capsule is then pushed forward through the biopsy channel until it reaches the third portion of the duodenum (about 10 to 15 cm ahead of the distal tip). After firing the capsule in the usual way it is extubated together with the gastroscope.

We have successfully used this technique in all of our last 30 patients undergoing jejunal biopsy. The whole procedure takes no longer than five to seven minutes, is devoid of complications, and does not require fluoroscopy. In addition it allows concurrent upper gastrointestinal endoscopy to be performed.

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Gastroenteritis in infancy

SIR,—Dr Tony Walsh (19 May, p 1539) considers that the main problem in the home treatment of infants with gastroenteritis is non-compliance with medical advice. After experience in hospital, general practice, and child health clinics, I conclude that much of the trouble lies in the advice commonly given to patients: to give only clear fluids so long as diarrhoea persists.

The infant's reaction to this near starvation, perhaps for several days, can be such that his mother is driven to despair. This ordeal is needless, for it has long been known that it does nothing but good to feed infants who have diarrhoea—provided that thirst is sated first.[1,2]

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[1] Levin S. Reflections on gastroenteritis and its treatment. *J Pediatr* 1958;**52**:227-44.
[2] Levin S. Stool-gazing. *Br Med J* 1958;ii:503-5.

Wilson's disease

SIR,—Dr David Parkes's leading article on Wilson's disease (21 April, p 1180) is a welcome reminder of how important it is to keep this rare but now treatable disease in mind. Perhaps the most grateful patient I ever had was the only one in whom I personally made the diagnosis of Wilson's disease during 30 years' experience of neurology. In this patient, once the diagnosis was considered clinically it took only seconds to see the confirmatory Kayser-Fleischer ring. Treatment with the then relatively new agent penicillamine resulted in complete recovery from a state of severe neurological and psychiatric disability, and this happy state continues more than 20 years later.

I was keen to repeat this experience when I returned to the United Kingdom from the United States in 1965 but to my regret I have not found another patient with Wilson's disease since then, while working in an area which provides the only neurological service for about half a million people. This experience, or rather lack of it, has been shared by my neurological and gastroenterological colleagues. One probable family was identified in the early 1960s. Three children had died of hepatic disease in the 1950s. A fourth member, without clinical evidence of liver disease or neurological disease although he was mentally retarded, was extensively investigated. Although ceruloplasmin and serum copper studies were normal, radioactive copper studies were thought to be in keeping with Wilson's disease and he has been treated with penicillamine ever since. He is still alive in a hospital for physically and mentally handicapped people and has shown no improvement in his mental status. This man, now 29 years old, is therefore the only known case of probable Wilson's disease in the region. The prevalence (not incidence, as stated in the leader) would appear to be very low (two in a million) in this region and even allowing for underdiagnosis this is still far short of the estimate of 30 in a million suggested by Scheinberg.[1] He also suggests (from his own series and from that of Walshe) that the percentage of cases of Wilson's disease presenting with neurological features is close to 40%. The expected number, therefore, in this region with neurological manifestations of Wilson's disease would be six, but in fact none has been identified. Presumably the gene frequency is low in this area, although it has its fair share of other recessive disorders. The estimated prevalence of 30 in a million is based on work largely in East Germany and in Japan, as well as in the United States, and may not be universally applicable.

It would be of interest to have more accurate figures for the rest of the United Kingdom. Such surveys might reawaken the interest of a new generation of neurologists and gastroenterologists and might lead to the uncovering of fresh cases. Diagnosis in cases presenting neurologically should be relatively easy, although slit lamp examination may be required to confirm the Kayser-Fleischer ring. Prevalence studies in such a disease may be very difficult and a search in our own hospital diagnostic coding system produced no fresh confirmed cases, although several suspected ones. Questionnaires sent to neurologists and gastroenterologists through their professional specialist societies could relatively easily give some more accurate estimate.

The initial delineation of this disorder by Wilson and the development of the remarkably effective treatment with chelating agents by Walshe should be a spur to the present generation of younger British neurologists and gastroenterologists.[2] I would be intrigued, however, to discover how many have had as small, or even smaller, a personal experience of this disease as myself.

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[1] Scheinberg IH, Sternlieb I. Wilson's disease. In: Smith LH, ed. *Major problems in internal medicine*. Vol 23. Philadelphia: W B Saunders, 1984.
[2] Walshe JM. Penicillamine, a new oral therapy for Wilson's disease. *Am J Med* 1956;**21**:487-95.

