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This Issue in the Journal

**Weight and height measurement: potential impact in obstetric care**
Emma Jeffs, Benjamin Sharp, Joanna Gullam, Helen Paterson

Our study identified that women are not having their weight and height accurately reported at the time of their first trimester combined screen. Height and weight (body mass index) impact on pregnancy. We recommend all women have their height and weight measured and their BMI calculated at their first antenatal visit, and have their weight measured and recorded at the time of their first trimester combined screen blood test.

**Management of excess weight in pregnancy in Otago: a qualitative study with lead maternity carers**
Diana Fieldwick, Helen Paterson, Melanie Stephen, Angus Cameron, Richard Egan, Sarah McFadden, Justus Pienaar, Celine Sinclair, Toby Struthers, Kirsten Taplin, Clara Watson

This paper aimed to investigate the knowledge and practice of midwives providing lead maternity care (LMC) in Otago, regarding gestational weight gain (GWG). We used focus groups to provide in depth discussion around how these midwives currently manage GWG and any issues that affect their management. We feel that clear guidelines along with increased education and collaboration between health professionals would improve the management of GWG in New Zealand.

**Duodenal switch— the initial experience in New Zealand**
Martyn L Humphreys, Steven J Robinson, Caryne McKeand, Hisham Hammodat

The duodenal switch (DS) has now established itself as an effective, durable and safe bariatric procedure (to aid weight loss for severely obese patients). We present our initial experience on 60 patients from May 2008 to November 2012. 36 patients have completed 1-year follow-up and 25 patients completed 2-year follow-up. The mean initial body mass index (BMI) was 52.8 kg/m² (range 40–66 kg/m²). The excess weight loss has been 72% at 1 year (n=49) and 72% at 2 years (n=36), respectively. The mean hospital stay is 5.08 days (range 3–18). In our short-term analysis DS appears to be very efficient in terms of cure rate for morbid obesity and its comorbidities (associated illnesses).
Timely cholecystectomy for acute gallstone disease: an ongoing challenge in a New Zealand provincial centre
Melissa J Welch, Andrew R Moot

Studies have shown that the best treatment for patients admitted to hospital with acute symptoms of gallstone disease is a cholecystectomy (surgical removal of the gallbladder). This paper determined the rate of cholecystectomies performed for patients presenting with acute gallstone disease for the first time to Hawke’s Bay Hospital. This rate was low (15%) which is poor compared with large hospitals around New Zealand. Many patients who are discharged without an acute operation re-presented to hospital with additional complications and associated financial expense. There are several reasons why it is difficult to provide an acute cholecystectomy service in a provincial hospital such as Hawkes Bay and some pragmatic solutions are proposed in this paper.

When medical reports become expert medical evidence: judgments of the Court
Koenraad Kuiper

Medical professionals write reports particularly to one another for many purposes. When such reports are presented to the Court, they undergo editing by the Court when they appear in the judgments of the court. The Court also comments from time to time on the quality of the medical evidence as it relates to the purposes of the Court. Such comments provide explicit injunctions on what the Court does and does not accept as useful for its purposes. These comments provide useful guidelines for the report writing of medical professionals.

Measurement of kidney cadmium in embalmed New Zealand cadavers
Ian C Shaw, Emma Spencer, Tessa Lambert, Meike Holzenkaempfer, Sally Gaw, Mark Stringer

Cadmium is higher in NZ soils than some other parts of the world because we use cadmium-contaminated fertilisers. This might mean that NZers have higher levels of cadmium in their bodies than people from other parts of the world. Since cadmium is toxic (i.e. causes cancer), higher body levels might have health implications. We studied cadmium levels in kidneys (cadmium is concentrated in the kidney) from bodies used for medical student dissection and found variable results. Further studies showed that the embalming fluids used to preserve the bodies leach cadmium from tissues, which explains the variability and means that embalmed bodies cannot be used to study cadmium in tissues. Despite this, our findings suggest that cadmium levels in NZers are of the order of those found in other parts of the world.
Are the amounts of vitamins in commercially available dietary supplement formulations relevant for the management of psychiatric disorders in children?  
Julia J Rucklidge, Amy Harris, Ian C Shaw

Micronutrients are a potentially viable way forward for the treatment of psychiatric illness but doses and ingredients vary from one formula to the next. What is unknown is whether the multivitamins sold over-the-counter hold any therapeutic effect for children with mental illness. This study compared the ingredients and doses of those formulas that have been shown to have some benefit for treating psychological/psychiatric symptoms with the ingredients and doses of those formulas available over-the-counter (i.e. in supermarkets/pharmacies). We found that the doses of the research formulas were significantly higher across almost all vitamins as compared with those sold over-the-counter. Therefore, we cannot assume that over-the-counter products will produce any psychological benefits for children. Our research also showed that in order to achieve a mental health benefit, children are likely to need more than the recommended daily allowances (RDA).

Medically facilitated discharge of adult diabetic ketoacidosis admissions: precipitants and average length of stay 
King W Yong, M Peter Moore, Helen Lunt

We wanted to see if a specialist registrar (i.e. a consultant doctor-in-training) could help patients in hospital with out-of-control type 1 diabetes, by working out how to discharge them from hospital, both more quickly but also safely. We found that these patients were already being discharged very quickly and efficiently by international standards, so little help was needed! The main reason these patients were admitted to hospital in the first place was because they forgot to take their insulin injections. Maybe future research efforts should focus on preventing admissions, by working out ways of helping patients to remember their insulin injection?
The sugar debate and nutrition: obesity and ‘empty calories’

Anne-Thea McGill

There is little doubt that refined high energy food, such as added sugar, contributes to being overweight.¹ This is a problem for more than two-thirds of adult New Zealanders, but also over 20% of children.² United Nations (Food and Agriculture Organization) data for our country indicates that, on average, we consume about 147 grams per day (37 teaspoons) of added sugar.³ However the low quality nutrition or lack of other micronutrients in these foods may be the greatest health risk—the ‘empty’ part of ‘empty calories’ does matter.⁴

We now have new science information on why we eat so much refined high energy food and what the metabolism is behind obesity and associated poor health. By briefly putting the issues together we may be able to see where sugar fits into obesity-related illness.

We, as a society, are still tough on our very overweight members. Embarrassment and distress is very common in those of us who know we can’t control our body weight and shape,⁵ and that is most of us. The problem is that the over-appetising, refined high energy food is so irresistible, and sugar is a major player.⁶ Why have we ‘not noticed’ how strong the drivers are that make so many of us become obviously overweight and/or unwell.

Those who put on large amounts of weight in peripheral (hip-thigh-buttock),⁷ subcutaneous areas have problems with moving around/walking,⁸ and psychological concerns about body shape.⁵ For others, without the capacity for storage in these lower-body metabolically-safe areas,⁷ ‘toxic’ lipids accumulate in organs, such as liver, and as upper body or central obesity.

The risks for type 2 diabetes, cardiovascular disease, and later-life cancers and neurodegenerative disease are serious, but even so, changing away from refined energy rich food patterns is not easy. Whist management of weight loss and/or metabolic improvement are different for both body types,⁸ managing the desire for refined high energy food is not.

Sugar addiction has become hard to contest from the extensive literature.²,⁵,⁶ Sugar—especially combined with starchy, fatty and salty food—activates addiction pathways in the brain. Drugs of addiction are similar to neurochemicals in the primordial reward system for acquiring high-energy food.⁶

What’s more, we all really know what these foods are; test question—what foods would you go down to the local food store for, late at night? Answer—sugary drinks, confection including ice cream/blocks and chocolate, salty fatty potato chips, biscuits/crackers with sweet or savoury toppings and other ‘junk food’. Not apples, not plain cooked meat, and not even sweet dried fruit.
Many of us know that sometime or other, for health and wellbeing, we will have to move to a diet without (or with highly controlled allocations of) addictive foods, and we’ll need help.

Although there have been a great many studies in the last few decades on what foods cause fat gain, much research did not follow early leads, which in hindsight appear correct.

Sugar addiction and obesity has been mooted since mid-last century. Lawrence wrote in 1941 “…animals build up reserves of fat from carbohydrate [sugars and starch] … ‘Diabetic obesity’ is very common … in the earliest stages and again after insulin treatment”.

The switch to believing that dietary fat caused heart disease seems to have come from the Seven Countries study. Although some data appeared in 1995, and methodology shortcomings were critiqued shortly after, the main study was published in 1970. By the early 1980s ‘saturated fat caused atherosclerosis’. Many heart health guidelines since then advise low saturated fat diets. Recently, it has been shown that when industrial ‘transfats’ (and non-free range animal fats) are excluded, saturated fats are not the major contributor to weight gain or metabolic problems, but that sugars are.

The upshot was that (refined) carbohydrates were pushed with great haste and vigour into our processed food supply—they were cheap, already deemed ‘staple’ foods and in mass production. They are highly palatable in combination (starchy food plus fat) or easy to hide in other items to ensure purchase (sugar added to drinks or sauces).

Food staples occur in agricultural societies, and are single or few items that are consumed most days. They are usually easily available, cheap, often storable, and are usually the major energy supply with variable other nutrients. Staples can be fruit (coconut, bananas), seed (pulses, grains), roots/tuber (turnip, potato), oil (olive), and sometimes animals or fish.

We did not evolve with staples, starch or otherwise. Westernised food is probably at least 72% different from most pre-agricultural forager or Palaeolithic diets. Prehistoric foods were typically a mix of myriads of useful nutrients from omnivorous diets. Highly energy-dense food was relatively rare and/or seasonal.

At this point, controversial clinical nutrition points arise that relate to starch. We can ask ourselves the following. Are there issues with:

(1) Highly bred grain/tuber starch foods becoming staples in our post-agricultural (10,000 years duration) diets.

(2) Starch, which breaks down into glucose, initially with the salivary enzyme, amylase, for use by mouth bacteria that can cause caries, and rapid absorption from the duodenum into the blood.

(3) The new bit of science that glucose in overload can flow through the polyol pathway, via fructose and rapidly form liver fat.

(4) There being so few studies investigating diets controlling for whole grain alone, and that one that has been done showed no benefit of added grain, which is bred mainly for energy not micronutrients, and
5) Addiction, with respect to grain/tuber and other concentrated starch?

There is evidence for the above scattered in a wide literature. Compared to lower-fibre grain-starch foods, higher-fibre grain is associated with fewer heart disease risks.\textsuperscript{20} However, somewhere along the line, high fibre became equated with highly studied whole grain fibre.\textsuperscript{19,20}

Insoluble (bran) and soluble grain fibres are, of course, carbohydrates, a term that the food industry uses to confuse. High fibre from fruit and vegetable diets also carries many micronutrients, but this effect, as opposed to cholesterol absorption and improved functional bowel health due to purified fibre, is less well differentiated.\textsuperscript{21}

The processed food industry has carefully maintained an advertising commentary of ‘high carbohydrate diets are good for you’ and ‘added sugars can be part of a balanced diet’. The sugar industry lobbies the public health bodies, and are currently pressing hard to prevent the World Health Organization (WHO) from changing their advice to ‘added sugar should make up <5%, [not <10%], of dietary energy’.\textsuperscript{22,23,24}

Meanwhile, the Internet blogs and media reports become a vehicle for ‘mixing and matching’ all information—research and opinion. Variations of high and low ‘carb’, fat, protein diets, and variable glycaemic, fat saturation, gluten free, ‘Palaeo’ and other themed diets, with other camps discussing food addiction are found; all are trying to make each theory work in our current nutrition environment.

There is a risk of relegating the most topical, interesting theories to this forum, which includes a very interested public, and declining to air them in well-known journals. This means that health and medical practitioners, on the ground, will be left trying to decipher which information is useful to help patients lose weight and improve health.

It appears that neither the researchers/clinicians nor food processing companies really want to discuss the idea that any food could be addictive. It is hard to come to the realisation that food, including our refined staples, can be addictive. We thought that dietary (saturated) fats contributed to problem in cardiovascular (CVD) disease, and had much evidence to believe that vegetarian-oriented diet was healthy. The inclusion in all healthy diets of grain/tuber staples (with or without its fibre) was therefore assumed. Often patients say they eat one-third less food after bariatric surgery, so if that one-third of omitted items was addictive refined high-energy food, then the main change to the food supply would be processed food.

All the evidence points to processed food companies understanding and exploiting addiction to all refined high energy foods. The umbrella term of ‘carbohydrate’, has become a foil for not having to mention sugar and refined starch. Tobacco and alcohol marketing ploys of obfuscation, setting up cues and habit formation are not uncommon.\textsuperscript{23–25}

The success of traditional Mediterranean diet studies—epidemiological and prospective—on improving health\textsuperscript{26} is unlikely to only be due to polyphenols in olive oil and wine.\textsuperscript{27} Studies of basic whole food diets, what were once ‘common or garden’, and all their nutrients, has been seen as too difficult to pursue. However, there is strong evidence that that human metabolism is particularly geared to use high food micronutrient diets in a very efficient manner, both for cellular upkeep, repair and healthy longevity.\textsuperscript{27}
There is now the technology to study thousands of chemicals in the diet, so the ‘thus far omitted’ studies of controlled whole food diets, from any and all traditional ethnic backgrounds, could now proceed.29

As health professionals, perhaps we can all now begin put the real ‘common sense’ science together, with evidence from evolution, many epidemiological surveys, thousands of inconclusive studies that did not control for whole food micronutrients, plenty of negative data from supplement studies,30 a few whole food diet studies26 and the food processing industry.23

We then see non-commercial diets which include natural levels of sugars in fruit, some starch in coloured vegetables, fats and protein in low processed plant and animal food, are still likely to be the main way to manage our chronic diseases.

It is heartening to see local public health scientists presenting topical research31 to the public recently, which has been picked up by various media.32 The mood of the community is to demand that the processed food industries’ distribution and marketing of refined, addictive sugary sweetened beverages be decreased through regulation.

However, this may be a distraction to the real health disaster that is upon us. The most pressing problem is the absolute and relative lack of food micronutrients available to and consumed by those of us who are the developing young, the older and the unwell in the community. We must attend to the ‘empty’ in empty calories; the serious gaping hole.

Obesity is usually a micronutrient deficient state, and a marker of serious malnutrition and metabolic problems.23 Neglect in ensuring that high quality food is available to all, feeds into the perpetuation of poverty, and curtails the cognitive, physical and reproductive potential of upcoming generations.

We cannot wait whilst we negotiate our societies’ addiction to calories. Certainly whole foods, even raw nuts and dried fruit, are never going to make the easy profits that processed food companies are used to. Negotiating non-sugar additives in non-nutritious beverages with processed food marketeers is not the answer.

We can now easily test food for quality, and grade items for food micronutrient to macronutrient ratios and content, and add terms for non-nutritive additives/toxins. With such an important issue, is it not timely for processed food producers to be taxed on low quality food, with poor micronutrient content? Accordingly, perhaps only high quality food production should attract direct or indirect public-funded subsidies.24

It seems ironic with our hugely sophisticated, technological society that we still need to provide basic nutritious food to our vulnerable and poor; many of whom are children and mothers.

It seems to me that public health organisations and our community must ensure that, against every impediment and apparent cost, it returns to basics. We must feed all children and mothers; at home, at kō hanga reo, at preschool, at school, at work, on the Marae (Māori meeting house), at church, nutrient rich, palatable, healthy food irrespective of the energy content—now.
Competing interests: Nil.

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Advice to reduce total and saturated fat, revisited

Lisa Te Morenga, Rachael McLean, Murray Skeaff, Jim Mann

The validity of advice to reduce total and saturated fat in order to reduce obesity and coronary heart disease (CHD) has been questioned from time to time.\textsuperscript{1,2} International guidelines\textsuperscript{3–6} recommend intakes of saturated fatty acids (SFAs) of below 10% of total energy, and polyunsaturated fatty acids (PUFAs) sourced mainly from plant oils, nuts, seeds and fish of 5–11% of total energy.

Implementation of this advice would involve a modest to substantial reduction in saturated fat intake in most Western countries and some increase in polyunsaturated fat. More recently it has been suggested that a wider range of intakes of total fat than had previously been suggested, is acceptable: up to 40% of total energy in the Nordic recommendations.\textsuperscript{7} Recommended amounts of monounsaturated fatty acids (MUFAs) are derived by differences (% energy from total fat – [combined % total energy from SAFA + PUFA]), implying a similarly wide range of acceptable intakes.

Such evidence-based recommendations are derived from a consideration of the totality of evidence relating to a wide range of health issues, most importantly cardiovascular disease, and obesity and its wide ranging consequences. It is timely to review the suitability of advice relating to fats in the light of recent publications, and the increasing rates of obesity in New Zealand.

A recent systematic review and meta-analysis examining the association between dietary fatty acids and coronary heart disease (CHD) by Chowdhury et al concluded that “current evidence does not clearly support guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats, and that nutritional guidelines may need reappraisal to reflect the current evidence”.\textsuperscript{8}

Fierce criticisms regarding a number of errors and omissions in the paper resulted in its undergoing revisions a day after publication. However the conclusions of the corrected version, now republished, remain unhelpful and a leading group of nutritional epidemiologists from Harvard have continued to call for the article to be retracted for misrepresenting the evidence on dietary fats.\textsuperscript{9} Here we consider the limitations of this new review and whether its conclusions are valid.

Chowdhury et al suggest that their review offers substantially new insights into the effects of dietary fats on CHD. They reported finding no significant association between CHD outcomes and intakes of SFA, MUFA, and both omega-3 (n-3 PUFA) and omega-6 polyunsaturated (n-6 PUFA) fats. The review relates principally to observational cohort studies, which have examined dietary intake in relation to subsequent CHD.

As reported in several previous reviews and meta-analyses there was no association between intakes of saturated, monounsaturated and n-6 PUFA and cardiovascular disease when the fatty acid groups were considered in isolation of other dietary
determinants.\textsuperscript{10,11} To some extent this could be due to the limitations inherent to dietary assessment methods, which make accurate measurement of the exposure variable difficult.

Chowdhury et al attempted to account for these limitations by including prospective cohort studies which examined associations between CHD and fatty acid intake biomarkers – a theoretically more objective measure of intake.\textsuperscript{8} However, the use of biomarkers to estimate fat intake is not well validated; for example the even-chain SFA biomarkers reflect both SFA intakes and endogenous synthesis from carbohydrates and alcohol.

Thus the finding of no link between fat intakes and coronary outcomes by Chowdhury et al. is likely to be confounded by other nutrient intakes, particularly carbohydrate, for which there has been no adjustment.

A rather different result emerges when considering also the nutrients which replace saturated fat (as would be the case in real life) rather than individual nutrients in isolation. The review by Jakobsen et al (2009)\textsuperscript{12} did just that in a meta-analysis of studies in which individual participant data was used as distinct from other meta-analyses based on aggregated study results.

This pooling approach is methodologically superior and made it possible to adjust for the same set of confounders across all of the data and to standardize the outcome measures. They found that substitution of 5\% of energy from SFA with 5\% of energy from PUFA was associated with a 26\% reduction in risk of coronary death based on 2155 deaths amongst 344,696 subjects followed-up for 4–10 years. Substitution of SFA with carbohydrate or MUFA was not associated with benefit.

A meta-analysis of eight randomised controlled trials by Mozaffarian et al (2010) provides further evidence that reducing SFA and replacing with PUFA lowers the incidence of CHD events.\textsuperscript{13} Results showed that each 5\% of total energy replacement of SFA by PUFA was associated with a reduction in CHD risk of 10\%, a finding consistent with the expected change in total cholesterol to HDL cholesterol ratio due to alteration in fatty acid intakes.

The apparent discrepancy between the two meta-analyses of the randomised controlled trials\textsuperscript{11,13} may be explained by the inclusion in Chowdhury et al of data from the recent re-analysis of the Sydney Diet Heart Study which reported a significant increased risk of coronary heart disease with replacement of SFA with PUFA—a finding at odds with the other studies included in the analysis.\textsuperscript{2}

This study involved dietary supplementation with large quantities (15\% of total energy intake) of plant oils and margarine containing primarily n-6 PUFA. This is well in excess of current recommendations for PUFA intake. In fact the addition of this trial did little to change the point estimate of relative risk, a benefit of a 14\% reduction in CHD events associated with replacement of SFA by PUFA, merely a widening of the confidence intervals.

Additional epidemiological and experimental evidence complements the data from the cohort studies and randomised controlled trials. Reductions in SFA intakes over the past several decades have been accompanied by substantial reductions in mortality from CHD\textsuperscript{14} which have occurred in parallel with serum cholesterol concentrations in
much of the Western world.15–17 This has occurred at all ages, so cannot be entirely attributed to treatment with statin drugs.

In Sweden, an increase in reported intakes of SFAs since 2004 has been associated with an observed increase in serum cholesterol levels.16 These ecological observations are also supported by findings relating the association between blood cholesterol and CHD in cohort studies. A meta-analysis of prospective cohort studies involving over 900,000 adults shows a linear association between blood cholesterol concentrations and CHD mortality.18

Innumerable controlled dietary studies, some dating back to the 1950s, confirm the potential of saturated fat to elevate cholesterol when compared with PUFA and MUFA.19,20 Thus we submit that the totality of evidence overwhelmingly supports the current guidelines and that the paper by Chowdhury et al adds no convincing new evidence to suggest that these should be revised.

Whether or not to recommend a reduction in total fatty acids is arguably a more contentious issue. Some have argued that there is no need to do so provided fatty acid composition is appropriate.7 Others claim that reducing total fat encourages an increased intake of carbohydrate, especially sugars.1

Of particular relevance to this debate is the finding of Hooper and colleagues who found in a carefully conducted meta-analysis, that reducing total fat intakes has the potential to facilitate weight loss and improve cardiovascular risk factors. With ever increasing rates of obesity this would be an appropriate justification for not further liberalizing recommended intakes of total fat.

Although it is appropriate to regularly review nutrition recommendations in the light of new evidence, given the overall understanding at the present time, we believe that the best quality evidence supports the current advice to reduce the intake of SFA and to replace this with healthier fats from sustainably managed fish, plant oils, nuts and seeds. However saturated fat should not be replaced with refined carbohydrates.

For those who are not overweight there may be no need to appreciably reduce total fat. However for individuals who are overweight and for populations with high rates of overweight and obesity, restricting total fat intakes should remain an important component of dietary advice.

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Weight and height measurement: potential impact in obstetric care

Emma Jeffs, Benjamin Sharp, Joanna Gullam, Helen Paterson

Abstract

Aim To assess the accuracy of reported weight and height in a pregnant population.

Methods Participants were recruited when attending their nuchal translucency scan if they attended with an ‘antenatal screening for Down syndrome and other conditions’ laboratory form (used for the maternal serum screening in the first trimester (MSS1) blood test) that had weight and/or height recorded. Participants’ weight and height were measured by trained recruitment centre staff and body mass index (BMI) was calculated. Differences in reported (MSS1) and measured weight, height and BMI were analysed using Bland-Altman plots.

Results 248 women participated. Only 23% (n=56) of participants had a weight recorded on the MSS1 laboratory form that was within ± 0.5 kg of measured weight: 62% (n=155) had an under-reported weight, and 15% (n=37) an over-reported weight. 30% (n=74) of participants had a correctly reported height: 26% (n=63) an under-reported height, and 44% (n=107) an over-reported height. 6% (n=14) of participants had a correctly reported BMI: 69% (n=166) had an under-reported BMI, and 25% (n=60) an over-reported BMI. 17% of participants (n=40) were incorrectly classified by BMI category based on MSS1 data.

Conclusion Our study suggests that there are considerable inaccuracies in the recording of weight and height during pregnancy in New Zealand. This results in a false reduction in BMI in many women which can affect clinical care.

It is widely reported in the literature that self-reported data tend to underestimate weight and overestimate height. Since weight and height are two of the most commonly used anthropometric measurements in clinical practice, inaccuracies in the measurement or recording of these indices could result in errors in clinical management. Throughout pregnancy weight, height and hence body mass index (BMI = weight [kg]/height$^2$ [m$^2$]) are routinely used to inform care.

Weight and BMI can significantly affect clinical care during pregnancy. Both inadequate and excess weight gain in pregnancy can affect a woman’s own health, as well as that of her infant. In view of this, it is recommended women be advised on ideal weight gain in pregnancy according to their pre-pregnancy or early pregnancy BMI (Table 1), and that weight gain throughout pregnancy be monitored.

At between 9 weeks and 13 weeks 6 days gestation, pregnant women are routinely offered an antenatal screen for ‘Down syndrome and other conditions’. This screening test includes the maternal serum screening in the first trimester (MSS1) blood test, which incorporates maternal weight in its prediction of risk algorithm for ‘Down syndrome and other conditions’, and the nuchal translucency (NT) scan.
The ‘Guidelines for Consultation with Obstetric and Related Medical Services’ (“Section 88”) recommend women with a BMI >35 be referred for consultation with an obstetric specialist, and that for women with a BMI >40 there is transfer of care to the specialist and anaesthetic consultation. It is also recommended that customised growth charts (‘GROW’ charts, accessed at: www.gestation.net) which consider maternal weight and height in their generation be used in pregnancy as these help to improve the recognition of small for gestational age (SGA) infants.

Table 1. 2009 Institute of Medicine Weight Gain in Pregnancy Guidelines

<table>
<thead>
<tr>
<th>BMI category</th>
<th>BMI range (kg/m²)</th>
<th>Recommended total weight gain (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
<td>12.5–18</td>
</tr>
<tr>
<td>Normal weight</td>
<td>18.5–24.9</td>
<td>11.5–16</td>
</tr>
<tr>
<td>Overweight</td>
<td>25–29.9</td>
<td>7–11.5</td>
</tr>
<tr>
<td>Obese</td>
<td>≥30</td>
<td>5–9</td>
</tr>
</tbody>
</table>

* Pre-pregnancy or early pregnancy BMI.

Figure 1. Antenatal screening for Down syndrome and other conditions laboratory form
Therefore, inaccurate documentation of weight and height (hence BMI) may affect the accuracy of education provided to women, the information given to women and their healthcare providers, and routine monitoring. When completing the ‘booking form’ at the beginning of pregnancy, there is evidence to suggest that women are being asked to report their weight and height, rather than being measured.\textsuperscript{13}

Weight and height are recorded on the ‘Antenatal screening for Down syndrome and other conditions’ laboratory form\textsuperscript{10} (MSS1 laboratory form, Figure 1). Women often have blood for the MSS1 test taken when attending their NT scan, if the testing centre is equipped to do so. This provides the opportunity to compare reported weight and height (on the MSS1 laboratory form) with measured weight and height at this time.

The aim of this study was to assess the accuracy of reported weight and height in a population of pregnant women, by comparing weight and height on the MSS1 laboratory form with measured weight and height at the time of the NT scan.

