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.

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Continuous combined hormone replacement therapy and endometrial hyperplasia

Risk of developing cancer is very low

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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.[1 2] 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.[3]

Most clinical trials of continuous combined hormone replacement therapy have been for one year in order to obtain regulatory approval for the products.[4] In some instances two and three years of use have been reported, but these data are limited.[5] 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.[6]

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.[4] The same is true for endometrial cancer after use of unopposed oestrogen.[1 2] The rate of endometrial hyperplasia was no different for continuous combined hormone replacement and placebo in a Cochrane meta-analysis.[4] 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.[7] 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).[8] 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.[1–3] These data should, however, be taken in context with the formulation of oestrogen and progestin used in the study—oestradiol-

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17β and norethindrone. Other formulations of oestrogen and progestin may not result in the same outcome. This is a speculative statement, based on the fact that each progestin has a different biological profile. On the basis of biochemical parameters, norethindrone could be considered a more potent progestin than either medroxyprogesterone acetate or progesterone.[9] To date endometrial morphology has been used to determine the safety of the progestin used with oestrogen in hormone replacement preparations. The end point in clinical trials has been the morphological changes seen in the endometrial tissue acquired through biopsy. The accuracy of the interpretation of the histology of the endometrium between pathologists has been questioned because of the discrepancies found in the interpretation of the endometrium in clinical trials.[10]

Better markers of endometrial stimulation and inhibition than that of histology alone are needed. Until these are available, we must rely on the pathological interpretation of the findings, as was done in this study, to reassure us that the endometrium is protected with continuous combined hormone replacement therapy. For clinicians this means that investigation of a woman taking continuous combined hormone replacement without bleeding is not required, and with bleeding and spotting the chances of finding a neoplasm are low to non-existent.

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Prevention and cure of type 2 diabetes

Weight loss is the key to controlling the diabetes epidemic

The Department of Health has published the first part of the national service framework defining standards of care for people with diabetes. The substance—how these standards will be achieved—is now awaited. Type 2 diabetes, however, is reaching epidemic proportions, and epidemics are seldom controlled unless their causes are addressed. Obesity is strongly and causally linked to type 2 diabetes. Recent data suggest that the prevention of diabetes is feasible if weight management is addressed adequately in individuals at high risk. More controversially, weight management also has the potential to make a significant impact in those with established type 2 diabetes.

The most common definition of obesity is a body mass index greater than 30 kg/m^2. In the nurses' health study the risk of type 2 diabetes in women with an index of 29-31 was 28-fold increased compared with women with an index lower than 22, and an index greater than 35 carried a 93-fold increased risk.[1]

The overall prevalence of self reported diabetes in the United States has reached 7.3%, and 15% in people over 60 years of age, driven by epidemic obesity.[2] There is no room for complacency in the United Kingdom. The prevalence of known and new type 2 diabetes, detected by oral glucose tolerance test, was 20% in Europeans, 22% in Afro-Caribbeans, and 33% in Pakistanis in urban Manchester.[3] Obesity and physical inactivity were the principal factors associated with diabetes, and waist circumference, a measure of intra-abdominal fat, was the strongest predictor of glucose tolerance. Similarly, obesity related diabetes in childhood, already common worldwide, has now reached the United Kingdom.[4]

So, could we prevent type 2 diabetes? In a prospective study of 84 941 female nurses followed for 16 years, a combination of five modifiable risk factors related to dietary behaviour, physical activity, weight, and cigarette smoking was identified that was associated with a remarkable 91% reduction in the risk of developing diabetes.[5] Even with a family history of diabetes the risk reduction was 88%. In theory, therefore, most diabetes could be preventable, largely irrespective of genetic background.

Two pioneering studies show that this is feasible. In the Finnish diabetes prevention study weight loss in overweight subjects with impaired glucose tolerance, averaging just 3-4 kg over 4 years, led to a 58% reduc-

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