

Homocysteine and vitamin status in older people in Perth

Leon A Flicker,* Samuel D Vasikaran,[†] Jenny Thomas,[‡] John G Acres,[§] Paul E Norman,^{||} Konrad Jamrozik,^{**} Nicola T Lautenschlager,^{††} Peter J Leedman,^{##} Osvaldo P Almeida^{§§}

* Professor of Geriatric Medicine, ‡ Research Nurse, School of Medicine and Pharmacology, § Research Fellow, School of Medicine and Pharmacology and School of Psychiatry and Clinical Neurosciences, ¶ Associate Professor of Surgery, †† Senior Lecturer in Psychiatry of Old Age, ††† Professor of Medicine, §§ Professor of Psychiatry of Old Age; University of Western Australia, Royal Perth Hospital, Box X2213, Perth, WA 6000. † Head, Department of Core Clinical Pathology and Biochemistry, Royal Perth Hospital, Perth, WA. ** Professor of Primary Care Epidemiology, Imperial College London, London, UK. leonflic@cyllene.uwa.edu.au

TO THE EDITOR: Elevated levels of homocysteine (Hcy) have recently been associated with increased risk of vascular events¹ and dementia.² The clearance of Hcy is dependent on three vitamins — folate, B₆, and B₁₂. Vitamin B₁₂ deficiency has been described in older people for over 40 years,³ and may have wide-ranging effects through this vitamin's influence on Hcy. The aims of this study were to examine serum B₁₂ and folate status, and their relationships with plasma Hcy concentrations, in community-dwelling healthy older people living in Perth.

Older men and women were recruited from two different sources: 299 men aged 75 years and over were recruited from a large population-based study of

screening for abdominal aortic aneurysm,⁴ where 70% of those invited joined the project; and we recruited 273 community-dwelling women aged 70 years and over through advertisements. Exclusion criteria for both groups included significant cognitive impairment, severe physical illness and current use of B-group vitamin supplements. The Human Research Ethics Committee at the University of Western Australia approved the study, and all participants provided informed consent.

Fasting total plasma Hcy, serum B₁₂ and folate concentrations were measured in all participants, and serum creatinine concentration was measured in the men only to calculate glomerular filtration rate (cGFR). For analyses, the variable plasma Hcy was heavily skewed to the right and natural logarithmic transformation was used. Pearson's product moment correlations were calculated for univariate analyses of continuous variables.

Descriptive statistics are presented in Box 1. Fourteen per cent and 1% of the men, and 6% and 1% of the women, were deficient in B₁₂ and folate, respectively. Hcy concentrations above upper reference limits (15 µmol/L for men and 13 µmol/L for women) were found in 24% of both men and women. There were significant ($P < 0.001$) positive correlations between age and log Hcy concentration for men ($r = 0.23$; 95% CI, 0.12–0.33) and women ($r = 0.25$; 95% CI, 0.13–0.36), inverse correlations

between B₁₂ and log Hcy concentrations for men ($r = -0.25$; 95% CI, -0.14 to -0.35) and women ($r = -0.30$; 95% CI, -0.19 to -0.41), and inverse correlations between folate and Hcy concentrations for men ($r = -0.43$; 95% CI, -0.33 to -0.52) and women ($r = -0.28$; 95% CI, -0.16 to -0.39). Plots of log Hcy against B₁₂ and folate concentrations for all participants are presented in Box 2. Under multiple regression, the association of B₁₂ and folate concentrations with log Hcy concentration remained after adjustment for age and cGFR in men only; beta values (SE) were:

- -0.00060 (0.00011) for B₁₂ concentration;
- -0.0155 (0.0017) for folate concentration;
- -0.0029 (0.0008) for cGFR; and
- 0.012 (0.005) for age.

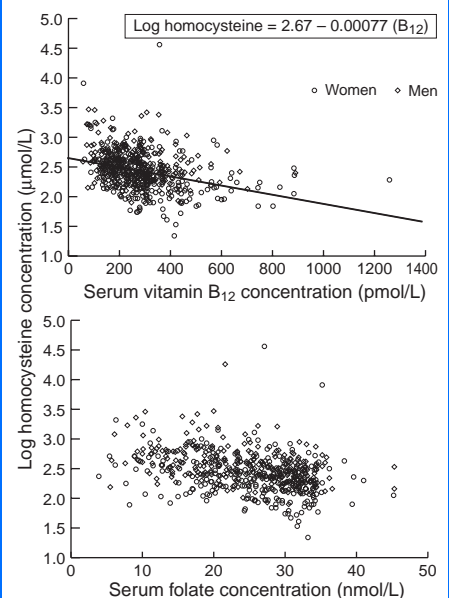
In this sample there were high prevalences of B₁₂ deficiency and hyperhomocysteinaemia. Although the prevalence of folate deficiency was substantially lower, there were still moderate inverse associations between serum folate and Hcy concentrations. Unfortunately, vitamin B₁₂ deficiency of this kind may not be universally corrected with small doses of oral supplements,⁵ and this has intensified concerns about precipitating neurologi-

1: Demographic characteristics, serum B₁₂ and folate, and plasma homocysteine in 299 older men and 273 older women