**Methods**

Participants were taking part in a larger study that aimed to describe women’s knowledge and perceptions of the risks of excess weight in pregnancy.\textsuperscript{14} Recruitment was undertaken at four community radiology centres in Christchurch, New Zealand in 2011. Participants were recruited when attending their NT scan at between 11 weeks and 13 weeks 6 days gestation. Participants who presented with a MSS1 laboratory form with weight and/or height recorded and who consented to have their weight and height measured were included in this study. Once recruited, participants were weighed on calibrated SECA 813 electronic scales, and had their height measured using SECA 206 or SECA 217 stadiometers.

Instruction on the correct use of both the scales and stadiometers was provided by research staff according to instructions given in the 2008/09 Adult Nutrition Survey (Accessed at: \url{www.health.govt.nz/publication/methodology-report-2008-09-nz-adult-nutrition-survey}, page 28). The weight and height documented on the MSS1 laboratory form were recorded for comparison. MSS1 weight was considered correct if within \(\pm 0.5\) kg of measured weight. Height in metres was rounded to two decimal places (dp). Raw data was used to calculate BMI to 1dp for both weight and height recorded on the MSS1 laboratory form and that measured at the time of scan form using the equation: 

\[ \text{BMI} = \frac{\text{weight (kg)}}{\text{height}^2 \text{ (m}^2)} \]

Differences in weight, height and BMI were analysed using Bland-Altman Plots and descriptive statistics were calculated, with the support of a biostatistician, using Microsoft Excel 2011.

**Results**

Data were available for both measured and MSS1 data for 248 participants for weight, 244 participants for height, and 240 participants for BMI (not all participants consented to having their weight and height measured, or had height and weight recorded on the MSS1 laboratory form).

The difference in weight measurements was from -15 kg (over-reported) to +12 kg (under-reported). In total, 62% (n=155) of participants had an under-reported weight on the MSS1 laboratory form. This is highlighted in Figure 2 as the majority of the data points (◆) are above the X-axis, representing a positive difference which is indicative of under reporting on the MSS1 laboratory form.
As measured weight increased, weight recorded on the MSS1 laboratory form became more discrepant, such that participants were more likely to have an under-reported weight. Only 23% (n=56) of participants had a weight recorded on the MSS1 laboratory form that was within ± 0.5 kg of measured weight, and 15% (n=37) had an over-reported weight.

The difference in height measurements was from -13 cm (over-reported) to +13 cm (under-reported) (Figure 3). Overall, 26% (n=63) of participants had an under-
reported height on the MSS1 laboratory form, 30% (n=74) a correctly reported height, and 44% (n=107) an over-reported height.

Figure 4. Bland-Altman plot of body mass index (BMI)

![Bland-Altman plot of BMI](image)

The difference in BMI measurements was from -3.8 kg/m² (over-reported) to +5.2 kg/m² (under-reported) (Figure 4). Overall, if based on reported not measured data, 69% (n=166) of participants would have had an under-reported BMI, 6% (n=14) a correctly reported BMI, and 25% (n=60) an over-reported BMI.

As measured BMI increased, BMI calculated from the MSS1 laboratory form data became more discrepant, such that participants were more likely to have a BMI that was less than measured BMI, and to a greater magnitude.

According to the MSS1 laboratory form data, 40 participants (17%) would have been incorrectly classified by BMI classification when compared with measured data. Twelve (26%) obese women would have been misclassified based on MSS1 data.

Discussion

This study identifies that documented height and weight recorded on the MSS1 laboratory form are not accurate at the time of the NT scan. Inaccurate recording or measurement of weight and height affects BMI calculation and BMI plays a significant role in recommended clinical care, specialist consultation and referrals in pregnancy.

In June 2012, the New Zealand Perinatal and Maternal Mortality and Morbidity Review Committee (PMMRC) recommended all women be advised on healthy weight gain in pregnancy according to their pre-pregnancy or early pregnancy BMI.8 The 2009 Institute of Medicine (IOM) guidelines for healthy weight gain in pregnancy (Table 1) have been internationally accepted and are the guidelines recommended by the PMMRC.
Weight gain in excess of the IOM guidelines (regardless of baseline BMI) has been associated with an increased risk for pregnancy-associated hypertension, gestational diabetes mellitus (GDM), complications during labour and delivery, postpartum weight retention and subsequent maternal obesity. An incorrect BMI could result in the failure to recognise a pregnancy as high risk and a woman being wrongly advised on appropriate weight gain, contributing to unnecessarily increased risk in pregnancy. Furthermore, if weight is not routinely monitored in pregnancy, inadequate or excess weight gain may not be identified and this could further contribute to increased pregnancy risk.

The Guidelines for Consultation with Obstetric and Related Medical Services (“Section 88”), which outline Crown payments to maternity providers providing primary maternity services, recommend women with a BMI >35 be referred for consultation with an obstetric specialist. For women with a BMI >40 it is recommended there is transfer of clinical responsibility and anaesthetic consultation. A number of adverse pregnancy outcomes are associated with maternal obesity (BMI ≥30) and these are well described within the literature. Such complications include GDM, hypertensive disorders of pregnancy, pre-eclampsia, preterm delivery, post-dates pregnancy, induction of labour, caesarean section delivery, infection and haemorrhage.

Risk associated with maternal obesity typically occurs as a continuum: there is increased likelihood of negative health outcome as maternal excess weight increases. In 2010 in New Zealand, BMI data was available for 92% of mothers of perinatal deaths. Over half (51.2%) of the mothers of perinatal deaths were overweight or obese, and 27% were obese. Thus, it is important that such high risk women are identified and referred appropriately.

GROW (gestation related optimal weight) charts are customised antenatal growth charts, recommended for use in all pregnancies. The chart is based on the calculation of an individualised weight standard for the duration of the pregnancy, adjusted for the physiological variables of maternal weight, height, parity and ethnicity.

If weight or height is incorrectly measured or reported it may lead to an incorrect GROW chart being created. Clinically, this could result in the under diagnosis of SGA and the overdiagnosis of large for gestational age (LGA). This is significant as SGA is associated with adverse outcomes including prematurity and stillbirth, whilst LGA might be the first presentation of GDM, which can present with both a large baby and polyhydramnios.

In February 2010 quality improvements to antenatal screening for ‘Down Syndrome and other conditions’ were introduced nationally in New Zealand. These recommend a first trimester combined screen which includes the MSS1 laboratory test taken at between nine weeks and 13 weeks 6 days gestation, analysing levels of pregnancy-associated plasma protein-A (PAPP-A) and beta human chorionic gonadotrophin (beta hCG), combined with an ultrasound scan, completed between 11 and 13 weeks six days, that measures the nuchal translucency. A risk assessment is calculated incorporating both of these results. Weight influences the risk assessment by means of a dilution effect on the PAPP-A and beta hCG.
reported on the MSS1 laboratory form could lead to the over or underestimation of risk (Table 2). If a woman is assessed as being of high risk (i.e. risk greater than 1:300) she will be offered an amniocentesis test which comes with a one percent risk of pregnancy loss.\(^{30}\)

**Table 2. An example of the impact of weight on risk prediction for Trisomy 21 (Down syndrome)**

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>Risk T21(^a)</th>
<th>Uncorrected 0.43(^b) PAPP-A MoM(^d) corrected for weight</th>
<th>Uncorrected 3.40(^c) Beta hCG MoM corrected for weight</th>
<th>NT MoM</th>
</tr>
</thead>
<tbody>
<tr>
<td>41</td>
<td>80</td>
<td>0.29</td>
<td>2.62</td>
<td>0.9</td>
</tr>
<tr>
<td>51</td>
<td>95</td>
<td>0.34</td>
<td>2.88</td>
<td>0.9</td>
</tr>
<tr>
<td>61</td>
<td>240</td>
<td>0.4</td>
<td>3.16</td>
<td>0.9</td>
</tr>
<tr>
<td>71</td>
<td>280(^e)</td>
<td>0.46</td>
<td>3.46</td>
<td>0.9</td>
</tr>
<tr>
<td>81</td>
<td>320</td>
<td>0.54</td>
<td>3.79</td>
<td>0.9</td>
</tr>
<tr>
<td>91</td>
<td>370</td>
<td>0.63</td>
<td>4.16</td>
<td>0.9</td>
</tr>
<tr>
<td>101</td>
<td>410</td>
<td>0.73</td>
<td>4.56</td>
<td>0.9</td>
</tr>
<tr>
<td>111</td>
<td>460</td>
<td>0.85</td>
<td>5</td>
<td>0.9</td>
</tr>
<tr>
<td>121</td>
<td>580</td>
<td>0.97</td>
<td>5.43</td>
<td>0.9</td>
</tr>
</tbody>
</table>

\(a\) T21: Trisomy 21, Down syndrome  
\(b\) Uncorrected MoM for PAPP-A for this patient  
\(c\) Uncorrected MoM for beta hCG for this patient  
\(d\) Multiple of median  
\(e\) Information supplied


If this woman was recorded as having a weight of 71kg, she would have been classified as being of high risk for Trisomy 21. If her weight was recorded as 81kg, she would have been assessed as being lower risk.

**Note:** Example presented with permission from: National Screening Unit, Ministry of Health, New Zealand

Whilst this study presents descriptive statistics pertaining to the measurement and recording of weight and height within pregnancy, it must be appreciated that each point on Figures 2–4 represents a woman whose pregnancy care may have been affected by inaccurate measurement or recording of weight or height.

A number of participants, particularly the 17% for whom an inaccurate BMI classification could have been given, could potentially have had care not in accordance with recommended practice (example: Table 3).
Table 3. An example of potential difference in care: a participant from this study

<table>
<thead>
<tr>
<th>Participant # 419</th>
<th>Measured data</th>
<th>MSS1 data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age: 25 years</td>
<td>80</td>
<td>70</td>
</tr>
<tr>
<td>Parity: 3 Gravida: 2</td>
<td>1.6</td>
<td>1.64</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>31.3</td>
<td>26</td>
</tr>
<tr>
<td>Height (m)</td>
<td>Obese</td>
<td>Overweight</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>5–9 kg</td>
<td>7–11 kg</td>
</tr>
<tr>
<td>BMI category</td>
<td>Low risk</td>
<td>High risk</td>
</tr>
<tr>
<td>IOM weight gain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>recommendation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSS1 risk (Down Syndrome)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Study limitations**—The main research, of which this was a sub-study, was not originally powered to consider the statistics presented in this paper and consequently this study reports descriptive statistics.

This study did not investigate the length of time between the Lead Maternity Carer (LMC) completing the MSS1 laboratory form and participants attending the NT scan, it was assumed that women attending their NT scan with the MSS1 laboratory form would have been intending to have their bloods taken at the time of the scan.

The period of time between completion of the MSS1 laboratory form and the NT scan may have resulted in small differences in weight. The IOM, however, note that average weight gain in the first trimester is less than 2 kg. This time difference would not affect the findings regarding height.

**Conclusion**—Weight, height and BMI play an important role in pregnancy care and this study presents evidence to suggest the measurement of these indices is not being correctly conducted, or that such indices are being estimated and not measured in all cases. This results in a false reduction in BMI which can affect clinical care.

**Implications for practice**—All women of childbearing age should have a weight measured and documented, and BMI calculated as part of routine clinical practice. When pregnant, BMI should be calculated from measured weight and measured height at booking/first visit (ideally before 10 weeks gestation) by the LMC.

**Competing interests:** Nil.

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**References:**
Management of excess weight in pregnancy in Otago, New Zealand: a qualitative study with lead maternity carers

Diana Fieldwick, Helen Paterson, Melanie Stephen, Angus Cameron, Richard Egan, Sarah McFadden, Justus Pienaar, Celine Sinclair, Toby Struthers, Kirsten Taplin, Clara Watson

Abstract

Aim To investigate the knowledge and practice of midwives providing lead maternity care (LMC) in Otago, regarding gestational weight gain (GWG).

Methods A qualitative study was conducted using three semi-structured focus groups and one in-depth interview. A total of 12 midwives, including one student midwife, were interviewed. Transcripts were analysed using generic coding and thematic analysis. At the conclusion of the focus groups no new themes were emerging.

Results Themes discussed included midwives' knowledge of GWG, methods used to identify BMI and weight gain throughout pregnancy, current management, barriers to management and tools used to overcome these barriers. There was satisfactory knowledge of the risks associated with excess GWG, however, adherence to current New Zealand guidelines and awareness of international guidelines in this area was limited. Management of GWG was highly varied and the weighing of pregnant women was not common practice. Sensitivity around the topic of weight management was identified as a major barrier to care.

Conclusions The management inconsistencies highlighted in this study have identified a need for New Zealand guidelines for the management of GWG. Clear guidelines along with increased education and collaboration between health professionals would help alleviate the current uncertainty regarding weight management in pregnancy.

Excess gestational weight gain (GWG) is associated with numerous risks for the mother and her child, contributing to serious health issues such as gestational diabetes, preeclampsia, maternal and childhood obesity.

The term GWG encompasses both the management of obese pregnant women and the management of weight gain during pregnancy. With recent statistics indicating the majority (64%) of New Zealand adults are obese or overweight, the issue of excess GWG is at the forefront of obstetric care and management is a challenge for all health professionals.

As midwives provide the majority of lead maternity care (LMC) in New Zealand; their knowledge and practice in this area has the biggest impact on the management of GWG.

Although there are no New Zealand guidelines for the management of GWG; the New Zealand College of Midwives and the New Zealand Perinatal and Maternal Mortality...
Review Committee (PMMRC) recommend that height and weight be measured at the first antenatal visit, and body mass index (BMI) be calculated.  

Obtaining an accurate baseline BMI is important to determine appropriate GWG and to identify at risk women who need specialist input. The Ministry of Health Referral Guideline for Consultant Obstetricians and Related Medical Services identifies having a BMI over 35 as a criteria for referral to an obstetrician, and recommends that if the BMI is over 40 the LMC must recommend a transfer of care to an obstetrician.  

International evidence-based guidelines for the management of women with obesity in pregnancy are available. These include protocols for monitoring weight gain, screening for high risk complications such as gestational diabetes and pre-eclampsia, and information about peripartum management. As there is no published research in this area involving LMC midwives in New Zealand information about adherence and knowledge of these guidelines is unknown.

In 2009, in response to the growing body of evidence on the risks of excess GWG, the American Institute of Medicine (IOM) updated their guidelines on recommended GWG based on pre-pregnancy BMI. Multiple international studies have looked at GWG in relation to the IOM guidelines including a multicentre study including participants from Auckland. This study found that over 74% of women were gaining more weight than suggested by the IOM. They also found that excess GWG was more likely in women who were overweight and obese at the beginning of their pregnancy.

Recent research which surveyed a group of New Zealand women identified that 70% of women were unaware of how much weight gain is appropriate in pregnancy. These findings suggest that New Zealand women may be lacking in education about GWG. Lack of, or incorrect, provider advice is independently associated with GWG above the IOM guidelines. It is primarily the role of the LMC to provide women with this information; with input from a general practitioner.

International studies on the knowledge and practice of maternity practitioners regarding GWG have found that GWG is often given a low priority by midwives and other maternity practitioners. Studies identified there are often gaps between the guidelines and practice concerning weight in pregnancy. Reasons for this included; limited education on this topic, the perceived lack of guidelines or being unfamiliar with guidelines regarding weight gain in pregnancy, the midwives’ own perspectives of weight gain during pregnancy, a lack of confidence in dealing with this sensitive topic, and limited resources. Despite this, both pregnant women and health care providers recognise pregnancy as a time of increased motivation for lifestyle change and an opportune time to educate about healthy lifestyle.

A literature search identified no publications on midwives’ knowledge and practice regarding GWG in New Zealand. This study aimed to explore the knowledge and current practice of LMC midwives in the Otago region, using a qualitative methodology to obtain an in-depth understanding. The focus group method was preferable in this setting as it allows for an intimate exploration of the midwives understanding, perceptions and experiences in this area in a group setting where these issues can be discussed in context to shed light on the practicalities and complexities within which the midwives practice.
The content of the conversation was able to be directed by the midwives for a thorough exploration of all pertinent themes. The focus group method is therefore not restricted to predetermined themes, a benefit over using a quantitative approach. Finally, focus groups were chosen as they provide a large amount of data in a short period of time without large numbers of participants.

Methods

This was a generic qualitative study using three semi-structured focus groups and one in-depth interview. The research team included nine trainee interns and their supervisors. Ethical approval for the study was obtained according to University of Otago Ethics protocols (Category B). The eligible population included the 100 practising LMC midwives in the Otago region. A mixture of purposive sampling and snowball sampling was used. Recruitment was assisted by a local midwifery council representative, and was undertaken via meetings, email and posters.

Three focus groups of two to five midwives and one key informant interview were conducted totalling 11 midwives and one student midwife from Dunedin, Balclutha and Wanaka. Focus groups took place at Dunedin Public Hospital and Balclutha Hospital during April 2013. Focus groups were facilitated by one researcher and where possible were co-facilitated by a midwife, as recommended by the ethics representative.

The focus groups and interview were semi-structured to establish open discussion based on predetermined points introduced by the facilitator. These questions were based around the key themes of; knowledge of gestation weight gain (GWG), identification of women with excess weight in pregnancy, risks associated with excess GWG, current management and challenges in managing women with excess weight in pregnancy and initiatives that work well to overcome these challenges. The facilitator acted as a prompt for discussion points and was largely removed from the intricacies of the conversation. By limiting the focus group guidance to several key questions it was hoped that the themes that emerged would be those considered important to the midwives.

All of the focus groups and the interview were recorded and transcribed verbatim. Focus groups and interview lasted 30 to 50 minutes.

Content analysis focusing on thematic analysis was undertaken, using a general inductive approach as described by Thomas (2006). This approach aims to condense raw textual data to a brief summary which can then be used to link findings with research objectives, allowing the emergence of a framework of underlying processes and experiences. Using this technique a number of codes were derived from close reading of the transcripts.

Over-arching themes were chosen guided by research objectives and focus group content. Categories and subcategories were created to reflect themes emerging from the text and codes. A description of each subcategory was written and quotations were selected that conveyed the core essence of the major themes. The local midwifery council representative was consulted about the thematic analysis and description of categories and subcategories. Saturation sampling was suggested by a lack of new emerging categories in the final interview.

Results

The 11 midwives and one student midwife ranged in both age (27–60 years) and experience (0–35 years). The average age was 43 and the average clinical experience was 12 years. All the participants were female.

Knowledge of gestational weight gain—Overall there was a good understanding of the risks associated with excess weight in pregnancy, and an awareness across all focus groups for the need to record client BMI. Most midwives were unsure of the exact details of New Zealand guidelines regarding specialist referral and there was varied knowledge on appropriate diabetes screening. Table 1 shows representative quotes from the midwives about their knowledge of gestational weight gain (GWG).
Table 1. Theme 1 – knowledge of gestational weight gain

<table>
<thead>
<tr>
<th>Category</th>
<th>Subcategory</th>
<th>Representative quotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risks</td>
<td>FG1 (M2)</td>
<td>“Do you want the tick list? Hypertension, pre-eclampsia, small for dates, large for dates, gestational diabetes, poor birth outcomes.”</td>
</tr>
<tr>
<td></td>
<td>FG2 (M1)</td>
<td>“Malpresentation, you can’t feel.”</td>
</tr>
<tr>
<td>Protocols</td>
<td>FG2 (M2)</td>
<td>“There is a section you have to fill out on the height and weight bit.”</td>
</tr>
<tr>
<td>Identification</td>
<td>FG2 (M2)</td>
<td>“For the obstetric appointment I would refer over 40, BMI. It’s supposed to be over 35 isn’t it?”</td>
</tr>
<tr>
<td>Referral</td>
<td>FG1 (M4)</td>
<td>“I haven’t done (an HbA1c) but I’m wondering if I should.”</td>
</tr>
<tr>
<td>Diabetes screening</td>
<td>FG2 (M5)</td>
<td>“I do HbA1c mostly, but I have my own glucometer. Sometimes polycose, but if they have risk factors I go straight to GTT, because I have suspicions, I don’t trust those polycose too much.”</td>
</tr>
</tbody>
</table>

Key: FG = focus group INT = interview  M = midwife  SM = student midwife.

Identification of baseline BMI and weight gain throughout pregnancy—The most common method for obtaining a baseline BMI was using a woman’s self-reported height and weight. Visual estimation and measurement of height and weight were also used. The issues highlighted as barriers to obtaining BMI and monitoring weight were a lack of appropriate equipment, sensitivity around the topic of weight, and the varied consensus within the midwifery community about standard practice.

None of the midwives weighed their clients at every visit and the majority did not use scales to obtain BMI. Despite the common practice of relying on self-reported weights and heights, many midwives did recognise that this is often inaccurate. Table 2 shows representative quotes from midwives discussing the theme “identification of baseline BMI and weight gain throughout pregnancy”.

Current management of women with excess weight in pregnancy—Current management for women with excess gestational weight involved education, referral, increased monitoring and tighter restraints around the birth plan. Most of the midwives reported discussing issues around diet and exercise with their clients. These were mostly verbal discussions, however; one midwife gave her client written information about how much weight to gain during pregnancy, and another gave written information about diet.

The majority of midwives acknowledged the importance of involving other health care providers such as dieticians and obstetricians, however, only a small proportion referred women with a BMI over 35 to antenatal clinic as the Ministry of Health advises. Most midwives organised more frequent ultrasound scans for women with higher BMIs and tailored their diabetes screening. How and when these screening tools were employed was variable however; both between the midwives and between her clients.

The majority of the midwives avoided home birth for women with a “high” BMI, and preferred vaginal delivery. Table 3 shows representative quotes from midwives discussing their current management of women with excess weight in pregnancy.
Table 2. Theme 2 – Identification of baseline BMI and weight gain throughout pregnancy

<table>
<thead>
<tr>
<th>Category</th>
<th>Subcategory</th>
<th>Representative quotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current methods of identification</td>
<td>Visual estimation</td>
<td>FG1 (M3): “I... well, I do (measure the BMI). I have a look at them.”</td>
</tr>
<tr>
<td></td>
<td>Measure height and weight</td>
<td>FG2 (M2): “You can tell by looking at a woman what her BMI is anyway.”</td>
</tr>
<tr>
<td></td>
<td>Self-report</td>
<td>FG1 (M3): “If they look really overweight or underweight, I calculate it. And I do weigh them, but not absolutely strictly.”</td>
</tr>
<tr>
<td>Barriers to obtaining BMI and monitoring weight gain</td>
<td>Equipment</td>
<td>FG2 (M2): “Sometimes the weight is a bit inaccurate because I don’t carry scales around with me.”</td>
</tr>
<tr>
<td></td>
<td>Sensitivity</td>
<td>FG2 (M1): “Just because I don’t have scales I think is the only reason I haven’t really.”</td>
</tr>
<tr>
<td></td>
<td>Variation in practice</td>
<td>FG2 (M2): “It’s a really sensitive issue for me... I have a lot of trouble talking to women about their weight...the concept of weighing a woman each time I see her. If she’s a normal weight then I probably wouldn’t.”</td>
</tr>
</tbody>
</table>

FG2 (M1): It hasn’t been standard practice for me or anyone else I’ve worked with. Even as a student I didn’t work with any midwives who routinely weighed people. It sort of became not the done thing and now it’s sort of just coming back.

Table 3. Theme 3 – Current management of women with excess weight in pregnancy

<table>
<thead>
<tr>
<th>Category</th>
<th>Subcategory</th>
<th>Representative quotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>Diet and exercise</td>
<td>INT: “I do a diet assessment”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG1 (M3): “I have diet sheets and I talk to them about what weight I would like them to put on.”</td>
</tr>
<tr>
<td>Referral</td>
<td>Timing of referral and transfer of care</td>
<td>FG1 (M1): “don’t bother with 35 anymore...you just don’t get anywhere.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG1 (M4): “I’m more inclined to send them (to antenatal clinic) when I’ve got a 28 week glucose tolerance test.”</td>
</tr>
<tr>
<td>Increased monitoring</td>
<td>Screening</td>
<td>FG3 (M1): “I give them serial growth scans because it’s harder to palpate.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG2 (M2): “we can order an HBA1c at booking...or maybe a GTT at 16-18 weeks instead of 24.”</td>
</tr>
<tr>
<td>Impact on birth plan</td>
<td>Location and mode of delivery</td>
<td>FG1 (M3): “I tell them...persuade them, that they should come to Queen Mary to have their babies.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG1 (M2): “we should be promoting an upright...staying upright, staying mobile because you’re not going to heal from a caesarean.”</td>
</tr>
</tbody>
</table>

Challenges and barriers to management—Many challenging aspects of managing excess weight in pregnancy were identified during the focus groups. Communication difficulties was a major challenge emphasised in the focus groups. This was predominantly due to a perception that excess weight in pregnancy is a sensitive issue and that discussion around it could offend women.
Other challenges highlighted were cultural difficulties, factors relating to the perceptions and context of the pregnant woman including a poor understanding of the consequences of excess GWG, and lack of resources for making changes. The main barriers were the widely perceived lack of usefulness of antenatal referral and attitudes to weight, in particular a tendency to normalise high BMI.

Table 4 shows representative quotes from the midwives discussing the challenges and barriers to management.

