patient was confused, which was attributed to a severe electrolyte imbalance. Repeat x-rays of the chest and abdomen revealed no abnormality, although by now an ill-defined swelling was palpable in the right lumbar area.

At this time the diagnosis was thought to lie between abdominal tuberculosis, repeated internal haemorrhages, appendix abscess, and peritonitis due to bacteroides infection. Laparotomy revealed pus in the right paracolic gutter with adhesions: the right tube was grossly inflamed, while the left tube and appendix were both normal. The pus grew *Staphylococcus aureus* and *Bacteroides* spp. Treatment with clindamycin and metronidazole was started. The patient remained very ill and her weight fell to 32.2 kg. Blood (2.2 l) was transfused to correct the anaemia. By the 19th day the patient was deteriorating, with a swinging pyrexia. A Heaf test was doubtfully positive. Blood culture now grew *Staph aureus*. No malaria parasites could be found. The Widal reaction was negative. Yet another chest x-ray showed clear lung fields and repeated scanning showed no evidence of a sub-phrenic abscess. The fluorescent amoebic antibody test was negative. The patient became critically ill. Apart from cachexia and moderate tenderness in the right hypochondrium pyrexia and tachycardia were the dominant signs. There was no leucocytosis. The Heaf test was now definitely positive, but the fundi were considered normal. Anti-tuberculosis therapy was started with ethambutol, isoniazid, and rifampicin. Just at this time a chest x-ray showed miliary tuberculosis, 46 days after delivery. Recovery was dramatic, the pyrexia and tachycardia settling within eight days. Fourteen weeks later the patient was discharged from the tuberculosis unit fit and well.

In all, just over 100 pathology and radiology reports were filed before the diagnosis was made, when peritoneal biopsy¹ or even possibly salpingectomy would almost certainly have revealed the true state of affairs.

I thank Dr Roger Williams for making the diagnosis and Dr J T Hutchison for curing the patient's tuberculosis. Guy's Hospital Medical School kindly performed the fluorescent amoebic antibody test.

ALAN M SMITH

New Cross Hospital,
Wolverhampton

¹ Das, P, and Shukla, H S, *British Journal of Surgery*, 1976, **63**, 941.

Campylobacter enteritis

SIR,—Dr M B Skirrow (2 July, p 9) is to be congratulated on drawing attention to campylobacters as a cause of enteritis.

Our investigations have led us to believe that the reservoir of the organisms is the wild bird population and that they are transmitted to humans by healthy poultry, meat, and possibly even by other foodstuffs contaminated with bird droppings and inadequately cleaned.

In the United States Smibert¹ isolated campylobacters (*Vibrio fetus* var *intestinalis*), from the intestinal contents of 11 of 15 sparrows, one of nine starlings, one of five pigeons, and one blackbird which he had trapped in and around sheep and cattle barns. He also isolated the organisms from the intestinal contents of sheep, aborted ovine fetuses, the intestinal contents of turkeys with transmissible enteritis, and the bile of chickens with vibronic hepatitis.

We have been informed that vibronic hepatitis in chickens is virtually non-existent in the United Kingdom and our preliminary investigations suggest that the organisms are commonly found in the intestine of healthy birds. Recently we examined the caecal contents of 50 freshly killed and apparently

previously quite healthy chickens and found campylobacters in seven (14%). Vacuum packing could provide one of the conditions necessary for their multiplication.

When a discovery like Dr Skirrow's is made there is always the danger of over-reaction, and it is important for us to keep things in perspective. Chickens are now our biggest source of animal protein. Salmonellas may be isolated from more than 30% of those subsequently offered for sale. We are not complacent and there is more work to be done, but, as Dr Skirrow indicates, it is logical to expect the food hygiene measures which have been advocated for the prevention of salmonellosis to control enteritis caused by campylobacters.

N A SIMMONS
F J GIBBS

Department of Clinical Bacteriology
and Virology,
Guy's Hospital,
London, SE1

¹ Smibert, R M, *American Journal of Veterinary Research*, 1969, **30**, 1437.

"Measles" in previously immunised children

SIR,—Doctors are often bothered by measles-like rashes in children with a history of live measles vaccination. Professor J A Dudgeon has recently (2 July, p 44) stressed the need "to carry out some virological tests to try and determine whether such cases were in fact measles or resulted from other viral infections."

