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Pethidine in emergency departments: promoting evidence-based prescribing

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TO THE EDITOR: We congratulate Kaye and colleagues on their efforts to educate and influence prescribing practice in reducing the use of pethidine.¹ The adverse effects of pethidine and its lack of efficacy over other opiates have been known and taught since the early 1990s.² A decade on, we are still seeing significant use of this drug,³ which has multiple disadvantages when compared with other opioid analgesics.

The difficulty lies in doctors' attitudes to quality improvement and change in health care. It has been noted that doctors' responses to concern about the quality of health care range widely, from opposition to whole-heartedly embracing legitimate opportunities for improvement.⁴ While there is such variance, the implementation of evidence-based medicine into practice will lag, sometimes by decades, resulting in unnecessary adverse effects in patients.

With clinical guidelines in place, a rigorous education campaign and many hours of research time and resources, Kaye and colleagues have significantly reduced, but not eradicated, pethidine prescribing in New South Wales. In comparison, O'Connor et al report combining a similar educational program with formulary restrictions to effectively eliminate the use of meperidine (pethidine)

in their single centre study.⁵ We can only conclude that clinical evidence, even when combined with quality improvement campaigns, remains less effective than policy changes which restrict doctors' behaviour.

From available evidence, the liberal use of pethidine may cause adverse effects which are preventable by a simple system-oriented approach — in this case, the appropriate risk-management step is restricting pethidine use to very limited situations. We cannot continue to justify use of a drug with poor efficacy, toxicity and serious drug interactions.

- 1 Kaye KI, Welch SA, Graudins LV, et al. Pethidine in emergency departments: promoting evidence-based prescribing. *Med J Aust* 2005; 183: 129-133.
- 2 Clark RF, Wei EM, Anderson PO. Meperidine: therapeutic use and toxicity. *J Emerg Med* 1995; 13: 797-802.
- 3 Seifert CF, Kennedy S. Meperidine is alive and well in the new millennium: evaluation of meperidine usage patterns and frequency of adverse drug reactions. *Pharmacotherapy* 2004; 24: 776-783.
- 4 Blumethal D. Quality of health care. Part 4: the origins of the quality-of-care debate. *N Engl J Med* 1996; 335: 1146-1149.
- 5 O'Connor AB, Lang VJ, Quill TE. Eliminating analgesic meperidine use with a supported formulary restriction. *Am J Med* 2005; 118: 885-889. □

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TO THE EDITOR: I was interested to read the article by Kaye and colleagues about reducing pethidine use in the emergency department by means of evidence-based prescribing.¹

In view of published reports describing the disadvantages of pethidine, it was decided in late 1996 to attempt to reduce the amount of pethidine prescribed in the emergency department of Launceston General Hospital. Narcotics were supplied as ampoules of

100 mg pethidine, 10 mg morphine, 15 mg papaveretum (the use of which was trivial) and fentanyl, which was used mainly for anaesthetic induction. Before mid-1996, 50%–72% of all ampoules of narcotics used in the emergency department were of pethidine. Of narcotics used for acute pain management, pethidine would have been much higher as a proportion because it was not used for acute pulmonary oedema, ischaemic myocardial pain, anaesthetic induction and in patients being ventilated. The use of narcotics was monitored by quarterly reports from the pharmacy department of the quantities of the various parenteral narcotics supplied to the emergency department. Papaveretum was removed from the pharmacopoeia in 1999.

In 1996, an informal education program was instituted within the emergency department, strongly supported by the nurses, with the aim of convincing junior medical staff on rotation from other areas within the hospital to prescribe morphine rather than pethidine. It had long been observed that such staff prescribed pethidine almost exclusively for acute pain management, and a cultural change was required. As shown in the Box, the percentage of narcotics dispensed as pethidine was steadily reduced over the following years, reaching 5% in 2002. After 2 years at this level it was decided to remove pethidine from the pharmacopoeia. In February of 2005, hydromorphone was introduced and pethidine removed. There have been no complaints or problems as a result, and the whole process was unexpectedly painless and successful.