Table 4. Theme 4 – Challenges and barriers to management

<table>
<thead>
<tr>
<th>Category</th>
<th>Subcategory</th>
<th>Representative quotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Communication</td>
<td>Sensitivity</td>
<td>FG3 (SM): “It’s a sensitive subject, we don’t want to hurt people’s feelings.” FG1 (M4): “It is interesting about when you introduce that notion though of the added risk of birth outcome, because, if you introduce it early, you run the risk of upsetting someone.”</td>
</tr>
<tr>
<td></td>
<td>Cultural challenges</td>
<td>FG2 (M1): “It’s kind of the norm isn’t it. Everyone in their family is big…” FG2 (M3): “…But Pacific Island women love to be big. It’s just a nightmare when they get to the 45-50 category.”</td>
</tr>
<tr>
<td>Perceptions and context of pregnant women</td>
<td>Attitudes to clinics and interventions</td>
<td>FG2 (M1): “It’s easy enough to refer, it’s actually getting women to go…” FG1 (M1): “I’ve only had 2 referrals for high BMI and neither of them went to clinic.”</td>
</tr>
<tr>
<td></td>
<td>Lack of understanding</td>
<td>FG3 (SM): “I think often it’s a “I’m pregnant, free for all, I’m going to get fat I may as well get fat”, you know what I mean.” FG3 (SM): “They do worry that they shouldn’t restrict their food in pregnancy because they may restrict the baby’s growth.”</td>
</tr>
<tr>
<td></td>
<td>Lack of resources</td>
<td>FG2 (M5): “it’s expensive for a woman in a low socio-economic environment…they can’t afford to go to the gym…” FG2 (M1): “Junk food is cheaper than healthy food.”</td>
</tr>
<tr>
<td>Referral</td>
<td>Perceived lack of usefulness of antenatal referral</td>
<td>FG1 (M3): “I don’t refer them. Why go down to the clinic – ‘hello, how are you? Yeah your BMI is this…’ They’re not going to do much more.” FG1 (M2): “They don’t do anything differently than what we would do. We can order all of those blood tests and do all of those things.”</td>
</tr>
<tr>
<td></td>
<td>Variable approach by antenatal clinicians</td>
<td>FG1 (M2): “The other thing (about antenatal clinic) is that registrar’s comfort level with talking about weight really, really varies.” INT: “There is no consistency with who they see and I see this as a barrier to care.”</td>
</tr>
<tr>
<td></td>
<td>Variable success in referral to dietician/scans</td>
<td>INT: “Access to the community dietician would help a bit. There is already a 6 week waiting list to be assessed.” FG1 (M1): “I had (radiology service) question the need for serial scans on a woman who had a large BMI.”</td>
</tr>
<tr>
<td></td>
<td>Transfer of care</td>
<td>FG1 (M1): “Wouldn’t actually transfer care, always remain involved. Involve them in it maybe, but not transfer.” FG1 (M2): “even though the referral guidelines say transfer, there’s no offer.” (M3): “Yeah I know, it’s stupid.”</td>
</tr>
<tr>
<td>Attitudes to weight</td>
<td>Normalisation of higher BMI</td>
<td>FG2 (M1): “My biggest women went on to have completely normal pregnancies and labours.” FG1 (M1): “Because I mean you can have women with very large BMIs that have birthed well before ….”</td>
</tr>
</tbody>
</table>

**Overcoming barriers**—The midwives had a range of opinions on strategies to overcome some of the barriers in managing women with excess GWG. These
included the importance of continuity of care, maintaining a practical focus, and utilising other health professionals. They also emphasised a need to improve access to dieticians, and some recognised a need for clearer guidelines.

It was suggested that a guideline should be developed around how to speak to women about excess gestational weight to facilitate consistent levels of care. Another suggestion was to combine the Healthy eating in pregnancy brochure with the Food safety brochure and add a section on recommended weight gain in pregnancy. Table 5 shows representative quotes from the midwives discussing the theme “overcoming barriers”.

Table 5. Theme 5 – Overcoming barriers

<table>
<thead>
<tr>
<th>Category</th>
<th>Subcategory</th>
<th>Representative quotes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midwifery approach</td>
<td>Continuity of care</td>
<td>FG1 (M2): “This is core midwifery stuff, and I think anything that is going to be around supporting women to control excess weight gain, the best place to do that is within the midwifery relationship.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG2 (M2): “We need to make it a habit from booking so they know it’s what happens every time, it’s like BP now, it’s habit. We should do the weigh.”</td>
</tr>
<tr>
<td>Practical focus</td>
<td>Attainable goals</td>
<td>FG1 (M2): “Focus on do-able objectives, achievable goals.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG1 (M2): “I’ve found some women are happy to focus on weight, on limiting weight gain, and other women are happier to focus on goals around diet and exercise, which is just as effective.”</td>
</tr>
<tr>
<td></td>
<td>Acquiring scales</td>
<td>FG2 (M4): “But I do think that if there was a bulk order of scales that we could all access it would increase the likelihood that midwives would…”</td>
</tr>
<tr>
<td>Collaborative approach</td>
<td></td>
<td>FG1 (M2): “I do think that offering the referral and for them to go along and have a chat with the doctor who says this is a significant issue is of benefit…”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG3 (SM): “Being able to have more than one health practitioner involved in looking after them because at least they reiterate what you’re saying, what it’s about. Talking to different people it’s better than just one person…”</td>
</tr>
<tr>
<td>Suggested improvements</td>
<td>Dietician</td>
<td>FG1 (M2): “I would love it if antenatal clinic was offering dietician, nutrition, exercise, all that kind of stuff.”</td>
</tr>
<tr>
<td></td>
<td>Guidelines</td>
<td>FG2 (M1): “I do think that the obstetric unit can do with a full time dietician.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG3 (SM): “Establishing a guideline about how to speak to women, something we take women through and discuss what, I guess how to approach it better.”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FG2 (M4): “Consistent information from healthcare providers. So you know, clear guidelines of weight gain. Clear guidelines of healthy eating.”</td>
</tr>
</tbody>
</table>

Discussion

This study found that there is significant variability in midwife knowledge and practice regarding excess weight in pregnancy. The LMC midwives in these focus groups were all dedicated to providing the best care for their clients and exhibited notable empathy for women in this sensitive area. The midwifery approach is characterised by working in partnership with women to provide the necessary support, care and advice during pregnancy, labour and the postpartum period.

This study reveals that a substantial amount of the variability in practice in this area arises from this partnership approach, in which midwives balance the perceived benefits of discussing and managing excess GWG against perceived potential harms.
This highlights the complexity of this issue, and how practice could be influenced by guidelines that clearly promote best practice, and practical tools that address communication difficulties.

We identified that while knowledge of the risks associated with excess GWG was satisfactory, knowledge of guidelines was vague and the current practices of many midwives are not supported by international best practice guidelines.

Management is significantly affected by communication difficulties that arise from the sensitivity surrounding the topic of excess weight. This is consistent with international publications in which health practitioners identified a lack of confidence in communicating about weight as a major barrier to optimal management of gestational weight.\textsuperscript{18-22}

All of the midwives included in this study were aware of the need to record the baseline BMI. However, contrary to the Midwifery College recommendation,\textsuperscript{6} the majority of the midwives used visual estimation or self-reporting to establish BMI. As management must be guided by BMI,\textsuperscript{8} accuracy is important.

Research suggests that both visual estimation and self-reporting of BMI are unreliable.\textsuperscript{28,29} Calculation of BMI is advocated by national and international bodies,\textsuperscript{5,7,9,10} and should be recommended as the standard of care in New Zealand; the fact that it is not may indicate that midwives are not aware of these recommendations and guidelines. Weighing throughout pregnancy to monitor GWG and providing written information about appropriate GWG was not routine practice for the midwives.

Research indicates that regular weighing of overweight women during pregnancy can help to reduce excess GWG.\textsuperscript{30} Correct provider advice about appropriate weight gain is associated with GWG within the IOM guidelines.\textsuperscript{13,15} Education is a vital part of the LMCs’ role and increasing awareness on the importance of educating clients about appropriate GWG is needed.

Most of the midwives interviewed agreed that increased ultrasound scanning (USS) and diabetes screening were important for management of women with excess weight in pregnancy. However, there was no clear consensus of how this should be done.

The lack of uniform management shown in this study highlights the need for New Zealand guidelines. There are many international guidelines; however, implementation of a New Zealand guideline advocated by both the New Zealand College of Midwives and the RANZCOG could go a long way to ensuring the optimal standard care is provided for all pregnant women in New Zealand.

The majority of midwives interviewed were aware of the Ministry of Health referral guideline, however, adherence to the recommendation regarding maternal BMI was not common practice.

The midwives felt that specialists had nothing more to offer in the way of education, monitoring, or practical tools for the women than what they would offer themselves, and none of the midwives agreed with the idea of transferred care based solely on BMI. Improving collaboration between midwifery and obstetric communities and providing a more practical focus at antenatal clinics may assist with adherence to this government guideline.
The issue of sensitivity has been recognised among many groups of LMCs as a barrier to both weighing pregnant women and talking to them about GWG. In this study sensitivity was discussed by all the midwives as a major barrier affecting their practice. They were concerned about the feelings of their clients, and felt that often the more overweight clients would not want to know their weight.

The midwives felt insisting on weighing a client or lecturing about GWG could have a negative impact on the LMC-client relationship. Increased training in this area to provide LMCs with new communication tools to address this subject may help overcome this barrier.

**Limitations**—This study had a small number of participants (12). However, using a qualitative approach we were able to examine the midwives knowledge and practice in depth and the number of participants was sufficient to meet our aim. Nearly all participants identified themselves as being of NZ European ethnicity and although thematic saturation was suggested by the lack of new themes emerging in the final interview, it is possible that further sampling of other midwives, for example of different ethnicities, could have revealed other important themes. Risks imposed by this method of research include issues around confidentiality and anonymity. However, as the midwives were all familiar with each other these risks were largely avoided.

**Conclusion and clinical implications**—Excess GWG and excess weight in pregnancy are important issues and this was recognised in the knowledge of GWG theme. Adequate monitoring of GWG is necessary for the health of both the pregnant woman and her baby; however, as identified in theme three, there is significant variation in current management.

In light of the challenges and barriers to management identified by the participants of this study in theme four; our key recommendation is for the creation of New Zealand guidelines for the management of GWG to help close the gap between knowledge and practice. Concise GWG guidelines that are accepted by all health professionals will help to ensure the best possible care is provided for pregnant women in New Zealand. Table 6 summarises our recommendations based on the data arising from this study.

**Table 6. Recommendations to improve management of gestational weight gain (GWG)**

| Guidelines | For LMCs managing all pregnant women  
Protocols on:  
Measure baseline BMI and monitoring gestational weight gain.  
Written early pregnancy education on healthy lifestyle, IOM recommended weight gain, and the risks of excess GWG.  
Diabetes screening for women with raised BMI  
Ultrasound scanning for women with raised BMI  
Referral criteria for dietician, obstetrician, anaesthetist  

| Education | For midwifery and medical health care providers:  
To increase knowledge of existing guidelines  
Provide health care professionals with better communication tools to boost confidence and improve management  

| Collaboration | Between midwives and obstetricians:  
To address the issue of transfer of care  
Improve access and outcomes of clinic referrals  


Future work—Further research into this area is needed to provide a strong base of evidence to assist with the development of GWG management guidelines.

Competing interests: None.

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Acknowledgements: We acknowledge Clare Cameron for her involvement and thank all the LMC midwives who took part in the research, with a special thanks to Prue Thompson.

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References:
Duodenal switch—the initial experience in New Zealand

Martyn L Humphreys, Steven J Robinson, Caryne McKeand, Hisham Hammodat
(This paper is based on a presentation given to the Royal Australasian College of Surgeons Annual Scientific Meeting, Auckland, May 2013)

Abstract

Aims The duodenal switch (DS) has now established itself as an effective, durable and safe bariatric procedure. We present our initial experience on 60 patients from May 2008 to November 2012.

Methods Retrospective case series from a prospective database. 94.8% follow-up over 4 years.

Results 45 patients have completed 1-year follow-up and 28 patients completed 2-year follow-up. The mean initial body mass index (BMI) was 52.8 kg/m$^2$ (range 40–66 kg/m$^2$). The excess weight loss has been 69.5% at 1 year (n=45) and 73.1% at 2 years (n=28) respectively. The mean hospital stay is 5.08 days (range 3–18). The range of bowel motions at 1 year is one to two movements per day. Comorbidity resolution rates were 95% (n=18) for diabetes, 100% (n=9) for obstructive sleep apnoea, 72% (18/25) or hypertension, and 92% (33/36) or dyslipidaemia. One death from liver failure occurred 9 months following surgery resulting from poor compliance with follow-up and intake of multivitamins.

Conclusion In our short-term analysis DS appears to be very efficient in terms of cure rate for morbid obesity and its comorbidities. In terms of risk/benefit DS has appeared safe with adherence to the appropriate follow-up regimen.

Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPL</td>
<td>Biliopancreatic limb</td>
</tr>
<tr>
<td>DS</td>
<td>Duodenal switch</td>
</tr>
<tr>
<td>LSG</td>
<td>Laparoscopic sleeve gastrectomy</td>
</tr>
<tr>
<td>OGTT</td>
<td>Oral glucose tolerance test</td>
</tr>
<tr>
<td>RYGB</td>
<td>Roux-en-Y gastric bypass</td>
</tr>
<tr>
<td>LAGB</td>
<td>Laparoscopic adjustable gastric banding</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>

Duodenal switch (DS) is an improved version of the initial biliopancreatic diversion Scopinaro-type operation (BPD-SC) where the distal gastrectomy was replaced by a sleeve gastrectomy for preserving gastric function and the common channel was lengthened to increase the role of biliopancreatic secretions.$^{1–3}$ See Figure 1.

The hallmark of the DS is pylorus preservation. The distal gastrectomy component of Scoparino’s BPD had two main problems; marginal ulceration at the jejunal side of the anastomosis and dumping syndrome.$^{1,4–6}$ By substituting a sleeve gastrectomy for the distal gastrectomy, the pylorus is preserved and marginal ulceration and dumping syndrome are virtually eliminated.
The goal in diverting pancreatic secretions and bile was to decrease caloric and fat absorption while preserving normal nourishment. Later additional benefits were discovered including remission of diabetes. The mechanism of action is complex but by bypassing the proximal intestine and reaching the distal small bowel directly, there is an alteration in the secretion of intestinal hormones but synergistically act to induce remission of diabetes and prevent obesity.\textsuperscript{4–7}

Historically there has been resistance to anatomical routing to create a change in the physiological pathway, previous experience in the 1960s with an operation called “intestinal bypass”, which whilst initially demonstrated good results became associated with malabsorptive side effects, mainly because of the very long intestinal bypass and the creation of a long blind loop of bypassed intestine.

With a greater understanding of the physiological disturbance underpinning type 2 diabetes these changes in the physiological pathway offer a mechanism for the remission of diabetes.\textsuperscript{7,8}
Marceau et al. after 10 years’ experience with the original BPD-SC changed to DS, due to fewer side effects and improved absorption of both protein and fat soluble vitamins without compromising weight loss. After 20 years of performing DS as the primary procedure for all morbidly obese patients Marceau et al have published their long-term results, which includes the follow-up of over 1423 patients.

Resolution of diabetes and other comorbidities

Defining remission or cure of diabetes is not as straightforward as it may seem. Diabetes is defined by hyperglycaemia, which exists as a continuum and may be impacted over a short time frame by treatment or events (medications, diet, activity, intercurrent illness).

A consensus group agreed upon the following definitions, which are the same for type 1 and type 2 diabetes:

- Remission is defined as achieving glycaemia below the diabetic range in the absence of active pharmacological (anti-hyperglycaemic medications) or surgical (ongoing procedures such as repeated replacements of endoluminal devices).

- A remission can be partial or complete. Partial remission is defined as sub-diabetic hyperglycaemia (HbA1C <6.5%) of at least 1 year’s duration in the absence of active pharmacologic therapies or ongoing procedures. Complete remission is defined as a return to normal measures of glucose metabolism (HbA1C in the normal range) for at least 1 year’s duration.

In 2004, Buchwald et al published a meta-analysis of various types of bariatric surgery on weight loss and resolution of type 2 diabetes and other obesity related comorbidities.

DS was shown to be superior to RNYGB, gastroplasty and laparoscopic gastric banding (LAGB) in terms of weight loss, resolution of type 2 diabetes, hyperlipidaemia treatment, hypertension treatment and obstructive sleep apnoea (OSA).

Body composition—Strain et al showed that DS resulted in better overall weight loss and greater fat loss and better preservation of lean body mass than RNYGB, LAGB and LSG.

Nutritional complications—In a randomised study of vitamin status after LRYGB and DS, Aashiem et al found that patients who underwent DS may be associated with a greater risk of Vitamin A and D deficiencies in the first year after surgery. Thiamine deficiency was also more common in the DS group in the first few months after surgery.

Materials and Methods

Definition of cohort and baseline demographic characteristics—All patients offered a Duodenal Switch as a primary weight loss procedure at North Shore Hospital, Auckland, New Zealand between May 2008 and November 2012 were included in this cohort. Patients were identified using the local registry of bariatric patients, all observations were entered prospectively into a local bariatric database by a single surgeon. Baseline demographic (Age, sex, ethnicity, height, weight BMI) and clinical (prior diabetes, hypertension, liver disease) data were
collected. Details of the bariatric procedure and preoperative BMI were recorded. The last known preoperative BMI prior to surgery was recorded.

Height and weight was recorded and updated during outpatient clinic visits at 3 weeks, 6 weeks, 3 months, 6 months, 9 months, 12 months and annually thereafter.

Operative details—All procedures were performed open through an upper midline vertical laparotomy. The sleeve gastrectomy was fashioned over a 36 F Bougie with multiple firings of a linear stapler device beginning at 6 cm proximal to the pylorus and continuing to the angle of His. An ultrasonic dissection device was used to mobilise the greater curvature of the stomach. The first part of the duodenum was mobilised for approximately 4 cm distal to the pylorus; the duodenum was Kocherised.

The duodenum was divided with a linear stapler 4 cm distal to the pylorus. Small bowel length was measured with a 15 cm umbilical tape. The small bowel was divided at 50% of its total length from the ileocaecal valve using a linear stapler. A retrocolic end to side double layered anastomosis was performed between the proximal duodenum and the alimentary limb of the small bowel using continuous 3/0 PDS (Ethicon) suture.

The biliopancreatic limb was anastamosed side to side using a 60 mm linear stapler with a common channel at 10% of total small bowel length but not less than 100 cm from the ileocaecal valve. Mesenteric defects were closed with 3/0 PDS (Ethicon). The greater omentum was reattached to the neostomach with 3/0 PDS (Ethicon).

Nutritional supplements/support—All patients received the following daily supplementation postoperatively:

- Cholecalciferol 1.25 mg once weekly
- Ferrous fumarate 310 mg and folic acid 350 mcg twice daily (as Ferro-F)
- Vitamin B₁₂ 1000 mcg once monthly
- Calcium 2458 mg as calcium citrate powder 1.5 teaspoon once daily
- Centrum® Advance 50+ (Pfizer) once daily
- Vit ABDECK™ (Pharmaco) capsule twice daily
- Vitamin A 10,000 iu daily

The cost of these supplements at the time of writing is approximately NZ$2 per day.

All patients received a one-to-one 1-hour nutritional counselling session with a qualified bariatric dietician prior to surgery. Patients were seen 2-4 weeks prior to the surgery date at the surgical outpatient clinic where the technical details of the operation were discussed in addition to the expected recovery and potential complications. Patients were handed over a surgical consent form for them to take home, read and bring back to the hospital on the admission day. On both occasions written information was given regarding diet and supplements. Further dietician sessions were provided at 1, 3, 6, 9, 12, 18 and 24 month follow-up visits. Telephone numbers were provided for the dietician and bariatric nurse specialist in the event of queries arising between clinic visits.

Excess weight loss—Height and weight are measured routinely at all attendances for postoperative follow-up.

In the absence of a pre-existing diagnosis of diabetes mellitus, preoperative glucose and HbA1c measurements were used to identify undiagnosed cases, using WHO criteria.

Remission of diabetes—All available glucose levels were analysed using WHO criteria.

Data was analysed using STATA® v.11 for Windows® software (StataCorp, Texas). Appropriate parametric and non-parametric methods were used as necessary. Survival analysis was performed using the Cox proportional hazards model. Statistical significance was set at p=0.05.

Results

Baseline demographic data is summarised in Table 1 below.
Table 1. Baseline demographics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>22–59 (43)</td>
</tr>
<tr>
<td>Gender</td>
<td>47F 13M</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>40–66 (52)</td>
</tr>
<tr>
<td>Weight at presentation (kg)</td>
<td>113–235 (150)</td>
</tr>
</tbody>
</table>

Numbers in parentheses are median values.

94.8% follow-up of these patients over the last 4 years, including clinical biochemistry evaluation; 45/49 have completed 1-year follow-up and 28/33 have completed 2-year follow-up.

The median initial body mass index (BMI) was 52.8 kg/m² (range 40–66 kg/m²). After a mean follow-up of 2.4 years (range 2 months to 4 years), the excess weight loss has been 69.5% and 73.1% at 1 year (n=45) and 2 years (n=28), respectively. See Figure 2.

**Figure 2. Weight loss post surgery**

![Graph showing weight loss over time](image)

The mean hospital stay is 5.08 days (n=61), range 4–6 days. The range of bowel motions at 1 year is one to two movements per day.

Remission of diabetes was achieved (i.e. medication was discontinued) in 94.7% (18/19).
The nutritional impact has been acceptable with one case of albumin deficiency (<30 g/L), one case of vitamin A deficiency and seven cases of ferritin deficiency. See Table 2 below.

Table 2. Nutritional impact

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin deficiency (&lt;30 g/L)</td>
<td>0</td>
<td>1 (2.0%)</td>
</tr>
<tr>
<td>Vitamin D deficiency (&lt;50 nmol/L)</td>
<td>33 (67.3%)</td>
<td>5 (10.2%)</td>
</tr>
<tr>
<td>Raised parathyroid hormone (&gt;7.3 pmol/L)</td>
<td>1 (2%)</td>
<td>5 (10.2%)</td>
</tr>
<tr>
<td>Folate (&lt;7 nmol/L)</td>
<td>3 (6.1%)</td>
<td>1 (2.0%)</td>
</tr>
<tr>
<td>Vitamin B12 deficiency (&lt;170 pmol/L)</td>
<td>2 (4.1%)</td>
<td>4 (8.2%)</td>
</tr>
<tr>
<td>Vitamin A deficiency (&lt;0.7 µmol/L)</td>
<td>0</td>
<td>3 (6.1%)</td>
</tr>
<tr>
<td>Ferritin deficiency (&lt;20 µg/L)</td>
<td>5 (10.2%)</td>
<td>11 (22.4%)</td>
</tr>
</tbody>
</table>

Four patients have suffered significant postoperative complications including pancreatic fistula, myocardial infarction, pneumonia and intraoperative splenectomy in different individuals.

See Table 3 below. No patients have required reversal of their procedure in this series. One death from liver failure occurred 9 months post surgery.

Table 3. Early and late complications

<table>
<thead>
<tr>
<th>Early (&lt;30 days postoperatively)</th>
<th>Late (&gt;30 days postoperatively)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 pneumonia</td>
<td>5 incisional hernia</td>
</tr>
<tr>
<td>1 pancreatic fistula</td>
<td>4 wound sinus</td>
</tr>
<tr>
<td>1 myocardial infarction</td>
<td>1 gallstones (laparoscopic cholecystectomy)</td>
</tr>
<tr>
<td>1 splenectomy</td>
<td>1 adhesional bowel obstruction</td>
</tr>
<tr>
<td>Zero 90-day mortality</td>
<td>1 death (liver failure) 9 months postoperatively</td>
</tr>
</tbody>
</table>

The use of CPAP apparatus was discontinued in 100% (9/9) with remission rates of 72% (18/25) and 91.7% (33/36) for hypertension and dyslipidaemia, respectively. See Table 4 below.

Table 4. Resolution of comorbidities

<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>Number of patients (% of total)</th>
<th>Remission (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>19 (42)</td>
<td>18 (95)</td>
</tr>
<tr>
<td>Obstructive sleep apnoea (sleep clinic diagnosis and use of CPAP* machine)</td>
<td>9 (20)</td>
<td>9 (100)</td>
</tr>
<tr>
<td>Hypertension (BP**&gt;135/85 mmHg) and requiring drug therapy</td>
<td>25 (56)</td>
<td>18 (72)</td>
</tr>
<tr>
<td>Dyslipidaemia (total cholesterol &gt;4 mmol/L and/or LDL cholesterol &gt;2 mmol/L and/or triglycerides &gt;2.2 mmol/L)</td>
<td>36 (80)</td>
<td>33 (92)</td>
</tr>
</tbody>
</table>

*Continuous positive airway pressure.

**Blood pressure.
There were several incidental intraoperative findings. These are detailed in Table 5 below.

Table 5. Incidental intraoperative findings

<table>
<thead>
<tr>
<th>Intraoperative finding</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver cirrhosis</td>
<td>2</td>
</tr>
<tr>
<td>Incomplete regression of pancreatic ring around duodenum</td>
<td>1</td>
</tr>
<tr>
<td>Large duodenal diverticulum</td>
<td>1</td>
</tr>
<tr>
<td>Duodenal lipoma</td>
<td>2</td>
</tr>
<tr>
<td>Meckel’s diverticulum</td>
<td>3</td>
</tr>
<tr>
<td>Carcinoid tumour of ileum with regional lymph node involvement*</td>
<td>1</td>
</tr>
</tbody>
</table>

*Surgery converted to sleeve gastrectomy.

Discussion

Morbid obesity is a chronic, metabolic disease that requires lifelong treatment. The DS has been shown to produce greater excess weight loss than either gastric band or gastric bypass; and higher remission rates of obesity-related comorbidites including type 2 diabetes mellitus (T2DM).14,17

Despite the advantages of DS there has been reluctance amongst bariatric surgeons to adopt the procedure. The main concern surrounds the longer malabsorptive component of the operation, which can result in relatively higher rates of metabolic complications, nutrient deficiencies and life-threatening protein-calorie malnutrition compared to RNYGB. DS is a longer and technically more demanding procedure than other bariatric surgeries and is difficult to undertake laparoscopically.16,18

This article describes the first 60 cases performed by a single surgeon in our unit. Our unit has progressed from performing open DS surgery to the point where we are now beginning to offer laparoscopic DS both as a single-stage primary bariatric surgery and as a 2 stage approach after sleeve gastrectomy in selected patients.

Initial patient selection and an intensive postoperative follow-up protocol is vital to ensure good outcomes from DS surgery.

Biertho et al17,19 showed a complication rate comparable to that of RNYGB in their series of 1000 patients. Indeed they only had 1 death in the 1000 patients (0.1%). Only 5 patients required revisional surgery (0.5%). This large series shows that acceptable safety rates can be achieved with DS surgery.

Biertho et al have also shown that long-term nutritional deficiencies are rare with close long-term follow-up and careful adjustment of nutritional supplementation. They report a series of 810 patients who have undergone DS with a mean follow-up of 103 months. In this study, they showed that the risk of protein malabsorption after DS was comparable to that after distal RNYGB. Around 4% of patients may require rehospitalisation and nutritional support for severe protein deficiency or food intolerance. Severe protein deficiency was detected in 1% of patients in this series17,18.

The obvious statistic in our results is the death from liver failure at 9 months postoperatively. The patient was a 46-year-old female with a preoperative weight of
235 kg and a BMI of 62 kg/m\(^2\). She had pre-existing steatohepatitis and colonic dysmotility as well as a history of depression. Adherence to the recommended dietary intake and multivitamin/mineral supplementation had been difficult for this patient. She presented acutely to hospital with a 2-month history of jaundice and hepatic encephalopathy secondary to liver failure from protein-calorie malnutrition; which is a recognised complication of DS.

Baltasar et al also reported a death from hepatic failure after DS.\(^{19,20}\) One death in our series of 60 patients gives a mortality rate of 1.66%. This is much higher than the 0.1% seen by Biertho et al in their series of 1000 patients. They comment that a reduction in mortality from DS came with a move to laparoscopic surgery from the open procedure.\(^{17,21}\) We hope that our adoption of the laparoscopic technique will yield similar benefits.