An investigation by Gerike and Sandow has already given an answer.¹ The authors studied serologically 247 cases of presumed vaccine failures. Only 60 cases (24.3%) could be identified as measles, 100 cases (40.5%) showed at least a fourfold increase in the titre of rubella antibodies, and the remaining 87 cases (35.2%) were undiagnosed. Vaccine failures are therefore more rare than some doctors suppose.

W EHRENGUT

Hamburg,
W Germany

¹ Gerike, E, and Sandow, D, *Deutsches Gesundheitswesen*, 1976, **31**, 1949.

Bromocriptine and TRH-induced growth hormone release in anorexia nervosa

SIR,—Thyrotrophin-releasing hormone (TRH) has been shown to stimulate the release of growth hormone (GH) in some patients with anorexia nervosa (AN),¹ in contrast to normal subjects. As bromocriptine lowers the raised plasma GH levels in an oral glucose tolerance test (OGTT) in AN² the effect of the drug on TRH-induced GH release was studied for comparison.

Six female patients aged between 15 and 36 years with typical AN³ and weighing between 50 and 88% of their standard weights were studied. After an overnight fast 500 µg of TRH (Roche) was injected intravenously. Blood samples for GH assay were withdrawn through an indwelling needle immediately before and at intervals of 20, 30, 60, 90, 120, and 180 min after the injection of TRH. The patients were then started on treatment with bromocriptine, initially 2.5 mg daily with food and increasing by 2.5 mg every three days to a maximum of

Plasma GH levels (µg/l) following intravenous injection of TRH (500 µg) in six patients with anorexia nervosa before and during treatment with bromocriptine

Patient	Before treatment		During treatment	
	Basal	Peak	Basal	Peak
1	18	36	15	17
2	13	15.3	6.8	14.1
3	56	72	19	27
4	2.7	6.6	1.6	5
5	17	18	14	25
6	2.2	8.8	9.3	6.2
Mean ± SEM	18.2 ± 8	26.1 ± 10	11 ± 2.6	15.7 ± 3.8

Before treatment paired *t* test showed that the difference between the basal and peak levels is probably significant ($P < 0.05$) but no significant difference was found between the basal levels or peak levels before and during treatment or between basal and peak levels during treatment.

10 mg/day. After 14 days' treatment in hospital without changing the dietary regimen the tests were repeated. Plasma GH was measured by specific radioimmunoassay.⁴

The basal and peak plasma GH levels before and during treatment with bromocriptine are shown in the table. Using the same criteria as Maeda *et al*¹—namely, a twofold or greater increase in plasma GH over the basal level and an increment greater than 5 µg/l—two patients (Nos 1 and 6) showed a rise in plasma GH levels before treatment only. One patient (No 2), however, had a rise in plasma GH concentration while on treatment which did not occur before treatment. In addition, we would normally have considered a rise in plasma GH to have occurred in patients 3 and 4 before treatment and patients 3, 4, and 5 after treatment, all these results implying that any rise in plasma GH in response to TRH is not affected by bromocriptine.

It is unlikely that the dose of bromocriptine, 10 mg/day, was so low that this would account for its failure to depress the response to TRH, since the same dose lowered plasma GH levels significantly during an OGTT in patients with AN.²

These results suggest that bromocriptine does not suppress GH release in AN in whole or in part by a direct action on the somatotroph, since stimuli such as TRH are able to bypass any such potential effect of the drug.

A D B HARROWER
P L YAP
I M NAIRN
J A STRONG

Department of Medicine and
Metabolic Unit,
Western General Hospital,
Edinburgh

¹ Maeda, K, *et al*, *Acta Endocrinologica*, 1976, **81**, 1.
² Harrower, A D B, *et al*, *British Medical Journal*, 1977, **2**, 156.

³ Lundberg, P O, *et al*, *European Journal of Clinical Investigation*, 1972, **2**, 150.

⁴ Schalch, D S, and Parker, M L, *Nature*, 1964, **203**, 1141.

Survival of specialist societies

SIR,—Scientific medical societies have proliferated with increasing specialisation. In order to keep up to date and maintain contacts many doctors have found it essential to belong to six or more societies and the cost of regularly attending scientific meetings seemed reasonable until recently. Now inflation, high taxation, and frozen earnings are making it financially impossible for most to travel long