	Men		Women	
	Mean (SD)	Range	Mean (SD)	Range
Age (years)	78.9 (2.8)	68–86	74.8 (4.4)	70–92
Weight (kg)	78.4 (1.2)	50.6–119.5	69.3 (1.3)	39.0–120.0
Height (cm)	171 (6.5)	150–197	159 (6.7)	132–176
Body mass index (kg/m ²)	26.6 (3.5)	16–37	27.4 (5.3)	17–52
Ever smoked	66%		41%	
Ever drank alcohol	95%		66%	
Serum folate (nmol/L)	24.3 (7.6)	5.5–45.3 (RI, 7–34)	25.3 (7.6)	3.9–45.2 (RI, 7–34)
Serum B ₁₂ (pmol/L)	254.5 (116.7)	57–890 (RI, 140–646)	313.5 (158.7)	59–1270 (RI, 140–646)
Plasma Hcy (µmol/L)	13.50 (5.3)	6.7–70.5 (RI, 6.0–15.0)	11.46 (6.8)	3.8–96 (RI, 5.0–13.0)
Glomerular filtration rate (mL/min)	78.3 (16.3)	35.8–142.4		

SD = standard deviation. Hcy = homocysteinaemia. RI = reference interval.

2: Plot of serum B₁₂ and folate concentration against log homocysteine concentration (with regression line for B₁₂) in 299 older men and 273 older women



cal complications by population-based folate supplementation.⁶ There is a need for intervention studies of B-group supplements to evaluate whether the risks associated with hyperhomocysteinaemia can be ameliorated.

Acknowledgements: This work was supported by grants from the National Health and Medical Research Council of Australia, and untied research grants from Pfizer CVL, Australian Rotary Health Research Fund and the Australasian Menopause Society. We are grateful to the men and women who participated in this study.

1. Hankey GJ, Eikelboom JW. Homocysteine and vascular disease. *Lancet* 1999; 354: 407-413.
2. Seshadri S, Beiser A, Selhub J, et al. Plasma homocysteine as a risk factor for dementia and Alzheimer's disease. *N Engl J Med* 2002; 346: 476-483.
3. Cape RDT, Shinton NK. Serum vitamin B₁₂ concentration in the elderly. *Gerontologia Clinica* 1961; 3: 163-172.
4. Jamrozik K, Norman PE, Spencer CA, et al. Screening for abdominal aortic aneurysm: lessons from a population-based study. *Med J Aust* 2000; 173: 345-350.
5. Seal EC, Metz J, Flicker L, Melny J. A randomised, double blind, placebo controlled study of oral vitamin B₁₂ supplementation in elderly patients with subnormal or borderline serum vitamin B₁₂ concentrations. *J Am Geriatr Soc* 2002; 50: 146-151.
6. Reynolds EH. Benefits and risks of folic acid to the nervous system. *J Neurol Neurosurg Psychiatry* 2002; 72: 567-571. □

Failed sterilisations and the unwanted child: a new medicolegal minefield?

Malcolm H Parker

Associate Professor of Medical Ethics, School of Medicine, University of Queensland, Herston Road, Herston, QLD 4006. m.parker@uq.edu.au

TO THE EDITOR: Gerber¹ appears sympathetic to the following reasons for rejecting damages in cases of wrongful birth, quoted from the High Court minority in *Cattanach v Melchior*,² and judges in similar cases:

- doctors do not owe a duty of care that protects the economic interests of patients;
- the birth of a healthy child should not be regarded as a legal harm, because the birth of a healthy child is a good thing, the cost of rearing a child does not exceed the value of parenthood, and parents ought not to enjoy the advantages of parenthood without the concomitant responsibilities;
- awarding damages would indicate to the child that he or she was unwanted; and
- doctors should not be liable for most of the costs of rearing a child, because parents have a choice of rearing the child or surrendering it for adoption.

As to the first point, why should doctors be exempt from any loss that results from their negligence? Depicting this

duty as one of “protecting economic interests” distorts the nature of the duty of care, which requires the duty holder to avoid foreseeable damage, which includes economic loss consequent on the negligence.

Regarding the second point, balancing the costs of rearing the child against the value and advantages of parenthood is conceptually misleading. The implication is that the value of parenthood should render the costs of rearing the child relatively trivial. A more coherent interpretation is that, because the costs of rearing the child and the value of parenthood are incommensurable, the natural love for the child and the value of parenting that follow the birth ought not discount the damage.

This interpretation also informs responses to the third and fourth points. Not wanting another child, and not wanting the particular child once it exists, are distinct concepts. The idea that Jordan Melchior was not wanted at the time his parents took steps to avoid further pregnancies is incoherent. Wanting to avoid further pregnancies and wanting to nurture the child who is born are perfectly consistent positions, whether the case involves forgotten contraception or negligent sterilisation.

Finally, the idea that, because they have the choice to keep or surrender the child, the parents should bear the costs if they keep it suggests that, once born, the particular child can be regarded as a commodity — something that the general position against awarding damages for wrongful birth repeatedly disavows. One of the judges quoted by Gerber claimed that such children would come to think of themselves as unwanted, and that this was “obscene”. The same judge’s glib claim that the parents can choose to keep or surrender the child strikes me as the obscenity.

