currence is common even after cure with plaster of paris boots with a rubber rocker.

May I emphasise that the diabetic ulcer is caused, and relapses, because the tissues are waterlogged and respond badly to trauma. The prime object of treatment should be to dry out the foot and keep it dry. This can be achieved quickly and cheaply in the patient's home. The affected foot is raised to the maximum tolerated height above heart level (vertical leg drainage regimen).¹⁻³ This leads to rigorous dehydration of the foot, and the ulcer heals quickly. This method is especially successful where there is deep sepsis and the presence of a sequestrum. The latter is extruded as part of the healing process.

The main advantage of the vertical leg drainage regimen is the prevention of relapse during the rest of the patient's life. At the first sign of swelling the patient lies down for a few hours with the leg raised as nearly vertical as possible. Apart from a few cases¹⁻³ the regimen is justified by the results in the leg ulcer clinic at Hammersmith Hospital during 1959-71.

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¹ Bourne IHJ. Treatment of gravitational ulcers and oedema of the lower limbs by postural drainage. London: University of London, 1972:236. MD Thesis.

² Bourne IHJ. Vertical leg drainage of oedema in treatment of leg ulcers. *Br Med J* 1974;ii:581.

³ Bourne IHJ. Results of treatment of swollen legs by vertical leg drainage recorded by serial displacement volumetry and measurement of leg circumferences. *Folia angiologica* 1975;XXIII:228.

Treatment of bacterial meningitis

SIR,—Professor H Lambert (5 March, p 741) highlights the difficulties in choosing antibiotics for treating *Haemophilus influenzae* meningitis. He also suggests that the newer cephalosporins are important additions to the drugs available for Gram negative bacterial meningitis. I would like to draw your attention to one of the drawbacks of using cefuroxime, which occurred when this drug failed to cure *H influenzae* meningitis in an infant using the optimal dosage and regimen.¹

A 12 month old boy was admitted to our general paediatric ward with a three day history of vomiting, drowsiness, refusal of feeds, and fever. He had been treated by the family doctor with penicillin V 125 mg by mouth four times daily for what was thought to be an upper respiratory tract infection. On admission to hospital he was lethargic and feverish with a temperature of 38.5°C. There was no clinical evidence of meningitis. A lower respiratory tract infection was clinically diagnosed, though not confirmed radiologically. In view of the child's age and clinical presentation a lumbar puncture was performed; the cerebrospinal fluid obtained was normal on microscopy and culture. Throat swab, blood culture, and urine culture were also sterile.

Treatment was started with ampicillin 125 mg intramuscularly every six hours without clinical improvement. Another blood culture taken on day three was again sterile. Ampicillin was stopped after four days of treatment, and as the fever continued and there was no localising sign clinically intravenous cefuroxime was substituted at a daily dose of 80 mg/kg. Another blood culture taken on day five was once again sterile. After five days of intravenous cefuroxime treatment there was no clinical improvement and the patient was still feverish and generally unwell. Treatment was stopped and another blood culture was taken (fourth blood culture). Twenty hours after cefuroxime was stopped the patient was still feverish with a temperature of 39.6°C. A second lumbar puncture was performed and mildly turbid cerebrospinal fluid was obtained. Microscopy

showed pleocytosis, and Gram staining showed numerous Gram negative rods. Culture of the cerebrospinal fluid subsequently confirmed the organism to be *Haemophilus influenzae* type b. On the same day that the second cerebrospinal fluid specimen was obtained the fourth blood culture (taken soon after cefuroxime was stopped) had also grown the same *Haemophilus influenzae* type b. Chloramphenicol 250 mg four times a day, given at first intravenously and subsequently orally, was started when the report of the microscopy of the cerebrospinal fluid was known. A swift clinical response was seen, and after 30 hours the patient was well, without fever, and feeding normally. The boy made a smooth, uneventful recovery.

At a review in the outpatient department four weeks after discharge no neurological deficit was seen and the parents described the child as being "back to normal." What worried us most was the possibility of this infant having developed bacteraemia and meningitis while being treated with cefuroxime. The dosage used was in accordance with the data sheet instructions. The *Haemophilus influenzae* isolated was also sensitive to cefuroxime *in vitro*. The cefuroxime used was not out of date. Collection of blood and bacteriological techniques were performed by standard methods. It was most unlikely that the procedures undertaken during his treatment—namely, lumbar puncture, venepunctures, and infusion cannula—could have been responsible for the septicaemia.

