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EDITORIALS

Patients, professionalism and politics: a new beginning

The role of health professionals in our health service has changed. The move from paternalism to autonomy has strengthened patients' rights. This we applaud. At a collective level, however, changes initiated politically have diminished the role of professionals in health decision-making. The management of health in New Zealand has been taken over by managerialists philosophy following accusations of "provider-capture", waste and expense.

Our hospitals were made subject to the Commerce Act, reinforcing dominance of the financial model. Competition through tendering was proposed to reduce costs of goods and services. However, the new system was complex and resulted in expansion of the health finance bureaucracy. The tendering process was expensive, often excluded involvement of health professionals and led to numerous faulty decisions. This reduced flexibility to meet individual and local needs. Gaps in patient services inevitably appeared forcing increases in spending. After years of the competitive model there has been little nett gain in the care of patients in hospital other than that due to technical advances.

Doctors and other health professionals in the hospital service were largely excluded from effective participation in planning and policy. Whilst some formal mechanisms have been introduced to try to correct this, in many hospitals the underlying attitude of distrust and antagonism has precluded open consultation and effective planning. There are parallels with the U.K. where "the substitution of market mechanisms and competition has fractured the traditional mechanisms for local accountability. National Health Service providers are governed by trust boards, with no democratic or legal mechanisms to ensure that they uphold the interests of the local communities from which they draw patients."[1] In New Zealand the harmful effects were made worse by the reduction in government spending on social services which mirrored the views of the World Bank and International Monetary Fund at the time. This created further unmet demand and encouraged international for-profit investment in our traditional public services. The extension of a free market in these services is promoted by the World Trade Organisation and poses a serious threat to "universal coverage, solidarity through risk-pooling, equity, comprehensive care, and democratic accountability."[1] It endangers our ability to plan and provide a system of health delivery that is fair to everyone, including the poorest groups.

The change of government in December 1999 to one claiming a commitment to the strengthening of social services offers a time for health professionals to engage government, official agencies and hospital administrators in dialogue to promote a collaborative approach. The Council of Medical Colleges in New Zealand has stated: "Medical Colleges want to contribute to open dialogue with community and government agencies that will lead to the development of sustainable, quality, and cost-effective solutions to the problems facing the health system."[2] This approach presupposes the democratic involvement of individuals and organisations through open consultation. Such shared decision-making was not possible in the environment created by our last government.

Strengthening of professionalism amongst doctors is important to support the aspirations of the community. A recent article in the New England Journal of Medicine directed attention to this issue: "Professions protect not only vulnerable persons but also vulnerable social values. Many values are vulnerable: individuals and societies may abandon the sick, ignore due process in judging the guilt or innocence of a person accused of a crime, provide inadequate support for education, propagate information that suits those in power while stifling different perspectives, and so on... When professionalism in these core social activities becomes unsteady, it marks the emergence of societal problems... Respect for human worth, trustworthiness, and protection of important values are not the exclusive province of professionals; neither is competence. But they are particular obligations of professionals."[3] The article argues that doctors must resist incentives that place at risk the trust which exists between them and their patients. They should uphold the needs of human life and health, recognising the vulnerability and potential of everyone for health or illness.

It is time to re-orientate management of the health system so that these professional values and the interests of patients are our central goal. Whilst the new government will wish to see cost-effective expenditure it is important that financial control systems are not ends in themselves and do not create gaps, and failure of quality, in our health service. Involvement of the public and accountability to the public must again be fundamentals of the management of health. We need to ensure that professional values and the science of medicine are restored to a central role in planning and are not put at risk by transient economic theories.

The Editors

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Soy formulas and the effects of isoflavones on the thyroid

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In November 1998 the New Zealand Ministry of Health (MOH) issued an update on its position on soy formulas.[1] The position paper was a response to questions raised about the safety of soy formulas. These concerns focused chiefly on the high levels of exposure to phytoestrogens for soy-formula-fed infants. The concerns are not new, having first been raised more than a decade ago by phytoestrogen researcher Kenneth Setchell. He compared the phytoestrogen exposure in soy formulas with the occurrence of clover disease in sheep, which was an infertility syndrome that led to permanent histological changes in the uterus and ovaries.[2] Setchell subsequently quantified the levels of phytoestrogens in soy formulas.[3]

Others have also expressed concerns regarding the potential developmental toxicity of phytoestrogens. In 1993 Daniel Sheehan, who is Director of the United States Food and Drug Administration's National Centre for Toxicological Research Estrogen Base Program, in presenting the case for expanded phytoestrogen research noted that the potential risks to infants from phytoestrogens should not be ignored.[4]

A risk assessment, in which daily levels of exposure to phytoestrogens in infants fed soy formulas were calculated,[5] led to calls for stricter control of soy formula sales.[6] This call was based on the fact that infants fed soy formulas were exposed to higher relative levels of phytoestrogens than those found to disrupt the menstrual cycle of adult women[7] and that the same compounds were known inhibitors of 17-β-hyroxysteroid oxidoreductase *in vitro*.[8] It was argued that there was, therefore, potential for the modification of key imprinting events affecting the development of many physical, physiological and behavioural characteristics in the neonate.

Internationally, there has been increasing concern regarding the potential for adverse effects in infants fed soy formulas. In July 1996, the UK Department of Health warned that phytoestrogens found in soy formulas could affect the health of infants. In issuing advice to health professionals the Chief Medical Officer said soy formulas should only be given to babies on the advice of a health professional.[9] That same year the UK Government's Food Advisory Committee asked manufacturers to investigate the removal of phytoestrogens from soy formulas.

In 1998 the United States Environmental Protection Agency's Endocrine Disruptors Screening and Testing Advisory Committee examined priorities for research into human exposures to endocrine disruptors. The phytoestrogen content of soy formulas was established as one of six topics requiring priority research.[10] In 1998 the Australian College of Paediatrics stated that the use of soy formulas might not be without side-effects as the long-term effects of contaminants, such as phytoestrogens, were unknown.[11]

The New Zealand MOH position statement discusses the appropriate use of soy formulas in paediatric practice and the risks posed by phytoestrogens. In particular it emphasises the potential for soy formulas to cause thyroid disorders in infants. On this issue MOH have recommended that:

- Clinicians who are treating children with a soy-based infant formula for medical conditions should be aware of the potential interaction between soy infant formula and thyroid function and consider assessment of thyroid function if satisfactory growth and development is not achieved or maintained.
- Clinicians treating infants with hypothyroidism should closely monitor thyroxine replacement in infants fed with soy-based infant formula or consuming high levels of soy-containing infant foods, as some of these infants may require a higher than usual dose of thyroxine to maintain a euthyroid state.

The MOH is correct in its assessment of the risks of thyroid harm in infant fed soy formulas. This paper seeks to define these issues more clearly and also to detail the risks to others who are exposed to high levels of soy phytoestrogens.

The goitrogenic effect of soy

Scientific reports of the goitrogenic effect of soy were first reported in the 1930s when it was found that goitre could be produced in rats fed soybeans[12] and in chickens fed a ration containing 25 per cent soy meal.[13] Further investigation of the goitrogenic effect of soy was limited until 1976 when it was found that rats fed soy developed malignant hyperplastic goitre if iodine was deficient. However, the goitrogenic factors were not identified at that time.[14]

As soy became part of the western diet, reports of thyroid disorders emerged. Several papers from the late 1950s and early 1960s reported cases in which infants fed soy formulas developed goitre.[15-18] Manufacturers subsequently increased the levels of iodine and reports of goitre in soy-formula-fed infants ceased. However, it has been shown that the thyroxine requirements of hypothyroid infants fed soy formulas are higher than in those not consuming soy,[19-20] indicating that the increased levels of iodine may not have entirely eliminated the earlier problems.

Recently it has been shown that soy can have profound effects on thyroid function in adults. A study by Japanese researchers concluded that intake of soy by healthy adults could cause enlargement of the thyroid and suppress thyroid function.[21] That study, from the Ishizuki Thyroid Clinic, recorded the effects of feeding 30 g of soybeans per day on thyroid function. During the course of the investigation iodine intake (via seaweed) was reported as normal in all subjects. The investigators observed a significant increase in thyroid-stimulating hormone (TSH) levels in a group of 20 adults fed for one month (group 1) and in a group of 17 adults fed for three months (group 2). In two individuals, TSH levels increased from approximately 1 mU/mL to 7 mU/mL. Mean thyroxine levels were lower in both groups fed soy compared with a control group, although the difference was not significant. However, a significant increase in free thyroxine was observed in the group 2 subjects after they ceased the regime of soy consumption.

The changes to thyroid hormone levels were of clinical significance. Diffuse goitre and hypothyroidism appeared in three of the group 1 subjects and eight of the group 2 subjects. Group 2 subjects also had symptoms associated with hypothyroidism: constipation in 53% of subjects, fatigue in 53% of subjects and lethargy in 41%.

The goitre in the 11 subjects was a diffuse goitre, with degrees I and II enlargement. One subject in group 1 developed subacute thyroiditis. Goitre size was reduced in nine of the 11 subjects after cessation of soy but goitre still persisted in two individuals. These two subjects received thyroxine treatment and their goitres reduced in size after two to six months. Hence, a moderate amount of soy was found to have a marked goitrogenic effect on adult humans even though iodine was sufficient.

Isoflavones: the goitrogenic agents in soy

Many plants contain compounds that possess goitrogenic activity.[22] Among the best known are flavonoids, which are polyhydroxyphenolic compounds, based on the compound 2-phenyl-4H-1-benzopyran-4-one, or flavone. It is well known that flavonoids possess potent and diverse anti-thyroid properties.[23] The goitrogenic activity of flavonoids is commonly understood in terms of their ability to inhibit thyroid peroxidase

(TPO), the enzyme that catalyses the oxidation and organification of thyroidal iodine, through competitive inhibition. However, flavonoids also inhibit the peripheral metabolism of thyroid hormones and affect serum thyroid hormone binding.[24]

Perhaps the most extensively studied goitrogenic flavonoids are those found in Pearl millet and Fonio millet. These grains contain significant quantities of glycosides of apigenin and luteolin. Iodine deficiency coupled with the consumption of large quantities of these flavonoids has resulted in endemic goitre in the Sudan[25] and the Republic of Guinea.[26]

Isoflavonoids, which are based on 3-phenyl-4H-1-benzopyran-4-one, or isoflavone, are structurally related to the flavonoids and also possess goitrogenic activity. Soybeans are rich sources of isoflavonoids, the best known of which are genistein (5,7-dihydroxy-3-(4-hydroxyphenyl)-4H-1-benzopyran-4-one) and daidzein (7-hydroxy-3-(4-hydroxyphenyl)-4H-1-benzopyran-4-one). These isoflavones are frequently referred to as phytoestrogens since at dietary levels they exert oestrogenic effects in diverse animal species.[27]

The goitrogenic effect of soy has recently been attributed to the presence of genistein and daidzein, which have been found to possess potent anti-thyroid activity.[28] In fact in terms of its ability to inhibit TPO, genistein is more potent than either apigenin and luteolin or either of the well known anti-thyroid drugs, methimazole and 6-propylthiouracil (Table 1).

Table 1. Concentrations of various compounds producing 50% inhibition of thyroid peroxidase.

Compound	IC_{50} (μM)
Apigenin	3.4[29]
Luteolin	13.2[29]
Genistein	3.2[28]
Daidzein	7.6[28]
Methimazole	4.2[25]
6-propylthiouracil	7.2[25]

Isoflavones: levels of dietary exposure

In their study of the relationship between millet consumption and endemic goitre in the Sudan, Gaitan et al found that 100 g of Pearl millet contained approximately 102 mg of the glucosylflavonoids glucosylvitexin, vitexin and glucosylorientin. In the gut these glucosylflavonoids are readily hydrolysed; in this manner 100 g of Pearl millet might release up to 35 mg of apigenin and 14 mg of luteolin. A 70-kg adult consuming 500 g of Pearl millet could have an intake of 2.5 mg/kg-body weight of apigenin and 1.0 mg /kg-body weight of luteolin per day. Gaitan et al estimate that this degree of exposure is equivalent to a 1 to 5 mg dose of methimazole per day.[25]

In vivo, Pearl millet flavonoids can exert goitrogenic effects even in the presence of high iodine intake.[24] Given the degree of intake of potent goitrogens, it is not surprising that goitre is endemic in Sudanese for whom Pearl millet is a staple food and iodine intake is low.

