autoantibody to smooth muscle antigen (S.M.A.). Autoantibodies have not previously been reported in this disease.

A 3½-year-old girl was admitted to hospital with a six-months history of cough, exertional dyspnoea and cyanosis, and severe weight loss. On examination she was apyrexial, emaciated, and had marked intercostal indrawing but no added breath sounds. Chest x-rays showed a fine general mottling of both lung fields. Repeated sputum cultures for tubercle bacilli were negative, as was the Mantoux test. The haematological findings, including differential white cell counts and blood and bone marrow films, were normal. The serum aspartate aminotransferase (SGOT) level (54 units) was slightly elevated for the laboratory technique used and, on one occasion only, she had a raised alkaline phosphatase level (62 King-Armstrong units). All the other liver function tests were normal. Paper electrophoresis of the serum showed only a slightly raised α_2 -globulin level. However, estimation of the individual immunoglobulin levels showed a low IgA (<40 mg/100 ml) with normal IgM (248 mg/100 ml) and IgG (920 mg/100 ml) levels. Her serum was screened for autoantibodies by the recommended immunofluorescence technique³ and there was a strong reaction(+++) with S.M.A. A lung biopsy showed the classic histological appearance of alveolar proteinosis. Immunofluorescence studies of frozen tissue showed no abnormal deposition of immunoglobulins in the lung, nor could autoreactive lung-specific antibodies be demonstrated in her serum. Despite repeated bronchial lavage her condition deteriorated rapidly and she died 10 months after the onset of the illness. Permission for necropsy was refused.

This case demonstrates the two important (and possibly related) differences between the adult and childhood forms of alveolar proteinosis: (1) In children the disease has a strikingly more serious prognosis. Of the 23 patients reviewed by Colón, the majority died in less than one year, and by the second year the mortality was 100%. In Davidson's⁴ review of 139 patients (135 of whom were adults) only 38 died from alveolar proteinosis or its complications, many living for several years and 34 recovering spontaneously. (2) In children the disease is associated with immunological abnormalities. In Davidson's comprehensive review of the adults no immunological abnormalities are reported, whereas in at least 13 out of the 24 childhood cases reported there is evidence of disordered immunity.

Interestingly, a low serum IgA level was the most consistent immunoglobulin abnormality found by Colón. As IgA is responsible for the local protection of mucus-secreting epithelial surfaces it is possible that these children have a lowered resistance to a normally innocuous organism, possibly a virus, which provokes the accumulation of pneumocytes and the excessive surfactant production which characterizes alveolar proteinosis.

Low serum IgA levels are also found in some cases of coeliac disease.⁵ Though no association between coeliac disease and alveolar proteinosis has been reported, in the majority of childhood cases there is very severe weight loss, usually associated with chronic diarrhoea. These symptoms are infrequent in the adult cases.

The presence of antibody to S.M.A. is particularly interesting in view of the recent work by Farrow *et al.*⁶ on the nature of S.M.A. They have shown it to be situated on microfilaments just beneath the surface membrane of certain cells. They suggest that the presence of serum antibodies to S.M.A. in patients with liver disease is caused by viral damage to the liver cell exposing the

microfilaments, thus allowing the (S.M.A.) antigen to become immunogenic. Since these microfilaments have been observed in fetal lung tissue, it is possible that a similar process could induce the production of S.M.A. in some lung diseases. In support of this, such antibodies have been demonstrated in two patients with idiopathic pulmonary disease.^{7,8}

The two-year mortality in children with alveolar proteinosis is 100% and in adults it is less than 20%. It is possible that this is due to the differing immunological status of the two populations, that of the affected children being abnormal.—I am, etc.,

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- 2 Colón, A. R., Lawrence, R. D., Mills, S. D., and O'Connell, E. J., *American Journal of Diseases of Children*, 1971, 121, 481.
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- 6 Farrow, L. J., Holborow, E. J., and Brighton, W. D., *Nature New Biology*, 1971, 232, 186.
- 7 Lancaster Smith, M. J., Benson, M. K., and Strickland, I. D., *Lancet*, 1971, 1, 473.
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Cancer and the Patient

SIR,—I greatly enjoyed and endorse Mr. Charles Wright's Personal View (6 October, p. 45) that, as in many other social situations and problems, we lag behind the United States in free, uninhibited communication. I am persuaded also that a principal barrier to good doctor/patient relations in Britain is often a lamentable failure on the part of doctors to take the time and trouble to explain in a humane and intelligible way what is wrong, what is to be done, and what it is likely that the future holds.

I have, nonetheless, a slight reservation regarding cancer. This frightening, primitive word seems to have imbued such a nightmarish image that many patients are unable to endure it, and so there are cases in which a tactful little charade is played out by doctor, patient, and family, all well aware that the illness is cancer, yet managing to diminish emotional suffering by refraining from referring to it in direct terms. It is a pity that we do not have readily available a less horrifying name; maybe "prolifera" would do.—I am, etc.,

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SIR,—I was very interested in Mr. Charles Wright's Personal View (6 October, p. 45) and in the ensuing correspondence. During the whole of my active professional life I was particularly interested in the management of cancer—in my case, of the ear, nose, and throat—and for 25 years conducted a combined follow up clinic with my radiotherapy colleague. This long association enabled us to make an accurate long term assessment of the patient's reactions to his disease and its treatment.

