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**Vitamin D deficiency in pregnant New Zealand women**
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Vitamin D is a key factor in bone development and is primarily produced in the skin from sunlight exposure. The term “rickets” describes the bone abnormalities associated with abnormal mineral deposition in the growing skeleton. This study investigated the prevalence of vitamin D deficiency in pregnant women of a Wellington general practice population where 10 cases of childhood rickets had been diagnosed over the past 3 years. It showed 61.2% of women in this multicultural population had severe vitamin D deficiency. All vitamin D deficient women in the study were treated. This study highlights the magnitude of vitamin D deficiency in the pregnant population in a New Zealand setting; this vitamin D deficiency is responsible for the re-emergence of childhood rickets.

**Reliability of ultrasound estimation of fetal weight in term singleton pregnancies**
A Colman, D Maharaj, J Hutton, J Tuohy

By measuring ultrasound images of a fetus, it is possible to estimate the baby’s weight at birth. The purpose of this Wellington Hospital study was to assess the reliability of this ultrasound measurement in pregnant women at 37 or more weeks. 1177 babies were studied. The accuracy of ultrasound estimations was at least similar and sometimes better than that reported in other studies. For one in four women, however, the estimated fetal weight was more than 10% different from the actual birth weight of the baby—ultrasound measurements had a tendency to overestimate the weight of small babies while underestimating the weight of both large babies and the babies of diabetic mothers. As the reliability of ultrasound estimation to detect larger babies was poor, its use in predicting macrosomia (large babies) at birth should be avoided.

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# The few: New Zealand's diminishing number of rural GPs providing maternity services

Don Simmers

Recently, the Royal New Zealand College of General Practitioners (RNZCGP) calculated there were 54 general practitioner obstetricians (GPOs) still providing intrapartum care within New Zealand's maternity system.[1] To many, including the author, this came as a surprise.

For the last few years, estimates have always put the number somewhere between 10 and 20 with the inevitable caveat that many of those left had given firm indications they too would stop in the near future. Although 54 is still a pitifully small number (and, predictably, soon to reduce to 52), the vital part they play in delivering maternity care, particularly in rural areas, must be recognised. Questions again need to be asked about whether New Zealand's maternity system is in any position to let this once proud cornerstone disappear altogether.

Before this statement is dismissed as misty-eyed nostalgia, a few facts need to be considered:

- Of the 55,000 women who give birth annually, just under a third live in rural areas.[2]
- Many rural non-GPO GPs are continuing to experience emergency obstetric situations where action is required immediately to either effect an immediate delivery or stabilise a clinical event prior to transportation.

  Rural GPs caught in these situations are often able to manage because of their previous experience in delivering obstetric care—but within a decade, all of this knowledge will be gone and no expertise will be available to attend these often perilous events.

  These situations are occurring because the:

  - Woman's lead maternity carer (LMC) is genuinely unavailable;
  - LMC lives in another (larger) town
  - LMC does not have the secondary care skills required for such emergencies.

  In contrast to GPs, LMC midwives cannot survive in many smaller locations simply because there is not enough work for them. Also, LMC midwives have been reluctant to take on women with medical problems who will inevitably require base hospital delivery.

  Some enlightened district health boards (DHBs) have recognised these problems and are paying GPs to look after their antenatal patients in a shared care arrangement with hospital maternity services. Sadly, however, many

more DHBs continue to struggle with their current lack of understanding of what is required in primary care.

- There is a workforce crisis among New Zealand's provincial hospitals where there are shortages of obstetricians, anaesthetists, and paediatricians.[3,4] There are now many provincial centres (e.g. Wanganui, Greymouth, Invercargill, Masterton, Gisborne) where at best specialist cover is less than ideal and at worst is imminently threatening closure of secondary care maternity unit services. Are rural women really prepared to accept this previously unthinkable situation?

Many of the remaining LMC GPOs work in areas distant from secondary services, or where obstetricians are already in short supply. Despite Section 88 payment rules discriminating against them for doing so, they continue to look after their own patients—however it is in their role of providing elements of secondary care to other LMCs' patients where they are of irreplaceable value to a local service.

Because of this availability, birthing women have more confidence in the service and are more willing to use it. Queenstown is a classic example of what can be achieved with some local expertise and what is not achieved when that expertise is absent. In 1990, there were over 100 deliveries performed locally and in 2003 only 31 deliveries were performed.[2] Given the continuing population explosion in this area, if the 1990 conditions were rekindled, the annual local birthing rate would be well over 200.

Evidence has always supported women birthing in their own communities, as opposed to travelling either acutely or electively to a distant major centre.[5] Ironically, the evidence even includes studies from New Zealand,[6] but most of it comes from Canada,[7] and Australia[8] where the problems of providing rural and provincial obstetric services have been similar to ours.

In response to this research, these first world health systems have (over the last 5 years) embarked on programmes to upskill generalists in provincial areas giving them sufficient skills to perform instrument-assisted deliveries, caesarean sections, and neonatal and maternal emergency care.[9,10]

Of greater importance, however, is the acquisition and maintenance of decision skills around referral to base hospitals these generalists bring to provincial and rural maternity units. Research again has determined that while peripheral units that have access to these sorts of skills thrive and prosper, "high outflow" units, where there is not good support by the local population, wither and die.[5]

Work is currently underway in New Zealand to develop a vocational training programme that will result in specialist recognition for rural hospital doctors. So far, there has been no suggestion of including a maternity care skill set in the training programme. Clearly, this needs to happen.

But of even more urgency is the need for our health system to recognise and reverse the neglect it has shown towards the provision of maternity services in rural and provincial areas compared with other countries. This neglect has resulted in needless disruption to young couples' lives, unwarranted cascades of intervention, and (on rare occasions) the death of a neonate.

This change in attitude has to begin with abandoning the deliberate handicapping of GPOs through the Section 88 framework. Instead, these 52 GPOs, who have battled for a decade against bureaucratic indifference and the anti-doctor mindset of our health system, need to be congratulated and encouraged to continue.

The best encouragement of all for the Churchillian few would be the development of a new framework that would inspire and encourage doctors in training to pursue a career that includes provision of primary maternity care, the acquisition of a few technical skills that until now have been considered the preserve of secondary care, and the ability to know when to refer.

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# New Zealand's labour resources in general practice—should we worry?

James Reid

It was reassuring to hear the Minister of Health (when opening the *Education and Research in Rural Health Conference* in Dunedin recently) state that "there is no shortage of general practitioners in rural areas in this country."[1] (In fact, he was sick of being told that there was a problem.) It is worrying to know that his opinion is in isolation, however.

Indeed, at the same conference, Professor Paul Worley (Director of Flinders University Rural Clinical School, Adelaide, Australia) after a study tour of the rural teaching facilities of the Dunedin School of Medicine, stated that the situation in rural areas was always one doctor short of a crisis.[2]

While it is reassuring for the citizens and current general practitioners of Levin, Kapiti, Timaru, Gisborne, Waimate, Twizel, and the entire West Coast to know that the current Minister of Health does not think there is a shortage of doctors in their areas, it is worrying to know that he is wrong, however.

While absolute doctor numbers are static in rural areas, and generally have not declined, the face of general practice as a discipline has changed and will continue to change. With increasing compliance requirements on general practice from the bureaucrats, increasing complexity of presenting illness, increasing age of patients with comorbidities, and increasing patient expectations, the "short" or "one problem" consultation has become a rarity.

The requirements of doctors in the 21st Century have also changed—no longer is it acceptable to be on call 24 hours a day, 7 days a week; no longer is it acceptable to work excessive hours to absorb demand; and no longer is it acceptable to work all night on call, and be expected to work a normal day following.

Yes, the wind of change has blown through, with increased expectations from young doctors of a "normal life" with adequate remuneration. In addition, a change in gender balance in the profession, with greater than 50% of graduates now being female, has added to this stance.

It is generally agreed that the general practice workforce is aging. For example, 73% of rural GPs are older than 40 years[3] and 60% of all GPs in New Zealand are over 46 years of age, with 37% being over 50. Even more alarming is the fact that more than 33% of the current workforce intend to move out of general practice within the next 5 years.[4]

Currently, 12 years are required before a medical student entering medical school today can practise general practice independently, so even if numbers entering medical school are increased, there will be a considerable time lag before any correction will occur.

In addition, there has been am alarming decline in the number of New Zealand students wanting to enter general practice. In the past, about 50% have become general practitioners, but this has declined in recent years, with many students perceiving GPs to have low status and pay, increasing paperwork, and general practice providing less stimulation overall than hospital medicine.[5] Large student loans are also a factor.

Current GPs are concerned about never-ending change, bureaucracy, poor earnings, time pressures, lack of adequate resources for patients, threat of litigation, and burnout.[6] Thus there is evidence that the general practice workforce is diminishing more quickly than it is being replenished. Indeed, there is direct evidence of this trend, with numbers of practising GPs declining by 249 between 2000 and 2002.[4]

One critical issue is the number of visits a patient makes to the doctor each year.[7] With the evolving *Primary Health Care Strategy* resulting in lower GP fees, it is likely that the number of visits by each individual patient will rise. If these visits generally rise above an average of four/year, than there will be an enormous increase in workload.

A large number of our doctors, especially in rural areas, are overseas trained and often come from developing countries which can ill afford to lose them. In addition, New Zealand GPs are being wooed across the Tasman with offers of conditions and salary that New Zealand GPs can only dream about.[8]

The problem is compounded, especially in rural and provincial regions, with GPs becoming too busy to cope, and as a result they leave for the cities with reduced workload.

Is there a solution? Unfortunately there is no quick fix, and denial by the Minister that a reality exists is not part of this. General practice must be made more attractive to young graduates. If it is to compete with hospitals, the status and pay of GPs (although it has been improved over the last 2 years) needs to be addressed as does workload.

As with hospital doctors, paid study leave and sabbatical leave should be available. Relief is required for GPs to take holidays—currently 6 weeks for hospital doctors! The after hours and on call situation also needs to be remedied.

The increase in funding for primary health (after neglect for so many years) is to be applauded, but more needs to be done if a crisis is to be averted.

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# Vitamin D deficiency in pregnant New Zealand women

Annie Judkins, Carl Eagleton

**Abstract**

**Aim** This aim of this study was to identify the prevalence of vitamin D deficiency in pregnant women of a Wellington general practice where 10 cases of childhood rickets had been diagnosed over the past 3 years.

**Methods** Ninety pregnant women were screened for vitamin D deficiency by measuring 25-hydroxy vitamin D by DiaSorin radioimmunoassay. Recruitment into the study was over a 12-month period. A second appointment was arranged for clinical review and drawing of blood for parathyroid hormone, adjusted calcium, and alkaline phosphatase.

**Results** 100% of women presenting to the general practice for antenatal care consented to the study. 87% of women had 25-hydroxy vitamin D levels below 50 nmol/L. 61.2% of women had a vitamin D level below 25 nmol/L consistent with severe vitamin D deficiency. 10 women had an elevated parathyroid hormone consistent with secondary hyperparathyroidism. Only 22% of our patients were veiled, and included a diverse ethnic population, including African, Maori, European, Middle Eastern, and Polynesian women.

**Conclusions** Vitamin D deficiency is common in young pregnant women in this general practice, and it was not only confined to veiled women or women with dark skin. This highlights the magnitude of vitamin D deficiency in the pregnant population in a New Zealand setting; this vitamin D deficiency is responsible for the re-emergence of childhood rickets.

Vitamin D deficiency (and consequently rickets) is re-emerging as a major primary health care and public health issue throughout the world.[1–5] Although there is no true international consensus on the best ways to treat vitamin D deficiency, it is acknowledged that the prevalence of vitamin D deficiency and its associated morbidities are higher than previously thought worldwide.[6–11]

Nozza and Rodda from their review of children with rickets in Melbourne, Australia have recommended pregnant women with dark skin pigment or “veiling” should have their vitamin D level checked. If vitamin D levels are low (<50 nmol/L) then supplementation should be given to the mother as well as supplements to the breast feed infants of deficient mothers.[3]

During pregnancy and lactation, the current consensus is that the vitamin D status of the infant is strongly influenced by the vitamin D status of the mother during pregnancy. During pregnancy, 80% of the fetal skeleton is mineralised in the third trimester, so maternal adaptations to fetal calcium demands are most important in the third trimester. The major adaptive process in humans is a two-fold increase in maternal intestinal calcium absorption, mediated by increases in 1-25 dihydroxy vitamin D. Therefore the level of 25-hydroxy vitamin D and the parathyroid hormone

levels are felt to give the best indication of the body's balance of vitamin D during pregnancy.

Prompted by anecdotal reports of an increase in childhood rickets in South-East Wellington, we decided to examine the prevalence of vitamin D deficiency in a general practice population. Newtown Medical Health Services (NUHS) is part of a small Primary HealthCare Organisation (SECPHO) in South-East Wellington (latitude 41S) providing integrated medical and midwifery care for a population of approximately 7000 patients. Currently this includes a patient base of 437 children under the age of 5 years. Each year the number of births within the practice is approximately 120.

A review of their database for children under the age of 5 years identified 10 cases with a diagnosis of rickets over the last 3 years (Figure 1 and 2). Because of the re-emergence of childhood rickets in this practice, and in many countries around the world, a prospective clinical study was undertaken. We offered vitamin D screening for all pregnant women presenting for antenatal care to this general practice.

**Figure 1. Six-month-old child with rachitic changes showing expansion of the costochondral junction (arrowed)**

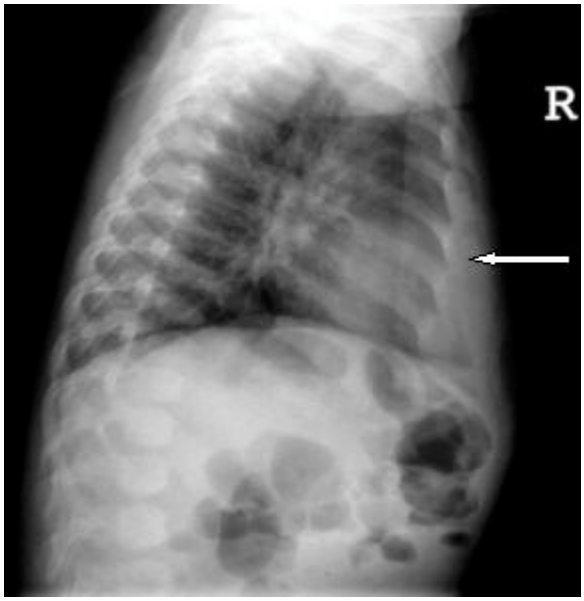
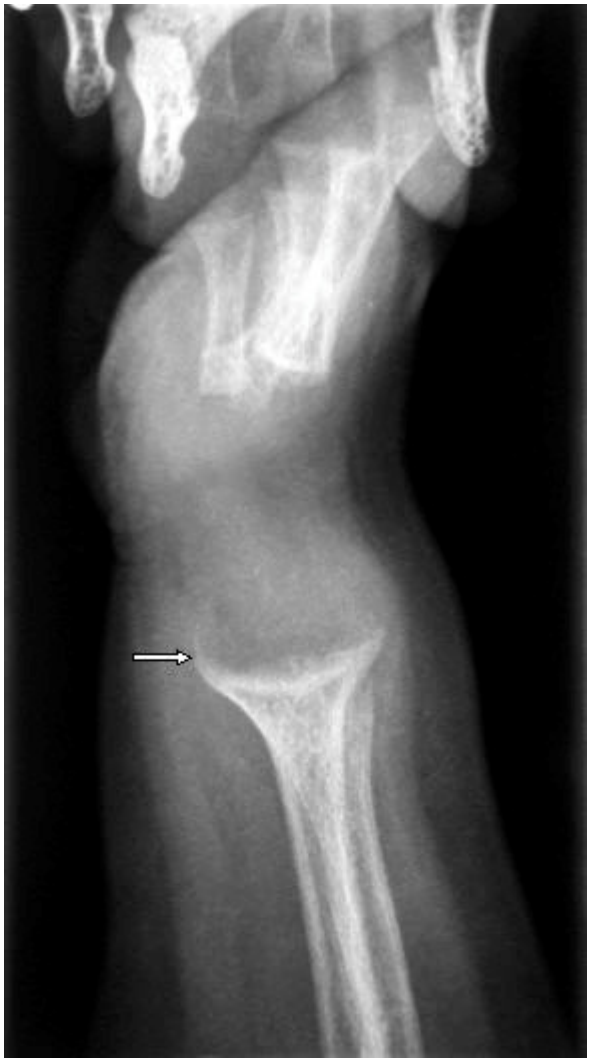


**Figure 2. Classic rachitic metaphyseal changes: cupping, fraying, widening, and fuzziness of the zone of provisional calcification immediately under the growth plate (arrowed)**



The study aim was to identify the prevalence of vitamin D deficiency in pregnant women of this small, busy primary health organisation (PHO). With an intention to treat vitamin D deficiency in order to prevent neonatal or childhood rickets in this population. We aimed to recruit 120 women to the study and replace vitamin D with ergocalciferol (vitamin D2) in those who were deficient from as early as 13 weeks gestation and continue this treatment throughout pregnancy and up to 6 months postpartum.

## Methods

In addition to the first antenatal bloods done routinely at commencement of maternity services, a 25-hydroxy vitamin D level was performed after consent was obtained. Each woman was asked to complete a simple dietary and sun exposure questionnaire. At the same time, an information sheet about vitamin D was provided and was available in several languages.

25-OH vitamin D was measured by acetonitrile extraction followed by DiaSorin radioimmunoassay (Stillwater, MN, USA). Patients with values less than 50 nmol/L were bought back for a further consultation and were considered to be significantly vitamin D deficient and offered replacement treatment.