Is it also possible that consumers of soy might suffer similar effects due to isoflavonoids even when iodine intake is sufficient? The groups most at risk are infants fed soy formulas, high soy users and those taking isoflavone supplements. Soy consumption and the use of isoflavone supplements have increased in recent years in response to the promotion of the theory that isoflavones may be protective against a variety of hormone-dependent diseases, such as breast and prostate cancer. But since these same isoflavones are goitrogenic, it is important to determine the level of soy consumption, or the dose of soy isoflavones, that might be required to affect the thyroid function of humans.

The observations of Ishizuki et al[21] indicate significant, goitrogenic effects in subjects fed 30 g soybeans per day. Based on the concentrations of isoflavones found in Japanese soybeans,[30] 30 g of soybeans could contribute up to 23 mg total genistein and 10 mg of total daidzein. For a 70-kg adult this would equate to an intake of 0.33 mg/kg body weight of genistein and 0.14 mg/kg body weight of daidzein per day. This amount of isoflavone consumption is three to four times higher than the amount commonly consumed in Japan, which is 0.08 to 0.13 mg/kg body weight of total genistein per day for a 70-kg adult.[31]

For infants fed soy-formulas, the exposure to isoflavones is greater than in any other population group. Infants less than six months of age, who are solely fed soy formula, have an intake of up to 5.4 mg/kg body weight of genistein and 2.3 mg/kg body weight basis of daidzein per day.[32] Hence, soy-formula-fed infants are exposed to more than twice the equivalent amount of goitrogenic compounds consumed in the endemic goitre regions of Sudan and approximately 16 times more than that of subjects in the Ishizuki study.

The concentrations of isoflavones found in products available in New Zealand,[33] indicate that a diet of 500 g of soy milk plus 50 g textured vegetable protein (TVP) per day would result in the consumption of up to 135 mg total genistein and 80 mg total daidzein. For a 70 kg-adult this equates to an intake of 1.9 mg/kg body weight of genistein and 1.1 mg/kg body weight of daidzein per day. This degree of exposure to anti-thyroid agents is broadly similar to that found in regions of Sudan where goitre is endemic and more than five times that of subjects in the Ishizuki Thyroid Clinic investigation.

Users of isoflavone supplements may consume up to 40 mg of genistein per day. For a 70 kg adult this is equivalent to 0.57 mg/kg, body weight basis of genistein per day, which is about 1.7 times more than that found to have goitrogenic effects.

Clearly there is potential for certain individuals to consume levels of isoflavones in the range that could have goitrogenic effects. Most at risk appear to be infants fed soy formulas, followed by high soy users and those using isoflavone supplements.

Isoflavones: the risks to consumers

For soy-formula-fed infants the risks to thyroid health due to exposure to isoflavones are significant. Thyroid hormones regulate growth, development and differentiation and, therefore, are essential in the physiology of humans.[34] Alteration in thyroid hormone levels or responsivity to thyroid hormone during the neonatal period may lead to disorders of the central nervous system and abnormal psychomotor development.[35]

High plasma concentrations of isoflavones are found in infants fed soy formulas. This is a consequence of regular feeding throughout the day, the ready absorption of isoflavones by the infant gut and the reduced body clearance of these compounds. Moreover, infants fed soy formulas from birth may experience such exposure for durations of 12 months or longer.

It has been suggested that although the plasma concentrations of total isoflavones in infants fed soy formulas are within the range required to inhibit TPO, the levels of free (active) isoflavones in plasma are very low.[1] This comment is based on preliminary data[36] which found no detectable free isoflavones in the plasma of infants (although plasma concentrations of total isoflavones were similar to those reported previously) and appears to suggest that isoflavone conjugates do not possess goitrogenic activity. However, the textbook view that conjugation leads to harmless metabolites that are readily excreted is not valid. In fact, there is considerable variability in xenobiotic conjugation and conjugates with enhanced biological activity and toxicity may be produced.[37] Besides, the work of Ishizuki et al[21] appears to confirm the *in vitro* activity of isoflavones even though adults readily conjugate these compounds *in vivo*.

Hence, long-term feeding of soy formulas may result in persistent inhibition of TPO and continually elevated TSH

levels. MOH has recognised this potential and it is entirely appropriate for them to suggest that thyroid problems due to soy formulas may be 'under-recognised'. Given that the clinical symptoms of hypothyroidism can be subtle due to the maintenance of normal thyroid hormone status, MOH advice that infants fed soy formulas should undergo tests to monitor thyroid hormone status is also warranted.

The lack of recognition of potential thyroid problems attributed to soy formulas may be due to difficulties in establishing a cause and effect relationship and even experienced soy researcher's may be ignorant of the connection between isoflavones and goitre. Hence, claims that soy formula feeding has occurred for many decades with no reports of adverse effects due to isoflavones[38] fail to account for the reported cases of goitre that have occurred in infants fed iodine sufficient soy formulas. It is also worthwhile noting that there is evidence that soy-formula-fed infants may have a greater incidence of anti-thyroid antibodies than breast-fed infants[39] and of an association between feeding soy formulas and the development of autoimmune thyroid disease in later childhood.[40]

Monitoring the thyroid status of high-soy consumers and users of isoflavone supplements may also be warranted. The goitrogenic effect of soy is consistent with the mechanism by which isoflavones inhibit TPO *in vitro*. Subjects fed 30 g soybeans per day experienced elevated levels of TSH although thyroxine levels were about normal. This defines subclinical hypothyroidism, a state in which a reduction in thyroid hormone secretion is compensated for by increased TSH production in order to maintain a clinically euthyroid status.

Subclinical hypothyroidism is a common condition that may eventually evolve toward overt hypothyroidism especially in persons with anti-thyroid antibodies. It is a condition of increasing importance and its prevalence appears to be growing such that studies aimed at defining its evolution are warranted. Dietary factors may well play a major role in the development of this condition since high goitrogen intake can increase TSH secretion.[41]

High-soy consumers and users of isoflavone supplements might, therefore, exhibit classic hypothyroid symptoms without recognising a dietary connection. Unfortunately there are few data as to what constitutes an appropriate level of soy intake, although it appears that some western consumers may now be eating far greater amounts of soy than that taken as part of a traditional Asian diet. The potential for 'mega-dosing' on isoflavone supplements has been raised before[32] but advertising of over-the-counter isoflavone products commonly claims benefits without any indication of risk to thyroid function.

Enhanced secretion of TSH is also associated with an increased incidence of thyroid cancer and those consumers who use large amounts of soy sporadically may face particular risk. Here, exposure to high levels of isoflavones might increase TSH secretion until, in the absence of soy, plasma isoflavone levels diminish and normal thyroid activity is restored; therefore in high-soy consumers a sporadic pattern of use could result in cycling between elevated and normal levels of TSH secretion.

Stimulating the thyroid in such a manner is the classic method for inducing thyroid tumours in laboratory animals.

Conclusion

MOH has found that infants with a history of thyroid dysfunction should avoid soy formulas and soy milks. Additionally, there is potential for isoflavone exposure to cause chronic thyroid damage in all infants fed soy formulas. The fact that infants fed soy formulas are subject to the highest isoflavone exposure of any population group has led Daniel Sheehan to warn that infants fed soy formulas have been placed at risk in a "large, uncontrolled, and basically unmonitored human infant experiment".[42] This level of exposure is unnecessary and the risk of harm could be avoided if manufacturers removed isoflavones from soy formulas. In the interim, it is appropriate for medical practitioners to monitor the thyroid status of infants fed soy formulas.

There is also evidence that adult exposure to isoflavones in high-soy users and users of isoflavone supplements may have adverse effects on the thyroid health. Therefore a more cautionary approach to the use of soy and isoflavone supplements is warranted.

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ORIGINAL ARTICLE

Thirty years of universal home dialysis in Christchurch

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Abstract

Aim. To review 30 years of universal home dialysis in a single dialysis unit.

Method. Analysis for patients using home dialysis since 1969 of information from hospital visits, clinical case notes and demographic and survival data from the Australia and New Zealand Dialysis and Transplant Registry.

Results. Since 1969 treatment options at the Christchurch Nephrology Unit for patients with end-stage renal disease have been home haemodialysis (HD), renal transplantation and, since 1979, continuous ambulatory peritoneal dialysis (CAPD). No long-term, hospital-based treatment has been offered. During this time 493 patients, aged 3-82 years, began treatment. The mean training time for home HD was 79 days (range 23-268) and for home CAPD 7 days (range 1-35). The mean HD treatment time was 7 hours x 3 per week (range 10-36 hours/week). Between 1980 and 1995, less than 5% of patients took antihypertensive drugs and 73% of those aged 18-65 years were in full or part-time employment. The mean age of patients commencing treatment increased from 41.8 years in the 1970s to 50.1 years in the 1990s. The median patient survival from 1970-1997 was 7.75 years on home HD and 2.1 years on home CAPD. Median survival on dialysis fell in the 1990's as more diabetics and older patients with comorbidity started treatment.

Conclusions. Home HD allows good rehabilitation, long treatment times and good blood pressure control which may all contribute to the superior survival of home versus hospital HD. CAPD survival in Christchurch was worse than HD, but this is probably due to patient selection. A policy of universal home dialysis is still workable provided there are sufficient resources for training and support of patients in the community.

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The first home dialysis training programmes began in the USA in the early 1960s.[1] Hospital dialysis facilities were scarce at that time and home haemodialysis (HD) provided the only means to treat the growing number of patients with end-stage renal disease (ESRD). It was soon realised that home HD was a safe and effective treatment which allowed excellent patient rehabilitation. In the early 1970s, 40% of long-term haemodialysis patients in the USA were on home HD.[1] Since then, however, there has been a steady decline in the use of home HD worldwide. Currently less than 2% of HD in the USA is performed at home.[2] Most HD patients are treated in a hospital or dialysis centre where they spend less than 12 hours per week on a dialysis machine. In contrast, the Christchurch Nephrology Unit has always used long HD sessions and long-term, hospital-based treatment has not been available. We here present the results of our 30-year experience with universal home dialysis.

The Christchurch Home Dialysis Training Unit opened in 1969 under the direction of Dr Peter Little and initially provided home dialysis services for all of New Zealand. From the mid-1970's it provided services for the South Island only and, since 1979, for Canterbury and Westland only. The unit currently serves a population of 480 000 spread over an area of 64 682 km^2. All dialysis training is conducted in a converted house away from the hospital campus by clinical technicians and nurses. Support is provided by doctors, a dietitian, a clinical psychologist and a social worker.

Methods

Since 1985, patient data have been collected prospectively at every clinic and hospital visit, and stored on a computer database (Proton™, Clinical Computing Ltd, London). Data on antihypertensive drug use, fistula surgery and the employment status of patients treated before 1985 were obtained from the clinical case notes. Demographic and survival data were provided by the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry,[3] a computer database updated six-monthly with reports from every nephrology unit in Australasia. Patients who died or had renal transplants within 90 days of starting dialysis are excluded from survival analyses and renal transplantation after 90 days is a censoring event.

Patients. Acceptance criteria for the dialysis programme were a willingness by the patient to accept treatment and the absence of imminently fatal comorbidity. By 31 December 1998, 493 patients aged 3-82 years had begun treatment. Currently there are 94 patients undertaking treatment at home (62 HD and 32 continuous ambulatory peritoneal dialysis, CAPD). The patient demography is notable for there being fewer Maori and Pacific Islanders than in other New Zealand centres (Table 1). Glomerulonephritis was the leading cause of ESRD in Christchurch. The incidence of diabetic nephropathy increased from 4.2% in the 1970s to 13.9% in the 1990s. Over this period the mean age of patients starting dialysis also increased, from 41.8 to 51.4 years.

Table 1. Patients starting dialysis (all forms) from 1970-1996.

	Christchurch	New Zealand	Australia
Number	405	2929	14836
Male:Female (%)	59:41	57:43	58:42
mean age (yrs)	45.6	46.1	49.9
Renal disease (%):			
Analgesic nephropathy	4.7	1.1	11.7
Diabetic nephropathy	12.5	25.2	11.1
Glomerulonephritis	31.3	32.6	36.5
Reflux nephropathy	11.1	6.8	6.5
Hypertension	10.6	10.9	7.7
Other	29.6	23.2	26.4
Race (%)			
Caucasian	89.6	56.2	87.6
Maori	6.6	30.6	0.3
Polynesian	1.7	9.3	1.1
Australian aborigine	0	0	4.8
Other	1.9	3.8	6.2

An audit detailing the reasons for declining treatment to patients with ESRD has been published previously.[4] Between 1988 and 1993, 139 patients started dialysis and 27

died of ESRD without receiving dialysis. Treatment was offered to, but was declined by, 11 (40%) of these patients. The remaining 16 patients were not offered dialysis for medical reasons, the most common being the presence of severe cardiac disease or metastatic carcinoma.[4]

Dialysis methods. All home HD patients have had arteriovenous fistulae. Before 1996 over 90% of fistulae were formed or had their formation directly supervised by one surgeon (JBM). Less than 1% needed polytetrafluoroethylene (PTFE, Teflon) grafts. Whenever possible, fistula surgery was performed before the expected start date of dialysis and most patients begin HD training using their fistula. Tenckhoff peritoneal catheters (straight, double-cuff) were used for CAPD access. All current CAPD patients use a disconnect system and exchange 8-10 L of fluid daily.

