratio tests were used to assess whether each variable in the full model contributed to a significantly better model fit compared with a model without that variable. Table 3 reports the OR of amenable mortality rates in each ethnic group compared with the European group, and how this association changes with stepwise addition of each new variable to the model.

All analyses were carried out in a secure IDI environment (Datalab), using SAS Enterprise Guide V.8.3. Confidentiality rules in the IDI required suppression of small numbers (<6

Table 1: Crude amenable mortality rates in New Zealand residential population younger than 75 years old, New Zealand, July 2009 to June 2019.

		Person years ^c	Amenable deaths ^c	Rate
		%	n	per 100,000
Total	All	42,223,521	52,371	124
Sex	Male	21,140,766	31,482	149
	Female	21,081,780	20,889	99
	Other	975	S	S
Age (years)	0–14	9,072,957	495	5
	15–24	6,373,227	2,307	36
	25–34	5,937,483	2,073	35
	35–44	5,985,627	3,588	60
	45–54	6,202,086	7,938	128
	55–64	5,051,727	13,467	267
	65–74	3,600,420	22,506	625
Total response ethnicity	Māori	7,738,731	12,471	161
	Pacific	3,737,439	4,266	114
	Asian	5,618,157	2,325	41
	MELAA	821,577	618	75
	Other	913,161	738	81
	European	30,463,104	36,354	119
	Missing ^a	144,087	S	S
Area-level deprivation index	Lowest	8,559,297	6,636	78
	Low–middle	8,299,827	7,878	95
	Middle	8,163,987	9,330	114
	High–middle	8,167,776	11,742	144
	Highest	8,697,423	16,359	188
	Missing	335,214	432	129
Income from last 5 years (25+ years old)	Lowest	5,481,678	10,896	199
	Low–middle	5,491,296	15,153	276
	Middle	5,474,013	11,514	210
	High–middle	5,486,571	7,233	132
	Highest	5,483,376	5,010	91
	Missing	14,806,593	2,565	17
Morbidity index (M3)	0 ^b	38,360,733	20,085	52
	>0–1	3,270,708	12,276	375
	>1–2	415,527	8,043	1,936
	>2	176,556	11,967	6,778

Table 1 (continued): Crude amenable mortality rates in New Zealand residential population younger than 75 years old, New Zealand, July 2009 to June 2019.

		Person years ^c	Amenable deaths ^c	Rate
		%	n	per 100,000
Geographic Classification for Health	Urban 1	26,655,423	28,113	105
	Urban 2	7,560,306	11,424	151
	Rural 1	4,947,597	7,725	156
	Rural 2	2,317,179	4,017	173
	Rural 3	484,377	936	193
	Missing	258,642	162	63

^a Missing only refers to prioritised ethnicity.

^b Includes people with no hospitalisation in the last 5 years.

^c Random rounded to base three.

S = suppressed due to small numbers fewer than six.

MELAA = Middle Eastern/Latin American/African.

Table 2: Relative odds of amenable mortality, July 2009 to June 2019, New Zealand.

		Unadjusted	Adjusted for age and sex	Adjusted for all other variables in the table	Likelihood ratio test (evidence variable improves model fit)
		OR (95% CI)	OR (95% CI)	OR (95% CI)	
Sex	Male	1	1	1	<0.001
	Female	0.66 (0.64–0.67)	0.65 (0.64–0.66)	0.62 (0.61–0.63)	
Age (years)	25–34	0.06 (0.05–0.06)	0.06 (0.05–0.06)	0.12 (0.12–0.13)	<0.001
	35–44	0.10 (0.09–0.10)	0.10 (0.09–0.10)	0.19 (0.18–0.20)	
	45–54	0.21 (0.20–0.21)	0.20 (0.20–0.21)	0.34 (0.33–0.35)	
	55–64	0.43 (0.42–0.44)	0.43 (0.42–0.43)	0.57 (0.55–0.58)	
	65–74	1	1	1	

Table 2 (continued): Relative odds of amenable morality, July 2009 to June 2019, New Zealand.

		Unadjusted	Adjusted for age and sex	Adjusted for all other variables in the table	Likelihood ratio test (evidence variable improves model fit)
		OR (95% CI)	OR (95% CI)	OR (95% CI)	
Prioritised ethnicity	Māori	1.66 (1.62–1.69)	2.49 (2.44–2.54)	1.46 (1.43–1.50)	<0.001
	Pacific	1.32 (1.27–1.36)	2.03 (1.96–2.10)	1.18 (1.14–1.23)	
	Asian	0.33 (0.31–0.34)	0.56 (0.53–0.58)	0.54 (0.52–0.57)	
	MELAA	0.48 (0.43–0.53)	0.84 (0.76–0.93)	0.72 (0.65–0.80)	
	Other	0.62 (0.58–0.67)	0.68 (0.63–0.74)	0.73 (0.67–0.79)	
	European	1	1	1	
Area-level deprivation index	Lowest	1	1	1	<0.001
	Low–middle	1.21 (1.17–1.25)	1.27 (1.23–1.31)	1.13 (1.10–1.17)	
	Middle	1.48 (1.44–1.53)	1.60 (1.55–1.65)	1.27 (1.23–1.31)	
	High–middle	1.93 (1.87–1.99)	2.13 (2.07–2.20)	1.44 (1.40–1.49)	
	Highest	2.79 (2.71–2.87)	3.27 (3.18–3.37)	1.67 (1.62–1.73)	
Income from last 5 years	Lowest	2.31 (2.23–2.39)	2.57 (2.48–2.66)	1.86 (1.80–1.93)	<0.001
	Low–middle	4.89 (4.74–5.05)	3.57 (3.45–3.68)	1.92 (1.86–1.99)	
	Middle	2.21 (2.14–2.29)	2.68 (2.59–2.77)	1.68 (1.62–1.74)	
	High–middle	1.25 (1.21–1.30)	1.56 (1.50–1.62)	1.24 (1.20–1.29)	
	Highest	1	1	1	
Morbidity index (M3)	0	1	1	1	<0.001
	>0-1	5.92 (5.78–6.06)	4.22 (4.12–4.32)	3.72 (3.63–3.81)	
	>1-2	28.27 (27.53–29.04)	17.82 (17.34–18.32)	13.74 (13.36–14.14)	
	>2	99.36 (97.02–101.75)	60.27 (58.79–61.79)	48.64 (47.42–49.89)	
Geographic Classification for Health	Urban 1	1	1	1	<0.001
	Urban 2	1.42 (1.39–1.45)	1.22 (1.19–1.25)	1.10 (1.07–1.12)	
	Rural 1	1.42 (1.38–1.45)	1.14 (1.11–1.17)	1.08 (1.05–1.11)	
	Rural 2	1.58 (1.53–1.64)	1.25 (1.21–1.29)	1.08 (1.04–1.12)	
	Rural 3	1.71 (1.60–1.83)	1.34 (1.25–1.43)	1.05 (0.98–1.13)	

Logistic regression analysis reporting ORs. Geographic Classification for Health urban/rural definitions are available from Whitehead et al.²³ Person-years (N) for all regression models was 26,511,750.

OR = odds ratio comparing likelihood of amenable mortality in this group compared with the reference group; 95% CI = confidence interval; MELAA = Middle Eastern/Latin American/African.

deaths), and random rounding of all raw counts to base 3. Also, rates that were based on small numbers had to be suppressed. Missing data were presented in tables wherever possible.

Ethics

Research was performed in accordance with the Declaration of Helsinki. The University of Otago Minimal Risk Health Research Committee gave ethics approval on 20 May 2024 (H24/0108). All administrative data used in this study on individuals were deidentified; thus, individualised consent for participation was not required.

Results

The New Zealand residential population, less than 75 years old, across 10 cohorts (2009 to 2018), was followed-up for 42-million person-years. There were 52,371 amenable deaths, with a crude rate of 124 per 100,000 person-years.

The descriptive table (Table 1) reports amenable mortality rates. There were higher crude amenable mortality rates in older age groups (55–74-year-olds), males, Māori, people living in more rural and higher deprivation areas, individuals with the lowest two quintiles of income and, particularly, individuals with an M3 index score greater than 0. The group with a >1 to 2 score had a 2% risk of amenable mortality in the next year, and the group with a >2 score had nearly a 7% risk (Table 1).

Key study results provide evidence demonstrating that ethnicity was a significant independent marker of health need, over and above the effects of age, sex, rurality, socio-economic position and morbidity (Table 2). In the fully adjusted model, Māori (OR 1.46, 95% CI 1.43–1.50) and Pacific (OR 1.18, 95% CI 1.14–1.23) had significantly higher odds of amenable mortality than European. Asian had the lowest odds of amenable mortality compared with European (OR 0.54, 95% CI 0.52–0.57). The inclusion of ethnicity to the fully adjusted model significantly improved the model fit (likelihood ratio tests all $p < 0.001$).

At the same time, age, sex, rurality, area-level deprivation, income and morbidity also each significantly improved the fit of the model (likelihood ratio tests all $p < 0.001$). Like for ethnicity, the independent effects of rurality, deprivation, income and morbidity remained statistically significant after adjustment for all other study variables. Morbidity had a very large effect on amenable mortality. Amenable mortality was 48.6 (95% CI 47.4–49.9) times as likely for people with

a morbidity index score of >2 compared with 0. Socio-economic position was also an important predictor. The odds of amenable mortality were 1.67 (95% CI 1.62–1.73) times greater for people living in the highest vs lowest deprivation areas, and 1.86 (95% CI 1.80–1.93) times greater for people on the lowest vs highest incomes.

Stepwise regression (Table 3) was used to investigate the impact of each additional variable on the magnitude of the association between ethnicity and amenable mortality. Deprivation and morbidity index were associated with the greatest attenuation in ethnicity effects, e.g., the Māori compared with the European OR decreased from 2.46 to 1.91 with deprivation added to the model, and decreased from 1.84 to 1.46 with morbidity added.

Discussion

Summary of results considering the literature

Ethnicity remained a significant predictor of health need after accounting for the effects of age, sex, deprivation, income, morbidity and rurality. Māori and Pacific peoples had 1.46 and 1.18 greater odds of amenable mortality than European peoples after adjusting for other factors. These findings are consistent with research in New Zealand and internationally.^{24,25} Ethnicity has been found to be an independent predictor of ED attendances and ambulatory-sensitive hospitalisation after adjusting for deprivation (Index of Multiple Deprivation), morbidity (M3) and multiple other factors.¹⁴ Our analysis shows that this is also the case for amenable mortality.

Including ethnicity as an independent marker of health need in health funding and prioritisation is important to address known differences in health need, but also to capture the wide-ranging effects of racism on health⁵ that may be otherwise poorly measured within administrative datasets.^{6,7} For Māori, the ongoing existence of ethnic inequities in health and government inaction to address these are a breach of Te Tiriti o Waitangi⁸ and Indigenous rights.⁹

Additionally, higher M3 morbidity index, greater deprivation and lower income were important predictors of greater amenable mortality (health need), over and above age and sex and ethnicity. High levels of the M3 index (>2) were (predictably) strongly associated with amenable mortality, experiencing 48 times the odds of amenable mortality in the subsequent year. This level of morbidity affected approximately one in 150 people in the

Table 3: How the relative odds of amenable morality by ethnicity varies with addition of rurality, deprivation and comorbidity variables in a stepwise logistic regression model, July 2009 to June 2019, New Zealand.

	Population	Unadjusted	Adjusted for age and sex	+ Adjusted for rurality, GCH	+ Adjusted for deprivation quintile	+ Adjusted for income quintile	+ Adjusted for morbidity, M3
	Person-years	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Māori	2,676,225	1.66 (1.62–1.69)	2.49 (2.44–2.54)	2.46 (2.40–2.51)	1.91 (1.87–1.95)	1.84 (1.80–1.89)	1.46 (1.43–1.50)
Pacific	1,081,869	1.32 (1.27–1.36)	2.03 (1.96–2.10)	2.11 (2.04–2.19)	1.45 (1.40–1.51)	1.35 (1.30–1.40)	1.18 (1.14–1.23)
Asian	2,481,327	0.33 (0.31–0.34)	0.56 (0.53–0.58)	0.58 (0.56–0.61)	0.53 (0.50–0.55)	0.46 (0.44–0.48)	0.54 (0.52–0.57)
MELAA	301,026	0.48 (0.43–0.53)	0.84 (0.76–0.93)	0.87 (0.78–0.96)	0.78 (0.70–0.87)	0.69 (0.62–0.76)	0.72 (0.65–0.80)
Other	392,817	0.62 (0.58–0.67)	0.68 (0.63–0.74)	0.68 (0.63–0.73)	0.67 (0.62–0.73)	0.69 (0.63–0.74)	0.73 (0.67–0.79)
European ^a	12,045,102	1	1	1	1	1	1

^a European, sometimes referred to as Sole-European, is the reference ethnicity group.

OR = odds ratio comparing likelihood of amenable mortality in this group compared to the reference group; 95% CI = confidence interval; MELAA = Middle Eastern/Latin American/African.

study population who were younger than 75 years old, highlighting its usefulness in health funding allocation.

Internationally, approaches to constructing funding formulae have been described with three purposes; a) identifying factors that predict differential need (such as those in this analysis), b) adjusting for other cost factors (e.g., rurality and visitors), and c) correcting for unmet need (e.g., that which is not evident from historical utilisation).²⁶ In a review of funding formulae, most jurisdictions included at least one measure of morbidity in the funding formula (but not New Zealand or Stockholm). None of the reviewed jurisdictions²⁶ used a combination of ethnicity, deprivation and morbidity together. The New Zealand population-based funding formula has been used to distribute regional district health board spending. The formula includes deprivation (NZDep index, quintiles 4 and 5) and ethnicity (Māori, Pacific, Other)²⁶ but not morbidity. Conversely, the 2026 revised primary care funding formula plans to include deprivation and morbidity but not ethnicity. Our findings support the importance of combining all three ethnicity, deprivation and morbidity measures in funding formula calculations, alongside age, sex and rurality adjusters.

Strengths and limitations

The retrospective cohort design reduces potential biases by defining model variables before amenable mortality occurred (except for sex and ethnicity, which are not time-stamped). A decade of linked data allows for high statistical power in the main analysis. We selected variables and databases available to the health sector to improve the applicability of our findings for use in directing health resources and health services/interventions. We used locally validated indexes of deprivation and morbidity, which are strong predictors of health need. Amenable mortality was a useful measure of health need because it does not depend on healthcare seeking and potentially incorporates an element of unmet need. However, it has been criticised as a poor causal measure of health system performance.²⁷ Amenable mortality also has limitations in that it considered only a specific group of conditions, ignoring morbidity and excluding a broader set of conditions that may still be preventable. We expect that our findings, e.g., on the importance of ethnicity, would be similar, however, if we had chosen a different measure like ambulatory-

sensitive hospitalisation.¹⁴

Adding other or more precise deprivation or morbidity predictors, or interaction terms, may improve this model's prediction ability. Such factors would need to be available for the whole population and their marginal value may be limited. For example, adding personal income to the model already containing deprivation made a small difference to the magnitude of the ethnicity effects (i.e., the Māori OR decreased from 1.91 to 1.84) and further socio-economic variables may have less additional predictive value. More precise modelling of health need is possible but ensuring modelling is applicable to resourcing and targeting decisions was our focus.

Morbidity data depend on access to hospitalisation and may be under-estimated for those with less hospital and healthcare access. Additional morbidity measures may make further useful contributions to accurately predicting amenable mortality, especially for morbidity that is defined outside of the hospitalisation dataset, e.g., mental health conditions. For example, in other countries multimorbidity measures based on medication prescribed have been found to be useful for predicting health care utilisation,²⁸ including a simple count of medications prescribed.²⁹ A mortality index (P3) using pharmaceutical prescribing from primary and secondary care has been developed in New Zealand to predict mortality,³⁰ but was found to have only marginal benefit for predicting mortality compared with using the M3 index alone (0.2–0.5% improvement), and thus might be expected to have a limited additional predictive effect on amenable mortality in this analysis.³⁰ This would be a useful area for future research, noting that P3, for example, is still dependent on whether people have access to healthcare and is not adjusted for unmet need.

Misclassification of ethnicity data may affect our results, e.g., if the IDI-ERP undercounts Māori, our estimates of the impact of ethnicity on amenable mortality may be conservative. This study used prioritised ethnicity, where each person is only counted in one ethnic group. This approach will not impact Māori results but will result in the undercounting of Pacific who also identify as Māori, as they will only be classified as Māori in this analysis.

Implications for public health

Ethnicity is an important marker of health need in New Zealand and remains an important marker of health need after accounting for age,

sex, rurality, deprivation, income and morbidity. Markers of ethnicity are critical in the allocation of health resources and targeting of services, alongside other markers such as deprivation and morbidity. Failure to include ethnicity in decision making about access to services and programmes, the primary care funding formula, and other relevant activities will negatively impact on our ability to deliver equal health outcomes to

all people in New Zealand, including Māori and Pacific people.

Conclusions

Ethnicity, in addition to socio-economic position and morbidity, is an important independent marker of health need that should be included in funding formulae, prioritisation and targeting of health services.

COMPETING INTERESTS

The authors declare that they have no other known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

A University of Otago research grant 2024 funded all authors.

ACKNOWLEDGEMENTS

Thank you to Professor James Stanley for producing the M3 index code, adapting it to run in the IDI and for advice on survey analyses. Thank you to June Atkinson for advice on socio-economic variables.

AVAILABILITY OF DATA AND MATERIALS

These results are not official statistics. They have been created for research purposes from the IDI, which is carefully managed by Stats NZ. For more information about the IDI please visit <https://www.stats.govt.nz/integrated-data/>. Applications can be made to Stats NZ to access research data from the IDI in an approved facility. Access to the data used in this study was provided by Stats NZ under conditions designed to give effect to the security and confidentiality provisions of the *Data and Statistics Act 2022*. The results presented in this study are the work of the authors, not Stats NZ or individual data suppliers. The results are based in part on tax data supplied by Inland Revenue to Stats NZ under the *Tax Administration Act 1994* for statistical purposes. Any discussion of data limitations or weaknesses is in the context of using the IDI for statistical purposes, and is not related to the data's ability to support Inland Revenue's core operational requirements.

