corticosteroids in patients with advanced cancer as they do in patients with other conditions.

As an essential safeguard, therefore, doctors should state clearly in their notes why a corticosteroid is being prescribed and tell their patients why. Except where the aim is to control the tumour, the corticosteroid should be prescribed initially on a trial basis for no more than a week: the chances of obtaining a better response after this time are poor.[7] Treatment should be continued only if subjective or objective benefit occurs. Using corticosteroids for their general effects (those on appetite, mood, and strength) should be avoided as far as possible in anxious patients and in patients with diabetes because of the risk of worsening the associated condition.

Stopping corticosteroids abruptly after a week is safe if no more than prednisolone 40 mg a day or its equivalent (methylprednisolone 32 mg or dexamethasone 6 mg a day), has been taken.[14] Short courses of larger doses and longer courses of lower doses will suppress the hypothalamic-pituitary-adrenal axis for prolonged periods, and doses must be tapered off over several days or weeks according to circumstances.

Needham *et al* also point out that advanced cancer and polypharmacy tend to go hand in hand. Stopping drugs that are not yielding benefit will therefore help to ease the patients' burden of tablet taking and may improve compliance with other drugs. Furthermore, because the biological half lives of corticosteroids are relatively long (for example, 18-36 hours for prednisolone and 36-54 hours for methylprednisolone)[15] they should be taken once a day unless the number of tablets precludes this.

An important unresolved question is the choice of dose; in controlled trials to treat anorexia the dose has varied between the equivalent of 15 mg and 40 mg of prednisolone a day.[7 8 16 17] It may be better to start with a relatively high dose in order not to miss an effect of treatment and then to reduce to a lower maintenance dose if treatment is to continue beyond seven days. In patients receiving anticonvulsants such as phenytoin and phenobarbitone, starting with an even higher dose may be advisable because these drugs enhance the metabolism of corticosteroids.[18]

Finally, well documented alternatives for treating anorexia exist. For example, many patients benefit from megestrol acetate, and the effect is still detectable after two months.[19 20] Megestrol is, however, considerably more expensive. Given the 50% response to placebo,[16] the best initial step may well be dietary advice with or without multivitamin tablets.

ROBERT TWYCROSS

Macmillan Clinical Reader in Palliative Medicine,
Oxford University,
Oxford OX1 2JD

1 Hanks GW, Trueman T, Twycross RG. Corticosteroids in terminal cancer: a prospective analysis of current practice. *Postgrad Med J* 1983;**59**:702-6.
2 Gilbert RW, Kim JH, Posner JB. Epidural spinal cord compression from metastatic tumours: diagnosis and treatment. *Ann Neurol* 1978;**3**:40-51.
3 Carter RL, Pittam MR, Tanner NSB. Pain and dysphagia in patients with squamous carcinomas of the head and neck: the role of perineural spread. *J R Soc Med* 1982;**75**:598-606.
4 Flombaum CD, Schroy P, Watson R, Vanamee P. Treatment of acute obstructive renal failure with high-dose methylprednisolone. *Arch Intern Med* 1986;**146**:58-61.
5 Minton MJ, Knight RK, Rubens RD, Hayward JL. Corticosteroids for elderly patients with breast cancer. *Cancer* 1981;**48**:883-7.
6 Tannock J, Gospodarawicz M, Meakin W, Panzarella T, Stewart L, Rider W. Treatment of metastatic prostatic cancer with low-dose prednisolone: evaluation of pain and quality of life as pragmatic indices of response. *J Clin Oncol* 1989;**7**:590-7.
7 Bruera E, Roca E, Cedaro L, Carraro S, Chacon R. Action of oral methylprednisolone in terminal cancer patients: a prospective randomized double-blind study. *Cancer Treat Rep* 1985;**69**:751-4.
8 Moertel CG, Schutt AJ, Reitemeier RJ, Hahn RG. Corticosteroid therapy of preterminal gastrointestinal cancer. *Cancer* 1974;**33**:1607-9.
9 Needham PR, Daley AG, Lennard RF. Steroids in advanced cancer: a survey of current practice. *BMJ* 1992;**305**:999.
10 Weissman DE, Dufer D, Vogel V, Abeloff MD. Corticosteroid toxicity in neuro-oncology patients. *J Neurooncol* 1987;**5**:125-8.
11 Capell H. Selected side-effects: steroid therapy and osteonecrosis. *Prescribers' Journal* 1992;**32**:32-4.
12 Schell HW. This risk of adrenal corticosteroid therapy in far-advanced cancer. *Am J Med Sci* 1966;**252**:641-9.
13 Guslando M, Titobello A. Steroid ulcers: a myth revisited. *BMJ* 1992;**304**:655-6.
14 Byyny RL. Withdrawal from glucocorticoid therapy. *N Engl J Med* 1976;**295**:30-2.
15 Speight TM, ed. *Avery's drug treatment*. 3rd ed. Edinburgh: Churchill Livingstone, 1987:562-4.
16 Willox JC, Corr J, Shaw J, Richardson M, Calman KC, Drennan M. Prednisolone as an appetite stimulant in patients with cancer. *BMJ* 1984;**288**:27.
17 Twycross RG, Guppy D. Prednisolone in terminal breast and bronchogenic cancer. *Practitioner* 1985;**229**:57-9.
18 Gambertoglio JG. Corticosteroids and anticonvulsants. *Drug Interactions Newsletter* 1983;**12**:55-8.
19 Tchekmedyian NS, Tait N, Moody M, Greco FA, Aisner J. Appetite stimulation with megestrol acetate in cachectic cancer patients. *Semin Oncol* 1986;**13**:37-43s.
20 Loprinzi CL, Ellison NM, Schaid DJ, Krook JE, Altmann LM, Duse AM. Controlled trial of megestrol acetate for the treatment of cancer anorexia and cachexia. *J Natl Cancer Inst* 1990;**82**:1127-32.