At the second appointment, additional blood tests for parathyroid hormone (PTH), adjusted calcium, alkaline phosphatase, and phosphate were taken. Parathyroid hormone was measured by a Roche immunoassay (Basel, Switzerland). In pregnant women of 13 weeks gestation and over, with a vitamin D level of less than 50 nmol/L, ergocalciferol (vitamin D2) was prescribed as one 1,000 IU tablet per day. Follow up of the women later in pregnancy and in the postpartum period is the focus of the continuing prospective study. Daily supplementation of 400–1000 IU/day in pregnancy is felt to be safe.[1,12–16]

The study received ethics approval from the Wellington Ethics Committee. Vitamin D in the form of ergocalciferol (vitamin D2) was purchased from New Hope Nutrition Ltd (Browns Bay, Auckland, New Zealand). The only active ingredient was vitamin D as ergocalciferol (vitamin D2) 1,000 IU (release limits 900–1650 IU).

## Results

To date, of the 90 pregnant women that have been seen at Newtown Union Health, 100% consented to be study participants. The community of this general practice is multicultural and was reflected in the diverse ethnic groups included in the study. Of the 90 pregnant women, 78 (87%) were vitamin D deficient—with 25-hydroxy vitamin D levels below 50 nmol/L. (See Table 1 below.)

**Table 1. Pregnant women (n=90) screened at Newtown Union Health (Wellington) for vitamin D deficiency**

| Ethnicity | Number screened | Vitamin D deficient |
|---|---|---|
| Middle Eastern | 15 | 15 (100%) |
| Indian | 2 | 2 (100%) |
| African | 21 | 18 (86%) |
| European | 12 | 8 (67%) |
| New Zealand Maori | 10 | 9 (90%) |
| Samoan | 18 | 17 (94%) |
| Tokelauan | 1 | 1 |
| Chinese | 3 | 2 |
| Other | 5 | 4 |
| Cook Islander | 2 | 2 |

Two patients with vitamin D levels above 50 nmol/L had had previous children with rickets and had been treated with vitamin D replacement before pregnancy. 25-OH vitamin D levels ranged from <7.5 to 112 nmol/L in the study group; 61.2% of women had a 25-hydroxy vitamin D level less than 25 nmol/L, 24.4% between 25 and 50 nmol/L, and only 14.4% had a level of 50 nmol/L or greater. Seventeen of 39 women who returned for their repeat blood testing had an adjusted calcium below the normal range of 2.25 mmol/L.

Amongst the 78 vitamin D deficient women, 10 had secondary hyperparathyroidism with a parathyroid hormone (PTH) of greater than 6.0 pmol/L (1.5–6.0 pmol/L). All those with a PTH of greater than 9.5 pmol/L had a 25-hydroxy vitamin D level less than 12 nmol/L.

## Conclusions

Deficiency of vitamin D is common in this general practice's (Newtown Union Health's) population of pregnant women. This practice does serve a diverse population and will not be the same for all other New Zealand general practices. However some of the significant findings include the high incidence of vitamin D deficiency (in our study group) in New Zealand Maori, Samoans, and other Pacific Islanders.

The African, Middle Eastern, and Asian groups showed a very high incidence of vitamin D deficiency, which was perhaps not unexpected. Secondary hyperparathyroidism and hypocalcaemia due to the vitamin D deficiency were also common, reflecting the severity of the deficiency.

Previously described risk factors for vitamin D deficiency include veiling with traditional dress. This only contributed to a minority of cases in our series with only 22% wearing veils. Sunlight exposure may also be related to the ability to sit outside for periods of time but only 33 of the 90 women were living in apartments.

In a general practice with a re-emergence of rickets in children, this study highlights the potential magnitude of this preventable disease. All these women have gone onto receive treatment where they have elected to continue with the pregnancy. Further follow up on the efficacy of treatment is planned.

Because of the high incidence of vitamin D deficiency in this group of pregnant women we seriously believe that further population studies are needed. It is also important to make midwives, general practitioners, endocrinologists, and obstetric medicine physicians aware that vitamin D deficiency is common in the pregnant patient.

Indeed, in our study population, vitamin D deficiency was not only common in the veiled and dark-skinned patients but among all ethnicities in our pregnant general practice population.

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# Reliability of ultrasound estimation of fetal weight in term singleton pregnancies

Atalie Colman, Dushyant Maharaj, John Hutton, Jeremy Tuohy

**Abstract**

**Aim** To assess the reliability of ultrasound estimation of fetal weight undertaken antenatally at Wellington Hospital (Wellington City, New Zealand) in women with a singleton pregnancy ≥37 weeks gestation.

**Method** Data were collected retrospectively for pregnant women who had undergone ultrasound estimation of fetal weight <7 days prior to a term delivery (≥37 weeks gestation) over the period of July 1998–June 2005. Stillbirths and multiple pregnancies were excluded. Ultrasound fetal weight estimations, calculated using a locally modified Woo formula, were compared with the infant's actual birth weight.

**Results** A total of 1177 infants were studied. The mean absolute error and mean signed error (±SD) of ultrasound fetal weight estimations were 7.0±5.7% and -0.2±9.0%, respectively (n=1177). Three-quarters of estimations were within 10% of birth weight. Ultrasonic estimation of fetal weight tended to overestimate the weight of small infants (<2500 g; mean signed error = +3.5%±9.1%, n=98) and underestimate the weight of large infants (≥4000 g; mean signed error = -3.3±8.7%, n=170). Both large and normal weight infants of women with diabetes tended to have their weight underestimated (mean signed error = -5.1±9.2%, n=48).

Sensitivity, specificity, positive predictive value, and negative predictive value for ultrasonic detection of fetal weight ≥4000 g in non-diabetic women were 61%, 96%, 69%, and 94%, respectively. For detection of fetal weight ≥4500, the figures were 50%, 98%, 47%, and 98%, respectively.

**Conclusion** The accuracy of ultrasound estimations of fetal weight performed at Wellington Hospital within 7 days of delivery in term singleton pregnancies was at least similar and sometimes better than that reported in other studies. For one in four women, however, the fetal weight estimation was more than 10% different from the actual birth weight of their infant. Ultrasound measurements had a tendency to overestimate the weight of small infants while underestimating the weight of both large infants and the infants of diabetic mothers. As the reliability of ultrasound estimation of fetal weight to detect larger babies was poor, the use of such an objective measurement in the management of suspected macrosomia in term singleton pregnancies should be avoided.

The ultrasound estimation of fetal weight in term pregnancies is used to determine growth, and this may affect the timing and route of delivery.[1–4] Although antenatal care has focused more on the diagnosis of fetal growth restriction, the delivery of macrosomic infants is associated with higher rates of adverse outcomes for both mother and infant in comparison to the delivery of normal weight infants. Increased risks to the large infant include shoulder dystocia, brachial plexus injury, perinatal

asphyxia, and neonatal death.[5–7] Adverse maternal outcomes include prolonged labour, genital tract trauma, postpartum haemorrhage, and a higher rate of caesarean delivery.[6–8]

Whilst early delivery is the obvious management option for growth-restricted term or near term infants,[9] management of suspected fetal macrosomia is less certain.[2,10] Macrosomia has variously been defined as birth weight >4000 g, >4500 g or >90$^{th}$ centile for weight by gestation.[11] One of the causes of fetal macrosomia is maternal diabetes.[7,8] Ultrasound fetal weight estimations are undertaken as part of the routine management of pregnant women with diabetes. Ultrasound estimations of fetal weight are also undertaken in cases where there is a clinical suspicion of abnormal growth. The appropriate clinical response to an ultrasound diagnosis of macrosomia is unclear, in part because the predictions have been considered unreliable.[2,10,12,13]

The aim of this study was to determine the reliability of ultrasound estimation of fetal weight performed antenatally at Wellington Hospital in singleton term pregnancies delivered within 7 days of the ultrasound assessment, including the particular accuracy of the diagnosis of fetal macrosomia when defined as >4000 g or >4500 g.

## Method

Data were collected retrospectively for the 7-year period from 1 July 1998 to 30 June 2005. The study cohort consisted of term infants (≥37 weeks gestation) who had undergone an ultrasound estimation of fetal weight at the Wellington Perinatal Ultrasound Unit, and who delivered <7 days after the measurement. All multiple pregnancies and stillbirths were excluded. Estimated fetal weight data as recorded in the ultrasound database were matched with data derived from the hospital's Perinatal Information Management System, in which maternal and infant information, including the diagnosis of diabetes, was recorded perinatally. Where electronic information was incomplete or uncertain, individual patient records were obtained and viewed to affirm accuracy.

All ultrasound measurements were performed by trained sonographers or obstetric specialists. Estimation of fetal weight was determined using measurements of biparietal diameter (BPD), abdominal circumference (AC), and femur length (FL), which were applied within the formula:

Estimated fetal weight = EXP [2.3026*(1.385 + 0.06739*BPD + 0.03591*AC – 0.00006883*BPD*AC + 0.1312*FL – 0.002675*AC*FL)].

This is a local modification of a formula described by Woo[14] and used at Wellington Hospital since 1996. It was developed after analysis of 581 cases between 1990–1995, which showed this formula to have the best prediction of birth weight (Personal Communication, Paula Carryer, 2002).

Statistical analyses were performed using the Student's t-test and linear regression analysis for parametric data and the non-parametric Kruskal-Wallis, Mann-Whitney U tests, and Chi-squared tests with $p<0.05$ considered significant. Data are presented as mean ± standard deviation (SD).

## Results

Of the 20,649 term live-born singleton infants delivered at Wellington Hospital during the study period, 1177 (5.7%) had undergone ultrasound estimation of fetal weight <7 days preceding birth.

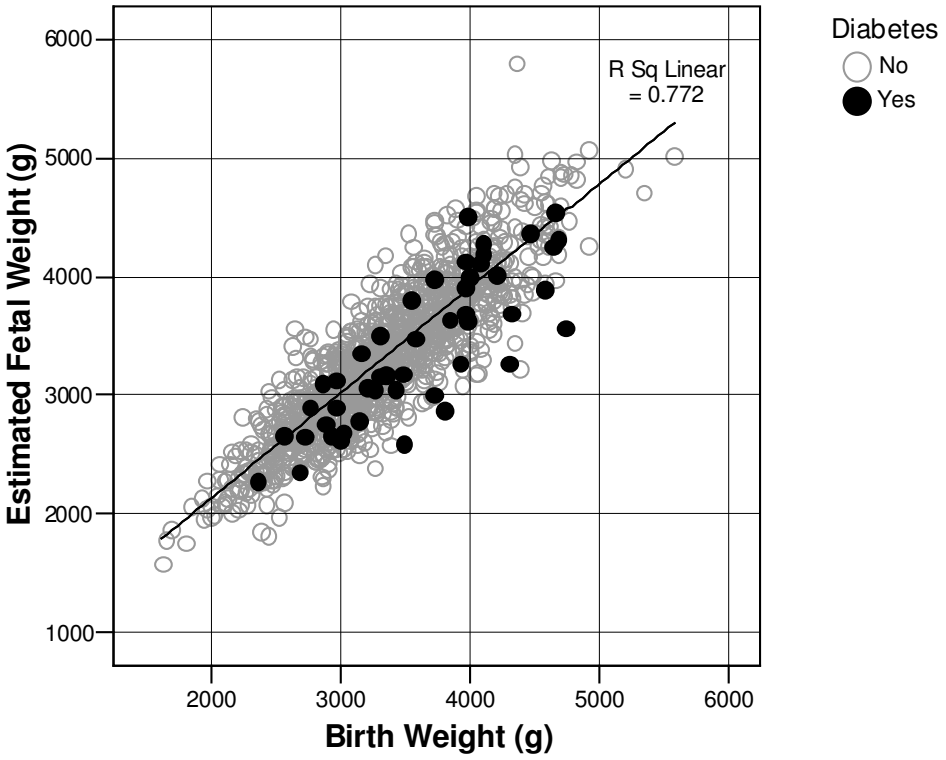
The mean actual birth weight within the study cohort was 3325 g (range 1620–5580 g). Ninety-eight infants (8%) weighed less than 2500 g, and 170 (14%) weighed more than 4000 g. Of the 170, 36 weighed more than 4500 g and three babies weighed more than 5000 g.

Forty-eight (4.1%) women in the study cohort had diabetes in pregnancy. The birth weight of the infants born to these women was significantly higher (3603±629 g) than in non-diabetic women (3314±604 g, n=1129) (p=0.001).

The mean time interval between ultrasound estimation of fetal weight and delivery was 2.9±1.8 days (n=1177) and did not differ significantly between diabetic and non-diabetic women, nor amongst infants in different birth weight categories. Within each birth weight category (<2500 g, 2500–4000 g and ≥4000 g), birth weight did not significantly differ between diabetic and non-diabetic women.

The ultrasonic estimation of fetal weight significantly correlated with actual birth weight for all infants (R=0.879, p<0.001) (Figure 1). Seventy-five percent of all fetal weight estimations were within 10% of actual birth weight; in one out of four women, the error was >10% (Table 1). The difference was >20% in 3% of the weight estimations.

**Figure. 1 Scatter plot showing correlation between birth weight and the ultrasound estimation of fetal weight (R=0.879, $R^2$=0.772, p<0.001, n=1177)**




The mean absolute error of fetal weight estimations was 7.0±5.7% (n=1177). This did not differ significantly between infants of different birth weights (<2500 g, 7.3±6.4%, n=98; 2500–3999 g, 6.9±5.6%, n=909; ≥4000 g, 7.3±5.8%, n=170) nor between diabetic and non-diabetic pregnancies (diabetic 8.3±6.5%, n=48; non-diabetic 6.9±5.6%, n=1129).

**Table 1. Error distribution of estimated fetal weights**

| Ultrasound weight estimation | Birth weight (g) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | <2500 | | 2500–3999 | | ≥4000 | | All weights | |
| | n | % | n | % | n | % | n | % |
| **Non-diabetic women** | | | | | | | | |
| Within 10% of birth weight | 71 | 73% | 659 | 75% | 116 | 74% | 846 | 75% |
| >10% below birth weight | 3 | 3% | 101 | 12% | 32 | 20% | 136 | 12% |
| >10% above birth weight | 23 | 24% | 115 | 13% | 9 | 6% | 147 | 13% |
| Total | **97** | **100%** | **875** | **100%** | **157** | **100%** | **1129** | **100%** |
| **Diabetic women** | | | | | | | | |
| Within 10% of birth weight | 1 | 100% | 24 | 71% | 9 | 69% | 34 | 71% |
| >10% below birth weight | 0 | 0% | 9 | 27% | 4 | 31% | 13 | 27% |
| >10% above birth weight | 0 | 0% | 1 | 3% | 0 | 0% | 1 | 2% |
| Total | **1** | **100%** | **34** | **100%** | **13** | **100%** | **48** | **100%** |

Although the absolute percent errors for fetal weight estimation were similar in each of the birth weight categories, the direction of the error (i.e. underestimation vs overestimation) differed. The percentage of infants whose birth weight was underestimated by more than 10% rose from 3% of babies in the low birth weight group to 21% in the macrosomic group. Conversely, the incidence of ultrasound overestimation of weight by more than 10% dropped from 24% of infants in the low birth weight group to 5% in the macrosomic group. Infants in the normal weight group were equally likely to have their weight underestimated (12%) or overestimated (13%) by >10%. These trends remained the same even when non-diabetic pregnancies were analysed separately (Table 1).

Although the absolute percent errors for fetal weight estimation were similar for diabetic and non-diabetic pregnancies, infants of diabetic mothers showed a tendency towards marked underestimation of weight (27% of infants) rather than overestimation (2% of infants), and this trend was seen in both normal weight infants and macrosomic infants (Table 1). This underestimation of weight of infants of diabetic pregnancies (27% [CI: 17–41%]) was significantly more common in comparison to infants of non-diabetic pregnancies (12% [CI: 10–14%]) ($p<0.01$). Only one infant of low birth weight was born to a diabetic mother; its fetal weight estimation was within 10% of birth weight.

These trends were also evident when signed percent errors were examined in the different groups. The mean signed error for all fetal weight estimations was -0.2±9.0%—but when infants were analysed by weight category, the mean signed errors were as follows: <2500 g, +3.5±9.1% (n=98); 2500–3999 g, 0.0±8.8% (n=909); ≥4000 g, -3.3±8.7% (n=170).

Thus the calculation of weight based on ultrasound measurements tended to overestimate the weight of low birth weight infants while underestimating the birth weight of large babies. Mean signed error was 0.0±8.0% in non-diabetic women (n=1129) but was -5.1±9.2% in diabetic women (n=48), indicating that ultrasound tended to underestimate fetal weight in women with diabetes in pregnancy. Linear regression analysis showed that birth weight and diabetic status each had a significant and independent influence on mean signed error (birth weight, $p<0.001$; diabetes, $p=0.001$).

The data were examined to determine the influence of time interval between the ultrasound scan and delivery on accuracy of the fetal weight estimation. There was no significant difference in the absolute error between estimations made ≤3 days prior to delivery (7.0%±5.7, n=724) and those performed 4–6 days before delivery (7.0%±5.7, n=453). Ultrasound measurements carried out 4–6 days prior to delivery tended to result in a slight underestimation of fetal weight (mean signed error = -1.3%±8.9, n=453, p<0.01) whereas ultrasound examination performed ≤3 days before delivery resulted in a mean signed error that was not significantly different from zero (mean signed error = +0.5%±9.0, n=724).