1. Gerber P. Failed sterilisations and the unwanted child: a new medicolegal minefield? *Med J Aust* 2004; 180: 123-125.
2. *Cattanach v Melchior* [2003] HCA 38 (16 July 2003). □

Paul Gerber

Honorary Reader in Legal Medicine, University of Queensland, Brisbane, QLD.

IN REPLY: Parker’s letter does little more than repeat the arguments that found favour with the majority in the High Court. He approaches the issues raised by this controversial litigation from an

ethical perspective. Alas, the parents’ claim for the cost of raising a healthy child, conceived as a result of the alleged negligence of the defendant gynaecologist, raises the *legal* issue of restitution: does the law of tort recognise this head of damages as a “loss” for which parents may be compensated? Courts have, in the past, answered this question by reference to general principles based upon *legal* values. In the *Melchior* case,¹ the majority departed from that hallowed principle. So be it.

Does that make Jordan Melchior a “commodity”, having a commercial value? Yes! The plaintiffs faced the choice of either keeping their son, or mitigating their “loss” by placing him for adoption. They chose the former. Priestley JA had, in an earlier case, put the issue succinctly: “After that decision was made, the defendant was not legally responsible for the parents’ financial cost of rearing the child”² (this was restated by Kirby J in the *Melchior* case¹).

Parker may not like it, but the law of tort has — up till now — shown more caution in awarding damages for what is called *pure* economic loss (ie, loss affecting purely financial interests) than it has in relation to conduct that causes damage to person or property. Before the *Melchior* case, that distinction had been firmly embedded in the law of tort and formed the basis of established rules governing liability for damages.³ That distinction has now been blurred by placing a financial value on the parent-child relationship.

1. *Cattanach v Melchior* [2003] HCA 38 (16 July 2003).
2. *CES v Superclinics (Australia) Pty Ltd* (1995) 38 NSWLR 47.
3. Felthusen BP. The recovery of pure economic loss. 4th ed. Toronto: Carswell Legal Publications, 2001: 10-11. □

Octreotide treatment for sulfonylurea-induced hypoglycaemia

Bronwyn AL Crawford,* Channa Perera†

* Endocrinologist and Clinical Senior Lecturer, Royal Prince Alfred Hospital and the University of Sydney, Sydney, NSW 2050; † Endocrinologist, Orange Base Hospital, Orange. brccrawfo@mail.usyd.edu.au

TO THE EDITOR: Prolonged hypoglycaemia in patients taking a sulfonylurea may be refractory to intravenous glucose treatment with fatal consequences, as described by Veitch and Clifton-Bligh.¹ Although these authors briefly mention

the use of octreotide, we believe that an additional point in the "Lessons from practice" should have been: *Octreotide may be an effective therapy in refractory sulfonylurea-induced hypoglycaemia.*

We describe the first two patients in whom we used this therapy.

A 76-year-old man with type 2 diabetes was admitted after an acute myocardial infarction and cardiac arrest. He was successfully resuscitated and underwent emergency bypass surgery. His diabetes was controlled with gliclazide 80 mg twice a day. After surgery, he developed cardiac failure, renal impairment (serum creatinine level, 0.22 mmol/L) and frequent hypoglycaemic episodes. The gliclazide was stopped but, despite good oral dietary intake, hypoglycaemia worsened and failed to respond to vigorous intravenous glucose therapy. He suffered a hypoglycaemic seizure (blood sugar level, 0.8 mmol/L). Over the next day, he received more than 300 g of glucose in the form of a 10% glucose intravenous infusion, but, despite this, went into hypoglycaemic coma. Blood results were: insulin, 472 pmol/L (reference range [RR], 15–60 pmol/L); C-peptide, 7616 pmol/L (RR, 300–800 pmol/L); and gliclazide, 9.4 mg/L (steady state average, 2.5 mg/L). He was given an intravenous infusion (30 ng/kg per minute) of octreotide.² Within an hour, his blood sugar level rose to 7.9 mmol/L and continued to rise. Dextrose and octreotide infusions were ceased within 13 hours, with no further episodes of hypoglycaemia. He was discharged home 2 days later.

A 75-year-old man with type 2 diabetes was taking glibenclamide 2.5 mg each morning. He was transferred from a rural hospital with acute on chronic renal failure (serum creatinine level, 0.4 mmol/L), as well as recurrent hypoglycaemia. The glibenclamide was stopped, but blood sugar levels remained low, and he became comatose despite boluses of 50% dextrose and a continuous infusion of 10% dextrose. The high volume of intravenous fluid precipitated cardiac failure and pulmonary oedema, requiring inotropic support. Blood results were: blood sugar, 1.5 mmol/L; insulin, 1250 pmol/L; C-peptide, 20 949 pmol/L. As an alternative to the high-dose, continuous infusion of octreotide used in our first patient, we administered a single subcutaneous injection of octreotide

50 µg. One hour later, the patient's blood sugar level had risen to 9.0 mmol/L. He had no further episodes of hypoglycaemia. Eight hours later, insulin and C-peptide levels had fallen markedly (insulin, 153 pmol/L; C-peptide, 5654 pmol/L). He made a full recovery.

