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Smoking, lung function, and body weight

SIR,—The recent report by Dr B Nemery and others (22 January, p 249) conducted on manual steelworkers aged 45-55 found smokers to weigh significantly less than non-smokers and ex-smokers. Furthermore, smokers with airflow obstruction, defined as having a forced expiratory volume in one second/vital capacity (FEV_1/VC) less than 66%, weighed less than smokers with normal FEV_1/VC ratios.

We recently reported[1] the changes in lung function in smokers and ex-smokers with pulmonary emphysema. As part of this study the body weights were recorded. For each patient the linear regression coefficient of weight on time was calculated. A weighted mean linear regression coefficient on time together with its standard error was then calculated for both smokers and ex-smokers. Fifty six male patients were followed for a minimum of three years (range 3-13). Thirty seven patients, mean age 53·7 years, comprised the group of smokers who smoked during or throughout the study period. There were 19 in the group of ex-smokers, mean age 56·6 years, and all had ceased smoking before the start of the study and did not smoke thereafter. At initial assessment there was no significant difference between the smokers and ex-smokers with respect to age, lifetime tobacco consumption (kg), duration of smoking (years), and both FEV_1 and VC after treatment with a bronchodilator. There was no significant difference between the weights of either group expressed as a percentage of the predicted value[2] (mean (1SD); smokers 82·2 (11·0); ex-smokers 84·4 (15·6)).

The mean weight loss of the smokers was 0·05 (0·13) kg a year, a loss which was not significant. The ex-smokers gained weight at a mean rate of 0·53 (0·13) kg a year, a significant increase ($p<0·001$). The difference between the two groups was also significant ($p<0·001$).

The mean body weight of all 56 patients with emphysema was only 85% of the predicted value, supporting the findings of Dr Nemery and others that it is the subjects with abnormal lung function who have significant changes in body weight. The explanation of weight gain in patients with emphysema who cease smoking, however, remains speculative. Several ex-smokers noticed an increased appetite while some patients who continued to smoke used the excuse that weight gain had occurred when the smoking habit was abandoned.

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Double indemnity in oesophageal carcinoma?

SIR,—In our view Mr R M Kirk's leading article "Double indemnity in oesophageal carcinoma" (19 February, p 582) does something of a disservice to patients with oesophageal carcinoma. The suggestion that a 30% operative mortality can be expected following oesophagectomy comes from an exhaustive but unselective review of the literature over a long time.[1] Analysis of reports—quoted in this review—from centres of excellence in more recent years indicates that it is perfectly possible to achieve an operative mortality of 15-20% following oesophageal resection. Thoracic surgeons all over Britain contributing to the register compiled by the Society of Thoracic and Cardiovascular Surgeons report an operative mortality of 17·4% in 1122 resections.[2]

We agree that evidence is accumulating to suggest that oesophageal carcinoma is difficult to cure by resection because of early metastases and distant submucosal spread but contest the issue that patients who cannot be guaranteed a cure should be denied the opportunity for the best long term palliation possible—an operation to resect macroscopic tumour and to restore continuity and normal swallowing.

Furthermore, we doubt whether Mr Kirk's small series[2] represents sufficient evidence to advocate the use of total oesophagectomy with colonic or jejunal interposition as the treatment of choice for carcinoma. Bypass procedures leaving often fungating tumour in direct continuity with the patient's mouth are in our view unnecessary and unpleasant.

The outlook for patients with oesophageal carcinoma is certainly gloomy, but not nearly as black as it is painted in your leading article. Patients with resectable tumour attending a centre of oesophageal surgery can expect an over 80% chance of leaving hospital well and swallowing normally with at worst the prospect of many months or years palliation and at best a 10-15% chance of a cure.

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SIR,—Mr R M Kirk's article (19 February, p 582) is, I believe, misleading and fails to refer to some of the most recent publications on the subject from centres in the United Kingdom with large series of patients.[1 2]

No one denies the depressing overall long term results of the surgical treatment of this disease. Let us, however, be clear that the primary objectives of surgery in cancer of the oesophagus are the relief of obstruction and the establishment of normal swallowing (ideally) for as long as the patient lives, bearing in mind that: (*a*) the average age of the sufferer nears the average life span; and (*b*) most patients are referred when the disease is widespread.

The mortality of resection can be high, but none of the experienced groups and the recent statistics put it above 18-20%. In my own series of nearly 400 resections the hospital mortality was 11·5% and that of Dark[2] 9·2%.

Several points raised in Mr Kirk's article need re-evaluation. There is no evidence to show that the long term results are better by allowing a 12 cm instead of 5-6 cm minimum clearance. If the benefit is judged by relief of dysphagia resectional surgery (when possible) is more beneficial to patients than any other method of treatment. Admittedly, however, in the presence of widespread disease particularly when dysphagia is not complete the patient is best left alone and reassessed later in the light of symptoms. Patients with lymph node involvement should not be excluded and will still benefit from resectional surgery—a bypass operation makes sense only when the tumour cannot be removed.