Morbidly obese patients show a high prevalence of hepatic steatosis and steatohepatitis.\(^{19,20}\) Questions have been raised about performing the DS in patients with pre-existing liver disease.

Baltasar et al report a series of 470 DS patients in which 10 developed postoperative clinical hepatic impairment. 1 patient died from hepatic failure. Deaths from liver failure have also been reported after other types of bariatric surgery.\(^{19}\)

Keshishian et al found in a series of 697 patients that DS improves hepatic steatosis; there is a mild worsening of liver function in the first 6 months postoperatively but then an improvement over preoperative levels after 1 year. They conclude that DS should be beneficial in reducing the rate of cirrhosis and liver failure from the progression of non-alcoholic steatohepatosis (NASH) in obese patients.\(^{21}\) Kral et al have also shown reversal of severe fibrosis and cirrhosis in obese patients following biliopancreatic diversion (BPD).\(^{22}\)

Two of our patients had Child-Pugh A liver cirrhosis found incidentally at surgery. They also had type 2 diabetes. Both have had good results achieving 100% and 70% weight loss at 2-year follow-up post DS surgery, with normalisation of their liver function tests and their diabetes remain in remission. One of them had a liver biopsy at 2 years post DS, the histology result on which confirmed resolution of steatosis but fibrosis was still present.

DS may also have an increasing role as a two stage procedure in patients who have previously undergone a sleeve gastrectomy. Such patients who have done well with excess weight loss but have not had satisfactory resolution of comorbidities may be candidates for a two-stage DS. In order to be considered for the two-stage procedure patients should have demonstrated good understanding and compliance with healthy eating and regular exercise since the sleeve gastrectomy.

We believe that DS surgery can be safely performed in a unit with facilities to provide intensive follow-up. Rates of operative mortality and nutritional sequelae similar to RNYGB can be achieved. DS does require larger doses of nutritional supplements and careful long-term follow-up to achieve these safety levels.

Potential patients need to be carefully selected for their willingness to attend all follow-up visits and their ability to understand the postoperative nutritional requirements as well as the risks that are associated with non-adherence.
Competing interests: Nil.

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References:


Timely cholecystectomy for acute gallstone disease: an ongoing challenge in a New Zealand provincial centre

Melissa J Welch, Andrew R Moot

Abstract

Aims To review the prior management of patients who underwent cholecystectomy for gallstone disease at a provincial centre over a 1-year period, with a particular focus on potentially preventable morbidity by performing index cholecystectomy (IC).

Methods Retrospective case note review was performed for patients who underwent cholecystectomy at Hawke’s Bay’s hospitals between 1 March 2009 and 1 March 2010.

Results 148 cholecystectomies were performed over the study period. Ninety-one patients (61%) were admitted acutely prior to receiving cholecystectomy. The IC rate was 15%. Seventy-seven patients who were admitted acutely could have been suitable for IC, but were discharged. These 77 patients subsequently had an additional 17 readmissions (72 bed-days), 26 ED presentations and 51 outpatient clinic (OPC) visits prior to receiving their eventual operation. Ten patients (13%) developed a complication or recurrence of their acute gallstone disease whilst awaiting surgery.

Conclusion Hawke’s Bay has a low rate of IC and fails to meet current international standards for timely surgical management of acute gallstone disease. A large proportion of those not operated on during their index admission re-present with further morbidity. There are significant barriers to improving these standards in a provincial centre with limited acute surgical resources.

Early laparoscopic cholecystectomy has been shown to be the preferred treatment for acute presentations of gallstone disease. Traditional fears of increased operative morbidity during acute episodes of gallbladder inflammation have now been allayed, with several meta-analyses demonstrating the safety of early surgery for acute cholecystitis.¹

For patients presenting with simple biliary colic, cholecystectomy performed during the index admission prevents further morbidity due to gallstone-related complications, which may otherwise occur during the waiting period for elective cholecystectomy.²

UK guidelines for the management of acute pancreatitis published in 2004 recommends cholecystectomy for acute gallstone pancreatitis during the index admission or within 2 weeks of discharge.³ Without prompt definitive treatment, there is significant risk of a further episode of acute pancreatitis, which may be life threatening.

Despite the known benefits of early cholecystectomy, it remains an international challenge to achieve this in a timely manner. Lack of resource availability and
insufficient institutional organisation have been cited as major contributors to the published low rates of IC. In New Zealand, significant efforts have been made in recent years to improve the timely surgical management of acute biliary pathology. Following a concentrated clinician driven change in practice, Christchurch Public Hospital improved their IC rate from 15% in 2005 to 78% in 2007.

Provision of timely cholecystectomy for acute gallstone disease remains low in provincial hospitals however, where acute surgical services face significant challenges in resource allocation. This was demonstrated in a recent study from Nelson Hospital, who described an IC rate of only 17%.

Hence the aim of this study was to review the prior management of patients who underwent cholecystectomy in Hawke’s Bay over a 1-year period, with a particular focus on the timing of surgery and the morbidity which may have been prevented by performing cholecystectomy during an index admission.

Methods
A retrospective case note review was performed for all patients who underwent cholecystectomy at Hawke’s Bay’s Hospitals (Hawke’s Bay Hospital Soldiers’ Memorial, and Royston Hospital) between 1 March 2009 and 1 March 2010. These patients were identified via the gallbladder specimens received by the Hawke’s Bay Hospital laboratory over this time period.

Patients who were admitted from ED to a Hawke’s Bay Hospital inpatient ward with acute gallstone-related pathology over the same time period were also identified from the Hawke’s Bay DHB patient management system. These were identified via International Classification of Diseases, 10th revision coding as follows; cholelithiasis (k80x) – including choledocholithiasis with/without cholangitis, cholecystitis (k81x) and acute biliary pancreatitis (k85.1).

Data concerning patients who met inclusion criteria were collected from the Hospital’s Electronic Clinical Application database. This included discharge summaries following inpatient admissions or ED presentations, as well as OPC letters. Basic demographic data and waiting list times were also recorded.

Indication for surgery was recorded based on standard diagnostic criteria for acute gallstone-related diseases. Biliary colic was defined as the presence of upper abdominal pain with radiological evidence of cholelithiasis. Acute cholecystitis was defined as biliary colic in addition to at least one of; temperature >37.5°C, increased white cell (>10x10^9/L) or neutrophil count (>7.5x10^9/L).

Choledocholithiasis was defined as radiological evidence of at least one gallstone within the common bile duct. Cholangitis was defined as the presence of choledocholithiasis, temperature >37.5°C, increased white cell or neutrophil count and jaundice.

Gallstone pancreatitis was defined as radiological evidence of cholelithiasis and at least two out of three of upper abdominal pain, elevated blood pancreatic enzyme levels and radiological evidence of pancreatic inflammation. Where more than one diagnosis existed, cholangitis/gallstone pancreatitis took precedence over acute cholecystitis which itself took precedence over biliary colic/choledocholithiasis.

The index admission was defined as the first admission to an inpatient ward meeting the aforementioned criteria for acute gallstone disease. IC was defined as a cholecystectomy performed during this admission. Acute cholecystectomy was defined as cholecystectomy performed during an unscheduled acute admission (but not necessarily the index admission) for acute gallstone disease.

The primary end points of the study were IC rate and excess morbidity caused by not performing IC in cases where this could have been suitable. Excess morbidity was defined as additional admissions, ED presentations or OPC visits incurred between the index admission and eventual cholecystectomy.

Nominal data is presented as actual numbers. Continuous data is presented as medians (range). The statistical software used was R version 2.15.2 (Vienna, Austria). Kruskall-Wallis test was used to compare ordinal and nominal data. Wilcoxon signed rank test was used to compare groups with
continuous variables. Fisher’s exact test was used for categorical data. $P<0.05$ was considered significant. Missing data was excluded from the analysis.

Ethical approval was not required as this study met the definition of an audit as outlined in the guidelines published by the New Zealand National Ethics Advisory Committee.

**Results**

Over the 1-year study period, 186 cholecystectomies were performed in the Hawke’s Bay at two hospitals. Thirty-eight patients were excluded. Twenty-four underwent cholecystectomy in private for an unknown reason, as they had had no contact with the public hospital. Eight underwent incidental cholecystectomy for cholelithiasis noted at the time of abdominal surgery for a separate pathology. Six patients underwent cholecystectomy for reasons other than gallstone disease—four for acute acalculous cholecystitis and two for unexplained right upper quadrant pain without radiological evidence of cholelithiasis. This left a study size of 148. The median age was 53 (range 18–92) and 78% were female.

126 (85%) operations were performed in the public sector at Hawke’s Bay Hospital. The remaining 22 (15%) operations were performed privately at Royston Hospital.

Patients’ diagnoses at time of surgery are summarised in Figure 1. Sixty-six patients (45%) underwent cholecystectomy for biliary colic, 39 (26%) for acute cholecystitis, 23 (16%) for gallstone pancreatitis, 12 (8.1%) for cholangitis, and eight (5.4%) for choledocholithiasis. Seventeen patients received their operation acutely during an admission. For nine of these patients this was during their index admission and was thus an IC. Five patients receiving acute cholecystectomy were already on the waiting list for elective surgery.

**Figure 1. Diagnosis at time of surgery for all patients undergoing cholecystectomy (n=148)**

![Figure 1](image-url)
Figure 2. Prior management and outcome for patients undergoing cholecystectomy in Hawke's Bay between 01 March 2009 and 01 March 2010

- Patients receiving cholecystectomy $n=148$
  - Ever admitted acutely $n=91$
    - Suitable for IC $n=86$
    - Unsuitable for IC $n=5$
    - IC $n=9$
    - No IC - Discharged $n=77$
      - Waiting list $n=29$
      - OPC $n=41$
      - Other; Private $n=5$
        - GP $n=2$
  - Never admitted acutely $n=57$
    - Elective cholecystectomy (Private=10)
      - ED presentations $n=26$
        - (16 patients)
      - OPC visits $n=51$
        - (38 patients)
      - Re-admission $n=17$
        - (15 patients)
      - Acute cholecystectomy $n=5$
    - Elective cholecystectomy $n=72$
      - (Private=12)
During the same study period 61 patients were admitted with symptomatic gallstone
disease. This included the nine patients who underwent IC. The IC rate was therefore
15%.

Figure 2 summarises the prior management of the 148 patients who underwent
cholecystectomy during the study period. Ninety-one (61%) of these patients were
admitted acutely for symptomatic gallstone disease at some point prior to their
surgery.

Of these patients, nine received IC while a further 77 could have been suitable for
index surgery but were instead discharged. Five patients would have been unsuitable;
three were deemed medically unfit for surgery at that time by either the surgeon or
anaesthetist and two patients declined an acute operation. Discharged patients were
either placed directly on the waiting list (29–38%) or followed up in OPC (41–53%).
Five patients (6.5%) went to the private sector while two (2.6%) were discharged to
their general practitioner.

Of the 77 patients who could have been suitable for IC but were discharged without
an operation, ten (13%) subsequently developed one or more
complications/recurrences (total 13) of their gallstone disease. These are summarised
in Table 1.

Fifteen patients (19%) (total 17 admissions) were readmitted with further
symptomatic gallstone disease. This resulted in an additional 72 inpatient days. Of the
patients who were readmitted, five subsequently underwent acute cholecystectomy.
Within this group of patients, there were also an additional 26 ED visits (16 patients)
and 51 OPC visits (38 patients) prior to their eventual operation.

Table 1. Development of complications following index admission for patients
who could have been suitable for index cholecystectomy

<table>
<thead>
<tr>
<th>Index diagnosis</th>
<th>Complication</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Choledocholithiasis</td>
<td>Recurrent choledocholithiasis post ERCP</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Acute cholecystitis</td>
<td>2 (1 patient)</td>
</tr>
<tr>
<td></td>
<td>Cholangitis</td>
<td>1</td>
</tr>
<tr>
<td>Acute cholecystitis</td>
<td>Recurrent acute cholecystitis</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Cholangitis</td>
<td>1</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>Recurrent cholangitis</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Acute cholecystitis</td>
<td>1</td>
</tr>
<tr>
<td>Gallstone pancreatitis</td>
<td>Recurrent gallstone pancreatitis</td>
<td>3 (2 patients)</td>
</tr>
</tbody>
</table>

Total: 13 (10 patients)

Of the three patients who were medically unsuitable for IC at the time of their index
admission, one subsequently developed recurrent pancreatitis and another developed
recurrent acute cholecystitis (four times). All three patients eventually underwent
elective cholecystectomy.
Overall median length of stay (including the operative admission) was unchanged between the IC and delayed surgery (for those who could have been suitable for IC) groups (5 versus 6 days respectively) as shown in Table 2.

### Table 2. Number of admissions and hospital stay for IC and delayed cholecystectomy groups

<table>
<thead>
<tr>
<th>Outcome</th>
<th>IC n=9</th>
<th>Delayed surgery n=59*</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median number of admissions</td>
<td>1</td>
<td>1 (1–4)</td>
<td>0.10</td>
</tr>
<tr>
<td>Median overall length of stay</td>
<td>5 (3–13)</td>
<td>6 (3–33)</td>
<td>0.56</td>
</tr>
</tbody>
</table>

IC=index cholecystectomy; *For those in whom IC could have been suitable, excluding patients with missing data.

Of the patients who underwent IC, seven (78%) were completed laparoscopically, compared with 86% in the delayed surgery group. One patient required a conversion to open, compared with three in the delayed surgery group. There were four (6.8%) bile leaks in the delayed surgery group and none in the IC group (overall bile leak rate of 2.7%). No major bile duct injuries were sustained.

### Table 3. Operative comparisons between IC and delayed cholecystectomy groups

<table>
<thead>
<tr>
<th>Outcome</th>
<th>IC n=9</th>
<th>Delayed surgery n=59*</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laparoscopic</td>
<td>7 (78%)</td>
<td>51 (86%)</td>
<td>0.51</td>
</tr>
<tr>
<td>Conversion rate</td>
<td>1 (11%)</td>
<td>3 (5%)</td>
<td>0.41</td>
</tr>
<tr>
<td>Bile leak</td>
<td>0 (0%)</td>
<td>4 (6.8%)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

IC=index cholecystectomy; *For those in whom IC could have been suitable, excluding patients with missing data.

The median waiting list time for the 112 patients who underwent public elective cholecystectomy was 129 days (3–1552). Waiting times by diagnosis are shown in Table 4. Twenty-three patients underwent cholecystectomy for gallstone pancreatitis. Of these patients, 22 were admitted acutely, with five cholecystectomies performed acutely, and a further two performed within 2 weeks of discharge.

### Table 4. Median waiting time to surgery by diagnosis for public elective cholecystectomies (n=112)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Median waiting time to surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biliary colic</td>
<td>138 (7-1552)</td>
</tr>
<tr>
<td>Choledocholithiasis</td>
<td>174 (16-459)</td>
</tr>
<tr>
<td>Acute cholecystitis</td>
<td>136 (10-805)</td>
</tr>
<tr>
<td>Cholangitis</td>
<td>121 (8-465)</td>
</tr>
<tr>
<td>Gallstone pancreatitis</td>
<td>61 (3-1231)</td>
</tr>
<tr>
<td><strong>Overall:</strong></td>
<td><strong>129 (3–1552)</strong></td>
</tr>
</tbody>
</table>
Discussion

Good quality evidence suggests that the definitive management of patients with acute gallstone disease should involve early cholecystectomy. Ideally this should be performed during the index admission in order to prevent morbidity associated with further gallstone-related complications\(^1\).

Hawke’s Bay Hospital’s IC rate is 15%. This compares poorly with figures published by tertiary centres around New Zealand, such as Christchurch Public Hospital (78\%\(^6\)), Middlemore Hospital (66\%\(^7\)) and Auckland Public Hospital (63\% - gallstone pancreatitis only\(^10\)). Of concern is the low rate of surgery performed within two weeks following presentation with acute gallstone pancreatitis (32\%). The median waiting list time for those not operated on acutely was 61 days – well outside the time frame recommended by the UK guidelines\(^3\).

Forty-three patients (29\%) underwent cholecystectomy for pathology relating to the presence of common duct stones (choledocholithiasis, cholangitis or gallstone pancreatitis) – a group of patients with the most potential to become critically unwell. This is a high figure, reflecting a regional population receiving delayed operative management.

Patients with simple biliary colic typically wait many months for elective cholecystectomy. In our study, the median waiting time for these patients was 138 days. This was relatively similar to waiting list times for patients with acute cholecystitis, cholangitis and choledocholithiasis.

Patients with more severe complications of gallstone disease should be operated on with priority over biliary colic and our study shows this prioritization needs review. Improvements in communication should be made with waiting list booking staff regarding the comparative urgency of particular diagnoses.

Studies have shown that rates of admission with acute gallstone pathology increase in accordance with length of time spent on the waiting list\(^11\). Overall hospital stay is significantly shorter in groups of patients receiving early treatment\(^12\).

In our study however, there was no significant difference in total length of stay between the index and delayed surgery groups. This was unexpected given the number of readmissions observed within the delayed surgery group. It therefore demonstrates the inefficient way in which patients receiving acute surgery were managed - including time spent obtaining radiological investigations and waiting on the acute operating list prior to theatre.

In our study, 86 patients admitted acutely prior to their eventual operation could have potentially undergone IC. Only nine patients actually did. The re-presentation rate with interval complications or recurrence of symptoms of those treated conservatively was unacceptably high. During the time between first admission and eventual surgery, there were an additional 17 admissions (total of 72 inpatient days), 26 ED presentations and 51 OPC visits, all of which could have been potentially avoided.

While a formal cost–benefit analysis was not performed, financial data supplied by the Hawke’s Bay DHB enables the cost of these additional presentations to the public
sector to be estimated at $59,520 (assuming one overnight surgical bed = $450, one ED presentation = $334 and one OPC visit = $250). It must be mentioned however, that reducing one patient bed-day does not necessarily save $450, as the ward and staffing infrastructure are already in place. Reducing unnecessary stays over time will however translate into financial savings long term.

The cost of readmissions is only the tip of the iceberg in terms of the financial cost of delaying cholecystectomy. The cost of procedures, investigations, pharmaceuticals, admissions to other hospitals and presentations to GP’s are an additional financial burden to be considered. Furthermore, health system costs fail to take into account the social cost of patient suffering and lost income. Overall, it would seem more cost effective to perform early cholecystectomy.

It is clear however that significant barriers exist to performing surgery in accordance with published guideline recommendations. A survey of UK surgeons identified surgeon reluctance, delays to confirming diagnosis and lack of acute surgical facilities as key barriers to early cholecystectomy. When compared with tertiary centres in New Zealand, different constraints exist at provincial hospitals contributing to low rates of IC. Identification and recognition of these barriers is the first step towards improving timely operative management.

Like many provincial centres, Hawke’s Bay Hospital has difficulties with access to acute surgical facilities. During the study period, Hawke’s Bay had one acute surgical theatre, shared between all surgical specialties. Cholecystectomy should ideally be performed during the day due to the potential requirement for intra-operative cholangiogram, further constraining available theatre time. Granted, time taken up by acute cholecystectomy is time taken away from other specialties, with the relevant incurred costs to those departments. Since the study period however, Hawke’s Bay Hospital has opened a further theatre specifically for orthopaedic acute cases. It is anticipated that this additional resource will increase theatre access for acute cholecystectomy cases.

Hawke’s Bay Hospital has accreditation to supervise SET trainees up to year three only. A consultant surgeon must therefore be available to supervise registrars performing acute cholecystectomies. Due to the low number of acute admissions compared with tertiary centres, the on call consultant is routinely otherwise occupied with their own elective list or clinic, thereby limiting their availability for acutes.

With the Ministry of Health’s focus on provision of elective surgical procedures, there is reluctance by hospitals to cancel elective patients in order to manage the acute load. With this study demonstrating the financial consequence of delaying surgery for acute gallstone disease it would seem prudent that elective surgeries may need to be postponed from time to time. With just 61 patients admitted with acute gallstone pathology over the one year study period, it would only require a few additional cholecystectomy cases per week to dramatically improve the IC rate.

Hawke’s Bay Hospital employs one gastroenterologist able to perform ERCP, with only one list per week. Patients requiring common bile duct clearance are therefore routinely discharged to return for elective ERCP at a later date. By the time this occurs, the opportunity for early cholecystectomy may have elapsed. A pragmatic
solution would be to operate in the first instance and manage ductal stones postoperatively when ERCP is more readily accessible.

Our paper reports an IC rate similar to that published by Nelson, another provincial hospital. It is likely that this is the case throughout many provincial centres in New Zealand. Sakowska et al in Nelson proclaim that taking all these barriers to consideration could mean that provision of an acute cholecystectomy service may be unrealistic in a provincial centre and that the focus should be shifted to providing an operation on an elective list within a minimum safe timeframe.

A definitive date should be provided on discharge from hospital for those patients who would be suitable for IC. In our study 41 (53%) patients who could have received IC were followed up in outpatient clinic, compared with 29 (38%) who were booked onto the waiting list directly. In patients without suspicion of choledocholithiasis and who do not require additional investigations, routine outpatient follow-up prior to booking patients onto the waiting list would seem unnecessary and time delaying.

Patients undergoing cholecystectomy in private were included in this study to demonstrate the not insignificant number of patients accessing the private service in order to obtain timely surgery following an acute admission (19%).

In conclusion, provincial centres face significant challenges in improving timely operative management of acute gallstone disease with respect to current international standards. Hawke’s Bay in particular has a very low rate of IC, with a considerable proportion of those discharged without an acute operation re-presenting to health services with the associated additional morbidity and financial expense.

We have suggested a number of pragmatic solutions that may improve the rates of acute surgery for this disease whilst working within the constraints of a provincial surgical service. It will be important to implement these strategies and reassess progress. As found by Christchurch, a coordinated clinician driven approach will be crucial to this process.

**Competing interests:** Nil.

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**Acknowledgement:** Thank you to Dr Ross Scott-Weekly (Anaesthetic Fellow, Department of Anaesthetics, Dunedin Hospital) for his statistical work on this paper.

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3. Working Party of the British Society of Gastroenterology; Association of Surgeons of Great Britain and Ireland; Pancreatic Society of Great Britain and Ireland; Association of Upper GI


When medical reports become expert medical evidence: judgments of the Court

Koenraad Kuiper

Abstract

Aim This paper analyses what happens when medical reports are reframed to become expert evidence in the New Zealand District Court. The aim is to understand what judges do with, and want from, medical reports.

Method Fifty medical reports were analysed for their properties as instances of a specialised genre. Twenty judgments of the District Court were then analysed to see how medical reports are edited and reframed in such judgments. The analytic techniques used were those of genre analysis.

Results When they are edited and become expert evidence in a court, medical reports written for one purpose are used for a different purpose. In a court, medical reports are assessed for the way they bear on the matter at issue and for their quality as expert evidence.

Conclusions The judgments of the medical reports made by judges can provide guidelines which may assist medical professionals in writing medical reports.

When medical professionals write reports, the intended audiences are other medical professionals. From a medical professional’s perspective such reports are the appropriate way to communicate in writing about a patient since medical professionals belong to a professional discourse community. Such reports constitute a specialised genre with its own rules.

Genres exist at the intersection of two sets of factors. The socio-cultural factors include the cultural location, the prototypical producers and receivers and purpose, of the genre.

The second set of factors is the textual properties of the genre. Genres always have a more or less fixed set of conventions relating to their form, their language. Such formal properties, if they are recurrent and characteristic, are the text type features of the genre. Any linguistic property, such as the intonation of a racing commentary, may be characteristic of a genre but there are also non-linguistic type features such as the formatting requirements of formal business letters. It may be the presence or absence of such a property, but it can also be its probability of occurrence, which is significant in identifying a feature of form as characteristic of the genre. Once established, many aspects of the genre, both as a social mode of action and as a text type, come to be conventional.

Medical reports as one specialised genre can be employed as expert evidence and then are quoted in part in judgments of the Court. Judgments of the Court are a different genre. Judges, who are responsible for the production of judgments of the Court, make judgments in favour either of the appellant or the respondent on the basis of the
weight they assign to the medical evidence with which they are presented. They may also comment on the quality of the original reports containing that evidence. Thus a close reading of legal judgments can provide guidance to the writers of medical reports.

Method
Fifty medical reports were analysed for their individual properties in order to determine some of the generic properties of the report genre. Additionally, 20 legal judgments in which edited medical reports were quoted as expert evidence were analysed to see how the editing and reframing process had altered the form and context of the reports. The context is that of medical reports which are framed in appeals to the District Court of New Zealand against determinations of the Accident Compensation Commission. The resulting judgments can be found at http://www.nzlii.org/nz/cases/NZACC/

Results
Reports are used as expert evidence in the determinations of the District Court and any such report may become part of the expert evidence presented to the Court.

Reports are written under at least four different circumstances. The first is when a patient presents with an accident-related condition to a General Practitioner (or A and E) who may then refer the patient to a specialist and/or to diagnostic services. The resulting reports are sent to the patient’s GP. The second circumstance arises when the ACC is approached to cover a patient for an accident-related condition and, either then or later, determines not to provide cover or to withdraw cover. This can lead to a request for a review by ACC. And, third, if mediation then takes place, further medical reports can be produced by both the ACC and the patient. At this point the reports are no longer communications between medical professionals but are commissioned by and written for the parties in dispute.

Should the matter not be resolved by mediation, an application can be made for a hearing by the Accident Compensation Appeal Authority. Hearings are held under the aegis of the New Zealand District Court. For the purpose of such a hearing the parties may present further medical evidence in the form of additional reports to the Court. The Court may also request further medical evidence. This could be seen as a fourth circumstance. Hearings result in judgments of the District Court. For a summary of these processes see http://www.acc.co.nz/making-a-claim/what-if-i-have-problems-with-a-claim/index.htm

Judgments have a standard legal form which begins with a standard header. The judgment is then divided into paragraphs, each distinguished by numbers in square brackets. Sequencing is either in terms of sub-headings or the sub-headings are given in the first paragraph devoted to the relevant section. Sections are:

[1] The issue—In this section the judgment lays out the issue for the Court to determine.

[2] The relevant background—in this section the judgment lays out in chronological order the events which are relevant to the appeal. For example, the following is the first part of such a chronology:
Meyrick v Accident Compensation Corporation [2010] NZACC 86 at [5] ‘The background facts relevant to the issue in this appeal may be stated as follows:

- On 29 November 1996 the appellant, then aged 36 and employed as a builder, fell approximately 3 metres from a roof onto a concrete surface, landing on his head.
- He was briefly unconscious and taken immediately to Wellington Hospital.
- The principal injury suffered by the appellant was an occipital skull fracture extending towards vertex.
- He also suffered some small contusions and the only other injury identified was a fracture of the left clavicle.
- A CT scan of his cervical spine identified it as being satisfactory.
- After several months of rehabilitation, the appellant was able to reengage in his pre-injury employment as a builder, eventually becoming a self-employed builder in his own business.’

