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CORRESPONDENCE

Correspondents are asked to be brief

Acute Appendicitis and Salmonella Infections

SIR,—We agree with Dr. J. V. Dadswell (24 March, p. 740) that while the true incidence of appendicitis associated with salmonella infection of the bowel is not known, it is probably higher than is generally realized. Now that the summer months are approaching, with increased travel abroad, particularly to Mediterranean resorts, we would like to draw further attention to the combination of appendicitis and salmonella infections, as illustrated by the following case which occurred in June last year.

Two days after returning from holiday in Ibiza Spain, a 24-year-old married woman, eight weeks pregnant, began to complain of abdominal pain, vomiting, and diarrhoea. Within 24 hours the pain localized to the right iliac fossa. She was mildly febrile (temperature 99·2°F (37·3°C), pulse 100/min) and had tenderness and guarding over the appendix. At operation the appendix was mildly inflamed and faecal impaction of the terminal ileum was observed. Appendicectomy was performed. She was discharged from hospital one week after her operation. Later the same day she was readmitted complaining of colicky abdominal pain and diarrhoea. There was no pyrexia. Physical signs of early obstruction were present and conservative treatment was undertaken with intravenous fluids and suction. She did not respond to this regimen, and a laparotomy was performed 36 hours later. At operation the terminal ileum was distended proximally and adherent to an abscess in the appendicular stump region. The adhesions were divided and a peritoneal drain inserted. Bacteriological examination of the pus grew a mixture of *Escherichia coli* and a *Citrobacter* species. Her postoperative course was uneventful and she was discharged home 14 days later. Within two days she was again admitted with a pelvic abscess some 6-7 cm in diameter presenting in the left iliac fossa. This was incised and drained. Bacteriological examination of the pus yielded *Salmonella typhimurium*. This organism was subsequently isolated from her stools. Initial treatment with ampicillin was changed to co-trimoxazole when the results of antibiotic sensitivity tests were known. Her final recovery occurred approximately six weeks after the start of her illness, but not before a further complication in the form of abortion. The *Salm. typhimurium* isolated was phage type U 129, a type frequently found in Spain.

Thus while operation must be carried out if there are clinical indications, it is well to remember that such patents can on occasion have a stormy passage on their way to eventual recovery, particularly with *Salm. typhimurium*.

Within the same month a further case occurred in a 14-year-old Asian boy. He presented with the typical picture of acute appendicitis and at operation an acutely inflamed appendix was removed. At no time was there any diarrhoea or history of diarrhoea. Progress was satisfactory for the first 48 hours after operation, but then a low-grade pyrexia ranging between 99 and 100°F (37·2 and 37·8°C) became evident. By the sixth day the appendicectomy wound was seen to be mildly inflamed and tender. Since he was allergic to penicillin, a course of tetracycline was started. Three days later a small collection of pus localized in the wound. Bacteriological examination of the pus showed *Salm. saint-paul*. The wound slowly dried and healed, his temperature settled, and he was discharged 14 days after his operation. The organism was not subsequently isolated from a stool sample.

In conclusion, may we suggest that in all such cases of suspected appendicitis where the history is suggestive of intestinal infection, or where the postoperative course after appendicectomy is not typical, stools be submitted to the laboratory for bacteriological examination for intestinal pathogens, preferably before antibiotics are prescribed?

We thank Mr. H. B. Young and Mr. W. R. S. Hutchinson, the consultant surgeons, for allowing us to quote the clinical details of their patients

—We are etc.,

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Pulmonary Disease after Amitriptyline Overdosage

SIR,—Drs. A. J. Marshall and K. C. Moore (24 March, p. 716) reported the case of a patient who died from diffuse pulmonary consolidation after ingesting an overdose of amitriptyline. They thought it likely that this was a direct effect of the drug and suggested that the mechanism might be specific inhibition of surfactant production. We do not think it necessary to invoke such specificity of action to explain the findings in their patient. We have previously reported similar complications, albeit with a happier outcome, in a 26-year-old woman who was unconscious from an overdose of quinalbarbitone.[1]

We would suggest that pulmonary consolidation may develop in any unconscious patient in whom there is a prolonged disturbance of the normal pattern of breathing. The absence in such a patient of intermittent deep inspiration may impair the regeneration of the pulmonary surfactant layer,[2] and absorption-collapse develops distal to closed airways in the dependent zones of the lungs. From the account given by Drs. Marshall and Moore, we infer that objective assessment of respiratory function was not made in their patient until the sixth day of admission, when the changes we have described could have resulted in extensive secondary damage to the lungs.