Octreotide is a somatostatin analogue that inhibits the secretion of a number of neuropeptides, including insulin. It is a safe and effective therapy for sulfonylurea-induced hypoglycaemia when initial therapy with oral or intravenous glucose fails.²⁻⁵ It is particularly useful in elderly patients with renal or cardiac complications, in whom fluid overload may be a limiting factor in intravenous dextrose therapy. Octreotide may also break the vicious circle that can occur in sulfonylurea-induced hypoglycaemia in which repeated dextrose boluses further stimulate insulin release.

1. Veitch PC, Clifton-Bligh RJ. Long-acting sulfonylureas — long acting hypoglycaemia. *Med J Aust* 2004; 180: 84-85.
2. Boyle PJ, Justice K, Krentz AJ, et al. Octreotide reverses hyperinsulinemia and prevents hypoglycemia induced by sulfonylurea overdoses. *J Clin Endocrinol Metab* 1993; 76: 752-756.
3. Braatvedt GD. Octreotide for the treatment of sulfonylurea-induced hypoglycaemia in type 2 diabetes. *N Z Med J* 1997; 110: 189-190.
4. Graudins A, Linden CH, Fern RP. Diagnosis and treatment of sulfonylurea-induced hyperinsulinemia hypoglycemia. *Am J Emerg Med* 1997; 15: 95-96.
5. Krentz AJ, Boyle PJ, Justice KM, et al. Successful treatment of severe refractory sulfonylurea-induced hypoglycemia with octreotide. *Diabetes Care* 1993; 16: 184-186. □

Peter C Veitch,* Rory J Clifton-Bligh†

*Specialist in Geriatric Medicine, Department of Aged Care and Rehabilitation Medicine; †Registrar in Endocrinology, Department of Endocrinology, Royal North Shore Hospital, Clinic 1, Level 3, Pacific Highway, St Leonards, NSW 2065. rclifton@med.usyd.edu.au

IN REPLY: Crawford and Perera highlight a very important point with respect to hospital-based treatment of sulfonylurea-induced hypoglycaemia. Whereas, in our article,¹ we wished to emphasise the importance of recognising and preventing this condition in primary care settings, we agree that octreotide is an effective therapy in treating this condition. Octreotide inhibits the specific effect of sulfonylureas (glucose-stimulated β -cell insulin release) and prevents rebound hypoglycaemia, which, in this situation, may occur with the use of glucose.² Case reports, including those elegantly presented and referenced by Crawford and Perera, clearly illustrate the safety and efficacy of octreotide in treating sulfonylurea toxicity when initial

therapy with glucose fails. Octreotide may be administered either intravenously or subcutaneously, and its effect is maintained after a short course of therapy.

At our own institution, we have now adopted guidelines for the treatment of refractory sulfonylurea-induced hypoglycaemia, which include the administration of octreotide (50 µg subcutaneously) every 8 hours for up to three doses, although, as Crawford and Perera note, some patients will have a sustained response after a single dose.

1. Veitch PC, Clifton-Bligh RJ. Long-acting sulfonylureas — long acting hypoglycaemia. *Med J Aust* 2004; 180: 84-85.
2. McLaughlin SA, Crandall CS, McKinney PE. Octreotide: an antidote for sulfonylurea-induced hypoglycaemia. *Ann Emerg Med* 2000; 36: 133-138. □

Diagnosis and management of hyperthyroidism and hypothyroidism

Malvinder S Parmar

Medical Director (Internal Medicine), Timmins and District Hospital, Suite 108, 707 Ross Ave East, Timmins, ON P4N 8R1, Canada. atbeat@ntl.sympatico.ca

TO THE EDITOR: I wish to add another cause of exogenous hyperthyroidism to those mentioned in the comprehensive review on hyper- and hypothyroidism by Topliss and Eastman.¹ Inadvertent ingestion of animal thyroid ("hamburger" thyrotoxicosis), although rare, is worth mentioning. Meat may be inadvertently contaminated with thyroid tissue through the process of "gullet trimming" during butchering. While this process has been prohibited in most countries since the recognition of outbreaks of hamburger thyrotoxicosis,^{2,3} it may still occur when farm animals or wild game are prepared for consumption by farmers, hunters or local butchers unaware of the prohibition.

I recently reported a case of a woman living on a farm in Canada who had five episodes of transient hyperthyroidism over a decade.⁴ These were initially diagnosed as episodes of "silent thyroiditis", but were later attributed to consumption of meat patties contaminated with thyroid tissue, as the local butcher was not aware of the prohibition on gullet trimming. A history of eating wild game or locally prepared meat should be considered before a diagnosis of silent thyroiditis is made. Thyroid uptake of radioiodine is low in both conditions, but

serum thyroglobulin level is raised in thyroiditis and decreased during the hyperthyroid phase of exogenous hyperthyroidism.

