and V and no significant decrease by men in socioeconomic group V or women in socioeconomic group III.

Our data do not exclude the future impact of health publicity or, indeed, of a reduction or cessation in advertising on smoking by lower socioeconomic groups, and we think it important that there should be research into and evaluation of health promotion and advertising aimed at these groups.

JOY TOWNSEND

Senior scientist

PAUL RODERICK

Consultant in public health medicine

JACQUELINE COOPER

Statistician

MRC Epidemiology and Medical Care Unit,
Wolfson Institute of Preventive Medicine,
Medical College of St Bartholomew's Hospital,
London EC1M 6BQ

1 Reid D. Effects of health publicity on prevalence of smoking. *BMJ* 1994;309:1441. (26 November.)

2 Townsend J, Roderick P, Cooper J. Cigarette smoking by socioeconomic group, sex, and age: effects of price, income, and health publicity. *BMJ* 1994;309:923-7. (8 October.)

Nestlé abides fully to WHO code on baby milk

EDITOR,—Andrew Radford justifies his attack on the British Paediatric Association by giving a reference to a booklet produced by the International Baby Food Action Network, *Breaking the Rules 1994*.^{1,2} He adopts his usual stance of assuming that if his organisation makes an allegation then the rest of the world must believe it to be true, even though many of the accusations are imprecise and unverifiable, have been hoarded for many months, or concern products not covered by the World Health Organisation's marketing code.

Nestlé's investigations indicate that among 455 allegations counted in *Breaking the Rules 1994* and three supporting letters from Baby Milk Action only three charges required corrective action.³ In one case this had been effected before the publication of the booklet. Not only do our investigations show the unreliability of the "monitoring" by International Baby Food Action Network but a number of independent third parties have confirmed the inaccuracy of the accusations. For example, in one case the network's monitor, an adviser on breast feeding, herself declared the accusation regarding her clinic to be "false and unfounded"; in another case the head of a maternal and child health clinic declared the allegations to be "in complete contradiction to what is happening here in the field." Investigations into the unpublished reports from Unicef similarly indicate the inaccuracy of the accusations.

The only conclusion that can be drawn from *Breaking the Rules 1994* is that the British Paediatric Association is fully justified in adopting the position it has done, and not that Nestlé is breaching the code. Nestlé has no wish to intervene in any decision that the British Paediatric Association takes with regard to commercial sponsorship. The association can, however, remain assured that Nestlé abides fully by the WHO's code and all other national agreements.

R F CLAYDON
Manager

Nestlé UK,
Corporate Affairs Department,
Croydon,
Surrey CR9 1NR

1 Radford A. Nestlé's donation. *BMJ* 1994;309:1585. (10 December.)

2 International Baby Food Action Network. *Breaking the rules 1994. A worldwide report on violations of the WHO/Unicef international code of marketing of breast milk substitutes*. Cambridge: Baby Milk Action, 1994.

3 Nestlé UK. *Marketing of infant formula—Nestlé response to "Breaking the Rules 1994."* Croydon: Nestlé, 1994.

Nocardia pericarditis

EDITOR,—S J Tabrizi lists the clinical contexts with which nocardial infection has been associated.¹ Malnutrition due to anorexia nervosa should be added to this list. We recently saw a 29 year old Greek man with a long history of anorexia nervosa who had been referred to Britain. He was receiving inpatient psychiatric and dietary treatment in a specialist eating disorders clinic when he complained of a dry cough of 12 days' duration, which became productive with bloodstained sputum. A Heaf test had yielded a positive result when he was a schoolboy. Three years previously he had weighed 92 kg, but after finishing his military service he had started to diet and had rapidly lost weight. There was no history of diarrhoea or self induced vomiting. He was tall (180 cm) and emaciated, weighing only 37 kg. His body mass index was extremely low at 11.4 (normal range 20-24).

Physical examination showed dullness to percussion over the right apical region of the chest anteriorly and oedema of the ankles. Chest radiographs, which had been normal 32 days before, showed a cavitating lesion in the right apex, and the erythrocyte sedimentation rate had risen to 156 mm in the first hour. *Nocardia asteroides* was isolated from a fibreoptic bronchoscopic lavage specimen and subsequently from three separate sputum specimens. No mycobacteria were seen or isolated by culture.

He was initially treated for tuberculosis as the most likely diagnosis and subsequently with sulphadimidine 1 g four times daily for three months. The results of liver function tests, full blood count, and erythrocyte sedimentation rate returned to normal, and the chest radiological appearances resolved. He also slowly gained weight and achieved the mean weight in a matched population.