All home HD is currently undertaken with hollow-fibre cuprophan (0.8 m^2) dialysers, a 200 mL/min blood pump speed and acetate based dialysate (sodium 138 mmol/L, calcium 1.6 mmol/L). Universal dialyser and bloodline re-use ceased in 1983. The HD "dose" chosen for each patient was that judged likely to minimise dietary restriction and the use of antihypertensive drugs. Dialysis machine run-logs were used to check probable compliance with prescribed dialysis times.

Therapy with antihypertensive drugs was stopped within one week of starting dialysis (unless indicated for cardiac disease) and was only restarted if a patient had sustained hypertension despite increased ultrafiltration. Once home dialysis training was completed, patients attended their general practitioners for non-dialysis related problems. All patients had monthly blood tests for biochemistry and haematology. All HD patients measured their own weight and blood pressure (with mercury sphygmomanometers or electronic devices) before and after each dialysis. At annual review clinic, the patient blood pressure recordings were scrutinised and a dietary history and anthropometric measurements were taken.

Results

The mean training time for haemodialysis was 79 days (range 23-268) and for CAPD 7 days (range 1-35). The mean treatment time was 7 hours, three times per week (range 10-36 hours/week). Of those patients treated between 1980 and 1995, less than 5% took antihypertensive drugs and 73% of those aged 18-65 years were in full-or part-time employment. From 1970-1997, the median patient survival on home HD was 7.75 years, while on home CAPD (from 1979-1997) it was 2.1 years. Technique survivals (until a switch to the other form of dialysis was necessary) for home HD and CAPD were 7.38 and 1.84 years, respectively. The median patient survival fell from 5.4 to 3.9 years in the 1990s. This fall was attributable to more CAPD patients having a worse outcome (Table 2). Survival curves for non-diabetics on HD in Christchurch, New Zealand and Australia are shown (Figure 1).

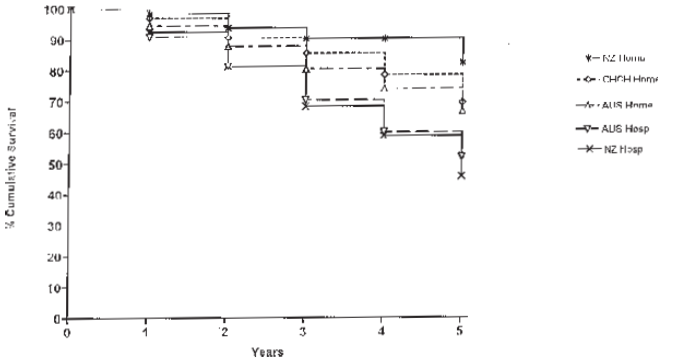

Figure 1. Survival curves for non-diabetic haemodialysis patients treated at home or in hospital centres in Australia and New Zealand from 1970-1997.

Discussion

We have presented results from our 30-year experience with universal home dialysis. No similar report has been published previously and we are unaware of any centre which has used home dialysis exclusively over such a prolonged period. The fall in patient survival on dialysis in the 1990s was probably due to more older patients and diabetics starting treatment. Our treatment methods have not changed significantly and our HD is still done using long sessions, gentle ultrafiltration and acetate-based dialysate – a type of HD now very rare worldwide. The success of our home HD programme meant that when CAPD was introduced it was perceived initially as offering few advantages. This was in contrast to units overseas, where CAPD provided for many patients the only means to be treated out of hospital. Since 1990, more of our patients have chosen CAPD, often those who were less suitable for home HD because they were elderly or because they had other medical problems, e.g. cardiac disease. The superior survival on HD compared to CAPD reflects this patient selection. In a recent study of 168 consecutive patients (data not shown), multivariate analysis found age, diabetes and systolic hypertension (or hypotension) to be important determinants of survival but treatment modality was not significant when other risk factors were considered.

Previous studies have found that patients on home HD have a lower mortality than those treated with HD in a hospital setting.[5,6] This difference has been attributed to patient selection since young, fit patients are more likely to receive home HD.[7] Prior to 1990, our home HD patients were less selected, because hospital HD was not available, which may explain why their survival was inferior to home HD patients elsewhere in New Zealand. Selection bias may not fully explain why home HD patients live longer. In a recent study, Woods et al[7] used a proportional hazards model to study a randomly selected series of 4892 USA dialysis patients and found that the survival advantage of home versus hospital HD persisted after adjustments were made for age, diagnosis and comorbidity.

Table 2. Age and survival of patients treated in Christchurch.

Year	1970-79			1980-89			1990-97		
Treatment	Total	HD	CAPD	Total	HD	CAPD	Total	HD	CAPD
Patients	95	92	3	136	127	9	180	115	65
Mean age (yr)	41.8	41.3	58.4	45.7	44.9	56.7	50.1	46.3	56.8
Median survival (yr)	5.4	9.1	1.5	5.4	6.5	2.1	3.9	7.0	2.2
2 yr survival (%)	81	83	0	77	80	51	71	88	53
5 yr survival (%)	53	57	0	51	56	0	42	75	16

HD: home haemodialysis; CAPD: continuous ambulatory peritoneal dialysis.

Home HD has several advantages over hospital HD which may contribute to a better long-term outcome. Home HD allows greater flexibility in choosing when to dialyse. Patients can dialyse for long periods while still maintaining an acceptable lifestyle, resulting in improved urea clearance and blood pressure control. Less than 5% of our dialysis patients required antihypertensive drugs which is in marked contrast to most hospital dialysis units where antihypertensives are prescribed for over 50% of patients.[8,9] Home dialysis patients, in general, have a better quality of life[10-12] than hospital-treated patients. The flexibility of home HD may make it easier for patients to continue in employment. Many of our patients dialyse overnight and are able to continue working full-time. Other quality of life characteristics such as patient autonomy and "empowerment" are also more evident in home dialysis patients than in their institutionalised counterparts.[10-12] Their possible contribution to longevity is, of course, difficult to quantify.

There are several prerequisites needed for successful home dialysis training. Firstly, patients must be willing and able to learn the technique and be prepared to take over responsibility for their own dialysis. Secondly, patients must have durable vascular (or peritoneal) access. In this regard, the presence of an experienced, dedicated vascular surgeon has been critical to the success of our programme. Considerable importance has been placed on fistula care and correct needling techniques and as a result very few alternative forms of access have been needed. Thirdly, all staff involved need to be experienced in home dialysis training and be committed to a philosophy of encouraging patient autonomy. A factor which may be contributing to the continued under-utilisation of home HD in the USA is that many younger nephrologists now have little experience with this form of treatment.

Finally, for home dialysis to prosper there must be no financial disincentive to using it. Although home dialysis is 30-50% less expensive than hospital dialysis,[13-15] a major reason for its decline in the USA was the introduction of legislation in 1973 which changed the method of reimbursement, encouraging physicians to use hospital treatment.[1] We are fortunate that the practice of home dialysis in New Zealand has not been hampered by similar government regulations. However, while we are thankful that home dialysis can thrive here, it does so partly because there is a lack of funding for hospital dialysis facilities. The commencement rate for elderly patients on dialysis is much lower in New Zealand than in Australia or the USA.[16] If more older people are referred for treatment, as has occurred overseas, there is likely to be a greater demand for hospital dialysis.

In summary, home dialysis has advantages over hospital dialysis in terms of cost, quality of life and survival. In our experience there are few patients who cannot be successfully taught to dialyse at home provided there are adequate resources for training and a consistent determination from dialysis staff to do so. The predicted increase in the numbers of older patients requiring dialysis will challenge the continuation of a universal home dialysis policy. However, geographical isolation and ongoing financial constraints should ensure that home dialysis remains important to the provision of dialysis services in New Zealand.

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An investigation into the incidence of toxoplasmosis in pregnancy in New Zealand

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Abstract

Aim. To estimate the incidence of toxoplasmosis in pregnancy in New Zealand and consider whether there is a case for screening women in pregnancy.

Methods. The risk of maternal and fetal infection with toxoplasmosis was derived by first determining the rate of maternal seroconversion based on seroprevalence studies. The age-specific number of seroconversions in pregnancy was then estimated from the birth rate. Using reported fetal infection rates after primary maternal infection, the expected number of congenitally infected infants in one year was estimated. These incidences were compared with the number of recognised cases of toxoplasmosis infection in pregnancy and the actual number of positive IgM results at the Wellington Hospital laboratory. Using national births data, this incidence was extrapolated to estimate the number of expected cases in New Zealand.

Results. The annual seroconversion rate was 0.62% (95% confidence interval 0.39-0.86). On this basis, 164 primary maternal infections are expected annually with 66 fetuses being infected. Ten patients tested positive for IgM

in Wellington, which averaged only one case per year being identified over the time examined in this study.
Conclusions. Very few of the expected cases in pregnancy are diagnosed. Reporting rates were low when toxoplasmosis was a notifiable disease. Other means of improving detection, reporting and the avoidance of infection are discussed. More information is required before screening can be recommended in New Zealand.

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Toxoplasma gondii infection during pregnancy is a cause of serious congenital morbidity which, unlike other infective causes such as cytomegalovirus, can be treated. Central nervous system calcification[1] and seizures,[2] for example, may be reversed by neonatal treatment with pyrimethamine and sulfadiazine.

Congenital toxoplasmosis is a social and financial burden on the community. There is increasing recognition of the scope of morbidity and disability it can cause. In the USA in 1990, the lifetime financial burden of children infected in one year was estimated to be at least $US369 million.[3]

International literature shows wide geographical differences in seroprevalence[4] and in some countries, such as France and Austria, pregnant women are routinely screened for toxoplasmosis infection. New Zealanders face risk factors for toxoplasmosis transmission. The cat is the definitive host for *T gondii* and it has been stated in commercial surveys that New Zealand has one of the highest cat-per-person ratios in the world (Effem Foods 1997, personal communication), exposing New Zealanders both directly and through cat faeces in garden soil. Many New Zealanders also consume improperly cooked meat from barbecues or the microwave,[5] which may contain viable toxoplasma cysts.[6] Thus women are susceptible to a potentially dangerous infectious agent which surrounds them.

Toxoplasma infection during pregnancy often has few symptoms or signs and may pass clinically unnoticed. Over the last ten years, national discharge summary data show only 27 cases of toxoplasmosis in women of child-bearing age (NZHIS personal communication). Only one case of congenital toxoplasmosis was notified to the Institute of Environmental Science and Research Limited (ESR) between 1987 and 1996, when it was a notifiable disease. This was obviously due to under-reporting because National Health Institute (NHI) testing data from only ten months of 1988 show nine babies with clinically and serologically diagnosed toxoplasmosis.[7]

It is important, therefore, to ascertain the local proportion of IgG-negative women in the child-bearing age group if consideration is to be given to screening in the New Zealand population. There would appear to have been only four publications with age-specific seroprevalence reported for New Zealand. It is the aim of this paper to estimate the disease burden of congenital toxoplasmosis in New Zealand and determine if there is a case for screening pregnant women.

Methods

Estimating the risk and number of maternal infections. All the New Zealand studies with age-specific seroprevalence of toxoplasmosis were sourced to provide data for this investigation. Data from a number of surveys in different parts of New Zealand reported in four seroprevalence studies of IgG *T gondii*- specific antibody[8-11] were collated to determine the infection rates.

With a constant rate of infection, seroconversion rates may be calculated by the expression log (1-cumulative incidence), which is equal to the seroconversion rate multiplied by age.[12] Constants developed from the ten surveys in the four published studies used, were added to allow for different infection rates at ages younger than those published. This modelled infection rate for all the studies over the age range required for pregnancies. The national cumulative incidence was estimated from the average intercept (estimated from these constants). The rate of seroconversion was estimated with a generalised linear model with binomial errors. The age-specific number of maternal seroconversions was then estimated from the number of live and stillbirths in 1995.[13] Included in this estimate was an additional 15% to allow for first trimester miscarriages. Terminations of pregnancy were not included as these do not add to the disease burden of toxoplasmosis with advancing gestation.