The ability of ultrasound fetal weight estimation to predict fetal macrosomia in non-diabetic women when defined as ≥4000 g or ≥4500 g is shown in Tables 2 and 3, respectively. The cohort contained only 48 diabetic pregnancies, of which only 13 resulted in macrosomic deliveries; thus it was not possible to perform a meaningful analysis of macrosomia prediction in pregnant women with diabetes.

**Table 2. Ultrasound prediction of birth weight greater than 4000 g in pregnancies of non-diabetic women**

| Estimated fetal weight (g) | Actual birth weight (g) | | Total |
|---|---|---|---|
| | <4000 | ≥4000 | |
| <4000 | 929 | 62 | 991 |
| ≥4000 | 43 | 95 | 138 |
| **Total** | **972** | **157** | **1129** |

Sensitivity: 61% (CI: 53–68%), specificity: 96% (CI: 94–97%), positive predictive value: 69% (CI: 61–76%), negative predictive value: 94% (CI: 92–95%); CI=confidence interval.

**Table 3. Ultrasound prediction of birth weight greater than 4500 g in pregnancies of non-diabetic women**

| Estimated fetal weight (g) | Actual birth weight (g) | | Total |
|---|---|---|---|
| | <4500 | ≥4500 | |
| <4500 | 1073 | 18 | 1091 |
| ≥4500 | 20 | 18 | 38 |
| **Total** | **1093** | **36** | **1129** |

Sensitivity: 50% (CI: 34–66%), specificity: 98% (CI: 97–99%), positive predictive value: 47% (CI: 32–63%), negative predictive value: 98% (CI: 97–99%); C=confidence interval.

There were no cases of macrosomia in infants when the estimated fetal weight was <3000 g. In the non-diabetic women, 3% (10/349) of infants with fetal weight estimations of 3000–3499 g had actual birth weights of more than 4000 g and 19% (52/273) of infants with fetal weight estimations of 3500–3999 g had actual birth weights of more than 4000 g. Conversely, 41% (41/100) of infants of non-diabetic women with fetal weight estimations in the range 4000–4499 g actually weighed less than 4000 g at birth. Sixteen percent (16/100) of estimated fetal weights in the range 4000–4499 g resulted in infants weighing more than 4500 g, and 53% (18/34) of

estimated fetal weights in the range 4500–4999 resulted in infants weighing less than 4500 g.

## Discussion

This analysis of 1177 pregnancies is the largest study of the reliability of ultrasound fetal weight estimation in New Zealand. The ultrasound estimation of fetal weight at Wellington Hospital was associated with a mean absolute error of 7%, a figure that compares favourably with other published data.[15–17]

Three out of four (75%) fetal weight estimations were within 10% of actual birth weight—this rate is as good or better than in most published studies (63%,[15] 74%,[16] 23–78%,[17] 52%,[18] 60%,[19] and 74%[20]).

Although the accuracy of our estimations was comparatively good, one out of every four fetal weight estimations was more than 10% different from actual birth weight. Ultrasound measurements give the appearance of precision, but the accuracy of ultrasonic estimations of fetal weight is limited by the fact that the mature fetus is an irregular, three dimensional structure of varying density, the weight of which cannot be calculated with certainty from biometric measurements.[12] It is therefore not surprising that the Australasian Society for Ultrasound in Medicine states that "No formula for estimating fetal weight has achieved an accuracy which enables us to recommend its use,"[21] despite the large number of formulae available.[17,22]

In our study there was an association between fetal size and the direction of the weight estimation error. Thus, for the one in four infants whose fetal weight estimation was more than 10% different from actual birth weight, the error was generally one of overestimation in the case of the small infants and an underestimation in the case of the macrosomic infants. These trends have previously been documented in a systematic review of ultrasonic estimation of fetal weight.[22]

However, our study did not confirm the findings of others[15,18] that the ultrasound estimation of fetal weight was less accurate in macrosomic infants than in non-macrosomic infants. In our study both the mean absolute percent error and the percentage of infants whose estimated fetal weight was within 10% of birth weight were similar in all three weight groups.

The tendency of the ultrasound estimation of fetal weight to err towards normal when the infant was subsequently found to be either <2500 g or ≥4000 g is important because the estimation of fetal weight is of relevance in clinical decision-making at these extremes. The relationship between birth weight and the direction of the estimation error was not due to a bias in the time interval between ultrasound and delivery (as might occur if smaller infants were scanned more regularly) as there was no relationship between infant birth weight and the time interval between ultrasound and delivery.

The reliable estimation of fetal weight is especially important in diabetic pregnancies because these pregnancies are at greater risk of macrosomia.[7,8] Amongst pregnancies complicated by fetal macrosomia, shoulder dystocia occurs more commonly in diabetic than non-diabetic women.[5] Fetal weight estimations have been reported to be less accurate in women with diabetes by some authors,[23,24] whereas others have found no difference in accuracy.[15]

In our study there was no significant difference in the accuracy of fetal weight estimation between women with diabetes and non-diabetic women. There was, however, a systematic underestimation (-5%) of fetal weight in the women with diabetes. This underestimation was also noted by Wong et al (2001),[24] who attributed it to the greater liver size and the increased subcutaneous fat that commonly occurs in fetuses of women with diabetes not being reflected in the formulae used in ultrasound fetal weight estimation.

In this study, the ultrasound estimations of fetal weight were performed <7 days prior to delivery. Although some authors studying reliability of ultrasound estimation of fetal weight have included estimations performed up to 14 days prior to delivery,[25] others have restricted their data to estimations performed within 7 days[24] or 3 days,[15,20] or have attempted to correct for the time elapsed between the ultrasound and delivery by the addition of 25 g per day[26] or 12.4 g or 13.0 g per day (Nahum et al, 2003).[17] Although fetal weight estimations made 4–6 days before delivery tended to slightly underestimate birth weight in our study, the error was small (-1.3±8.9%).

Amongst the non-diabetic cohort, ultrasound estimation of fetal weight detected only three out of every five infants weighing more than 4000 g and only half of the infants weighing more than 4500 g. Our findings thus confirm those of others,[10,13,20] that ultrasound does not reliably detect macrosomia, at least in non-diabetic mothers. Until more reliable methods are developed to determine fetal macrosomia, the use of ultrasound to assess fetal weight in singleton term pregnancies must be interpreted with caution.

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# Myotonic dystrophy in Otago, New Zealand

Chris Ford, Alexa Kidd, Graeme Hammond-Tooke

**Abstract**

**Aims** To determine the prevalence of myotonic dystrophy (DM) in Otago, the ethnic distribution of the disease, any founder effect, the complications and adequacy of health care, and the quality of life of sufferers in this region.

**Methods** DM patients were identified through hospital records and assessed using a structured questionnaire, neurological examination, and review of hospital records. Quality of life was evaluated using the SF-36 Health Survey, and compared to patients with other neuromuscular conditions and New Zealand norms.

**Results** 21 patients were identified, giving a prevalence of 11.6 per 100,000. All were of European descent. There was no evidence of a common ancestor. Not all patients had had essential investigations such as electrocardiogram and many had not been seen by the genetic service. DM patients had higher scores on the bodily pain subscale of the SF-36 Health Survey, compared to neuromuscular controls and the general population. Subjects differed significantly from New Zealand norms on four of the eight subscales.

**Conclusions** DM is relatively common in Europeans in Otago, but we found no cases in other ethnic groups. The disease affects aspects of quality of life, and management could be improved by use of a clinical care pathway.

Myotonic dystrophy (DM) is a dominantly inherited, multisystem disorder, with at least two genetic variants—type 1, due to a trinucleotide (CTG) repeat expansion on chromosome 19; and the rare type 2, due to an expanded CCTG repeat on chromosome 3q.[1] The clinical features include weakness, myotonia, cataracts, cardiomyopathy, and gonadal atrophy.

DM is the most common form of adult muscular dystrophy, and the prevalence in the Western World has been reported to range from 2.2–5.5/100,000.[2] Studies of DM in South Africa[3,4] and Canada[5] have shown evidence for a founder effect—where most individuals are descendants of a common ancestor. The prevalence in New Zealand is not known, and there has been no report of a founder effect.

There are also striking ethnic differences, DM being less prevalent in South-East Asians and extremely rare in central and southern Africans.[6] There have been no reports of the prevalence in different ethnic groups in New Zealand, but anecdotally, DM seems to be rare in Polynesians.

Because DM is a multisystem disorder, patients require management by multiple specialties, and it is important to ensure well-planned and consistent care.[7] It is also likely to impact on quality of life, but there has been no study of quality of life issues in a pure population of DM sufferers.

The aims of this study were therefore multiple. To determine:

- The prevalence of the condition in Otago;
- The ethnic distribution;
- Whether there is a founder effect in Otago;
- Whether patients receive adequate medical care and whether their quality of life is affected.

## Methods

Patients with a clinical diagnosis of DM and resident in Otago were identified through a search of computerised medical records in the Neurology Department at Dunedin Hospital, and through a database of members of the Muscular Dystrophy Association of New Zealand. Other involved family members were identified through the index cases. Subjects were recruited via a letter inviting them to take part in the study.

Patients were assessed at the hospital or in their home. They were given a structured interview, a full neurologic examination, and they also completed the SF-36 Health Survey.[8] The investigators were not blinded to the diagnosis, as the typical facial appearance usually made this impossible.

For the quality of life survey, age and sex matched control subjects with other neuromuscular diseases were identified from Neurology Department records and recruited by letter. Results were also compared with New Zealand norms.[9]

Student's t-test was used to evaluate the results of the SF-36 Health Survey, using the SPSS-PC software package (SPSS Inc, Chicago, Illinois, USA).

The study was approved by the Otago Ethics Committee and informed written consent was obtained from all participants.

## Results

**Prevalence and demographics of myotonic dystrophy in Otago—**Twenty-one patients with a diagnosis of DM were identified, of whom 18 agreed to participate. With a population base of approximately 181,539 (New Zealand 2001 Census[10]) this represents a prevalence of 11.6 per 100,000. Ten patients had DNA confirmation of the diagnosis, including two that did not participate. For those patients without DNA testing, diagnosis was based on clinical findings and electromyography. No cases of type 2 DM were identified, either clinically or with molecular genetics.The mean age of our patients was 45 years with a range of 16 to 74 years (Table 1); 61% were male and all were of European descent but born in New Zealand, with no knowledge of Māori ancestry.

**Table 1. Demographic characteristics of myotonic dystrophy (DM) patients and controls**

| Variable | Age (years), SD | Range (years) | Sex (n (%)) | |
|---|---|---|---|---|
| | | | Male | Female |
| DM Patients | 45.0±13.9 | 16–74 | 11 (61) | 7 (39) |
| Controls | 47.8±15.0 | 15–75 | 11 (61) | 7 (39) |

**Genetic aspects—**There were 12 families: 4 parent-child pairs, 2 sibling pairs, and 6 patients with no other family members in Otago with DM. When the pedigrees of the 12 families were investigated, one previously unknown link was found between two

of the families. In most families it was possible to trace back the lineage only by two generations. But it was nevertheless clear that the families were otherwise unrelated.

**Clinical features—**Fourteen patients were mildly affected by the disease. Their most prominent symptom was muscle stiffness (myotonia), with mild weakness and minimal impact on gait and walking. Three were moderately affected—patients found it difficult to walk large distances, and had some dysphagia. One was severely affected, with difficulties with walking and swallowing. All subjects were ambulant.

The most common initial symptom was myotonia, noted in 7 (38%). Fatigue was the most debilitating symptom according to 8 (44%) patients; 17 of the 18 patients reported significant myotonia, and 4 reported significant muscle pain.

All had weakness of typical distribution and myotonia, either clinically or electrophysiologically, leaving little doubt about the diagnosis.

Several complications of the disease were present in the patients, as shown in Table 2.

**Table 2. Complications of myotonic dystrophy**

| Complications | n (%) |
|---|---|
| **Respiratory** | |
| Sleep apnoea | 2 (11) |
| Type II respiratory failure | 1 (6) |
| REM-related hypoventilation | 1 (6) |
| **Cardiovascular** | |
| 1st-degree AV block | 3 (17) |
| Ventricular tachycardia and pacemaker | 2 (11) |
| **Swallowing** | |
| Minor swallowing difficulty | 13 (72) |
| PEG tube | 1 (6) |
| **Ophthalmologic** | |
| Cataract operation | 9 (50) |
| Minor visual problem | 1 (6) |
| **Endocrine** | |
| Diabetes mellitus | 2 (11) |
| Libido/impotence problems (in men, n=11) | 5 (45) |

AV=atrioventricular; PEG=percutaneous endoscopic gastrostomy.

**Management and access to specialist services—**Five patients lived in rural settings (more than 1 hour from a major hospital), and 13 lived close to (or in) an urban centre (less than 1 hour from a major hospital).

Six patients had been referred to a respiratory specialist for sleep problems (4 from urban communities, and 2 from rural locations); 15 patients had seen a cardiologist (11 from urban communities, and 4 from rural locations); 16 had had an ECG on record and 11 had had an echocardiogram; 5 patients had been referred to a speech language therapist (all from urban communities); and 9 patients had been referred to an ophthalmologist (5 from urban communities, and 4 from rural locations). Only four patients recalled having been seen by genetic services. Seven were members of the Muscular Dystrophy Association of New Zealand.

**Quality of life—**18 out of 22 invited control subjects with neuromuscular disorders agreed to participate. Their diagnoses were polymyositis (3 patients), dermatomyositis (1), myasthenia gravis (4), Charcot-Marie-Tooth disease (5), Becker muscular dystrophy (1), facioscapulohumeral muscular dystrophy (2), myotonia congenita (1), and limb girdle muscular dystrophy (1). They were not significantly different from the DM patients in age and sex ratio, and like the DM patients they were all of European descent (Table 1).

There was no significant difference between the DM patients and their matched controls in overall scores as measured by the SF-36 Health Survey. However, the DM patients had significantly higher scores on the bodily pain subscale than their paired controls (mean difference=20.22, t=2.692, p=0.015), and the New Zealand norms (Table 3). Both the DM patients and the controls differed significantly from the New Zealand norms on the subscales for physical functioning, role physical, general health and vitality, but not for social functioning, role emotional, and mental health (Table 3).

**Table 3. Myotonic dystrophy (DM) patients and controls compared to New Zealand (NZ) norms[9].**

| Variable | | SF-36 mean | NZ Mean[9] | T statistic | P |
|---|---|---|---|---|---|
| Physical functioning | DM | 65.00±26.07 | 86.00 | -3.42 | 0.0033 |
| | Controls | 50.83±29.42 | | -5.08 | 0.0001 |
| Role physical | DM | 52.78±37.27 | 80.70 | -3.18 | 0.0055 |
| | Controls | 48.61±38.80 | | -3.51 | 0.0027 |
| Bodily pain | DM | 88.11±17.20 | 77.90 | 2.52 | 0.0220 |
| | Controls | 67.89 | | -1.49 | 0.1547 |
| General health | DM | 45.78±28.16 | 73.80 | -4.23 | 0.0006 |
| | Controls | 53.11±26.69 | | -3.29 | 0.0043 |
| Vitality | DM | 39.44±19.55 | 65.60 | -5.69 | 0.00003 |
| | Controls | 42.22±23.34 | | -4.25 | 0.0005 |
| Social functioning | DM | 86.81±17.92 | 86.60 | 0.05 | 0.9609 |
| | Controls | 73.61±26.39 | | -2.09 | 0.0521 |
| Role emotional | DM | 81.48±32.78 | 85.00 | -0.46 | 0.6546 |
| | Controls | 81.48±32.78 | | -0.46 | 0.6546 |
| Mental health | DM | 79.78±12.76 | 78.00 | 0.59 | 0.5621 |
| | Controls | 74.44±17.74 | | -0.85 | 0.4062 |

## Discussion

This study shows that the Otago region has a prevalence of DM that is more than double that reported in Western Europe.[1] The population base was derived from the 2001 census[10], and the Otago population may have increased slightly, but this is unlikely to affect the result by more than 2%, which was the growth of the Otago population between 1996 and 2001. The reason for the high prevalence is unclear, and it may just be chance that produced this result, as the population studied was quite small.

As the neurology service for the region is concentrated in our hospital, with limited private practice, case ascertainment may have been better than previous studies. Our department's subspecialty interest in neuromuscular diseases was not a factor, because we excluded patients not domiciled in Otago. There was no evidence for a founder effect. As we may have missed patients who had not been diagnosed or had not been referred and were not members of the Muscular Dystrophy Association of New Zealand, the true prevalence of DM may be even higher.

All patients indicated that they were of purely European descent and none were Māori. This may reflect the high proportion of Europeans (93.7%) and low proportion of Māori (6%) in the Otago population,[10] or indicate that Māori are less likely to seek medical care. However, an informal (unpublished) survey of all neurologists in New Zealand found no DM patients of Māori or Pacific Island ancestry.

DM is rare in sub-Saharan Africans, and this is reflected in a lower frequency of large-sized normal alleles (CTG repeats) in this part of Africa.[6,11] Outside of Africa, the frequencies of large-sized normal alleles and the prevalence of DM are highest in West Europeans and Japanese and lowest in South-East Asians[6]. As larger alleles are more unstable, it is postulated that people with large-sized normal alleles provide a pool of individuals, who may have descendants with DM.

The original expansion of CTG repeats into the large-sized normal range may have occurred in a north-eastern African population prior to the migration of the ancestors of the European and Asian population out of Africa, and most cases of DM may be descendants of these individuals.[6]

The prevalence of DM in Polynesian populations is unknown, but our inability to identify any cases in the Māori or Pacific Islander populations suggests that it is low. If so, the prediction would be that Polynesians have a low frequency of large-sized triplet repeats, perhaps reflecting their origins in South-East Asia. However, the frequency of large sized alleles is high in Micronesian and Australo-Melanesian populations (also thought to originate from South-East Asia), while the prevalence of DM is unknown.[6] In Polynesians, neither the prevalence of DM or the frequency of large alleles is known, and more research is clearly needed.