I suggest that when cefuroxime is used in the management of bacterial meningitis this may have to be given in much higher doses than those currently recommended.

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¹ Bailey B, compiler. Cefuroxime dose in infants. *Data sheet compendium*. London: Datapharm Publications, 1981:451.

Intraperitoneal hydrocortisone in eosinophilic peritonitis

SIR,—Dr A C T Leung and others (5 March, p 766) report a case of eosinophilic peritonitis associated with continuous ambulatory peritoneal dialysis which responded to treatment with intraperitoneal hydrocortisone.

The increasing number of patients treated by continuous ambulatory peritoneal dialysis has resulted in the reappearance of the syndrome of eosinophilic peritonitis, which was first described 15 years ago.¹ Most workers accept that this syndrome is benign, asymptomatic, resolves spontaneously after a variable time, and does not require any treatment.¹⁻⁴ Some authors have even used the term "peritoneal fluid eosinophilia" to describe it.⁶ The factor(s) responsible for this eosinophilia and its clinical importance remain unknown.

The patient described by Dr Leung and others had an allergic history, but there was no differential white cell count in the effluent at an early stage. Also there was an initial improvement in white blood cell count when no drugs were used, and only after the first relapse was intraperitoneal hydrocortisone started.

In our unit we have seen dialysate eosinophilia along with peripheral eosinophilia in two patients on continuous ambulatory peritoneal dialysis. Neither of these patients had a previous allergic history. When peritoneal fluid eosinophilia was diagnosed no treatment was given. In the first case the episode cleared spontaneously after seven days. The second patient had two relapses on the 10th and 35th days respectively (figure). Finally, the con-

Percentage of eosinophils in peritoneal dialysis fluid.

dition improved without any treatment and no relapse occurred during six months' follow up.

We believe that no treatment is needed in peritoneal fluid eosinophilia even if it reoccurs and that hydrocortisone should be given with caution considering its unknown effects on phagocytosis or the bactericidal action of the peritoneum itself.

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¹ Lee SL, Schoen I. Eosinophilia of peritoneal fluid and peripheral blood associated with chronic peritoneal dialysis. *Am J Clin Pathol* 1967;47:638-9.

² Gokal R, Ramos JM, Ward MK, Kerr DNA. "Eosinophilic" peritonitis in continuous ambulatory peritoneal dialysis (CAPD). *Clin Nephrol* 1981;15:328-30.

³ Humayun HM, Ing TS, Daugirdas JT, et al. Peritoneal fluid eosinophilia in patients undergoing maintenance peritoneal dialysis. *Arch Int Med* 1981;141:1172-3.

⁴ Steiner RW. Clinical observations on the pathogenesis of peritoneal dialysate eosinophilia. *Peritoneal Dialysis Bulletin* 1982;2:118-9.

⁵ Vas SI. Peritonitis during CAPD: A mixed bag. *Peritoneal Dialysis Bulletin* 1981;1:47-9.

Smoking, lung function, and body weight

SIR,—Dr B Nemery and others (22 January, p 249) have shown in a sample of steel workers in Belgium that smokers had significantly lower values of lung function, as measured by forced expiratory volume in one second/vital capacity (FEV₁/VC), than ex-smokers and asymptomatic non-smokers. In table II they divided these smokers according to severity of airflow obstruction into smokers without airflow obstruction (FEV₁/VC > 66.6%) and smokers with airflow obstruction (FEV₁/VC < 66.6%). The former group had the same mean ratio (73.4%) as asymptomatic non-smokers (73.9%). From this observation we might infer that lung function is unaffected among smokers in the absence of severe airflow obstruction. This is not so.