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amenable-mortality

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Anastomotic leak rates between powered and non-powered circular staplers in left-sided colorectal resection; a retrospective cohort study

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ABSTRACT

AIM: Anastomotic leak (AL) is associated with major post-operative morbidity and mortality. The circular stapler, widely utilised in colorectal anastomosis, has seen a technological change from manual firing stapler (MFS) to powered automated firing stapler (PAFS). PAFS may reduce user error and technique variation and may be associated with reduced AL rate. The primary aim of the study was to assess differences in AL rate between MFS and PAFS. Secondary aims were to assess differences in length of stay (LOS) and 30-day mortality.

METHODS: This was a retrospective, single surgeon review of patients undergoing resection with anastomosis using a circular stapler between 2016 and 2023. A historical MFS group (n=105) and a study PAFS group (n=112) were identified. Demographics, comorbidity, operation type, neoadjuvant therapy, AL, LOS and 30-day mortality were recorded.

RESULTS: The populations were comparable, with no significant difference in demographics, BMI, ASA grade, neoadjuvant radiotherapy use or type of operation. The PAFS group contained more non-malignant cases, 35% vs 18% (p=0.01). AL rate was 11.4% in the MFS group and 3.6% in the PAFS group (p=0.04). Fifty-eight percent of the anastomotic leaks in the MFS group needed surgery, compared to zero from the PAFS group (p=0.09). Mean LOS was 10 days in the MFS group and 6 days in the PAFS group (p = 0.01). Thirty-day mortality was 0.9% from the MFS group and zero from the PAFS group (p=0.48).

CONCLUSION: While acknowledging confounders may have affected outcomes, in this study PAFS was safe and associated with a significant reduction in AL and LOS.

Anastomotic leak (AL) is a feared complication among colorectal surgeons that remains common despite improved understanding of technical and patient-related factors. Frequency of AL varies by centre and type of bowel resection. Large international studies have reported a frequency of AL for left-sided resections between 8% and 15%.^{1,2,3} Specifically in Aotearoa New Zealand, a review of anterior resections at a provincial hospital found an AL rate of 10.5%.⁴ Furthermore, the effects of colorectal AL are devastating both in terms of patient outcomes and financial implications for the healthcare system. AL is associated with a five-fold increase in 30-day mortality and a two-fold increase in local malignancy recurrence at 5 years.⁵ With regard to financial impact, AL in America have been shown to cost over US\$30,000 per index admission more than cases without AL.⁶ While in Europe the cost is even more stark at €54,000 additional cost per case

with AL.⁷ Given the sequelae of AL, performing a colorectal anastomosis is a critical step and all efforts should be made to minimise the rates of AL in colorectal surgery.

Traditionally colorectal anastomoses were hand-sewn; however, by the late 1970s, the end-to-end anastomosis (EEA) circular stapler for left-sided colorectal anastomosis formation came into use. After several iterations, the late 2010s saw the introduction of the powered automated firing stapler (PAFS). Proposed benefits of powered firing compared to a manual firing stapler (MFS) include reduced force required to fire the stapler, improved stapler head stability and equal compression of tissues throughout the anastomosis.⁸ Contemporary studies suggest a reduction in AL rates when using the PAFS for left-sided colorectal anastomosis.^{9,10} However, the PAFS has been associated with a higher rate of staple-line bleeding.¹¹

We hypothesise that use of the PAFS for left-sided

colorectal anastomosis will lead to a reduction in AL rate and therefore better patient outcomes when compared to manual firing stapler (MFS) use. The primary aim of our study is to compare AL between MFS and PAFS. Secondary aims of the study are to assess 30-day mortality, re-operation rate and length of hospital stay (LOS).

Methods

This was a single centre retrospective cohort study of consecutive cases performed at a regional hospital in Aotearoa New Zealand from June 2016 to October 2023. All left-sided resections, i.e., left hemi-colectomy, sigmoidectomy and high, low and ultra-low anterior resections, performed by a single colorectal surgeon, prospectively recorded on a personal database, were included in the study. The surgeon had been a consultant colorectal surgeon for 4.5 years and had performed approximately 360 colorectal resections by the start of the data collection period. During this period patients were managed according to well-established surgical checklist and Enhanced Recovery After Surgery (ERAS) protocol.¹² The surgeon switched to preferential utilisation of 29mm PAFS (ETHICON ECHELON CIRCULAR™) from 28mm MFS (COVDIEN EEA™) in June 2020.

Operation notes of all eligible patients were reviewed retrospectively, and patients who underwent an anastomosis with an EEA circular stapler met inclusion criteria for the study population. Patients were excluded if the anastomosis was hand-sewn or if there was uncertainty regarding which stapler was utilised. All patients had routine intraoperative “bubble-test” of the newly formed join.

The primary outcome, AL, was defined either radiologically, as documented on post-operative imaging report, or as documented in operative notes if taken back to theatre. Patients were investigated for AL only if clinically indicated.

Data were collected from online medical records by two authors. Outcomes included AL, post-operative complications defined by Clavien-Dindo (CD) score three or above, LOS and mortality at 30 days. Demographic data included age, gender, ethnicity, pre-operative ASA grade and BMI. Disease and surgical factors included underlying pathology (malignant vs benign), neoadjuvant treatment, height of anastomosis (high, low, ultra-low) and formation of covering ileostomy were also recorded.

Data for the two cohorts were analysed using

R Studio 3.6.1, with statistical significance of differences for categorical data assessed using Fisher's exact test, and statistical significance of differences for continuous data assessed using the Two-Sample *t*-Test. A *p*-value of less than 0.05 was deemed to be statistically significant.

Results

A total of 222 patients had left-sided anastomosis between October 2016 and October 2023. Anastomoses formed between October 2016 and May 2020 used the MFS, and anastomoses formed between June 2020 and October 2023 used the PAFS. Between October 2016 and May 2020 there were 106 left-sided bowel resections. One case was excluded as they had end stoma formation rather than an anastomosis.

A total of 116 left-sided colorectal resections were performed between June 2020 to October 2023. Four cases were excluded; two because they had stoma formed rather than anastomoses, two more because they had hand-sewn anastomoses (see Figure 1).

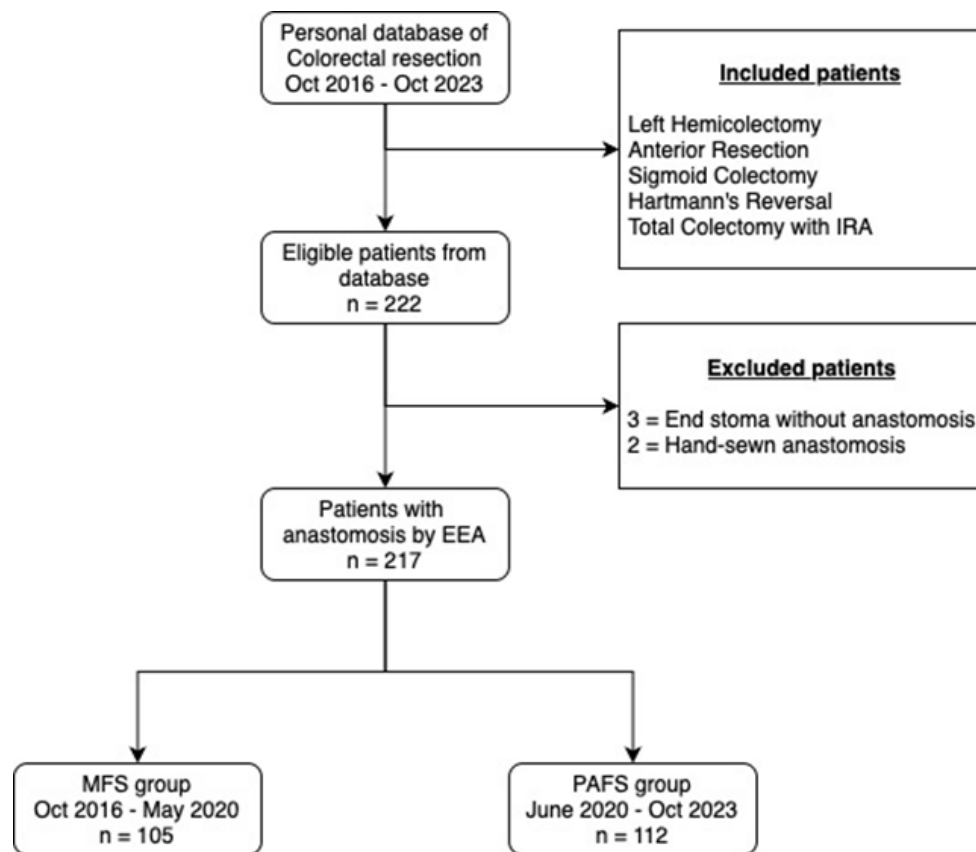
After exclusion, there were 105 patients who had anastomosis performed by MFS and 112 by PAFS for analysis.

Both cohorts showed marked homogeneity with regard to demographics and pre-operative comorbidity (see Table 1). There was no significant difference regarding type of operation, inclusion of covering ileostomy or use of neoadjuvant radiotherapy between each group (Table 2). There was a significantly higher rate of resections for malignancy in the MFS group, 82% (n=84), than the PAFS group, 65% (n=70, *p*=0.01).

The MFS group had an AL rate of 11.4% (n=12) compared to 3.6% in PAFS (n=4), which was statistically significant (*p*=0.04). The risk difference was 7.8%, giving a number needed to treat of 13, suggesting that for every 13 anastomoses performed with PAFS rather MFS, one leak would be prevented.

The mean LOS for the MFS group was 10.3 (SD=8.6) days, while for the PAFS group it was 5.9 (SD=4.9) days (*p*=0.01). Excluding patients who had AL, patients who had an anastomosis formed with the MFS had a mean LOS of 9.7 days, whereas the PAFS group had a mean LOS of 7.6 days (*p*=0.01).

For patients found to have AL, both MFS (n=12) and PAFS (n=4) patients had a mean LOS of 15 days (*p*=0.5). There was no significant difference among the leak cohort between the rate of

Figure 1: Study recruitment & exclusion flow chart.**Table 1:** Demographic and comorbidity characteristics of each cohort.

	Manual firing stapler (n=105)	Powered firing stapler (n=112)	P-value
Mean age (years)	65 (SD=14.4)	64 (SD=13)	0.9
Sex			
Male	55% (n=58)	54% (n=61)	0.99
Female	45% (n=47)	46% (n=51)	
Ethnicity			
NZ European/Pākehā	82% (n=86)	81% (n=91)	0.96
Māori	10% (n=10)	11% (n=12)	0.82
Other European	8% (n=8)	5% (n=6)	0.59
Other/Not recorded	<1% (n=1)	3% (n=3)	0.62

Table 1 (continued): Demographic and comorbidity characteristics of each cohort.

Mean BMI (kg/m ²)	27 (SD=5.3)	28 (SD=8.1)	0.15
Mean ASA	2 (SD=0.6)	1.9 (SD=0.5)	0.25

Table 2: Operation type, ileostomy formation rate and neoadjuvant radiotherapy rate. *ULAR = Ultra-low anterior resection, LAR = Low anterior resection, HAR = High anterior resection.

	Manual firing stapler (n=105)	Powered firing stapler (n=112)	P-value
Elective operation	96% (n=101)	97% (n=109)	0.71
Laparoscopic	69% (n=72)	64% (n=72)	0.56
ULAR*	31% (n=33)	26% (n=29)	0.37
LAR*	14% (n=15)	15% (n=17)	0.85
HAR*	33% (n=35)	40% (n=45)	0.33
Other	22% (n=23)	19% (n=21)	0.61
Covering ileostomy	40% (n=42)	36% (n=32 out of 88 complete data)	0.09
Neoadjuvant radiotherapy	18% (n=19)	18% (n=20)	1

pre-operative radiotherapy, covering ileostomy nor malignant pathology.

Fifty-eight percent of the AL occurring with the MFS (n=7) required a return to theatre, whereas none of the AL occurring with the PAFS needed operative management (p=0.09). One patient died at 30 days in the MFS group, whereas none of the PAFS group died at 30 days (p=0.48).

Of the 151 patients that had resections for malignancy, 82 were in the MFS group and 69 were in the PAFS group. Nine (11%) of the patients in the MFS group who had resection for malignancy developed AL, whereas three (4%) of the patients in the PAFS developed AL. However, this difference was not statistically significant (p=0.23)

The cost of the MFS was NZ\$850 per unit, compared to the PAFS at NZ\$1,067. The cost of a surgical bed at our hospital was NZ\$1,150 per night. Comparing the costs of each stapler use

based on average LOS, PAFS cost NZ\$4,383 less per use than MFS.

Discussion

This study found a significant reduction in the incidence of left-sided colorectal AL with the PAFS, with results comparable to recent international studies.^{6,7,11} While we did not look specifically at the reasons why the PAFS may have lower AL rates, this is likely to be multifactorial. We posit that consistency in the firing mechanism of the PAFS, and the reduced force needed to be applied to fire the stapler, are important factors in forming reliable anastomoses. However, the change of stapler from MFS to PAFS involved a change in manufacturer and therefore further differences in stapler technology may also have influenced outcomes.

Current literature suggests that malignant resections have a higher risk of AL than anastomoses formed after resection of non-malignant pathology.¹³ Our results showed a trend towards reduced rate of AL for anastomoses formed with PAFS during malignant resections, however the results did not reach statistical significance.

PAFS use was associated with a significantly shorter average LOS, even when accounting for AL. Furthermore, none of the patients who had an AL with the PAFS required a return to theatre. That said, reduced LOS and reduced return to theatre may also reflect factors such as evolution in clinical practice and experience gained over time.

As the data pertained to a single surgeon's experience, the technical skills for each operation are consistent. However, there may be a learning effect seen with outcomes improving over time. The firing of each stapler is usually performed by the assistant, rather than the primary operator. The study did not record who fired the stapler on each occasion, making it impossible to comment on the experience or learning curve of each stapler user.

Our results associate the introduction of PAFS with a financial saving, with a basic cost analysis showing a saving of over NZ\$4,000 with each use of the PAFS. This excludes additional costs such as further interventions or critical care required to manage AL, but it does not account for inflation over time. There is a green cost implicated with powered stapler use, however, that may be offset by reduced AL rate, subsequent interventions and LOS, and it is not explored in this study.

As a small, retrospective study, accuracy was dependent on medical records; this was a limitation of our study. To further the evidence base in this field it would be interesting to do a multi-centre, multi-operator study.

In conclusion, we found a reduced rate of AL and a reduced LOS when switching from MFS to PAFS for left-sided colorectal anastomoses. This adds to the body of evidence that the PAFS appears to be safe and may lead to improved patient outcomes. As the body of data regarding the use of this stapler grows it will be interesting to see what further conclusions may be drawn.

COMPETING INTERESTS

The authors declare that they have no financial or material interests that relate to the research described in this paper. There are no conflicts of interest to declare.

ACKNOWLEDGEMENTS

Ethics approval: Formal locality was approved for the study to be conducted in Health New Zealand – Te Whatu Ora Hauora a Toi, Bay of Plenty (study reference 2024:300).

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<https://nzmj.org.nz/journal/vol-139-no-1628/anastomotic-leak-rates-between-powered-and-non-powered-circular-staplers-in-left-sided-colorectal-resection-a-retrospective-coho>

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Percutaneous endoscopic gastrostomy in atypical parkinsonian syndromes: survival and aspiration outcomes from a retrospective international cohort

Tim Ruttle, Edward Jones, Cindy Towns

ABSTRACT

INTRODUCTION: Dysphagia frequently occurs in movement disorders, leading to malnutrition and aspiration. Percutaneous endoscopic gastrostomy (PEG) provides nutrition directly into the stomach, bypassing the dysfunctional swallow. However, PEG insertion is a complex decision, both clinically and ethically. Although PEG outcomes are reported in other neurological disorders, there is limited research in atypical parkinsonian syndromes such as multiple system atrophy (MSA), progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD). Insertion rates remain variable, reflecting a paucity of research and lack of consistent guidelines. Basic mortality and morbidity data would help inform practice. To our knowledge, this is the first international study of PEG insertion and its impact on survival and aspiration pneumonia in atypical parkinsonian syndromes.

METHOD: This was an international retrospective study of 72 patients with MSA, PSP or CBD. Survival was recorded from reported onset of dysphagia to death. Secondary outcomes included hospital admission rate for aspiration pneumonia.

RESULTS: Median survival was 17.4 months (95% confidence interval [CI] 14.0–24.9) in non-PEG patients versus 48.8 months (95% CI 44.8 to not reached) in PEG patients, hazard ratio (HR) 0.38 (95% CI 0.18–0.81; $p=0.013$). PEG was not associated with reduced risk of aspiration pneumonia; 0.76 versus 0.68 admissions per patient-year, incidence rate ratio (IRR) 1.41 (95% CI 0.74–2.68; $p=0.297$).

CONCLUSION: PEG insertion may improve survival in atypical parkinsonian syndromes, though we found no evidence of reduced aspiration risk. Given the rarity of these conditions, international registries may help to determine the safety and efficacy of PEG use.

Dysphagia occurs frequently in atypical parkinsonian syndromes. Subsequently, aspiration pneumonitis or pneumonia are common complications and significant causes of mortality.¹ Percutaneous endoscopic gastrostomy (PEG) tubes can be used in the setting of dysphagia to provide nutrition. In bypassing the abnormal swallow, PEG feeding was also originally thought to avoid or reduce the risk of aspiration pneumonitis or pneumonia. However, aspiration risk has proven to be more nuanced than this, with data showing no benefit or, at times, increased risk with PEG feeding.²

A number of neurodegenerative disorders have a significant evidence base to inform decision making and informed consent processes for PEG insertion.³ However, there is a paucity of research to guide decision making in movement disorders. This is particularly challenging in the atypical parkinsonian syndromes where the lower prevalence and reduced life expectancy further hamper research.

This international retrospective study analysed 72 patients with atypical parkinsonian syndromes across two regions in the United Kingdom (UK) and New Zealand. Patients with a diagnosis of multiple systems atrophy (MSA), progressive supranuclear palsy (PSP) or corticobasal degeneration (CBD) were included. The objective was firstly to determine whether PEG feeding increased life expectancy and secondly to assess whether PEG feeding reduced the risk of significant aspiration complications.

The aim is to provide clinicians and their patients with quantitative data on survival and aspiration risk to help inform and facilitate clinical decision making.