We attempted to assess the standard of care of patients with DM. As this is a multisystem disorder, management can be complex and requires input from several specialties. It is not clear from our data whether patients received adequate input from other specialties. However, it is concerning that not all patients had a recent electrocardiogram, and there was one patient with visual symptoms who had not yet been referred for ophthalmological evaluation.

The low rate of referral to genetic services probably reflects the lack of a genetic service in this region at one time, but does suggest a degree of inertia in referring patients already known to our service. Review of the notes suggested that care was haphazard and non-systematic. It has been recognised that clinical guidelines and integrated clinical care pathways are important in the management of complex genetic disorders.[7] Our findings emphasise the need to make use of such protocols in myotonic dystrophy, and we plan to introduce a clinical care pathway into our clinical practice.

Although overall quality of life, as measured by the SF-36, was no different to patients with other neuromuscular conditions, DM patients had significantly higher scores on the bodily pain subscale than paired controls and New Zealand norms. Surprisingly, only four DM patients reported significant muscle pain during the interview, so it was not clear why they scored so high on this subscale. Muscle pain has been commonly reported in other studies however.[1]

Both the DM patients and the controls had worse scores for physical functioning, role physical, general health, and vitality, when compared with New Zealand norms. DM patients and neuromuscular controls were not significantly different to the general population for social functioning, role emotional, and mental health, which is surprising for a disabling group of diseases.

No previous studies of muscular dystrophy have utilised the SF 36 Health Survey. However, several Swedish studies have examined quality of life in muscular dystrophies, including myotonic dystrophy, using the Sickness Impact Profile and the Kaasa test.[12–15] In these studies, no significant differences were found between types of muscular dystrophy, and increasing disability over 5 years was correlated with decreased coping and quality of life. Quality of life was significantly related to forced vital capacity and fatigue, but less so to other respiratory and cardiac parameters and performance of activities of daily living.

Myotonic dystrophy is an important neuromuscular disease in Otago, affecting quality of life and requiring significant health resources. Management of these patients could be improved with the use of an appropriate clinical care pathway.

The apparent low prevalence of myotonic dystrophy in Māori and other Polynesians is intriguing and, if confirmed, may help inform theories of the genetic origins of these populations.

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# Erucism in New Zealand: exposure to gum leaf skeletoniser (*Uraba lugens*) caterpillars in the differential diagnosis of contact dermatitis in the Auckland region

José Derraik

**Abstract**

There are no indigenous caterpillars known to be associated with erucism, but the recently established gum leaf skeletoniser (*Uraba lugens*) has venom-containing spines that cause adverse reactions in humans. Symptoms are usually characterised by a stinging sensation, followed by itching and the formation of wheals. Exposure to *U. lugens* should be considered by medical practitioners in the differential diagnosis of contact dermatitis in the Auckland region.

## Introduction, biological notes, and distribution

'Erucism' is the term generally used to refer to the adverse reactions resulting from contact with urticating caterpillars, the larval forms of the insect order Lepidoptera (moths and butterflies).[1,2] Although erucism is a relatively common public health problem throughout the world,[1] there are no indigenous species of Lepidoptera in New Zealand whose caterpillar is known to cause adverse reactions in humans (Brian Patrick, Otago Museum, personal communication; 2006).[3] As a result, erucism has never been a human health issue in this country, and is therefore a condition somewhat unknown to local medical practitioners.

The situation has changed however, since the establishment of the gum leaf skeletoniser *Uraba lugens* Walker (Lepidoptera: Nolidae), an Australian moth whose caterpillar feeds on the foliage of gum trees (*Eucalyptus*) and other closely related genera.[4] The younger larvae avoid feeding on the oil glands and veins found in the leaves, which are consequently 'skeletonised'.[3] More mature larvae will however eat the whole leaf.[3]

*Uraba lugens* is a significant pest for *Eucalyptus* forestry. Infestation by *U. lugens* may kill young trees if there is repeated defoliation.[5] This process on larger trees may reduce wood production for several seasons.[6] Outbreaks of this species seem to periodically occur in natural forests in Australia, but these eventually recover, even though defoliation can be severe.[7]

*Uraba lugens* was first discovered in New Zealand in 1992, and it is now firmly established in the Auckland region, over an area of at least 20,000 ha.[4] Due to its wide distribution, eradication was deemed to be not feasible.[8] *Uraba lugens* is now the focus of a long-term management programme aiming particularly at filling current knowledge gaps and controlling the existing population.[8]

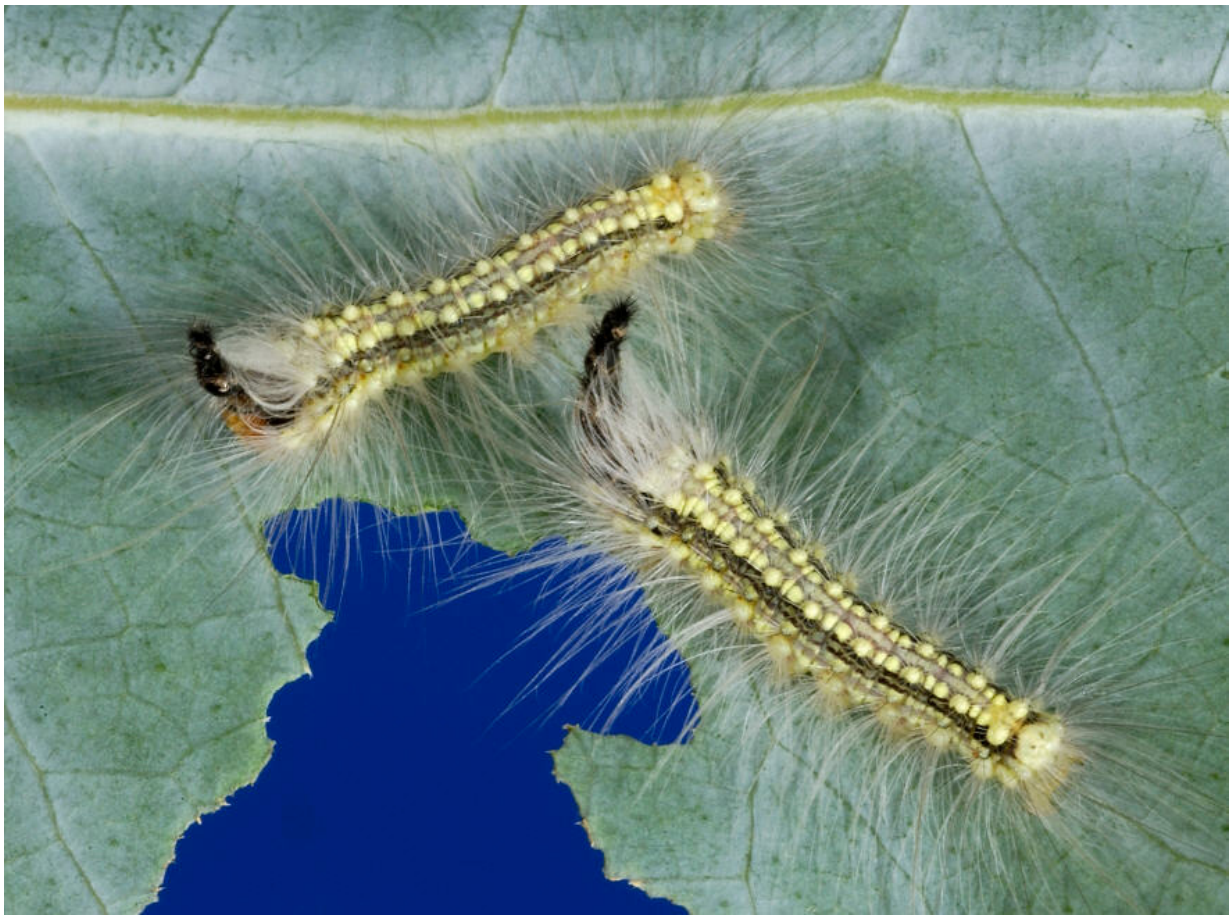
The approximate distribution of *U. lugens* currently goes as far north as Takapuna and as far south as the Bombay Hills, including the area between the Hunua Ranges in the east and the Manukau Heads and Waitakere Ranges in the west.[7] The highest density

of this organism seems to occur in southwest Auckland.[9] Results from climatic models indicated that *U. lugens* could potentially establish throughout New Zealand. To date, the population is not known to be established outside the Auckland region, but specimens have recently been captured in pheromone traps in Katikati (Bay of Plenty) and Warkworth (Northland).[18]

The Auckland populations of *U. lugens* have two generations per year (bivoltine), with larvae usually present from January to March (summer) and May to October (winter).[10] *Uraba lugens* caterpillars vary in length from 1 to 25 mm depending on the stage of development.[3.] There are between 11 and 13 larval stages.[4,5]

*Uraba lugens* are extremely hairy caterpillars with yellow and brown markings, and when mature they retain the head capsules of previous instars that are pushed up to top of the head, which makes *U. lugens* easy to identify (Figure 1).[5] Each body segment of the larvae has 10 tubercles, four of which are located dorsally and have short, stiff, brown-tipped bristles that are hollow and contain venom that can be injected into the human skin upon contact.[3]

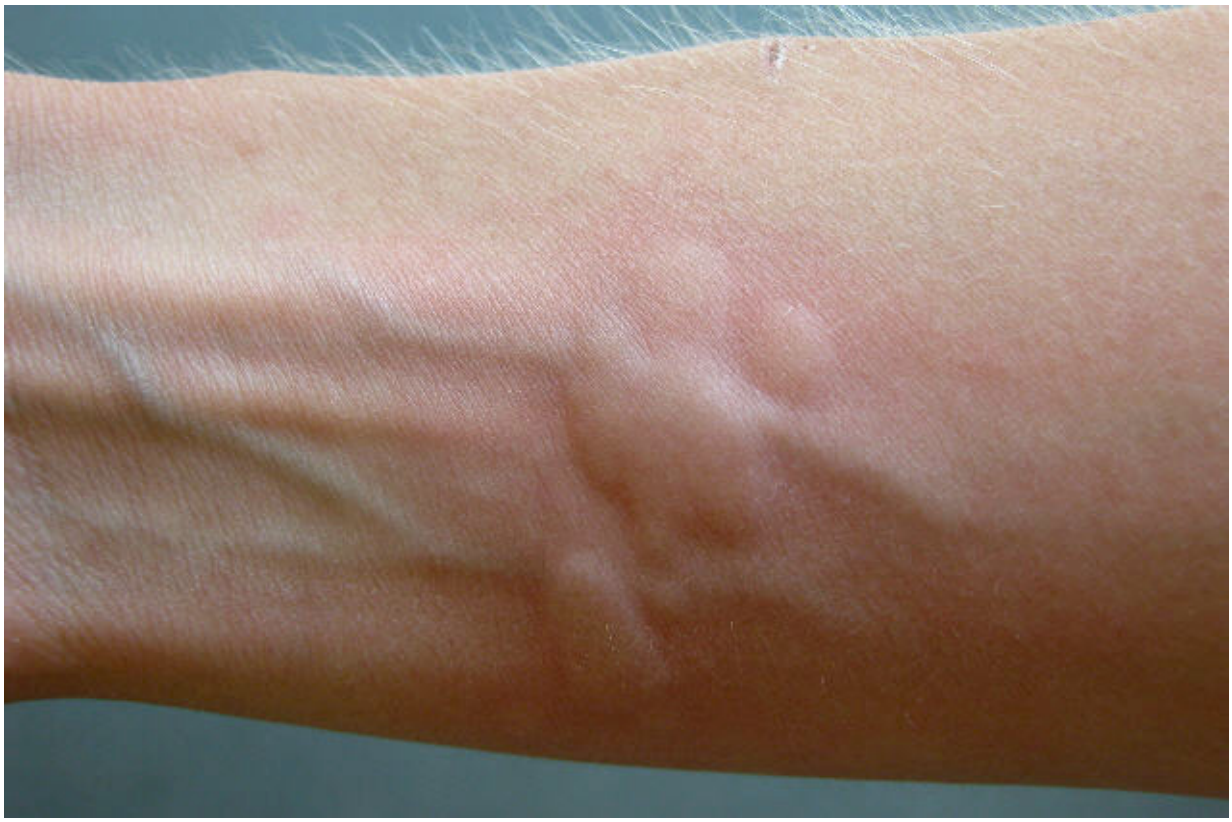
**Figure 1. *Uraba lugens* caterpillars (Photo courtesy of Ensis)**



## Exposure, symptoms of envenomation, and possible treatment

The exposure of people to stinging caterpillars is greater when these creatures are occurring at high densities, as numerous caterpillars may be found in the direct vicinity of an infested tree. Large infestations of stinging caterpillars can become a serious public health issue, and in some severe cases it has led to the closure of schools.[2]

**Figure 2. Wheals formed approximately 30 minutes after exposure to the urticating spines of *Uraba lugens* (Photo courtesy of Ensis)**



Most cases of harmful exposure to caterpillars seem to occur in young children, and in one study of 365 cases of exposure to *Lophocampa caryae* Harris (Lepidoptera: Arctiidae), 80% of the records were paediatric exposures.[11] Caterpillars are a source of curiosity to children due to their easy accessibility and slow mobility,[11] and also due to these creatures' generally bright colours. Young children tend to have thinner skin and smaller bodies than adults, both of which may increase the extent of the reaction.[12]

Approximately 150 species of Lepidoptera from more than a dozen families have been described to cause some form of injury to humans.[1,13] The caterpillars of many species have chitinous spines that are capable of penetrating human epidermis, and injecting venom parenterally.[13] Exposure to stinging caterpillars can result in a variety of reactions, which vary according to the species,[14] but adverse reactions range from

moderate to severe local effects, usually characterised by severe pain, and less commonly systemic effects,[14] including renal failure and intracerebral hemorrhage.[1]

The nature of *U. lugens* venom is largely unknown,[12] but it contains histamine and most likely also a proteinaceous substance.[3] Skin contact with the envenomating bristles of *U. lugens* will immediately cause a sharp stinging sensation, which may be severe.[3] Local pain is followed by the associated formation of flat itching wheals (Figure 2),[15] which may remain visible for a few weeks.[3]

The skin reaction may cause a high degree of discomfort, and an adult woman described the reaction as "violent and distressful for 3–4 days".[3] As a result, adverse reactions to *U. lugens* venom may be particularly distressing for young children. It is important to note that even the spines on the youngest caterpillars are capable of stinging, and that these continue to sting even after the insect is dead, and also following the shedding of skin.[3]

Ingestion of caterpillars of other species by children has been described in the literature,[1,14,16] with some adverse effects consisting of pain, difficulty swallowing, drooling, and shortness of breath.[16] There seem to be no records of *U. lugens* ingestion, but it would be likely to require hospitalisation. While life-threatening reactions are unlikely to occur, the possibility of serious adverse reactions, such as anaphylaxis, cannot be discarded.[12] Although severe and systemic reactions to *U. lugens* have not been described, eye lesions could be potentially serious and should be dealt with by a specialist. Note that there is no evidence of sensitisation from repeated exposures to *U. lugens*.[3]

Contact with some caterpillar species such as the white-stemmed gum moth (*Chelepteryx collesi*) leads to a very large number of hairs becoming embedded in the skin.[17] Even though for some species attempts to remove the hairs seem to be unsuccessful,[17] the careful removal of spine(s) with adhesive tape is a commonly prescribed initial treatment of urticating caterpillar stings.[2,14] However, this is not likely to be an issue with *U. lugens*, as there seem to be no reports of its spines becoming embedded in human skin, especially in Southcotts's detailed descriptions of numerous cases of exposure.[3] Instead, the application of ice packs, and oral or topical administration of antihistamines to attenuate itching and burning sensation, is advised.[1,2,14] Intense inflammatory reaction may be locally relieved by topical or oral corticosteroids.[1,2]

## Current incidence and recommendations

There are no available data on the incidence of exposure to *U. lugens* in New Zealand. Biosecurity New Zealand has information on at least two confirmed cases, where members of the public have contacted the agency following adverse reactions to an 'unknown' caterpillar (Mark Ross, personal communication; 2006). However, based on the distribution of the caterpillars in the Auckland region and its relatively high density in some areas, one could expect the actual number of cases to be considerably higher. The author would welcome information on any confirmed cases of exposure to *U. lugens* in New Zealand.

Even though *U. lugens* is the target of a long-term management programme, this species is well-established in the Auckland region and will not be eradicated. Since Auckland is the most populated region in the country, human exposure to the

caterpillars is likely to occur on a regular basis, particularly among children. As a result, general practitioners should consider exposure to *U. lugens* in the differential diagnosis of contact dermatitis, where symptoms such as wheals are present.

Prevention is an important tool, and in cases where exposure to *U. lugens* is confirmed, the access of children to areas adjacent to infested trees should be restricted, and a reputable pest controller should be contacted for mitigating action. In case the infested tree is located on public land, the local or regional authority should be notified.

Avoidance is a fundamental preventive tool, and children should be educated not to touch or handle *U. lugens* caterpillars. It should be also noted that this species' potential establishment in *Eucalyptus* plantations in New Zealand may lead to occupational safety and health concerns, as a result of the likelihood of exposure to forestry workers.[12]

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# Colitis and bronchiolitis obliterans organising pneumonia—the treatment or the disease?