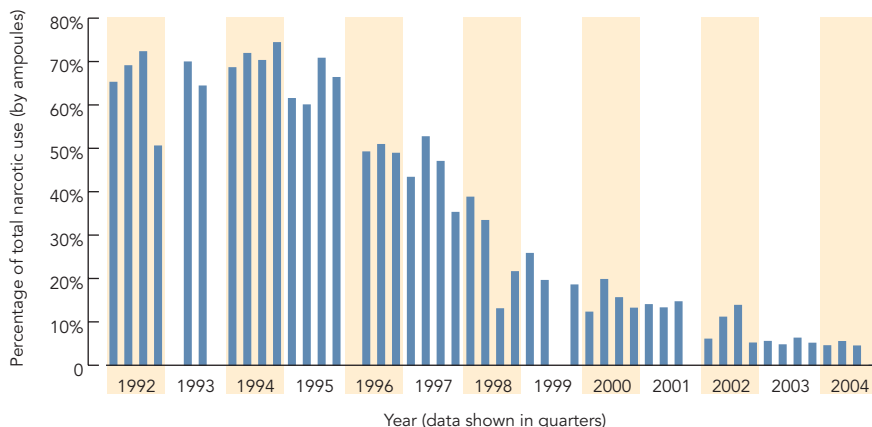
- 1 Kaye KI, Welch SA, Graudins LV, et al. Pethidine in emergency departments: promoting evidence-based prescribing. *Med J Aust* 2005; 183: 129-133. □

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IN REPLY: As Mitra and Cameron point out, policy change can be effective in influencing prescribing practice. Indeed, use of a restric-

Pethidine prescribing as a percentage of total narcotics in Launceston General Hospital emergency department, 1992–2004



Note: data not available for some quarters. ◆

tive formulary is a strategy used by drug and therapeutics committees in most Australian hospitals. However, where support among clinicians for policy change is lacking, significant time and effort is required by those responsible for policy implementation. Confrontation and lack of interdisciplinary cooperation can be expected.

Quality use of medicines (QUM) means selecting management options wisely, choosing suitable medicines if a medicine is considered necessary, and using medicines safely and effectively. Australia is fortunate in having a National Medicines Policy¹ and a national strategy for QUM.² This strategy recognises the importance of active and respectful partnerships, and of consultative, collaborative, multidisciplinary activity to improve the quality use of medicines.

To attain QUM, "... key partners must be involved at all stages in designing, implementing and evaluating QUM programs... Multiple activities and strategies are needed to raise awareness about issues related to QUM. Attitudes, knowledge, skills and behaviours that support QUM need to be developed and maintained".²

Our approach was based on these principles. Pielage provides another example of the successful use of this approach to limit pethidine prescribing in a large teaching hospital, which should be applauded.

Doctors, pharmacists, nurses and consumers are important partners in QUM. An educative, multidisciplinary approach that respects each partner is the most appropriate way to ensure sustained practice change and promote QUM in hospitals and the wider community.

1 The National Medicines Policy Document. National Medicines Policy 2000. Canberra: Australian Government Department of Health and Ageing, 2000. Available at: <http://www.health.gov.au/internet/wcms/publishing.nsf/Content/nmp-objectives-policy.htm> (accessed Nov 2005).

2 Quality Use of Medicines (QUM) strategy. Canberra: Australian Government Department of Health and Ageing, 2000. Available at: <http://www.health.gov.au/internet/wcms/publishing.nsf/Content/nmp-pdf-natstrateng-cnt.htm> (accessed Nov 2005). □

Hospitalisation and costs attributable to tobacco smoking in Australia: 2001–2002

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TO THE EDITOR: Previous analyses of costs to the Pharmaceutical Benefits Scheme and costs for stroke and acute myocardial infarction hospitalisations suggest that tobacco control programs are a good investment.^{1,2} To further highlight the economic benefits of reducing smoking rates, I estimated the hospitalisation costs attributable to cigarette smoking in Australia for 2001–2002 by applying aetiological fractions to hospitalisation data.³

Aetiological fractions were calculated using 2001 National Health Survey smoking prevalence data and relative risks of hospitalisation for current and former smokers, as previously calculated by English and colleagues through linkage of data from the Busselton health survey and the Western Australian Hospital Morbidity Data system.⁴ Counts of separations (hospitalisations), bed-days, and average costs for hospitalisations in Australia in 2001–2002, by sex and 5-year age category, were obtained from the Australian Institute of Health and Welfare. They had been sourced from the National Hospital Morbidity Database (<http://www.aihw.gov.au/hospitaldata/morbidity.html>) and the National Hospital Cost Data Collection (<http://www.health.gov.au/casemix>), linked by the common variable "DRG4.2".