The long term survival is affected by the extent of lymph node involvement. In our series of patients among those who underwent surgery[3] we found: 48% of patients (32% of total) had no lymph node involvement and of these 42% survived more than four years; 27% (18% of total) had proximal lymph node involvement and 12% of these survived four years but 40% lived for two years; 24% (17% of total) had proximal and distal lymph node involvement and none of these survived more than three years but 30% lived for 12-14 months.

Only rarely was recurrence of dysphagia due to recurrence of tumour at the site of anastomosis; more commonly it was due to tumour associated with the mediastinum, diaphragm, and porta hepatis. I believe, therefore, that resecting the whole of the stomach and the oesophagus will not improve the long term results (even Mr Kirk's small series confirms this[4]).

The message from the leading article should be: early and direct referral of patients to centres which deal with oesophageal surgery routinely and not once in a while, which can provide a better immediate result and a more expert overall management. Finally, the future of oesophageal cancer treatment must be seen in combination therapy. In this respect a pilot study of high dose concentrated preoperative radiotherapy followed by early surgery in our department appears to show improvement of long term results with no increase in hospital mortality (Moghissi K. Annual meeting of the Society of Thoracic and Cardiovascular Surgeons of Great Britain and Ireland, 1981).

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Locking up patients with psychiatric illness

SIR,—Dr Greg Wilkinson is to be congratulated on his article "Locking up patients with psychiatric illness (19 February, p 581). While the use of a quiet room where a patient can recover his composure after a violent emotional outburst may be permissible, to my mind the locking up of people in a bare room without access to toilet or washing facilities, particularly if the intent is disciplinary, has no place in a modern psychiatric treatment programme.

May I suggest that the commission con-

sidering the new Mental Health Act should not only take a close look at seclusion but give a clear lead to the caring professions by clarifying its use and abuse.

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SIR,—Dr Greg Wilkinson's subject of the use of seclusion in the nursing of psychiatric patients (19 February, p 581) is an important one for discussion. Its use in an emergency to help cope with disturbed behaviour is of particular interest to me. Guidelines are in use in many hospitals and are useful to safeguard not only patients but also nursing staff. Anyone managing a violent person is at risk of returning the violence in some form or of being accused of doing so. Psychiatric nurses should be protected from both.

Winnicott[1] points out that psychotic patients inspire hate in their therapists as a clinical phenomenon. This may be difficult enough to master at any time and is often handled by doctors by discharge, admission, or simply termination of an interview, all of which serve to remove the patient from the therapist and so alleviate the therapist's distress and likelihood of acting directly on the feeling.

The nurse with a psychotic, non-voluntary patient is in a similar position but with fewer options. If the patient's aggression is not only emotional but also physical the difficulty increases. The nurse must handle the patient physically with just enough force to contain him and no more and must also tolerate the emotions that the psychotic generates. In other words the nurse must contain the patient's feelings and the patient's body, non-punitively.

I agree with Dr Wilkinson that hospitals should, as a minimum safeguard, have strict regulations about the use of seclusion. Any action which compromises the freedom of a human being must be constantly subject to scrutiny and particularly so when concerned with people who have already lost their freedom on account of mental illness. The option of seclusion not only protects the patient from damage to others but protects the staff. Perhaps discussion should not only focus on concrete methods of control but should look also at the emotional aspects which have led to this physical expedient. Any discussion of seclusion must place emphasis on helping nurses in this difficult situation which few of us would be willing or able to tackle.

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Is dexamethasone deleterious in severe malaria?

SIR,—Dr D A Warrell and others (4 December, p 1652) suggest that I may have misconstrued (6 November, p 1357) some of the facts in their paper.[1]

In denying that dexamethasone may prolong life for 20-30 hours, however, they seem to contradict themselves. Perhaps part of the confusion is their use of two terms: loss of consciousness, which was not defined but apparently derived from histories taken from relatives, and coma, unrousable unconsciousness—the admission point into the dexamethasone/placebo trial. Thus the average fatal case in the dexamethasone group (placebo group) became unconscious 8·4 (15·5) hours before quinine was started, went into coma, was started on dexamethasone (placebo) 17·4 (23·0) hours after losing consciousness, and died 70·7 (48·1) hours after losing consciousness. From the entry point into the trial (coma, unrousable unconsciousness) the first placebo patient died in 12·75 hours, and all were dead within 40·75 hours. The dexamethasone deaths occurred between 31 and 117·5 hours, which according to the authors was a significant difference. Do they now attribute this significance to a small and possibly unreal difference—seven hours—in the onset of quinine treatment relative to the imprecise "loss of consciousness?"

Dr Warrell and others imply that the dose of dexamethasone was not large, but that the 128 kg patient was misleading. At least this patient makes the dexamethasone dosage of 10 mg every six hours regardless of weight seem more modest. In fact a 50 kg man would have been given about 600 mg of prednisone equivalent over a 42 hour period. Dr Warrell and others point out that tissue concentrations persist for 15 hours after ceasing treatment, but during these 15 hours three patients died. What is known of the consequences of the abrupt cessation of massive dose steroid treatment in patients moribund with an illness liable to terminate in shock (algid malaria)?

