

against the use of medication to treat depression in children.<sup>11</sup> Children are less likely than adults to receive adequate analgesia for physical pain.<sup>12</sup> There is a danger that, because of limited research and some antagonism to drug treatment, the same thing may happen with depression.

Philip Hazell *professor*

University of Newcastle, Australia, Child and Youth Mental Health Service, Locked Bag 1014, Wallsend, NSW 2287, Australia  
 hazell@mail.newcastle.edu.au

- 1 Poznanski EO, Mokros HB. *Children's depression rating scale—revised*. Los Angeles: Western Psychological Services, 1996.
- 2 Sawyer M, Arney FM, Baghurst PA, Clark JJ, Graetz BW, Kosky RJ, et al. *The mental health of young people in Australia*. Canberra: Mental Health and Special Programs Branch, Commonwealth Department of Health and Aged Care, 2000.
- 3 Birmaher B, Ryan ND, Williamson DE, Brent DA. Childhood and adolescent depression: a review of the past 10 years, part I. *J Am Acad Child Adolesc Psychiatry* 1996;35:1427-39.

- 4 Garralda E, Rangel L, Levin M, Roberts H, Ukoumunne O. Psychiatric adjustment in adolescents with a history of chronic fatigue syndrome. *J Am Acad Child Adolesc Psychiatry* 1999;38:1515-21.
- 5 Hazell P, O'Connell D, Heathcote D, Henry D. Tricyclic drugs for depression in children and adolescents. *Cochrane Database Syst Rev* 2002;(2):CD002317.
- 6 Hazell P. Depression in children and adolescents. In: Barton S, ed. *Clinical evidence*. Issue 6. London: BMJ Publishing Group, 2002:278-84.
- 7 Harrington R, Whittaker J, Shoebridge P, Campbell F. Systematic review of efficacy of cognitive behaviour therapies in childhood and adolescent depressive disorder. *BMJ* 1998;316:1559-63.
- 8 Clarke GNP. Targeted Prevention of unipolar depressive disorder in an at-risk sample of high school adolescents: A randomized trial of a group cognitive intervention. *J Am Acad Child Adolesc Psychiatry* 1995;34:312-21.
- 9 Emslie GJ, Rush AJ, Weinberg WA, Kowatch RA, Hughes CW, Carmody T, et al. A double-blind, randomized, placebo-controlled trial of fluoxetine in children and adolescents with depression. *Arch Gen Psychiatry* 1997;54:1031-7.
- 10 Avci A, Diler RS, Kibar M, Sezgin F. Comparison of moclobemide and placebo in young adolescents with major depressive disorder. *Ann Med Sci* 1999;8:31-40.
- 11 Breggin PR. *Reclaiming our children: a healing plan for a nation in crisis*. Bethesda: Lake House Books, 2000.
- 12 Petrack EMM. Pain management in the emergency department: patterns of analgesic utilization. *Pediatrics* 1997;99:711-4.

## Mr Milburn's good hospital guide

*One star for trying*

The self proclaimed aim of Health Secretary Alan Milburn's star awarding exercise, rating the performance of trusts, is simple. It is "to provide patients and the general public with comprehensive, easily understandable information on the performance of their local health services."<sup>1</sup> No one could argue with that. But execution is another matter. Two dilemmas arise when constructing a summary measure of performance in an organisation as complex and heterogeneous as the NHS. On the one hand, how "comprehensive" can it be while remaining "easily understandable?" Sophistication can all too easily turn into mystification. On the other hand, can the same sort of exercise meet the requirements of the multiple audiences involved? The general public apart, these include ministers and their officials, the boards of trusts as well as the doctors, nurses, and others working in them, and the commissioners of services. Attempting to meet everybody's expectations may mean frustration all round.

The Department of Health's performance ratings are the product of a complex game of statistical snakes and ladders.<sup>2</sup> For acute trusts, they are based on three very different sets of information (the methodology is different for ambulance and mental health trusts). Firstly, there are the department's own political targets: nine in all, dominated by financial performance and various waiting targets. Secondly, there are the judgments of the inspectors of the Commission for Health Improvement, marking the reviewed trusts on seven dimensions. Thirdly, there are 29 performance indicators split into three groups—with a clinical, patient, and staff focus, respectively—that together make up a so called balanced scorecard.

The various inputs have their own individual technical problems. So, for example, there are statistical problems about the presentation of clinical indicators.<sup>3</sup> The Commission for Health Improvement's ratings are based on a fragile, still evolving methodology.<sup>4</sup> But

the biggest problem lies in the process of converting 38 indicators into a summary measure. If a trust "significantly underachieves" on three of the Department of Health's key targets, it falls automatically into the category of the damned: zero stars and faces the prospect of visits from the missionaries of the NHS Modernisation Agency at best and the threat of being taken into the pupilage of a successful trust at worst. A highly critical report from the Commission for Health Improvement has the same effect. But thereafter all simplicity vanishes. If a trust achieves all of the Department of Health's key targets, it is not automatically guaranteed three star status and what goes with it: the promise of £1 000 000, earned autonomy, and keys to the heaven of foundation status. It may lose one of its stars, failing a satisfactory review from the Commission for Health Improvement or adequate balanced scorecard performance (defined as being outside the lowest 20% of the distribution for all three areas and within the top 50% in one area). Conversely, a moderate level of underachievement on the key targets may be compensated by a satisfactory balanced scorecard performance or review from the Commission for Health Improvement, turning one star into two.