Methods

Data collection for UK patients from the York and Scarborough Teaching Hospitals NHS Foundation Trust came from two sources. The

first was a prospective register maintained by one of the authors, comprising patients reviewed by himself and one other specialist in movement disorder clinics at the Scarborough and Bridlington Hospitals (2009–2024). This register was updated according to each patient's most recent clinical diagnosis prior to death, in line with International Parkinson and Movement Disorder Society criteria. From this source, 78 deceased patients with a definitive diagnosis of MSA, PSP or CBD were identified. The second was the hospital register of patients seen throughout the hospital trust who had a clinic code of "atypical parkinsonian syndrome". This identified a further 286 deceased patients at time of search in 2024. The two lists of patients were cross-checked and duplicate patient entries were identified and eliminated.

Data collection for New Zealand patients in the Wellington Region was sourced from the hospital register for the Capital and Coast District Health Board between 2010 and 2024. This database included patients seen at Wellington Regional Hospital, Hutt Hospital and Kenepuru Community Hospital. A search was completed for patients who were deceased and had a clinic code of "multiple systems atrophy", "progressive supranuclear palsy", "degenerative disease of basal ganglia, unspecified", "extrapyramidal and movement disorder, unspecified" or "dementia with Lewy bodies". This identified 189 patients.

Patient letters were extensively reviewed and those without a diagnosis of MSA, PSP or CBD at death were excluded. Patients who were lost to follow-up or had insufficient clinical data were not included in the final analysis. Where there was no evidence of reported dysphagia, these patients were also excluded. A summary of patient identification and exclusions is shown in Figure 1.

First report of dysphagia was defined from the earliest date of any of the following: clinic letter describing episodes of aspiration or the patient noticing a problem with dysphagia, referral to speech and language therapy for swallow assessment or presentation to the emergency department or hospital admission for an aspiration event. Survival time was measured from the onset of dysphagia until death. Hospital admission for aspiration pneumonia was recorded if the discharge letter had "aspiration pneumonia" as a diagnosis, or the chest X-ray on initial assessment demonstrated a right lower lobe pneumonia. Presentations to the emergency department that did not result in admission were

not included.

Statistical analysis

All patients were deceased at database closure (31 December 2024), so follow-up times represent uncensored durations measured from the onset of dysphagia. Survival was assessed using Kaplan–Meier (KM) methods and Cox proportional-hazards regression. Median survival times with 95% confidence intervals (CIs) were estimated from KM curves. Hazard ratios (HRs) with 95% Wald CIs and p-values were obtained from Cox models.

Incidence rates for aspiration pneumonia admissions were calculated as the number of events divided by person-years of observation, with 95% CIs obtained from exact Poisson limits. To compare rates between groups, Poisson regression models were fitted with log person-time as an offset, and incidence rate ratios (IRRs) with 95% Wald CIs and p-values were reported.

For both Cox and Poisson models, age and sex were evaluated as potential confounders. Adjustment was considered meaningful if it changed the effect estimate (HR or IRR) by >10%, in which case adjusted estimates were reported as primary. Additional sensitivity analyses were applied to address outcome-specific residual biases. For survival, an age-range restriction (52–73 years) was applied to mitigate age imbalance between non-PEG and PEG groups. For aspiration outcomes, we produced a stage-matched comparison of late-stage non-PEG versus post-PEG follow-up. Late-stage non-PEG was defined as the final 37.4% of each individual's follow-up (corresponding to the median fraction of observation time to PEG insertion among PEG patients, 0.626).

All analyses were conducted in R (version 4.4.2; R Foundation for Statistical Computing, Vienna, Austria).

Ethics statement

This study was conducted as an observational retrospective analysis using fully anonymised patient data. No ethics approval was required, as per institutional and national guidelines. Patient confidentiality was strictly maintained. No identifiable information was used in data collection, analysis or reporting.

Results

The study sample characteristics are described in Table 1. In the non-PEG group (n=60), the

median age at dysphagia onset was 76.0 years (interquartile range [IQR] 69.2–80.0; range 54.0–95.0). In the PEG group (n=12), the median age was 64.0 years (IQR 60.0–69.5; range 52.0–73.0).

The full breakdown of survival outcomes is presented in Table 2. Median survival from dysphagia onset was 17.4 months (95% CI 14.0–24.9) in non-PEG patients versus 48.8 months (95% CI 44.8–upper limit not reached) in PEG patients. In Cox regression, PEG was associated with a lower risk of death (age/sex-adjusted HR 0.38, 95% CI 0.18–0.81; $p=0.013$). The crude estimate was similar (HR 0.32, 95% CI 0.16–0.65; $p=0.001$), indicating limited confounding by demographics. In a sensitivity analysis restricted to patients aged 52–73 years, the survival benefit of PEG persisted (age/sex-adjusted HR 0.34, 95% CI 0.15–0.81; $p=0.015$). Full KM survival curves are shown in Figure 2. Median time to PEG insertion was 28.3 months, and median survival following PEG insertion was 12 months.

The full breakdown of aspiration pneumonia outcomes is presented in Table 3. In the total cohort, non-PEG patients experienced 34 aspiration pneumonia admissions over 106.3 patient-years (0.32 per patient-year, 95% CI 0.22–0.45), while PEG patients experienced 24 admissions over 46.0 patient-years (0.52 per patient-year, 95% CI 0.34–0.78). The crude IRR was 1.63 (95% CI 0.96–2.74; $p=0.066$), suggesting a higher incidence among PEG patients. After adjustment for age and sex, the IRR attenuated to 1.41 (95% CI 0.74–2.68; $p=0.297$), indicating that demographic confounding accounted for much of the apparent difference.

In the stage-matched analysis, late-stage non-PEG patients had 27 aspiration pneumonia admissions over 39.8 patient-years (0.68 per patient-year, 95% CI 0.45–0.99), while post-PEG patients had 13 admissions over 17.2 patient-years (0.76 per patient-year, 95% CI 0.40–1.29). The crude IRR was 1.12 (95% CI 0.56–2.12; $p=0.744$), suggesting that disease progression also contributed to confounding of aspiration outcomes. After adjustment for age and sex, the IRR increased to 1.40 (95% CI 0.57–3.38; $p=0.452$), closely aligned with the adjusted total cohort estimate. This pattern suggests that while disease progression contributed to confounding, age and sex exerted greater influence on aspiration outcomes.

Discussion

PEG feeding presents a clinically and ethically complex decision. National Institute for Health

and Care Excellence guidance offers general recommendations for the broader population, advising PEG insertion for patients likely to require nutritional support for more than 4 weeks.⁴ International guidelines from the European Society for Clinical Nutrition and Metabolism cover the use of PEG feeding in stroke and amyotrophic lateral sclerosis (ALS).³ However, movement disorders lack robust data on enteral feeding outcomes, further complicating decision making.

The low prevalence of atypical parkinsonian syndromes significantly limits opportunities to conduct research and establish guidelines. While idiopathic Parkinson's disease is a relatively common condition, the prevalence of PSP and CBD is markedly lower, at 6.92 and 3.91 per 100,000 population respectively.⁵ As a result, despite drawing on databases from two tertiary centres dating back to 2009, our sample size remained small. This contributed to considerable heterogeneity in baseline patient characteristics.

In addition to challenges posed by low prevalence, high variability in local clinical practice presents a further obstacle to generating consistent data in this population. In our study, the PEG insertion rate for dysphagic patients was 22.9% (n=11) in the UK compared with just 4.2% (n=1) in New Zealand. Although this reduces the generalisability of our findings, it underscores the need for clearer guidance. The study is limited to just two sites and did not aim to elucidate the many patient and clinician characteristics that influence decision making. However, the observation is consistent with the variability seen in PEG placement for other conditions.

Atypical parkinsonian syndromes represent unique challenges in terms of PEG feeding decision making. Although these conditions share some clinical features with idiopathic PD, their disease trajectories—particularly with respect to dysphagia—differ markedly. Dysphagia typically emerges late in PD, whereas in MSA, PSP and CBD it appears earlier and is often more severe.^{1,6} One retrospective study reported the median latency to dysphagia as 130 months in PD, compared with 67 months in CBD, 64 months in PSP and just 42 months in MSA.⁶ Atypical parkinsonian syndrome patients also experience more rapid functional decline, partly due to poor responsiveness to dopaminergic therapy.¹ Hence, while dysphagia in PD often coincides with late-stage frailty, patients with atypical parkinsonian syndromes may require decision making regarding enteral support earlier in the disease course.

The key finding of this study is the demonstration that life expectancy increased with PEG placement. For patients and physicians wanting mortality data specifically for atypical parkinsonian syndromes this is important. However, limitations should be noted as selection bias, attrition rate and immortal time bias may have had a significant impact.

The retrospective design limited the ability to comprehensively document patient characteristics. While the results remained significant after adjusting for age, a more relevant measure would have been clinical frailty. Patients with rapidly progressive disease or advanced frailty are unlikely to be deemed suitable for PEG insertion. Consequently, survival benefit may have been exaggerated by selection bias, with fitter patients being more likely to receive PEG, and therefore more likely to survive. This is corroborated by clinic documentation showing gastrostomy was not discussed for 70% of the non-PEG group. Cognitive status was also not recorded. This is important given there is strong evidence that PEG insertion is of limited benefit in dementia patients.⁷ Although both frailty and cognition were likely taken into consideration by treating physicians in individual patient discussions, a prospective approach would enable use of a standardised clinical frailty score and cognitive assessment tools.

Beyond selection for PEG insertion, attrition also affected baseline recruitment. A significant drop-off was observed between initial patient identification and final inclusion. This largely reflected the broad clinic codes used in our searches, which captured a wide range of conditions. In the UK, many patients coded as “atypical parkinsonism” were later excluded due to overlap with other diagnoses (e.g., Lewy body dementia, vascular parkinsonism, drug-induced parkinsonism, essential tremor, chorea, myoclonus, normal pressure hydrocephalus). In New Zealand, non-specific codes such as “degenerative disease of basal ganglia” and “extrapyramidal and movement disorder, unspecified” produced similar challenges. Given the diagnostic uncertainty of atypical parkinsonian syndromes, this approach was necessary to avoid missing eligible patients. However, it meant that only a minority had a confirmed diagnosis of MSA, PSP or CBD at death. Very few patients were excluded solely for lack of dysphagia, although the exact number was not recorded.

Alongside selection and attrition, immortal time

bias represents another source of overestimation. Survival in the PEG group was calculated from the onset of dysphagia, therefore patients who survived long enough to undergo PEG insertion contributed a period of guaranteed survival before the intervention. This inherent bias may have artificially inflated the apparent survival benefit associated with PEG, and future prospective studies would require predefined entry points to mitigate these effects.

Kobylecki et al. (2024) conducted a similar UK study of patients with MSA, PSP and CBD, but restricted their cohort to individuals who had been offered PEG insertion.⁸ Outcomes were compared between those who proceeded with PEG and those who declined, with survival measured from the date of recommendation. This design reduced the impact of both selection and immortal time bias. Median survival was 24 months (95% CI 14.9–33.1) in the PEG group and 12 months (95% CI 8.2–15.8) in the non-PEG group. Their findings align with the survival advantage observed in our study, though the smaller effect size likely reflects the reduction in bias.

The second major outcome of this study is that PEG placement did not reduce aspiration pneumonia in atypical parkinsonian syndrome patients. This is consistent with previous research showing that PEG does not necessarily lower aspiration risk,¹ and it is an important consideration when discussing risks and benefits with patients. Several factors may explain this result, including confounding by disease progression, age and sex, as well as the inherent challenges of isolating the influence of PEG insertion on aspiration pneumonia.

With advanced disease progression, aspiration of oropharyngeal secretions (“silent aspiration”) remains a major risk factor even in the absence of oral intake. The stage-matched analysis was intended to mitigate this influence by aligning disease stage between groups. While the unadjusted IRR showed a smaller difference in aspiration risk, adjustment for age and sex yielded an estimate almost identical to the adjusted total cohort. This indicates that age and sex exerted greater confounding influence than disease progression in this study. Given that increasing age and male sex are established risk factors for aspiration, this finding is not unexpected. More broadly, it highlights the difficulty of assessing aspiration outcomes retrospectively, since even basic demographic factors can substantially distort results.

There are further limitations when attempting

to isolate the exact effect of PEG insertion on aspiration risk. Aspiration pneumonia is a difficult outcome to measure, as it relies on clinician judgement rather than objective criteria. The presence of a PEG tube may also bias clinicians towards attributing respiratory infections to aspiration. In addition, some enterally fed patients continue limited oral intake for comfort, further complicating assessment. This study encountered common methodological difficulties in recording aspiration data. We sought to minimise under-ascertainment by including right lower lobe pneumonia in data extraction, but this may have inadvertently captured cases of community-acquired pneumonia. While admission rates are a common method of measuring aspiration, some patients may have had conservative escalation plans, with events managed in the community rather than in hospital.

Overall, these findings suggest that retrospective analyses are limited in their ability to disentangle the influence of PEG on aspiration outcomes. Given the strong effect of basic demographic confounders and the inherent challenges of quantifying and controlling for aspiration, a substantially larger and higher-powered study would have been required to address this question definitively.

A multicentre study by Marois et al. (2017) investigated outcomes following gastrostomy in 32 patients, 78% of whom had an atypical parkinsonian syndrome.⁹ Median post-procedure survival was just 186 days, and aspiration pneumonia was the most frequent complication. Their findings are consistent with our observation that PEG insertion does not prevent aspiration in this population. However, median post-PEG survival in our cohort was substantially higher at 365 days. This variation highlights the need for larger international registries to better characterise the risks, benefits and optimal timing of PEG insertion in atypical parkinsonian syndromes.

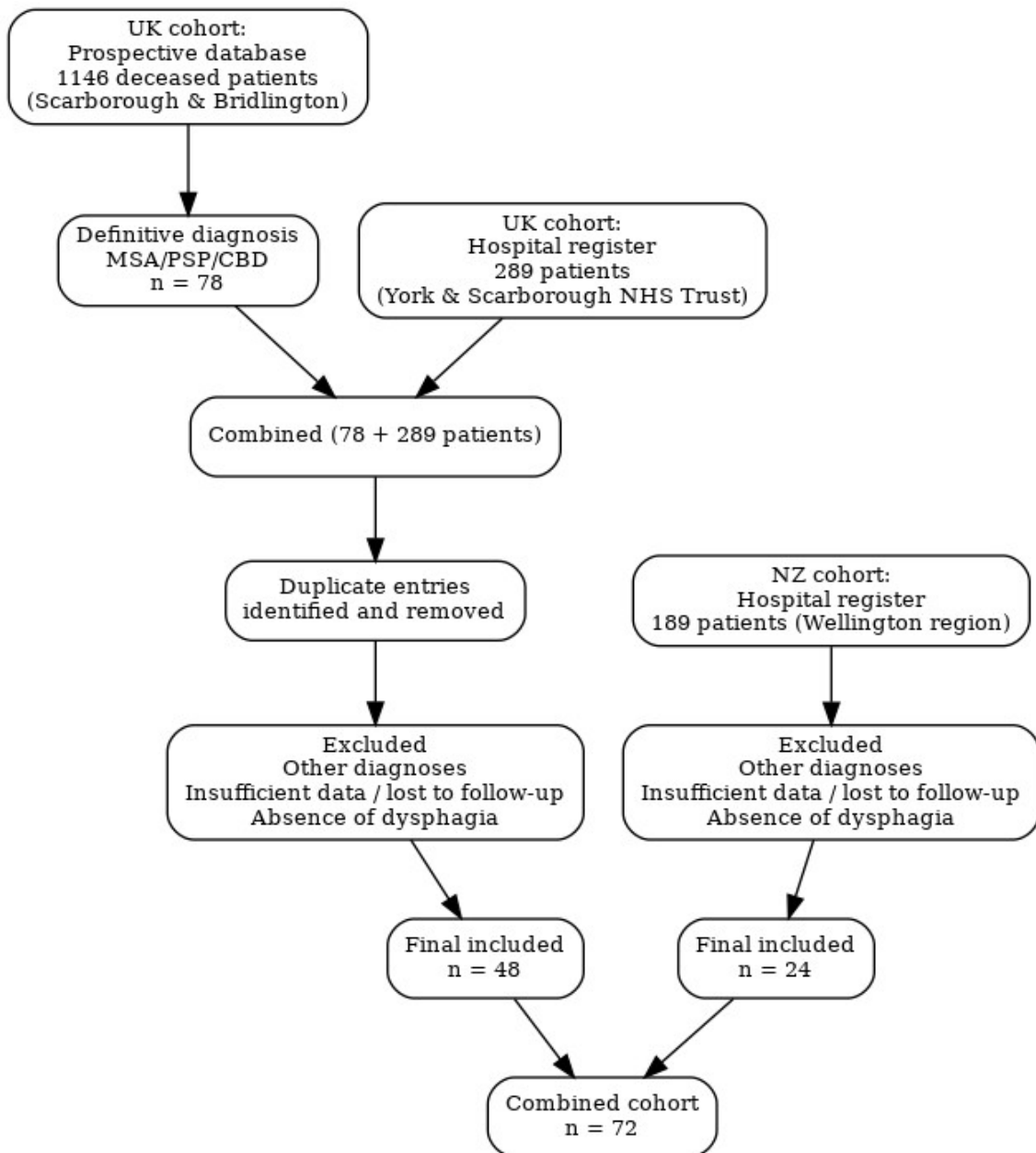
While survival data and aspiration risk can inform PEG decision making, patients and families often focus on other meaningful factors, such as quality of life (QoL) and nutrition. Existing

evidence demonstrates an association between inadequate nutrition and sarcopenia, frailty, fatigue, immunodeficiency, cognitive decline, depression and hospitalisation.¹⁰ Hence, it would be intuitive to expect enteral support to improve QoL.

Given their comparable neurological burden, studies with ALS populations may offer relevant comparators for atypical parkinsonian syndromes. In a small study of 13 ALS patients, Körner et al. (2013) found that 84.6% reported improved QoL, with 76.9% experiencing weight gain or stabilisation.¹¹ In contrast, a qualitative study by van Eenennaam et al. (2023), which explored the lived experiences of 14 ALS patients, found the relationship with PEG feeding to be more nuanced.¹² Some participants were pleased with functional improvements such as weight gain and described relief after PEG insertion alleviated the distress of mealtimes. Others expressed a loss of independence (needing help to manage feeds) and a sense of being altered by the presence of the tube. These views emphasise the importance of individualising care and recognising that clinical benefit does not always align with what patients value most.