1. Topliss DJ, Eastman CJ. Diagnosis and management of hyperthyroidism and hypothyroidism. *Med J Aust* 2004; 180: 186-193.
2. Hedberg CW, Fishbein DB, Janssen RS, et al. An outbreak of thyrotoxicosis caused by the consumption of bovine thyroid in ground beef. *N Engl J Med* 1987; 316: 993-998.
3. Kinney JS, Hurwitz ES, Fishbein DB, et al. Community outbreak of thyrotoxicosis: epidemiology, immunogenetic characteristics, and long-term outcome. *Am J Med* 1998; 84: 10-18.
4. Parmar MS, Sturge C. Recurrent hamburger thyrotoxicosis. *CMAJ* 2003; 169: 415-417. □

Ngairé T Jones

Medical Practitioner, Beaconsfield, WA.
ngairej@bigpond.com

TO THE EDITOR: The recent article on thyroid disorders by Topliss and Eastman notes that “around the world, iodine deficiency still remains the predominant cause of hypothyroidism” and furthermore that “mild iodine deficiency is re-emerging in Australia”.¹ Indeed, the Journal has recently published at least two articles suggesting that the iodine status of the Australian population needs to be further explored.^{2,3}

My question therefore is: when treating a patient who has results indicating clinical or subclinical hypothyroidism, would it be relevant and important to test for iodine deficiency (by 24-hour urine collection)? This seems analogous to undertaking iron studies in a patient with a falling haemoglobin level. In the same way that iron deficiency can exist and produce symptoms, even in the absence of anaemia, may not iodine deficiency affect health and well-being? Without the elemental “building blocks” of iron and iodine, the relevant systems are put into overdrive to no avail.

It seems simple to test routinely for this possibility, correct any deficiency and then recheck thyroid function. There may be more “clinically significant iodine deficiency” than we realise.

As it will no doubt be some time until further studies in the Australian population shed more light on this, is it not relevant meanwhile to at least check for this possibility in individual patients?

1. Topliss DJ, Eastman CJ. Diagnosis and management of hyperthyroidism and hypothyroidism. *Med J Aust* 2004; 180: 186-193.
2. McElduff A, McElduff P, Gunton JE, et al. Neonatal thyroid-stimulating hormone concentrations in northern Sydney: further indications of mild iodine deficiency? *Med J Aust* 2002; 176: 317-320.

3. McDonnell CM, Harris M, Zacharin MR. Iodine deficiency and goitre in schoolchildren in Melbourne, 2001. *Med J Aust* 2003; 178: 159-162. □

Duncan J Topliss,* Creswell J Eastman†

* Director, Endocrinology and Diabetes, Alfred Hospital, Commercial Road, Melbourne, VIC 3004;
† Director, Institute of Clinical Pathology and Medical Research, Westmead Hospital, Sydney, NSW.
Duncan.topliss@med.monash.edu.au

IN REPLY: Jones asks if iodine excretion should be measured routinely in all patients with hypothyroidism in Australia as part of the initial assessment. We do not advocate this for the following reasons.

Urinary iodine estimations are unreliable for assessing individual patients, as urinary iodine levels can vary considerably from day to day with iodine intake. These measurements should be reserved for population studies to provide an overall assessment of iodine nutrition in that population.

In Australia, it is probable that virtually all cases of primary hypothyroidism are caused by chronic autoimmune lymphocytic thyroiditis, ablative therapy for Graves' disease, or inadequate thyroxine replacement therapy in these conditions, as is the case in the United States and the United Kingdom.¹⁻³ This contention is supported by data from the Busselton (Western Australia) survey on thyroid peroxidase antibody levels,⁴ which suggest that these antibodies will be of great diagnostic assistance, in contrast to the dubious clinical value of individual measurement of iodine excretion. In support of this view, in a large survey of the US population, where iodine intake has probably fallen similarly but not to the same degree as in Australia, there was no association between low urinary iodine levels and increased serum levels of thyroid stimulating hormone (TSH).⁵ In that study, the significant association between raised TSH and female sex disappeared after controlling for the presence of thyroid peroxidase antibodies, while the prevalence of clinical hypothyroidism was strongly correlated with positive results for these antibodies.

In Australia, current information indicates that iodine deficiency, where it exists, is mild. The prevalence and regional variation are currently the subject of the National Iodine Nutrition Survey, which is surveying iodine excretion and thyroid size in primary school children. There is no evidence that this

mild deficiency is associated with an increased prevalence of hypothyroidism. Jones's suggestion that iodine deficiency impairs health by a mechanism other than impairment of thyroid function is not supported by any scientific evidence.

Any consideration of advocating routine assessment of iodine status should await the results of the ongoing national study. However, iodine nutrition would appear to be best addressed as a public health issue, by promoting use of iodised salt and ensuring adequate iodine nutrition in pregnant and breastfeeding women and their infants.