Sometimes this section contains more extensive documenting of all the events up until the appeal to the District Court. In other reports, only the initial steps in the process are included under this section.

[3] The medical evidence—In this section the relevant medical evidence, in chronological order of it being obtained, is either summarised and/or edited excerpts from the medical reports are recorded.

[4] Counsels’ submissions—Here the judgment summarises the submissions made to the court on behalf of the contending parties by their legal counsels.

[5] The decision—The form this section takes varies among the presiding judges but always involves weighing up the relevant law relating to the situation, the nature of the medical evidence and its relationship to the relevant law and the submissions of counsel. On the basis of this weighing up, the judge finds for or against the appellant.

The report concludes with a statement relating to costs.

The judgment is then dated and signed.

The language in which a judgment is phrased is formal but not as technical as that of medical reports. Many of the terms and phrases are in common use but have developed legal denotations and a history in legal precedent. There are also a number of formulae in evidence which can be shown from their regular re-appearance. A phrase such as on the balance of probabilities is used frequently and while it has a clear meaning in standard English, it also has a track record in specialised legal use as a legal test. Other examples are causative nexus, take him/her as (s)he is found. Many of the formulae of legal judgments consist of the main clause of a complex sentence with a noun clause object where the noun clause object is a slot to be filled. Examples are:

I accept that, I find it to be the case that, it is accepted by the respondent that, it must be established on the balance of probabilities that, the fact of the matter is that, the
medical evidence in this case is that, counsel submitted that, the Court can take judicial notice of that the fact that.

There is an emphasis on clarity and lack of ambiguity which leads to a high degree of explicitness.

So how is expert medical evidence presented in such judgments? It is done by means of selective editing without reference to the original report writers.

First, all evidence of a report’s original communicative function is removed. This includes the headers of the original letters, the salutations such as Dear Dr, acknowledgement of patient formulae such as I reviewed [patient], thank you formulae such as Thank you for asking me to see [patient], hand over formulae such as I am happy to return him/her to your care, sign off formulae such as Kind regards.

Second, in the judgment only evidence which is regarded as relevant to the matter in dispute is presented. Reports presented by an appellant or respondent are not represented in the judgment if they are not considered relevant. The judgment will refer to them and indicate why they are not considered relevant.

Third, the extracts from the relevant medical reports are reframed in a chronological narrative as one of a sequence of items of medical evidence.

Such editorial processes convert a medical report into expert medical evidence for the purpose of the Court which is to ‘identify, based on the medical evidence, precisely what the nature and extent of the covered injury may be.’ Clegg v Accident Compensation Corporation [2010] NZACC 209 at [6]

In the process of editing medical reports judges provide, implicitly or explicitly, desiderata for the purpose of converting medical reports to expert evidence.

1. The Court values clarity and precision.

   For example in Simpson v Accident Compensation Corporation [2009] NZACC 206 at [38] the Court notes that

   ‘Mallon J has reiterated to ACC the importance of clarity of evidence and having a sufficient basis to suspend a claimant’s entitlements.’

2. The Court values expert evidence that is both necessary and sufficient for the conclusion reached by the medical professional.

   For example in Wyatt v Accident Compensation Corporation [2006] NZACC 9 at [65] the Court notes that

   ‘The authors provide no detail or comment on the specific limitations caused by the appellant’s disability. The authors simply state, with no reasoning or explanation offered, that the appellant cannot work for more than 10 hours per week. With respect, such a statement requires more than bare assertion.’

3. Since it is for the Court to determine the matter at issue, it does not wish medical experts to stray outside the limits of their expertise.

   For example in Johnston v Accident Compensation Corporation [2009] NZACC 46 at [28] the Court notes that
‘In Mr X’s report of the 16 January 2008, he goes on to say: “My feeling in terms of fairness, is that this man should be recognised as having an injury related process and must be covered for his surgery.” Of course Mr X is an expert on medical matters, as distinct from legal issues and whether they can take account of fairness.’

4. The Court does not take seriously expert evidence from the same person if that person changes their mind without good reason.

For example in Meyrick v Accident Compensation Corporation [2010]
NZACC 86

at [21] the Court notes that

‘The appellant relies on the advice of Mr. X, which has been set out above. His first comment is that he can neither confirm nor deny causation. He appears not to have been aware of the fact that no part of the appellant's spine was injured in the 1995 fall, and indeed he says that it was his opinion that if all the degeneration in the spine had been caused by the fall, spinal symptoms at the time of that injury would have been overwhelming.

at [22] That is simply not the case on the facts either medical or general, as we know that the appellant recovered from that injury and was able to resume his employment.

at [23] Mr X in his next breath, as it were, then says that it was his opinion that it was more likely that the condition was due to non-injury related factors, but then somehow, as he says, to give the patient and his injury the reason of the doubt, he settles on a 50:50 figure.

at [24] Frankly, I find the reasoning of Mr X quite confusing and from a medico-legal perspective it is entirely unconvincing as establishing causation.’

5. As noted earlier, the Court is concerned that what it gets by way of expert evidence contains only evidence that is relevant to the matter in dispute.

For example in Singh v Accident Compensation Corporation [2010] NZACC 38

at [18] the Court notes that

‘I must note that in addition to the medical reports that were obtained over the years on the appellant's medical condition, there was a psychological report provided by a Dr Y, in February 2005, who was looking at the appellant's situation from a psychological perspective and who gave as her opinion that there was a significant component of malingering in the appellant's presentation.

at [19] The Court must consider that comment in the light of the many reports which have identified that the appellant does have a chronic pain syndrome, and I find that whilst there might be psycho-social features in the total clinical picture, as identified by Dr Y, nevertheless the medical evidence from the medical specialists is to the effect that the chronic pain is real.’
6. The Court does not value highly expert opinion which is not backed up by detailed factual evidence.

For example in Wilson v Accident Compensation Corporation [2009] NZACC 189 at [7] the Court notes that

‘The opinion was given without further explaining of the medical reasons to ascribe a degenerative cause rather than a traumatic injury cause.’

7. The Court expects expert evidence to assess the relevant facts comprehensively.

For example in Wilson v Accident Compensation Corporation [2009] NZACC 189 at [23] the Court notes that

‘The ultrasound in July 2007 suggested a partial thickness tear. That did not show up in the MRI in February 2008. The difference was attributed to possible healing over that period, or to obscuring in the MRI of a possible tear because of the position of the shoulder at the time of the scan. The opinion of Dr. X was restricted to the conclusions that could be drawn from radiology alone, without reference to the history and symptoms observed by treating practitioners.’

Discussion

The above examples illustrate the Court’s concerns that medical reports have all of the qualities listed in [1]–[7]. This is because the Court’s duties in relation to the appellant and the respondent are different from those of a medical professional in their duty of care for a patient.

The Court’s views of the quality of the evidence provided by medical experts are in the public domain, as is the experts’ identity. (In the quotations above X and Y replace the full names of medical professionals which are in the original judgments.) It is a feature of the editorial processes conducted by the Court that, when appellants proceed to appeal, those who provide medical evidence are moved from writing privately to having extracts from their reports appear in the public domain.

Views of the Court as to the quality of medical reports also appear in the public domain as comments in judgments of the Court. If one looks at the desiderata of the Court for medical reports and at their use as expert evidence, these desiderata which are apposite for the Court’s purposes may also be valuable when the reports are written for medical purposes. For example, taking into account the relevant medical evidence and only the relevant medical evidence should probably be a hallmark of medical reports as such.

Given that the comments of the Court cited above are a selection taken from only twenty judgments, it seems that more attention might be paid to the quality of medical report writing, not only by those writing directly for appeals. ACC and HDC both run courses on report writing which give clear instruction on what is required.

As to the sometimes critical comments on the reporting of medical professionals by judges, these are matched by the potentially critical review of judgments and judges (also named) made by higher courts. Criticism is the price one pays not only for the growth of knowledge but also for living in a democracy under the rule of law.
Competing interests: Nil.

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References:
Measurement of kidney cadmium in embalmed New Zealand cadavers

Ian C Shaw, Emma Spencer, Tessa Lambert, Meike Holzenkaempfer, Sally Gaw, Mark Stringer

Abstract

Cadmium (Cd) is a toxic (carcinogenic) metal found in food because of its presence in the environment. New Zealand has elevated levels of Cd in soil due to the long term use of Cd-containing phosphate fertilisers. It is therefore likely that New Zealanders have a high Cd body burden which might have health implications. The aims of this study were to determine whether Cd levels in kidney cortex samples are affected by the embalming process and whether kidney cortex samples from embalmed cadavers (e.g. those used for medical student teaching purposes) can be used as a reliable indicator of Cd body burden. Kidney cortex samples from cadavers preserved with different embalming fluids were analysed for Cd by inductively coupled plasma mass spectrometry (ICP-MS). A perfused pig kidney model was used to investigate the effects of embalming on kidney cortex Cd levels. We report considerable variability in Cd levels in kidney cortex samples according to the embalming fluid used; this suggests that the embalming fluid influences tissue Cd concentrations. All pig kidney model perfusions resulted in leaching of Cd from the kidney cortex. We conclude that analysis of Cd in embalmed tissues does not give a reliable indication of in-life Cd levels.

Cadmium (Cd) levels are high in the New Zealand diet because of the volcanic origins of the country (Cd is known to be dispersed by volcanic activity¹) and the extensive use of Cd-containing fertilisers²; this has resulted in elevated concentrations of Cd in agricultural soils.

There have been no detailed studies of Cd levels in the kidneys of New Zealanders despite the evidence that dietary intakes of Cd are high³ and Cd is accumulated in the kidney¹. Since Cd is nephrotoxic⁴ and a non-genotoxic carcinogen⁵ an assessment of body burden, using kidney Cd levels as an indicator, might be useful in studying the magnitude of potential effects of exposure to Cd.

For these reasons we studied Cd levels in the kidneys of New Zealand cadavers. Kidney samples from embalmed cadavers undergoing dissection by medical students were used because their donors had already agreed to the use of their tissues for medical research.

Embalming processes have been shown to alter levels of organic compounds, including drugs, in tissues.⁶ Our studies investigated whether these effects also apply to Cd.

We investigated Cd levels in embalmed New Zealand cadaver kidney samples and then extended our studies in an embalmed pig kidney model system to explore the possibility that the embalming fluids might leach Cd from the kidney tissues. These
findings are important because they indicate the utility of measuring Cd (and perhaps other heavy metal) concentrations in embalmed tissues as a reflection of the \textit{in vivo} situation.

**Materials and methods**

**Chemicals**—All chemicals were of general laboratory reagent grade except nitric acid (Tracepur®, Merck KGaA, Darmstadt, Germany), water (Milli-Q (18.2 MΩ), Millipore, Billerica, USA; Ultrapure Organex cartridge) and a mixed metal analytical standard for the ICP-MS analyses (Inorganic Ventures, Christianburg, USA).

Dodge® embalming fluids were supplied by Regal Manufacturing Ltd, Wellington, New Zealand.

**Human cadaver samples**—Cadavers (n=19; 9 male, 10 female) used for student dissection originated from people who had lived in New Zealand for many years and were almost all of New Zealand European origin (see Cornwall et al (2012)\(^7\) which included the cadavers used in the present study). The bodies had been embalmed with Crosado mix\(^8\) (n=11; 22 kidney samples analysed), Dodge Anatomical® fluid (n=3; 4 samples analysed), or Dodge Emory® fluid (n=5; 9 samples analysed) and stored for at least 6 months at a cool ambient temperature prior to dissection.

The age of death of the cadavers was 81±10 years and none had died of kidney-related causes. All tissue samples were from the upper poles of the kidneys and stored in airtight containers at 4°C prior to analysis. The renal cortex was dissected out and subjected to Cd analysis. Samples from both kidneys from 16 of the cadavers were included in the study; only one kidney sample from each of the remaining 3 cadavers was included (i.e. total number of kidney samples analysed=35) because of difficulties obtaining sufficient cortex for analysis from these kidney sample.

All cadavers had been donated to the Department of Anatomy, Otago School of Medical Sciences, for teaching and research in accordance with the New Zealand Human Tissue Act 2008. Approval for use of the kidney tissues in this research project (conducted in University of Canterbury laboratories) was granted by the University of Canterbury Human Ethics Committee (Approval Reference HEC 2010/70/LR).

**Pig kidney model embalming system**—Fresh pig kidneys were purchased from a local butcher (this means that we do not know whether the kidneys originated from the same or different animals, but this is not thought to be a significant issue in the current study). They were transported to the laboratory on ice and stored for 24 h at 4°C prior to embalming. Cannulae (2 mm internal diameter rigid polythene tubing) were inserted into the renal artery and renal vein of individual pig kidneys (n=7) and secured with nylon thread.

The kidneys were perfused for 1 min via the renal artery cannula (using a 20 mL plastic syringe) with approximately 50 mL Crosado Mix (n=2), Dodge Anatomical® (n=2) or Dodge Emory® (n=1) embalming fluid or isotonic saline (0.5% w/v aqueous NaCl; n=2). Perfusates were collected from the renal vein cannula and stored at 4°C prior to Cd analysis.
Cannulae were removed from the kidneys following the perfusion process and the kidneys were stored at 4°C prior to the cortex being dissected out and subjected to Cd analysis.

**Cadmium analysis**—Pig kidney cortex samples (1 g) taken from the apex of the kidney (i.e. approximately the same sampling position as the human kidney samples used in this study) were homogenised in 1 mL water in 2 mL microtubes (Sarstedt & Co, Nümbrecht, Germany) using a Bead Rupture 24 homogeniser (Omni International, Kennesaw, USA).

Human kidney cortex samples (accurately weighed, approx 3 g) were homogenised in 5 mL water in nitric acid washed (overnight) 50 mL polycarbonate centrifuge tubes (ThermoFisher Scientific, Scoresby, Australia) in an Ultra Turrax (IKA Laboratory Technology, Selangar, Malaysia). The homogenates were digested with 5 mL of concentrated nitric acid at 85°C for 60 min from commencement of reflux. One mL aliquots of the pig kidney perfusates were digested with 5 mL 50% nitric acid (v/v (aq.)). The acid extracts were cooled (overnight), made up to 20 mL with water and diluted fivefold with 2% v/v (aq.) nitric acid. The diluted acid digests were analysed for Cd by inductively coupled plasma mass spectrometry (ICP-MS) equipped with a collision cell (He mode) (Agilent 7500cx, Agilent Technologies, Tokyo, Japan).

103Rh was used as the internal standard. A procedural blank was digested with each batch of samples and Cd concentrations were below the detection limits. Samples were analysed in triplicate. The detection limits were 10 ng/g for kidney tissue and 10 ng/mL for the pig kidney perfusates. The recovery of Cd from a certified reference material (Bovine Liver, reference 1577c; National Institute of Standards and Technology, USA) was 82%.

**Results & Discussion**

Cd levels in human cadaver kidney cortex samples from embalmed cadavers varied considerably according to the type of embalming fluid used to preserve the cadaver (Table 1), thus suggesting that the embalming process changes Cd levels in the tissues. In addition, Cd levels in left and right kidneys from the same cadaver also varied (Fig 1); for this reason, kidney Cd levels were used as individual data points (rather than as kidney pairs) in our assessment of the effects of embalming.

**Table 1. Cadmium (Cd) concentration in human kidney cortex from embalmed cadavers**

<table>
<thead>
<tr>
<th>Embalming Fluid</th>
<th>Sample Size</th>
<th>Kidney cortex [Cd] ng/g tissue (wet weight) Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crosado Mix</td>
<td>22</td>
<td>3212 ± 5302*</td>
</tr>
<tr>
<td>Dodge Anatomical®</td>
<td>4</td>
<td>256 ± 92</td>
</tr>
<tr>
<td>Dodge Emory®</td>
<td>9</td>
<td>419 ± 155</td>
</tr>
</tbody>
</table>

*The variability of these results was large because two samples had very high Cd levels. If these data are removed mean ± SD = 1610 ± 1159.
The Cd levels in the cadavers’ kidneys were very variable (range=84–19,505 ng/g wet weight; mean (n=37)=2,044 ng/g wet weight), but Cd levels in the pairs of kidneys from individual cadavers correlated well; e.g., the two highest levels were samples 429L=18,965 ng/g and 429R=19,504 ng/g and the lowest levels were 423L=84 ng/g and 423R=182 ng/g.

The between cadaver variability might reflect natural variations in Cd levels in kidneys due to individual variation in exposure to Cd, or it might relate to variability in the leaching effects of embalming fluids on tissue Cd. From this study it is not possible to conclude which is the case. However, comparison of our findings with previous post mortem human studies in Sweden\(^9,10\), Poland\(^11\), the UK\(^12\), Canada\(^13\) and Greenland\(^14\) (Table 2) suggest that the Cd levels we found were low except for the two highest Cd concentration samples which had Cd levels of the same order as those found in the two Swedish studies (Figure 1).

These findings might indicate that exposure to Cd in New Zealand is low or that the embalming process leaches Cd from the kidney.
Table 2. Kidney cadmium (Cd) concentrations from published international studies

<table>
<thead>
<tr>
<th>Country</th>
<th>Mean kidney cortex [Cd] ng g tissue (wet weight)</th>
<th>Sample number</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweden</td>
<td>18,400</td>
<td>20</td>
<td>9</td>
</tr>
<tr>
<td>Sweden</td>
<td>15,000*</td>
<td>109</td>
<td>10</td>
</tr>
<tr>
<td>Poland</td>
<td>47,500</td>
<td>64</td>
<td>11</td>
</tr>
<tr>
<td>UK</td>
<td>15,300</td>
<td>933</td>
<td>12</td>
</tr>
<tr>
<td>Canada</td>
<td>15,120†</td>
<td>11</td>
<td>13</td>
</tr>
<tr>
<td>Greenland</td>
<td>15,971</td>
<td>91</td>
<td>14</td>
</tr>
</tbody>
</table>

*Live human whole kidney biopsy study
†Post mortem whole kidney study

In order to explore the possibility that embalming fluids leach Cd from kidney tissues, a pig kidney embalming model system was used. Studies in this model system showed that the embalming process resulted in loss of Cd from the kidney cortex with its subsequent appearance in the embalming fluid perfusate (Table 3).

Table 3. Cadmium (Cd) concentration in pig kidney cortex and perfusate from an embalmed pig kidney model system

<table>
<thead>
<tr>
<th>Perfusate</th>
<th>Kidney cortex [Cd] ng g tissue (wet weight) Analysis mean ± SD (n=3)</th>
<th>Perfusate [Cd] ng mL Analysis mean ± SD (n=3)</th>
<th>Approx. <em>%Loss</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>Control† 1</td>
<td>338 ± 43</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Control† 2</td>
<td>398 ± 29</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Saline 1</td>
<td>295 ± 63</td>
<td>43 ± 2</td>
<td>23</td>
</tr>
<tr>
<td>Saline 2</td>
<td>269 ± 71</td>
<td>63 ± 2</td>
<td></td>
</tr>
<tr>
<td>Crosado Mix 1</td>
<td>277 ± 7</td>
<td>65 ± 1</td>
<td>38</td>
</tr>
<tr>
<td>Crosado Mix 2</td>
<td>179 ± 7</td>
<td>128 ± 5</td>
<td></td>
</tr>
<tr>
<td>Dodge Anatomical® 1</td>
<td>155 ± 14</td>
<td>87 ± 4</td>
<td>56.5</td>
</tr>
<tr>
<td>Dodge Anatomical® 2</td>
<td>166 ± 34</td>
<td>81 ± 2</td>
<td></td>
</tr>
<tr>
<td>Dodge Emory® ††</td>
<td>299 ± 30</td>
<td>281 ± 38</td>
<td>19</td>
</tr>
</tbody>
</table>

*Calculated from kidney cortex [Cd] data.
†No perfusate used
††Only one Dodge Emory® kidney was used as the second ruptured during perfusion.
Even perfusion with saline perhaps resulted in some loss of Cd from the kidney cortex. Dodge Anatomical® resulted in the greatest consistent loss of Cd (approx. 56.5%; Table 3). The Cd leaching results for the Crosado mix perfusion were very variable while Dodge Emory® appears to have little effect (Table 3). The differences in leaching by different embalming fluids are likely to reflect the chemical composition of the fluids and their ability to remove Cd from tissue sequestered forms. It is clear from our results that embalming fluids can remove Cd from kidney tissues.

The chemical mechanism of Cd leaching from tissues probably involves displacement of the metal from its tissue binding site. Cd is known to bind to the metal carrier protein, metallothionein\(^1^5\) and the tripeptide, glutathione\(^1^6\) via the sulphydryl groups of their cysteine residues. It is possible that other metals in the embalming fluids either displace Cd from its sulphydryl binding sites or that the embalming fluid causes a metallothionein conformational change which disrupts the metal binding cleft thus releasing Cd.

Unfortunately, for commercial reasons, we were unable to obtain the formulae of the embalming fluids and so cannot explore this hypothesis further. However, it is known that embalming fluids can contain metal chelating agents such as EDTA which act as anticoagulants\(^1^7\) and EDTA would be expected to bind strongly to Cd\(^{2+}\) in a manner analogous to its strong affinity for Ca\(^{2+}\). Saline is very unlikely to change the conformation of metallothionein or disrupt Cd’s binding via cysteine sulphydryls; however, Cd forms strong complexes with Cl\(^-\) and this process has been shown to extract Cd from other matrices including soil\(^1^8\) – this might explain the possible Cd-leaching effect when pig kidney was perfused with saline.

It is clear from these findings that tissues from embalmed cadavers cannot be used to reliably measure Cd levels in kidney because the embalming process reduces tissue Cd levels. This conclusion can perhaps be extended to other heavy metals (e.g. mercury) that are sequestered in tissues via metallothionein or other sulphydryl-based metal binding peptides.

Despite our concerns about the reliability of Cd measurements in embalmed tissues, our results show that all except one of 19 (approx. 5%) cadavers had kidney cortex Cd levels (mean=1,062 ng/g wet weight) approx. 94% lower than levels seen in other countries (see data above; mean=18,553 ng/g wet weight).

It is impossible to know what proportion of Cd would leach from kidneys in stored embalmed cadavers and so it is equally impossible to conclude whether these cadavers had ‘normal’ levels of Cd for their age based on world data prior to embalming. However, one cadaver had significantly higher levels (mean of left and right kidney cortex samples=19,235 ng/g wet weight); therefore, taking account of leaching, this cadaver would likely have had Cd kidney cortex concentrations very considerably higher than ‘normal’ world levels. Therefore, our data point to only a small proportion (a single cadaver, i.e. 5%, in our study) of kidney cortex Cd levels in New Zealanders being significantly higher than ‘normal’ world levels. Since this is appears to be a sporadic case it is more likely to reflect occupation or smoking habits (cigarette smoke is an important source of Cd\(^1^0\)) than diet.
Competing interests: Nil.

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References:


Are the amounts of vitamins in commercially available dietary supplement formulations relevant for the management of psychiatric disorders in children?

Julia J Rucklidge, Amy Harris, Ian C Shaw

Abstract

Aim To investigate whether micronutrient supplements shown through research to have perceived benefits in the treatment of psychological/psychiatric symptoms in children have similar vitamin ingredients and doses to over-the-counter dietary supplements.

Methods We conducted a systematic review to identify studies that used micronutrients for the treatment of psychological/psychiatric symptoms in children with documented benefits; 13 different supplements were identified that included vitamin ingredients. They were compared with the vitamin composition of 22 over-the-counter child-targeted supplements available in New Zealand.

Results The vitamin ingredients were comparable across the research and commercially available supplements. However, the median vitamin daily doses in research supplements were found to be greater than those of over-the-counter supplements, with most mean differences being significant, including vitamins B1, B3, B6, B7, B12, C and D (p<0.05), B5 and B9 (p<0.001), but not vitamins A or B2.

Conclusions Micronutrient supplements found to show potential benefit in research with a focus on improving psychological/psychiatric symptoms in children have a significantly greater vitamin dose than over-the-counter supplements. Therefore, the results found in micronutrient research studies cannot be extrapolated to over-the-counter supplements. Comparing the myriad ingredients and dosages in micronutrient supplements is, however, a complex process and further investigation is required to understand fully the importance of our findings.

There is a plethora of micronutrient supplements for children available over-the-counter in New Zealand (NZ). They are regulated under the NZ Dietary Supplements Regulations1 (amended 20102; under revision). Since they are not covered by medicines legislation they cannot have medicinal claims; despite this their use in the general population is substantial. Indeed, a 1997 study in the USA reported that 54% of three year olds were given a dietary supplement, and of these, 85.4% were given a micronutrient supplement.3

Similarly, in a sample of 100 New Zealand children under 12 years old, 70% were given complementary or alternative medicines (CAM), of which multivitamins were among the most common types of CAM given.4

Doses of dietary supplements are set in accordance with the recommended daily intakes for the vitamin and minerals they contain, and since experimental efficacy data are often limited to specific age groups, setting maximum daily doses for
children is usually achieved by extrapolation from adult studies using recommended daily doses from North American Daily Recommended Intake (DRI) panels. Whether supplement doses are clinically effective or not is a subject of much debate.

Even though dietary supplements are not regulated as medicines, some commercial products infer clinical benefits by referring to published studies that report benefits. Given the current rise in studies being published looking at the effectiveness of micronutrients in the treatment of psychological/psychiatric disorders, it is important to determine to what extent commercial products are comparable with respect to the components that might be effective in the management of these psychological disorders, and whether the amounts of these components in the supplements’ formulations are sufficient to result in beneficial psychological effects when administered in accordance with the manufacturer’s instructions.

Perceived benefits of multi-micronutrient supplements have been reported in autism, Attention-Deficit/Hyperactivity Disorder (ADHD), mood disorders, and antisocial behaviours, with the evidence supporting their use ranging from case controlled studies, open label (OL) trials, database analyses to randomized controlled trials (RCTs). Acknowledging the relative newness of the field and therefore limited evidence base, and that the perceived benefits are not definitive in the context of properly conducted clinical trials, researchers have proposed a variety of mechanisms by which micronutrients could be beneficial in mental illness including the correction of in-born errors of metabolism, regulating emotions and behaviour, exerting an effect on the dopamine inhibitory system, improving mitochondrial function, promoting healthy gastrointestinal functioning and reducing inflammation, and acting as cofactors for various metabolic processes.