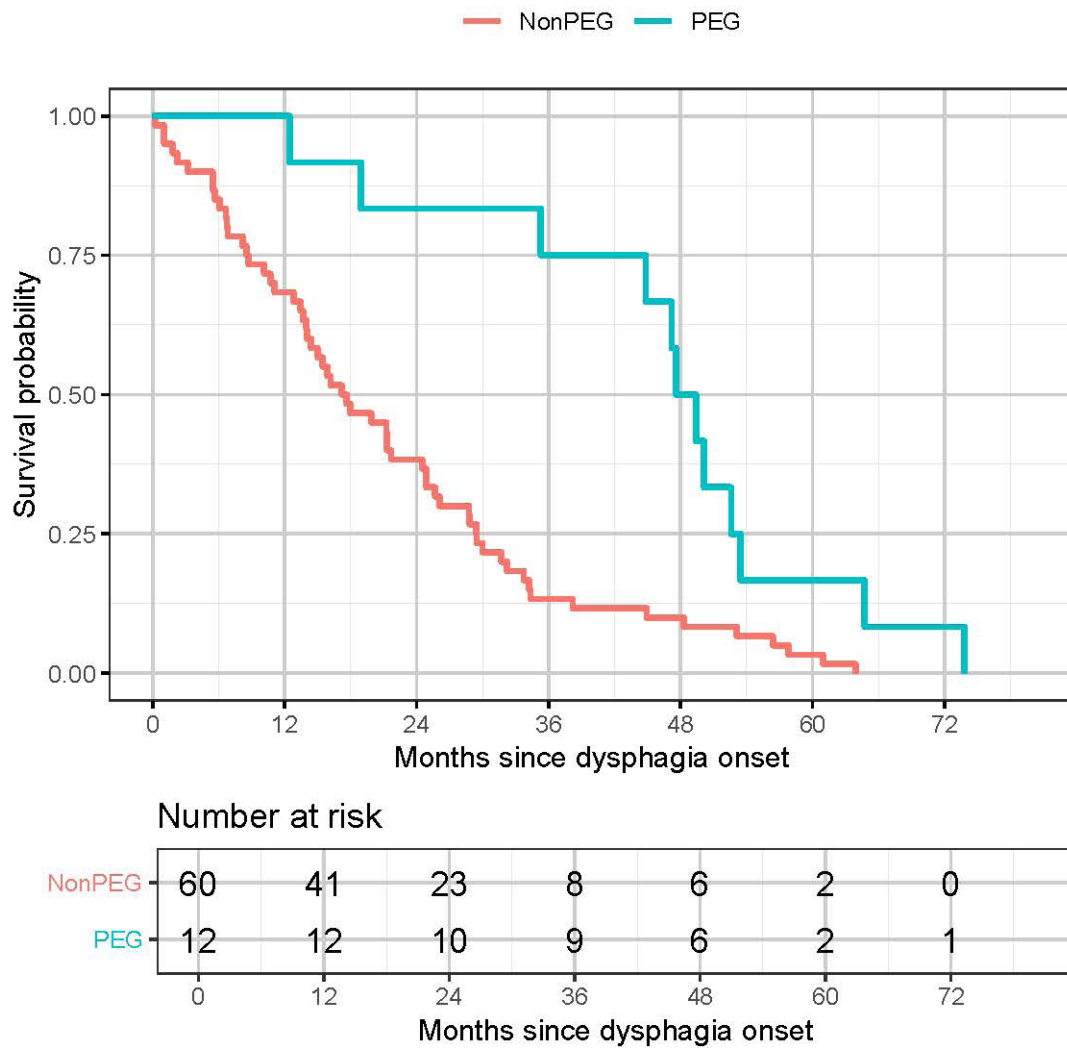
In conclusion, this study represents the first international research examining life expectancy and aspiration risk following PEG placement in a cohort of atypical parkinsonian syndrome patients. The results demonstrate that PEG insertion was associated with improved survival, with a median advantage of 27.4 months. This is broadly consistent with findings from similar populations. However, interpretation must account for substantial heterogeneity and selection bias. While aspiration pneumonia is often a key concern for patients and clinicians, we found no significant difference in hospital admission rates.

Prospective studies are needed to further evaluate the risks and benefits of PEG feeding in atypical parkinsonian syndromes, with particular attention to nutritional outcomes and QoL. Future research in this field will benefit from multicentre collaboration to enable recruitment of larger and more representative cohorts.

Figure 1: Patient selection flow diagram.

Patients identified from United Kingdom (UK) and New Zealand databases were screened for atypical parkinsonian syndromes. After removal of duplicates (UK) and exclusion for alternative diagnoses, insufficient data/lost to follow-up, or absence of dysphagia, 48 UK and 24 New Zealand patients were included, giving a combined cohort of 72.

Figure 2: Kaplan–Meier survival curves comparing percutaneous endoscopic gastrostomy (PEG) and non-PEG patients from the onset of dysphagia.



The red line represents patients who received PEG, while the blue line represents those who did not.

Table 1: Characteristics of patients with and without percutaneous endoscopic gastrostomy (PEG) insertion.

Characteristic	PEG not inserted N/60(%)	PEG inserted N/12(%)
Gender		
Female	18 (30)	3 (25)
Male	42 (70)	9 (75)
Diagnosis		
MSA (multiple system atrophy)	17 (28)	5 (42)
PSP (progressive supranuclear palsy)	31 (52)	6 (50)
CBD (corticobasal degeneration)	12 (20)	1 (8)
Country of residence		
United Kingdom	37 (62)	11 (92)
New Zealand	23 (38)	1 (8)

Patient demographics, diagnoses and country of residence are shown by PEG status. Baseline characteristics, except for age and country of residence, were broadly similar between both groups.

Table 2: Survival analysis (percutaneous endoscopic gastrostomy [PEG] versus non-PEG).

Analysis	Group	N patients	Median survival, months (95% CI)	HR (95% CI, p)	HR sex/age adjusted (95% CI, p)
Total cohort	Non-PEG	60	17.4 (14.0–24.9)	-	-
	PEG	12	48.8 (44.8–NA)	0.32 (0.16–0.65, 0.001)	0.38 (0.18–0.81, 0.013)
Sensitivity (52–73 years)	Non-PEG	25	17.9 (14.4–32.2)	-	-
	PEG	12	48.8 (44.8–NA)	0.39 (0.19–0.83, 0.015)	0.34 (0.15–0.81, 0.015)

95% CI = 95% confidence interval; HR = hazard ratio.

Median survival times and hazard ratios (HRs) from Cox regression are shown for the total cohort and for a sensitivity analysis restricted to patients aged 52–73 years. Both crude and age/sex-adjusted estimates are presented.

Table 3: Aspiration pneumonia outcomes (percutaneous endoscopic gastrostomy [PEG] versus non-PEG).

Analysis	Group	Admissions	Person-years	Rate per PY (95% CI)	IRR (95% CI, p)	IRR sex/age adjusted (95% CI, p)
Total cohort	Non-PEG	34	106.3	0.32 (0.22–0.45)	-	-
	PEG	24	46.0	0.52 (0.34–0.78)	1.63 (0.96–2.74, 0.066)	1.41 (0.74–2.68, 0.297)
Stage-matched	Late-stage non-PEG	27	39.8	0.68 (0.45–0.99)	-	-
	Post-PEG	13	17.2	0.76 (0.40–1.29)	1.12 (0.56–2.12, 0.744)	1.40 (0.57–3.38, 0.452)

PY = person-years; 95% CI = 95% confidence interval; IRR = incidence rate ratio.

Incidence rates and incidence rate ratios are shown for the total cohort and for a stage-matched analysis (late-stage non-PEG versus post-PEG). Both crude and age/sex-adjusted estimates are presented.

COMPETING INTERESTS

EJ received support from Bial Pharma UK for attending the International Movement Disorder Society Conference in Philadelphia, October 2024.

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<https://nzmj.org.nz/journal/vol-139-no-1628/percutaneous-endoscopic-gastrostomy-in-atypical-parkinsonian-syndromes-survival-and-aspiration-outcomes-from-a-retrospective-int>

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Riluzole use and reasons for non-use in people with amyotrophic lateral sclerosis in Aotearoa New Zealand

Natalie Gauld, James Cleland, Sarah Buchanan, Joanna Hikaka, Chris Frampton, Stephen Buetow

ABSTRACT

Amyotrophic lateral sclerosis (ALS), the most common form of motor neurone disease (MND), is a neurodegenerative condition with typically short life expectancy. Riluzole, the only survival-prolonging medication funded in Aotearoa New Zealand, has high uptake in other developed countries.

AIMS: To quantify riluzole use in New Zealand, identify factors associated with its use and explore reasons for non-use.

METHODS: In 2025, people in New Zealand diagnosed with MND were invited to self-complete questionnaires. Data were collected via Qualtrics, exported to Excel and analysed using descriptive and inferential statistics. Respondents with progressive muscular atrophy or primary lateral sclerosis diagnoses were excluded from this analysis.

RESULTS: Of 115 respondents, 55 (48%) were currently taking riluzole, 14 (12%) had taken it previously and 42 (36%) had never taken it. Common reasons for non-use included riluzole not being offered and concerns about lack of effectiveness and/or side effects. Uptake was lower with bulbar onset than limb onset ($p < 0.05$).

CONCLUSIONS: People with ALS in New Zealand have low uptake of riluzole, despite its survival benefits. Prescribers and people with ALS need up-to-date information about riluzole's benefit-risk profile to increase uptake and confidence in prescription and use. Liquid riluzole is needed in New Zealand to aid uptake.

Amyotrophic lateral sclerosis (ALS) is a devastating neurodegenerative disease that causes progressive paralysis, with patients facing a median survival of around 3 years from symptom onset.^{1,2} Limited therapeutic interventions improve survival, though non-invasive ventilation³ and a multidisciplinary team approach⁴ have demonstrated meaningful benefits.

In Aotearoa New Zealand, riluzole stands as the sole approved and funded medication prolonging survival in ALS patients. Pooled analysis of three randomised controlled studies (RCTs) found at 12 months an average survival benefit of 2–3 months and slower decline in limb and bulbar function.⁵ One of these studies found reduced mortality of 38.6% at 12 months and 19.6% at 21 months.⁶ However, methodological limitations likely underestimated riluzole's therapeutic potential,⁷ with participants beginning therapy an average of 2 years post-diagnosis, relatively short study durations and exclusion of participants surviving the trial period.⁷ Observational evidence is inherently less robust than well-conducted RCTs but can address some of these deficiencies. Most more recent observational

studies, which include longer follow-up and earlier therapy initiation, suggest that riluzole may confer a median survival benefit of 6–19 months.^{7,8}

Pharmac, New Zealand's drug funding agency, has funded riluzole tablets in New Zealand since 1 October 2013,¹⁰ under special funding arrangements (Table 1).¹¹ Because people with ALS often have swallowing difficulties or rely on feeding tubes, the lack of a liquid option in New Zealand is a major practical challenge to administration. Australia, the United Kingdom, the United States and Spain, among other countries, address this problem by providing and publicly funding riluzole in tablet and liquid form.

The study aim was to assess riluzole uptake by people with ALS in New Zealand, explore reasons for non-use and discontinuation and examine adherence patterns to inform policy decisions around riluzole funding and formulation provision.

Methods

The Central Health and Disability Ethics Committee approved the MND Insight Research (2025 EXP 21804). This nationwide study explored the

Table 1: Subsidy criteria for riluzole in New Zealand.¹¹

<p>Special authority criteria for funding</p> <p>Initial application</p> <p>This application can only be made by a neurologist or respiratory specialist, with approvals valid for 6 months. The criteria require that the patient:</p> <ul style="list-style-type: none"> • has amyotrophic lateral sclerosis with disease duration of 5 years or less, and • has at least 60% of predicted forced vital capacity within 2 months prior to the initial application, and • has not undergone a tracheostomy, and • has not experienced respiratory failure, and • is ambulatory, or can use upper limbs or is able to swallow <p>Renewal applications</p> <p>These applications are from any relevant practitioner with approvals valid for 18 months before reapplication is necessary. The criteria require that the patient:</p> <ul style="list-style-type: none"> • has not undergone a tracheostomy, and • has not experienced respiratory failure, and • is ambulatory, or able to use upper limbs or is able to swallow
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experiences and needs of people affected by motor neurone disease (MND) and their families. It recruited three participant groups: people with MND, family members and bereaved individuals. Only participants with MND were asked about riluzole use. Eligible participants were over 16 years of age, had a diagnosis of MND and lived in New Zealand. Family carers and the bereaved were defined as the key supporter of a person diagnosed with MND who either was currently living with MND (family carers) or had died in the last 2 years (bereaved). Exclusion criteria applied to anyone under 16, individuals not residing in New Zealand and those without a diagnosis of MND.

Recruitment occurred through two primary channels from 11 March to 20 April 2025. Motor Neurone Disease New Zealand (MND NZ) emailed study invitations and participant information sheets on four occasions to clients accepting communications, reaching 247 people with MND. Those without email addresses who accepted communications received postal invitations once. MND NZ support advisors could also mention the research at their discretion to clients not on the communications list. Additionally, the New Zealand MND Registry emailed invitations four times to their 201 registrants with MND, most of whom were likely also MND NZ clients.

The research tailored questionnaires for each participant group. This paper reports on the questions for people with MND ascertaining riluzole use (including reasons for non-use and discontinuation), self-reported riluzole adherence, clinical history (diagnosis and symptom onset dates, plus initial symptoms), the healthcare setting for the diagnosis (public versus private), demographic information, and the ALS Functional Rating Scale Revised (ALSFRS-R). Participants reported their specific MND diagnosis from options including ALS, primary lateral sclerosis, progressive muscular atrophy, progressive bulbar palsy, and “*I only know it as MND*” or “*other, please specify*”. Incidental references to riluzole by family members and bereaved participants in any open-ended responses were included in the data analysed.

Disease progression rates were calculated using a standardised formula: subtracting the current ALSFRS-R score from 48 (the maximum possible score) and dividing this by the number of months since symptom onset. Given that New Zealand restricts riluzole funding to people with ALS, the analysis excluded respondents with diagnoses of primary lateral sclerosis, progressive muscular atrophy, neuronal intranuclear inclusion disease or any other variants that two neurologists agreed were unlikely to be ALS. Multiple completion methods accommodated

participant preferences and abilities. People with MND could complete questionnaires online (with Qualtrics), on paper or by telephone. Family members and the bereaved used online or paper formats only. All paper and telephone responses were entered into Qualtrics for consistency.

Data were exported from Qualtrics into Excel. Duplicates and those with insufficient data were removed. For people with MND, responses from the three questionnaires were matched by individual participant to create one complete file. The analysis employed descriptive statistics as the primary approach, with Fisher's exact tests for nominal data and Mann–Whitney U tests for interval data to assess statistical significance. Qualitative responses were analysed by grouping similar responses, with quotes based on typicality or informativeness.

Results

Of the estimated 285 people with MND who received written information about the research, 142 completed the first questionnaire (49.8% response rate). This represents 35.5% of the estimated 400 people with MND in New Zealand. Twenty-seven responses were excluded based on a diagnosis of primary lateral sclerosis (n=18), progressive muscular atrophy (n=6) or other diagnoses considered atypical ALS (n=3), leaving 115 responses for analysis.

The sample comprised 74 males (64.3%) and 41 females (35.7%). Most participants were born in New Zealand (n=95; 82.6%). Most (87.8%) identified as NZ European, with seven (6.1%) identifying as Māori, two (1.7%) as Pacific peoples, three (2.6%) as Chinese, two (1.7%) as Indian and six reporting other ethnicities—all European (5.2%). Two-thirds of the participants (n=76, 67.1%) had been diagnosed in 2022 or later (range 2000–2025). The median age was 67 years with 25% aged 58 years or less, and 25% aged 72 years or older. Thirty respondents (26.3%) appeared to have bulbar onset, 84 (73.7%) had spinal onset and one onset was unclear.

Sixty per cent of the sample had taken riluzole before, with 48% currently taking this medication (Table 2), reflecting a 20.3% discontinuation rate.

Those who had never taken riluzole cited various reasons (Table 2), with most reporting having never been offered it, never having it prescribed or having not heard of it. Five respondents said their neurologist had recommended against it, did not recommend it or would not prescribe it

for them.

Concerns about lack of effectiveness were common, with participants sometimes reporting that their neurologist had indicated the medication had little effect. Two respondents reported that a 3-month survival gain was insufficient justification.

“After being given the information that I may only get around 3 extra months of life I opted not to take it and instead started taking natural supplements...”

Other reasons for declining riluzole included the view that the risk of side effects and impact on quality of life outweighed the potential benefits:

“After consultation with my neurologist, I decided that the possible slight life extension was not worth the side effects. Plus I would've had to give up coffee, and coffee brings me joy.”

Indeed, among the 14 respondents who had discontinued riluzole, the most common reason was side-effects, including itchy rash, tiredness, nausea, diarrhoea, blurred vision and “kidney issues”. One person reported that riluzole “compromised my immune system”, resulting in hospitalisation with a high temperature. Two of the three respondents who stopped because of concerns about lack of effectiveness noted no benefit.

Of those currently taking riluzole, 43 (81.1%) reported missing no doses in a typical week. Reasons for missing doses were attempting to time doses to avoid fatty meals or food entirely (n=5), forgetting (n=4), changes in routine (n=3) and tiredness or oversleeping (one each):

“I sometimes miss the evening one. I intend to take it two hours after dinner (empty stomach) and then I forget.”

One respondent missed doses because of swallowing difficulties but achieved complete adherence after having a feeding tube inserted. Another respondent had only ever been prescribed riluzole once daily. Several factors were associated with riluzole use (Table 3). Uptake was significantly lower among those diagnosed in the private health system, those with bulbar onset, and those diagnosed before 2013 when riluzole funding started.

One family respondent reported difficulty with

Table 2: Riluzole usage and reasons for non-use.