Dr Warrell and others believe that the side effects are especially worrying, particularly in a district hospital. But they used huge doses of steroids in an intensive care unit. A district medical officer might use a fifth of their dose with a different expectation of side effects. Are the authors convinced that these side effects—aspiration pneumonia, "late" gastrointestinal haemorrhages, bed sores, convulsions, and psychoses—were all due to the 48 hour steroid regimen and not, at least in part, to its withdrawal?

In explaining the low parasite counts Dr Warrell and others say that it is "well known that in cerebral malaria scanty peripheral parasitaemia may be associated with sequestration of parasitised cells in deep vascular beds." *Plasmodium falciparum* parasitised red cells are always sequestrated in deep vascular beds. The parasite spends 50% or more of its 48 hour life span, from large ring, young schizont to mature schizont, merozoite and minute ring, sequestrated in deep vascular beds. Characteristically it is only the ring forms (trophozoites) aged from 6 to 24 hours or so that are seen in the peripheral blood. There is a possibility, perhaps because of a single brood infection, that the peripheral film may be transiently even devoid of parasites but it is amazing how rarely this has been convincingly documented in severe malaria. Multiple broods are the rule, and their presence in the peripheral circulation immediately before treatment—the initial parasite count—is the most reliable guide in prognosis. In Malaya[2] a gradual increase in mortality was observed over progressive increases in the initial parasite count: three of 2251 patients dying with counts below 25 000/μl, 11 of 974 with counts of 25 000-100 000/μl; 12 of 147 with counts of 100 000-250 000/μl, 14 of 46 with counts of 250 000-500 000/μl, and 17 of 32 with counts over 500 000/μl. Most of the patients in the Bangkok trial seem to have started antimalarial drugs before reaching hospital so that Dr Warrell and others presumably used an intratreatment count in determining seriousness of infection and assessing the success of their randomisation. A count taken during or after treatment may have little if any prognostic value. A patient presenting with an initial count of 500 000/μl may still die even though with treatment the counts during and after treatment fall steadily to zero.

It is a pity that although a multitude of tests were performed to assess the comparability of the two groups in terms of prognosis the single most valuable prognostic indicator was not used. If it had been the trial might have been much smaller, restricted to patients with initial parasite counts greater than 250 000/μl, and with the certainty that the groups were prognostically equivalent.

Dr Warrell and others propose mandatory lumbar puncture for all patients comatose with severe malaria, quoting a patient with coincidental cryptococcal meningitis. Most doctors working in the tropics will have encountered and recognised situations such as this but they are usually alerted by clinical experience and suspicion rather than by a blanket of investigations. At the Kenyatta National Hospital we are looking after a boy at the moment who presented with kala-azar, *Escherichia coli* septicaemia, and tuberculous meningitis, but this is hardly grounds for recommending mandatory lumbar puncture in kala-azar. With the same logic lumbar puncture could be proposed as mandatory in other conditions such as diabetic coma. The consequences do not need describing.

It does seem that this trial of steroids has been too large. Nine of the 50 placebo patients died, six of these from pulmonary oedema and possibly two others from septicaemia. Clearly an opportunity for steroids to affect mortality was in the reduction of pulmonary oedema. That they might do this is suggested by the death of only three of the 50 dexamethasone patients from pulmonary oedema, quite possibly a chance result, but if the *initial* parasite count only had been used to set up a smaller trial with the patients at greatest risk then this doubt might well have been answered.

I hope that Dr Warrell and others can see that their trial has raised the possibility that steroids may be useful in severe (initial pretreatment parasite count over 250 000/μl) malaria, both by prolonging life, thus allowing time for effective chemotherapy, and by combating pulmonary oedema. Could they not put this to the test this time starting steroids as soon as the seriousness of the situation has been realised, in a moderate dose, and for a reasonable period of time? Field and Shute[2] described a parasite count of 2·8 million/μl as almost incredible, but recently[3] a patient was seen in England with a parasite count of 3·5 million/μl. Could it be that he tolerated this enormous parasitaemia and did not succumb earlier with a more usually fatal parasite count because he was on steroids for his asthma?

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Chemoprophylaxis of malaria in Africa

SIR,—Dr G H Rée suggests that it was inappropriate for his patient to have been taking Maloprim (pyrimethamine and dapsone) as malaria prophylaxis in the area where he lived (12 February, p 562). The patient was resident in Okapa, which lies at an altitude of about 1800m in the highlands of Papua New Guinea. Effective transmission of malaria may occur at altitudes of up to 2100m, although the mosquitoes become less efficient at high altitude. The penetration of malaria into the highlands corresponds with the building of roads (Sharp P. Fifteenth annual symposium of the medical society of Papua New Guinea, Goroka, Papua New Guinea, 7 September, 1979), and Okapa has good road connections with the main highway to the coast. The local people regularly travel to and