1. Hamburger JI. Factitious elevation of thyrotropin in euthyroid patients. *N Engl J Med* 1985; 313: 267-268.
2. Tunbridge WM, Evered DC, Hall R, et al. The spectrum of thyroid disease in a community: the Whickham survey. *Clin Endocrinol* 1977; 7: 481-493.
3. Sawin CT, Geller A, Hershman JM, et al. The aging thyroid. The use of thyroid hormone in older persons. *JAMA* 1989; 261: 2653-2656.
4. Hawkins BR, Cheah PS, Dawkins RL, et al. Diagnostic significance of thyroid microsomal antibodies in randomly selected population. *Lancet* 1980; 2: 1057-1059.
5. Hollowell JG, Staehling NW, Flanders WD, et al. Serum TSH, T(4), and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). *J Clin Endocrinol Metab* 2002; 87: 489-499. □

Management of chronic low back pain

David S Elder

Occupational Physician, 517 St Kilda Road, Melbourne, VIC 3004. delder@bigpond.net.au

TO THE EDITOR: In Bogduk's review of the management of low back pain,¹ he cited several international guidelines but did not address the effect of returning the patient to work. Disappointingly, return to work was mentioned only as an outcome of multidisciplinary therapy, with no mention at all of a planned and purposeful return to work in the suggested approach. This is surprising, given the literature available^{2,3} and the significant adverse effects of being out of work.⁴

Further, the algorithm in Box 3 (general practice management of chronic low back pain) appears to have a never-ending loop: I am cautious of the adverse effects that the reductionist model can have,⁵ and it appears possible in this algorithm to be forever stuck in the investigative loop. An additional pathway from this loop to intensive therapy would allow progression in some cases.

The inclusion of a return to work in management of low back pain has been extensively analysed in the Australian setting and shown to significantly reduce disability.⁶ This advice should be included in any clinical update on management of low back pain.

1. Bogduk N. Management of chronic low back pain. *Med J Aust* 2004; 180: 79-83.
2. Victorian WorkCover Authority. Guidelines for the management of employees with compensable low back pain. Melbourne: Victorian Workcover Authority, 1996.
3. Waddell G. The back pain revolution. Edinburgh: Churchill Livingstone, 1998.
4. Gerdtham UG, Johannesson M. A note on the effect of unemployment on mortality. *J Health Econ* 2003; 22: 505-518.
5. Ober KP. Uncle Remus and the cascade effect in clinical medicine. Brer Rabbit kicks the tar-baby. *Am J Med* 1987; 82: 1009-1013.
6. Buchbinder R, Jolley D, Wyatt M. 2001 Volvo award winner in clinical studies. Effects of a media campaign on back pain beliefs and its potential influence on management of low back pain in general practice. *Spine* 2001; 26: 2535-2542. □

John Salmon,* Anna Hilyard†

*Pain Management Specialist, Bethesda Hospital, 25 Queen Mary Drive, Claremont, WA 6010; †Director, Achieve Pain Control Group, Perth, WA. salmon8@bigpond.com

TO THE EDITOR: Bogduk's article on management of chronic low back pain¹ was disappointingly retrogressive as a guide for general practitioners. Compartmentalising back pain management as monotherapy, multidisciplinary therapy or "reductionism", and favouring the last, reinforces the medical model which has singularly failed to stem the epidemic of low back pain disability affecting the developed world.

The biopsychosocial model of chronic spinal pain is now widely accepted and rationally emphasises the multi- or interdisciplinary model of management.^{2,3} Bogduk's preference for the reductionist approach may be reasonable in a specialised centre and as a basis for research, but must justify its practical relevance in the face of the following:

- Available data on the reductionism approach are meagre, conflicting and mainly derived from pain-clinic populations likely to differ from patients presenting to GPs.
- Diagnostic joint and disc injection procedures and radiofrequency treatment performed to the required standard are available in only a very few centres.
- Radiofrequency lesioning of the nerve supply to symptomatic joints has been shown to provide pain relief limited to 9–18 months.⁴ Repeat lesioning may be less

effective and is impracticable in the long term.

At best, these treatments could be considered palliative.

Of course, patients can only benefit from accurate diagnosis and reduction of pain from identified peripheral generators. Unfortunately, for most people with chronic back pain, it is not that simple. Usually there are multiple pathologies and pain generators, multisegmental dysfunction, disrupted motor control and interacting peripheral and central neural sensitisation mechanisms. And that is just the "bio" of the biopsychosocial model. There is then the interplay with the individual's psychological and social environment.

It is often a challenge to communicate the diagnosis effectively in the face of conflicting input from other health providers, the media and patient preconceptions. Just "plonking" "the diagnosis" before a patient and dangling a seductive "techno fix" that does not deliver in the long term is precisely what renders patients with chronic pain increasingly bewildered, dysfunctional and desperate to try one passive treatment after another.

The biopsychosocial model provides a basis for management in both general and specialist practice. Appropriate interventions to reduce pain-generator input are embedded in a cognitive behavioural management matrix that imbues patients with accurate, relevant knowledge of their conditions and common-sense self-management techniques to maintain appropriate activity levels, goal setting and psychological positivity.

For a time-challenged GP, collaboration with an activation- and exercise-oriented physiotherapist can be effective. The GP's role is to provide the "white coat authority" so vital in recruiting patient confidence.