	N (%)
Currently take riluzole	55 (47.8)
Previously took riluzole	14 (12.2)
Never taken	42 (36.5)
Missing data	4 (3.5)
Total	115 (100)
Reasons riluzole was never taken*	N=42
Never offered it/never prescribed it/never heard of it	13 (30.9)
Concern about lack of effectiveness	10 (23.8)
Concern about side effects	7 (16.7)
Specialist recommended against it or would not prescribe it for them	5 (11.9)
Not needed	3 (7.1)
About to start (one having respiratory test first)	2 (4.8)
Other	3 (7.1)
No reason provided	3 (7.1)
Reasons for stopping riluzole*	N=14
Side effects	10 (71.4)
Insufficient effectiveness	3 (21.4)
No reason given	2 (14.3)

*Some respondents gave more than one reason.

crushing riluzole with the “casing” not dissolving and getting stuck going into the feeding tube. Another family respondent stated:

“The specialist was terrible... When asked about any meds that might help, [they] said there is one that has a history of adding 1-to-2-months, but never offered it and basically dismissed it. Well with MND, 1-to-2-months extra to a person diagnosed with it is a lot.”

Discussion

This research found that 48% of respondents with MND were currently taking riluzole, with

many not taking it due to concerns about lack of effectiveness and side effects. Some reported not being offered the medication or being advised against it. Reasons for discontinuing treatment included possible side effects and concerns about lack of effectiveness. High self-reported adherence was noted. Uptake was reduced in respondents with bulbar onset, those diagnosed by a private specialist and those diagnosed before 2013. Our uptake rate is substantially lower than rates from four European ALS centres (83% taking riluzole),⁹ an audit of French ALS centres (100% offered and 88% initiated within 2 months of diagnosis),¹² an audit from Northern Ireland (91%),¹³ an audit from a tertiary ALS centre in Pennsylvania, United States (91% initiated riluzole)¹⁴ and an

Table 3: Riluzole use by sub-groups from respondents who had ALS.

		Ever taken riluzole N (%)
Sex	Female (n=39)	23 (59.0)
	Male (n=72)	46 (63.9)
Feeding tube	Needs tube feeding (n=19)	12 (63.2)
	Does not need tube feeding (n=92)	57 (62.0)
Progression	Slow progression* (n=48)	27 (56.3)
	Intermediate progression* (n=57)	38 (66.7)
	Fast progression* (n=6)	4 (66.7)
Where diagnosed**	Diagnosed in the private system (n=50)	23 (46.0)
	Diagnosed in the public system (n=60)	45 (75.0)
Site of onset**	Limb onset (n=81)	55 (67.9)
	Bulbar onset (n=29)	13 (44.8)
Year diagnosed**	Diagnosed before 2013 (n=11)	2 (18.2)
	Diagnosed after 2013 (n=100)	67 (67.0)
Ethnicity	European (n=98)	59 (60.2)
	Māori or Pacific (n=8)	6 (75.0)

*Slow progression is ≤ 0.31 ALSFRS-R change/month, intermediate is 0.32-1.17/month, fast is ≥ 1.18 /month.

** $P < 0.05$, Fisher's exact test.

audit from three Italian regions (61-85%).¹⁵ A 2025 Australian MND patient survey found a 76% uptake, although the response rate was 8%.¹⁶ The Pooled Resource Open-Access ALS Clinical Trials (PRO-ACT) data reported 80% riluzole uptake in phase two and three ALS clinical trial participants versus 65% in the United States ALS patient registry.¹⁷ However, our uptake was higher than the rate documented in Scotland (86% offered and 40% taking riluzole).¹⁸ High uptake might reflect a different experience for patients in ALS centres versus treatment in neurology or gerontology outside of an ALS centre as is common in New Zealand. Supporting this suggestion is a finding from Spain of 30% riluzole uptake under a general neurologist versus 89% for those treated in a multidisciplinary care clinic.⁴

The special authority criteria might reduce uptake in New Zealand; however, it is expected that most people with ALS would meet the diagnostic criteria at diagnosis and none discontinuing

riluzole cited cessation of funding. New Zealand has nearly identical access criteria to Australia,¹⁹ but it likely differs from other countries.

Other research has found lower riluzole uptake with increasing age of the person with MND,^{15,18} but age was not associated with uptake in our study.

Our discontinuation rate of 20% was higher than registry data in Scotland (15%),¹⁸ a single hospital audit in Portugal (14%)²⁰ and a single clinic audit in Pennsylvania (17%)¹⁴. In the Portuguese study,²⁰ discontinuation was primarily attributed to concerns about lack of effectiveness, with only one person discontinuing due to side effects. Reasons for the Pennsylvania discontinuations included side effects (32%) with some restarted on a lower dose, and cost (15%).¹⁴ The high rate of self-reported adherence in our study aligns with the Portuguese study²⁰ and is similar to Pennsylvania where adherence was lower immediately after initiation and late in the disease.¹⁴

Implications

The low uptake in New Zealand appears largely driven by beliefs about ineffectiveness stemming from early findings that showed a small 2-to-3-month survival benefit, alongside concerns about side effects. Given the significant methodological deficiencies in these studies, and the fact that most observational studies suggest extended survival benefits of 7–11 months,^{7,9} better dissemination of information about effectiveness to diagnosing clinicians appears necessary. The riluzole data sheet (prescribing information) still references the 2-to-3-month survival benefit,²¹ and, until recently, so did the webpage for patients from MND NZ. One family member's observation of a benefit of even a short additional survival period provides a potent reminder to present survival information objectively without pre-judging treatment decisions. Other concerning prescribing practices include one person reporting being prescribed only one tablet daily and another believing they could not use riluzole with caffeine. While listed in the riluzole data sheet, caffeine is not known to be a clinically relevant inhibitor of CYP1A2 and no clinically significant interaction is expected.²² Some respondents identified concern about side effects as a reason for non-use, potentially unaware that riluzole is generally well-tolerated.²⁵

Liquid riluzole is unavailable in New Zealand, unlike many other health systems. Given that people with MND frequently have swallowing difficulties and some require feeding tubes necessitating tablet crushing—a time-consuming task that reduces drug delivery²³ and risks tube blockage—liquid riluzole is urgently needed in New Zealand. This could improve uptake in people with bulbar presentation (who had lower use) and reduce the care burden on family members who already undertake many tasks for people with ALS who often have poor hand function.

Strengths and weaknesses

The study achieved a good response rate and, beyond understanding uptake and adherence patterns, identified specific reasons for non-use, discontinuation and adherence challenges with riluzole. However, several limitations should be acknowledged.

The reliance of the research on self-reporting

may have over-estimated adherence and reduced the accuracy of other data collected, such as the ALSFRS-R scores. We used self-reported diagnoses, and some participants reporting only “MND” might have had primary lateral sclerosis or progressive muscular atrophy. However, given the life-changing nature of the disease, persons with MND likely remember accurately their onset time and diagnosis date. Disease progression rates were calculated using ALSFRS-R scores at questionnaire completion, divided by months since onset. The non-linear nature of ALSFRS-R progression²⁴ may have affected calculations compared to studies calculating this metric earlier in the disease course.

Our sample may not represent all people in New Zealand with MND. It included relatively few fast progressors and many slow progressors, likely reflecting the survival benefit of slow progression and the challenges faced by those with rapid decline. The sample included more people with higher function than lower function, possibly reflecting reduced energy and capability for completing the questionnaire as function declines. The 6-week data collection period may have limited participant numbers and faster progressor inclusion. We had relatively few non-European respondents, although this could partly reflect the relatively low numbers of Māori with MND,²⁵ and the MND prevalence in countries with high non-European ethnicity is often lower than those with European ethnicity.²⁶ We did not ask reasons for taking riluzole, which may have revealed differences in discussions with the prescriber.

Conclusion

Riluzole uptake among people with ALS in New Zealand is suboptimal, which is particularly concerning given that it is the only funded medicine shown to lengthen survival in ALS. Health services and clinicians should promote its appropriate use and update guidance to address outdated perceptions of riluzole's effectiveness and safety. Making liquid riluzole available and funded would facilitate prescribing and use in people with swallowing challenges, including those with bulbar onset ALS, and those using a feeding tube.

COMPETING INTERESTS

NG has amyotrophic lateral sclerosis and has no other interests to declare. All other authors have no interests to declare.

We accept full responsibility for the conduct of the study, for our access to the data and for our decision to publish.

ACKNOWLEDGEMENTS

We thank those from the MND community who provided input into the development of the questionnaires and who participated in the study. We thank staff at MND NZ for input into the questionnaires and informing the community about the study and the MND Registry for informing their registrants about the study. We thank health providers and our Māori reviewer for helpful input.

This research was funded by a Health Research Council Research Activation Grant, a Motor Neurone Disease New Zealand grant and some voluntary time by the authors.

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Childhood blindness prevention in Aotearoa New Zealand

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ABSTRACT

AIM: While less common than adult blindness, childhood blindness has a significant burden in terms of the total number of “blind years”. We aim to determine if there is scope for improved strategies in the prevention of childhood blindness in Aotearoa New Zealand.

METHOD: We conducted a review of New Zealand childhood blindness data.

RESULTS: In New Zealand, there is a paucity of data on childhood blindness. However, significant scope remains for prevention through optimising maternal health, neonatal care, increasing uptake of immunisations and attendance at vision screening programmes, as well as the earliest possible detection of myopia and keratoconus.

CONCLUSION: Ophthalmologists and the Royal Australian and New Zealand College of Ophthalmologists must continue to actively collaborate with obstetricians, paediatricians, general practitioners, optometrists, national screening units, vaccination programmes, epidemiologists and Health New Zealand – Te Whatu Ora to promote primary prevention strategies and improve visual outcomes for our tamariki.

Childhood blindness, while representing only 5% of worldwide blindness, ranks second as the leading cause of “blind years”. One-point-five million blind children account for 70 million blind years.¹ Childhood blindness definitions vary from visual acuity of less than 3/60 in the better eye (World Health Organization [WHO]) to 6/24 or less in Aotearoa New Zealand.^{1,2} Children under the age of 5 years have the highest incidence of blindness. Early onset blindness adversely affects the development of psychomotor, social and emotional skills. In addition to disability affecting their opportunities for education, employment and earning potential, blind children have a higher mortality rate.³

In New Zealand, more population data on childhood blindness have been published in recent years, but knowledge gaps remain. We, therefore, often extrapolate from Australian data; however, our populations differ. In New Zealand, the burden of blindness is inequitable, with nearly a quarter of blind children being Māori.³ The leading causes of childhood blindness in New Zealand are cortical vision impairment (CVI; 31.5%), retinopathy of prematurity (ROP; 18.2%) and optic nerve hypoplasia (ONH; 9%).⁴ In 2023, the Royal Australian and New Zealand College of Ophthalmologists (RANZCO) published *Vision 2030 and Beyond*, which aims to provide a road map to deliver better eye care nationwide despite challenging resource constraints in the public health sector.² This article aims to review New Zealand’s specific prevention strategies for childhood blindness.

Prevalence, incidence and cost of childhood blindness in New Zealand

The prevalence of childhood blindness in New Zealand has been reported as 5 per 10,000 children.³ We do not have data on incidence currently.

In Australia, the prevalence of childhood blindness is fairly similar at 3.4 per 10,000 children, and the incidence rate is close to one per 10,000 live births.⁵ Estimated direct healthcare costs in Australia have been evaluated at around AU\$30,000 per year per child, while indirect costs such as education, support services and caregivers’ loss of productivity have been estimated at around AU\$45,000 per year per child.^{5,6} The health burden in terms of Disability-Adjusted Life Years (DALYs) lost due to paediatric visual impairment in Australia has been estimated at 7,011 annually.⁶ Using an estimated Value of a Statistical Life Year of AU\$187,200, the total monetary value of the disease burden of 7,011 DALYs amounts to AU\$1.3 billion in 2015.⁶

Main causes of childhood blindness and preventive interventions

CVI

CVI is a decreased visual response to a stimulus due to brain injury rather than ocular disease. Brain injury can occur before, during or shortly

after birth.⁷ CVI is the leading cause of preventable childhood blindness in high-income countries, with as many as 50% of cases being preventable.⁸ CVI accounts for 31.5% of childhood blindness in New Zealand.⁴ Of these cases, CVI is idiopathic in 36% but is caused by perinatal hypoxia/asphyxia in 18–25% and non-accidental injury in 8%.⁷ Rarer causes include hydrocephalus, severe epilepsy, neonatal central nervous system infections, prematurity or genetic conditions. It has also been associated with antenatal maternal drug use. There is no treatment for CVI; therefore, prevention is a priority.⁷ To reduce the prevalence and impact of this condition, we summarise potential interventions in Table 1.

ROP

ROP affects premature infants weighing $\leq 1,250$ grams and born before 30 weeks gestation. It is

caused by incomplete retinal vascularisation secondary to oxygen-induced damage, although rates have been lower in recent years. There are multiple risk factors, including gestational weight/age and oxygen supplementation. ROP can lead to a spectrum of visual changes ranging from myopia, strabismus, amblyopia and anisometropia to blindness from retinal detachment.⁹ Worldwide, ROP is the primary cause of blindness in premature infants. In New Zealand, ROP accounts for an estimated 18% of childhood blindness and low vision.⁴ There is a higher rate of prematurity among Māori children.⁹ To reduce the prevalence of this condition, we summarise potential interventions in Table 2.

ONH

ONH is a congenital condition in which the optic nerve under-develops during pregnancy.

Table 1: Specific interventions to prevent cortical vision impairment.

Type of prevention	Specific interventions
Primary prevention	Optimal access to, and provision of, antenatal care to prevent premature birth.
	Optimal access to, and provision of, obstetric and perinatal care to prevent perinatal hypoxic brain injury.
	Optimal access to, and provision of, postnatal care.
	Interventions to reduce maternal alcohol and other drug use (e.g., opioids, methamphetamine).
	Interventions to reduce sexually transmitted infections (e.g., to prevent congenital syphilis and congenital herpes simplex virus infection).
	Interventions to reduce intentional and non-intentional injuries in pregnancy (e.g., family violence preventive interventions; seat belt use and other road safety interventions).
Secondary prevention*	Screening for maternal alcohol/drug use and then appropriate provision of treatment services.
	Screening for family violence or other exposure to intentional injuries during pregnancy.
Tertiary prevention**	Access to assessment for early diagnosis and intervention to optimise visual function (e.g., management of associated refractive errors, strabismus and amblyopia).
	Referral to the Blind and Low Vision Education Network New Zealand to reduce the impact of disability.

*Secondary prevention involves detecting disease at an early stage and intervening to halt or slow its progression.

**Tertiary prevention involves reducing the impact of an already established disease by preventing complications and improving quality of life.

Table 2: Specific interventions to prevent retinopathy of prematurity.

Type of prevention	Specific interventions
Primary prevention	Optimal access to, and provision of, antenatal care to prevent premature births.
	Administration of steroids to women with impending premature delivery. ⁹
	Minimisation of mechanical ventilation when not absolutely indicated. ⁹
	Minimisation of oxygen saturation fluctuations. ⁹
	Minimisation of blood transfusions when not absolutely indicated. ⁹
	Optimal access to, and provision of, neonatal and postnatal care: specifically, adequate nutrition and use of human milk where possible to encourage good postnatal growth. ⁹
Secondary prevention	Ensure ophthalmic screening of all infants $\leq 1,250\text{g}$ at birth and/or born at <30 weeks gestation to detect treatable disease.
	Early treatment with anti-vascular endothelial growth factor (VEGF) injections and laser if required.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.
	Access to ophthalmic care to manage associated refractive errors, strabismus and amblyopia, which can worsen visual function.

Table 3: Specific interventions to prevent optic nerve hypoplasia.

Type of prevention	Specific interventions
Primary prevention	Interventions to reduce maternal smoking, alcohol and other drug use (e.g., opioids, methamphetamine).
	Appropriate sexuality education in schools to reduce the incidence of teenage pregnancies.
	Optimal access to, and provision of, family planning/sexual health services to reduce the incidence of teenage pregnancies.
	Optimal access to, and provision of, antenatal care.
Secondary prevention	Screening for maternal smoking/alcohol/drug use and then appropriate provision of treatment services.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.
	Access to ophthalmic care to manage associated refractive errors, strabismus and amblyopia, which can worsen visual function.

It can present unilaterally or bilaterally and is not progressive.^{10,11} It can occur in isolation; however, it is commonly associated with cerebral midline structure abnormalities and pituitary axis hormone deficiencies.¹¹ In New Zealand, ONH accounts for an estimated 9% of childhood blindness and low vision.⁴ It is the third most common cause of visual impairment in Māori children. Visual acuity can range from near normal to no light perception. A current hypothesis is that ONH is caused by vascular disruption during pregnancy. The risk factors are increased first-trimester bleeding, maternal smoking, maternal alcohol consumption, young maternal age, maternal diabetes, preterm labour, primiparity and use of abortifacients, anticonvulsants or antidepressants.¹⁰ There is no treatment for ONH; therefore, prevention is a priority.⁸ To reduce the prevalence of this condition, we summarise potential interventions in Table 3.

Refractive error (RE)

In 2006, the WHO recognised uncorrected RE as an important cause of vision loss. By broadening this definition, the estimated total number of visually impaired people worldwide effectively doubled.¹²

Twelve-point-eight million children aged 5–15 years are visually impaired from uncorrected RE worldwide, representing 8.3% of all visual impairment. There are three main types of RE: myopia, hyperopia (or hypermetropia) and astigmatism.¹³ In New Zealand, an estimated 24% of 7–10-year-olds had RE on a recent school-based screening project. Only half of those children were regularly wearing glasses.¹⁴

Myopia is predominantly caused by an increased axial length. While genetics play an important role, increasing evidence suggests that environmental factors—particularly limited exposure to natural light and extended periods of near work (such as reading, screen use or other close-up visual tasks) in low-light conditions—also contribute significantly.¹⁵ There are predictions that half the world will be myopic by 2050.¹⁶ Hyperopia is thought to be genetic and can delay visual development. It is a significant risk factor for strabismus and amblyopia. Astigmatism is mainly caused by an excessive corneal curvature. RE commonly coexists with other paediatric eye disorders. Therefore, the management and treatment of RE should be the initial strategy for all eye conditions.^{2,13} The main focus of RE prevention lies in preventing myopia and reducing

Table 4: Specific interventions to prevent refractive error.