The results (Box) show that, in 2001–2002, almost 300 000 hospitalisations, costing \$682 million, were attributable to cigarette smoking.

English and colleagues estimated previously that, in 1992, 129 000 hospital separations and over 1.1 million bed-days were

attributable to cigarette smoking.⁴ Although the proportion of the Australian population who are smokers has decreased since then, from 26% to 23% in 2001,⁵ cigarette smoking is still associated with substantial health care utilisation and costs.

The actual costs are even greater than the \$682 million per annum estimated by my analysis, as the following were not considered: hospitalisation costs for those aged 80 years and over; pharmaceutical costs (estimated at \$126 million for cardiovascular drugs on the Pharmaceutical Benefits Scheme¹); community care costs (such as general practitioner visits); and patient contributions to hospitalisation costs.

In stark contrast to the \$682 million spent on hospitalisations attributable to smoking, the Australian Government has committed an average of only \$2 million per year over the last 10 years to tobacco harm minimisation programs.⁶

Acknowledgements: This research was supported with funding from the Victorian Health Promotion Foundation and The Cancer Council Victoria. These funding bodies, per se, had no role in the design, conduct or interpretation of the study. I am a consultant to The Cancer Council Victoria. Dallas English provided advice on the methodology used by English et al.⁴

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2 Hurley SF. Short-term impact of smoking cessation on myocardial infarction and stroke hospitalisations and costs in Australia. *Med J Aust* 2005; 183: 13–17.

3 English DR, Holman CD, Milne E, et al. The quantification of drug caused morbidity and mortality in Australia 1995. Canberra: Department of Human Services and Health, 1995.

4 English DR, Vu HTV, Knuiman MW. The impact of smoking on use of hospital services: the Busselton study. *Aust N Z J Public Health* 2002; 26: 225–230.

5 White V, Hill D, Siahpush M, Bobevski I. How has the prevalence of cigarette smoking changed among Australian adults? Trends in smoking prevalence between 1980 and 2001. *Tobacco Control* 2003; 12 (Suppl II): ii67–ii74.

6 VicHealth Centre for Tobacco Control. Tobacco control: a blue chip investment in public health. Melbourne: VicHealth Centre for Tobacco Control, 2003. Available at: http://www.vtcc.org.au/publ/reports/TC_S1April2003.pdf (accessed Aug 2005). □

Hospitalisations, bed-days and costs attributable to cigarette smoking in Australia in 2001–2002*

	Hospitalisations		Bed-days		Costs [†]	
	Proportion	Number	Proportion	Number	Proportion	\$(millions)
Men	7.6%	138 000	14.6%	891 000	7.6%	\$339
Women	8.6%	153 000	9.8%	581 000	8.6%	\$342
Total	8.1%	291 000	12.2%	1 472 000	8.1%	\$682

*For people aged 40–79 years.

† Estimated costs are conservative, as they are based on average cost per hospitalisation for the total population. However, the higher proportion of bed-days than hospitalisations attributable to smoking suggests smokers tend to have longer stays and thus higher than average costs. ◆

Severe renal failure and nephrocalcinosis in anorexia nervosa

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TO THE EDITOR: The recent article by Roberts et al¹ requires further comment. In anorexia nervosa, hypercalcaemia is extremely unusual because patients are likely to be under- or malnourished, with consequent hypocalcaemia and hypovitaminosis D, rather than the opposite, as implied by the authors. In one of their references,² hypercalcaemia is only briefly included, and the mechanism is not discussed or substantiated. Another of their references³ does not include hypercalcaemia at all as a metabolic disturbance. In addition, hypercalcaemia in itself is not a diagnosis, and indicates a significant underlying pathophysiological disturbance whose differential diagnoses need to be carefully dissected.