1. Bogduk N. Management of chronic low back pain. *Med J Aust* 2004; 180: 79-83.
2. Waddell G, Burton K. Evidence review. In: Carter JT, Birrell LN, editors. Occupational health guidelines for the management of low back pain at work — principal recommendations. London: Faculty of Occupational Medicine, 2000. Available at: www.facocmed.ac.uk (accessed Feb 2004).
3. Molloy AR, Blythe FN, Nicholas MK. Disability and work-related injury: time for a change? *Med J Aust* 1999; 170: 150-151.
4. Lord SM, Barnsley L, Wallis B, et al. Percutaneous RF neurotomy for chronic cervical zygapophyseal joint pain. *N Engl J Med* 1996; 335: 1721-1726. □

Nikolai Bogduk

Director, Department of Clinical Research, Royal Newcastle Hospital, Newcastle, NSW 2300. mgillam@mail.newcastle.edu.au

IN REPLY: There is a difference between wishful thinking and evidence. Elder advocates a focus on return to work. Elsewhere, I have described how this should be pursued.¹ However, the evidence supports success only in the context of acute and subacute pain. I was commissioned to write on low back pain. In that context, evidence is lacking. Even Waddell, whom Elder cites,² conspicuously avoided the issue of chronic low back pain; his evidence pertains only to acute low back pain.

Salmon and Hilyard promote the biopsychosocial model. Indeed, this model is now widely accepted. Even our own studies have shown how successful it can be to recognise and treat patients' fears and mistaken beliefs.³ However, the evidence of success is limited to acute and subacute low back pain. The predictions of the biopsychosocial model have not been fulfilled in the context of chronic low back pain. Although better than no therapy, behavioural therapy is not more effective than other therapies, and does not "reduce pain generator input", as Salmon and Hilyard contend. Insurers, who pay for this treatment, do not share their enthusiasm for it.⁴

Salmon and Hilyard also repeat the commonly held view that patients have multiple pain generators. There is no actual evidence for this assertion, while the available evidence indicates the opposite. When investigated comprehensively, fewer than 10% of patients have more than one simultaneous pain generator.⁵

Further, Salmon and Hilyard consider that complete relief of pain for 9–18 months amounts to palliative therapy. Yet the opposite is true. Not relieving pain by behavioural therapy is palliative. They also deprecate radiofrequency neurotomy with the accusation that it "may be less effective" when repeated, but fail to cite the literature showing that this is not the case.

They are correct in stating that reductionist procedures performed to the required standard are available in only a few centres. However, this does not invalidate these procedures; it reflects only a political and ideological problem in healthcare delivery. They also fail to reveal that in many places where these

procedures are available, they are not performed according to best-practice standards. It is not the procedures, but misguided and unscrupulous practitioners, who render patients bewildered and dysfunctional.

1. Bogduk N, McGuirk B. Medical management of acute and chronic low back pain. An evidence-based approach. Amsterdam: Elsevier, 2002.
2. Waddell G. The back pain revolution. Edinburgh: Churchill Livingstone, 1998.
3. McGuirk B, King W, Govind J, et al. The safety, efficacy, and cost-effectiveness of evidence-based guidelines for the management of acute low back pain in primary care. *Spine* 2001; 26: 2615-2622.
4. Mastroianni T. Personal reflections. Australian Pain Society newsletter, Feb 2004: 6-7.
5. Schwarzer AC, Aprill CN, Derby R, et al. The relative contributions of the disc and zygapophyseal joint in chronic low back pain. *Spine* 1994; 19: 801-806. □

Risk-taking behaviour of young women in Australia: screening for health-risk behaviours

Gordon Broderick

Executive Director, Distilled Spirits Industry Council of Australia, 1st Floor, 117 Ferrars Street, South Melbourne, VIC 3205. gordonb@dasic.com.au

TO THE EDITOR: In their article on risk-taking behaviour among young Australian women, Carr-Gregg and colleagues make a number of statements about alcohol consumption among young women.¹ Unfortunately, these statements are not supported by the facts.

The authors assert, citing a national study of 14762 women aged 18–23 years,² that “seventy percent of young women engage in ‘binge drinking’ (5 or more drinks on one occasion) at some time, with 19% doing so on a weekly basis”. The level and frequency of alcohol consumption that constitutes “binge drinking” is a matter of conjecture. The National Health and Medical Research Council (NHMRC), in guidelines released in 2001,³ state that “binge drinking” is “not a preferred term due to its lack of consistent and specific meaning”.

The NHMRC guidelines on short term risk specify 5–6 alcoholic drinks for a female on any one day as being “risky” for health, and 7 drinks or more being “high risk”. For long term risk, 3–4 drinks on an average day, or 15–28 drinks a week, is considered “risky”, with any more constituting “high risk”.

Applying these guidelines to the Women’s Health Australia dataset shows that 5.1% of young women engage in drinking that is “risky” or “high risk” in

the long term. Of the remaining 94.9%, 14.4% drink 5 or more drinks weekly or more, and 51.9% drink five or more drinks monthly or less. This is a more revealing (and accurate) picture than the blanket statement that “70% of young women are ‘binge drinkers’”.

Carr-Gregg and colleagues also claim that “22% of females aged 14–19 years drink between 9 and 30 alcoholic drinks a day”. The source for this statement is a survey conducted for the Salvation Army.⁴ The survey has several limitations, not least the small sample size. The survey sampled 614 respondents, of whom 70 were aged 14–19 years. The assertion that 22% of females in this age category were “binge drinkers” is based on just seven respondents. This number is well below what is required for any reliable statistical estimation.

Encouraging responsible drinking among younger people is a major goal of health professionals and the alcohol industry. A constructive policy debate on this issue requires sound, objective evidence about alcohol consumption among younger people. The article by Carr-Gregg et al does not represent progress towards providing that evidence.