Using a reported fetal infection rate after primary maternal infection of 40%,[4] the number of congenitally infected infants in one year in New Zealand was estimated. It is likely that approximately the same number of women would be acutely infected in each of the three trimesters of pregnancy. However, the risk of fetal infection differs by trimester. The incidences of fetal infection per trimester, as found by Forestier,[14] were used to estimate the number of fetuses infected in each trimester. These percentages are 10% in the first trimester, 30% in the second trimester and 60% in the third trimester.

Estimating the number of recognised cases in Wellington. From the total number of positive IgM results at the Capital Coast Hospital, Wellington, all positive results from women aged >13 and <50 years and infants ≤1 year of age were selected and the case notes reviewed or the referring clinician contacted where the patient had not been admitted. The total number of positive IgM results at the Capital Coast Health (CCH) Laboratory for the nine year period, 1989 to 1997, was also determined. The CCH Laboratory tests for IgM using the VIDAS EIA-immunocapture test, which is unaffected by rheumatoid factor or antinuclear antibody (ANA). The number of recognised cases of toxoplasma infection during pregnancy in the Wellington region was counted. The number of observed cases was compared with the number expected. This was based on the estimates derived above. The number of cases expected nationally was based on extrapolation from the percentage of births occurring in the Wellington region[13] (which was 12%).

Clinical data from the cases of suspected or proven toxoplasma infection during pregnancy that had been identified via the CCH Laboratory positive IgM test results were collected and are presented in tabular form. Evidence of intrauterine or postnatal infection and the presence of neonatal and infant morphological or functional abnormalities was also sought. Treatment of the mother and/or the child was noted, as were follow-up plans or actions. The indications for toxoplasmosis serology testing were also examined.

Results

A 0.62% (95% confidence interval 0.39-0.86) annual seroconversion rate in the New Zealand population was estimated from the seroprevalence studies and is shown in Figure 1. Cumulative incidence was estimated by the expression $1\text{-}e^{(-0.363-0.062 \times age)}$. From this estimate, 164 primary maternal infections were expected, including the correction for the numbers of women who miscarry, which accounted

for nine infections. Using the 40% fetal infection after primary maternal infection 66 fetuses would be predicted to be infected each year. On the basis of the reported findings of Forestier,[14] it would be expected that there would be seven fetuses infected in the first trimester, 19 in the second trimester and 40 in the third trimester.

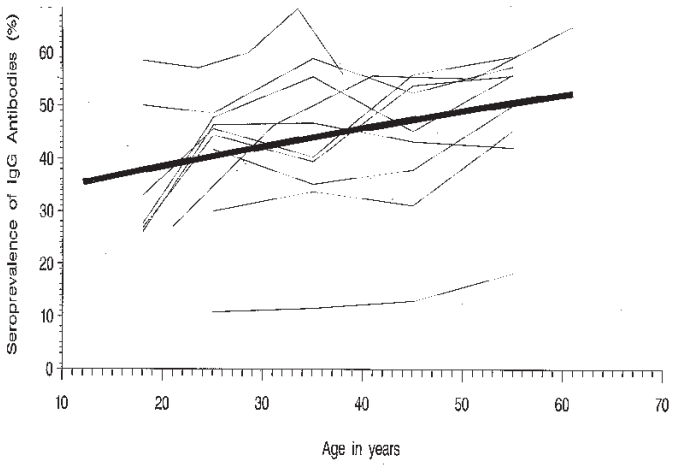

Figure 1. Age-specific prevalence percentage of IgG antibodies to toxoplasmosis in New Zealand women of reproductive age. ——: each published seroprevalence study; ————: calculated average (refer to methods section).

The clinical data are presented in Table 1. Ten pregnant patients tested IgM-positive between 1989 and 1997 at Wellington Hospital. This equates to approximately one identified case per year. One case suffered a miscarriage and there was one intrauterine death at 22 weeks gestation, some days after fetal testing in the presence of maternal seropositivity and clinical signs of infection. In addition, sixteen patients have received *toxoplasma* antibody tests for recurrent miscarriages at Wellington Hospital in the last two years.

Cases of toxoplasmosis in pregnancy generally presented with lymphadenopathy or a flu-like illness. General practitioners diagnosed 50% of the cases. Four cases were diagnosed by hospital doctors performing a TORCH screen. Referral letters from general practitioners revealed symptoms or signs as the indication for testing. There were no symptoms suggestive of toxoplasmosis infection recorded in the histories taken from obstetric patients whilst in hospital.

Two of the ten cases reviewed, returned a positive IgM *Toxoplasma* test after the patient had been discharged. Where the differential diagnoses of cytomegalovirus, Epstein-Barr virus or tuberculosis also returned a positive test, these were the preferred diagnosis over toxoplasmosis. There had been four clinical cases in 1997, which was about five times the number in preceding years.

Treatment was given in only three of the ten cases in pregnancy. In two cases, this was with the combination regimen of spiramycin +/- sulfadiazine, pyrimethamine and folinic acid and, in one case, piramycin alone was used.

Four fetuses suffered intrauterine growth retardation, however, the mothers in three of these cases were smokers. Three babies were delivered prematurely. Only three had a cord blood sample taken for *Toxoplasma* antibody analysis. Follow-up plans varied from full paediatric and ophthalmic assessment to no follow-up. Where there had been a miscarriage or intrauterine death, the mother received obstetric specialist review. Only one of all those tested for toxoplasmosis was HIV positive but this was not in pregnancy.

Discussion

The fact that only a small proportion of the expected number of cases of toxoplasmosis in pregnancy is diagnosed is likely to be due in part to the relatively asymptomatic nature of *Toxoplasma* infection and in part to a lack of awareness amongst many of the care-givers. Systematic, compulsory screening of pregnant women, as is done in France, allows serological diagnosis to be made without the need for patient and doctor to recognise the symptoms and/or signs and respond accordingly. Internationally there has been much debate over the value of screening. Studies in the USA,[15] Finland,[16] Norway[17] and Germany[18] have predicted it to be cost-effective, however, studies in the United Kingdom have found insufficient evidence of benefits outweighing risks,[19] as well as no financial benefit in screening.[20] Screening remains controversial and more evidence is needed before it could be advocated in this country. The cost of the IgG/IgM toxoplasmosis screen tests done on a request basis approximates $11 per test. This cost would be considerably less if performed in large numbers as part of antenatal booking blood tests. However, raising medical awareness of toxoplasmosis during pregnancy in New Zealand carries very little cost and could reap benefits. Understanding how to interpret positive serology is important to avoid causing unnecessary anxiety in patients with past infection.

It is of concern that few clinical cases of infection in the second or third trimester are being detected. When the risk of fetal infection increased in the first trimester, miscarriage may occur but fetuses infected in the second and third trimesters may be normal at birth, yet as many as 40% suffer sequelae in later life.[20] These children may well be lost to

Table 1. Characteristics of the cases of toxoplasmosis in pregnancy in Wellington, 1989-1997.

Case	Age (years)	Ethnic group	Diagnosis made by	Gest at diagnosis (weeks)	Gest at delivery (weeks)	Symptoms and signs	Therapy	Cord IgM tested	Follow-up
1	34	PI	GP	10	38	Lymphad	Full	No	Full
2	32	Other	GP	25	32	Flu	Full	No	Full
3	26	Other	Obstetric specialist	12	41	None	None	Yes	M/W
4	27	Other	Registrar	NK	34	None	None	No	Trans
5	40	Other	GP	Pre Conc.	13	Lymphad	None	Yes	N/A
6	24	Other	Obstetric specialist	10	38	NK	None	No	Prev Rx
7	29	PI	Registrar	NK	22 IUD	NK	None	No	Nil
8	19	Maori	Not Known	NK	40	Sweats	None	No	Full
9	26	Other	GP	19	22	Fatigue	Spiram	Yes	N/A
10	26	Other	GP	NK	40	Lymphad	None	No	Full

NK: not known; Lymphad: lympadenopathy.

follow-up, becoming an unrecognised burden on their families and cost to the country.

Increased awareness of the modes of transmission of *T gondii* is required by doctors so they can improve their history-taking and give advice to pregnant women on how they can minimise the risk of becoming infected. Widely recognised transmission routes are faecal-oral via stale cat faeces or soil in which cats have defecated and by the consumption of undercooked meat which contains toxoplasma cysts. Possible risk factors of particular relevance to New Zealand include the high rate of cat ownership and the use of microwave cooking or barbecuing, which may not destroy cysts in meat.[6] Women who do not own a cat may not realise that they are at risk of infection when gardening through exposure to soil contaminated by cats living in the neighbourhood. There are potential risks to New Zealand female veterinarians, who comprised 80% of a recently graduating veterinary class, of immunising themselves with the live toxoplasma vaccine during pregnancy (Wilks C, personal communication,1997). Pregnant women are made aware of many useful lifestyle changes to avoid infection and chemicals which are potentially harmful to the fetus, such as avoiding seafood, delicatessen foods and smoking. Education about avoidance of the toxoplasmosis risk factors should be added to these.

Toxoplasmosis should be considered as a differential diagnosis when a pregnant woman presents with lymphadenopathy or a non-specific, flu-like illness. She should be tested, for IgG specific antibodies. A positive result indicates she has been infected at some time. A negative result indicates that she has not been previously infected. Blood samples which return an IgG latex agglutination test titre of ≥1/16 are automatically tested for IgM specific antibodies by ELISA-immunocapture assay at the Capital Coast Health Laboratory. As IgM may remain detectable in the blood for ≥one year, reliable diagnosis of an acute infection requires demonstration of a rise in IgM titre between two samples taken three weeks apart and run in parallel.

Toxoplasma tests for the investigation of recurrent miscarriages are unnecessary. The development of IgG antibodies after an initial acute infection is thought to make the mother immune to any further *Toxoplasma* infection challenges, including those she may face in future pregnancies. It would only be useful to test pregnant women with AIDS for *Toxoplasma* antibodies but, despite the increasing literature on toxoplasmosis reactivating in AIDS patients, we did not find this is to be a major component of seropositivity and indication for testing in New Zealand.

Congenital toxoplasmosis was removed from the list of notifiable diseases in 1996, as only one case had been reported in the preceding ten years. Official notification by clinicians seems virtually never to occur, probably because congenital toxoplasmosis is not a readily accepted diagnosis. Further, discharge summary data record less than 5% of the estimated cases. However, official notification of cases could indirectly increase awareness of the condition and improve follow-up plans, particularly for the 85% of congenitally infected newborns that appear normal at birth.[21] A direct notification link between the laboratory and the ESR may prove much more effective than the past scheme. ESR could then monitor national trends, or detect outbreaks more effectively, as recently illustrated in British Columbia.[22] The Ministry of Health could also provide relevant information to clinicians as necessary, to facilitate better management of toxoplasmosis during pregnancy in New Zealand. Such material might include the information summarised in this discussion.

The incidence of primary infection with toxoplasmosis in pregnancy in New Zealand remains unknown and there are very limited seroprevalence data in the child-bearing age group. Before it is possible to decide on the value of screening in New Zealand and whether to treat IgM-positive pregnant women with spiramycin to prevent transplacental transmission of the parasite, it is necessary to estimate the disease burden. The present study aimed to do this and has suggested considerable underestimation of the disease burden in this country. A prospective study of women in the child-bearing age group and their offspring is needed to answer the questions about seroprevalence, seroconversion and intrauterine transmission of toxoplasmosis.

One of the drugs used for treatment, spiramycin, is thought to be safe in pregnancy and may be given without the confirmation of fetal infection. A polymerase chain reaction test on amniotic fluid is the investigation of choice to confirm fetal infection, as it is rapid, safer than fetal blood sampling and as accurate. It has a reported sensitivity of 97.4%.[23] Polymerase chain reaction tests on amniotic fluid are available in New Zealand from the Virology and Immununology Department at Auckland Hospital.

The therapeutic combination of sulfadiazine, pyrimethamine and folinic acid should only be added once congenital infection has been confirmed since the safety profile of these drugs in pregnancy is less clear.

Ultrasound screening of the unborn child is routinely performed in New Zealand. Up to 45% of congenital infection cases have been found to have abnormalities on ultrasonography.[24] Therefore, increased awareness amongst those who perform ultrasonography in New Zealand may provide supportive evidence in a diagnosis of congenital toxoplasmosis.