Pet birds and lung cancer

Smoking is still a confounder

Cigarette smoking accounts for about 80% of Britain's 40 000 deaths from lung cancer each year.[1] The contribution of other causes of deaths from lung cancer in the general population is thus small. It may, however, be increasing,[2 3] and natural radiation, occupational exposures, dietary intake of vitamin A, and familial predisposition have all been implicated.[4-7] A more recent hypothesis, advanced and tested by Holst *et al* in 1988,[8] is that some cases of lung cancer may be caused by exposure to pet birds. This hypothesis is independently tested in two studies published in this issue (pp 986-9, 989-92).[9 10]

The original study by Holst *et al* compared 49 patients with lung cancer with 98 randomly selected community controls.[8] With adjustment for smoking the relative risk of lung cancer from exposure to any pet bird five to 14 years before diagnosis was estimated at 6·7 (95% confidence interval 2·2 to 20·0). The two studies published in this issue are both larger but arrive at smaller estimates of risk: Kohlmeier *et al* report an adjusted odds ratio of 2·12, which was significant,[9] and Gardiner *et al* an unadjusted value of 1·58, which was not.[10] Gardiner *et al*, however, also analysed the effects of exposure to individual bird species and, though cautious about the validity of this subgroup analysis, found a significant fourfold increase in risk associated with exposure to pigeons. Thus there are now at least three independent reports describing an increased risk of cancer associated with exposure to pet birds. How likely is it that these findings are valid?

This will depend on the extent to which the investigators have eliminated bias and controlled for confounding in their study design and analysis. In case-control studies bias arises principally from the methods by which cases and controls are selected and exposure is measured, and once present it is difficult to remove. Confounding by factors that are related to both the exposure and the disease can be dealt with in the analysis so long as the confounding exposure is recognised and measured. The main potential source of confounding in studies of the aetiology of lung cancer is smoking, and, because both smoking and the keeping of pet birds tend to occur in lower socioeconomic groups, confounding of these effects is inherently likely.

Controlling successfully for confounding by smoking requires either that cases and controls are closely matched for

smoking history or that detailed and reliable information on smoking history is entered into the analysis. Sadly, although detailed smoking histories were taken in at least two of the three studies, smoking was included in their regression analyses only as a binary variable discriminating those who had ever smoked from non-smokers. None of the studies has therefore excluded the possibility that keepers of pet birds may tend to be heavier smokers. The influence of smoking may also have contributed to the differences in the estimates of the odds ratio between the studies: both Holst *et al* and Kohlmeier *et al* selected healthy age and sex matched community controls, who are less likely to have been heavy smokers than the cases[8 9]; Gardiner *et al* used as controls hospital inpatients, who are generally more likely to be smokers, particularly those admitted because of heart disease.[10] Such selection bias among the controls may have contributed to the smaller odds ratio in this study.

Gardiner *et al's* findings may also have been influenced by the tendency for cases to be interviewed by one investigator and controls by others. The authors have attempted to adjust for this systematic bias, but, without measuring the extent of the bias, it is probably not possible to remove it simply by adjusting for the interviewer in the analysis. A further problem is that the studies used exposure to pet birds from five years before diagnosis. For some types of tumour this is likely to include exposure occurring after rather than before the development of the cancer, raising the possibility of reverse causation.[11] Holst *et al* argued, however, that this was unlikely to be the case since increased exposure to birds was evident in cases up to 35 years before diagnosis.[8]

Despite the possible sources of error in these studies it is essential to give the work credit. If valid, the association with pet birds would not only identify an easily avoidable cause of disease but also open new avenues for pathogenetic research. The immediate priority is to build on these findings by conducting investigations that control properly for the effects of smoking. One solution would be to study lung cancer only in lifetime non-smokers, perhaps by combining data on non-smokers from these three studies. The finding in the two papers in this issue of an association with intake of vitamin A also highlights the potential value of investigating dietary intervention in the prevention of lung cancer.[6] Smoking may remain the most important cause of lung cancer, but we should not ignore other possible causes.