Type of prevention	Specific interventions
Primary prevention	Public awareness campaigns targeted at parents of young children. Ministry of Education policy changes about outdoor time at schools and minimising near activities (or following the 20/20/20 rule) for children. Such outdoor time of course requires appropriate sun protection (shade cover, hats and sunblock etc) for some of the year.
Secondary prevention	Screening: Add an autorefractor to the Year 7 (age 11–12) vision and hearing technician school vision check to detect and refer early myopia to appropriate clinics.
	Screening and early optometry care: Provide broader public funding to improve access to glasses and optometry care (especially for more deprived communities).
	Family history of refractive error is to be actively sought by general practitioners, paediatricians, midwives and Plunket nurses.
	Provision of funding by Pharmac for appropriate provision of atropine eyedrops, which are proven to slow myopia progression. ¹⁶
	Support the New Zealand Association of Optometrist's recommendation for enabling funding of MiYOSMART and Stelless myopia-prevention spectacles. ²
	Establish publicly funded optometry care (for example, at public hospitals or via funding contracts for community optometrists) to actively manage children with progressive myopia.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.

the prevalence of this condition. We summarise potential interventions in Table 4.

Amblyopia

Amblyopia can be caused by uncorrected RE, strabismus or deprivation. A 2022 review of 97 studies, including 4,645,274 children and 7,706 with amblyopia, reported an overall worldwide pooled prevalence of amblyopia of 1.36%.¹⁷ The only New Zealand data detected a prevalence of 1.8%.¹⁸ The *National Vision and Hearing Screening Protocols*, updated in 2021, specify the training and procedural requirements for vision-hearing technicians responsible for conducting childhood vision screening during the B4 School Check for 4–5-year-old children.¹⁹ The uptake of such screening in New Zealand is 92%.¹⁹ The long-term functional impacts of living with amblyopia are limited, and the main consequence of childhood amblyopia is bilateral vision impairment if loss of vision in the better eye occurs. Two New Zealand studies have shown a low positive predictive value (31%) but also a high negative predictive value (98%) for screening, so there is scope for optimising the prevention of childhood amblyopia. Firstly, the uptake of New Zealand screening should be increased to match the rates of 99% in Europe. Secondly, the screening protocol could be refined to target the detection of amblyopia with visual acuity of 6/15 or worse, as individuals with 6/12 vision in an amblyopic eye generally retain sufficient visual function for driving, employment and everyday tasks. Thirdly, it is important to ensure that bilateral amblyopia,

representing roughly 6% of all cases, is reliably detected across all screenings. The revised 2021 national protocol supports the adoption of an automated refraction device, which will contribute to improved outcomes.²⁰ To reduce the prevalence of this condition, we summarise potential interventions in Table 5.

Keratoconus

Keratoconus is characterised by progressive corneal thinning and protrusion, leading to irregular astigmatism and visual impairment. It is a bilateral, asymmetrical disease that develops during or before puberty.²¹ As keratoconus ectasia progresses, vision correction requires glasses, specially fitted (and expensive) contact lenses and, potentially, corneal transplant surgery. The prevalence is estimated at 1.38 per 1,000 people globally.²² In Wellington high school students, keratoconus affected one in 45 Māori students, compared with one in 191 New Zealand European adolescents. This study also found that eight out of 10 individuals with keratoconus identified by screening were unaware they had this condition and thus were not actively seeking appropriate care.²³ Therefore, in New Zealand, we have substantially higher rates, with an overall prevalence of 5.2 per 1,000 and up to 22.2 per 1,000 among our Māori population. Multiple New Zealand studies have demonstrated that Māori and Pacific people have a higher prevalence of keratoconus, present with more severe disease and have a more rapidly progressing disease form.^{23,24} As a result, New Zealand has the highest proportion of corneal transplants for

Table 5: Specific interventions to prevent amblyopia.

Type of prevention	Specific interventions
Secondary prevention	Screening: Increase the uptake of vision screening at the B4 School Check. ¹⁹
	Further optimise the screening protocol to only detect amblyopia of 6/15 or worse.
	Use an autorefractor in the screening process to better detect amblyogenic factors and bilateral amblyopia.
	Early treatment of amblyopia with patching, optical penalisation and/or atropine eye drops.
	Prompt management of amblyogenic risk factors including strabismus surgery and refractive error within the amblyogenic time frame.
	Improve public funding of patches and spectacles to improve access, especially in deprived populations.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.

Table 6: Specific interventions to prevent keratoconus.

Type of prevention	Specific interventions
Primary prevention	Consideration of educational campaigns targeted at parents and teachers around children minimising eye rubbing and having any allergic eye disease appropriately managed (including lifestyle measures and the use of olopatadine eye drops).
Secondary prevention	Screening: Add an autorefractor and corneal topography to the Year 7 (age 11–12) vision and hearing technician school vision check to detect and refer early keratoconus.
	Public funding to improve access to optometry assessment for those identified as at risk for keratoconus by parents, school teachers or general practitioners, especially those in deprived communities.
Tertiary prevention	Improve access to prompt tomography diagnosis and corneal cross-linking.
	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.
	Use of the Aotearoa Research Into Keratoconus registry to ensure high-quality management of cases.
	Increase awareness among health workers of corneal donor (tissue donors) impact and need.

keratoconus among the Organisation for Economic Cooperation and Development (OECD) countries.²⁵ To prevent vision loss and decrease disease burden, timely diagnosis and management of keratoconus are crucial. Early detection with diagnostic imaging modalities, such as corneal topography or tomography, leads to early intervention in the form of corneal cross-linking, which is effective at stopping the progression of ectasia. A Cochrane review found an 80–90% relative risk reduction in progression over 12 months following corneal cross-linking.²⁶ This intervention is also cost effective.²⁵ We propose a targeted screening programme for keratoconus in New Zealand for high-risk populations. Automated refraction and corneal topography could easily be incorporated into the National Vision and Hearing Screening Programme for children in Year 7 (ages 11 and 12). Further screening should be undertaken in the later teen years to rescreen for new cases of keratoconus. Several pilot studies are currently underway in New Zealand to identify the most cost-effective screening methods. To reduce the prevalence of this condition, we summarise potential interventions in Table 6.

Genetic eye diseases

Genetic eye diseases are now a leading cause of childhood blindness. Their impact on vision ranges from mild (particularly in carriers) to bilaterally blinding, and they may be associ-

ated with systemic syndromic manifestations. Hereditary eye conditions include congenital cataracts, congenital glaucoma, inherited retinal dystrophies, retinoblastoma (RB), optic atrophy and eye malformations (including corneal opacities), along with other rarer conditions. The main inherited retinal dystrophies are retinitis pigmentosa (54%), Stargardt disease (12%) and macular dystrophy (8%), as highlighted in a 20-year retrospective observational study in Western Australia.²⁷ RB is a malignant ocular tumour with an incidence of 1/18,000 live births. With early detection, survival rates for RB are more than 95%, but it can be associated with significant visual impairment post-treatment.⁸ Preventive measures for genetic eye disease in New Zealand include genetic counselling for affected people considering their pregnancy options and pre-implantation genetic testing to select embryos that do not carry the gene for the disease. Early diagnosis, counselling and support are essential for people who are carriers of genetic disease or parents of children born with genetic eye disease. Ophthalmology is leading human genetic therapies with approximately 10 approved genetic eye therapies worldwide, and the first New Zealand child was treated recently with Luxturna.²⁸ To reduce the prevalence of these conditions, we summarise potential interventions in Table 7.

Table 7: Specific interventions to prevent genetic eye diseases.

Type of prevention	Specific interventions
Primary prevention	Genetic counselling for affected individuals to inform their decisions about future pregnancies.
	Education on consanguinity.
	Access to in vitro fertilisation and pre-implantation screening.
	Germline therapy.
Secondary prevention	Prenatal diagnosis, allowing counselling and consideration of termination of pregnancy.
	Early diagnosis with routine red reflex assessments at birth and 6 weeks. ²⁹
	Early treatment of affected infants/children.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.
	Inform parents of available national and international support groups.
	Patient enrolment to the New Zealand national Database of Inherited Retinal and Optic Nerve Disease, which can offer the option to participate in genetic studies and therapies. ³⁰

Infectious eye diseases

The most significant advance in paediatric ophthalmology in the twentieth century was childhood vaccination. The following infections can all result in visual impairment or blindness; however, vaccines are available to prevent their occurrence. These are mainly but not limited to rubella (congenital cataract, keratitis, Fuchs heterochromic iridocyclitis, glaucoma); measles (keratitis, uveitis, optic neuritis); mumps (keratitis, retinitis, optic neuritis); *Corynebacterium diphtheriae* (corneal scarring); *Streptococcus pneumoniae* (keratitis, endophthalmitis); *Neisseria meningitidis* (conjunctivitis, endophthalmitis); *Haemophilus influenzae type b* (orbital cellulitis, uveitis, vaso-occlusive retinal vasculitis, neuroretinitis, exudative retinal detachment, optic neuritis); varicella and herpes zoster (keratitis, uveitis, chorioretinal scars).³¹ The recent measles outbreak in Samoa and New Zealand highlights the need for ongoing and widespread emphasis on vaccination requirements. This has become an increasing challenge since the COVID-19 pandemic and the rise of anti-vaccine ideology. To reduce the prevalence of these conditions, we summarise potential interventions in Table 8.

Eye trauma

It is estimated that each year, 3.3–5.7 million eye injuries affect children worldwide.³² In 2019, the New Zealand childhood ocular trauma study determined an incidence of 719 cases per 100,000 children per year.³³ This study also highlighted that they occurred more commonly in males (63.2%), between the ages of 0 and 4 years (30.7%) and among those of New Zealand European ethnicity (60.8%). These injuries predominantly involved being “struck by an object” (53.7%), were typically in the home setting (50.9%) and reported protective eyewear use was very low at the time of injury (2.7%). Around one-fifth of cases (19.7%) admitted for tertiary assessment and treatment had final visual outcomes that were 6/12 or less, and Māori and Pacific people were over-represented in that category.³³ To further compound these statistics, up to 90% of ocular traumas are preventable. Prevention strategies, such as parental education, legislation (e.g., restriction on sales of hazardous toys and lasers) and the introduction of eyewear protection (e.g., for sporting activities, especially cycling, football and ball sports) effectively reduce the incidence of ocular trauma.³² RANZCO also recommends developing and

Table 8: Specific interventions to prevent infectious eye diseases.

Type of prevention	Specific interventions
Primary prevention	Promote and provide ready access to vaccination to increase coverage levels of all childhood vaccines. Elimination of some of these diseases is feasible (e.g., measles, mumps, rubella and <i>H. influenzae</i> type b).
	Prompt and effective control of outbreaks (e.g., measles, meningococcal disease outbreaks) if these arise.
Secondary prevention	Prompt identification and treatment of cases may reduce the risk of sequelae (e.g., early antibiotic treatment for meningococcal disease).
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of disability.

Table 9: Specific interventions to prevent eye trauma.

Type of prevention	Specific interventions
Primary prevention	Mass media campaigns (e.g., funded by Accident Compensation Corporation [ACC]) to promote avoidance of hazardous situations for eye injury and to promote increased use of protective eyewear.
	Legislation to prevent the sale of high-risk toys, lasers and fireworks.
	Legislation to require eye protection use during specific sporting activities such as cycling, football and ball sports.
	Public funding of protective eyewear (e.g., from ACC).
Secondary prevention	Prompt and effective treatment to maximise recovery and minimise the risk of sequelae.
Tertiary prevention	Referral to Blind and Low Vision Education Network New Zealand to reduce the impact of any disability.

implementing a national strategy and awareness campaign to prevent ocular trauma.² To reduce the prevalence of ocular trauma, we summarise potential interventions in Table 9.

Conclusions

While less common than adult blindness, childhood blindness in New Zealand has a significant burden in terms of the total burden of blind years. Many prevention strategies must be initiated well

before vision loss occurs, and ophthalmology care can typically only prevent the worsening of vision rather than the restoration of vision. Ophthalmologists and RANZCO must continue to actively collaborate with obstetricians, paediatricians, general practitioners, optometrists, national screening units, vaccination programmes, epidemiologists and Health New Zealand – Te Whatu Ora to promote primary prevention strategies and improve visual outcomes for our tamariki.

COMPETING INTERESTS

J Rodier has received payment or honoraria from the University of Sydney Microsurgery Course as a lecturer and for providing tutorial.

ACKNOWLEDGEMENTS

We would like to give special thanks to Professor Nick Wilson (co-head of the Department of Public Health, University of Otago, Wellington, New Zealand) for his valuable input in the write-up of this paper.

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Putting communities at the centre for a more effective and equitable health system in Aotearoa New Zealand

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ABSTRACT

Community-led action is essential for building a more effective and equitable health system. Yet Aotearoa New Zealand's history of top-down structural reforms has undermined progress toward “healthy futures for all”. We draw on complexity science and system-change principles to explain why genuine devolution and community engagement are not just ideological preferences but practical necessities in a complex adaptive health system. Community agency and locally tailored innovation can drive emergent, system-wide improvements, but only if central structures enable and sustain these relationships. A key step is reframing our mental model of the health system from a linear machine to a complex system. We discuss how the turbulence of current policy changes fits into long-running patterns and why a clearer conceptualisation of complexity can guide policymakers toward tangible actions that reorient the system towards patients and communities. Finally, we outline some essential ingredients for how New Zealand can transition from rhetoric and good intentions to the effective implementation of an equitable, community-centred health system.

In Aotearoa New Zealand (New Zealand), decades of effort have failed to achieve equitable health outcomes.^{1,2} A stark socio-economic gradient in health is well documented, and institutional racism within health and social services further compounds material inequities, especially for Māori and Pacific peoples.^{3,4} Recent high-level inquiries and reviews have reiterated that successive governments have struggled to effectively devolve resources and deliver services at the local level for all communities.^{5,6} Meanwhile, we know that in communities where the quality of service delivery is poorer and harder to access, the quality of the broader determinants of health, such as income, employment, housing and food, is also poorer.⁷

In 2021, at the same time as the country was dealing with the COVID-19 pandemic, New Zealand embarked on another round of health system reform in pursuit of “healthy futures”, *Pae Ora*.⁸ These Labour Government reforms aimed to create a more sustainable, accessible and fair system through several key changes. The *Pae Ora (Healthy Futures) Act 2022* (the *Pae Ora Act*) established a national health charter to guide system-wide stewardship; replaced 20 district health boards (DHBs) with a single national entity and four regional divisions under Health New Zealand – Te Whatu Ora; and created a co-governance arrangement with Māori through Te Aka Whai Ora, a new Māori

Health Authority—a key recommendation from the Waitangi Tribunal's *Hauora* report.^{5,9} The *Pae Ora Act* also introduced geographically defined “localities”; a model intended to grow and strengthen local networks of providers and community partnerships to drive better local decision-making and resource allocation. Localities promised not just another structural reorganisation, but the possibility of real devolution and genuine power-sharing with communities. The *Pae Ora Act* also reinforced the focus on local need by establishing Iwi-Māori Partnership Boards (IMPBs) as a key mechanism to embed Māori and local voices into health system governance. The IMPBs were to represent local Māori perspectives on health needs, priorities and service design.

In late 2023, the incoming coalition government signalled a significant shift in direction, returning to short-term, more easily measured targets and moving away from equity as an explicit health system goal. Under urgency, the *Pae Ora* reforms were “reset”, resulting in the disestablishment of Te Aka Whai Ora – Māori Health Authority, a move seen by some as undermining decades of advocacy for independent Māori health leadership.¹⁰ This rollback of Māori influence on health decision-making has been followed by other top-down decisions impacting trust within Māori communities, such as questioning current progress on Te Tiriti o Waitangi (Te Tiriti), de-emphasising te reo

Māori and halting the use of ethnicity as an indicator of health need.¹¹

Nonetheless, this reset did retain IMPBs, intended to give whānau and hapū a direct role in identifying what is working and what needs improvement in health services and the wider system, thereby providing some accountability to Māori communities. However, recent legislative changes have seen the IMPBs lose agency, retaining only an advisory capacity.^{12,13} Some were already arguing that to function effectively, IMPBs would require dedicated funding and infrastructure, and better, more accessible data and information.¹⁴ While IMPBs currently remain—albeit in a less potent form—the rollout of “localities” has been paused, delaying their mandatory establishment and locality plans until July 2029 and 2030, respectively. This delay leaves the reform’s promise of more integrated and locally responsive health services uncertain.

Persistent inequities and past attempts at devolution

Multiple high-level reviews in recent years point to the urgent need for directing resources and decision-making power towards communities in need and addressing the determinants of health. The Waitangi Tribunal’s *Hauora* report⁵ identified breaches of Te Tiriti by the Crown, specifically in terms of health sector leadership, and recommended structural reform. Similarly, the *Health and Disability System Review*⁶ that underpinned the *Pae Ora* reforms highlighted system fragmentation and a pattern of underserving communities, calling for substantial changes to achieve a genuinely population-focussed system.

The rhetoric of shifting the health system toward primary care and community-based approaches is decades old and has been central to influential frameworks in health, such as the Ottawa Charter for Health Promotion.¹⁵ New Zealand’s *Primary Health Care Strategy* (2001) envisioned a community-oriented, prevention-focussed model of care. At this time, some steps were taken: Primary Health Organisations (PHOs) were formed and some services were devolved to DHBs and community providers. However, much decision-making power and accountability remained centralised, with underlying incentive structures, including fee-for-service funding and private ownership in general practice, meaning profit interests continued to dominate.¹⁶ Further, the Waitangi Tribunal (2019) identified systemic

underfunding of Māori primary health care organisations and providers from the outset, compounded by limited Crown data that would enable effective monitoring and tracking of its own performance in achieving health equity.¹⁷ In short, past reforms, despite stated intention, did not substantially alter the system’s power dynamics.¹ There was little shift toward genuine community involvement or shared local health goals.

Centralised decision-making and control have persisted through successive reforms,⁸ despite strong evidence that community-centred health systems are more efficient and effective, particularly over the long term.^{18,19,20} Evidence shows that community-led or devolved initiatives have achieved measurable health gains, especially in populations with lower life expectancy linked to socio-economic deprivation. In these settings, the most enduring improvements arise from effective action on the determinants of health.^{21–25} Indeed, health systems that embrace intersectoral approaches also tend to be more resilient to complex challenges such as climate change and pandemics,²⁶ reflecting the Sustainable Development Goals’ emphasis on local resilience and partnership.²⁷ By deliberately accounting for local context, health organisations and policymakers can align action with the assets, relationships and capacities already present within communities, ensuring that services and intervention are more responsive and locally acceptable.²⁸

Despite the recognised benefits of devolution, a persistent challenge has been the integrity of its implementation. When poorly executed, devolution can lead to reduced expertise and capacity at the local level,²⁹ not because of weak intent, but because responsibility is transferred without the corresponding knowledge, authority or resources. Like many other high-income countries, New Zealand’s health system has remained shaped by paternalistic policy paradigms, often disconnected from community realities.¹⁸ Policy and management decisions have tended to prioritise financial control, technological solutions and institutional or political interests over local experience and lived realities. This helps to explain why successive attempts at “devolution” or “partnership” have struggled to deliver in practice.