It is important to emphasize that whilst the data on primary toxoplasmosis infection in pregnancy remain incomplete, advising women on means of avoiding exposure is as important as offering screening testing. Increased knowledge and awareness of the condition by health-care professionals are necessary, if the public are to be made more aware of the potential risks to pregnant women.

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Emerging clinical governance: developments in independent practitioner associations in New Zealand

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Abstract

Aims. To document and analyse the development of independent practitioner associations and similar groups in New Zealand.

Methods. A questionnaire was sent to the 30 independent practitioner associations in August 1998 and followed-up by a number of reminders.

Results. The 28 respondents (93%) represent 97% coverage of the estimated membership of independent practitioner associations and similar groups. Membership of the 28 responding organisations ranged between seven and 340, with an average of 74 members and a total of 132 employed staff. Twenty-one had appointed a chief executive officer or general manager. The respondents' most important goals were "achieving better health outcomes for patients" and "making better use of primary care resources". They reported almost total implementation of computerised age/sex registers in their practices. There was strong support for independent practitioner associations to manage the clinical activity of members, to move from historical to equitable, needs-based funding and for formal patient enrolment. The majority of respondents supported integrated and capitated primary care budgets but few supported capitated budgets for separate general medical services, laboratory and pharmaceutical services. Important recent initiatives include a wide range of integration projects and increasing involvement of local communities.

Conclusion. Independent practitioner associations have made significant progress in increasing membership levels, in establishing a framework for managing clinical activity of members and in developing their infrastructure, including information systems. They have established a wide range of new relationships within primary care, with their communities and with primary and secondary care providers. In managing increasing amounts of public money to achieve public goals, these groups may be developing a new model of clinical governance, which could be of international importance.

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The term independent practitioner association (IPA), as used in the paper, includes not only large groupings of practitioners using the designation IPA but also larger practices and other groupings associated with the IPA movement. IPAs are recognised to be an important development arising from the 1991 health reforms.[1-3] They were initiated by a few enthusiasts prepared to risk collegial disapproval and/or opposition, and now claim an overwhelming majority of general practitioners (GPs) in New Zealand as members. An initial survey of IPA development in 1994 established a baseline of information. This more recent survey (late 1998) aimed to assess subsequent developments. The specific objectives were to determine: the nature and extent of IPA development; the goals and policies of IPAS; IPA achievements; the changing relationships between IPAs and the Health Funding Authority (HFA), the Hospital and Health Services (HHSs) and the communities they serve; and progress towards integration.

Methods

In September 1998 the authors sent a questionnaire to all recognised IPAs, to contracting group practices and other organised, but non-IPA, GP groups. A follow-up of non-responders in late 1998 resulted in a total of 28 responses out of a possible 30 (93%). IPAs were asked to discuss the questionnaire at their board level and to provide, as far as possible, a representative view on questions of opinion. It is not known to what extent this request was complied with. Respondents did not answer some questions.

Results

Membership and scope. The total membership of the 28 respondent organisations was 2 000, approximately 97% of total estimated membership of IPAs and similar groups. The size of respondent organisations varied widely with the smallest a group practice of seven and the largest an IPA with a membership of 340. The average size was 74 members. Seven of the 28 respondents indicated that their membership included health professionals other than GPs. Of those groups which have formed legal entities, 17 are limited liability companies, four incorporated societies, two non-profit trusts and four partnerships. Twenty-one of the 28 groups had either a chief executive officer or general manager. Staff totalled 132 and averaged 6.3. IPA governing boards averaged 5.6 directors including 17 community representatives.

Goals and policies. IPAs were asked to rate a list of goals on a five-point scale from very important to unimportant and then select their most important goal (Table 1). The highest rating goal was "achieving better health outcomes for your patient," which was also the most frequent response, and "making better use of primary care resources".

Several questions sought information about the organisations' policies, including computerised registers, capitation payments and financial risk management. Computerised age/sex registers are totally supported with almost total coverage of members' practices. There was majority support (19) for formal patient enrolment, with only four opposed.

With respect to financing and risk-management policy there was more diversity. In terms of current financing, the main source for IPAs was HFA budget holding (14), with some receiving HFA special grants (5), and HFA special projects (3). 'Other' reported sources of funds were uncommon, but included investment income, contracts for services (e.g. with the Ministry of Health or local HHS), computer services and software, and management fees.

Table 1. Rating of the goals of IPAs and similar groups.

Goal	Goal weighting (out of possible 5)	Most important goals, number of responses
Achieving better health outcomes for your patients	4.8	7
Making better use of primary care resources	4.8	1
Improving the health of the community you serve	4.7	3
Improving standards of general practice	4.5	2
Integrating your services with other providers of primary health care, nurses etc.	4.4	0
Becoming a stonger negotiating body with the HFA	4.3	0
Improving services for patients through holding integrated budgets	4.2	1
Protecting the status of general practice	4.2	1
Integrating primary and secondary care services	4.0	1
Shifting the balance of services from secondary to primary care	3.7	0
Involving communities you serve more actively in service provision	3.7	2

In 1998 the HFA signalled its intention to establish integrated, capitated budgets.[4] Most respondents opposed or were uncertain about individual budgets for general medical services and associated services, laboratory or pharmaceutical services. A majority (14 out of 24) supported integrated budgets for all of these combined. Only five groups indicated opposition. There was also strong support (21) for a move from historical to population needs-based funding, with only three opposed to this. Eleven supported the acceptance of funding to purchase secondary care services, six opposed this and seven were uncertain. Seventeen of the 28 respondents opposed taking on the risk of going over budget. This indicates less opposition than in previous surveys. On the other hand, almost all respondents (26) opposed retaining savings as personal benefits. There was no particular pattern with respect to these responses except that those opposed to enrolment were also opposed to holding budgets and taking on risk.

Reported achievements. IPAs were asked to rate a list of achievements as being "very successful" (scaled as 3), "quite successful" (2) and "not very successful" (1). Items included in the list were assessed by the authors to have been important in the development of IPAs and similar groups. From Table 2 it can be seen that the four most important achievements identified were "establishing an infrastructure" (2.7 out of 3) "collaboration between members" (2.5), "developing information systems" (2.4) and primary care resource management (2.3).

Moderate successes included: establishment of new services, development of integrated care initiatives, collaborative external relationships with other providers, collective accountability for primary care resource management, and collective accountability for quality of care. Community involvement, at 1.7, was the lowest rated achievement. A few additional achievements were mentioned, including an effective partnership with Maori, a good working relationship with the HFA, effective management arrangements for the IPA and being a good advocate for integrated care developments.

All respondents reported that, as far as they could tell, their members gave 'strong' or 'moderate' levels of support to group goals, leadership, communication, the services provided and the opportunities to participate in the activities of their groups.

External relationships. A number of questions enquired about perceived relationships with the HFA and HHSs. Most improvements had occurred with HHSs. Ten respondents noted that there had been no change in relationships with the HFA over the past 12 months but equal numbers indicated either a deterioration or improvement (eight each).

Table 2. Rating of achievements by IPAs and similar groups.

Achievements	Not very successful	Quite successful	Very successful	Average score
Establishing an infrastructure (27)	1	6	20	2.7
Collaboration between members (26)	0	13	13	2.5
Developing information system (26)	1	13	12	2.4
Primary care resource management (26)	4	11	11	2.3
Establishment of new services (24)	8	9	7	2.0
Integrated care initiatives (25)	7	10	8	2.0
Collaborative external relationships with other providers (26)	7	11	8	2.0
Collective accountability for primary care resource management (25)	4	16	5	1.9
Collective accountability for quality of care (26)	7	14	5	1.9
Community involvement (26)	12	9	5	1.7

Average score based on 'very successful'=3; 'quite successful'=2; 'not very successful'=1; Numbers of respondents in brackets.

In terms of relationships with the community, IPAs demonstrated some support for community involvement. Fourteen indicated it to be "quite important", eight "slightly important" and six "not important". Respondents provided information to the community mainly through newsletters and pamphlets. A number of consultative processes have developed such as public meetings (10), submission on written documents (12), complaints advocacy procedures (13) and surveys of community views (9). Ten respondents had direct community representatives on their boards, seven had established community advisory boards and five had established joint ventures with community groups. Respondents also reported a variety of other community initiatives including: involvement on the boards of other

health agencies, provision of health information (e.g. via a website, through local media), participation in projects such as a health survey and health promotion meetings, and involvement with other groups in more formal health planning projects.

Clinical and service initiatives. There was very strong support by IPAs for managing the clinical activity of members through a range of strategies such as guidelines and quality management. This included managing the cost as well as quality of services. Outcome-related performance indicators (20 respondents) and multidisciplinary practice teams are both strongly supported (21 of respondents). On the other hand, only 12 supported sharing of information between IPAs and similar groups.

Opinion was also sought on two service integration options. There was strong support for joint venture projects between primary and secondary care providers (19) but much less support for the purchase of secondary care from a primary care base (5). Twelve respondents reported service integration projects either under way or about to be implemented. The projects mentioned fell into four broad groups: disease management (11 projects, e.g. diabetes, asthma); target populations (six projects, e.g. Maori, elderly, children); service arrangements (ten projects, e.g., joint project with ACC and physiotherapists, community nursing services); and infrastructure (three projects, e.g. community pharmacy, joint project with the New Zealand Health Information Service).

Discussion

Membership of IPAs now covers nearly 70% of the nearly 3000 GPs practising in New Zealand,[5] although, as indicated by a recent unpublished Auckland study, in full-time equivalent terms the number of GPs in some form of contracting relationship with the HFA is likely to be nearer 80%. This is a significant increase on the estimated 50% reported in the 1994 survey.[1]

IPAs and other surveyed groups have become much more sophisticated organisations than they were in 1994, well staffed and with senior managers appointed. Despite being made up of private practitioners they are managing large and increasing amounts of public expenditure. Almost all have taken on budget-holding responsibility for pharmaceutical and laboratory services but they largely continue to reject both financial risk and retaining savings as personal benefits, seeing the latter as both unprofessional and unethical.

Overall ranking of goals is similar to that of 1994.[1] Management of resources, including shifting savings from lower to higher priority areas, was seen as a critical factor in achieving their stated primary goal of better health outcomes for patients and communities. Process goals, such as "becoming a stronger negotiating body with the HFA" and "protecting the status of general practice", have become somewhat less important. This reflects increased confidence arising from developments over the last four years in working through contentious matters with the purchasers.

Although relationships with the new HFA appear improved, continuing uncertainty is partly due to concerns over the HFA's promotion of capitated primary care.[4] Previous surveys indicated increasing support for capitation and, in this survey, there is still majority support for integrated, capitated budgets, inclusive of general medical services, practice nurse, laboratory and pharmaceutical services and strong support for moving from historical to population needs-based funding.

The adoption of this approach by IPAs, which could then negotiate capitated arrangements with their practices, is likely to be much more acceptable than compulsory imposition of a capitated general medical services regime by the HFA.

A new feature in this survey is the development of external relationships by IPAs. Still in the early stages, collaborative relationships may prove a more successful way of promoting community participation than past efforts through election or appointment to hospital or area health boards. Community input to primary care is more relevant than to secondary care as patients are closer to services and important issues of quality, access and service priorities may be better dealt with at this level. Community input into the primary care sector in the past has been almost totally neglected as there has been no organised entity through which such input could be established. IPAs now provide such opportunities.

Further evidence of the development of external relationships has been efforts towards service integration with other primary care providers, such as nurses, midwives and community health groups, and with secondary care services. Most IPAs have shifted their aspirations from purchasing secondary care services from a primary care base to establishing a wide range of collaborative service initiatives between themselves and secondary care clinical specialists.

This survey and related studies have demonstrated evidence of the emergence of a comprehensive model for the management of primary care,[6] the key features of which are summarised in Table 3. These achievements, from recent discussion in the literature, may be considered to be an advanced model of clinical governance.[7-12] One recent definition from the North Thames Region states that clinical governance can be defined as "the means by which organisations ensure the provision of quality clinical care by making individuals accountable for setting, maintaining and monitoring performance standards".[11] This is elaborated by Donaldson, "the term clinical governance resonates with that of corporate governance, a set of financial duties, accountabilities, and rules of conduct".[8]

Table 3. Key functions and achievements of IPAs and similar groups relevant to the concept of clinical governance.