Problems of tuberculosis in decline

SIR,—Dr N W Horne's leading article (28 April, p 1249) cited a report from this department describing two cases of adrenal crisis precipitated by rifampicin.[1] This has prompted us to comment on estimation of cortisol concentrations during antituberculous chemotherapy.

Since submitting that report we have estimated cortisol concentrations in patients before and from three to four weeks after commencing antituberculous chemotherapy. This is the time of maximum enzyme induction by rifampicin. A total of 17 paired basal specimens were obtained. The range for the pretreatment specimens was 210-1031 nmol/l (7·6-37·3 μg/100 ml) and for the second specimens 251-827 nmol/l (9·0-29·9 μg/100 ml) (normal range 130-690 nmol/l (4·7-25 μg/100 ml). All were, therefore, within or above the normal range. Two patients with the lowest levels had tetracosactrin challenge, which showed a normal response.

We have also recently seen one patient who had initially a normal cortisol value of 688 nmol/l (24·9 μg/100 ml); for reasons of non-compliance further specimens were not obtained, but he had no evidence of hypoadrenalism throughout his treatment. After six months of antituberculous chemotherapy, however, which included 450 mg of rifampicin daily, he died from a massive haemoptysis secondary to pulmonary tuberculosis. At postmortem examination the adrenals were macroscopically atrophic, weights at the lower limit of normal, and histologically there were fibrotic changes consistent with old tuberculosis.

In the recent series of deaths occurring in newly notified patients with pulmonary tuberculosis by Humphries *et al* 69% were noted to have occurred within the first month of chemotherapy.[2] These patients were white and mainly elderly with extensive disease. In Ellis and Webb's series of 60 patients, 16 died suddenly and unexpectedly during the early stages of treatment.[3] Although many other factors are implicated, the role of rifampicin in increasing cortisol turnover and inducing adrenal insufficiency in patients with existing adrenal impairment must be considered.

Our results suggest that cortisol values alone are inadequate, and perhaps tetracosactrin challenge is necessary to identify patients with impaired adrenal function who would be unable to increase cortisol production to cope with increased cortisol turnover induced by rifampicin.[4]

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[1] El-Ansary EH, Earis JE. Rifampicin and adrenal crisis. *Br Med J* 1983;**286**:1861-2.
[2] Humphries MJ, Byfield SP, Darbyshire JH, *et al*. Deaths occurring in newly notified patients with pulmonary tuberculosis in England and Wales. *Br J Dis Chest* 1984;**78**:149-58.
[3] Ellis ME, Webb AK. Causes of death in patients admitted to hospital for pulmonary tuberculosis. *Lancet* 1983;i:665-7.
[4] Ohnhaus EE, Park BK. Measurement of urinary 6 β-hydroxycortisol excretion as an in vivo parameter in the clinical assessment of the microsomal enzymes inducing capacity of antipyrine, phenobarbitone and rifampicin. *Eur J Clin Pharmacol* 1979;**15**:139-45.

Necrobacillosis

SIR,—Dr John Moore-Gillon and others' lesson of the week (19 May, p 1526) reminded us of two cases we have seen in the last three years which also showed the classical features of necrobacillosis (Lemierre's syndrome).

The first was a 20 year old West Indian man with a history of a sore throat, cough, chest pain, and a painful swollen left knee. He had been given co-trimoxazole by his general practitioner. On examination he was feverish (39·8 C) and confused with a pulse rate of 154 beats per minute and blood pressure of 110/70 mm Hg. He had inflamed fauces with a yellow exudate but no cervical lymphadenopathy. The left knee was tender with a marked effusion. A chest radiograph showed patchy consolidation of the right lung with cavitation and a right pleural effusion. Laboratory results were haemoglobin concentration 14·4 g/dl, white cell count $18·5 \times 10^9$/l (84% neutrophils), normal concentrations of urea and electrolytes, bilirubin concentration 20 μmol/l (12 mg/l) and alkaline phosphatase activity 118 IU/l. A sickle cell test gave negative results. The sputum was purulent but no organisms were isolated from it. A throat swab grew *Candida* only. A midstream urine specimen contained 100 white cells per ml with no bacterial growth. Purulent fluid aspirated from the left knee was sterile. Antibiotic treatment was started with intravenous benzylpenicillin and flucloxacillin, but because of poor response and increasing pyrexia (42 C) gentamicin and metronidazole were added. On the third day two blood cultures taken on the day of admission grew *Fusobacterium necrophorum*, and the antibiotics