The passing of the *Pae Ora Act* in 2022 launched an ambitious suite of reforms aimed at finally breaking the cycle of inequities in health. The reform agenda recognised that structural reorganisation was needed not only at the centre, but also of the structures that reached into local

communities. By creating formal local structures for partnership and community input, the reforms were attempting to move beyond tokenistic consultation toward genuine co-ownership with local communities. Early on, there was cautious optimism, tempered by the lessons of previous attempts at “devolution”. Earlier reforms, such as the establishment of Area Health Boards in the 1980s and DHBs in 2001, failed to transfer real agency to communities.^{30,31} Despite new governance structures, decision-making remained highly centralised, with continued tight control over funding and narrow accountability requirements that constrained local flexibility.³²

The reforms initially held promise. From the outset, there was recognition that structural change alone would be insufficient, without accompanying cultural and relational change. Transformation would require shifts in the ways people and organisations worked together across the system.⁸ Yet, as implementation unfolded, this focus on culture change was largely lost. In practice, the creation of new central entities, on their own, cannot alter entrenched system behaviours if existing practices and power relationships remain. The minority view on “Māori commissioning” within the *Health and Disability System Review*⁶ similarly cautioned that, without integrity in implementation, a genuine commitment to equity and shared decision-making, *Pae Ora* risked becoming yet another missed opportunity for meaningful change.

Viewed through the lens of complexity science, this missed opportunity points to a deeper need to understand how system behaviour emerges from relationships, incentives and feedback. It is these dynamics that need to change if any future reforms are to successfully improve the performance of the whole health system.

What can complexity science tell us about implementation and whole-system reform?

Globally, health systems are increasingly understood as complex adaptive systems.^{33,34} Population distributions of health outcomes emerge from countless interactions among diverse actors and forces: hospitals, primary care, public health agencies, communities, patients, social services, economic and political dynamics, cultural norms and more.³⁵ In a complex system, relationships and interactions drive outcomes.³⁶ Effects are often non-linear, with small changes amplified

through feedback loops. Population-level patterns, such as persistent health inequities, are emergent properties: they arise from systemic interactions over time, rather than from any single cause or policy that can be adjusted in isolation. Health systems themselves are not only complex, but they are nested within broader complex social systems. Grasping complexity is not just an academic exercise, it is essential for identifying actions that lead to meaningful change. For example, the socio-economic gradient in health is not simply the result of personal choices or bad policy, but rather the interplay of economic, educational, healthcare and social factors, along with government responses, that reinforce each other within places and across generations.³⁷

Because of these dynamics, intended actions often have unpredictable effects. A policy that succeeds in one community may fail in another because local histories, relationships and resources differ.³⁸ Yet across sectors, complex systems display recognisable patterns of behaviour; feedback loops, adaptation and path dependence mean that while specific outcomes cannot be predicted, broad tendencies can. In health systems, for example, we can anticipate that when decision-making remains highly centralised and community knowledge is excluded, inequitable health outcomes are likely to persist. Similar patterns can be seen in other domains, such as education or environmental management, where top-down initiatives struggle when they overlook local realities. Conversely, changing the “rules of interaction”—for instance, through community co-design of services, shared governance or directing more funding toward locally led prevention—can trigger new system dynamics that support innovation and longer-term improvement.³⁹

Knowledge of complexity helps explain how we are reproducing patterns of health outcomes over time. It strengthens the argument for empowering local communities to participate in creating conditions where new configurations of practice, service delivery, and innovation can emerge, thereby altering the system’s trajectory. Essentially, a “butterfly effect”, where small, early changes can generate large effects.^{40,41}

The “butterfly effect” and local community-led innovation

The concept of sensitivity to initial conditions (the “butterfly effect”), illustrates how small changes can lead to significant differences in outcomes

over time. In health systems, this suggests that local, community action can have disproportionately large, long-term impacts. For example, a small community-led initiative might shift a feedback loop by building local trust, increasing engagement with preventive care, or modelling an integrated service which others then replicate, thus putting the system on a new trajectory. Over time, these local “seeds” can grow into widespread change (analogous to a butterfly’s tiny wings eventually altering the weather).

New Zealand’s COVID-19 response demonstrated the importance of community-led action—both in a crisis and, by extension, in system change. Faced with urgent threats, many bureaucratic barriers fell away, resources were rapidly mobilised and, in some cases, communities were empowered to act. The government’s early pandemic response showed unprecedented co-ordination across sectors. Community providers, including many Māori and Pacific health organisations, NGOs, iwi and hapū, played central roles in testing, vaccination, outreach and social support, filling longstanding gaps left by inadequate policy.^{42,43} Māori providers, backed by iwi leadership, established health hubs, distributed care packages and delivered tailored public health messages in te reo Māori and Pacific languages. Pacific church and community leaders mobilised to boost vaccination uptake. Parts of the pandemic response demonstrated that trust-based, flexible, community-embedded approaches yielded faster and more effective solutions than centralised control could achieve on its own. The national public health response tapped into pre-existing relational infrastructure built on trust and local connections. We saw a glimpse of the system becoming more adaptable and resilient when bottom-up action was enabled. But we also saw resistance to this from government and policy organisations, and ultimately a quick return to the status quo, even for those initially enabled.⁴⁴

This experience reinforced a critical lesson. Relationships and trust are not peripheral to system performance—they are causal.^{45,46} A major flaw of past New Zealand health reforms has been the repeated disruption of health system and community relationships. Each restructuring tends to reset institutional memory, weaken local networks and further distance the “centre” from community realities.

One principle of change in complex systems is the need for feedback mechanisms that keep the system aligned with its desired goals.⁴⁷ The

“localities” were intended to provide precisely that—channels through which information about what is working (or not) on the ground could flow back up, and through which communities could hold the system accountable. Without these feedback loops, the system risks once again becoming “insensitive to initial conditions” at the local level—small local issues risk growing into big problems before the centre notices.

The implications of putting localities “on hold” are significant. In their absence, it is unclear how the system will now ensure local health needs and priorities are identified and addressed. To date, no model has been articulated that states how the benefits “localities” promised will be achieved. Removing or weakening formal structures for local community input and action removes essential feedback loops, which were only just beginning to be built back into the system. It sets the stage for a return to the familiar scenario where a policy looks fine in theory but fails in practice because it was not cognisant of local context.

A way forward: embracing community-led action in a complex health system

Amid growing awareness of planetary challenges,⁴⁸ demographic change and economic constraint, New Zealand must find ways to make its health system both more effective and more sustainable. International evidence shows that community-led action is not a “nice-to-have” but essential for long-term system performance. This requires moving beyond short-term targets and centralised control, towards valuing local knowledge and long-term learning. Even if formal locality networks are paused, the intent behind them should continue through other mechanisms that strengthen community agency and cross-sector collaboration.

A complex systems lens reminds us that researchers, policymakers and health professionals are not external observers pulling levers—we are part of the system we seek to change.⁴⁹ Every intervention becomes an event within that system, interpreted and responded to in unpredictable ways. In community settings, interventions act less as fixed “doses” and more as catalysts that interact with existing conditions, often triggering ripple effects that cannot be fully anticipated.⁵⁰ This perspective demands both humility and continuous learning. Health equity cannot be mandated from above, it must be co-created by

working with the system's adaptive nature and supporting all actors—including communities—to shift practices and resource flows.²³ Complexity science also highlights that a system's underlying purpose and mindset are far more powerful levers of change than structural changes alone.⁴⁷ In human systems, true purpose is revealed through everyday practice. If the stated aim is better health for all, then the actions that shape funding, planning and accountability must consistently reflect that purpose.

Primary care remains a key nexus for change and must be better funded, focussed on universal access and incentivised to respond to local contexts.^{51,52} While capitation can improve access, it does not address workforce shortages, system capacity gaps or the higher costs of caring for people with complex needs.⁵³ Short electoral cycles also prioritise visible, short-term gains over enduring community health outcomes.¹ To change this dynamic, funding models could be tied to locally defined health goals, rewarding providers for improving population wellbeing rather than increasing service volume.

Investment should also extend beyond clinical settings to the places where health is created or lost—homes, schools, workplaces and neighbourhoods. This means shifting from treating illness in silos to promoting wellbeing across communities. Investment strategies should enable local organisations to better collaborate on shared goals, recognising that social determinants—such as housing, education and food systems—are integral to the health ecosystem. Lessons we already hold need to be taken up from initiatives, like Whānau Ora and Healthy Families NZ, which are demonstrating how to work across sectors and build local capacity.^{23,25}

An integrated prevention and primary care system should be re-centred around community need and insight, underpinned by stable, long-term funding that enables collaboration and innovation across local organisations. At present, financial, political and professional incentives often undermine locally led prevention efforts.⁵⁴ Instead, commissioning models should embed mechanisms that allow communities to define what success looks like, moving beyond top-down key performance indicators to measures that reflect local values and priorities.⁵⁵⁻⁵⁷

Centring communities requires more than consultation or co-design, it demands a rebalancing of relationships between central- and local-government and communities,⁵⁸ with a focus on increasing

local agency and genuine co-ownership of health goals. Community partnerships should hold real decision-making authority over portions of health budgets so they can direct resources toward local priorities. Central agencies, in turn, should act as enablers; setting broad outcomes and standards, while allowing flexibility in how local communities determine and achieve them. Critically, resources must align with responsibilities. Many Māori, Pacific and community providers operate with limited and insecure funding; strengthening their capacity is not ancillary but foundational to system resilience.^{25,44}

In a complex system, outcomes cannot be fully planned or controlled, so continuous learning and adaptation are vital. A learning health system relies on data and insight to refine programmes and policies in real time as conditions change, but it also depends on reflection and responsive action. Digital technologies and emerging tools, such as artificial intelligence, hold promise for addressing workforce shortages, improving timely access to care and creating more integrated data systems. However, even the most sophisticated technology and tools will not improve outcomes for all communities if the underlying conditions of the delivery system remain unchanged. If local and primary care organisations continue to face inadequate resourcing and barriers to collaboration they will struggle to respond effectively to community needs. Greater local data sovereignty must therefore be central to the design of learning health systems. Routinely collected data should be made available through trusted, free and user-friendly platforms, and communities should be supported to interpret and apply evidence to their own contexts.⁵⁹ Over time, data and local insights can become a shared asset for collective action rather than a mechanism of control. Tracking data in context can build a richer understanding of community needs and assets, supporting locally grounded solutions that are trusted and sustainable.²³

A learning and adaptive health system

History shows that top-down change, without grassroots agency, fails to improve the health of the whole population. The 2022 reforms wisely, but timidly, combined structural change with community empowerment. If implemented well, it is an approach that still holds promise. Eliminating the postcode lottery in health requires systems that foster inclusive and trusting relationships,

strengthen community networks, support local innovation and adapt in response to new information. Small, community-level changes can spark significant shifts, but only if the broader system supports and nurtures them. Community-led health action is not a threat to unity—it is an essential lever for system learning and improvement.

New Zealand needs to learn from its past missteps. Either we repeat failures or embrace pae ora. Without an ability to learn, we risk a future where: we are unable to respond to emerging local changes before they become larger problems; inequitable health outcomes persist or worsen

for some communities; public trust further erodes amidst continual restructurings; and we are unable to respond with speed to current and emerging health threats. But, if we equip and empower communities and frontline providers to act more collectively, we can create the feedback that supports resilience and enables the health system to learn, adapt and innovate. We need a health system rebuilt with patients and communities at the centre, not just redesigned for them. Only by shifting the locus of control closer to where health is created can we have lasting, meaningful improvements in health for everyone.

COMPETING INTERESTS

Nil.

ACKNOWLEDGEMENTS

We would like to acknowledge the many people and projects that have contributed to the knowledge and insights informing this paper, as well as the reviewers for their thoughtful feedback, which has helped us to strengthen and improve the manuscript.

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<https://nzmj.org.nz/journal/vol-139-no-1628/putting-communities-at-the-centre-for-a-more-effective-and-equitable-health-system-in-aotearoa-new-zealand>

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Are you sure it's Crohn's?

Winston Zheng, Zaal Meher-Homji, Minnie Au

A 57-year-old Australian female, originally from the Philippines, underwent endoscopy for asymptomatic iron deficiency. Colonoscopy demonstrated ileitis with ulceration and stenosis (Figure 1). Histopathology showed chronic inflammation with granulomas (Figure 2). Magnetic resonance enterography demonstrated moderately severe irregular ileocaecal valve and distal terminal ileum wall thickening, deep ulcerative foci and lymphadenopathy (Figure 3).

What are your differentials?

What would you do?

Discussion

The patient was empirically treated for Crohn's disease (CD) with prednisolone while awaiting immunomodulator screening. Two weeks later, Quantiferon-Gold was positive. Prednisolone was ceased, repeat biopsies confirmed *Mycobacterium tuberculosis* infection with a positive polymerase-chain-reaction (PCR) (Xpert MTB/RIF Ultra, Cepheid). No resistance mutation

genes were detected to rifampicin, isoniazid or fluoroquinolones. Mycobacterial cultures were negative after 6 weeks of incubation. She had no respiratory symptoms, and chest-X-ray showed no abnormalities.

She was diagnosed with intestinal tuberculosis and had an uncomplicated 6-month course of standard weight-adjusted, drug-susceptible anti-tuberculosis treatment of rifampicin, isoniazid, pyrazinamide and ethambutol. Post-treatment endoscopy demonstrated healing, though with a mild ileocaecal valve fibrotic stricture.

Gastrointestinal tuberculosis (GITB) is an example of extrapulmonary tuberculosis. Sites of GITB can include the gastrointestinal tract, peritoneum and lymph nodes. The ileocaecal is the most commonly infected (44–84%) due to it being a constricted region allowing for slower transit time and high rates of fluid absorption, and having an abundance of lymphatic tissue.¹

Clinical features can be non-specific, such as fever, weight loss and anorexia, or more localised, including abdominal pain, distension,

Figure 1: Colonoscopy view of the terminal ileum.

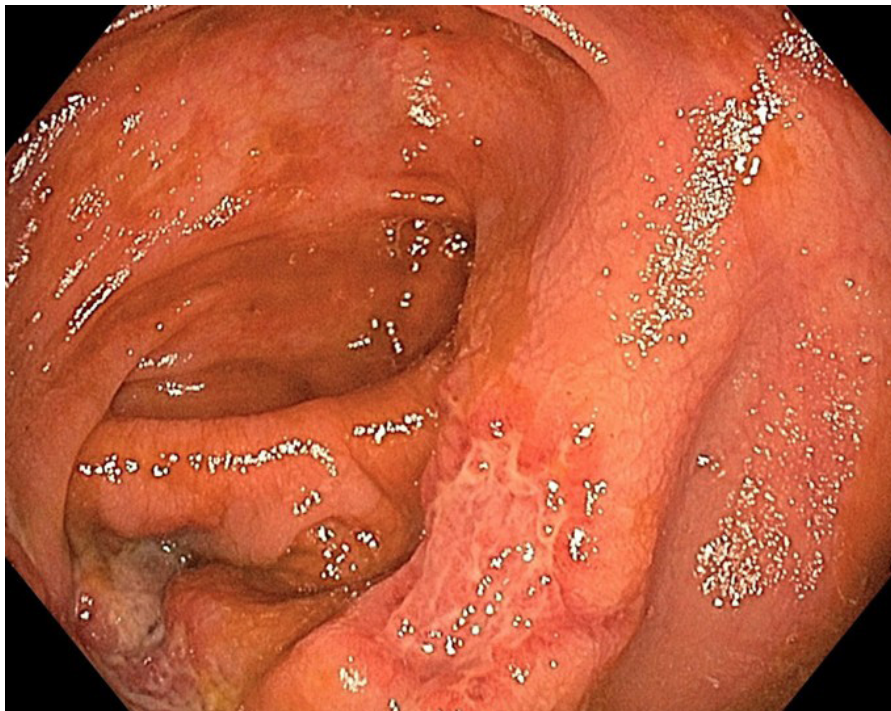


Figure 2: Histopathology of terminal ileum biopsies.