Developing collective professional accountability for the management of clinical activity of members to improve quality and make better use of primary care resources.
Managing new integrating relationships between members, between other primary care professionals and the community and between primary and secondary care.
Implementing a primary care infrastructure including staff appointments, an information system to computerise, merge and manage practice registers, analyse laboratory and pharmaceutical data and provide personalised feedback to members.
Peer group formulation of clinical guidelines and monitoring of performance to promote better quality, evidence-based practice.
Managing corporately an increasing set of primary and some secondary care resources to achieve better health outcomes for patients and communities.

IPAs, however, appear to be extending this in ways which owe more to Shortell and Kaluzny's broader understanding of governance as "the function which holds management and the organisation accountable for its actions and which provides management with overall strategic direction in guiding the organisation's activities".[13]

Conclusion

From this survey and other studies it is clear that IPAs and similar groups have made important recent progress especially in two areas: developing both internal and external

relationships and networks, and applying a new model of clinical governance. The emergence of integrating relationships, including those with other primary care providers and the community, as well as with secondary care providers, shows that IPAs are organisations serving more than narrow GP interests. This wider corporate relationship building, along with the commitment to the management of both clinical activity and health resources to achieve better health outcomes, is demonstrating an advanced model of clinical governance which may be of international importance.

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IN PRACTICE

Echocardiographic evaluation of stroke

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Stroke accounts for 10% of deaths and is a leading cause of long-term disability in New Zealand.[1] Identification and treatment of possible causative factors following stroke is important because of the high risk of recurrent neurological events.[2] Two-dimensional echocardiography has been used from the early 1980s to evaluate patients for cardiac sources of embolism which may account for 15% to 34% of all strokes.[3] Transthoracic echocardiography is commonly used to evaluate patients for potential sources of embolus in New Zealand hospitals. This technique has advantages of being non-invasive and widely available but has limitations of technically inadequate imaging in a proportion of patients. Transoesophageal echocardiography improves visualisation of cardiac structures but is invasive and is only available in hospitals staffed by cardiologists trained in the technique. At present there is uncertainty regarding which patients should have echocardiography after stroke and who should have transoesophageal rather than transthoracic evaluation.

Role of transthoracic echocardiography

Transthoracic echocardiography identifies a probable (i.e. thrombi, tumours or vegetations) source of embolism in about 4% (range 2% to 16%) of unselected stroke patients and in only about 1.5% (range 0% to 6%) of patients without clinically evident cardiovascular disease.[3-5] Left ventricular thrombi related to either recent myocardial infarction, left ventricular aneurysm or dilated cardiomyopathy are well visualised using transthoracic echocardiography.[6] In patients presenting with stroke and an intermediate or high probability of endocarditis, transthoracic echocardiography is the initial diagnostic procedure of choice.[7] A clinically suspected diagnosis of mitral stenosis can be accurately determined with transthoracic echocardiography. Left atrial myxomas are rare but may present with thromboembolism and features of a systemic illness.[8] Transthoracic echocardiography is generally highly sensitive for detecting myxomas although transoesophageal echocardiography may detect smaller or multiple myxomas.[9]

Applications of transoesophageal echocardiography

The low yield of transthoracic echocardiography in the evaluation of stroke in patients without clinical evidence of heart disease is related to the low sensitivity of the technique for definite sources of emboli such as left atrial and left atrial appendage thrombus[3] and protruding atheroma in the ascending aorta and aortic arch.[10,11] Transoesophageal echocardiography uses high-frequency ultrasound which, along with close proximity of the transducer to the heart and aorta, significantly improves identification of abnormalities definitely related to stroke, such as small cardiac tumours as well as left atrial thrombi and aortic lesions. The yield for any potential source of embolus (probable or possible) in stroke is doubled from 19% with transthoracic echocardiography to approximately 40% with transoesophageal echocardiography in patients without clinical evidence of heart disease.[12,13]

The majority of the potential sources of emboli identified with transoesophageal echocardiography include patent foramen ovale, small atrial septal defects, atrial septal aneurysm and spontaneous echocardiographic contrast.[12-15] Despite the increased sensitivity of transoesophageal echocardiography, there are no data to suggest identification of these potential (in contradistinction to definite or probable) sources of emboli significantly impacts on patient management.[16] This largely relates to the lack of firm evidence indicating whether active treatment of patients with these potential sources of emboli results in a reduction in subsequent stroke rate.

Risk factors for stroke

Aortic atherosclerotic disease in patients presenting with stroke has recently been established as an independent risk factor for recurrent stroke.[17] The risk of embolus is increased when there is complex aortic atheroma which is pedunculated and highly mobile.[11] The absence of cardiac disease does not exclude the presence of complex aortic atheroma which may have a prevalence of up to 5% in

patients after stroke or systemic embolus who are in sinus rhythm and have a normal transthoracic echocardiographic study.[18] Patients with a history of systemic emboli and mobile aortic atheroma treated with warfarin (international normalised ratio 2.0), tend to have a decreased risk of subsequent stroke compared to those treated with aspirin.[19] A more recent observational study has shown that patients with mobile aortic debris have a reduction in both combined events and mortality, when treated with anticoagulation compared to antiplatelet therapy.[20] Confirmation of these preliminary findings in a randomised trial is likely to strengthen the case for routine use of transoesophageal echocardiography to detect complex aortic atherosclerosis in patients with stroke.

Age influences the risk of cardioembolic stroke with an increased prevalence of left atrial thrombus and atherosclerotic plaques in patients older than 50 years.[21] Although the prevalence of these abnormalities is lower in those with stroke under the age of 45 to 50 years, up to 35% of these patients will have a cardiac source of embolus identified.[22] There is an increased prevalence of abnormalities of the interatrial septum, including patent foramen ovale, atrial septal defects and atrial septal aneurysm in younger patients.[18] Although the clinical significance of these abnormalities remains unclear, their detection is generally regarded as important especially as other detectable sources of embolism are less frequent in younger patients. Given the increased sensitivity of transoesophageal echocardiography for cardiac abnormalities in young patients after stroke[23] this investigation should be considered the procedure of choice in evaluating potential cardioembolic sources of emboli in this age group.

Transthoracic echocardiography is usually inadequate for obtaining a full assessment of prosthetic heart valve morphology and function. The use of transoesophageal echocardiography improves the assessment of prosthetic heart valves for evidence of vegetations, valvular dysfunction and associated thrombus. Transoesophageal echocardiography in patients with prosthetic valves and systemic emboli changes clinical management in approximately one third of cases[24] and should be used routinely in the evaluation of patients with prosthetic valves and stroke.

Atrial fibrillation is a strong risk factor for stroke and the highest risk of stroke in patients with atrial fibrillation (annual risk 12%) occurs in those with previous transient ischaemic attack or stroke.[25] Patients in atrial fibrillation constitute approximately 20% (range 7% to 36%) of all patients with embolic stroke but account for 50% of patients where left atrial thrombus is identified as the probable source of embolus.[13,15,26-31] Warfarin is more effective as secondary prophylaxis against stroke in patients with atrial fibrillation than aspirin and should be considered in all patients without contraindications to anticoagulation.[32] Initiation of warfarin in patients with atrial fibrillation and stroke is generally a clinical decision and is unlikely to be influenced by echocardiographic findings. However, transthoracic echocardiography may be useful in patients with atrial fibrillation to clarify cardiac pathology.

Indications for echocardiography

An approach to the echocardiographic evaluation of embolic stroke is outlined in Figure 1. If a patient is not a candidate for anticoagulation or surgery then echocardiographic evaluation for a source of embolus may not be warranted as the findings will not influence subsequent management. Patients in the younger age group are best investigated with transoesophageal echocardiography because of its increased sensitivity. In older patients with stroke and atrial fibrillation anticoagulation is usually indicated and echocardiography is only likely to have a role in clarifying aspects of cardiac pathology.

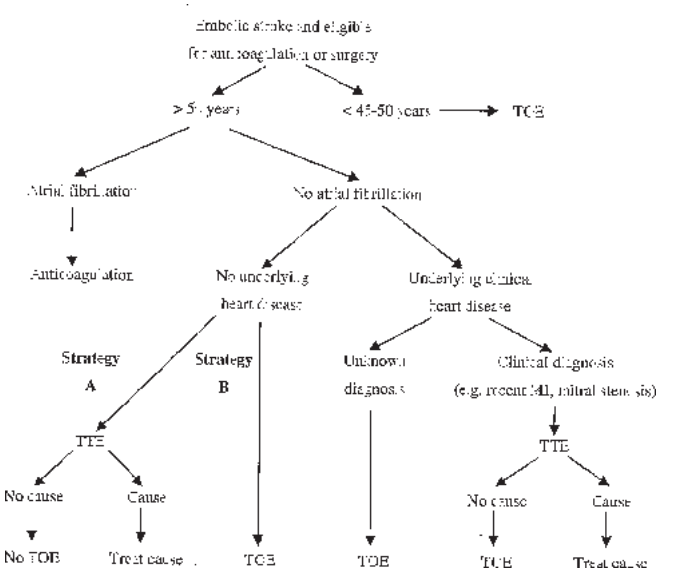

Figure 1. Algorithm for use of transoesophageal echocardiography (TOE) and transthoracic echocardiography (TTE).

Patients with clinical evidence of heart disease

Patients in sinus rhythm with stroke or systemic embolism may have up to an 18% prevalence of left atrium or left atrial appendage thrombi which will generally not be detected by transthoracic echocardiography.[12,29] Transoesophageal echocardiography is indicated in the majority of patients with stroke and clinical evidence of heart disease. Performing initial transthoracic studies in all these patients is time-consuming and is unlikely to significantly decrease the number of subsequent transoesophageal studies required for the exclusion of potential sources of emboli. Exceptions to this approach occur when there is a high pre-test probability of identifying an abnormality such as left ventricular thrombus after myocardial infarction or with clinically diagnosed mitral stenosis where a transthoracic study alone can be used to guide therapy. Patients without an identifiable cause after such a targeted transthoracic study should usually subsequently have a transoesophageal study.

Patients without clinical evidence of heart disease

Evaluation of stroke patients in sinus rhythm with no clinical evidence of heart disease, is more problematic because of the lower prevalence of probable or definite sources of emboli. Such patients have approximately a 1% prevalence of left atrial thrombus[15,30] and up to a 5% prevalence of complex aortic atheroma.[18] Two possible approaches to the investigation of stroke patients without clinical evidence of heart disease are outlined in Figure 1. Strategy A represents common practice in many hospitals where a transthoracic study is the only investigation performed or transoesophageal studies are performed only if abnormalities are identified on the initial transthoracic study. This approach avoids the requirement of performing large numbers of relatively low-yield transoesophageal studies but risks failure to diagnose thrombus or complex aortic atherosclerosis and commence anticoagulant therapy in a small number of patients who may benefit from treatment. Strategy B is to perform transoesophageal studies in all patients without clinically evident cardiac disease. Instituting

this approach would pose difficulties in New Zealand given the limited number of hospitals where transoesophageal echocardiography is available and also constraints on the availability of transoesophageal studies at facilities offering this investigation.

At present there is no definitive trial evidence to indicate which of the two strategies is more effective. A recent cost-effectiveness analysis concluded that performing only transoesophageal studies in all persons with stroke to detect left atrial thrombus was more cost-effective than using transthoracic echocardiography alone or in sequence with transoesophageal studies.[33] Transoesophageal echocardiography in patients with unexplained cerebral ischaemia has shown an increased risk of recurrent stroke in patients with atherosclerotic aortic plaque treated with aspirin alone.[34] Emerging evidence suggesting anticoagulation is effective as secondary prophylaxis in stroke patients with complex aortic atheroma[19,20] strengthens the case for routine transoesophageal echocardiography after embolic stroke. However, this approach needs to be formally evaluated against empiric anticoagulation therapy for patients with suspected stroke unexplained by carotid atherosclerotic disease.[34]

Conclusions

Transoesophageal echocardiography is indicated in patients with embolic stroke and clinical evidence of heart disease. Further guidance on the indications for transoesophageal echocardiography in patients without clinical evidence of heart disease and the effect of this investigation on management and patient outcome can only be determined from prospective randomised trials.

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Most leaders of American medicine are busy now just coping – trying to preserve the membership of their societies, struggling to increase their share of the market, striving to save their research and teaching missions, and conspiring to survive in the intensely competitive marketplace. Many acknowledge their deep concern about the system privately but publicly remain silent. Compromising care to control cost is a vexing social issue in which the integrity of the profession is at stake, and medicine must have a clear, strong voice in those public decisions. Before we face far more odious choices, we must come to grips with these difficult trade-offs. So far, except for a few voices in this country, the air is filled with a strained silence.