The methodology is open—inasmuch as it is available on the web—but hardly transparent. What is the public to make of it all? Even assuming that there is scope for choice, a prospective patient is more likely to be interested in the performance of a specific department or doctor than in an ambiguous star award. The system may well raise unnecessary anxiety among the public as well as anger among clinicians. So, for example, two of the "starless" trusts, Bath and Bristol, score considerably better on their clinical indicators than some with three stars. Also, it is not self evident that either ministers or trust boards need a star system to stir them into action: they know all about the Department of Health's key targets, which, for better or worse, are the main driving force. Further, it is not clear

News p 236

BMJ 2002;325:230-1

how much stability there is in the ratings: less than half the acute trusts retained their 2001 ratings with 47 moving up and 37 moving down. Although differences in methodology may be partly responsible, this hardly suggests that the rating system provides a solid base for policy making.

Next year the Commission for Health Improvement takes over responsibility for the assessment system and faces the challenge of making it less opaque and more comprehensible. In doing so, it might usefully consult the original exponent of the star system: the Michelin guide. In classifying hotels Michelin does not just award stars for the cooking. Nor does it try to collapse all aspects of an institution into one metric. Instead, it has an elaborate battery of symbols

for different aspects of the performance of the hotel. Something similar for trusts might be richer in information, provoke less anxiety or anger, and above all be more accurate because it is multidimensional.

Rudolf Klein *visiting professor*

London School of Hygiene and Tropical Medicine, London  
WC1E 7HT

Rudolfklein30@aol.com

- 1 Department of Health. *NHS performance ratings: acute trusts, specialist trusts, ambulance trusts, mental health trusts 2001/02*. London: DoH, July 2002
- 2 Department of Health. *Performance rating methodology: acute NHS hospital trusts*. <http://www.doh.gov.uk/performance/2002/method-acute.html> (accessed 27 Jul 2002).
- 3 Rixom A. *Performance league tables*. *BMJ* 2002;325:177-8.
- 4 Day P, Klein R. Who nose best? (sic) *Health Serv J* 2002;112:26-9.

## Continuous combined hormone replacement therapy and endometrial hyperplasia

*Risk of developing cancer is very low*

Papers p 239

The use of continuous combined hormone replacement therapy, consisting of an oestrogen and a progestogen taken daily by postmenopausal women, is increasing. Its possible benefits are the prevention of endometrial hyperplasia and reduction in the occurrence of endometrial bleeding with time. Daily exposure to oestrogen and progestin without a break may be more important than using oestrogen intermittently in prevention of disease. A major concern is the occurrence of endometrial cancer in women using cyclic or sequential hormone replacement with the progestin being given for either less than 10 days each month, 10-16 days each month, or every three months for 14 days.<sup>1,2</sup> The case-control studies indicate a significant increased risk in endometrial cancer with a reduction in the number of days of exposure to progestin. The use of continuous combined hormone replacement therapy not only does not increase the incidence of endometrial cancer but could even be protective compared with non-use of hormone replacement.<sup>3</sup>

Most clinical trials of continuous combined hormone replacement therapy have been for one year in order to obtain regulatory approval for the products.<sup>4</sup> In some instances two and three years of use have been reported, but these data are limited.<sup>5</sup> The end point in clinical trials is endometrial hyperplasia rather than endometrial cancer because of the low incidence of endometrial cancer in the general population. In clinical situations we assume that inhibition of endometrial hyperplasia implies endometrial protection. This assumption has been challenged recently, with a call for randomised prospective clinical trials to document the efficacy of progestins in preventing endometrial cancer.<sup>6</sup>

To date, all clinical trials of unopposed oestrogen at moderate and high doses have shown an increase in the incidence of endometrial hyperplasia, which is related to dose and duration.<sup>4</sup> The same is true for endometrial cancer after use of unopposed oestrogen.<sup>1,2</sup> The rate of endometrial hyperplasia was no different for continuous combined hormone replacement

and placebo in a Cochrane meta-analysis.<sup>4</sup> With use of sequential hormone replacement, the rates of endometrial hyperplasia were no different from placebo, although there was an increase in the occurrence of hyperplasia after 24 months (odds ratio 4, 95% confidence interval 1.2 to 14.0).

Doctors are confronted with women who have taken continuous combined hormone replacement for several years and then experience endometrial bleeding and spotting. Assessment of these women has entailed ultrasound imaging of the endometrium, hysteroscopy, and endometrial assessment through biopsy. The accuracy of ultrasonography in diagnosing endometrial disease in these patients is open to question.<sup>7</sup> The reason for this intensity of evaluation of the bleeding is that doctors have been trained to evaluate aggressively any endometrial bleeding in postmenopausal women. These investigations have usually failed to document any malignant cause of the bleeding in women taking continuous combined hormone replacement; rather, endometrial polyps or uterine fibroids seem to be the most common finding.

A paper in this issue (p 239) addresses the issue of limited published data in long term users of continuous combined hormone replacement by presenting a 5 year follow up of postmenopausal women taking a preparation of 2.0 mg oestradiol and 1.0 mg norethindrone acetate (Kliofem/Kliogest; Novo Nordisk, Denmark).<sup>8</sup> The paper found no evidence of endometrial hyperplasia after five years of continuous combined hormone replacement therapy. Moreover, 75% of the women had a final endometrial assessment. This is noteworthy because the usual attrition rates in clinical trials are higher than that in this study.

These data are reassuring because they are in agreement with case-control studies that have documented a reduction in the incidence of endometrial cancer in women taking continuous combined hormone replacement therapy.<sup>1-3</sup> These data should, however, be taken in context with the formulation of oestrogen and progestin used in the study—oestradiol-