1. Carr-Gregg MRC, Enderby KC, Grover SR. Risk-taking behaviour of young women in Australia: screening for health-risk behaviours. *Med J Aust* 2003; 178: 601-604.
2. Jones HA, Dobson AJ, Brown WJ. Patterns of alcohol consumption in young Australian women: associations with sociodemographic factors, lifestyle, health practices and physical health. *Aust N Z J Public Health* 2002; 26: 156-163.
3. National Health and Medical Research Council. Australian alcohol guidelines. Canberra: NHMRC, 2001. Available at: www.nhmrc.gov.au/publications/synopses/ds9syn.htm (accessed Jul 2003).
4. Salvation Army. Alcohol awareness study. www.salvation-army.org.au/media/2002/020903_alcohol.asp (accessed Jul 2003). □

MJA Advertisers' Index

Corinth Healthcare

Medical recruitment p496

Mayne Pharma

Androderm p491

Medical Defence Association

Medical indemnity p503

Novartis

Optifast VLCD p497

Roche

Dilatrend p536

Schering Pty Ltd

Androcur-100 Inside front cover

Servier Laboratories

Coversyl Outside back cover

The Medical Journal of Australia

Editor

Martin Van Der Weyden, MD, FRACP, FRCPA

Deputy Editors

Bronwyn Gaut, MBBS, DCH, DA

Ruth Armstrong, BMed

Mabel Chew, MBBS(Hons), FRACGP, FACHPM

Ann Gregory, MBBS, GradCertPopHealth

Manager, Communications Development

Craig Bingham, BA(Hons), DipEd

Senior Assistant Editor

Helen Randall, BSc, DipOT

Assistant Editors

Elsina Meyer, BSc

Kerrie Lawson, BSc(Hons), PhD, MASM

Tim Badgery-Parker, BSc(Hons)

Josephine Wall, BA, BAppSci, GradDipLib

Proof Readers

Christine Binskin, BSc

Richard Bellamy

Editorial Administrator

Kerrie Harding

Editorial Assistant

Christine Tsim

Production Manager

Glenn Carter

Editorial Production Assistant

Melissa Sherman, BA

Librarian, Book Review Editor

Joanne Elliot, BA, GradDipLib

Consultant Biostatistician

Val GebSKI, BA, MStat

Content Review Committee: Leon Bach, PhD,

FRACP; Adrian Bauman, PhD, FAFPHM; Flavia

Cicutinni, PhD, FRACP; Marie-Louise Dick, MPH,

FRACGP; Mark Harris, MD, FRACGP;

David Isaacs, MD, FRACP; Paul Johnson, PhD,

FRACP; Jenepher Martin, MEd, FRACS;

Adrian Mindel, MD, FRACP; Michael Solomon,

MSc, FRACS; Campbell Thompson, MD, FRACP;

Tim Usherwood, MD, FRACGP; Owen Williamson,

FRACS, GradDipClinEpi; John Wilson, PhD,

FRACP; Jeffrey Zajac, PhD, FRACP

Australasian Medical Publishing Co Pty Ltd

Advertising Manager: Peter Butterfield

Media Coordinators: Julie Chappell, Stephanie Elliott

The Medical Journal of Australia (MJA) is published on the 1st and 3rd Monday of each month by the Australasian Medical Publishing Company Proprietary Limited, Level 2, 26-32 Pyrmont Bridge Rd, Pyrmont, NSW 2009. ABN 20 000 005 854. Telephone: (02) 9562 6666. Fax: (02) 9562 6699. E-mail: ampco@ampco.com.au. The Journal is printed by Offset Alpine Printing Ltd, 42 Boorea St, Lidcombe, NSW 2141.

MJA on the Internet: <http://www.mja.com.au/>

None of the Australasian Medical Publishing Company Proprietary Limited, ABN 20 000 005 854, the Australian Medical Association Limited, or any of its servants and agents will have any liability in any way arising from information or advice that is contained in *The Medical Journal of Australia (MJA)*. The statements or opinions that are expressed in the Journal reflect the views of the authors and do not represent the official policy of the Australian Medical Association unless this is so stated. Although all accepted advertising material is expected to conform to ethical and legal standards, such acceptance does not imply endorsement by the Journal. All literary matter in the Journal is covered by copyright, and must not be reproduced, stored in a retrieval system, or transmitted in any form by electronic or mechanical means, photocopying, or recording, without written permission.

Published in 2 volumes per year.

Annual Subscription Rates for 2004 (Payable in Advance) to:

AMPCo, Locked Bag 3030, Strawberry Hills, NSW 2012

Individual Subscriptions (includes 10% GST)

Australia—\$A291.50, Medical students (Australia only)—\$A60.00

Overseas Economy Air—\$A370.00, Airmail—\$A505.00

NZ & PNG Economy Air—\$A340.00

Indexes are published every 6 months and are available on

request as part of the current subscription.

Single or back issues contact: AMPCo (02) 9562 6666.

Advice to Authors—

<http://www.mja.com.au/public/information/instruct.html>



27,721 circulation as at
31 March, 2004

ISSN 0025-729X