Jerome P Kassirer. Our endangered integrity – it can only get worse. N Engl J Med 1997; 336: 1666-7.

Book Review

Molecular Biology in Reproductive Medicine

B Fauser, A Rutherford, J Strauss, A Van Steirteghem. The Parthenon Publishing Group. Contains 523 pages. ISBN: 1 85070 994 7. Price not stated.

In the last twenty years there have been remarkable scientific discoveries about how the nervous and endocrine systems regulate the male and female reproductive systems. The identification of protein molecules, and the genes regulating their synthesis, is enhancing our understanding of reproductive function and dysfunction.

This book attempts to integrate all the basic science discoveries in reproductive biology with the clinical science of reproductive medicine. The advances are well reviewed, and scientists and students of reproductive biology will regard this new text as inspiring and comprehensive. Clinicians wishing to update on the scientific advances at a molecular level will probably be overwhelmed by the detail, and disappointed in the relevance of that detail to most of their clinical practice.

The text is well set out in a generally consistent manner with good supporting diagrams and vast lists of references. The authors of the chapters have obviously been carefully chosen.

The rapid advances in molecular biology mean, however, the useful lifetime for this textbook in science libraries and fertility clinics is unlikely to exceed five years.

John Hutton,
Wellington.

VIEWPOINT

Asthma in New Zealand: myths and realities

Shaun Holt, Clinical Research Fellow; Neil Pearce, Professor, Wellington Asthma Research Group, Department of Medicine, Wellington School of Medicine, Wellington.

Abstract

Asthma is a major health problem in New Zealand, generating large costs to the health services and industry. However, there are several myths concerning asthma in New Zealand. These are that it is more common and more severe in New Zealand than in the rest of the world, that it is more common in rural areas and that asthma mortality in New Zealand is the highest in the world. Using recently published data, it is now possible to show that none of these "facts" is true. In fact, asthma in New Zealand appears to have a prevalence and severity similar to those of the other major English-speaking countries and in this respect can be thought of as being first equal in the world. The relevance of correcting these myths is that they could influence future research. Previous research into the causes of asthma has sometimes focused on factors unique to New Zealand in an attempt to explain the increasing prevalence of asthma around the world. It may be more productive to direct future research towards identifying factors that the major English-speaking countries have in common.

NZ Med J 2000; 113: 39-41

Asthma is an emotive subject in New Zealand. It is well recognized as a serious problem affecting a large proportion of the population[1,2] and it is estimated to generate about $150 million per year in direct costs to the health services and almost as much in indirect costs such as lost productivity.[3] Most New Zealanders will have one or more asthmatics in the family, or will have a friend with asthma, and many New Zealand families have several members with a diagnosis of asthma. Furthermore, there has been considerable publicity about the epidemic of asthma deaths that occurred in New Zealand during 1976-1989.[4,5]

There has also been considerable speculation about possible causes of asthma in New Zealand and possible regional variations due to differences in exposure to allergens, agricultural sprays, air pollution, etc. It is thus not surprising that several "great New Zealand asthma myths" have developed, based largely on speculation or anecdotal evidence. Although there are few, if any, published references on these issues, our discussions with respiratory physicians, asthma educators and asthmatic patients, both in New Zealand and internationally, indicate that it is often assumed that: (1) New Zealand has the highest asthma prevalence in the world; (2) there are major urban/rural differences; (3) asthma is more severe in New Zealand than in other countries; and (4) asthma mortality in New Zealand is the highest in the world.

It is now possible to evaluate these common assumptions using recently published standardized international asthma prevalence data in children[2,6] and adults,[7] standardised regional comparisons of adult asthma prevalence in New Zealand[1] and international comparisons of asthma mortality time trends.[8]

New Zealand has the highest asthma prevalence in the world

Perhaps the most frequently quoted "myth" is that New Zealand has the highest asthma prevalence in the world. In particular, it is often claimed that asthma is more common in New Zealand than in other English-speaking countries such as Australia, the United Kingdom, Ireland, the United States and Canada. In fact, it is well established that, for reasons that are currently unknown, all of the major English-speaking countries have particularly high asthma prevalence rates.[6,7] However, there is no evidence that asthma is more common in New Zealand than in the other English-speaking countries. In particular, Figure 1 shows that the asthma prevalence in children aged 13-14 years is similar in all six English-speaking countries included in the International Study of Asthma and Allergies in Childhood (ISAAC) survey,[6] with the highest prevalence being recorded in the United Kingdom. There are similar patterns in adults, with the highest prevalence being observed in Australia, but with little variation between the five English-speaking countries that took part in the European Community Respiratory Health Survey.[7]

Collectively, the English-speaking countries have the highest asthma prevalence in the world, but the prevalence in New Zealand is similar to that in other English-speaking countries.

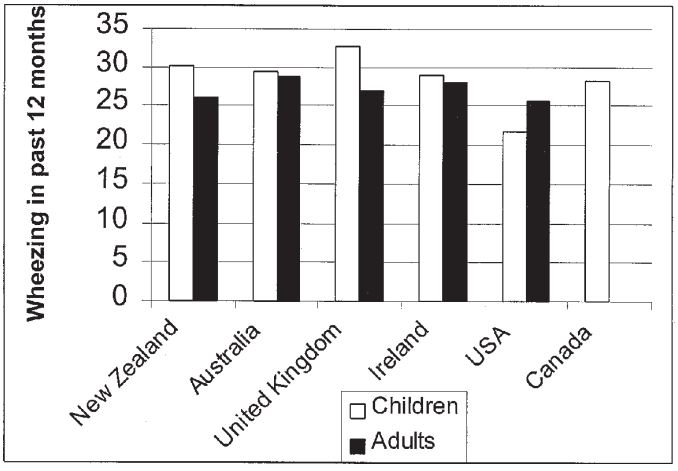

Figure 1. Asthma prevalence in 13-14 year-old children and in adults aged 20-44 years in English-speaking countries.

Asthma is more common in rural areas

It is also commonly assumed that asthma prevalence is higher in rural, rather than urban, areas.[9] This is probably because some environmental factors which have been shown to exacerbate asthma are more commonly found in rural areas. These include pollens and other grass allergens, allergens from animals and agricultural chemicals such as pesticides. However, geographical variations in asthma prevalence in New Zealand were examined in 1997 in a questionnaire survey of over 25 000 randomly selected adults aged 20-44.[1] The highest prevalence of asthma was seen in some areas of

Auckland and Wellington, although equally high rates were found in some rural areas such as Wairarapa. On both major islands, the lowest asthma prevalence was found in rural areas such as Rotorua and Wallace. The asthma prevalence was 15.5% in the main urban centres (Auckland, Wellington, Christchurch, Hamilton, Palmerston North, Dunedin), 14.7% in provincial centres and 13.8% in rural electorates. Thus, although the prevalence in some rural areas was very high, in some areas it was very low and the overall prevalence in rural areas was a little lower than in urban areas.

Further evidence comes from the six New Zealand centres included in the ISAAC study.[2] There were only minor differences between the six centres but the lowest rates were found in rural Nelson rather than the cities of Auckland and Wellington, in both 6-7 year-old and 13-14 year-old children. This is consistent with other research which shows little urban / rural asthma prevalence differences in Western countries and relatively higher prevalence in urban areas in developing countries.[10] This adds support to the hypothesis that the worldwide increasing prevalence of asthma may be due in some way to increasing urbanization and affluence.[6]

Asthma is more severe in New Zealand

It is also commonly believed that not only do more people have asthma in New Zealand but that those who have asthma are more severely affected. As with prevalence, the best available evidence suggests that although many New Zealand asthmatics could be classified as severe, the proportion is no higher than in other English-speaking countries. For example, the ISAAC study includes a question on "severe wheeze limiting speech".[2] The proportion of 13-14 year-old children who answered 'yes' to this question in New Zealand was 8.0% but the proportions were similar in Canada and Australia, and were higher in the United States (10.0%) and the United Kingdom (8.5%) (Figure 2). Again, New Zealand occupies an approximately "first equal" position, although it is actually fifth out of the six major English-speaking countries. This further validates the previously stated hypothesis that the high prevalence of asthma and severe asthma is not unique to New Zealand but is common to English-speaking countries. The European Community Respiratory Health Survey included a similar question. The prevalence of severe asthma in adults was highest in Australia, with New Zealand in second place, but once again a similarly high prevalence was shown across all of the English-speaking countries[7] (Figure 2).

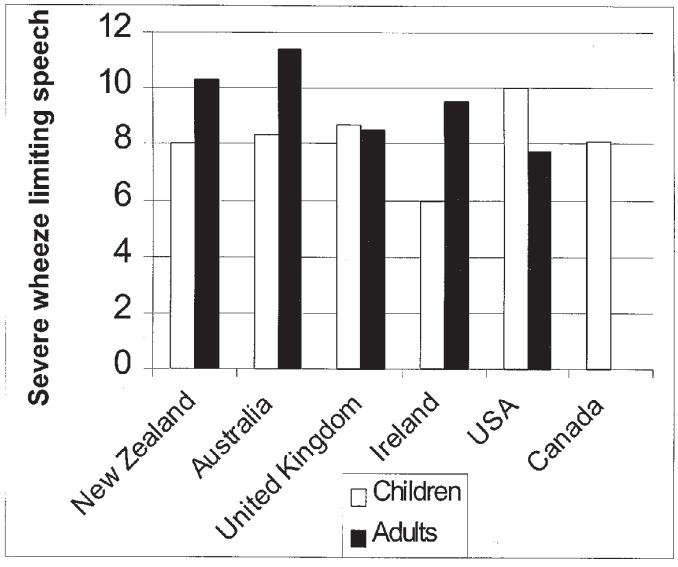

Figure 2. Prevalence of severe asthma in 13-14 year-old children and adults aged 20-44 years in English-speaking countries.

Asthma mortality in New Zealand is the highest in the world

The origins of the belief that asthma is more severe in New Zealand and that mortality rates are also the highest in the world, can perhaps be traced to the recent epidemic of deaths in New Zealand attributable to the inhaled beta-agonist drug, fenoterol.[11] There was a sudden increase in the number of asthma deaths in New Zealand in 1976 when fenoterol was introduced[5] and the New Zealand asthma death rate was the highest in the world until 1989. Time-trend data showed that there was no association between the epidemic of deaths and other possible causes such as total sales of inhaled beta-agonists, underprescribing of inhaled corticosteroids or social factors such as unemployment.[5] A New Zealand case-control study was published in *The Lancet* in 1989, which associated inhaled fenoterol with the epidemic of deaths.[4] Following a warning about the safety of the drug by the New Zealand Department of Health, the drug was withdrawn from the Drug Tariff in 1990. Fenoterol virtually disappeared from the market in New Zealand and there was a sudden dramatic fall in the death rate.[5] Since the removal of fenoterol from the New Zealand market, the asthma death rate in New Zealand is again similar to that of England and Wales, Australia, West Germany, Canada and the United States[8] (Figure 3).

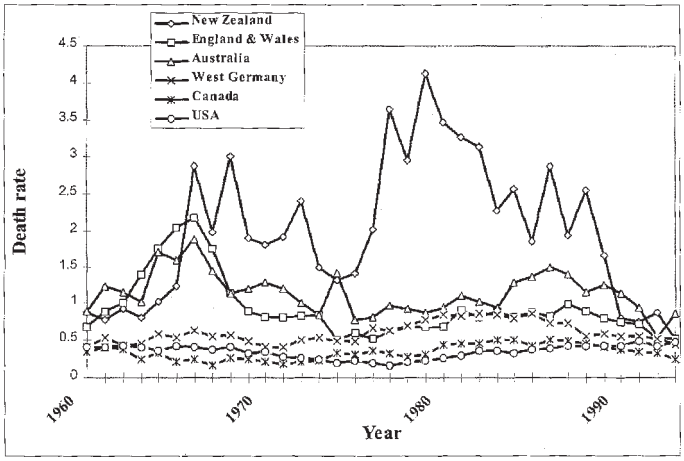

Figure 3. Asthma mortality in the 5-34 age-group in New Zealand and other Western countries.