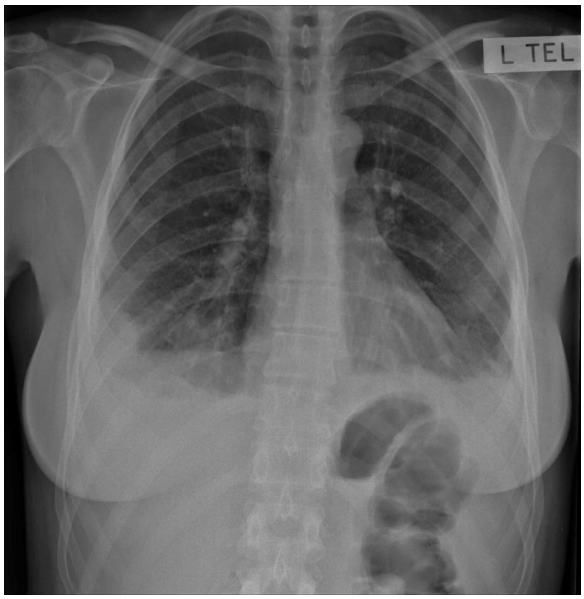
Oliver Menzies, Barry Colls

A 31-year-old white female was admitted to hospital with a slight fever, bilateral chest pain, and dyspnoea, which had been gradually developing over the past 12 months. Of interest was a similar admission 3 months previously, when no cause for her symptoms had been found, but a CT pulmonary angiogram (CTPA) showed small areas of parenchymal air space opacity in both lower lobes but no pulmonary embolism.

Her past history included fertility problems but she was currently pregnant after *in vitro* fertilisation. Ulcerative colitis had been diagnosed 1 year ago and she had been on mesalazine (5–aminosalicylic acid) at a dose of 4 g daily since that time.

Her physical signs included bilateral basal chest dullness to percussion and a low-grade fever. A chest X-ray (Figure 1) showed bibasal pulmonary/pleural opacities, more marked on the right. Ultrasound of the chest revealed minimal pleural effusions.

**Figure 1. X-ray showing bibasal pulmonary/pleural opacities suggestive of bronchiolitis obliterans organising pneumonia (BOOP)**



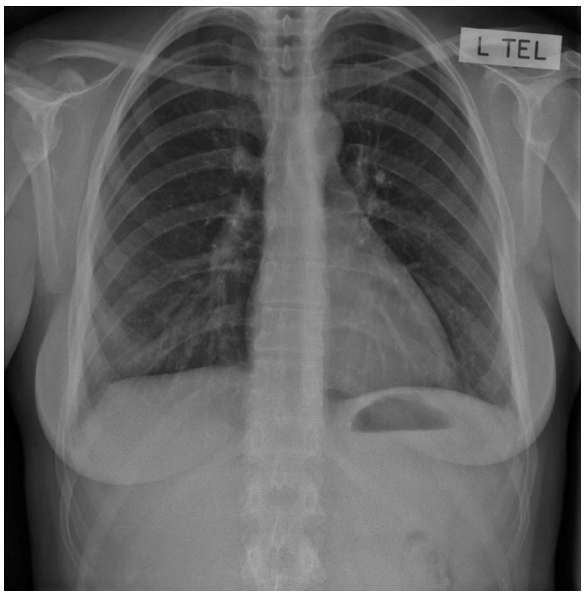

Blood screen: haemoglobin 106 g/L, white count 7·6 ($x10^9$/L) with normal differential, erythrocyte sedimentation rate (ESR) 35mm/hr. Serum sodium, potassium, creatinine, and liver function tests were all normal.

Lupus anticoagulant, anticardiolipin antibodies, and antineutrophil cytoplasmic antibodies (ANCA) were negative. Antinuclear antibody antibody (ANA) was positive at >1280 with a diffuse, chromosome-positive pattern. ENA, DsDNA, and complement 3 and 4 were normal. Ultrasound of the abdomen and pelvis revealed no evidence of ovarian hyperstimulation syndrome.

As pulmonary embolism and ovarian hyperstimulation syndrome seemed unlikely, she was treated with amoxicillin, but with no improvement. A pulmonary complication of her inflammatory bowel disease was considered but this was felt to be less likely as her ulcerative colitis was well controlled.

Mesalazine was stopped and prednisone 40 mg daily was instituted. Within a few days her symptoms improved. One month later she had no symptoms and her X-ray showed considerable improvement. Three months later she had a normal chest X-ray (Figure 2) and had resumed all her usual activities, including vigorous exercise.

**Figure 2. X-ray showing successful resolution of her symptoms**




Sulphasalazine which has been used in the past to treat inflammatory bowel disease is known to occasionally cause infiltrative lung lesions. It has generally been considered that the sulpha component of sulphasalazine is responsible for this adverse reaction.[1] Mesalazine (5-aminosalicylic acid) is now more commonly used as it does not contain sulpha and is less likely to cause adverse reactions. However, there is a small literature suggesting that mesalazine can cause bronchiolitis obliterans organising pneumonia (BOOP)[2].

A similar case was reported by Swinburn et al in 1988, although these authors felt that the syndrome in their patient was probably unrelated to the mesalazine.[3] Since that time there have been 20 or more case reports in the literature where mesalazine has been incriminated as a cause of BOOP.[2] In some of these cases, the exposure has been only for a few days (5 days in the case reported by LeGros),[1] but other cases have

been reported in which the exposure to the drug has been many months, or even years.[2]

The dose of mesalazine used has varied widely between 1 g and 4 g daily.[2] In all reported cases, withdrawal of the drug with or without steroid treatment has resulted in resolution of the pulmonary symptoms and x-ray abnormalities.[2]

Alternative diagnoses considered included diffuse interstitial pneumonia and systemic inflammatory response syndrome, but the timeline of her illness and its relationship to the inception and conclusion of her treatment with mesalazine made them unlikely. Inflammatory bowel disease may be associated with pulmonary manifestations such as our patient had, but the quiescence of the colitis over the entire symptomatic pulmonary disease process makes this alternative less likely.

Had this lady not been pregnant, further investigations such as high resolution computed tomography and/or transbrochial lung biopsy might have confirmed the diagnosis. Clearly these were not reasonable investigations in this case. Rechallenge with mesalazine was also not a sensible option.

The patient's respiratory symptoms commenced within days of the institution of mesalazine but intensified over the following year. Subsequently, the symptoms improved within days after the mesalazine was stopped. This sequence of events is highly suggestive that mesalazine was the underlying cause of the BOOP.

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# Quality improvement in New Zealand healthcare. Part 5: measurement for monitoring and controlling performance—the quest for external accountability

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**Abstract**

In this fifth article in the *Series* on quality improvement, we examine organisational performance indicators, the consequences of their use, and what policy-makers and clinicians need to do to minimise their potential adverse effects. The Ministry of Health (MOH) and district health boards (DHB) are increasingly using performance indicators to measure activity and are looking at how they can be used to incentivise provider performance. Two different approaches are used: report cards (comparing individuals and organisational performance) and pay-for-performance (which provides financial payments for those organisations or individual providers who do what the funder wants). Given the United Kingdom and the United States experience with both approaches, it would seem prudent for New Zealand to proceed cautiously with using performance indicators to modify clinician behaviour.

We argue that the integrity of the system overall is dependent on clinicians taking an interest in the indicators used by the Ministry of Health (MOH) and district health boards (DHBs). Furthermore, those using them must invest in learning about system variation and how this affects the external monitoring of performance.

> *In an attempt to arrive at the truth, I have applied everywhere for information but in scarcely any instance have I been able to obtain hospital records fit for any purpose of comparison. If they could be obtained they would enable us to answer many questions. They would show subscribers how their money was being spent, what amount of good was really being done with it or whether the money was not doing mischief rather than good.*[1]

This quote encapsulates what those measuring organisational performance are trying to achieve. The fact that Florence Nightingale said it in 1863 suggests that she was a woman ahead of her time as the issues are essentially unchanged.

The MOH, Treasury, and DHBs are still looking at ways to get useful information and have decided that investing in performance indicators is the way forward. This interest in performance management has been reinforced with the appointment of an experienced health manager—Stephen McKernan—as the new CEO of the Ministry of Health. We suggest that by this appointment the Government is signalling its intention to focus more on value for money considerations than it has in the past.

As noted in the previous article in this *Series*, Freeman[2] emphasises a useful distinction between indicators that are designed and used to improve healthcare

quality from within (*formative* clinical indicators) and those used in external performance monitoring (*summative* performance indicators).

In this article we will be focussing on summative measures as we examine the role of performance indicators and the two main ways that they are used in an attempt to improve quality of care: report card (or league tables) comparing performance across organisations, and the use of financial incentives attached to improved quality performance indicators (pay-for-performance).

## What are performance indicators and why use them?

Performance indicators are intended to enable outsiders to gauge how an organisation is performing, usually in comparison with other like organisations. Performance indicators measure numerically one aspect of an organisation's performance.

The 'performance' that is measured varies—it may be the financial performance of an organisation, the market share, or the productivity. Information from performance indicators can be used in a number of ways—to verify activity,[2] impose a policy agenda,[3] or to stimulate interest in quality improvement or the quality of care.

Another reason for performance indicators, particularly in publicly-funded institutions, is a quest to ensure that money is well spent. Treasury would argue that it has been putting extra money into healthcare and what have we got to show for it? Performance indicators can also be used to satisfy the desire to hold someone accountable.[4]

The history of performance indicators in New Zealand is somewhat chequered. In 1989, Helen Clark as Minister of Health led the MOH and area health boards into a contractual arrangement whereby the boards had to meet certain output targets in order to receive funding. This was the beginning of performance management of productivity in the New Zealand health system.

When Crown Health Enterprises (CHEs) were formed in 1993, performance monitoring began to expand as CHEs were encouraged to compete with each other. Early work focused on searching for efficiency indicators that could be used to 'improve management performance of individual crown health enterprises (CHE).[5]

With hindsight, some of the early performance indicators were plainly ridiculous—for example, CHEs performance in the area of public relations was gauged using an indicator that measured newspaper column inches of positive publicity as the numerator and total column inches of publicity as the denominator. A lot of this work developing performance indicators took place behind closed doors because of commercial sensitivity; neither clinicians nor patients were involved and the performance indicators chosen largely avoided scrutiny.

## Strategies to modify provider behaviour: *report cards*

Both the United States and the United Kingdom have invested heavily in public reporting of comparative performance indicators. The evidence suggests that patients and the public at large, are strongly in favour of publicly reported performance in principle. However, most studies conclude that they make little use of such reports.[6,7]

Purchasers and funders of healthcare also seem to be in favour in principle but also make little direct use of report cards. The key audience for public reporting appears to be the provider organisations themselves.[8]

Public reporting of performance indicators, or their use in league tables, demands very good data. If an organisation's or an individual clinician's reputations are at stake, then it needs to be established that the indicators are comparing 'like with like.' The *enthusiasm for public reporting is well ahead of the science*[8] and even the best 'risk adjustment' may not be able to accurately disentangle the key quality differences between organisations from those due to case-mix differences.

Organisational performance indicators, like clinical indicators, should be technically sound—derived from data that can be reliably obtained, be valid measures of what they are intended to measure, and focus on an area of importance.

The utility of an indicator is only as good as its ability to be measured accurately. As those in management and governance do not have knowledge about what goes on at the sharp end of service delivery, they can (and do) assume that data is being correctly obtained when in fact it often isn't. Indicator data entry may be delegated to ward clerks who, when faced with 'compulsory' fields, may enter nonsense data so that they can complete the process.

The validity of a performance indicator may be called into question if there is not a close relationship between what is being measured and the performance of interest. For example, the MOH requires DHBs to report on the rates of readmission to hospital within 28 days. It is unclear to clinicians why 28 days was chosen. If this indicator was measuring the performance of a hospital's discharge planning, then re-admission within 7 or 14 days would be a more useful indicator. By 28 days, many patients with chronic illnesses have developed a further exacerbation and 'perfect' discharge planning would not prevent their readmission.

Those in charge of funding allocation sometimes aggregate performance indicators for report cards into simple metrics such as ticks/crosses or even smiley/frowning faces.

There are two major problems with this:

- Reducing performance data into a series of smiley/frowning faces masks the complexity inherent in any data collection, it can hide the problems with validity and reliability of the measures themselves.
- Variation in the data may be an intrinsic property of the data (common cause variation) and not a reflection of any variation of the quality of care being measured. Organisations and their managers may be getting rewards (or punishment) for processes that are operating normally—not good, not bad—within the normal variation to be expected about a mean. If they react to this common-cause variation they can damage the system of care by wasting people's time and by making the system less stable.

Everyone using performance indicators should be knowledgeable about the difference between common-cause and special-cause variation and be comfortable with the limits that data can show.

## Strategies to modify provider behaviour: financial incentives—*pay-for-performance*

The conceptual and technical problems with performance indicators are compounded when they are linked to financial rewards or sanctions—a situation that has been described as *an ever expanding collection of carrots and sticks [in] the hope of influencing quality and cost control.*[9]

The use of financial incentives linked to performance is most evident in the UK, where hospitals with a 3-star rating have until very recently been eligible for a £1 million bonus (rescinded as too many hospitals achieved 3-star status). The performance indicators used included *key targets; patient focus; clinical focus*; and *capacity & capability focus* performance indicators.[10]

Examples of the *key targets* are:

- A&E emergency admission waits (<4 hours).
- All cancers: maximum 2-week wait.
- Breast cancer: 1 month diagnosis to treatment.

Breaches in performance indicators were aggressively managed by many National Health Service (NHS) trusts to ensure that their 3-star rating was protected, leading to some perverse behaviour. For example in response to the arbitrary key emergency care performance indicator: 'no patient should stay more than four hours in the ED' with a target of 98% compliance, some trusts aggressively managed any 'breaches.'[11]

Some of the dysfunctional behaviour included:

- Starting and stopping the clock at different stages in the patient journey.
- Opening a 'short-stay' ward next to the ED and moving patients into this if they threatened to 'breach.'
- Moving patients to an inpatient ward—even of their evaluation was not complete.
- When the indicator was measured quarterly, it was not unknown for trusts to hire more staff at the end of the audit period to inflate the percentages.
- Managers cajoling and sometimes bullying staff to meet the target.

Such unintended behaviour avoided the breach but did not necessarily address the patient's needs or improve the quality of care.

The NHS has also introduced an ambitious scheme in primary care, with the introduction of 146 performance indicators which if satisfied, provides approximately 30% of the general practitioner's salary (~£27,000 per GP). The first evaluation of this policy[12] has shown that targets were met for 83% of patients and primary care practices earned nearly 97% of the possible performance points.

As the policy-makers had estimated that practitioners would earn only 75% of available points, the initiative has contributed to the burgeoning NHS deficit.[13] Furthermore, early indications are that at least some of the bonuses were achieved by excluding large numbers of patients through 'exception reporting.'

Exceptions were defined at the outset and included factors such as: the patient had just joined the practice, had refused treatment, or despite three attempts had not attended for care.

Since 2001, the New Zealand Government has had a national primary care strategy[14] and is now primed to bring in performance measures and financial incentives. The exact level of this funding is still to be determined, but the lessons from the UK experience are important.

Our primary care performance measurement programme has identified a number of performance indicators including:

- Children fully vaccinated by their 2nd birthday.
- Influenza vaccinations in the elderly (over 65s).
- Cervical smears recorded in the last 3 years.
- Breast screening recorded in the last 2 years.

In addition, there are performance indicators which are intended to gauge the potential of Primary Healthcare Organisations (PHOs) to operate effectively and improve performance—for example:

- Percentage of valid National Health Index (NHI) numbers on PHO patient registers.
- Access for high needs enrolees.[15]

## Possible dysfunctional consequences of public reporting and pay for performance:

All performance indicators give rise to perverse incentives and unintended consequences,[16,17] and these are likely to be exaggerated when there are financial rewards or losses at stake (see Box 1). Both report cards and financial incentives are blunt instruments designed to change provider behaviour—in the hope that the change will be positive for the quality of care.

### Box 1. Possible dysfunctional consequences of public reporting of performance indicators.[21]

**Organisations or individuals may alter their behaviour by:**

- Concentrating on short-term goals (getting their rating higher) and neglecting the long-term strategy.
- Concentrating on those areas being measured to the detriment of other important areas.
- Placing great emphasis and energy on not being exposed as an outlier, rather than on a desire to be outstanding.
- Eschewing innovation for fear of failure.
- Altering their behaviour to gain strategic advantage ('gaming').
- Entering false or corrupt data.
- Avoiding the treatment of high-risk patients if this is going to reflect badly in a public report.
- Disengaging from quality improvement initiatives if the performance indicators do not seem relevant and are externally imposed.

Performance indicators by definition focus on one aspect of care—this may encourage organisations to concentrate on just those areas being measured and like clinical indicators, this means that those things which are not easily measured may be ignored.

Some aspects of healthcare quality lend themselves to measurement—e.g. waiting times in the emergency department and delays in surgery. Other important activities (e.g. accurate diagnosis and proficiency in discussing end-of-life issues with patients) are much harder to measure, and risk being ignored in the rush to report performance. Furthermore, unlike clinical indicators, externally imposed performance indicators cannot 'drill down' to provide information on what actions are needed to improve performance.

Organisations that gear themselves to 'do well' in report cards, or to increase their financial gains may be focusing on short-term goals, and in doing so, neglect the long term strategic vision and investment.