Where there are problems with the interpretation of calcium homeostasis, such as renal impairment and hypoalbuminaemia, ionised calcium should be measured because it is the physiologically active agent. In the first patient described by Roberts et al, although the investigations are incomplete, primary hyperparathyroidism (PHPT) needs to be carefully considered, as the parathyroid hormone (PTH) level is *not* normal in the setting of hypercalcaemia. The normal physiological response would dictate that the PTH level should be low to suppressed. Nephrocalcinosis and renal impairment then fit snugly into the diagnosis of PHPT.⁴ Phosphate level, expected to be low in this condition, might have been masked by exogenous phosphate supplement.

For Patient 2, stool electrolyte analysis could further support the presence of laxative misuse and aid in the interpretation of urinary results. Faecal fluid in this situation should be high in sodium, potassium and calcium concentrations. The low urinary sodium and calcium levels are therefore appropriate, and indicate relatively intact tubular function. The diagnosis of hypercalcaemia is thus difficult without a PTH measurement and in the presence of gastrointestinal confounders, even if all investigations were available. Nevertheless,

familial benign hypocalciuric hypercalcaemia (FBHH) is a strong probability given the low urinary calcium excretion, especially before iatrogenic manipulation of calcium and phosphate homeostasis. Nephrocalcinosis can theoretically occur in FBHH.⁵ It is important that the diagnosis is made in both cases, so that a familial study can be carried out if indicated given the patients' age. One may also wonder if the intermittent hypercalcaemia contributed to or aggravated the psychiatric disturbance in both patients.

- 1 Roberts MA, Thorpe CR, MacGregor DP, et al. Severe renal failure and nephrocalcinosis in anorexia nervosa. *Med J Aust* 2005; 182: 635-636.
- 2 Comerci GD. Medical complications of anorexia nervosa and bulimia nervosa. *Med Clin North Am* 1990; 74: 1293-1310.
- 3 Becker AE, Grinspoon SK, Kilbanski A, Herzog DB. Eating disorders. *N Engl J Med* 1999; 340: 1092-1098.
- 4 AAACE/AAES Task Force on Primary Hyperparathyroidism. The American Association of Clinical Endocrinologists and the American Association of Endocrine Surgeons position statement on the diagnosis and management of primary hyperparathyroidism. *Endocr Pract* 2005; 11: 49-54.
- 5 Sayer JA, Carr G, Simmons NL. Nephrocalcinosis: molecular insight into calcium precipitation within the kidney. *Clin Sci (Lond)* 2004; 106: 549-561. □

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IN REPLY: Disturbances of calcium metabolism in anorexia nervosa are complex, particularly with associated renal insufficiency. Although hypocalcaemia is observed in patients with anorexia nervosa, our article attempted to highlight nephrocalcinosis and hypercalcaemia.

We agree that hypercalcaemia is not in itself a diagnosis, and identifying the underlying pathology is essential. Ingestion of vitamin D preparation remains a likely explanation for the hypercalcaemia observed in Patient 1; however, primary hyperparathyroidism was considered as a possible differential diagnosis. Patient 1 had two normal parathyroid hormone tests in the setting of hypercalcaemia and renal impairment. This is consistent with secondary hyperparathyroidism and vitamin D ingestion as documented. The coexistence of primary hyperparathyroidism can-

not be excluded. Ionised calcium may be a useful measure if the patient had hypoalbuminaemia, and this would be our normal practice.

Patient 2 had ionised calcium measured twice (one result high, one low), but these added little to the case description and message of the article. Faecal electrolytes were not measured in Patient 2. However, we acknowledge the potential utility of this investigation when interpreting electrolyte disorders. We also agree that familial or genetic conditions should be considered if clinically appropriate. □

The aromatase inhibitors in early breast cancer: who, when and why?

Alan Rodger

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TO THE EDITOR: I note with concern that one of the authors of the article on aromatase inhibitors in early breast cancer (Nordman)¹ declares under competing interests that she received an honorarium (in the form of financial support to attend a conference) "for writing this article" from a manufacturer of one of the current licensed aromatase inhibitors.