I would entirely agree with Mr. R. M. Kirk (20 October, p. 164) that, at any rate up till now, the vast majority of British

patients do not wish to be told the diagnosis, and this attitude was well demonstrated over the years in the combined follow-up clinic. To anyone but the most unintelligent radiotherapy means only one thing, but nothing was ever said for some five or six years, after which a number of patients discussed the matter quite openly. By that time they realized that their chances of permanent recovery were good. In a clinic like ours the doctor/patient relationship was very close and friendly and I cannot remember any instance of a patient suggesting that he should have been told exactly what was the matter with him at the start.

My policy therefore was not to tell the patient unless he asked, and this I taught my students. On the other hand I also emphasized that, if a patient put the direct question, he must be told the truth. To lie to a patient is quite unjustifiable and morally wrong. It will destroy the confidence of both patient and relatives, though a slightly optimistic view of the prognosis could in some cases be permissible. I am surprised at the suggestion that British consultants habitually lie to their cancer patients, and do not believe that this is so. In my experience "going to pieces" very rarely occurred. There were two suicides in some 300 cases. One was a patient who had asked and been told the diagnosis and the other one who had not.

I am not submitting that the North American custom may not, in the long run, be right, but it will take the time to become accepted in this country and it would be a dangerous mistake to adopt it too suddenly.—I am, etc.,

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Mucosal Ulceration in Coeliac Disease

SIR,—We note the comments of Drs. P. F. Fitzpatrick and A. G. MacIver (1 September, p. 502) on our paper (28 July, p. 212). The diagnosis of coeliac disease was made in our patient as subtotal villous atrophy was present in two separate jejunal biopsy specimens taken at sites different from the ulcerated areas.

The differentiation of coeliac disease with mucosal ulceration from ulcerative jejunitis may indeed be difficult; other authors have classified the same case in different ways.^{1,2} We believe the term ulcerative jejunitis should include those cases in which the villous architecture of the jejunum at sites away from the ulcerated areas is normal; however, when subtotal villous atrophy is present a diagnosis of coeliac disease with ulceration should be made. There will remain cases in which the non-ulcerated jejunal mucosa shows partial villous atrophy or only patchy villous changes; the classification of these cases is difficult. A gluten challenge after healing has occurred may be of help, but in our patient we were unwilling to carry this out in view of the severity of the initial illness. With regard to the failure to respond to gluten withdrawal, this has been noted previously in coeliac disease with³ and without³ mucosal ulceration. Not only may the addition of corticosteroids initiate a response,³ but corticosteroids alone may produce partial or complete healing.⁴

The possibility of Crohn's disease was considered, but despite an extensive search

for granulomata in serial sections none were found. Further, we are not aware of sub-total villous atrophy occurring in those parts of the jejunum otherwise uninvolved in small-intestinal Crohn's disease.—We are, etc.,

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¹ Goulston, K. J., Skyring, A. P., and McGovern, V. J., *Australasian Annals of Medicine*, 1965, 14, 57.

² Bayless, T. M., Kapelowitz, R. F., Shelley, W. M., Ballinger, W. F., and Hendrix, T., *New England Journal of Medicine*, 1967, 276, 996.

³ Pink, I. J., and Creamer, B., *Lancet*, 1967, 1, 300.

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Seat Belts

SIR,—As the proposer of the successful motion at the Annual Representative Meeting at Folkestone in June on the need for improvement in the design of seat belts and the advisability of their compulsory use in cars (*Supplement*, 16 June, p. 94), I was gratified to see the letter from Mr. E. S. R. Hughes of Melbourne (29 September, p. 695) on the experience in Australia of the lowered mortality and reduced incidence of injuries resulting from their legislation.

How right Professor W. Gisane (30 June, p. 772) was in commending the action of the Australian legislature in introducing compulsory belt-wear! Let us hope that our own legislators will be as discerning and perspicacious.—I am, etc.,

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Hypotension in Tetanus

SIR,—I was interested to read the article by Dr. J. L. Corbett and others (25 August, p. 423), and would like to make two suggestions:

(1) Rather than leave the patients with an alarmingly low blood pressure for 15-20 minutes, to try immediate infusion of 500-1,000 ml of plasma or other fluid. A heart pumping at a control venous pressure of only 1.5 mm Hg as described in case 1 can be expected to respond to increased filling pressure with an increase in cardiac output and blood pressure, because of Starling's law of the heart. Increasing the blood volume is more effective than head-down tilt, and is surely safer than giving alpha-, beta-, or ganglion-blocking drugs to patients with hypotension. Patients on mechanical ventilation are likely to need a greater than normal blood volume, especially if autonomic function is impaired.