Performance indicators attempt to externally impose 'quality assurance,' however if clinicians do not have confidence in their validity, or if indicators do not align with professional values and assess an important clinical area, they may disengage from the process or worse still, 'game' the results. Gaming—'the alteration of behaviour to look good rather than the implementation of substantive improvements'[18]—can be damaging not only to the quality improvement effort but also to the involvement of clinicians in quality improvement work.

For example, problems exist with accreditation in New Zealand, where clinicians know that there are deep and serious problems with care provision, yet their organisation gets accredited without these problems being exposed. This leads to cynicism about the value of being involved in the process and to the questioning of the managerial drivers.

An even more important perverse behaviour is that potentially 'high risk' patients are avoided—if such high-risk patients are going to make the 'figures' look bad in a league table, there is a danger that there will be pressure to only operate on low risk cases, or concentrate on the easy to reach patients.[19]

Even when performance indicators are good measures, they may in fact threaten the trust necessary for clinicians to engage in quality improvement work. By elevating the status of external inspection they decrease that of the internal informal work that most organisations have. Indeed, it has been said that the 'indicator industry has begun to suffer from the regulators' delusion that central systems of oversight are the sole guarantors of quality.[20]

## What is the evidence that performance indicators improve quality of care?

Despite the enthusiasm for performance indicators and the millions of dollars spent on their development, relatively little is known about the actual impact on improving quality of care.

> *Performance indicators cannot capture the range and complexity of health service activity and are blunt and dangerous tools when used in pursuit of quality—that is, if they have any impact at all.*[22]

Healthcare is complex and less deterministic than traditional industries. The link between actions and outcomes in not necessarily strong or direct, and it is modified by non-healthcare factors (such as employment, deprivation) and patient-mix.[20] There is little evidence of a positive impact from performance indicators on health service delivery or health outcomes. [23] Even the oft-quoted example—the New York State league table published in a New York newspaper, where individual cardiac surgeons were ranked by the mortality rates of their coronary artery bypass patients—has been questioned. Yes, the mortality rates improved, but they also improved in other states that did not have public reporting. New England achieved similar benefits in mortality rates through confidential reporting and sharing best practices.[24] Furthermore in New York here was evidence that low volume surgeons stopped operating but also that high-risk cases went elsewhere.[25,26]

As discussed above, the blunt instrument that is public reporting resulted in unintended consequences.

## What can New Zealand learn from all this?

Firstly we need to be cautious about implementation of performance management systems.(Box 2) It may be useful to de-politicise public reporting.[8] Both the US and UK have agencies that have some political independence (the National Committee for Quality Assurance and the Commission for Health Improvement, respectively).

In order to get clinical buy-in, to command credibility, performance indicators need to address clinically important areas—for both clinicians and patients. We need to address as much attention as possible on the technical issues of performance indicators—validity, reliability, and case-mix. We also need to anticipate and monitor unintended consequences. It is important to ensure that the performance indicators relate to processes of care over which clinicians have control,[27] and that are not unduly affected by the non-healthcare factors discussed above.

**Box 2. Lessons for New Zealand—ways of reducing the dysfunctional responses to performance indicators**

- Anticipate and monitor unintended consequences—be prepared to modify indicators and their use accordingly.
- Consider an agency at 'arms length' from the Ministry of Health and the Treasury to develop and monitor a performance programme.
- Address technical issues for validity, reliability, and case-mix.
- Focus performance indicators on clinically important areas over which providers have control.
- Do not set financial incentives at a level which might distort clinical practice.
- Undertake formative evaluations of the effectiveness of the performance indicator programme as a whole, and learn from this.

Finally there is a substantial opportunity cost involved in developing performance indicators as well as collecting and analysing data, while seeking to risk adjust so that comparisons are useful. This is money that is not being spent on actually improving the quality of care delivered, and was a reason quoted by the Waitemata DHB CEO in his resignation—he cited stifling compliance issues as interfering with his ability to introduce patient safety initiatives. He urged the sector to refocus on concrete improvements to patient care, like better drug dispensing and infection control.[28]

When using financial incentives, they need to be large enough to influence performance, but not so large that they encourage distortions of the clinical processes and tenets of professionalism.

## Summary

Performance indicators, used in report cards or linked to financial incentives, seem to be firmly on the political agenda in New Zealand. If the NHS in the UK is any guide, we should expect to see increasingly vigilant surveillance by the MOH and DHBs of performance and quality in the New Zealand system.

While we endorse the MOH's interest in healthcare quality, there are serious pitfalls in how performance indicators are used. To be successful, they require funders and managers educated in the subtleties of performance management and indicator use (i.e. not to be content with aggregated measures of little validity) as well as clinicians who can challenge the indicator set.

> *It is clear that indicators of health care quality are not axiomatically good.*[2]

Two things can be guaranteed; performance indicator use by those funding health systems is on the increase, and indicator use is far from an exact science. What we have tried to do in this article is balance the sometimes zealous proponents of external performance monitoring, with the realities of the problems such monitoring can cause.

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# Notes on cholelithiasis—Case 1

*This case report was one of several written by Dr Martin and published in the New Zealand Medical Journal 1907, Volume 5 (21), p9.*

*Case 1.*—S.R. aet 42. Stout flabby man. Seen during an attack of acute abdominal pain. Pain general over right hypochondrium and over back posteriorly in the infra-Scapular region. No vomiting, no retching, no jaundice.

The point of most acute pain was midway between the 9th costal cartilage on the right side and the umbilicus. Liver not enlarged. Temperature 102 degrees. Pulse rapid and of small volume. The attack was ushered in by a smart rigor, next day he was still in considerable pain when he moved, but the acute stage had passed; Complexion then a muddy yellow not typically jaundiced. Urine contained bile. During the next few days, the pain gradually subsided leaving him with a "dull ache" in the epigastrium.

This was the third sharp attack of abdominal pain this man had had. During the previous three months he had felt constant nagging and uneasy sensations over the liver. His last acute attack was about six months before the time I saw him, and the attack before that was about six months further back. Owing to the intensity of the symptoms, and the fact that the man between the attacks did not feel well, I decided to operate.

Dr. Stowe gave the anaesthetic while Dr. Wilson assisted me. The usual vertical incision was made over the right rectus muscle and the gall bladder and ducts exposed. The Liver was rotated forwards. The cystic, hepatic, and common Bile Ducts were quite clear. The Gall Bladder was then shut off with sterilized cloths, opened and scooped clean.

Over six hundred and forty-nine calculi were removed. You will see on looking at the specimens that many of them are very minute and all are facetted. These calculi were lying in a mass of thick colloid bile substance, sticky to the touch and inky in appearance.

The mucous membrane of the Gall Bladder was then curetted, and douched with warm sterile salt solution. A rubber drainage tube was tied into the Gall Bladder and the Gall Bladder stitched to the fascia and peritoneum in the usual way. Bile drained freely after the operation. On the tenth day the stitches and drainage tube were removed. The after history was uneventful.

Three months after the operation, the sinus had closed completely. This case was simple to operate upon. There were no adhesions worth mentioning. Since the operation he has had no recurrences of symptoms, and now feels strong and active.

**NZMJ Note:** The NZMJ Production Editor (with assistance from Drs Iain Wakefield and Jon Wilcox) selects papers for *100 Years Ago*.

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# Death due to a stingray barb piercing the heart: a New Zealand case from 1939

The tragic recent death of popular Australian wildlife celebrity Steve Irwin—who was killed by a stingray on the Great Barrier Reef—has focused our attention on stingrays and whether anyone has been killed before by one in New Zealand waters.

Like Steve Irwin, this person was killed after being struck in the chest by the barb of a stingray.

## An Unusual Bathing Fatality

By J. B. Liggins

*This report was published in the New Zealand Medical Journal 1939, Volume 38, Pages 27–29.*

While drowning is a common accident in New Zealand, we are fortunate in that attacks from denizens of the sea are few and far between. Shark tragedies such as are experienced in Australia are uncommon.

An unusual fatality occurred in the Hauraki Gulf, ten miles from Thames, on a recent Sunday. A girl of 18 was bathing with her fiancé. There was a heavy off-shore wind blowing, making very smooth sea conditions in the shallow water. It was dead low water at the time—4 p.m. She was wading in under three feet of water close to the shore. Suddenly she called out for help and the man with her rushed to her aid. She collapsed immediately and was carried ashore. A passing motor van was requisitioned to take her to Thames. It was reported that she was breathing when placed in the van but stopped soon afterwards.

I saw her on her arrival at Thames. She was then dead. The Coroner ordered a post mortem examination which I carried out that evening, and I think my findings are of sufficient interest to record; first because of the unusual nature of the injuries, and secondly because of the possible medico-legal complications.

When I examined her after the accident I found her to be a well-developed female about twenty years of age. She was clad in a blue bathing suit which was wet, and there was sand adhering to her skin. She had died very recently.

On external examination, the bathing suit had a six-inch ragged tear over the precordium. Coinciding with this tear was an incised wound exposing the intercostal space. There were three wounds on the inner aspect of the left thigh.

At the post-mortem examination, which I performed later in the evening, the following findings were made

EXTERNAL INJURIES

1. *Chest Injuries*.—An incised wound two inches long and gaping open an inch wide was present running parallel to the fourth left intercostal space about 1½ inches from the mid-sternal line. In the middle of the depth of the wound the intercostal space had

been perforated and there was a circular hole which would admit the first finger. Blood issued from the perforation when pressure was put on the chest.

2. *Injuries to the Left Thigh*.—On the inner aspect of the left thigh in its upper third there were two parallel wounds two inches apart running from the anterior to the posterior aspect. These were clean incised wounds down to the deep fascia but not penetrating it. Between these two wounds was a further incision two inches long but involving skin only.

Close to the labia on the left side, but still on the thigh, was a superficial wound in which the skin was peeled back over an area the size of a form.

3. On both knees were superficial scratches and bruises.

INTERNAL INJURIES

Immediately under the perforation in the fourth intercostal space already described, a tear was found in the pericardium about 1½ inches long. On my opening the pericardium a. gaping, incision was found on the anterior surface of the right ventricle. It was a horizontal, clean-cut incision showing a slight double curve, and measuring an inch in length. The edges of the wound were slightly serrated and there was bruising of the tissues around the wound. There was a perforating wound of the interventricular septum and also of the left ventricle on its left border near the base. This wound of exit measured 3/8” in length. The heart had thus been transfixed by a somewhat rough, pointed instrument. The pericardium and the left pleural cavity were filled with blood.

The lungs were free from water and there was no other evidence of drowning.

At the inquest the man who helped the unfortunate woman from the water stated that when she called f or help she pointed at the water and he noticed a swirl and what he took to be a fin.

The Coroner found that the cause of death was haemorrhage from a wound transfixing the heart caused by the barb on the tail of a sting-ray.

The case I have described was undoubtedly a most unusual accident. Its medico-legal aspect is interesting. If the girl had been found by herself dead on the beach the police would certainly have looked for a murderer of the sexual pervert character. The wounds looked as if they had been made by the sharpest of knives and the penetrating wound of the heart was typically homicidal. The sting-ray must have been of very large size, the barb being at least eight to nine inches long, as the distance between the skin incision in the precordium and the exit perforation on the left ventricle was eight inches.

Reconstructing the fatality from the evidence a fair assumption would be the following:—While the sea was very calm due to a heavy off-shore wind and a very low tide, the bather walked on a sting-ray which was sheltering in the shallow water. It immediately lashed her with its barb, cutting her leg in three places. She was knocked off her feet and on falling forward the fish again brought its barb into action, this time penetrating the chest wall and heart.

I have seen three cases during the last few years in which perforating wounds were caused by sting-rays. They were received by fishermen who were either clearing the sting-ray from their net or dumping it overboard from their boat. Two of these

wounds were through the calf and the other through the wrist, severing several of the extensor tendons. All the wounds became septic, and when first seen were characterised by extreme pain needing ½ grain doses of morphia.

It is known that no poison is secreted by the sting-ray, but on examining a tail and barb recently I noticed a covering of dirty yellow slime, and it may be that this contains some acrid substance. In any case, the amount of pain was out of all proportion to the severity of the wound. It is known that, in common with most fish, a sting-ray will always swim away if disturbed. It has never been known to attack a person as does a shark, but will def end itself very ably if it cannot escape. This case shows that there is need for care in bathing on our northern beaches, which are infested with sting-rays. In such cases as do occur of injury by sting-ray barbs, the wounds are intensely painful and particularly prone to become infected.

## SUMMARY

1. A case is described in which a girl, whilst bathing in shallow water, was stabbed to death by a sting-ray.
2. The injuries simulated homicidal stab wounds of the heart.

 

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# Prandial tussiculation

Amer Ahmad Alkhatib

A 44-year-old man with small cell carcinoma of the lung presented with chest pain, productive cough, and weight loss. The patient was febrile (39ºC) and he had crackles in both of his lungs. A chest radiograph (Figure 1) and chest CT scan (Figure 2) were obtained. What is your diagnosis?

**Figure 1**

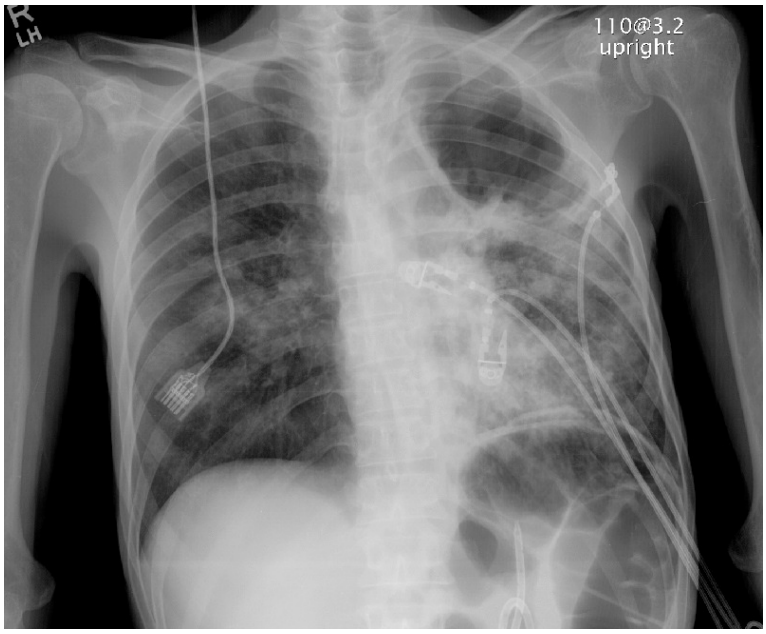
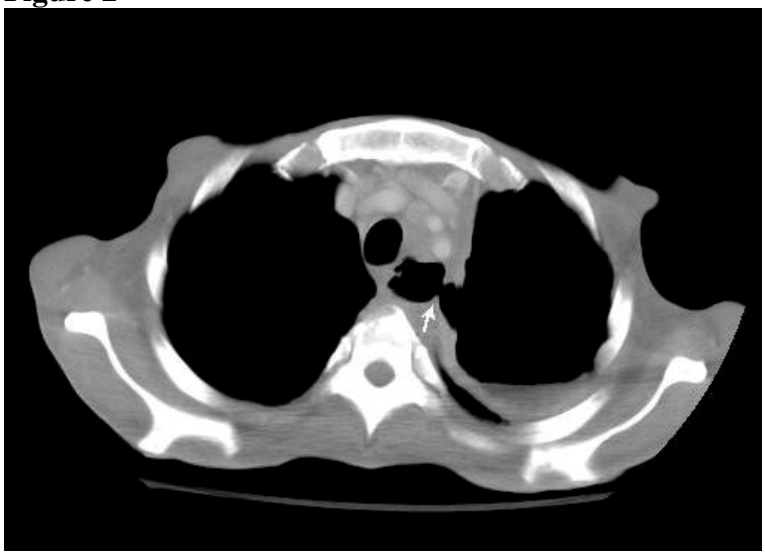



**Figure 2**



## Diagnosis

**Aspiration pneumonia secondary to malignant oesophagopulmonary fistula.**

- Figure 1 shows a left upper lung mass with an adjacent large cavity and alveolar opacities in both mid lungs and the left lower lobe.
- Figure 2 shows that the oesophagus and the left upper lobe are continuous (arrow) consistent with an oesophagopulmonary fistula.

## Discussion

Acquired oesophagopulmonary fistulas are classified into benign and malignant fistulas. Causes of benign fistulas include tracheal-cuff injury with prolonged intubation, corrosive burns, swallowed watch batteries, foreign bodies, penetrating trauma, gunshot wounds, postoperative and endoscopic lesions, oesophageal diverticula, pleural empyema, Crohn's disease, tuberculosis, and mycobacterium avium-intracellular infection.[1]

Most common causes of malignant fistulas are tracheal cancer, oesophageal cancer, and lung cancer.[1] Patients with oesophagopulmonary fistula usually presents with coughing while eating, dysphagia, recurrent pneumonia, dehydration, and weight loss.[2]

Double stenting of the oesophagus and the tracheobronchial tree appears to be the procedure that yields the best overall results for symptomatic relief for patients with malignant tracheoesophageal fistula. To ensure better nutrition and fluid intake, percutaneous enterogastric tubes placement is usually considered.

The prognosis of malignant fistulas secondary to lung cancers is dismal. Without therapy, the patient usually lives less than 7 weeks.[2]

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## Caesarean delivery rates and pregnancy outcomes

Rates of caesarean delivery have risen from about 5% in developed countries in the early 1970s to more than 50% in some regions of the world in the late 1990s. There are many reasons for this other than pure obstetric indication. These include obstetricians' defensive practice, changes in health systems, and patient demand. The question of pregnancy outcome vis-à-vis the increased surgical intervention arises. In this WHO sponsored study, data from 8 countries in Latin America—including Argentina, Brazil, Cuba, Ecuador, Mexico, Nicaragua, Paraguay, and Peru—has been collated. The researchers obtained data for 97,095 of 106,546 deliveries (91% coverage). The median rate of caesarean delivery was 33%, with the highest rates of caesarean delivery noted in private hospitals (51%).