Discussion

Thus, the available evidence indicates that asthma prevalence, severity and mortality in New Zealand are no higher than in other English-speaking countries. Furthermore, there is no evidence of systematic urban/rural differences. Why therefore have these "asthma myths" arisen? The reasons for this are unclear but they may stem in part from the considerable publicity about the epidemic of asthma deaths that occurred in New Zealand during 1976-1989. This was eventually linked to the beta-agonist drug fenoterol and mortality fell immediately following the restriction of the availability of this drug.[5] However, for a long period in the 1980s the cause of the epidemic was unknown and this led to considerable speculation about the possible causes of the epidemic and the possibility that asthma may be more common or more severe in New Zealand than in other countries. There has also been considerable related speculation about possible causes of asthma in New Zealand and possible regional differences due to differences in exposure to allergens, agricultural sprays, air pollution, etc.[9]

It should be emphasised that, although asthma is no more common or severe in New Zealand than in other English-speaking countries, and that New Zealand is only "first equal"

in the world for asthma prevalence and severity, this does not discount the huge burden of morbidity from asthma in New Zealand and the huge costs to the health services and the country.[3] Thus, asthma remains of major importance for the health services and for health researchers in New Zealand.

So do the myths matter? Unfortunately they do, because these common misconceptions have had a major influence on how asthma has been researched and managed in New Zealand. For a number of years it was assumed that asthma prevalence was uniquely high in New Zealand, and research has therefore focused on factors that appear to be relatively unique to New Zealand, e.g. the coastal humid environment, the high indoor and outdoor allergen levels, etc. However, it now appears that factors that are unique to New Zealand are most unlikely to be major contributors to the high asthma prevalence here. This therefore requires a major reorientation of asthma research to focus on factors that are common to all of the English-speaking countries, rather than factors that are unique to New Zealand. In particular, there is growing evidence that the increases in asthma worldwide, and perhaps also the very high prevalences in English-speaking countries, are not due to changes in exposures to "asthma triggers" (e.g. house dust mites and other indoor allergens)[12] and that, in any case, the proportion of asthma cases that are attributable to atopy is probably less than one-half.[13] Rather it now appears more plausible that these increases are due to changes in immune function and the underlying susceptibility to asthma, due to changes in exposure in utero and in infancy. In particular, there is increasing evidence that the "cleaner" environment and reduced rate of infections that results from increasing affluence and reduced family size may be "programming" the immune system and making it more susceptible to the development of asthma and allergy.[14] To understand this complex process and to ultimately develop appropriate preventive measures, it is necessary that asthma research, education and management in New Zealand should be evidence-based, and that we should critically examine common perceptions and myths about the causes of asthma in New Zealand.

Acknowledgement. The Wellington Asthma Research Group is supported by a programme grant from the Health Research Council of New Zealand.

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LETTERS

Letters to the editor should be signed by all authors, typewritten in doublespacing, not exceed 400 words and 10 references. References should be in the Vancouver style. Over long letters may be shortened without reference to the author unless it is specifically stated otherwise. Priority of publication may be given to short letters.

Excessive workloads for junior doctors

As a medical student reading your journal, I was alarmed to learn of the circumstances surrounding the medicolegal case published on December 10 1999 (NZ Med J 1999; 112: 477). In giving its decision, the Medical Council took into account the fact that the junior doctor involved in the case had been on duty for 24 hours as well as there being a heavy commitment of cover that night. What other occupation or profession would allow its junior members to make life threatening decisions at the end of a 24 hour shift? There is legislation to prevent truck drivers and airline pilots endangering the lives of others by working excessive hours. Are doctors not considered to be as susceptible to tiredness?

It is perhaps not surprising that one reads articles in the newspaper (for example, Waikato Times, Wednesday 15th December 1999) indicating that people are attracted to Australia due to the more reasonable working conditions. Is it not time that the Medical Council took action on this systematic failure of professional standards?

Joanna Lawrence,
Education Representative,
Auckland University Medical Students Association.

General practice obstetrics

Speaking as a general practitioner who previously practised obstetrics and who feels our representatives have been out-manoeuvred in the various negotiations, tribunals and reviews in recent years, I propose that the NZMA orchestrate the withdrawal of all general practitioner's who no longer deliver babies from the Maternity Benefits Scheme.

In my view, it is high time that our representatives led us to demonstrate some collective strength over this issue. The sorry result of endless negotiation has been one of the progressive destruction of general practice obstetrics, and it is time for action. As I understand it, general practitioners can ask in writing to resign from the scheme. This is then gazetted in Parliament. We are then no longer obliged to accept a fixed fee of $25 for antenatal consultations, and can charge our normal fee for service. For those general practitioners continuing to deliver I would not expect them to give up a significant part of their income, but for the rest of us, for which maternity care represents a small service part of our practice, financial losses should be minimal or non-existent given that we can then charge normal fees.

This move is not without precendent. When Dr Bassett was Minister of Health for the Labour Government coming out of the price freeze, a number of delaying tactics were employed to stop general practitioners having the then maternity benefits tribunal reviewing our fees. It was only when a number of colleagues and myself managed to sign up the entire general practitioner obstetric community on the North Shore to a letter of resignation from the Maternity Benefits system that this tribunal was allowed to proceed.

With the right leadership this can be done again, but only if it is orchestrated in an en masse letter of resignation as opposed to individuals resigning unnoticed.

M Hoogerbrug,
Browns Bay,
Auckland.

Quality assurance in colposcopy using video capture and the internet for individual audit

Recent studies have highlighted the fact that adverse events and errors occur in the healthcare industry frequently enough to cause concern, and to warrant continuing attempts at improvement.[1] There is a requirement by the RANZCOG for continuing education requirements to include quality assurance activities. There is also a demand from the community and health purchasers for the profession to address variations in practice and adopt policies of continuing improvement. Variations in colposcopy diagnosis can be wide, studies at unaccredited practices being poorly accurate.[2]

The authors who perform colposcopy in surgeries several hundred kilometres apart have developed a simple method for peer review in clinical practice. Colposcopes with beam splitters are connected to digital cameras thus enabling the cervical image to be displayed on a monitor. The patient is able to view her cervix if

she so desires. The image is captured through a video capture card and is then downloaded to a computer hard drive. A simple visual basic programme automates and manages the storage of the images.

The video pictures are sent as file attachments via email in JPEG format from one colposcopist to the other, coded to preserve confidentiality of patient identity. Accompanying the picture is a brief clinical history, the cervical cytology and the colposcopic diagnosis. The histology is provided later so that a final correlation between cytology colposcopy and histology can be made. As a further refinement JPEG images of the histology are available from collaborating laboratories.

The advantage of this system is that it provides any practitioner and especially those in professionally isolated locations the opportunity to share colposcopic images with colleagues. Such peer to peer collaboration does not usually occur at consultant level in clinical practice. The system involves low set up costs and minimal costs for information transfer. In addition the images can be stored on the computer as part of the patient clinical records. The patient and the referring doctor can receive computer printed copies and the files form an important litigation defence resource. We propose the system as an adjunct to quality assurance CME observance.[3,4]

Bernie N Brenner,
North Shore Day Surgery,
Auckland.

Al M Donoghue,
Lucanus Corporation,
Wanganui.

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Diabetes is still unrecognised in the 1990's: report of a hospital audit

Diabetes mellitus is under-diagnosed. New Zealand population based studies find one third of those with diabetes were undiagnosed[1] and a workforce study revealed 50% were undiagnosed.[2] Type 2 diabetes is suitable for screening.[3] Hospital admission provides an opportunity for screening and intervention.[4] We audited the response of hospital medical staff to the identification of hyperglycaemia.

Since there are no recognised criteria for the diagnosis of diabetes in a stressed state, we arbitrarily chose a random plasma glucose level (RPG) ≥ 7.8 mmol/L as indicating an abnormality in glucose handling on hospitalisation. Patients with a RPG above this level were divided into two subgroups, RPG 7.8-11.0 mmol/L and ≥11.1 mmol/L. From the hospital notes, demographic and clinical data were also extracted.

Of 2734 adult patients admitted to Wellington and Kenepuru hospitals during November 1997, 60% had a RPG measured. Of 317 patients who had a RPG ≥ 7.8 mmol/L we received the hospital notes of 280 (88.3%). One hundred and fifteen (41%) had known diabetes. Of the remaining 165 patients, 117 had a RPG between 7.8-11.1 mmol/L but in only 2.6% of cases was this recorded on the discharge summary. For those 48 patients with a RPG ≥ 11.1 mmol/L, 15 (31.3%) had some documentation in the hospital notes, but in only 14.6% did this reach the discharge summary.

There remains uncertainty in the literature as to just what the significance of "stress hyperglycaemia" is.[4,5] HbA1c has been used to separate hyperglycaemia due to stress from pre-existing diabetes mellitus.[6,7] In our audit, no patient had an HbA1c performed. Some patients with drug or other stress-induced glucose intolerance will develop diabetes each year. A three year follow up study of hyperglycaemia on surgical wards revealed that almost 50% of those with a RPG ≥/10 mmol/L on the ward and alive three years later had diabetes. A further one third had impaired glucose tolerance.[8]

It should not be assumed that "stress hyperglycaemia" is a benign state. It is an opportunity to make an early diagnosis of diabetes or to identify an individual at risk of future diabetes and therefore in need of regular screening. We believe that a high RPG should be documented not only in the hospital records but also for the general practitioner at discharge.

Jeremy Krebs,
Endocrinology Department
Wellington Hospital.

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Myocardial infarction and rupture

Recently the Health and Disability commissioner reported on a missed diagnosis of myocardial infarction (MI) in a 55 yr old patient who died of a ventricular rupture four days after the consultation. "There had been a three day history of tiredness, nausea and a feeling of something being stuck in his throat. He was continually burping and rubbing his chest to dislodge the discomfort. He was very stressed and his colour was taking on a greyish tinge."[1] The general practitioner recorded normal physical findings (heart sounds, rhythm and auscultation). Cardiac risk factors were not assessed. A diagnosis of gastritis and duodenitis was made. The commissioner found that "The general practitioner did not adequately consider that the patient's symptoms could be cardiac" and "The general practitioner failed to both undertake an appropriate examination and record details in the notes."

In the study by Baker and Koelmeyer,[2] 71% of patients with a myocardial infarction and rupture were not referred because the diagnosis was missed. They report that clinically unrecognised MIs are reported to be 20% to 60% of all MIs. The addition of rupture makes the diagnosis even more difficult and "nearly always fatal" for the patient. Baker and Koelmeyer's advice is to pay attention to those at risk with vague symptoms and to those who appear quite unwell (common symptoms in general practice). Is admission to hospital for assessment for all patients in this group to be encouraged?

Was the doctor mentioned above, negligent? There is reference to "reasonableness" in the Code of Health and Disability Services Consumer's Rights and this implies that the tests for negligence would be considered. Would a responsible general practitioner have diagnosed the MI with rupture and if he had diagnosed and admitted the patient would the patient have survived?

It is negligent for a doctor not to take an appropriated history and not to perform an appropriate physical examination, irrespective of outcome.[3] The acceptable time the general practitioner has to spend with the patient is 15 minutes. General practitioner's who perform 60 minute general physical examinations with patients in their underwear would be unlikely to miss as many difficult diagnoses. The financial burden to the patient may be unacceptable. Has the time come for general practitioner's to spend more time with their patients, have a higher index of suspicion of serious disease and be more willing to refer patients to hospital?

John Kennelly
Waiatarua,
Auckland.

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Notices

Graham Aitken Nuffield Medical Postgraduate Travelling Scholarship

Applications are invited from well qualified New Zealand medical graduates in the 25-35 age group for the above Scholarship. The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom. The Scholarship will provide up to three return air fares to the U.K., together with allowances amounting to $3000.

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the U.K. who will provide salary and facilities.

For further information pleases consult the Deans of the Schools of Medicine or write to Professor AD Campbell, Honorary Secretary, Managing Trustees, Graham Aitken Nuffield Trust, C/- Department of Chemistry, University of Otago, PO Box 56, Dunedin. Email: adc@otago.net.nz

Applications must be submitted to Professor Campbell by 31 March 2000.

Medical Council Election Results

The Medical Council announced the result of the election of four doctors to the Council in November 1999. Three current members have been re-elected and one new member. The successful candidates who will serve on the Council for the next three years are Dr Mark Adams (anaesthetic registrar, Hutt Hospital, currently on the Council), Dr Tony Baird (obstetrician and gynaecologist at National Women's, Auckland, currently President of the Medical Council), Dr John Neutze (Clinical Director, Paediatric Cardiology, Green Lane, Auckland) and Dr Ian St George (general practitioner of Wellington, currently Deputy President of the Council).

There were 24 candidates standing for election. This was only the second time that doctors have been able to elect their peers to the Council, the first being in 1996. The new Council took office on 10 December 1999 and will vote for their President in February 2000.