(2) Why not keep the patients sedated as well as paralysed? Reflex depression with barbiturates, diazepam, droperidol, or narcotics may well prevent these episodes of hypertension, which are quite common during light general anaesthesia. Perhaps normal subjects, without tetanus, would react in a similar way to prolonged curarization and mechanical ventilation as these three patients.—I am, etc.,

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Hypergastrinaemia in Rheumatoid Arthritis

SIR,—We read with interest Dr. K. J. Fraser's reply (29 September, p. 696) to our preliminary communication (30 June, p. 752) concerning elevated concentrations of plasma gastrin in rheumatoid arthritis. Dr. Fraser's initial comments on our lack of awareness of the literature on the incidence of achlorhydria in rheumatoid arthritis are perhaps unfortunate as no reference is made to the work of Armstrong,¹ who demonstrated no increase in incidence of parietal cell autoantibody in rheumatoid arthritis. He also demonstrated the organ-specificity of the antibody by means of gastric function studies. The omission of reference to this work carried out in this centre is out of step with Dr. Fraser's kindness elsewhere in referring to our work in the field of Sjögren's syndrome. We would like to point out in addition that the increased incidence of hypergastrinaemia may prove to be of much more fundamental importance if it is not due to gastric atrophy.

Our preliminary studies would suggest normal acid secretory status in rheumatoid arthritis in the presence of hypergastrinaemia, and recently have shown that the induction of adjuvant arthritis in rats is associated with a concomitant elevation of fasting plasma immunoreactive gastrin.² The significance of these observations remains unclear to date, but it seems unlikely that they merely reflect the presence of atrophic gastritis.—We are, etc.,

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¹ Armstrong, E. M., *Annals of the Rheumatic Diseases*, 1967, 26, 499.

² Rooney, P. J., Dick, W. C., Buchanan, K. D., and Imrie, R. C., *Nature New Biology*. In press.

Making Hospital Geriatrics Work

SIR,—Under this heading Drs. H. M. Hodgkinson and P. M. Jefferys (2 December, 1972, p. 536) discussed the work of a new geriatric department and advocated a policy of high turnover and no waiting list. They suggested that this policy was a matter of choice by the geriatricians.

Experience at Greenwich over recent years indicated that the work of a geriatric department is not quite such a matter of choice as Drs. Hodgkinson and Jefferys state. The figure shows the changes that have occurred over a 10-year period. From 1963 to 1968 the service at Greenwich was very similar to that at the Northwick Park unit with no waiting list, direct admission on general practitioner or Emergency Bed Service request, and high turnover (five patients per bed yearly), and the wait for transfer from other wards and hospitals was less than a month. At that time the work of the department was in equilibrium with the needs of the area served.

In 1968 22 beds were closed out of a total of 200 (11%) and the admission rate has fallen from 1,000 per year to 450—a drop of 55%. This disproportionate fall stems from a simple error. When the beds were closed it was argued that an 11% reduction in beds would act as a stimulus to the efficiency of an over-bedded department (200 beds for 18,000 over-65 population). What

was overlooked was that the department had taken on a high long-stay commitment to help out other agencies under stress. There were 150 long-stay patients, including a 33-bed psychogeriatric ward with demented patients who in many parts of the country would have been placed in a psychiatric hospital, and 26 patients had been accepted from welfare accommodation for whom Greenwich would not normally have been the responsible geriatric hospital. There were therefore only 50 admission and rehabilitation beds through which 1,000 admissions were passing each year. Within these 50 beds were about 25 very-high-turnover beds and about 25 slower-stream beds with a stay of three months or more before discharge, death, or transfer to long-stay accommodation. Clearly none of the long-stay or slower-stream beds could be closed and the department was left with virtually no high turnover beds.

My choice in this situation was to accept preferentially those patients whose stay was likely to be short and to give low priority to those referrals who were likely to need a prolonged stay. Nevertheless, the department has functioned over the last five years as a low-turnover/long-waiting-list unit. In spite of not now taking ambulant demented patients and in spite of now adopting a "legalistic" approach to the geographical catchment area the number of long-stay patients is now 141 (the total bed number having fallen this year to 171). There are signs, however, that a new equilibrium is being established since the number of patients in other hospital beds waiting for transfer to the department has fallen from a maximum of 70 in 1972 to 20 in September 1973. During the whole period the staffing (medical, nursing, and paramedical) has remained substantially the same in numbers but, as would be expected, morale has plummeted.

The suggestion by Drs. Hodgkinson and Jefferys that the work of a geriatric department is a matter of choice therefore appears to be an over-simplification. Given positive attitudes and adequate staffing, there appears to be a critical number of beds in any area with which a high-turnover/no-waiting-list service can be given. An important lesson that comes out of the Greenwich experience is that the total number of beds in a geriatric department is a misleading figure. In planning changes the number of admission and rehabilitation beds is a key