The results—caesarean delivery was positively associated with postpartum antibiotic treatment and severe maternal morbidity and mortality, even after adjustment for risk factors. Furthermore, caesarean delivery was associated with an increase in fetal mortality rates and higher numbers of babies admitted to intensive care for 7 days or longer, even after adjustment for preterm delivery.

The message seems clear.

Lancet 2006;367:1819–29

## Long-acting β-agonists (LABAs) and severe asthma

Much controversy has surrounded the use of β-agonists in patients with asthma ever since their introduction over 50 years ago. Regular β-agonist use is associated with tolerance of the drug's effects and a worsening of disease control. Clearly, if regular β-agonists are suspect so must LABAs be regarded with suspicion. Hence a meta-analysis of LABA versus placebo trials. Pooled results from 19 trials with 33,826 participants found that long-acting β-agonists increased exacerbations requiring hospitalisation (odds ratio 2.6). The risk for asthma-related deaths was increased (odds ratio 3.5). However, the absolute increased risk of death was small—about one extra death for every 1000 patients using these drugs for a year.

And the conclusion? LABAs are powerful and complex and should be used with care. An accompanying editorial favours their use when all other strategies, including maximal doses of inhaled steroids, have failed.

Ann Intern Med 2006;144: 904–12 & 936–7

## Endoscopic surgery vs open surgery for carpal tunnel syndrome

Surgery for carpal tunnel syndrome is one of the most often performed procedures. Apparently open carpal tunnel release may result in prolonged pain at the scar and proximal palm. Hence endoscopic procedures to release the carpal tunnel have been introduced with the presumed advantage of decreased postoperative pain and

subsequently faster return of patients to work. Intuitively one would assume that this would be the case. A randomised trial of these two surgical techniques has been reported from Sweden. Yes, endoscopic surgery was associated with less postoperative pain than open surgery, but the small size of the benefit and similarity in other outcomes make its cost effectiveness uncertain. The median length of work absence after surgery was 28 days in both groups.

BMJ 2006;332:1473–6

## Chronic otitis media and bacterial biofilms

Otitis media (OM) is the most common illness for which children visit a medical practitioner, receive antibiotics, or undergo surgery in the United States. This is also likely to be true of New Zealand, we suspect.

*Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis* are isolated from approximately 25% of children with OM with a middle ear effusion, but polymerase chain reaction (PCR)-based methods have demonstrated sequence-specific DNA and RNA for these pathogens in nearly 80% of cases. Biofilms consist of aggregated bacteria, usually adherent to a surface, surrounded by an extracellular matrix, and have been implicated in several chronic bacterial infections. The question investigated in this report is whether chronic OM is biofilm related. Well it was in 92% of 50 cases studied.

This throws some light on why chronic OM is rather resistant to antibiotic treatment.

JAMA;296:202–11

## Lifestyle of the cardiologist

Cardiologists offer a lot of lifestyle advice—what are they like at taking it themselves? A group of cardiologists in the United States (US) posed a questionnaire to their colleagues on this topic. 471 answered the questions—the average age of the participants was 48.6 years; 7.1% were women. The average body mass index (BMI) was 25 kg/m$^2$, and 8% were obese (BMI≥30 kg/m$^2$); 1.3% were active smokers; 89% exercised ≥1 time/week; and 72% had ≥1 alcoholic drink/week. Red wine was the most frequently consumed alcoholic beverage. Associated cardiovascular risks included dyslipidaemia (28%), hypertension (14%), and diabetes mellitus (0.6%). Four percent had experienced coronary events.

Compared with matched cohorts from the US population, cardiologists reported lower rates of hypertension, dyslipidaemia, and diabetes mellitus, and the rates of smoking and obesity were 1/18 and 1/3 those of the US population, respectively. These data suggest that cardiologists as a group appear to have healthier lifestyles than the general adult US population. This is likely to translate into improved health and longevity among cardiologists.

As expected—a healthy lot. But the 471 were only 59% of the cohort. One can only speculate on the health profile of the other 41%.

Am J Cardiol 2006;97:1093–6

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# New Zealand Food Safety Authority's response to the 'flies, fingers, fomites, and food' article on campylobacteriosis

Nelson and Harris's analysis of New Zealand campylobacteriosis data proposes flies as vectors and transmission agents for *Campylobacter*.[1] It uses ecological investigation of seasonal variation to try to infer causality.

The authors need to remember that associations identified in aggregate data may not apply to individuals. There are many potential *Campylobacter* transmission routes that can lead to illness in humans. Epidemiological studies indicate that campylobacteriosis is predominantly of food-borne origin with poultry consumption/handling considered to be a major risk factor in New Zealand.[2] Food from animals, including red meat, poultry, offal and raw milk can be contaminated. From the farm, the natural environment and the processing premises through to our homes, there are opportunities for spread to humans.

The potential for transmission of *Campylobacter* to humans is not limited to the meat itself or meat-handling utensils (including hands), there being opportunities for contamination during food purchasing and preparation. The relative importance of poultry as a reservoir of *Campylobacter* and cause of disease has yet to be accurately determined in New Zealand but available evidence is suggestive that it is important.[3] While climatic factors may contribute to any role that flies play in the spread of this organism, the higher rates of illness in urban rather than rural dwellers do not support the hypothesis of direct transmission by flies from cattle faeces to food.

We believe that a 'farm to fork' approach has to be taken to minimise food's contribution to New Zealand's unacceptable high rate of campylobacteriosis. There is no 'magic bullet' that can be adopted. Human exposure to *Campylobacter* should be minimised utilising a risk management based approach. A range of interventions involving primary production and primary and secondary processing are likely to be needed to progressively reduce the high load of *Campylobacter* which is entering our food chain. To this end, the New Zealand Food Safety Authority has commissioned work looking at laboratory methods, epidemiology, transmission routes, source attribution and interventions throughout the food chain, in association with both the Ministry of Health and the food industry, as described at http://www.nzfsa.govt.nz

While the fly theory is interesting and may, along with other postulated sources, merit further study in the long term, we agree with the authors that effective programmes to reduce the known risks of direct food-borne transmission must not be delayed.

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# Tobacco harm reduction in New Zealand

McCormick and colleagues' editorial (*Where to next with tobacco smokers?* NZMJ 21 July 2006; http://www.nzma.org.nz/journal/119-1238/2084) challenges us to use harm reduction to reduce tobacco smoking and its consequences.

Snus (pasteurised tobacco) is the best known such product. No one size fits all, and so the Clinical Trials Research Unit (CTRU)—as conveyed in their letter *Working with what we have before getting into bed with the tobacco industry*. NZMJ 18 August 2006; http://www.nzma.org.nz/journal/119-1240/2139)—is trialling a range of nicotine replacement therapy (NRT) and enhanced fast acting NRT products. Besides helping people stop smoking, these products may assist those with emphysema or schizophrenia who find it too difficult to quit. The more choices available to smokers, the more will find a product to their liking.

Cigarettes have killed some 200,000 New Zealanders so far. Moral outrage is justified, but should not blind us to future health gains obtainable by astutely using industry know-how for public health ends. The industry does not know how to make safe cigarettes, but it does know how to make much safer smokeless products such as snus.

Snus has decreased Swedish male smoking and cancer rates below New Zealand levels (14% of Swedish men smoke, 20% use snuff; 23% of New Zealand men smoke). Snus' clinical effectiveness needs testing by a randomised controlled trial. If New Zealand research can prove that nicotine-only products are just as effective, well and good.

Sweden has lowered male smoking rates dramatically while spending 23% as much on tobacco control per capita, while their cigarettes are twice as affordable as in New Zealand. (Swedish expenditure at community level was not counted.) Treasury and the Ministry of Health needs to take a hard look at the effectiveness of the New Zealand regulated market model of tobacco control, not to disinvest, or to dismantle it, but to consider tweaking it, to get more fully-researched harm-reduction products on sale alongside cigarettes, at half the price. Every smoker switching to smokeless means reduced tobacco mortality.

Further data on all known possible harm reduction options for smokers are found at http://www.smokeless.org.nz

**Conflict of interest:** SmokeLess is not in receipt of any tobacco, smokeless, nicotine or pharmaceutical company funding, nor is the writer.

Murray Laugesen
Chair, SmokeLess New Zealand Inc

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# Media report of rare rhabdomyolysis cases seems to have triggered reluctance among some New Zealanders to use statins

In October 2005, a news item was aired on TVNZ's *CloseUp* that detailed the side effect of rhabdomyolysis related to the use of simvastatin. Two members of the public were interviewed who had experienced rhabdomyolysis and had suffered permanent injury as a result. Two case fatalities in New Zealand were also discussed.[1] Eleven months later, cardiologists are finding that the public are apprehensive about being prescribed simvastatin because of an unjustified fear of a rare side effect.

The evidence supporting the use of statins in primary and secondary prevention of coronary disease is irrefutable.[2–6] Should 11 people with previous myocardial infarction stop taking their statin, one preventable coronary event within that group will occur within 5 years. If 25 people with a similar history were to stop taking their statin there would be one preventable death within the same period.[4]

The penetration of statin use however remains poor.[7] This is a problem primarily of access to health services but also touches upon the issue of public health education in primary prevention. The importance of cholesterol management is a subject that is probably well entrenched in the collective public consciousness. However what is perhaps not realised is that practise has changed and pharmacological management has become an accepted early intervention in individuals with vascular risk factors, in conjunction with dietary and lifestyle changes rather than following them.[8]

Rhabdomyolysis associated with the statin drug class is rare. Fatal rhabdomyolysis is considered to be an extremely rare complication of statin use, lower than one case per million prescriptions.[9] Myositis and myopathy are more common adverse reactions which when recognised early are reversible. Dose reductions, trialling alternative statins, or alternative lipid-lowering agents are all accepted methods of subverting this complication. Testimony to the safety of statins is the fact that the Medicines and Healthcare Regulatory Authority (MHRA) in the United Kingdom has recently approved sale of over-the-counter generic simvastatin.[10] The purpose of such a move was to make the drug more accessible, perhaps to those who cannot afford a doctor's appointment.

Concerningly, Pfizer and Merck have sited zero to negative sales growth in New Zealand for simvastatin and atorvastatin since October 2005.[11] With an aging population and reducing targets for LDL cholesterol management, the expectation would be an overall increase in total prescriptions.

Negative publicity has had a significant impact on the perception and uptake of an extremely effective life-saving medication. On a population basis, such an impact could have profound flow on effects. Doctors need to be aware of the potential rare side effects of statins and provide information to patients when prescribing, particularly as many drugs in New Zealand come without product inserts. However prescribers also need to reassure patients that serious adverse reactions are rare and that the potential benefits of remaining on treatment are profound.

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# What a performance

In their article *Quality improvement in New Zealand healthcare. Part 4: achieving effective care through clinical indicators* (http://www.nzma.org.nz/journal/119-1240/2131) Buchanan et al promise to tell us about performance indicators, key performance indicators (KPIs), and clinical indicators, defining what they are, how they differ, and how are they used. The authors also mention "continuous quality improvement," or CQI, a term so fraught that it would be better consigned to oblivion. So how far do we get with the others? KPIs get no further mention in the article, so we can we let that one go.

The authors define performance indicators not in terms of what they are, but of what they do. We learn that they can be either *summative* mechanisms for external accountability, and *formative* mechanisms for internal quality improvement. These mechanisms are mutually antagonistic, since they lead to fights over money, with CQI (continuous quality improvement) the helpless bystander.

Clinical indicators, we learn, are a "subset of performance indicators…variously defined…an objective measure of either the process or the outcome of patients care in quantitative terms…a powerful means of improving the effectiveness of patient care."

Sound nice, but it is not as easy as all that. "There are different objectives for clinical indicators depending on who is using [them] and whether the assessment is intended to be *summative* or *formative*." That is to say, CQI falls off when the money dries up, and the doctors have a clear obligation not only to keep spending but also to show that the money does some good.

As I see it, there is no way that clinical indicators are going to be of any help at all if they mean different things to different people. How can they be "objective" when they are "variously defined…do not measure quality directly", and are used by different people with different objectives? We have a semantic problem here.

What is significant is that was an initiative launched 20 years ago in the United States that got the summative/formative debate going. The health funds wanted to know what their money was being spent on, a question that has never been asked in this country.

The resolution of the matter lies not in a lot of useless data about performance indicators, KPIs, and clinical indicators. A full investigation of all third-party funding is long overdue. No country has either fully socialised, or fully privatised, its health services. We have chosen to lean well towards the socialised end of the spectrum and the collapse of the waiting lists now tells it own story.

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## Response

By way of explanation for the lack of detail on performance indicators in our article (Part 4 in the *Series*) we had initially intended to discuss performance indicators and clinical indicators in the same article but the topics proved too complex for that and so measurement for performance monitoring and control is described in more detail in the article in this edition (Part 5 in the *Series*).

Performance indicators are numerical measures of different aspects of organisational performance and a *key* performance indicator (KPI) simply reflects the concept that some aspects of performance that need to be maintained or improved are more important than others. When a range of these important aspects are selected and suitable measures chosen then a set of KPIs exists. The selection of KPIs is subjective and depends on the setting and circumstances. There are no "automatic" KPIs.[1]

In our article we state that clinical indicators "are essentially an objective measure of either the process or outcome of patient care in quantitative terms." Provided that the numerator and denominator of the indicator are clearly defined, and the data is collected and analysed properly, then the *measurement* is "*objective.*" Like any clinical measurement the result must then be interpreted by someone with the requisite knowledge and skill who understands the clinical context.

Dr Ridley-Smith has identified a semantic problem and asks how can clinical indicators be "objective" when they "have been variously defined," "do not measure quality directly" and "are used by different people with different objectives"? As indicated above, the numerical result for a clinical indicator is obtained by objective measurement but there is a subjective element in the interpretation. To maximise the objectivity in measurement and consistency of interpretation of clinical indicators each indicator must be carefully selected for the purpose, be very clearly defined and be accepted, understood and owned by the clinicians and managers who use it. One of the purposes of our article was to explain the attributes of clinical indicators and raise points for consideration in the selection of suitable indicators.

With regard to the tension that may arise when there are different objectives for clinical indicators depending on who is using them and for what purpose Dr Ridley-Smith is perceptive in his comment that "…CQI falls off when the money dries up…" The problem is, however, often one of management rather than funding and forsaking efforts to improve clinical indicators and the usefulness of the data derived from them is likely to hinder rather than help the resolution of such difficulties.

We have no particular quarrel with Dr Ridley-Smith's plea for an investigation of third-party funding, but believe that it is a separate issue from the assessment of quality and effectiveness of care by means of clinical indicators. Such assessment is important regardless of the arrangements for funding.

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## What is common sense?

Common sense means paying attention to the obvious. This is not as easy as it sounds. We all have vivid imaginations, and we tend to get lost in our fantasies.

When fantasy replaces common sense, life becomes farcical and even tragic. Life is a series of ordinary events that follow the laws of logic and probability. These ordinary events are indifferent to our fantasies and require the careful, accurate navigation of common sense.

I learned the lesson of common sense as a third-year medical student. I was doing an internal medicine rotation at a Veterans Affairs (VA) hospital and working with interns, residents, and attending physicians.

One day, on morning rounds, we examined a patient with a black tongue. The intern assigned to that patient had researched all the causes of a black tongue and was eager to demonstrate his new knowledge. As the intern started to lecture us, the attending physician interrupted him and asked the patient if he uses black cough drops. The patient smiled, opened the drawer of his night table, and took out a package of Smith Brothers black cough drops.

The intern's face turned red, and we all laughed. The intern was so focused on being a doctor that he forgot to ask his patient an obvious question. It's been 35 years since I was a third-year medical student, but I still have a vivid memory of that day and that lesson: use common sense and pay attention to the obvious.

My 30 years of medical practice have taught me the lesson of common sense again and again. Eventually, I realised that society in general, and modern medicine in particular, lack common sense. This is why societal and medical problems are rarely solved. Let's apply common sense to healthcare.

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# George David Abbott

*Paediatrician (15 Aug 1939—10 Aug 2006)*

Associate Professor George Abbott, who died after a short illness with lung cancer, was recognised on both sides of the Tasman as one of the most respected paediatricians of his generation.

George was born in Lahore and moved with his family to Christchurch after the partition of India. He graduated in medicine from the University of Otago in 1964 and gained MRACP in 1968 (FRACP in 1973). He completed his MD in 1971 on "Bacteruria in the Newborn".

Urinary tract infections and vesico-ureteric reflux remained major research interests over many years, and with the late Ross Bailey and others he contributed to knowledge of the familial nature of this condition and the prevention of long-term consequences.

George continued his training in microbiology and infectious diseases at the Hospital for Sick Children in Toronto, and then at Boston Children's Hospital. On returning to Christchurch in 1973, he was appointed as Senior Lecturer in the new Christchurch School of Medicine and, together with Professor Fred Shannon, developed the undergraduate teaching programme in Paediatrics. He became an Associate Professor in 1981.

George Abbott was an outstanding clinician, teacher, researcher, and advocate for child health. His reputation was such that George was always the paediatrician other paediatricians took their children to when they were sick. He was a general paediatrician but with many sub-specialist interests. He ran the Paediatric Oncology Service for 20 years and established specialist clinics, often bringing in an Adult Medicine colleague, in areas such as urology, endocrinology, rheumatology, and inflammatory bowel disease.