A key challenge associated with micronutrient supplement clinical effectiveness research is the applicability of the findings to commercial products available over-the-counter. Are commercial products comparable with formulations used in research? Can the reported research benefits be extrapolated to over-the-counter products? Such a comparison is important in order for the public to be aware that perceived benefits noted in research may not apply to products purchased in the supermarket, even if those products were not necessarily developed for the treatment of psychological disorders.

The present study identifies a broad range of over-the-counter micronutrient products for children, and compares their ingredients and recommended doses with supplements used in research studies that reported benefits in psychological disorders. Such an investigation may determine whether over-the-counter supplements sold in New Zealand are likely to provide psychological benefits in children.

**Methods**

**Inclusion of research micronutrient formulation data in the study**—Only published studies investigating psychological/psychiatric variables (e.g. stress, ADHD, psychosis) in children (<18 years) were included in our study. It is appreciated that this breadth of symptoms introduces heterogeneity in samples; however, as this is the first study of its kind, restricting inclusion to only diagnosed psychiatric disorders may conceal a body of literature that could well inform the direction of the field. The selection criteria for inclusion of research formulations required that a) the formulae must include ≥4 vitamins and/or minerals, and b) botanicals, amino acids and fatty acids were not included unless they were part of a formula including ≥4 vitamins and/or minerals and it was evident that the main
ingredients were the vitamins and minerals (this excluded supplements that were predominantly composed of amino acids).

Preparations that indicated that vitamins and minerals were contained within the formulation but that did not provide information on specific ingredients along with some information on dose were excluded. As there are so few RCTs using micronutrients, we included four experimental designs: RCTs, OL trials, case-control studies, and case studies with within-subject crossovers (ABAB). Such breadth also ensures that there was not bias towards RCT data because this can be problematic and acknowledges that single case research designs have been accepted as establishing empirical validity of therapies. No restrictions were placed on studies reviewed (e.g. sample size, trial length); however, it was a requirement of inclusion that the research report provided information about treatment responses. Only micronutrient supplements that conferred a treatment benefit were included; the rationale for only including those supplements with an observed treatment benefit was because the intent was to determine whether products sold over-the-counter, depending on how comparable they are to the research products in ingredients and doses, could equally confer a treatment effect for children with psychological disorders. Details of the full search can be found elsewhere.

Eighteen child trials were identified that met our inclusion criteria: five for the treatment of bipolar disorder, three for autism, seven for cognitive functioning, one for antisocial behaviours, and one for ADHD. Three studies were excluded as no effect was reported. This search resulted in 13 different micronutrient supplements being identified (referred to as the research supplements).

**Over-the-counter micronutrient products**—Over-the-counter micronutrient products were purchased from New Zealand supermarkets, health food stores, pharmacies and online. A micronutrient supplement was defined as any product that was labelled as a ‘multi-nutrient, multivitamin and/or multi-mineral, or micronutrient supplement’ and included ≥4 vitamins and/or minerals. For each micronutrient supplement, both the dose and largest recommended daily dose were recorded. The amount of each nutrient at the largest daily recommended dosage for a child was calculated. When recommended doses were based on age, the dose was calculated for the age range 3–13 years. When the recommended dose was body weight based, the weight bracket ≤45 kg was calculated. Twenty-two over-the-counter supplements were sampled (Table 1).

**Table 1. Over-the-counter micronutrient supplements included in the present study (daily doses of individual vitamins were calculated from amounts taken from the product labels)**

<table>
<thead>
<tr>
<th>Healtheries Fizz Bomb</th>
<th>Iceberg Labs’ KIDZ Multivitamins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healtheries Kidscare Chewables</td>
<td>Rainbow Light Essential Gummies Multivitamin &amp; Multimineral</td>
</tr>
<tr>
<td>Healtheries Tongue Fizzer</td>
<td>Essentials Children’s Multi with Acidophilus</td>
</tr>
<tr>
<td>Blackmores Kid’s Multivitamin</td>
<td>Healtheries Teen Multi</td>
</tr>
<tr>
<td>Centrum Kid’s Complete</td>
<td>Flinstone’s Gummies Complete (Bayer)</td>
</tr>
<tr>
<td>Nature’s Own Child Care Multivitamin &amp; Mineral</td>
<td>Children’s Probiotic &amp; Multivitamins (Lloyds Pharmacy)</td>
</tr>
<tr>
<td>Nature’s Sunshine Heroes Multiple Vitamin &amp; Mineral</td>
<td>Kindervital Tonic (Floradix)</td>
</tr>
<tr>
<td>Thompson’s Animals Junior Immunofort</td>
<td>Every Day Please (Microgenics)</td>
</tr>
<tr>
<td>Radiance Kid’s Multivitamin</td>
<td>Yummi Bears Multi-Vitamin &amp; Mineral (Hero Nutritionals)</td>
</tr>
<tr>
<td>Solgar Kangavite’s Complete Multivitamins &amp; Minerals</td>
<td>Kordel’s DHA Smart Multi</td>
</tr>
<tr>
<td>Emergen-C Kid’s Multi-Vitamin Fizzy Drink Mix</td>
<td>Nature’s Plus Animal Parade Chewable</td>
</tr>
</tbody>
</table>

**Establishing equivalency across ingredients and doses**—The dose of each ingredient across all supplements was normalised to either mcg or mg. This normalisation was done to allow for ease of comparison across the supplements. For
example, using the 30% International Unit (IU) conversion rate for vitamin A (retinol), 1 IU vitamin A was = 0.3 mcg.

Supplement ingredients were listed on the labels in a number of different ways for each vitamin or mineral. For example, Vitamin C was listed in four different forms: ascorbic acid, sodium ascorbate, calcium ascorbate and mixed mineral ascorbates.

To enable equitable comparison across the micronutrient supplements, the dose of the active compound (e.g. ascorbic acid) in each of the different forms was calculated. For example, if ascorbic acid (molar mass = 175 g/mol) was present in a particular formulation as sodium ascorbate (molar mass = 198 g/mol), the dose of ascorbic acid in the latter would be \((175/198) \times \text{sodium ascorbate dose mcg} = \text{ascorbic acid dose mcg}\).

Due to the difficulty of establishing consistent values across the minerals due to the complexity of identifying the correct chemical form used in each mineral variation, only the vitamins were selected for conversion. The active compound doses were calculated for vitamins A, B₁, B₂, B₃, B₅, B₆, B₇, B₉, B₁₂, and D.

Statistical analyses—Following conversion of each of the supplement vitamins to their active compound doses, the median, maximum and minimum daily dose were calculated. In order to provide the most accurate picture of the data, the median was chosen as the measure of central tendency due to a number of top end outliers.

Because the data did not follow a normal distribution, the two groups were compared using the non-parametric, two-tailed Mann-Whitney test, to identify whether the mean daily doses of the research and over-the-counter supplements were significantly different. The effect size was calculated for each Mann-Whitney test using \(N\) (total number of supplements) and the Z score.

The standard value of \(r\) for a weak effect size is 0.1, 0.3 for a medium effect size and 0.5 for a large effect size. Outliers were removed before conducting non-parametric tests and dose distribution graphs to improve the test accuracy and reduce the errors of inference. For each vitamin, no more than one outlier was removed from each of the supplement categories. Six outliers were removed from the research supplements and five were removed from the over-the-counter supplements.

Results

Ingredients and doses—Table 2 shows the frequency with which each vitamin appeared in the research and over-the-counter supplements. The ingredients in the research and over-the-counter supplements did not differ greatly. For example, vitamin B₁ (thiamine) appeared in 78% of the effective research supplements, and 86% of the over-the-counter supplements. Vitamin B₆ (pyridoxine) was an ingredient in all of the supplements.

Table 3 shows the median doses and dose ranges for the vitamins.
Table 2. Percentages of over-the-counter micronutrient supplements and research supplement formulations containing specific vitamins

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>% of research supplements containing specific vitamin</th>
<th>% of over-the-counter supplements containing specific vitamin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin A</td>
<td>78%</td>
<td>95%</td>
</tr>
<tr>
<td>Vitamin B₁ (Thiamine)</td>
<td>78%</td>
<td>86%</td>
</tr>
<tr>
<td>Vitamin B₂ (Riboflavin)</td>
<td>78%</td>
<td>82%</td>
</tr>
<tr>
<td>Vitamin B₃ (Niacin)</td>
<td>85%</td>
<td>91%</td>
</tr>
<tr>
<td>Vitamin B₄ (Pantothenic Acid)</td>
<td>78%</td>
<td>95%</td>
</tr>
<tr>
<td>Vitamin B₅ (Pyridoxine)</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Vitamin B₆ (Biotin)</td>
<td>46%</td>
<td>77%</td>
</tr>
<tr>
<td>Vitamin B₇ (Folic Acid)</td>
<td>78%</td>
<td>86%</td>
</tr>
<tr>
<td>Vitamin B₉ (Cyanocobalamin)</td>
<td>92%</td>
<td>100%</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>78%</td>
<td>100%</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>78%</td>
<td>91%</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>78%</td>
<td>95%</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>23%</td>
<td>14%</td>
</tr>
<tr>
<td>Choline</td>
<td>54%</td>
<td>41%</td>
</tr>
<tr>
<td>Inositol</td>
<td>54%</td>
<td>36%</td>
</tr>
</tbody>
</table>

Table 3. Median daily doses and dose ranges for vitamins in research supplement formulations and over-the-counter micronutrient products calculated from label and published formulations, showing that the doses of the research supplements tended to be higher than the doses of the over-the-counter supplements

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Research supplements</th>
<th>Over-the-counter supplements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Daily dose median±SD</td>
<td>Dose range</td>
</tr>
<tr>
<td>B₁</td>
<td>14.59±11.52 mg</td>
<td>0.7–243.18 mg</td>
</tr>
<tr>
<td>B₂</td>
<td>13.5±12.84 mg</td>
<td>0.85–200 mg</td>
</tr>
<tr>
<td>B₃</td>
<td>63.75±36.87 mg</td>
<td>10–750 mg</td>
</tr>
<tr>
<td>B₅</td>
<td>22.08±54.76 mg</td>
<td>0.92–450.8 mg</td>
</tr>
<tr>
<td>B₆</td>
<td>23.04±18.87 mg</td>
<td>0.9–287.95 mg</td>
</tr>
<tr>
<td>B₇</td>
<td>400±429.31 mcg</td>
<td>10–150 mcg</td>
</tr>
<tr>
<td>B₉</td>
<td>560±703.48 mcg</td>
<td>35–2048.5 mcg</td>
</tr>
<tr>
<td>B₁₂</td>
<td>900±490.71 mcg</td>
<td>1–500 mcg</td>
</tr>
<tr>
<td>A</td>
<td>1500±1416.36 mcg</td>
<td>300–4512.8 mcg</td>
</tr>
<tr>
<td>C</td>
<td>600±868.8 mg</td>
<td>26.6–1500 mg</td>
</tr>
<tr>
<td>D</td>
<td>10±13.59 mcg</td>
<td>2.5–40 mcg</td>
</tr>
</tbody>
</table>

Comparison of research supplements with over-the-counter supplements—The non-parametric comparison of the research and over-the-counter doses are presented in Table 4.
Table 4. Nonparametric tests of group differences in daily dose between research formulations and over-the-counter products (note that not all supplements were included in each analysis if the particular nutrient was not contained in its formulation); effect sizes are provided to highlight the magnitude of the group difference

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>( U ) (Mann-Whitney)</th>
<th>( Z )</th>
<th>( p ) (2-tailed)</th>
<th>( r^* ) (effect size)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B₁</td>
<td>36.5</td>
<td>-2.96</td>
<td>0.003</td>
<td>.55</td>
</tr>
<tr>
<td>B₂</td>
<td>62.5</td>
<td>-2.42</td>
<td>0.16</td>
<td>.43</td>
</tr>
<tr>
<td>B₃</td>
<td>41.5</td>
<td>3.16</td>
<td>0.002</td>
<td>.56</td>
</tr>
<tr>
<td>B₅</td>
<td>21</td>
<td>-3.95</td>
<td>0.001</td>
<td>.69</td>
</tr>
<tr>
<td>B₆</td>
<td>79</td>
<td>-2.88</td>
<td>0.004</td>
<td>.47</td>
</tr>
<tr>
<td>B₇</td>
<td>26</td>
<td>-3.08</td>
<td>0.002</td>
<td>.59</td>
</tr>
<tr>
<td>B₉</td>
<td>44.5</td>
<td>-3.25</td>
<td>0.001</td>
<td>.57</td>
</tr>
<tr>
<td>B₁₂</td>
<td>75</td>
<td>-2.68</td>
<td>0.007</td>
<td>.45</td>
</tr>
<tr>
<td>A</td>
<td>120</td>
<td>-1.06</td>
<td>0.289</td>
<td>.18</td>
</tr>
<tr>
<td>C</td>
<td>56.5</td>
<td>-3.06</td>
<td>0.002</td>
<td>.52</td>
</tr>
<tr>
<td>D</td>
<td>74.5</td>
<td>-2.19</td>
<td>0.029</td>
<td>.38</td>
</tr>
</tbody>
</table>

*Pearson’s r: weak effect size = 0.1, medium effect size = 0.3, large effect size = 0.5.

The research supplement vitamin doses were larger than the corresponding over-the-counter supplement doses. For example, the median vitamin B₁ daily dose for research and over-the-counter supplements was 14.59 mg and 1.22 mg respectively, \( U(29) = 36.5, p<0.01, r=0.55 \).

All of the doses, with the exception of vitamins A and B₂, were significantly higher in the research supplements when compared with the over-the-counter supplements.

Figure 1 shows the dose distribution across the vitamins with research doses on the left side of the graph and over-the-counter doses on the right, ordered in increasing magnitude of dose. It does not show an exhaustive list of the ingredients of all the products.
Figure 1. Individual daily doses of the vitamins for research formulations and over-the-counter products showing the consistent and significantly higher doses in the research formulations compared to over-the-counter-products.
Discussion

The empirical composition of research and over-the-counter supplements did not differ greatly. However, the doses of the vitamins were significantly greater in the research supplements compared with the over-the-counter supplements for all the vitamins studied except vitamins A and B2.

The most important ramification of these differences may be the inability to extrapolate micronutrient research findings to over-the-counter supplements. Therefore, research indicating a positive effect of supplements on, for example, ADHD, may not apply to patients treated or self-medicating with over-the-counter supplements. Similarly, claims or inferences on over-the-counter supplements’ labels relating to their benefits based on research studies published to date might be misleading unless specific studies are conducted on that specific product providing the evidence of efficacy.

Vitamin B6 was included in all the supplements studied, indicating that it might have an important role in the combination of nutrients used in micronutrient supplements. Vitamin B6 is involved in the synthesis of a number of neurotransmitters including serotonin, dopamine, acetylcholine, norepinephrine and GABA;39 these are all neurotransmitters known to affect depression, mood, sleep, attention, appetite, and anxiety. Although vitamin B6 was included in all of the supplements studied, the median dose of this vitamin was significantly higher in the research compared to the over-the-counter supplements.

The median over-the-counter supplement daily dose of vitamin B6 was close to the recommended daily intake (RDI) of 1 mg for New Zealand children aged between 9 and 13;4 the median daily dose in research supplements was much higher (approx. 23 mg). The higher research supplement dose may indicate that vitamin B6 levels significantly above the RDI are necessary to effectively impact on psychological symptoms and cognitive functioning.

Vitamin B12 was also included in the formulations of all over-the-counter supplements studied, and almost all of the research supplements, indicating that vitamin B12 might also play an important role in the combination of ingredients used in micronutrient supplements. The psychological effects of vitamin B12 deficiency include adverse effects on cognition from adolescence years onwards,40 indicating that vitamin B12 might have a role in improved cognition.
The RDI for New Zealand children aged between 9 and 13 is 1.8 mcg,\textsuperscript{5} which is lower than the 5 mcg over-the-counter supplement daily dose median and significantly lower than the research median dose of 900 mcg. This large difference indicates that achieving a significant change in psychological symptoms might require vitamin B\textsubscript{12} doses significantly greater than the RDI for New Zealand children.

The greatest differences in doses between research and over-the-counter supplements were for vitamins B\textsubscript{5} and B\textsubscript{9}. The significantly higher doses of vitamin B\textsubscript{5} and B\textsubscript{9} in research supplements are unsurprising given their roles in modulating mental health.\textsuperscript{41} As a component of coenzyme A (CoA), vitamin B\textsubscript{5} plays an important role in the synthesis of vitamins A and D as well as supporting adrenal function and cortisol production.\textsuperscript{41} Acetyl CoA is the acetyl group donor in acetylcholine synthesis and the latter is a neurotransmitter with important roles in memory, attention and cognitive functions.\textsuperscript{5}

The median dose of vitamin B\textsubscript{5} in research supplements (22 mg) is approximately four-times the RDI of 5 mg for boys and 4 mg for girls, whereas the vitamin B\textsubscript{5} dose in over-the-counter supplements was much closer to the RDI, with a median daily dose of 7.5 mg. The median daily dose of vitamin B\textsubscript{9} in research supplements of 560 mcg is 260 mcg above the RDI\textsuperscript{5} for children aged between 9 and 13. Although much higher than the 100 mcg over-the-counter supplement median, the research daily dose is still well below the human Lowest Observed Adverse Effect Level (LOAEL) of 5 mg/day.\textsuperscript{5}

These findings indicate that over-the-counter micronutrient supplements available in supermarkets, health food stores and pharmacies are unlikely to be as effective, if effective at all, compared with the supplements formulated specially for research when targeting behavioural changes. Despite this, some micronutrient products available in New Zealand advertise that their ingredients have psychological benefits (such as improved concentration).

Although the information of the nutrient effect that they present is correct, the inferred claim may be misleading because of the differences between the research and over-the-counter doses and the fact that the claims are likely to be based on research dose studies. Based on research supplement composition, we suggest that the psychological benefits that might result from micronutrient administration would only be obtained from doses much higher than those found in over-the-counter micronutrient supplements. It is the doses of the micronutrients, not simply their presence, that is important in predicting their biological effects.

It is likely that the over-the-counter products have lower doses of some nutrients because of statutory limits set in the Dietary Supplements Amendment Regulations 2010.\textsuperscript{2} For example, the Act specifies that a daily dose of vitamin B\textsubscript{9} (folic acid) in a supplement manufactured in New Zealand must not exceed 500 mcg even though the Tolerable Upper Intake Level (UL) is 1000 mcg. However, since the UL was set at 1000 mcg to prevent vitamin B\textsubscript{12} deficiency, if vitamin B\textsubscript{12} is consumed alongside vitamin B\textsubscript{9}, that statutory limit could be much higher without adverse effects.

The statutory limit is currently being reviewed in light of proposals for fortification of bread with folic acid and the development of the Natural Health Products Bill 2011, where the upper limit may increase to 1000 mcg or may even possibly be removed.
However, in the meantime, the current commercial products reflect these statutory limits (median 100 mcg) whereas research products often exceed the statutory limit for folic acid (560 mcg).

Likewise, the statutory daily dose limit for vitamin B$\textsubscript{12}$ is 50 mcg despite the fact that there is no evidence of harm associated with higher doses.$^5$ Again the research supplements exceeded this dose (median 900 mcg) whereas the over-the-counter products were substantially lower (median 5 mcg) and within the statutory limit.

Again, these values are being reviewed as part of the Natural Health Products Bill; however, the regulations may indeed have prevented manufacturers from providing the necessary dose to confer health benefits. This thinking is speculative due to the great number of ingredients contained within the products and the potentially complex biological interactions between them in the context of health benefits; however, it is possible that the government legislation of commercial products results in products that cannot reflect research and knowledge on biological effects, safety and ULs.

This research calls into question the usefulness of RDIs and Recommended Dietary Allowances (RDAs). Indeed, Fletcher et al$^{42}$ showed that the North American diet, as reflected by food intake, while sufficient to prevent vitamin deficiency diseases, is inadequate to support optimal health.

The United States (US) RDA guideline levels in 2002 were similar overall to those set by the National Health and Medical Research Council (NHMRC)$^5$ guidelines, with the RDA of some vitamins in the New Zealand guidelines set marginally higher, and others marginally lower. The US RDA levels were established to prevent acute vitamin deficiency disorders; however, this current study suggests that higher doses of some vitamins and minerals may be necessary to achieve optimal mental health for some people. The assessment made by Fletcher et al$^{12}$ that RDA values are set too low, is supported by the higher median vitamin daily doses (with the exception of vitamins B$\textsubscript{3}$ and B$\textsubscript{9}$ in the over-the-counter supplements) found in both the research and over-the-counter supplements. This difference was most evident in the median research vitamin B$\textsubscript{12}$ dose which was 500 times greater than the NHMRC RDA$^5$ guidelines.

This high B$\textsubscript{12}$ vitamin intake is unlikely to produce adverse effects, possibly because of the body’s ability to decrease absorption in response to high vitamin intakes.$^5$ The over-the-counter median daily dose of vitamin B$\textsubscript{12}$ was also greater than the RDA (i.e. 2.8 times greater than the recommended daily dose).

It is possible that the doses in over-the-counter supplements are significantly lower than the doses in research supplements because of the high cost of micronutrients.

There were 13 effective research supplements and the prices were available for two of these. Using these supplements as a cost guide, the average cost of a research supplement when used in a treatment setting would be NZ$6.90/day.

The average price of the 22 commercial products studied was NZ$0.64/day. This large difference in the daily cost of providing a child with a micronutrient supplement is likely to explain the low doses found in commercial products; it is unlikely consumers would be willing to pay for higher doses. This might, however, change if consumers were aware that the supplements they are buying are unlikely to have biological effects with respect to mental health symptoms.
Limitations—First, the frequency of nutrient inclusion comparison did not account for the different forms of each nutrient in micronutrient formulations. Indeed, vitamins and minerals were found in a number of different chemical forms across the spectrum of supplements.

In-depth analysis of the forms of each mineral and vitamin may provide further information to guide a decision as to whether or not these ingredients are comparable. This in-depth analysis is important because the form of a nutrient indicates the amount of specific mineral or vitamin contained. For example, retinyl palmitate which is a form of vitamin A, contains 54.7% vitamin A compared to retinol acetate, which contains 87%.\(^5\)

Also, the chemical form of the micronutrient is very likely to affect the absorption efficiency and thus bioavailability of the compound. For example, the palmitoyl ester of vitamin A is more hydrophobic than vitamin A itself and is therefore likely to be better absorbed from the gastrointestinal tract. Clearly these differences are important in the context of biological activity of micronutrients and should be further studied in order to understand fully their implications.

Finally, the range of study methodologies included to determine effectiveness of the ingredients and doses of the research supplements was broad given the dearth of RCTs that have been conducted. Given that this is the first comparison of its kind, we felt comfortable including a broad range of methodologies to identify potential effective nutrient levels and as the field becomes more rigorous, we should gain a greater understanding of optimal doses required in order to effect change in psychological symptoms.

Conclusions—There is significant debate about the usefulness and efficacy of micronutrient-based dietary supplements in the management of psychological disorders. The preliminary evidence that supports their use is mostly based on studies using formulations that contain significantly higher micronutrient doses than over-the-counter products.

Therefore, even if published efficacy data support the use of micronutrients in psychological disorders, it is not clear whether over-the-counter products will have benefits because of their significantly lower micronutrient doses.

Competing interest: Nil.

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References:


Medically facilitated discharge of adult diabetic ketoacidosis admissions: precipitants and average length of stay

King W Yong, M Peter Moore, Helen Lunt

Abstract

Aims To assess the impact of medically facilitated discharge by a specialist registrar on diabetic ketoacidosis (DKA) length of stay (LOS) and to collect data from these DKA admissions for a descriptive summary of their clinical and biochemical characteristics.

Method DKA admissions were identified through the electronic patient management system, Monday to Friday over a 9 month period. The admitting team was then offered assistance with discharge planning (‘study intervention’). The registrar also collected clinical information for all identified DKA admissions.

Results There were 71 DKA admissions; 92% had type 1 diabetes and 56% were overnight admissions. Following exclusion of four admissions with prolonged LOS secondary to major comorbidities, mean LOS fell from 3.7 (± 1.0) to 2.8 (± 0.3) days. Facilitated discharge had no major impact on LOS. The commonest precipitant for admission was insulin omission, accounting for 65% of admissions. Local practice was to discharge patients following resolution of acidosis, but prior to complete abolition of ketosis.

Conclusions The majority of DKA admissions were of short duration. Achieving further reduction in LOS is therefore difficult. Insulin omission was the commonest DKA precipitant. Diabetes clinical resources may be best allocated on preventing DKA admissions, rather than facilitating early discharge.

The mean LOS (length of stay) for patients discharged from hospital with DKA (diabetic ketoacidosis) has fallen over the last two decades.\textsuperscript{1-4} Historically, studies from the UK and USA have reported mean LOS of around 6.1 to 6.9 days,\textsuperscript{1-3} but the mean LOS in the United States for patients admitted with DKA as the primary diagnosis has fallen from 5.7 days in 1988 to 3.4 days in 2009.\textsuperscript{4} Multiple factors are likely to have contributed to this shortening of mean LOS. These include widespread use of clinical management guidelines,\textsuperscript{5,6} patients’ ability to make an earlier self-diagnosis of DKA\textsuperscript{7} and the involvement of specialist diabetes nurses.\textsuperscript{8}

Although patient mortality associated with DKA is low,\textsuperscript{9} DKA admissions have significant impact on patients’ quality of life and represent an ongoing cost to the health care system.

The primary objective of this prospective study is to assess the impact of providing a facilitated discharge service, with early involvement of an appropriately trained medical specialist\textsuperscript{10} (a senior endocrinology registrar attached to the local hospital’s Diabetes Centre) on LOS.
The secondary objectives are to describe the clinical characteristics of patients admitted with DKA, including the triggers for the development of DKA and patients’ biochemical characteristics, including discharge beta-hydroxybutyrate levels.

**Method**

**Setting**—The study was undertaken in a tertiary teaching hospital (Christchurch Hospital, New Zealand) that serves a population of predominantly European New Zealanders with a high prevalence of type 1 diabetes; the prevalence in the 20–24 year age group is 427 per 100,000 which approaches the level seen in high risk Scandinavian populations. The hospital serves a geographically well-defined population of around 500,000 people. It has 650 inpatient beds and admits in excess of 35,000 patients annually. The multidisciplinary diabetes team, which is located adjacent to the hospital in the Diabetes Centre, includes registrars undertaking advanced training in endocrinology / diabetes. Admissions with DKA that are not able to be treated quickly and ‘turned around’ by the Emergency Department team are usually cared for by the Department of General Medicine.

Only a minority of the General Medicine teams are led by consultants with a specialist interest in diabetes. The General Medicine teams can access input from both nursing and medical members of the multidisciplinary diabetes team but typically this might occur late during DKA admission. The study took place between February and November 2010.