In our study of steelworkers in South Wales¹ we examined the merits of three lung function indices: forced expiratory volume in one second (FEV₁), forced vital capacity (FVC), and forced expiratory volume in one second/forced vital capacity (FEV₁/FVC). Of these FEV₁ proved the most sensitive. A heavy smoker or a bronchitic may not make an all out effort to expel the air from his lungs during measurement of FVC for fear of coughing. This underestimates the FVC and for this reason FEV₁/FVC is not as reliable a measure of impaired lung function as FEV₁. The trend of lung function indices by smoking habits in the South Wales study² clearly showed the greater sensitivity of FEV₁ compared with FVC.

and FEV_1/FVC . In the figure the results, adjusted for age and height, are expressed relative to baseline values of the never smoked group for FEV_1 , FVC, and FEV_1/FVC as 100%. The index FEV_1 shows the steepest decline with amount smoked. In contrast, the ratio FEV_1/FVC is a misleading indicator as it fails to show deterioration of lung function for moderate, heavy, and very heavy smokers.

Relation of lung function indices to smoking habits in non-smokers, ex-smokers who have not smoked for eight or more years, and smokers by number of cigarettes smoked a day. Baseline values of non-smokers are taken as 100% for the indices FEV_1 , FVC, and FEV_1/FVC .

We suggest that at least two indices (FEV_1 , and FEV_1/FVC) should be reported as a matter of routine for all comparisons between groups. Both decline with age but only FEV_1 is positively correlated with height. Appropriate adjustments for age and height can be easily made.²

We are fairly confident that the subgroup of smokers without airflow obstruction studied by Dr Nemery and others would have been found to have lower FEV_1 values than the asymptomatic non-smokers. Also the smokers without airflow obstruction were lighter by about 2 kg, adjusting for height. This difference, although appreciable and in the expected direction, is not significant because of small numbers.

We agree entirely with their concluding statement: "From a practical point of view loss of excess weight in middle aged smokers should not always be regarded as favourable, since it could point to deteriorating lung function and chronic obstructive lung disease." To this we would add that it is much better to be an overweight non-smoker than to be an underweight smoker.³

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¹ Lowe CR, Pelmear PL, Campbell H, et al. Bronchitis in two integrated steel works. *Br J Prev Soc Med* 1968;22:1-11.

² Khosla T. Indices of ventilatory measurements. *Br J Prev Soc Med* 1971;25:203-9.

³ Khosla T. Obesity smoking and health. *Community Medicine* 1979;1:221-8.

Smoking in Papua New Guinea

SIR,—As Dr Euan M Scrimgeour and Mr Damien Jolley write (30 April, p 1414) there are at the moment no constraints on the marketing of tobacco in Papua New Guinea. This is likely to change, however, following a recent meeting in Port Moresby organised by the Institute of Medical Research, the anti-smoking committee of Papua New Guinea's medical society, and the International Union against Cancer. At the meeting the secretary for health announced new antismoking laws

that should be introduced in June. All the indications are that the new laws will be passed.

The legislation, drafted by the Minister for Health with expert help from the Institute of Medical Research, has the full backing of key ministers. The proposals are specific. Firstly, all advertising of tobacco products will be prohibited. This includes newspapers, magazines, radio, films, and outdoor advertising signs. Secondly, all cigarette packets must be marked with a universal warning symbol and a text pointing out the risks in the three main languages: Pidgin, Motu, and English. The text proposed is: "*Sapos yu smok long olgeta dei bai yu kisim bigpela sik* (if you smoke every day then you will suffer serious illness)." Thirdly, sale will be banned to minors. Fourthly, the health ministry will restrict smoking in public places such as hospitals, restaurants, places of entertainment, and public transport.

At the meeting further recommendations were put to the minister: sports sponsorship should be banned entirely, and all cigarettes should contain less than 15 mg of tar and less than 1 mg of nicotine. It was proposed to ban all duty free imports; and, finally, no loose cigarettes should be sold or given in place of small change. This is common practice that helps establish the smoking habit, especially among children.

The second thrust of the powerful antismoking group who organised the meeting is in public information and education. This is being aided by a responsible and helpful press, whose papers reach the smallest villages; a national radio network (in a village with a trade store someone will have a radio); and an established system of yearly inservice training for teachers. The need is there. Last year, *New Nation* magazine ran an article on the dangers of smoking and received an unusual volume of correspondence. "I was horrified. I had no idea smoking did such terrible things." Schoolchildren at Wabag High School wrote in to say they were campaigning to have the sale of cigarettes stopped in the school canteen. Even the educated in towns have little idea cigarettes are dangerous. At a recent forum at the university, most students had not heard of lung cancer. The few who had were surprised to learn that Western medicine had no effective cure.