JOHN BRITTON
Senior Lecturer

SARAH LEWIS
Statistician

Respiratory Medicine Unit,
City Hospital,
Nottingham NG5 1PB

1 Johnson K, Callum C, Killoran A. *The smoking epidemic. Counting the cost in England.* London: Health Education Authority, 1991.
2 Carstensen JM, Axelson O. Changes in non-smoking related lung cancer with special reference to mortality trends in Swedish women. In: Davis DL, Hoel DG, eds. *Trends in cancer mortality in industrial countries.* New York: New York Academy of Sciences, 1990:159-64.
3 Axelson O, Davis DL, Forestiere F, Schneidermann M, Wagener D. Lung cancer not attributable to smoking. In: Davis DL, Hoel DG, eds. *Trends in cancer mortality in industrial countries.* New York: New York Academy of Sciences 1990:165-78.
4 Clarke RH, Southwood TRE. Risks from ionising radiation. *Nature* 1989;**338**:197-8.
5 Spiro SG. Lung tumours. In: Brewis RAL, Gibson GJ, Geddes DM, eds. *Respiratory medicine.* London: Baillière Tindall, 1990:832-79.
6 Willett WC. Vitamin A and lung cancer. *Nutr Rev* 1990;**48**:201-11.
7 Samet JM, Humble CG, Pathak DR. Personal and family history of respiratory disease and lung cancer risk. *Am Rev Respir Dis* 1986;**134**:466-70.
8 Holst PA, Kromhout D, Brand R. Pet birds as an independent risk factor for lung cancer. *BMJ* 1988;**297**:1319-21.
9 Kohlmeier L, Arminger G, Bartolomeycik S, Bellach B, Rehm J, Thamm M. Pet birds as an independent risk factor for lung cancer: case-control study. *BMJ* 1992;**305**:986-9.
10 Gardiner AJS, Forey BA, Lee PN. Avian exposure and bronchogenic carcinoma. *BMJ* 1992; **305**:989-92.
11 Geddes DM. The natural history of lung cancer: a review based on rate of tumour growth. *Br J Dis Chest* 1979;**73**:1-17.

Mental health services for children

Receiving too little attention

The Health of the Nation's targets for mental illness are to improve the health and social functioning of mentally ill people and to reduce the suicide rate.[1] The white paper has little to say on the mental health of children and adolescents, though it acknowledges their vulnerability to disorders which, if untreated, may have serious consequences in adult life. Preventing behavioural disorders in children is listed as a possible future target.

Improving the mental health of children and adolescents should be an end in itself and not merely a means of improving adult functioning, though it would achieve this. For example, childhood bereavement and parental loss and repeated separations from attachment figures (even without death) are associated with increased rates of depression in childhood and later life,[2-4] and therapeutic intervention with children facing or experiencing loss improves their mental health and functioning—at least in the short term.[5 6]

Many child and adolescent mental health services, however, are so stretched that they cannot extend their services for assessment and treatment to offer preventive interventions. These services rely heavily on social workers with special skills in family therapy and parental counselling; all over the country these workers are being removed from child psychiatric clinics as the social services departments of local authorities feel the financial pinch.

What of youthful suicides? Of every 100 000 young people aged between 15 and 19, 400 attempt suicide each year and three succeed.[7] At least a quarter of these young people have serious psychiatric disorders,[8] and 10% of boys and 3% of girls who attempt suicide go on to kill themselves.[9] How are hard pressed child and adolescent psychiatrists to meet the demand for reducing suicide rates in young people in addition to other increasing demands on their time,[10] and dwindling resources? One solution is for health service managers to recognise the improvements in therapeutic services that would result from employing all therapists, rather than relying on departments of social services and education to fund many of them.

Only four qualified child psychotherapists are employed by the NHS north of Birmingham,[11] and the NHS employs only a handful of the family therapists working in child psychiatric clinics. Yet the effectiveness of both these therapies has been shown in some common conditions[7] and, although clinical psychologists, child psychiatric nurses, and consultant child and adolescent psychiatrists can provide some of the therapy, lack of specialised training in some cases and their small numbers preclude every troubled child who needs treatment from getting it.

In this context a guide produced for purchasers of mental health services for children and adolescents by the charity Action for Sick Children is welcome. *With Health in Mind*, which describes the disorders seen in child psychiatric clinics,