**Intervention**—Prior to commencement of this study, local diabetologists and endocrine trainees were asked to identify modifiable delays in the DKA discharge process. These included late switching of intravenous variable insulin infusion to subcutaneous insulin, not providing patients with an appropriate and timely prescription for diabetes related medical products and not replacing or upgrading diabetes medical devices. An additional delay related to the time associated with processing a referral from the admitting team to the multidisciplinary diabetes team.

The senior registrar’s intervention aimed to provide proactive, timely specialised input at an earlier time point than usual, thereby reducing the delays identified above. Prior to study commencement, the specialist registrar notified all General Medicine consultants and their junior team members about the aims of the study intervention.

**Patient identification**—The registrar identified adult (>15 years old) admissions with DKA, by checking the hospital’s electronic patient management system each weekday morning. Once a potential DKA admission had been identified, the admitting team was then offered specialist registrar facilitation of patient discharge planning, starting as early during the DKA admission as possible. The registrar was available on weekdays (Monday to Friday), thus patients admitted over the weekend could only be offered the intervention if they were still present on the ward the following Monday.

**Calculating length of stay (LOS)**—The Coding Department was asked to provide LOS data at completion of the study. Local coding practice is as follows: Irrespective of the time of day an admission occurs, LOS is recorded as Day 1 on the day of admission and the time from midnight is counted as Day 2, thus LOS data is treated as a categorical variable.

A calculation of the time saved on LOS by the intervention was made by the registrar, for each of the patients assessed. Time saved per admission (hours) was obtained by estimating the delay in discharge timing that would have occurred if usual care (typically General Medicine team led decision making) had occurred.

A couple of examples are provided below, to illustrate how these estimations were done: Typically, the transition from intravenous to subcutaneous insulin would be made after the post-acute consultant ward round and the variable insulin infusion would be stopped two hours after the next meal, thus the transition would be complete by the mid-afternoon. The specialist registrar’s early intervention typically allowed earlier transition onto subcutaneous insulin and the time difference between these two clinical processes was estimated.

Similarly, referrals to a diabetes multidisciplinary team member such as the diabetes nurse or dietitian were typically made by General Medicine team after the post-acute ward rounds, usually in the early afternoon. An estimate of the time difference was made between the early activation of these referrals by the specialist registrar and the time they would have been taken, if the General Medicine team had undertaken these referrals.
Reconciliation of diabetic ketoacidosis (DKA) admissions identified during the study and DKA discharges identified by the Coding Department—On study completion, DKA cases identified by the registrar were checked against the Coding Department’s discharge diagnoses, to ensure that all eligible cases were included in the secondary study outcome; the prospective audit of DKA clinical and biochemical characteristics.

Demographic, clinical and biochemical data—The diagnosis of DKA was based on the presence of hyperglycaemia (blood glucose greater than 10 mmol/L) and also the presence of ketoacidosis. Demographic, clinical and biochemical data that had been collected as part of the patient’s routine clinical care were collated on all identified admissions, including admissions that did not have registrar intervention (weekend admissions, admissions under clinical teams that did not require diabetes registrar involvement in the patient’s care). The type and level of registrar intervention needed for individual patients was recorded. The precipitants for DKA were also identified. Where possible this was done at the time of admission by interviewing the patient and also from collateral history.

If the patient was not directly assessed by the registrar, the relevant information was obtained from discussions with the admitting team or by case note review, during or as soon after the admission as was practically possible. Patients were identified as having recurrent DKA if they had had a previous admission with DKA within the 9-month study period.

In addition to the biochemical information collected routinely by the admitting teams, measurement of capillary beta-hydroxybutyrate was undertaken by the endocrine registrar close to discharge, in selected patients with a paucity of discharge biochemical information. This allowed assessment of the degree of ketosis at discharge for audit purposes. These capillary test results were not conveyed to the General Medicine team.

Unless otherwise stated, averages are given as a mean (± SD). Statistical comparisons were undertaken using standard parametric methods. The study had local Ethics Committee approval (Ethics reference: URA/10/EXP/025).

Results

Number of admissions with diabetic ketoacidosis (DKA)—After reconciliation of cases of DKA identified by the registrar with those identified by the Coding Department, 54 patients with 71 admissions for DKA were identified over the study period.

Forty-four patients had only 1 admission. Ten patients had recurrent admissions: four had 2 admissions, five had 3 admissions and one had 4 admissions. There were no DKA fatalities over the period of study. All subsequent data describes these 71 admissions.

Patient characteristics—Median age was 27 years and age ranged from 15 to 80 years, 40 (56%) DKA admissions were male. With regards to diabetes type, 66 (93%) had type 1 diabetes, four (6%) had type 2 diabetes and one (1%) had pancreatic insulin insufficiency from chronic alcoholic pancreatitis. Five (7%) admissions were for newly diagnosed diabetes; four (6%) type 1 diabetes and one (1%) type 2 diabetes.

Length of stay (LOS): description and exploration of clinical determinants of LOS—LOS ranged from 2 to 32 days, with 40 (56%) admissions staying for 2 days (overnight) and 67 (94%) admissions having a LOS of 7 days or less. Once admitted, no patient was discharged on the same day. LOS distribution for the DKA admissions is summarised in Figure 1.
Figure 1. Length of stay distribution for diabetic ketoacidosis (DKA) admissions (n=71)

![Figure 1](image)

**Note:** †No patient was admitted and discharged on same day, thus there were no “one day” admissions. Four DKA long stayers labelled as A, B, C and D.

There were four admissions with prolonged LOS and their clinical characteristics are detailed in Table 1.

Table 1. Clinical description of diabetic ketoacidosis (DKA) long stayers (n=4)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Discharge diagnosis and medical comorbidities</th>
<th>LOS (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>25</td>
<td>Type 1 DM, gastroparesis, anaemia requiring inpatient gastroscopy</td>
<td>12</td>
</tr>
<tr>
<td>B</td>
<td>51</td>
<td>Type 2 DM, concurrent septic illness, cognitive impairment, required rehabilitation</td>
<td>15</td>
</tr>
<tr>
<td>C</td>
<td>46</td>
<td>Alcoholic pancreatitis, alcohol withdrawal, prolonged ICU admission</td>
<td>18</td>
</tr>
<tr>
<td>D</td>
<td>80</td>
<td>New Type 1 DM, ICU admission, prolonged delirium, deconditioning, required rehabilitation and placement</td>
<td>32</td>
</tr>
</tbody>
</table>

**Note:** LOS=length of stay; DM=diabetes mellitus; ICU=intensive care unit.

Mean LOS for the total (71) admissions was 3.7 (±1.0) days. This fell to 2.8 (±0.3) days when the four longer stay admissions were excluded. The 52 admissions reviewed by the registrar had a median LOS of 2.0 (2.0–6.0) days and mean LOS of 2.7 (±0.3) days. For the remaining 15 admissions that did not receive registrar input, the median LOS was 2.0 (2.0–7.0) days and mean LOS was 3.0 (±0.8) days.

The American Diabetes Association’s DKA diagnostic criteria, defined in their 2009 consensus statement,\(^{13}\) are more conservative than this study’s more inclusive diagnostic criteria. Considering those admissions of 7 days duration or less (n = 67), 56 admissions satisfied ADA criteria for DKA and their mean LOS of 2.8 (±0.3) days
was identical to mean LOS for the 67 admissions, utilising this study’s inclusive criteria.

The mean age of overnight admissions was younger than the mean age of those requiring longer (3 to 7 days) admissions (27 years versus 38 years, p = 0.002). There was no relationship between LOS and the biochemical markers of DKA severity, collected on admission. (See below for details of these biochemical markers).

**Registrar intervention: estimated impact on length of stay (LOS)**—The registrar reviewed 55 (78%) of the 71 DKA admissions. The mean number of registrar visits per reviewed admission was 1.8 and the mean amount of time spent by the registrar on each reviewed admission was 2.5 hours.

Typical tasks undertaken by the registrar include assistance in the transition between variable intravenous insulin infusion and subcutaneous insulin treatment in 41 (58%) admissions, provision of new insulin prescriptions on 26 (37%) occasions and provision of ketone testing strips on 22 (31%) occasions.

New blood glucose meters were provided during 16 (23%) admissions. The registrar estimated the amount of time saved on LOS per admission receiving registrar input, to average 3.3 hours.

**Clinical and biochemical assessment of diabetic ketoacidosis (DKA) severity**—Three (4%) DKA admissions were initially managed by the Intensive Care Unit due to the severity of presentation and were later transferred to General Medicine teams. Two (3%) admissions had mild DKA and were managed with subcutaneous insulin injections without the need for intravenous insulin infusion. Capillary and plasma beta-hydroxybutyrate measurements on discharge were obtained for 44 (62%) and 21 (30%) admissions respectively and the majority of these results showed evidence of mild ketosis (see Table 2). DKA biochemical markers are summarised in Table 2.

<table>
<thead>
<tr>
<th>Biochemical marker</th>
<th>% admissions</th>
<th>Median (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Admission biochemistry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plasma glucose (mmol/L)</td>
<td>100%</td>
<td>30.8 (12.5–75.1)</td>
</tr>
<tr>
<td>Arterial pH‡</td>
<td>27%</td>
<td>7.16 (6.86–7.35)</td>
</tr>
<tr>
<td>Venous pH‡</td>
<td>89%</td>
<td>7.23 (6.80–7.34)</td>
</tr>
<tr>
<td>Plasma β-OH (mmol/L)</td>
<td>88%</td>
<td>7.18 (0.03–11.5)</td>
</tr>
<tr>
<td><strong>Discharge biochemistry</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial pH</td>
<td>16%</td>
<td>7.35 (7.28–7.43)</td>
</tr>
<tr>
<td>Venous pH</td>
<td>55%</td>
<td>7.35 (7.26–7.47)</td>
</tr>
<tr>
<td>Plasma β-OH (mmol/L)</td>
<td>30%</td>
<td>0.86 (0.03–4.74)</td>
</tr>
<tr>
<td>Capillary β-OH (mmol/L)</td>
<td>62%</td>
<td>0.10 (0–2.0)</td>
</tr>
<tr>
<td>HbA1c (mmol/mol)$</td>
<td>79%</td>
<td>108 (57–156)</td>
</tr>
</tbody>
</table>

**Note:** β-OH=beta-hydroxybutyrate; ‡Either an arterial pH and / or a venous pH was available for all 71 admissions; §HbA1c expressed in % units is 12.0% (7.4–16.4%).

**Triggers for the development of diabetic ketoacidosis (DKA)**—A single trigger was identified for 47 (66%) admissions, while 24 (34%) admissions had more than one trigger. Omitting or stopping insulin was the major factor in 46 (65%) admissions and in 24 (34%) admissions infection was a factor.
Alcohol was implicated in 9 (13%) admissions, 5 (7%) admissions were for the first presentation of diabetes and 2 (3%) admissions were related to recreational drugs. Triggering factors were unable to be identified in 7 (10%) of the admissions.

Discussion

The study was designed as feasibility study to ascertain whether it might be possible to reduce DKA LOS with a facilitated discharge model of care. A multicentre randomised controlled trial (RCT) designed to minimise contamination bias (that is, the effect of the intervention ‘spilling over’ into the control group), would then have been the next logical step to investigate the effectiveness of this care model. However, since no statistically significant reduction in LOS was found, the study then focussed on a description of the clinical findings of patients admitted with DKA, including descriptions of DKA severity and triggering factors.

The estimated amount of time saved on length of stay by the registrar intervention was 3.5 hours per reviewed patient. The median LOS during the 9 month study of facilitated discharge was 2 days. This median LOS is shorter than the 3 days (range 1 to 21 days) obtained retrospectively from the 101 DKA admissions from the previous year (2009 LOS data from Coding Department, not analysed for potential miscoding/misclassification), however this study’s mean LOS of 3.7 (±1.0) days is little different from the 2009 mean LOS of 3.1 (±0.5) days.

This lack of overt temporal difference in overall results and also the short median and mean LOS implies that; a) the registrar intervention had only a weak or no effect on overall LOS and that b) we are near the limits for reducing LOS for DKA admissions. Although the facilitated discharge intervention had minimal impact on LOS, it may have offered other benefits that were not measured in this study, such as a reduced amount of time spent on patient care by the admitting team and better patient self-care in the period immediately following discharge.

Patients’ admission biochemical profiles described in the current study are similar to that of earlier studies,1,7,14,15 thus the trend towards shorter LOS is unlikely to be due to secular differences in biochemical severity of DKA presentation. It was of passing interest to note that younger patients tended to have short overnight stays. This relationship between age and LOS has been described previously.1

Secondary analyses showed that insulin omission was the major precipitant for DKA admissions. This was in contrast to the findings from some retrospective studies15-19 where infection was the most commonly cited cause. Our findings were however in keeping with two other prospective studies14,20 which also found missed insulin injections to be the major factor for admission.

In contrast to a recent Australian report,21 recreational drugs did not feature prominently as a DKA trigger in our study. It is not known whether this represents a true difference in precipitating factors, reflecting differences in prevalence of substance abuse between Australian and New Zealand populations studied, or whether it represents under-reporting by the patients in our study.

Another secondary finding was that discharges were occurring at a time ketosis was settling, as judged by median plasma beta-hydroxybutyrate level at discharge of 0.86
mmol/L and median capillary beta-hydroxybutyrate of 0.1 mmol/L, rather than when patients showed complete metabolic recovery.

This study has several limitations. The time saved on LOS by the intervention was estimated by the registrar performing the intervention, rather than by an independent third party.

Another study limitation relates to a system limitation associated with the recording of LOS. The current study estimated LOS in number of days. In theory, measurement of LOS could have been undertaken in hours but it would have been difficult to interpret this measurement; the electronic patient management system allows LOS to be calculated in hours but it is based on the discharge summary completion timing rather than the time the patient left hospital. In our local hospital it is not uncommon for patients to leave some time after completion of their discharge summary, for example due to delays in arranging transportation or in arranging review by allied health personnel. The converse is also true, with some patients leaving the hospital when medically stable, prior to formal completion of the discharge summary.

Finally, the study focused on saving bed days (LOS) once admission had occurred, as the diabetes team thought that the later part of the admission was associated with the greatest modifiable delays. Maximal reduction in LOS is however dependent on effective management of the patient journey through all stages of hospital admission process. It may be possible for selected patients with minor DKA to be discharged on the same day as admission, if this event is planned for at the very beginning of the admission process, i.e. in the Emergency Department. However, as DKA admission volumes are low relative to overall admission numbers and as most DKA admissions are brief overnight stays, arguably there is limited incentive to focus on additional interventions aimed at reducing DKA bed day stay, when it is already very short.

Conclusions

The LOS for DKA admissions has fallen over the last two decades and we are probably now close to the limits for a safe LOS, with little capacity for any further reduction in LOS. The current study suggests that the commonest precipitant for DKA is insulin omission. Future resources might therefore best be directed towards reducing admissions and particularly reducing recurrent admissions, rather than trying to reduce further the number of days in hospital.

Competing interests: Nil.

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Hepatic complications in poorly controlled type 1 diabetes mellitus: a case report
Johanna Martin, Paul Tomlinson

Abstract
We present the case of a 13-year-old male with poorly controlled type 1 diabetes mellitus who developed significantly deranged liver transaminases following an episode of diabetic ketoacidosis. A liver biopsy diagnosed glycogenic hepatopathy (GH). We believe the combination of GH and ischaemic hepatitis led to his presentation.

The link between type 1 diabetes mellitus (T1DM) and hepatic abnormalities is well established. Hepatic complications in children are primarily associated with poor glycaemic control.¹

We present the case of a 13-year-old male with poorly controlled T1DM who developed severely deranged liver transaminases following an episode of diabetic ketoacidosis (DKA).

Discussed are two diagnoses we believe contributed to this clinical picture.

Case report
The subject is a 13-year-old male with poorly controlled T1DM (average glycosylated haemoglobin 90 mmol/mol (10.4%) and marked lipohypertrophy). Four years after diagnosis he presented following a school camp with respiratory distress, decreased level of consciousness (GCS 10), poor perfusion (capillary refill time 4 seconds) and pH 6.92. His heart rate was 152 and blood pressure was 150/86 mmHg. This was his fourth episode of DKA.

The next day, following administration of intravenous fluids and insulin, he was noted to have hepatomegaly. Investigations showed markedly raised transaminases—maximum ALT 1196 IU/L (30 × upper limit of normal [ULN]), AST 3969 IU/L (80 × ULN) and GGT 647 IU/L (15 × ULN). Synthetic liver function remained normal. Ultrasound revealed hepatomegaly (span 26 cm) with increased echogenicity but normal blood flow. He had associated renal impairment (maximum creatinine 167 micromol/L).

Hepatitis A, B, C, HIV, cytomegalovirus, toxoplasma and leptospira serology were negative. Epstein-Barr virus and parvovirus serology were consistent with past infection. Toxicology and metabolic screens (including ceruloplasmin, alpha-1 antitrypsin and alpha fetoprotein) were negative.

Autoantibody titres (including smooth muscle antibody) and immunology screens were negative apart from an ANA titre of 1/320.

Percutaneous liver biopsy showed normal architecture with glycogen accumulation in the hepatocytes. There was mild macrosteatosis but no inflammation or fibrosis.
His liver enzymes slowly improved with improved glycaemic control and normalised by day 16.

Discussion

In 1930 Mauriac noted hepatomegaly, impaired growth, pubertal delay and Cushingoid features in poorly controlled diabetic children. The term glycogenic hepatopathy (GH) has been applied to diabetic patients (including adults) with hepatic glycogen deposition in the absence of other features of Mauriac syndrome.

GH usually presents with hepatomegaly and moderately elevated transaminases (up to 10 times ULN) in the setting of poorly controlled T1DM. It is believed to be the consequence of recurring fluctuations in insulin and glucose levels.

During periods of hyperglycaemia, glucose passively diffuses into hepatocytes independent of insulin. Once treated, the presence of insulin promotes the conversion of glucose to glycogen inside the hepatocytes. GH is indistinguishable from non-alcoholic fatty liver disease without a biopsy and is reversible (transaminases usually normalise within 2 to 4 weeks) with improved glycaemic control.

Ischaemic hepatitis (IH) is characterised by a sudden reversible rise in serum transaminases in the setting of hypoxia or hypovolaemia. IH has been clearly described in adults and is generally associated with cardiac failure. We have only
found one case where IH was described in association with DKA and no liver biopsy was performed.  

While hypotension has previously been considered a key feature of IH, it does not need to be present for the diagnosis. Our subject was poorly perfused and subsequently developed renal impairment despite an absence of hypotension. Liver necrosis has been suggested as being a mandatory finding in IH, but its presence is not universal in patients who otherwise fit the criteria for this diagnosis. Our subject did not have evidence of necrosis on his biopsy.

Our subject had features of two pathologies—GH and IH. Individually, neither diagnosis sufficiently explained the overall clinical picture. Furthermore IH is commonly described in conjunction with other forms of liver pathology, for example in congestive heart failure.

Therefore, we believe the sudden, dramatic and reversible rise in liver enzymes in our patient occurred due to the combination of GH and IH. This would appear to be an important complication of poorly controlled diabetes in the setting of DKA.

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**References:**

FIZZ Sugary Drink Free Pacific by 2030—Symposium Declaration

Background—The Sugary Drink Free Pacific by 2030? Symposium was held on February 19th and 20th, 2014 at the University of Auckland, New Zealand. An initial draft of this declaration was presented to delegates. The final version was primarily authored by the symposium steering committee and was sent to all delegates. A list of delegates and other contacts that have supported this declaration is available on the FIZZ website: www.fizz.org.nz

Declaration—Sugar, also known as sucrose or table sugar, is found in many manufactured foods and drinks in the New Zealand food supply. It is distinguished from intrinsic sugar, generally present in whole fruit and vegetables. UN food balance sheet data states that New Zealanders, on average, consume about 147g per day (37 teaspoons) of added sugar (2009).

Nutrition surveys have reported that adult males consume, on average, 55g per day (14 teaspoons); and females 42g per day (11 teaspoons). In 2002, children consumed an average of 55g of sucrose (14 teaspoons) between the ages of 5 and 6 years, and 69g (17 teaspoons) between 11 and 14 years, from all sources per day. Recalled responses, such as from nutrition surveys, are likely to under-report actual sucrose intake by about 20%, when they are compared to more objective methods. The adult survey response rate of 61% also suggests that actual intake is likely to be higher than that reported, as respondents are more likely to have healthy diets, compared to those who refuse to take part.

Nutrition surveys show that the most important source of sugar for children, supplying about 25% of dietary sugar, is from sugar sweetened beverages, both powdered and soft drinks, although mostly from the latter. In this document, we define sugary drinks as those which are greater than 5% added sugar content, by weight. They include, but are not limited to, sugar-sweetened soft drinks, cordials, powdered drinks, fruit drinks and fruit juices.

In contrast to the average population intake, the American Heart Association recommends a safe upper limit of nine teaspoons per day (36g) of added sugar for men and six (24g) for women to limit weight gain and reduce risk of cardiovascular disease incidence. The upper limit for children was set at 3 teaspoons (12g) per day. Therefore, average New Zealand sugar intake is conservatively estimated at between 1.5 to 5 times higher than that recommended by this heart health organisation.

Dietary sugars have been widely accepted as a cause of weight gain and obesity. The association between free sugars and dental caries has also been established beyond reasonable doubt.
In addition, dietary sugar particularly that consumed in sugar-sweetened soft drinks, has been associated with:

- Cardiovascular disease\textsuperscript{9,10}
- Type-2 diabetes\textsuperscript{11–17}
- Raised blood pressure\textsuperscript{18}
- Dyslipidaemia\textsuperscript{19}
- Gout\textsuperscript{20,21}

These diseases are major contributors to New Zealand’s burden of disease, with cardiovascular disease, for example, accounting for a quarter of New Zealand’s disability adjusted life years lost due to illness.\textsuperscript{22} Increasing evidence also suggests that sugar is addictive among high consumers, and individuals may experience withdrawal symptoms when they attempt to control their intake.\textsuperscript{23}

Improvement in dental health and reduction in obesity and obesity-related diseases, and cardiovascular diseases will have flow-on benefits throughout New Zealand society including in the health sector, educational settings, and social services.

We believe that sugar-sweetened beverages, particularly soft drinks, are an unnecessary addition to children and adults’ diets. They provide little in the way of nutrition other than energy and other healthier or lower-energy beverage alternatives are readily available. Reduction of sugary drinks intakes is therefore an ideal target for dental caries and obesity prevention strategies.

Reducing sugary drink consumption is unlikely to do harm, even if achieved through a switch to low energy drinks. A randomised-controlled trial showed that children rated similar satiety levels after drinking sugar-sweetened soft drinks, compared with their responses after drinking sugar-free alternatives.\textsuperscript{24} This suggests that swapping sugary drinks for sugar-free versions will neither adversely affect children’s appetites, nor their intake of other food.

One way to reduce sugary drinks intakes is by raising the price of sugary drinks,\textsuperscript{25} such as through taxation. Recent econometric modelling has determined that a 20% sugary drink tax could result in a 4–5% reduction in the prevalence of obesity.\textsuperscript{26} In the UK, a tax on sugar-sweetened drinks has been proposed to reduce the prevalence of obesity.\textsuperscript{27} Other strategies include restricting the marketing of sugary drinks to children through regulation.\textsuperscript{28}

We, the delegates of this conference, endorse the view that sugary drinks in New Zealand and the Pacific region should be treated in the same manner as tobacco, with a view to eventually phasing these products from our food supply (<5% of total beverage intake).

Measures to reduce the intake of sugar which we endorse include:

- An excise tax on drinks containing sugar.
- Advertising restrictions applied to the marketing of sugary drinks (particularly to children).
• Health promotion and social marketing campaigns to increase public awareness of the negative health consequences of sugary drink intake.
• Sales restrictions, limiting purchases of sugary drinks only to people aged greater than 15 years.
• Policies to reduce sugary drinks availability in public sector settings and workplaces.

We encourage the development and promotion of sugar-free alternatives, including drinks which contain low energy sweeteners, or are unsweetened.

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Healthiness of popular fast food items in New Zealand: plenty of room for improvement

Fast food provides an increasing contribution to dietary energy and nutrient intakes in New Zealand (NZ). A recent (2014) World Health Organization (WHO)-commissioned study found that per capita fast food consumption in NZ increased 10% between 1999 and 2008; one of the sharpest increases in intake of all countries in the Organization for Economic Development (OECD).\(^1\) Such an increase is likely to be associated with ease of access given that the median distance to a fast-food outlet is less than 1 km.\(^2\)

The rise in fast food availability and consumption is concerning because this food is generally high in fat, sugar, sodium, and energy, and high intakes are associated with increased body mass index (BMI) and obesity risk.\(^1,3\) As such, there have been calls for guidelines around the nutrient composition of fast food and availability of healthier options.\(^4\)

We undertook a survey of the most popular items available for sale at NZ fast food restaurants to determine their mean nutrient content and contribution to recommended daily intakes (RDIs) of energy, saturated fat, sugar, and sodium. We included the four fast food chains with the highest number of outlets in NZ: McDonalds (McD; n=212 restaurants), Kentucky Fried Chicken (KFC; n=98), Pizza Hut (PH; n=84) and Burger King (BK; n=80). The most popular fast food items were determined using an online survey (n=104 NZ adults; January 2014) asking about fast food consumption over the past month. The nutrient content of fast food items was determined using the 2013 version of Nutritrack, a brand-specific supermarket and fast food composition database.\(^5\) Burgers, pizza, chicken, sandwiches, salads, sides/other, and beverages sold for lunch and dinner were included.

Our analysis showed that burgers (n=46) had the highest mean (SD) energy (2242 [709]kJ), saturated fat (8.6 [5.8]g), sugar (7.0 [3.1]g) and sodium (1063 [345]mg) content per serve. The most popular burger combo meals and pizza would contribute between one-third and a half of the adult RDI for energy and nutrients (Table 1). Combo meals provided at least 94% of the RDI for sugar when applying the new WHO guideline (5%RDI). Our findings are conservative because they focus on regular-size combo meals and pizza without large or extra-large portion size options.\(^6\)

Three of the four restaurants offered salads (BK, KFC and McD), which had the healthiest nutrient profile of all main menu items in terms of mean energy (305kJ), saturated fat (0.5g) and sodium (143mg) per serve (mean serving size 279g). However, on average they contained more sugar per serve (5.5g), than pizza (mean 3.1g), chicken (0.9g) and sides (2.3g). Further, the mean (SD) range in sodium content of salads available at different chains was wide, ranging from 133 (172)mg per serve at KFC to 967 (809)mg per serve at BK.