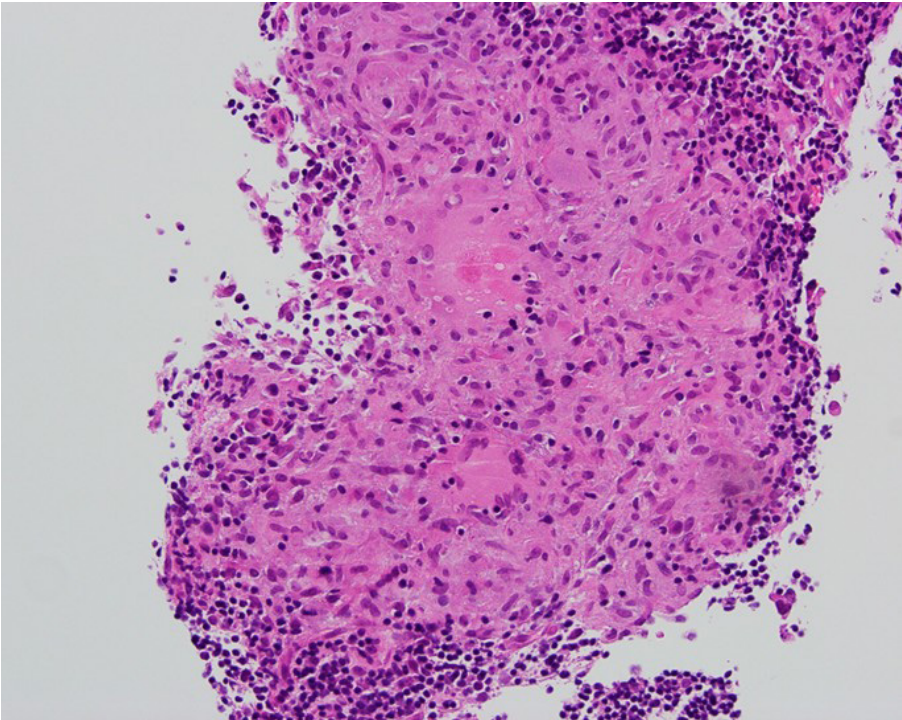
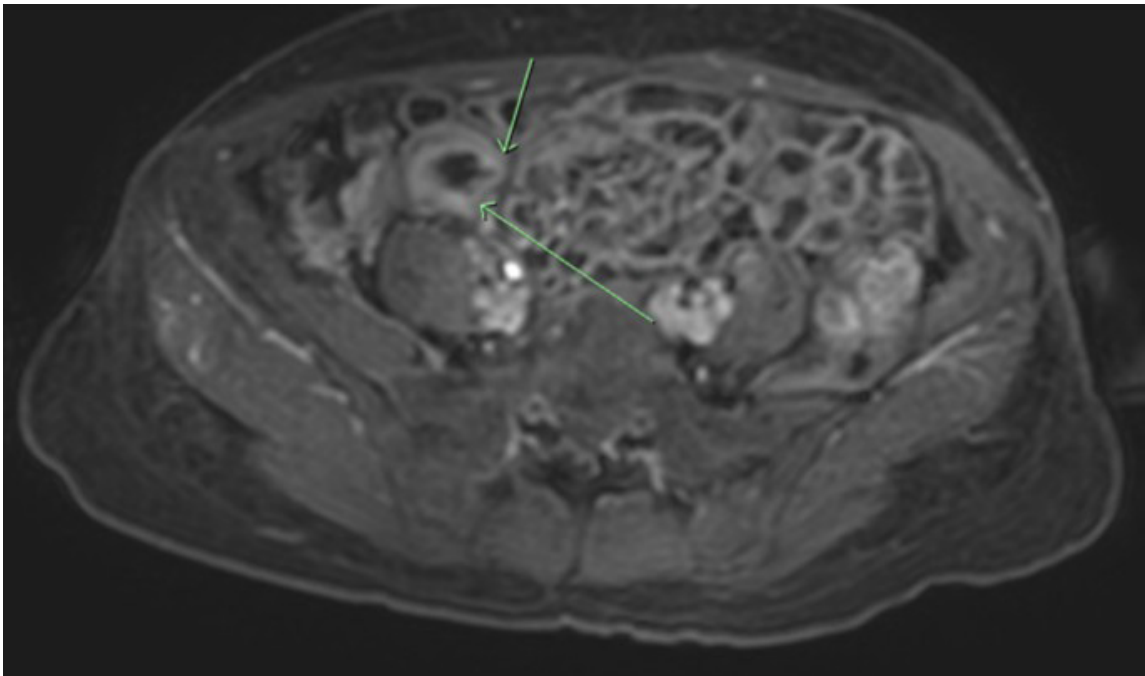


Figure 3: Magnetic resonance enterography.



nausea/vomiting, diarrhoea and blood in stools, with symptoms varying dependent on the location of the tract affected.¹ Serious complications can include intestinal obstruction, perforation, fistulas, collection and bleeding.¹ Symptoms of pulmonary tuberculosis may or may not occur concurrently.

Endoscopy findings of GITB generally reflect inflammatory features, including erythema, erosions, ulcers, nodules, pseudopolyps and strictures. Ulcerations and deformed ileocaecal findings are common in colonoscopy, while strictures are the most predominant finding on gastroscopy.¹ Histopathology from biopsies generally reveal non-specific chronic inflammation with features of chronicity, crypt distortion and cryptitis. Granulomas can often be seen, sometimes with central necrosis.²

PCR and mycobacterium cultures on biopsies should be performed. Acid-fast bacilli (AFB) smears on intestinal biopsy and culture positivity have high specificity and high positive predictive value, but low sensitivity and low negative predictive value.¹ Hence, while a positive test is helpful, a negative test does not necessarily rule out GITB.

Differentiating between GITB and CD can be difficult. Both have chronic granulomatous features, a predilection for affecting the ileocaecal region and non-pathognomonic clinical presentations, endoscopic and imaging findings. There is significant importance in differentiating between these conditions given their differing management:

GITB is an infection and requires anti-tubercular therapy, while CD requires immunosuppression. Delayed or misdiagnosis can lead to poor outcomes.²

There is significant overlap between the clinical presentations of CD and GITB, with no specific feature to help discriminate. CD typically has a more prolonged and indolent presentation, while GITB is typically shorter (<6 months). Constitutional symptoms, especially fevers, are infrequent in uncomplicated CD and are more suggestive of GITB. Pulmonary symptoms, especially productive cough with haemoptysis may suggest concurrent pulmonary tuberculosis. As demonstrated by this case, a careful epidemiological history regarding exposure to high-risk endemic areas for TB is important to help stratify risk. The most reliable parameters for favouring GITB over CD are AFB on smear, positive mycobacterium culture, granulomas with caseating necrosis on histopathology, and lymph node necrosis on radiology.²

It is also important to consider other differentials of chronic inflammatory changes and granulomas including histoplasmosis, cryptococcosis, sarcoidosis, chronic granulomatous disease.

The mainstay treatment approach for GITB remains primarily medical, with international guidelines recommending standard drug-susceptible anti-tubercular therapy for 6 months.³ Therapeutic response can be monitored through symptomatic, biochemical and endoscopic resolution.¹

COMPETING INTERESTS

None declared by the authors.

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<https://nzmj.org.nz/journal/vol-139-no-1628/are-you-sure-it-s-crohn-s>

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Chronic oscillopsia and neck dystonia: atlanto-occipital origin

Leonardo Furtado Freitas, Márcio Luís Duarte, Kevin J Abrams

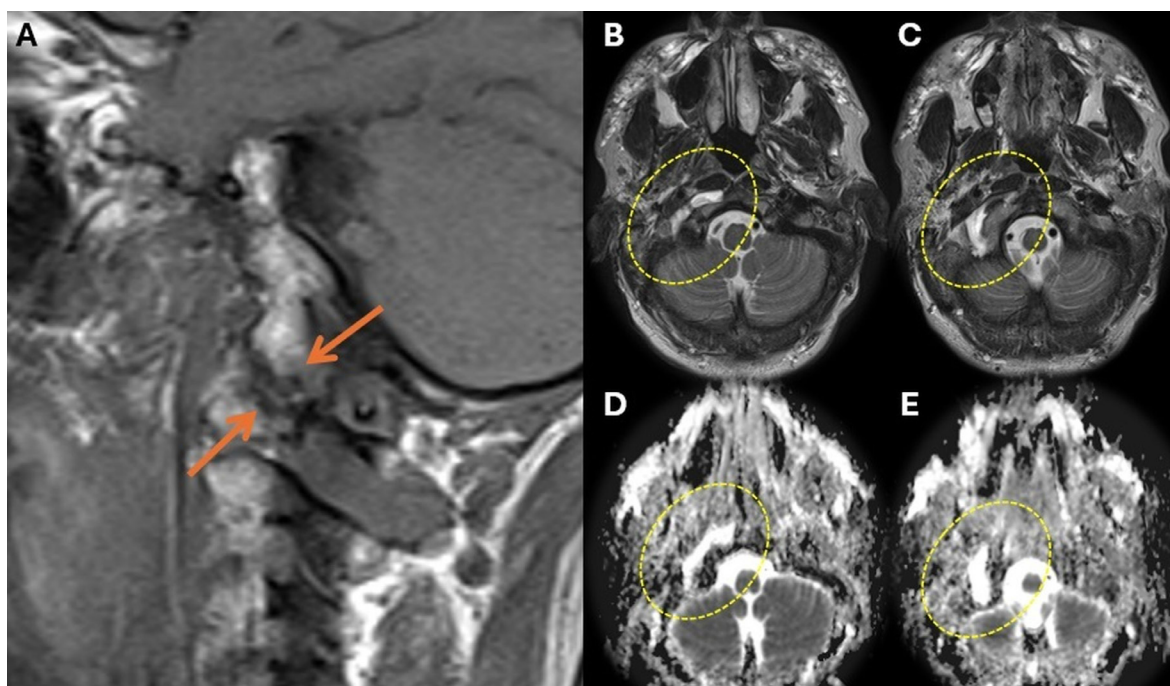
A 61-year-old woman presented with long-standing involuntary neck and head movements, characteristic of cervical dystonia, accompanied by oscillopsia. These symptoms severely impacted her daily activities, including basic tasks and overall quality of life. Cervical dystonia, a movement disorder caused by sustained or intermittent muscle contractions leading to abnormal postures or repetitive movements, was compounded in this patient by visual disturbances, including the perception of unstable surroundings.

To investigate a potential structural cause, magnetic resonance imaging (MRI) of the craniovertebral junction was performed. The imaging revealed significant right-sided atlanto-occipital arthropathy, evidenced by bony erosions, joint effusion and adjacent soft tissue changes

(Figure 1). These abnormalities suggested underlying degenerative or inflammatory pathology involving this critical joint, which plays a fundamental role in head and neck mobility. The findings clarified the likely origin of her symptoms, indicating that the arthropathy contributed to both her dystonia and vestibular complaints.

Atlanto-occipital arthropathy, particularly when associated with effusion, can disrupt the integration between vestibular and proprioceptive systems, essential for spatial orientation and balance.¹ Inflammation and effusion may interfere with visual-vestibular pathways, manifesting as vertigo and oscillopsia.² This case underscores the importance of considering craniovertebral structural pathology in patients with atypical or refractory dystonia, where MRI can play a pivotal diagnostic role.

Figure 1: Magnetic resonance imaging of the craniovertebral junction with sagittal T1 (A), axial T2 (B and C) and axial ADC map (D and E) sequences. There was unilateral right-sided atlanto-occipital arthropathy, with bony erosions (orange arrows) and effusion (dashed yellow circle).



COMPETING INTERESTS

No specific funding was received for this work. The authors declare that there are no conflicts of interest relevant to this work.

ACKNOWLEDGEMENTS

Ethics:

Name of the institutional review board or ethics committee that approved the study: no institutional review board (IRB) was requested for this case report. Informed consent for publication was obtained from the patient's legally authorised representative.

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<https://nzmj.org.nz/journal/vol-139-no-1628/chronic-oscillopsia-and-neck-dystonia-atlanto-occipital-origin>

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Strictures of the Ureter

NZMJ, 1926

By James A. Jenkins, *Dunedin*.

Strictures of the ureter are of common occurrence in the practice of one doing much urological investigation. Their presence as a cause of ill-health and suffering has not yet reached the majority of practitioners. The wide range of symptoms present in a series of cases makes their diagnosis impossible apart from instrumentation. Symptoms may suggest the possibility of their presence, but only the passage of bulbs of large calibre, together with urographs, gives definite information.

There are two main types :—(1) Congenital strictures of the ureter (rare) ; and (2) , acquired (common).

Congenital strictures have been described by several writers :— Bottomly ⁽¹⁾ in 1910 brings the literature to date with 58 cases, many of them from *post-mortems* and monstrosities. Eisendrath ⁽²⁾ brings the series up to 63 cases and reports one case with hydronephrosis and hydroureter and Ockerbald ⁽³⁾ and Caulk ⁽⁴⁾ have described cases of congenital dilatation of the ureter which can be compared to the condition of the sigmoid colon known as Hirschsprung's disease. In these cases there is no sign of back pressure upon the kidney. In other words there is no marked obstruction.

Cases of obstruction due to valves and other pathological conditions in the posterior urethra have been fairly commonly reported as occurring in children. Many have been found *post-mortem*, the children dying of hydronephrosis, and infec-

tion, and renal failure.

Acquired strictures of the ureter are a very real thing, both to the patient and to the surgeon who has the necessary equipment and skill to demonstrate them. Hunner, who has done so much to draw the attention of urologists to their frequent presence, has, after many years of careful study, had his work widely recognised.

Long-standing infection of the kidney and renal pelvis causes considerable dilation of the ureter, and these cases have to be distinguished from the cases of true stricture here described.

The cause of this condition is still not definitely known, but all the evidence goes to prove that localised infections of the wall of the ureter is the chief factor. Focal sepsis and elective localisation possibly play an important part.

The condition is bilateral in the vast majority of cases, though there may be symptoms on one side only when the patient first reports. (cases 2 and 3 illustrate this).

The symptoms of uterine stricture are varied. Hunner describes them as follows :—Pain.— Usually situated deep in the pelvis. It frequently occurs in the back, loins, kidney regions, and varies from a slight drag to great discomfort. Scaro-iliac joint and the appendix area is a common situation. Frequency of micturition and neuralgic pains in the bladder are common. Gynaecological symptoms.—Dysmenorrhoea, "ovarian neuralgias," and dyspareunia are amongst the commonest symptoms. Gastro intestinal symptoms.—Headache, mild uraemic symptoms, are said to occur.

George Earle Dunlop Brown



George Earle Dunlop (“Earle”) Brown FRACS, FRCS (Eng) was one of the formative figures in New Zealand plastic and hand surgery. He died on 8 November 2025. A quiet, courteous and meticulous surgeon, he combined technical innovation with a deep sense of service to his patients at Middlemore Hospital, to colleagues and trainees across the country and to communities throughout the Pacific.

Born in Stratford in 1936 and raised in Clyde, Central Otago, Earle graduated MB ChB from the University of Otago in 1960 and completed compulsory military training with the First Casualty Clearing Station and Otago University Medical Company. After early surgical posts, he joined the plastic surgery department at Middlemore Hospital in 1963 as a registrar under Sir William Manchester, beginning a professional association that

would shape both his career and the development of New Zealand plastic surgery. Further training in the United Kingdom led to the FRCS (England) in 1967, followed by FRACS in Plastic and Reconstructive Surgery in 1970.

His time at Canniesburn Hospital in Glasgow, under leaders such as Ian McGregor and Ian Jackson, left a lasting mark on his technique, philosophy and lifelong emphasis on careful tissue handling. Returning to Auckland in 1970, he became a senior registrar at Middlemore and was promoted to consultant plastic surgeon in 1972. Over his 40 years at Middlemore, Earle shaped modern plastic surgery in New Zealand. With Onkar Mehrotra, he co-founded the combined plastics–orthopaedic “Red Team”, pioneering hand surgery and completing one of the country’s earliest successful upper-limb replantation in

1974. His ingenuity, using jeweller's loupes and fine forceps to extend microsurgical capability, placed him at the forefront of surgical innovation. His papers on the indirect deltopectoral flap, oesophageal reconstruction, limb replantation and complex facial trauma reflected a career-long interest in solving difficult reconstructive problems and adapting emerging techniques to local conditions.

Earle's influence extended well beyond the operating theatre. He served as secretary of the New Zealand Association of Plastic Surgeons and of the New Zealand Society for Surgery of the Hand. He also represented New Zealand on the executive of the section of Plastic and Reconstructive Surgery of the Royal Australasian College of Surgeons and the Asian Pacific section of Plastic and Reconstructive Surgery. He was New Zealand's representative at the International Congress of Plastic and Reconstructive Surgery in Yokohama, Japan. At Middlemore he led the plastic surgery department for 7 years before becoming the clinical director of surgery and immediate care in the late 1990s, overseeing all surgical services during a period of significant growth and change.

He was also an exceptional teacher. For decades he organised weekly registrar teaching sessions. Later, he and his wife Gay even hosted Sunday evening tutorials at their home for trainees preparing for fellowship exams. Many surgeons who trained under him cite his generosity, clarity and kindness. A colleague describes how "*of all the consultants, apart from Sir William, Earle contributed the most to the department... We owe him a debt of gratitude for his work in promoting and running the department.*"

Earle was a prolific author. His major works include:

- *Introduction to Local Flaps: A Surgeon's Handbook* (with Michael Klaassen), 2011
- *An Examiner's Guide to Professional Plastic Surgery Exams* (with Michael Klaassen), Springer, 2018
- *Perfection: The life and times of Sir William Manchester* (with Michael Klaassen), Mary Egan Publishing, 2021
- *Middlemore Hospital: The First Two Decades* (with Wally Robbins), Mary Egan Publishing, 2024

Notably, they also include two collaborations with Professor Felix Behan, whose influential technique, the "keystone flap", Earle admired greatly:

- *Defining Local Flaps: Clinical Applications and Methods* (with Michael Klaassen and Felix Behan), 2016
- *Simply Local Flaps* (with Michael Klaassen and Felix Behan), Springer, 2018

These texts, along with his historical essay "War, Facial Surgery and Itinerant Kiwis", helped shape modern understandings of flap surgery and the history of plastic surgery in New Zealand.

These publications reflect two constants in Earle's life: his desire to make difficult surgery more accessible to others and his determination to record the story of those who built the specialty. Those same instincts underpinned his long association with the Sir William and Lady Lois Manchester Charitable Trust, where after retirement he served as a medical adviser.

Service also defined his work beyond New Zealand. From the 1970s onwards he participated in numerous voluntary surgical missions across the Pacific and Asia, including programmes in Papua New Guinea (Port Moresby and coastal hospitals), the Philippines (Mindanao and surrounding regions), Tuvalu, Kiribati, Fiji, Indonesia and the Solomon Islands. Working often in challenging conditions, with unreliable power, limited sterilisation, basic equipment and crowded wards, he treated patients with burns, cleft lip and palate, hand injuries and neglected tumours who would otherwise have had little or no access to specialist care. His mission diaries reveal not only surgical ingenuity and persistence, but also deep empathy for patients and respect for local staff.

Alongside this demanding professional life, Earle's anchor was his wife, Gay (née Grant), whom he first met when she was a theatre nurse in the Middlemore plastic surgery unit. She quietly showed the young registrar how to assemble a skin-grafting knife. In time they became lifelong partners in work, family and adventure. Gay's support was woven through every stage of his career, from long on-call nights and overseas meetings to their later years of shared interests in travel, reading and family history.

In retirement Earle and Gay turned some of their meticulous energy to genealogy, exploring the Brown, Buchanan, Grant, MacGregor and related Scottish and Irish lines, and visiting the places where their forebears had lived. Their collaborative family histories echo the same patient scholarship that characterised his medical writing.

Colleagues remember Earle as a true gentleman,

careful and exacting in theatre, quietly determined in committee rooms, unfailingly kind with trainees and patients and always ready with a story that illuminated both the past and the present. His legacy lives on in the thousands of patients

whose lives he improved, the surgeons he trained, the missions he served, and the written record he leaves of a formative era in New Zealand plastic surgery.