This is the first report of anorexia nervosa associated with nocardial infection. Malnutrition is recognised as predisposing to infections with various pathogens,² including mycobacteria,³ probably owing to its effects on the function of polymorphonuclear cells,⁴ antioxidant systems,⁵ and humoral immunity. Since anorexia nervosa is fairly common it is interesting that infections such as this are not seen more often.

A D MACKAY
Senior registrar in medical microbiology

G M SCOTT
Consultant in clinical microbiology

S L COHEN
Consultant in medicine

Clinical Microbiology Department,
University College London Hospitals,
London WC1E 6BD

E STONEHILL
Director

Eating Disorders Unit,
Charter Nightingale Hospital,
London NW1 6SH

1 Tabrizi SJ. *Nocardia pericarditis*. *BMJ* 1994;309:1495-7. (3 December.)

2 Rhoads JE. The impact of nutrition on infection. *Surg Clin North Am* 1980;60:41-7.

3 Gupta K, Sivakumar K. Anorexia nervosa and tuberculosis: case reports and a review of immune mechanisms. *Int J Eat Disord* 1994;15:301-4.

4 McMurray DN. Cell-mediated immunity in nutritional deficiency. *Prog Food Nutr Sci* 1984;8:193-228.

5 Vaisman N, Tabachnik E, Hahn T, Voet H, Guy N. Superoxide production during refeeding in patients with anorexia nervosa. *Metabolism* 1992;41:1097-9.

Risk factors for breast cancer

EDITOR,—Peter J Lewis comments on the fact that our article on risk factors for breast cancer did not include the role of xeno-oestrogens and progesterone deficiency.¹ The aim of the ABC series is to review the current state of knowledge succinctly. The article did not mention the two factors because

the evidence linking them with breast cancer is conflicting. While some studies have linked pesticides with breast cancer, others have not.² Definitive evidence that pesticides either induce or promote breast cancer has yet to be presented.

The role of progesterone in the breast is unclear. There is a body of opinion that progesterone deficiency, leading to unopposed oestrogen action, is associated with an increased risk of breast cancer.³ Others, however, have failed to show that women either at high risk of, or with, breast cancer have abnormal progesterone concentrations.⁴ Furthermore, there is an opposing view that high progesterone concentrations may be a determinant of risk and that the hazard of a prolonged reproductive life is mediated through cumulative exposure to an excessive number of ovulatory menstrual cycles.⁵ Progesterone also seem to be mitogenic in the normal breast, with increased proliferation being found in the luteal phase compared with the follicular phase of the menstrual cycle.⁶ Proliferation is also increased in women using combined oestrogen-progesterone contraceptive pills.⁶ Against this background, the case for giving progestogens to women at high risk of breast cancer is at best unconvincing and at worst potentially hazardous.

K MCPHERSON

Professor of public health epidemiology

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

C M STEEL

Professor of medical and biological sciences

University of St Andrews,
St Andrews, Fife

J M DIXON

Consultant surgeon

Edinburgh Breast Unit,
University Department of Surgery, Royal Infirmary,
Edinburgh EH3 9YW

W R MILLER

Professor of experimental oncology

ICRF Oncology Unit,
Western General Hospital,
Edinburgh EH4 2XU

1 Lewis PJ. Risk factors for breast cancer. *BMJ* 1994;309:1662. (17 December.)

2 Key T, Reeves G. Organochlorines in the environment and breast cancer. *BMJ* 1994;308:1520-1.

3 Cowan LD, Gordis L, Tonascia JA, Jones GS. Breast cancer incidence in women with progesterone deficiency. *Am J Epidemiol* 1981;114:209-17.

4 Moore JW, Thomas BS, Ang DY. Endocrine status and epidemiology and clinical course of breast cancer. *Cancer Surv* 1980;5:537-60.

5 Apter D, Vihko R. Early menarche, a risk factor for breast cancer, indicates early onset of ovulatory cycle. *J Clin Endocrinol Metab* 1983;57:82-6.

6 Anderson TJ, Miller WR. Morphological and biological observations relating to the development and progression of breast cancer. In: *Mammary tumorigenesis and malignant progression*. Dordrecht: Kluwer Academic, 1994:3-27.

Trials in palliative care

EDITOR,—In their discussion of the problems and pitfalls of evaluating a palliative care service McWhinney and colleagues mentioned three published trials in palliative care services.¹ Apparently only trials with additional care services were selected, and trials on palliative treatment were excluded. In the field of additional care services we found one other trial.² Many trials in palliative cancer care report problems with patient selection, accrual, attrition, contamination of exposure, and selection of adequate outcome measures. We also conducted a randomised trial on palliative care services, for patients with disseminated colorectal cancer. Our trial compared fluorouracil as single agent by continuous infusion at home versus combination therapy with leucovorin at an outpatient clinic. Main outcome measures were quality of life, use of care services, and satisfaction with care. Quality of life was assessed with a global assessment with a linear analogue scale, the medical outcomes study's 24 item short form, the affect balance scale, the

Rotterdam symptom checklist, the instrumental help for daily living scale, the activities of daily living scale, and the social experiences scale. Satisfaction was assessed with the Rand corporation's patient satisfaction questionnaire.