How is the cigarette industry retaliating? Dr Nigel Gray, director of the International Union Against Cancer, warned that it would appear, well funded and organised, in a bid to prevent legislation and offer a voluntary code that would not work effectively. Dr Michael Alpers, director of the Institute of Medical Research, and a driving force behind the new laws, thinks that the government's commitment to legislation is so strong that it will not yield to multinational business pressure. The tobacco salesman's arguments have been preempted. "Brand changing," an excuse used by companies, would not require hoardings in villages that had never seen a manufactured cigarette before. "Loss of employment" would affect only 702 people, all low wage earners. Professor Jackson, director of the Institute of Applied Social and Economic Research, stated that total abolition of the industry would cause no great hardship. In 1981 money earned by local production was less than a fifth of the value of imports. Twenty eight million kina (one kina is about 80p) was earned in excise, import duty, and employees' tax, but this income will decline gradually as sales decrease after legislation, allowing time for adaptation. As was suggested at the seminar, tax on each packet can always be increased.

Jean Kekedo, the ombudsman with a reputation that causes businessmen to quake, summed up the current mood here: "Papua New Guineans were a healthier people until the ways of the West got hold of them. Now they are slaves to tobacco . . . but now I feel that the tide may be ready to turn."

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Medical sense and nonsense in biography

SIR,—Mr Milo Keynes is correct in emphasising that a medical perspective is often helpful in literary analysis and biography (26 March, p 1025). It is easy, however, to take this approach too far, and I believe that he has done this in his assertion that Henry James must have used the symptoms of temporal lobe epilepsy as the basis for *The Turn of the Screw*. This medicalisation of literary invention was called "medical materialism" by William James, Henry's brother, the physician and philosopher, in his lecture on "Religion and Neurology."¹ In this he said: "Medical materialism finishes up St Paul by calling his vision on the road to Damascus a discharging lesion of the occipital cortex."

A more likely source of inspiration for the *Turn of the Screw* was the religious and mystical culture in which Henry James was brought up—for example, his father, relaxed and content at the dining table after a good meal, suddenly sensed a presence in the room "raying out from his fetid personality influences fatal to life."² This phenomenon had a permanent effect on his philosophy and life, leading him to Swedenborgianism, and this influenced Henry's upbringing. He too had his own metaphysical problems, notably his "obscure hurt," a "huge comprehensive ache" which prevented his participation in the civil war. This is not to argue that this is the source of *The Turn of the Screw*, only to advise caution in the use of medical materialism.

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¹ James W. *Varieties of religious experience*. London: Fontana, 1960:35.

² Edel L. *The life of Henry James*, vol I. London: Peregrine, 1977:29.

SIR,—Mr Milo Keynes (26 March, p 1023) emphasises the need for reliable medical facts in biographies. It is quite clear that a variety of health problems which have afflicted senior politicians have appreciably affected their judgments at the time.¹ But much meticulous research may be needed in order to reveal the salient medical facts and how they affected decision making. For the research to be really meaningful the biographer may need to be medically qualified as well as conversant with the relevant political events. This was so in the excellent study of Hitler by the Hestons.² But one has to agree with Dr L J Bruce-Chwatt (16 April, p 1281) that the erudite and conscientious historian may be as well qualified as anyone in interpreting vague and equivocal symptoms described many years ago.

I should like to make a strong plea for the possible inclusion in biographies of inherited factors when these might be relevant. In the case of serious late onset genetic disorders the person's fear that he might have inherited such a disease from an affected relative and eventually develop disabling symptoms could well colour a choice of career or the way in which a career is pursued. How, for example, might such knowledge have affected those in the political sphere such as George IV in the case of porphyria³ or Edward Grey in the case of familial (? recessive) retinopathy?⁴ Recent evidence suggests that the neuro-degenerative disorder which affected Delius, resulting in paralysis and blindness, may also