His knowledge was encyclopaedic. He served for 15 years (much longer than the usual term) on the RACP written examination committee. Rumour has it the committee had an "Abbott" test for new questions. If George knew the answer, then the question might be of some use; if he did not know, then the question was discarded as too obscure.

George also enjoyed teaching and he excelled at it. Many undergraduates and young doctors responded to his exacting standards and were inspired by him to take up careers in Paediatrics and often to follow an academic pathway. The student body in Christchurch recognised his contributions by awarding him their Best Consultant Teacher prize on three occasions and the Medical School awarded him their Gold Medal for teaching excellence in 2003.

George held many administrative roles in Christchurch and was Chairman of Paediatric Services from 1985–92 and Clinical Director from 1999–2006. He did not always enjoy this role but he was a good administrator and a very effective advocate for improved child health services. His development of an Acute Assessment Unit significantly reduced hospital admissions and he strongly supported the introduction of outreach services.

As a measure of the esteem in which he was held by his colleagues, George was elected to the two highest posts available to Paediatricians in New Zealand; President of the Paediatric Society of New Zealand (1993-97) and Chairman of the Board of Paediatrics, RACP (1995–2000). Through these positions he influenced the appointment of a Child Health Advisor in the Ministry of Health and a national review of Paediatric Tertiary Services.

He had many other RACP roles, including being Senior Paediatric Examiner (1989-95) and a member of Council (1997-00), and was awarded the John Sands Medal for outstanding service to the College and its Fellows in 2002.

George will be remembered as being strongly supportive of nursing, allied, and clerical staff, and as a clinician who worked in a truly collaborative way, asking for and valuing the opinions of all staff involved in caring for a child and their family. He was a private man but those who knew him well enjoyed his sense of humour and his ability as a mimic.

Away from work, George had a number of interests. These included a love of literature and particularly poetry. His personal landscape was the mountains and the rivers of Canterbury and he was an accomplished fly fisherman. He had a lifelong interest in many sports. He was a good cricketer and was honorary Medical Officer to Canterbury Cricket, and he enjoyed coaching junior teams which he did until just before his illness.

For all his achievements George would undoubtedly count his greatest contribution as being able to help and care for the many sick children and their families to whom he dedicated so much of his life. He will be sadly missed as a friend and colleague.

George is survived by his wife, Louise; children, Christopher, Bronwyn, Felicity, Jonathan, and Georgina; and four grandchildren.

We are grateful to Professor Brian Darlow for writing this obituary.

 

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# John Wellesley Evan Raine

*12 March 1919–12 July 2006*

John Wellesley Evan Raine died recently aged 87. He was born in Wellington, educated at Scot's College, and apart from time at Medical School and doing his surgical training he spent his entire life in that city.

Evan was descended from an old Wellington family of merchants.

He spent a year at Victoria University College before proceeding to Dunedin. He was a Blue at both hockey and golf.

He graduated in 1941, and after a year on the house at Wellington, he joined the RNZAF and served in the Pacific, notably at Guadalcanal and Bougainville. After the war he joined the Territorial Army, which he served in for many years, retiring as a Major.

During his time in the services he married Eleanor Luke who died in 1978. He subsequently married Pat Cryer.

After returning to Wellington with his Fellowship, he practised as a general surgeon advancing up the pyramid from junior consultant to become a senior consultant. He became Chairman of Surgical Staff in 1969.

After retiring from the hospital staff he continued in consulting practice and served as director of medical services for the Justice Dept and provided services to the Union Steamship Co, NZ Railways, and ACC. He enjoyed teaching, and remained an honorary postgraduate tutor in surgery for 20 years after he retired from the hospital.

Evan was awarded an OBE in 1980 and was elected a Fellow of the NZMA. He also had a long association with the Royal Australian College of Surgeons as an Examiner, including a term as President (1974–75). He devoted a lot of time to these roles.

Evan loved skiing and golf and was a devotee of classical music.

Surgeons worked hard, long hours and his family say that he was a doctor during the week and a father at the weekend. This was typical of the provider-father of his time. Fortunately, Evan had a long retirement in which he learned to relax and enjoy time with family and friends.

This is adapted by Roy Holmes from a longer obituary written by Mike Crean entitled *Surgeon of his times* that was published in *The Press* (Christchurch) on 29 July 2006. Additional information and the photograph were provided by Evan's family.

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# William Reisiswyl Trotter

*Smoke-filled waiting rooms, starched white medical coats, and housecalls—recollections of the old family GP*

Amidst today's heated debate and concerns over the medical system, the recent death of my father rekindled my fond memories of the GP system of old. My Dad, Dr William (Bill) Trotter, who passed away at the fine age of 91, epitomised the now rare sole-practice family GP.

I have always been immensely proud of my tall, handsome, doctor father—resplendent in starched white coat, always accessorised with a shining stethoscope, and, to my mother's consternation, a firmly clenched Peterson pipe—all set off by a scattering of burn holes which rather marred the professional image!

Dad was constantly puffing and gurgling away on his pipe, the occasional splutter sending a flurry of sparks flying. Family and patients alike were well practised at extinguishing various smouldering items of clothing—or furnishing—after such a paroxysm.

Bill had a dramatic way of commanding attention as he spoke. He would hold a lit match to his pipe, the flame growing ever closer to his fingers, all the while the tension of the onlooker mounting, until at the very last moment with a light shake of the hand the flame would die out.

Dad set up practice in Newmarket in the late 1940s which at the time was a working class area of Auckland. Rundown railway houses and a high street of shops, pubs, and light industry provided his main clientele.

Dad didn't bother with an appointment system—except in token on one "maternity" afternoon. Appointments just weren't feasible, given his fondness for becoming deeply entrenched in conversation with his patients. If they were interested in boating or fishing then so much the worse!

My mother, a nurse who Dad had fallen for—apparently in a broom cupboard at Greenlane Hospital—worked with Dad, and in the school holidays I would take over to give her a break and earn some pocket money. Consulting hours were loosely 12noon–4pm and then 7pm onwards each weeknight except Wednesday—Dad's one night off. He routinely celebrated this freedom with a roast dinner followed by a vanilla fudge-making episode—and preceded by a welcome gin.

The evening surgery was always a source of amusement. Under his regime, Dad's patients would gather in the street outside his surgery on Broadway awaiting his inevitable late arrival (caused by the nightly habit of misplacing his essential matches or tobacco). Then, as soon as the door was unlocked, the most able would race up the three flights of stairs to get first in line for their consultation. Presumably this sorted

out the level of severity of illness—to the detriment of the weak, but no doubt aiding diagnosis!

My job was to locate the handwritten patient cards in the black metal filing cabinets (not easy given Dad's writing) and place them in order of arrival. Many had multitudinous staples holding them tenuously together and occasionally I would accidentally knock this unstable pile over—a dreaded event, as the waiting group then had cause to argue and jockey to re-establish their position in the queue.

After organising his surgery desk, treasured ash tray, and ensuring his pipe was well alight, the magnificent, almost mystical, white-clad figure of Doctor Trotter would appear in the doorway in a cloud of smoke calling "who's first?"" He would always place a kindly arm around his patient's shoulder (probably not permitted today!) and without fail utter the same words "And what brings you to see me today?"

Knowing that they may have to wait several hours for their turn, Dad's patients brought books, cards, knitting, crosswords, and other hobbies with them to while away the time. Smoking was the norm in those days and as the night wore on the waiting room atmosphere became progressively thicker with smoke while 1YC hummed incessantly on the old mantle radio. Actually it could be quite an enjoyable social gathering some nights! My least favourite task was emptying and cleaning the ashtrays every half-hour or so—a job which happily put me off ever desiring to smoke.

In those days the medical subsidy seemed sufficient to cover the basic costs of the practice and Dad more often than not gave me his "No charge" look when his less financially fortunate patients eventually escaped his conversational trap!

Accounts were also in fashion and some patients built up a huge debt never likely, or expected, to be paid. Once in my youth, and enthusiasm for a new school-ball dress, I sent out an account with a note saying "Please pay immediately—this is not a charity." A very irate recipient rang Dad and he had to do quite a lot of talking to get us out of that trouble. I don't recall the bill being paid though! I do recall that the cost of a consultation with Dad was equivalent to buying a new good brand lipstick—and I have noticed that my "cosmetic index" remained true for many years!

The'60s marked the emerging drug problem in New Zealand. Once we arrived to find that an enterprising thief had hidden in the toilets and overnight sawn through the walls to gain access to the surgery—stealing a quantity of drugs.

Another time, Dad's medical bag was stolen from his car boot. We received an anonymous phone call instructing us not to call the police and to collect the bag from the phone box outside the Auckland Domain at 10.30am. With great trepidation we went—and indeed it was there—minus the drugs. In fact I still have that bag—I haven't the heart to throw it and the old medical instruments away after surviving such an adventure.

Another alarming incident occurred late one Friday night as Dad and I were leaving evening surgery with the week's takings. On reaching the carpark, a man leapt out in front of us with a rifle. Not one to let his hard-earned money be so easily taken, Dad pushed me to the floor and with the gears in neutral gunned the accelerator, causing the assailant to leap aside, and enabling our escape. I don't think we ever told the

police about those events—or realised in those more innocent days how serious they potentially were!

Dad's path to becoming a doctor intrigued and inspired me. Born the son of a Presbyterian minister in Greymouth, at the age of 3, Dad travelled to Dunedin over the Southern Alps in a horse-drawn carriage, where the family set up a ministry at St Leonard's.

The youngest of four (with three sisters) Dad reported his early life as frugal but enterprising. He fashioned his own (not particularly speedy!) skis from two lengths of wood and assembled a makeshift bike from parts unearthed at the local tip. He absolutely detested his school years and any form of academia. His youthful highlight was building himself a sailing boat in the coal shed. He subsequently raced her very successfully on Otago Harbour, which imbued him with a life-long love of the sea. He left school at the earliest opportunity to work in a number of unfulfilling positions including office clerking, shovelling a never-ending pile of phosphate at the Ravensbourne Fertiliser works, and as a builder's labourer.

At age 21 he finally decided he had more to give to life and vice versa, so returned to study—taking 2 years to gain a pass mark in the required Latin. He distinguished himself by gaining 3% in the exam the first time round, breaking all previous records of failure. However he persevered and eventually gained entry to Otago Medical School.

Money was short and Dad covered expenses by driving a taxi at night after lectures. Given that Dunedin was not exactly a thriving metropolis he was able to get a lot of study done and excelled at several subjects!

At Medical School, Dad befriended fellow student, the now Sir Tom Davis (destined to be PM of the Cook Islands, a three-star general, and work for NASA). He and Dad had many adventures.

Daily surgery in Newmarket had many memorable moments. One such event involved a very ample woman who climbed onto Dad's examination plinth, which unfortunately did not accommodate her girth and propelled her onto the two-bar radiant heater below. I can still clearly envisage the dual pattern emblazoned on a sensitive part of her anatomy. I could not look Dad in the eye—and am still wary of plinths!

Another time, Dad was making a housecall when he tripped on a floor mat while approaching the patient and landed up in bed with the woman in question. Actually Dad was rather handsome and desirable in those days so he evidently did not receive a complaint! In fact I think she made a miraculous recovery.

Of course the 50s and 60s were the long-lost days of house calls which Dad carried out every morning after delivering my sister and I to school. This daily journey provided one of the only times we had him to ourselves as children—since his work involved most evenings and weekends. Eventually Dad joined a roster of doctors for weekend work—but there was no such thing as weekends or even Christmas Day off in the early days.

Accompanying Dad on his weekend house calls armed with books and toys was a favourite activity. I was quite happy to wait in the car, and many of his patients rewarded my dedication! If they couldn't afford to pay with money there was always

fresh-laid eggs, crisp vegetables, fragrant flowers, delicious home baking, or handknitted toys instead. No-one went without care because of money. Christmas saw our house festooned with literally hundreds of cards from Dad's adoring patients.

In return, Dad knew each family intimately—he had brought many of their children into the world and been with them through their triumphs and failures—and some their eventual demise. He was a great psychologist and philosopher—always very aware that there were generally underlying reasons behind every complaint. He had a deep understanding of human nature and what made people tick.

His wisdom and plain common sense has again become fashionable and "politically correct" after many years. Never one to bow to fads, Dad persisted in eating eggs and butter daily, and always preferred to let nature take its course. He resisted putting antiseptics on wounds believing they would retard the natural healing. He would often say, "Well—your ailment will last 2 weeks with medication and a fortnight without".

When he retired, Dad was a bit lost without medicine, despite his many interests. He accepted a position as Medical Officer at the Mangere Immigration Centre and spent several very fulfilling years helping a new group—the Vietnamese refugees—into life in New Zealand.

Last year in July, we farewelled Dad. We put his Peterson pipe in one hand and a bottle of gin in the other. We all miss him dearly.

We are grateful to Barbara Harris (nee Trotter)—Deputy Director, Trade & Investment, British Consulate-General, Auckland—for writing this obituary.

 

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# Morbidity, Performance and Quality in Primary Care: Dutch General Practice on Stage

GP Westert, L Jabaaij, FG Schellevis (eds). Published by Radcliffe Publishing Ltd (Oxford), 2006. ISBN 1846190533. Contains 304 pages. Price GBP 29.95

This compendium of 31 short but informative chapters seeks principally to report results of the second Dutch National Survey of General Practice (DNSGP-2) in the Netherlands, during 2000–2002, as presented at a 2004 international conference, *Dutch general practice on stage*.

Organised broadly around the themes of this conference, this very readable book has six parts: Introduction, Health and disparities, Use of care, Organisation and communication, Quality, and International perspective and the future. It describes general practitioners' role and performance as gatekeepers of the Dutch health system.

By international standards, general practice in the Netherlands is advanced, and the book reflects growing international interest in monitoring and understanding how 'cottage industry' models of primary care can contribute to population health and contain mounting health costs.

The first two chapters of the book summarise respectively the major findings and design of the DNSGP-2, including comparison with the first Dutch National Survey of General Practice conducted in 1987. Involving 104 practices, 195 general practitioners, and approximately 400,000 patients, the DNSGP-2 detected a growth in demand for Dutch general practice services, and found these to be efficient and meet most patient needs for accessible, community-oriented and high quality care. Chapter 3 describes the organisation of the Dutch health system as of 2001, before noting that in 2006 compulsory standard insurance has replaced the Netherlands' two-tier system of public-private financing.

Subsequent chapters detail findings for specific, individual topics including disease prevalences, migrant health, prescribing practices, workload, and patients' perspectives on quality. Three chapters compare the Dutch experience with developments in the United Kingdom.

Parts of the book, such as 'organisation and communication,' could be better fitted, and chapters from outside the Netherlands, for example by Kawachi, sometimes sit uncomfortably despite their purpose of putting Dutch primary care into international perspective. Nevertheless, the editors have produced a comprehensive, representative and clear portrait of general practice in the Netherlands, which is particularly relevant to New Zealand because of the similarity between the two countries' models of general practice. International comparisons are difficult without common methods of data collection. But the DNSGP-2 complements our 2001–2002 National Primary Medical Care Survey, and provides lessons including technical advantages offered by the Dutch patient enrolment system.

The production quality of the book is good and its soft cover form is reasonable value at around NZ$90 for anyone interested in apprising themselves of recent international developments in primary care.

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# What Seems to be the Trouble? Stories in Illness and Healthcare

Trisha Greenhalgh. Radcliffe Publishing Ltd (Oxford), 2006. ISBN: 184619122X. Contains 128 pages. Price GBP 19.95

This book shows the importance of descriptive narrative (storytelling) in healthcare. Professor Greenhalgh finds stories everywhere, from patients and their carers, within hospital organisations, and even in the process of systematic review.

With great expertise, and drawing on diverse authorities ranging from Aristotle to websites, she analyses and then weaves the stories into a common theme, demonstrating their importance in everyday patient management, in teaching, in effecting change in healthcare organisations and even in protecting future research against ever-burgeoning bureaucracy. It is an undoubted tour-de-force.

Each chapter is heralded by a brief narrative, which is then analysed and used as a starting point for developing more general concepts. A simple example discusses the pregnant patient's fear of taking insulin, necessary to treat her diabetes, which becomes understandable in the light of the story of a close relative having died of complications shortly after starting insulin. No randomised double blind controlled trial will ever identify these issues, of crucial importance in management.

Such trials may help identify the best insulin regimen, but evidence-based medicine is only part of good care and is firmly put in its place. Another example looks at the annual UK National Confidential Enquiry into pregnancy deaths, which has traditionally been illustrated by case studies told by doctors and nurses about the people who died.

It was recently decided to remove these as part of a drive towards more 'evidence-based' healthcare. Anecdotes are unscientific and would introduce the reader to bias. But these stories have great emotional impact and are the most interesting part of an otherwise dismally dull document. The teenage girl frozen to death in the grounds of a hospital after being discharged late in the afternoon following a miscarriage cannot be reduced to bullet points. Stories embrace complexity and are of much greater impact.

For the more casual reader, some of the book is hard going. But it is well worth the effort. It's a rare and brave book that battles successfully against the overwhelming impression that good medicine means the exclusive use of evidence-based data, trials, protocols, and audit.

Especially in secondary care, it is so easy to treat the disease (as defined in our textbook) rather than the sick person defined by their story, and Professor Greenhalgh corrects the balance. She provides a rational basis for the importance of anecdote without which medicine is not just dry but, more importantly, very incomplete. She recognises the importance of stories at many levels and in ways not obviously evident. She even shows you how it may help get your application approved through the ethics committee!

My eyes have been opened to new ways of thinking from reading this book, something I hadn't expected from its cover. Buy it!

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