Our trial failed to attain the intended sample size too and we stopped the intake of patients. McWhinney and colleagues reported problems with patient recruitment, attrition, and the exposure of the control group to services equivalent to the home support team. In our case the major problem was patient accrual. As it happened, the number of eligible patients was nearly half that of previous years. To improve patient intake we invited on several occasions 11 associated centres to refer their patients. During two years only 22 patients entered the trial, of whom nine came from associated centres. Furthermore, 22 eligible patients refused to participate in the trial. This was suspected early on in the trial, and the reason for refusal and the characteristics of these patients were registered. Eight patients did not want chemotherapy, 10 refused either continuous infusion or randomisation, and four were not asked by the physician because of medical reasons. In contrast with McWhinney and colleagues' experiences, attrition did not have a major role—four patients died before the first evaluation at two months, and all other patients completed the questionnaires. A possible explanation for this difference could be that the patients in McWhinney and colleagues' trial had a more advanced disease than our patients.

Despite inclusion criteria allowing only patients with disseminated colorectal cancer, without metastasis to the brain, and with normal results of haematology tests, with normal liver and kidney function, World Health Organisation performance status of 2 or more, and no serious comorbidity to participate, our study population turned out to be rather heterogeneous. Intersubject variability of the measures of quality of life was great. Individual plots of quality of life scores over time seemed stable when the clinical situation was stable and seemed to decline around the time of disease progression. No apparent relations between quality of life and satisfaction were found. Small sample size and great intersubject variability do not allow for further statistical analysis.

In conclusion, randomised trials in palliative cancer care are difficult, but not impractical. Our trial failed because of difficulties in patient accrual, but other common problems, such as attrition, contamination of exposure, or selection of adequate outcome measures did not have a major role. Future randomised trials in palliative cancer care should consider multicentre participation and the possibility of patient entry without limitations to type of primary cancer. Moreover, the care intervention should be well defined, and adequate outcome variables should be selected.

GERDY RINCK
Junior clinical epidemiologist
JOS KLEIJNEN
Clinical epidemiologist

Department of Clinical Epidemiology and Biostatistics,
Academic Medical Centre,
University Hospital of Amsterdam,
PO Box 22700,
1100 DE Amsterdam,
Netherlands

TRUDI G A M VAN DEN BOS
Professor of social medicine and chronic diseases
Institute of Social Medicine,
Academic Medical Centre

HANNEKE J C J M DE HAES
Associate professor of medical psychology
Department of Medical Psychology,
Academic Medical Centre

EGBERT SCHADE
Professor of family medicine
Department of General/Family Medicine,
Academic Medical Centre

CEES H N VEENHOF
Associate professor of medical oncology
Division of Medical Oncology,
Department of Internal Medicine,
Academic Medical Centre

- 1 McWhinney IR, Bass MJ, Donner A. Evaluation of a palliative care service: problems and pitfalls. *BMJ* 1994;309:1340-2. (19 November.)
- 2 Ovesen L, Allingstrup L, Hannibal H, Mortensen EL, Hansen OP. Effect of dietary counseling on food intake, body weight, response rate, survival and quality of life in cancer patients undergoing chemotherapy: prospective randomized study. *J Clin Oncol* 1993;11:2043-9.

- 1 Dillner L. Scottish death rates published with health warning. *BMJ* 1994;309:1599-600. (17 December.)
- 2 Goldacre MJ. Hospital inpatient statistics: some aspects of interpretation. *Community Medicine* 1981;3:60-8.
- 3 Goldacre M, Seagroatt V, Hawton K. Suicide after discharge from psychiatric inpatient care. *Lancet* 1993;342:283-6.
- 4 Seagroatt V, Goldacre MJ. Measures of early post-operative mortality: beyond hospital fatality rates. *BMJ* 1994;309:361-5.
- 5 Gill L, Goldacre MJ, Simmons H, Bettley G. Computerised linking of medical records: methodological guidelines. *J Epidemiol Community Health* 1993;47:316-9.