It is reassuring to be informed that the company concerned had no role in the content of the article. However, is it wise and reasonable and, indeed, necessary for a medical oncology registrar to be financially rewarded by a pharmaceutical company for writing an article published in *The Medical Journal of Australia* about therapeutic products, one of which was developed and is now marketed by that company?

Two other authors and reviewers and editorial staff were involved in this otherwise excellent article. I commend Nordman for her openness and declaration. However, I question the role of the pharmaceutical industry in rewarding medical authors in this way.

Competing interests: Since 1992, I have received three "travel grants" from pharmaceutical companies — two for international meetings and one for a product launch. One company was involved with an aromatase inhibitor.

1 Nordman IC, Spillane AJ, Hamilton AL. The aromatase inhibitors in early breast cancer: who, when and why? *Med J Aust* 2005; 183: 24-27. □

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IN REPLY: In response to Rodger, AstraZeneca would like to reiterate that the company had no role in the content of the article published by Nordman and colleagues in the 4 July issue of *The Medical Journal of Australia*.¹

AstraZeneca has provided Sydney Cancer Centre with an unrestricted educational grant. Along with Rodger, we commend the authors on their transparency about how this grant was applied.

1 Nordman IC, Spillane AJ, Hamilton AL. The aromatase inhibitors in early breast cancer: who, when and why? *Med J Aust* 2005; 183: 24-27. □

Vitamin D and chronic mental illness

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TO THE EDITOR: It is well known that people with serious mental health problems are more likely to suffer substantial physical health problems, or die younger, than those in the general population.^{1,2} We would like to report some early results from a program that is aiming to improve primary health care for people with serious mental health problems.

The Stewart Lodge program was developed through cooperation between the local Moreland Community Health Service, two general practitioners and one registered nurse from the medical clinic collocated with the Community Health Service, the local area mental health service, and the managers of the Victorian Government Supported Residential Services program. The Stewart Lodge program includes a regular non-appointment doctor's session, complete health assessments for all residents, and regular case conferences involving all carers and clinicians. Initial establishment was funded through a Vic-

torian Government Department of Human Services GPs in Community Health Services strategy grant, which focused on improving integration and service coordination. There are around 85 people living in this community, most of whom have chronic schizophrenia or another serious mental health problem.

We plan to report the findings from our program in more detail when we have completed the assessments of most Stewart Lodge residents. However, we would like to report our interim findings on vitamin D levels, which are likely to be relevant to many others in similar circumstances. Of the 30 residents tested so far, three have had vitamin D levels in the normal range (> 50 nmol/L), 20 in the deficient range, (25–50 nmol/L) and seven in the severely depleted range (< 25 nmol/L).

An increased risk of low vitamin D levels has been previously reported in populations of older institutionalised people,³ and a recent position statement in the Journal on accepted levels of 25-hydroxyvitamin D (25-OHD) warned of risks of vitamin D deficiency for various groups in the community.⁴

We suggest that people with serious mental illness are another group that should be included in those at risk. The people we work with are at risk because of decreased exposure to the sun through inactivity, and because of their illness and medication. Of note, the median age of our residents is 49 years.

We aim to tackle this issue by giving Vitamin D supplementation (although this is currently problematic because there is no suitable vitamin D supplement supported by the Pharmaceutical Benefits Scheme). We will be encouraging more physical activity, particularly outdoors, as this is most likely to be of overall benefit to our residents' general health.

1 Lambert T, Velakoulis D, Pantelis C. Medical comorbidity in schizophrenia. *Med J Aust* 2003; 178 (9 Suppl): S67-S70.

2 Castle DJ, Pantelis C. Comprehensive care for people with schizophrenia living in the community. *Med J Aust* 2003; 178 (9 Suppl): S45-S46.

3 Sambrook PS, Cameron ID, Cumming RG, et al. Vitamin D deficiency is common in frail institutionalised older people in northern Sydney [letter]. *Med J Aust* 2002; 176: 560.

4 Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia. Vitamin D and adult bone health in Australia and New Zealand: a position statement. *Med J Aust* 2005; 182: 281-285. □

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