Nonetheless, salads were not popular with survey respondents: no-one indicated they had consumed a salad from one of the four fast food chains in the past month.
Table 1

<table>
<thead>
<tr>
<th>Combo Meals(burger, fries, soft/fizzy drink)</th>
<th>KFC (n=2)</th>
<th>Male</th>
<th>25-28%</th>
<th>5-6%</th>
<th>35-36%</th>
<th>100-101%</th>
<th>51-62%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td>34-38%</td>
<td>7-9%</td>
<td>48%</td>
<td>135-136%</td>
<td>51-62%</td>
</tr>
<tr>
<td>McD (n=4)</td>
<td>Male</td>
<td>23-30%</td>
<td>8-18%</td>
<td>33-35%</td>
<td>94-98%</td>
<td>50-62%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>31-41%</td>
<td>11-26%</td>
<td>44-47%</td>
<td>127-132%</td>
<td>50-62%</td>
<td></td>
</tr>
<tr>
<td>BK (n=4)</td>
<td>Male</td>
<td>26-41%</td>
<td>11-20%</td>
<td>36-49%</td>
<td>102-138%</td>
<td>32-76%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>35-54%</td>
<td>16-28%</td>
<td>48-65%</td>
<td>137-185%</td>
<td>32-76%</td>
<td></td>
</tr>
<tr>
<td>Pizza (3 slices)</td>
<td>PH (n=4)</td>
<td>Male</td>
<td>17-20%</td>
<td>15-19%</td>
<td>5-8%</td>
<td>13-24%</td>
<td>49-68%</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>23-26%</td>
<td>22-27%</td>
<td>6-11%</td>
<td>18-32%</td>
<td>49-68%</td>
<td></td>
</tr>
</tbody>
</table>

a. RDI values used include (for males/females respectively): energy 13,300/9900kJ; saturated fat 42.3/31.5g; sugar 117.4/87.4g; sodium 2,300mg.

b. WHO guideline for sugar intake (5%RDI).
Our results illustrate that there is plenty of room for improvement in the nutrient composition of fast food items in NZ. In addition, our data show that some popular items were considerably lower in saturated fat, sodium, and sugar than others, illustrating that it is possible to improve nutrient composition.

The major limitation of this study is the relatively small sample size (n=104 adults) used to determine the most popular fast food items. Electronic sales data, or data collected from the till receipts of consumers across a range of fast food restaurants would have been much better for this purpose.

Unfortunately, the fast food chains we surveyed refused to allow us to collect till receipt data from customers at their stores indicating some hesitance to cooperate with public health research. This analysis indicates the need for regulation, in particular around the nutrient composition of fast food and availability of healthier options.

International experience suggests that voluntary initiatives led by industry often may not have the intended results (a study conducted by the Yale Rudd Centre found that 97% of studied kids fast food meals failed to meet even the industry’s own Children’s Food and Beverage Advertising Initiative’s nutrition standards). Should this be the case for NZ then stricter government-led policies and guidelines should be introduced to achieve adequate public health outcomes.

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Employment, poverty and health: ideology or science?

Since July 2013 when the New Zealand Government’s benefit ‘reforms’ saw the replacement of sickness and a proportion of invalid benefits by a single job-seeker support, GPs have been signing Work Capacity Medical Certificates bearing a quotation regarding the health benefits of work from the Position Statement on that subject from the Australasian Faculty of Occupational and Environmental Medicine (AFOEM), which is running a campaign on this theme.

Those GPs who have been inducted by the Ministry of Social Development (MSD) as ‘designated doctors’ are meanwhile being exposed to the scientifically more dubious claim that long-term benefit dependency is a cause of ill health. There are several reasons why GPs should be skeptical of this attempt to manipulate them into disentitling claimants to disability support.

Firstly there is clearly an alternative hypothesis to account for the statistics of ill-health amongst beneficiaries, and that is poverty, the health impacts of which get no mention whatsoever in the AFOEM document (or in the work assessment application form!)

Included amongst the statistics adduced by the AFOEM to support its claim that unemployment causes ill-health are statistics of ill-health of children! Of the competing hypotheses, poverty is clearly the one that best accounts for this, and if it wholly accounts for it amongst children, it would be perverse to maintain that it did not do so for adults also.

The same Government that has (for reasons entirely different from any supposed health benefits) vowed to reduce benefit numbers by 40,000 has meanwhile denied that child poverty is as deep and widespread as its critics claim. Moreover it has now been revealed that the Deputy Prime Minister has been concealing a major underestimate of the true extent of child poverty. As long as it can claim support from the medical profession by appearing to address the poorly substantiated health effects of benefit dependency, the more likely the Government is to continue in its state of denial regarding any link between poverty and illness.

Increases in illness resulting from the higher levels of poverty attendant on the draconian administration of benefits is likely to far outweigh any decreases obtained by the miniscule number of beneficiaries finding healthy employment in an economic recession.

Thirdly, foremost amongst the promoters of the ‘health benefits of work’ is the holder of an academic chair sponsored by the World’s largest disability insurer. Even worse, that insurer is one that has been thoroughly discredited in the United States courts for denying benefits to rightful claimants.

Surely it is time the medical profession objected publicly and loudly to being manipulated by Government and the corporate interests it transparently serves.
The allegations made above are substantiated by links in an Open Letter to the Royal College of Physicians published on http://www.waitemataunite.blogspot.com

See also http://nzsocialjusticeblog2013.wordpress.com/

**Keith Henderson**
Hon Secretary
Waitemata Branch of Unite Union
Henderson, Auckland, New Zealand
Diagnostic testing of blood donor specimens

Most clinicians working within the New Zealand public service are accustomed to using the National Health Index Number (NHI). This is a common number used to identify patients when they access different parts of the health system.

The New Zealand Blood Service (NZBS) uses an entirely different number when testing donor blood. The current IT systems in NZBS have been set up so that they do not allow the NHI to be used. This number is not available to other clinicians and very few clinicians are aware of its existence.

Donors are informed that their information will not be made available to other agencies. When donor blood is tested, if abnormalities arise, the NZBS medical team will inform donors and will recommend that this information be provided to the GP and where appropriate seek consent to contact the GP to provide additional information. In the event that test results that might impact on future transfusion requirements then NZBS seeks permission from the donor to include the information on the patient side of the database (Peter Flanagan, Medical Director, NZBS, Personal Communication).

It is not clear how many clinicians in New Zealand know that test results from NZBS are not routinely available. Our reliance on the NHI as the index for all laboratory results may mean that abnormal tests or clinically significant changes in results ordered by NZBS go unnoticed. GPs and other treating clinicians may not know if their patients are regular donors and are very unlikely to know if there are relevant test results in an entirely different database.

This letter is to make all clinicians aware of the potential risk. To the best of my knowledge no significant adverse effects of this system have been recorded. I also hope to encourage debate about the ethical obligations to donors who potentially may be disadvantaged by the current system.

David Spriggs
Physician, Auckland District Health Board
Auckland, New Zealand
Public attitudes to new smokefree outdoor places policies in New Zealand: an analysis of 217 online comments

Background—There is very limited evidence in the qualitative literature about the reasons the public think smoking should or should not be allowed in outdoor areas. One study in Toronto, Canada found some smokers were more comfortable smoking away from non-smokers. Non-smokers reported discomfort, nuisance, health concerns and repugnance about cigarette butt litter.¹

In some media websites, readers can leave online comments alongside electronic news articles, and this has increased the ability of the public to comment on news articles.² There appear to be few published analyses of online comments provoked by tobacco-related news items. These have found conflicting views from smokers and non-smokers, as well as discourses around quitting, rights and evidence.³

Because of the restricted qualitative evidence about attitudes to them, we aimed to identify what themes could be found in online discussions provoked by news articles on smokefree outdoor public areas in New Zealand, and to explore the potential utility of this data source for public health research more generally.

Methods—Using the Factiva media database we searched for online public responses to New Zealand newspaper stories, from 1 June 2012 to 31 January 2013, that described possible smokefree outdoor policies. Using the search words ‘smoking’, ‘outdoor’ and ‘policy’ we found 10 such articles with accompanying online discussions, with a total of 375 online comments. Comments were excluded from analysis if they: (a) only concerned a total ban on tobacco smoking in New Zealand (not just outdoors), (b) focused only on critiquing/heckling other commenters or (c) only concerned another issue, for example air pollution from traffic.

The remaining relevant comments were coded and themes and sub-themes identified. For the relevant comments, the author’s support or opposition to the proposed smokefree policies, or whether they appeared to be neutral or unclear on their position, was determined.

Results—All 375 comments identified were relatively concise (mean: 79 words, range: 1–247 words). There were 217 relevant comments (58% of the total 375 comments). Of the authors who posted relevant comments, 41% appeared to support outdoor smokefree area policies, 48% opposed and 11% were either unclear in their sentiments or appeared neutral.

Four major theme groups emerged, with many associated themes and sub-themes:

- The first theme group consisted of concerns about smoking in public, including health issues, normalisation of smoking, the risk of cues for ex-smokers to smoke, pollution from tobacco smoke and repugnance towards smoking.
- The second major theme group supported or doubted the scientific evidence that smoking and secondhand smoke (SHS) harms human health. Myths were
commonly articulated; in particular that exposure to SHS is harmless and easily avoidable.

- The third theme group highlighted perceived rights in society and associated justice or equity issues. Many felt ‘people have the right to smoke in public if they choose’. Conversely many others felt ‘everyone has the right to clean air’.

- The fourth theme group concerned the appropriateness of proposed smokefree policies. Opinions ranged from viewing these measures as overly restrictive, to just right, and to not restrictive enough. The practicality of implementing the policies was a strong theme, and some commenters made suggestions on how to go about implementation. Smokefree policies in other parts of the country and overseas were frequently referred to.

Beyond the material in themes 2 and 3, there was considerable antagonism shown by commentators. Around half the commenters appeared to have negative attitudes towards smokers.

**Discussion**—The study of online comments appears to be a useful way to identify major themes relating to public knowledge and attitudes, in this case on smokefree outdoor area policies. Ideally, such comments would be used in combination with other data sources such as content analysis of media, and in-depth interviews or focus groups with key informants and the public. Quantitative studies (e.g., surveys) should also be considered in order to comprehensively understand the key drivers and barriers to new outdoor smokefree policies. However, as a qualitative data source, online comments have multiple advantages, including easy access, large volumes, and relative lack of inhibitions compared to other sources of opinions.

Because of the proportion of negative online comments, the politics around smokefree outdoor policies may be influenced away from the direction of majority opinion by the visibility and prominence of opposing views. The themes found could allow advocates and policymakers to plan for or take advantage of the responses. They have emerged widely in debates about smokefree place policies.4–6

Advocates and policymakers in New Zealand need to be aware of the very strong ‘rights’ discourse around smokefree outdoor places policies.7,8

A detailed report on this study is freely available online at [http://www.otago.ac.nz/wellington/otago067456.pdf](http://www.otago.ac.nz/wellington/otago067456.pdf)

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References:

Government Insurance Bill

Excerpt from Editorial published in 1912 March issue of the NZMJ.

There is a matter which directly affects the medical profession and which is engrossing its attention in the Old Country which deserves on an occasion like this a passing reference.

It is, as very many guess, the Government Insurance Bill. The object of this Bill is, apparently, to confer distinct benefits on the public, and no doubt this laudable object would be attained if the subject were dealt with in a proper manner; but by attempting to compel treatment by the medical profession at a starvation rate, no good can be done to the recipients and a great deal of harm will be inflicted on the profession.

Dr. James Milward writing to the Times recently, says:—"Cheap doctoring is the dearest luxury in which anyone can indulge." I agree with him that nothing is more objectionable and injurious, and anyone doubting what I say may seek evidence in support of it and probably find it without much difficulty.

Medical practice to-day is scientific, and does not consist in writing a hasty prescription, but involves a careful examination, resulting in most cases in an accurate diagnosis and intelligent treatment. And how, I ask, can this be done, including visiting at such a ridiculously low remuneration?

I will quote Dr. Milward again: "This system will continue to exact its annual preventable toll of lives, and unnecessarily prolonged illness of bread-winners as the penalty for ignoring what would be a platitude in any other walk of life—that you cannot buy a pound's worth of work for a shilling—"
Emergency and stroke physician combined consensus statement on thrombolysis for acute stroke

Annemarei Ranta, John Bonning, John Fink, Dominic Fleischer, John Gommans, Peter Jones, P Alan Barber, Michael Ardagh

Abstract

The New Zealand Faculty of the Australasian College for Emergency Medicine (ACEM) is the professional body representing the specialist emergency medicine physicians who work in and lead emergency departments of New Zealand. The National Stroke Network Leadership Group represents New Zealand stroke clinicians including stroke physicians and neurologists who work within and lead district health board (DHB) stroke services.

In an effort to promote their shared goal of ensuring patient safety while striving to achieve improved stroke outcomes, the two communities have set up a consensus group to develop this combined emergency physician and neurologist/stroke physician consensus statement on the use of intravenous alteplase in stroke (‘stroke thrombolysis’).

The New Zealand Faculty of the Australasian College for Emergency Medicine (ACEM) is the professional body representing the specialist emergency medicine physicians who work in and lead emergency departments of New Zealand. The National Stroke Network Leadership Group represents New Zealand stroke clinicians including stroke physicians and neurologists who work within and lead district health board (DHB) stroke services.

In an effort to promote their shared goal of ensuring patient safety while striving to achieve improved stroke outcomes the two communities have set up a consensus group to develop this combined emergency and stroke physician consensus statement on the use of intravenous Alteplase in stroke (‘stroke thrombolysis’). Once the statement was produced by the consensus group, wider emergency and stroke physician community consultation confirmed majority support.

Consensus Statement:

- Stroke thrombolysis with intravenous alteplase is applicable only to a minority of stroke patients and should be seen as a treatment option indicated in carefully selected stroke patients.

- Patients should be selected in accordance with agreed protocols with explicit inclusion and exclusion criteria. These protocols should be aligned with the published literature and established collaboratively between emergency physicians, neurologists, stroke physicians, and other relevant stakeholders. Treatment outside of agreed criteria might increase the risk of adverse outcomes.
• The strongest evidence for benefit of stroke thrombolysis is for patients treated within 3 hours of stroke onset. Emergency physicians, neurologists, and stroke physicians should work collaboratively to minimise treatment delays.

• This group was unable to reach a consensus about the utility of stroke thrombolysis between 3–4.5 hours of symptom onset.

• Inpatient stroke team pre-notification of a potential thrombolysis patient’s arrival is encouraged to facilitate rapid patient assessment and to assist with potential resource constraints in emergency departments.

• Appropriate infrastructure should be present including timely access to neuro-imaging (CT scanning) and timely interpretation of these scans prior to thrombolysis by consultant radiologists, neurologists, or stroke physicians/delegated radiology registrars with appropriate expertise and training.

• Informed consent for thrombolysis should be discussed and obtained by a medical specialist or delegated registrar with appropriate expertise and training in stroke assessment/management and with a sound knowledge of the benefits and harms of stroke thrombolysis.

• There should be appropriate care and documentation of progress after thrombolysis including recording of vital signs and neurological observations according to agreed protocols.

• Patients should be expeditiously transferred to a designated intensive monitoring ward area or Stroke Unit (ideally immediately following neuro-imaging prior to or immediately after administration of thrombolysis).

• Services who provide thrombolysis should audit their service regularly to monitor safety and measure outcomes. Audit results should be reported routinely to local clinicians and regional stroke networks. Ideally a national database should be established to audit all stroke patients, including individuals treated with thrombolysis, to measure outcomes, with the results made available to all clinicians involved in the care of stroke patients.

• The ongoing appropriate use of thrombolysis in stroke should be reconsidered as the results of audits, or further research, become available.

This group will continue to collaborate to improve stroke care for patients in New Zealand.

Combined Stroke Thrombolysis Consensus Group Members:

Dr Anna Ranta (Chair), Neurologist (MidCentral DHB)
Dr John Bonning, Emergency Physician (Waikato DHB)
Dr John Fink, Neurologist (Canterbury DHB)
Dr Dominic Fleischer, Emergency Physician (Canterbury DHB)
Dr John Gommans, Stroke Physician (Hawke’s Bay DHB)
Prof Mike Ardagh, Emergency Physician (Canterbury DHB)
Prof Alan Barber, Neurologist (Auckland DHB)
Dr Peter Jones, Emergency Physician (Auckland DHB)
GFR may not accurately predict aspects of proximal tubule drug handling
T Putt¹, S Duffull², J Schollum¹, R Walker¹

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Dose modification in renal impairment has traditionally been based on changes in estimated glomerular filtration rates (eGFR). However, many drugs are eliminated by tubular anionic and cationic transport where changes in eGFR may not necessarily reflect changes in tubular function. This study investigated the relationship between GFR and renal tubular function with reference to drug handling by using accepted drug probes.

Measured GFR (Cr⁵¹ EDTA clearance; mGFR) was compared to tubular anionic transport (urate clearance), tubular reabsorption (fluconazole clearance) and cationic transport (S-pindolol clearance). In addition, comparison was made to eGFR formulae in common use.

We demonstrated a strong correlation between mGFR and creatinine clearance and a moderately positive correlation between mGFR and proximal tubular anion transport and reabsorption. In contrast, cationic secretion correlated poorly with mGFR. The mGFR was shown to be significantly underestimated by eGFR calculations for those without established chronic kidney disease.

Given that drug-dosing schedules utilise eGFR values as the basis for modifying drug dosing, our results would suggest that a global recommendation of dose reduction according to eGFR alone should be treated with caution.

Erythromelalgia? A clinical study of people who experience red, hot, painful feet in the community
D Friberg, T Chen, G Tarr, A van Rij

Department of Surgical Sciences, Dunedin School of Medicine, University of Otago, Dunedin

Erythromelalgia is thought to be a rare debilitating condition affecting the distal extremities. Suffers have red hot, painful feet made worse with heat and improved with elevation and cooling. Little is known about the disease and diagnosis is made on clinic assessment because there are no definitive diagnostic investigations. We recruited a population of people from the Dunedin community who self-identify as having the symptoms of erythromelalgia to better characterise this population and measure their quality of life (QOL).
Ninety-two individuals completed the QOL surveys (SF-36 and the Otago Condition Specific Questionnaire), and 56 individuals were clinically assessed. There was a 3:1 ratio of females to males with an average age of 61 years (range 8–91). The estimated prevalence of individual with erythromelalgia in the Dunedin community was 15/100,000. Only 27% of people had received a diagnosis for their symptoms despite seeking medical attention. People in the study population had worse quality of life than the general New Zealand population ($p \leq 0.001$) and patient seeking varicose vein treatment in the public sector ($p \leq 0.001$). In the majority of participants symptoms had a mild-moderate effect on their quality of life.

The results of this study indicate that the number of people who have clinical symptoms of erythromelalgia is much greater than is commonly accepted and that the majority of these individuals go unrecognised by the medical profession despite seeking help. They have significantly diminished QOL with the majority of people having mild-to-moderate symptoms.

Sentinel node biopsy versus axillary lymph node dissection for breast cancer surgery in a provincial New Zealand hospital: An audit of morbidity

N Fischer$^1$, C Wee$^2$, S Aiono$^2$

$^1$Department of General Surgery, Dunedin Hospital, Dunedin. $^2$Department of General Surgery, Whanganui Hospital, Whanganui

Sentinel node biopsy (SNB) has reduced the need for routine axillary lymph node dissections (ALND) in clinically node negative patients with breast cancer. SNB has reduced the risk of morbidity associated with ALND, including lymphoedema, seroma and infection. SNB has been introduced into breast cancer surgical practice in New Zealand with satisfactory results compared with international standards. Routine use of SNB for breast cancer surgery was introduced in Whanganui Hospital in 2009. The aim of the audit was to compare the number of axillary dissections before and after the introduction of SNB for patients undergoing breast cancer surgery, and to review the complication rates for SNB alone versus ALND.

A retrospective audit was conducted examining patients’ clinical records and physiotherapy department records. All patients undergoing breast cancer surgery at Whanganui Hospital for 4 years from 2007 to 2011 were included. The number of SNB and ALND’s performed were recorded, as well as important outcomes measured comparing the incidence of seroma formation, infection, lymphoedema, chronic pain and haematoma between SNB alone versus ALND.

One hundred and seven patients (n=107) underwent breast cancer surgery; 39 patients before the introduction of SNB and 68 patients after. Thirty nine patients underwent SNB. Eighty-four patients underwent ALND. Eighteen patients had a negative SNB & did not require ALND (46%). The incidence of any complication in patients undergoing ALND was 52% The Incidence of any complication in patients undergoing SNB only was 28%. Ten percent of women who had ALND developed lymphoedema compared with no women in the SNB only group.
SNB can be adopted successfully to reduce the number of ALND’s and associated morbidity in a provincial New Zealand hospital for breast cancer surgery. Our complication rates are similar to those reported in the literature.
Dietary fibre intake and risk of cardiovascular disease

Numerous observational studies have reported greater fibre intake being associated with lower risk of cardiovascular disease, with others reporting no such associations. This systematic review evaluates this concept using a dose-response approach. 19 prospective observational studies with at least 3 years follow-up were included.

The researchers report that a lower risk of cardiovascular disease and coronary heart disease was associated with greater intake of total fibre, insoluble fibre, and fibre from cereals and vegetables. Greater fruit fibre was also associated with lower risk for cardiovascular disease. For each increase of 7g/day intake of fibre, separate risks for cardiovascular disease and coronary heart disease were each 9% lower.

It is noted that fibre intake could be a surrogate marker for other healthy lifestyle features. Also, dietary assessments are challenging, with measurement error being a particular difficulty.


Treatment of acute otorrhea in children with tympanostomy tubes

Tympanostomy tube insertion is indicated in children with persistent otitis media with effusion, which is often complicated by hearing problems. Acute otorrhea is a common sequel in children with tympanostomy tubes and is usually due to a bacterial infection. Treatment is therefore aimed at eradicating bacterial infection, with the options including broad-spectrum oral antibiotics and antibiotic eardrops with or without glucocorticosteroids.

This report concerns a trial in which 230 children, 1 to 10 years of age, who had acute tympanostomy-tube otorrhea, were randomised to receive hydrocortisone-bacitracin-colistin eardrops (76 children) or oral amoxicillin-clavulanate suspension (77) or to undergo initial observation (77). The primary outcome was the presence of otorrhea, as assessed otoscopically, 2 weeks after study-group assignment.

The eardrops were more effective than the other options. At 2 weeks, 5% of children treated with antibiotic-glucocorticoid eardrops had otorrhea, as compared with 44% of those treated with oral antibiotics and 55% of those treated with initial observation. The median duration of the otorrhea was 4 days in the eardrops group, 5 days in those treated with oral antibiotics and 12 days in the observation group.

Human papillomavirus (HPV)-based screening for prevention of invasive cervical cancer

Cytological screening for the prevention of cervical cancer is well established. Screening with HPV-based techniques is an alternative reviewed in this paper. Evidence from the four randomised trials comparing these techniques is evaluated. 176,464 women (aged 20–64 years) were randomly assigned to HPV or cytological testing. Follow-up was for a median of 6.5 years and 107 cancers were identified. Detection of invasive cancer was similar in both groups over 2.5 years but was significantly lower in the HPV-based group thereafter.

When the patients were reviewed at 3.5 and 5.5 years the results favoured the HPV cohort. The conclusion was that HPV-based screening provides 60–70% greater protection against invasive cervical carcinomas compared with cytology. The data support initiation of HPV-based screening from age 30 years and extension of screening intervals to at least 5 years.

Ian Ronayne, one of the earliest Residents at National Women’s Hospital, and later one of its Visiting Obstetricians and Gynaecologists, died recently, aged 93.

He was born in Wellington. He concluded his secondary education at Mt Albert Grammar School where he was a member of the Cricket First XI and the swimming team.

He graduated from Otago Medical School in 1947. During the war he served as a reservist in the Medical School Army Corps, spending a holiday on the Auckland Islands searching for enemy submarines and counting albatrosses!

Following house surgeon years in Auckland Hospital he spent two and a half years as a Registrar at the Cornwall (later National Women’s) Hospital Obstetric and Gynaecological Unit.

When he left to pursue his postgraduate studies in Britain, the senior medical staff gave him an Omega watch.

On arrival in London, in 1953, he contacted the Royal College of Obstetricians and Gynaecologists seeking assistance in obtaining a training position. To his surprise, that same evening, he was phoned by Mr Joe Wrigley (of forceps fame) offering him an immediate position at St Thomas’s Hospital. This included duties at the old General Lying In Hospital, one of London’s oldest maternity units. Later he moved to St Luke’s Hospital in Guildford as a registrar, working with his friend and surgical colleague, Keith Ewen, later to be his best man. While in Guildford he met his future wife, Veronica—a ward sister.

Ian passed his MRCOG in 1956, became a FRCOG in 1972 and a Foundation Fellow of the RNZCOG (later RANZCOG) in 1992.

He returned urgently to New Zealand in 1957 to take over the practice of Dr Fred Smale, Auckland’s busiest obstetrician at that time. He charged five guineas plus GMS for full obstetric care. He was soon appointed to a Visiting Obstetrician post at St Helen’s Hospital and became heavily involved in midwife training, once claiming ‘half the midwives in New Zealand were trained by me’. His contributions were marked by a plaque at the hospital.

Later he was appointed to a Visiting position on ‘B Team’ at National Women’s Hospital, working with Bruce Grieve, Bernie Kyle and Ron Elvidge in the gynaecology team. He had a close working relationship with Pat Dunn at the St Vincent’s Home for unmarried mothers whose babies were for adoption. They
delivered over 2000 babies. Pat became a Papal Knight, and Ian received a Papal Apostolic Blessing with its ‘pledge of heavenly favours’. Ian also worked with Pat Dunn in the Diabetic Clinic at National Women’s Hospital, co-authoring a 1983 paper ‘Fructosamine in Diabetic Pregnancy’, in The Lancet.

Ian was a practical, no-nonsense specialist: countless Residents enjoyed his forthright opinions. He was possibly the most conscientious obstetrician and gynaecologist of his era—refusing to take a holiday when any of his obstetric patients was due. He was immensely proud of his association with the Postgraduate Department, then in its heyday of research achievements.

He shared a clinic with Bill McIndoe, and more than anyone, provided friendship and support in the years Bill was battling the ‘Unfortunate Experiment’.

Ian’s move into retirement was gradual and he finally gave up practice in 1990. His diminishing workload gave him more time for leisure activities. Family issues to one side, his principal interest was golf at the Akarana Golf Club where, at an earlier time, he played off a handicap of 8.

We extend our sympathy to his three children (Richard, Clare and Felicity) who were at his bedside when he died.

Ron Jones wrote this obituary.