Interpreting hospital death rates

Can be difficult

EDITOR,—We agree that hospital death rates for myocardial infarction can be difficult to interpret.¹ In a guide to hospital statistics and their interpretation we reported hospital death rates in Oxford, which ranged from 8% (27/341) to 73% (73/100).² The hospitals were anonymised because our code of practice is not to identify individual hospitals in publications.² The hospital with a death rate of 8% admitted only convalescent medical patients: its statistics were selectively based on survivors. The hospital with a death rate of 73% was a geriatric hospital: many of its patients were admitted, with conditions other than myocardial infarction, for long stay care. Those whose deaths were certified as due to myocardial infarction contributed not only to the numerator but also to the denominator because they had a fatal infarct while in hospital.

Circumstances such as these should be easy to identify locally, but a more important problem is that of defining a hospital admission when patients with myocardial infarction die close to the time of admission. There may be considerable variation among hospitals in whether such patients have been formally admitted and therefore included in the hospital's statistics.² We also know currently of a hospital computer system in which, when the patient's disposal is unknown to the coder, the default option is that the patient is coded as alive. A useful check in studies of hospital death rates is to identify death certificates that specify that the patient died in hospital and see whether a corresponding hospital record abstract exists, and vice versa. Although problems like these arise, however, they should be addressed in the analysis and interpretation of hospital statistics rather than be regarded as reasons for not auditing hospital care.

England's problem has been not so much the lack of means to link records but the incompleteness of its data since the Körner changes. For example, in the Oxford region from 1979 to 1986 (when collection of data for hospital statistics was managed by the Oxford record linkage study) 97% of hospital activity analysis records (1 557 753/1 609 588) included diagnostic data. From 1987 to 1990 the figure dropped to 79% (1 095 711/1 383 973), and it has only gradually crept back towards earlier levels of completeness. The completeness of clinical data in the national hospital dataset in England fell from the mid-1980s, and the hospital inpatient enquiry and the mental health enquiry—previously rich sources of information—have not been available since 1985.

Mortality after care in hospital can be studied in England.^{3,4} Statistical information about hospital episodes, including data needed to link records, exists in patient administration systems in most hospitals. Death certificates can be made available locally for linkage: copies are held by district departments of public health. Software to match records for linkage has been developed to run on microcomputers, and copies of it can be made available to anyone in the NHS in England.⁵

MICHAEL GOLDACRE
Honorary director
LEICESTER GILL
Chief computer scientist

Oxford Record Linkage Study,
Department of Public Health and Primary Care,
University of Oxford,
Anglia and Oxford Regional Health Authority,
Oxford OX3 7LF

Report was intended to promote discussion

EDITOR,—The stated intention of the Clinical Resources and Audit Group in publishing its report on clinical outcomes is to promote discussion among clinicians to determine what lessons can be learnt to improve the treatment of patients—a worthy and proper aim.¹ Contrary to the group's advice, however, the data provided have been put into league tables. This has caused considerable and quite unnecessary anxiety to people living in Fife. Had the opportunity been afforded to trusts to comment on a draft of the document before publication this anxiety would have been avoided by the correction of a simple error—that is, the inclusion of elderly patients in the figures for myocardial infarction. Furthermore, the appropriate and more satisfactory figure would have been attributed to the appropriate trust—namely, the Queen Margaret Hospital Acute NHS Trust.

When it was first suggested that these tables should be published medical directors in Scotland expressed considerable disquiet, being concerned that wrong messages would be given to the public and that the primary purpose, of promoting open discussion among clinical staff, would be undermined. This view is now well justified. The expectation is that purchasers will use the information in the report to influence the contracting process. Therefore if the public is not to be caused unnecessary anxiety, and if the document is to have credibility for clinicians and in the contracting process, basic errors must be eliminated before any further publication.

N W HARRY
Medical director

Fife Healthcare NHS Trust,
Leven,
Fife KY8 5RG

- 1 Dillner L. Scottish death rates published with health warning. *BMJ* 1994;309:1599-600. (17 December.)

Asthma trends

EDITOR,—Helen Booth and colleagues suggest that differences in the output of DeVilbiss No 40 and No 45 nebulisers¹ may have influenced our estimates of the increase in the prevalence of airway hyperresponsiveness in children studied 10 years apart, in 1982 and 1992.² We expressed airway responsiveness both as a dose-response ratio (defined as the percentage fall in forced expiratory volume in one second divided by the dose administered) and as airway hyperresponsiveness (defined as a 20% fall in forced expiratory volume in one second at a dose of 3.92 μmol histamine). As reported, we normalised the dose-response ratios measured in 1992 to take account of methodological differences in collecting the data in 1982 and 1992 and so that we could more accurately compare the dose-response ratios measured in 1982 with those measured in 1992. To normalise the 1982 values we added a further constant to the dose-response ratio so that the cumulative frequency response curve for normal subjects studied in 1992 matched that for normal subjects studied in 1982. This adjusted for systematic methodological changes between the years, including nebuliser outputs, brand of spirometer,