severe, interstitial pneumonitis of undetermined cause. The remaining animals were anaesthetised, systemically perfused with buffered solutions of paraformaldehyde followed by glutaraldehyde, and tissue prepared for examination in 1 μm epoxy cross-sections stained with toluidine-blue. Examination of cortex, cerebellum, spinal cord, sciatic-nerve complex, and gastrocnemius muscle revealed no toxin-related pathological changes.

Thus authentic samples of contaminated oil did not induce neuromuscular disease in a species that is sensitive to most neurotoxins. The relative doses were much larger than the amounts ingested by victims of the Spanish oil syndrome and the exposure was longer. These negative data, coupled with the absence of identified neurotoxic agents in the adulterated oil sample studied, strengthen the view that the neuromuscular sequelae of this disease are attributable to factors other than a direct-acting neurotoxin.

The oil sample was kindly provided by Dr Alberto Portera Sanchez, 1° de Octubre Hospital, Madrid-26, Spain.

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TOXIN A (ENTEROTOXIN) FROM CLOSTRIDIUM DIFFICILE IN ANTIBIOTIC-ASSOCIATED COLITIS

SIR,—Pseudomembranous colitis and other cases of antibiotic-associated colitis are thought to be caused by intestinal overgrowth of *Clostridium difficile*. This organism produces in vitro two potent toxins, toxin A (enterotoxin) and toxin B (cytotoxin).[1-3] The search for toxin B in stool samples is now a routine diagnostic assay in many clinical laboratories. In contrast, toxin A in stool samples of patients (i.e., formation of this toxin in vivo) has not been reported. We have found toxin A activity in the stool of a patient with antibiotic-associated colitis; she also had a raised toxin A serum antibody titre.

A 76-year-old woman with chronic rheumatoid arthritis and a prosthesis of the knee, the origin of an osteitis, had, for a month before the onset of diarrhoea, been treated with cloxacillin, metronidazole, co-trimoxazole, and cefotaxime. Signs of antibiotic-associated colitis (i.e., voluminous, frequent non-haemorrhagic diarrhoea) persisted for 10 days. Endoscopy was not done.

Toxin B was demonstrated in stool filtrates by titration of the cytotoxic effect in cell cultures of human embryonic intestinal cells and the titre was determined as the highest dilution of the filtrate that affected 50% of cells. Cytotoxic activity was in all cases neutralised by an antiserum against *C. sordellii*.[4] Toxin A was determined by injection of 1 ml stool filtrate in a rabbit loop. IgG antibodies to toxins A and B were determined by enzyme-linked immunosorbent assay (ELISA) against purified preparations of the toxin on serum samples diluted 1/500.[5]

C. difficile was isolated on days 4 and 5 of the disease. A stool filtrate on day 5 was strongly positive for toxin A and this effect was neutralised by an antiserum specific for toxin A. In the same sample the cytotoxin assay showed a toxin B titre of 64 000 (table). Serum IgG titres against both toxin A and toxin B were raised (table).

TOXIN A AND B ASSAYS AND ELISA TITRES

	Toxin assays (stool)*		ELISA IgG titre (serum)†	
Day	Toxin A	Toxin B	Toxin A	Toxin B
4	<0·3	4000	..	..
5	1·6	64 000	..	..
9	..	..	35	22
15	<0·3	1000	335	44
17	..	..	570	105
19	<0·3	100	..	..
22	..	..	695	368
24	..	..	675	408

*Rabbit loop assay for toxin A (ml/cm) and cell test for toxin B (titre)
† Expressed as $A_{4\,m}$, × serum dilution; >200 defined as positive.

This is the first toxin-A positive stool filtrate our laboratory has found among 44 stool samples that were positive for toxin B (unpublished). Furthermore, it is the first time that an increased IgG titre has been found against toxin A in antibody studies in 122 sera from thirty-eight patients with prolonged diarrhoea or pseudomembranous colitis. Although an antibody response to toxin B was frequently demonstrated in the third week, no such response to toxin A was recorded among these patients.[5]

These data strongly suggest the formation of toxin A in vivo in this patient. With the methods available, this seems to be a rare finding in *C. difficile* associated diarrhoea. However, it encourages the discussion of the relative role of these (and other?) toxins in the pathogenesis of antibiotic-associated colitis.

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IS ABDOMINAL AUSCULTATION IMPORTANT?

SIR,—Abdominal auscultation is always included in the evaluation of patients with acute gastrointestinal symptoms. Bowel sounds come in an assortment of pitches, volumes, and tones which may suggest several disease processes. These are difficult to classify but most clinicians agree that it is important to ascertain the presence or absence of bowel sounds. In our experience few physicians auscultate the abdomen for more than a minute—indeed, most consider 15 seconds sufficient. Does abdominal auscultation for 15 seconds provide an accurate indication of the presence or absence of bowel sounds?

33 inpatients were auscultated twice daily, 11 on the paediatric surgery service and 22 on the general paediatric service. The patients' ages ranged from 1 month to 17 years (average 5 years). Abdominal auscultation of the right lower quadrant was done for 1 min in the morning before breakfast and in the evening 3 h after dinner.

Abdominal auscultation was done 223 times, an average of 6·8 auscultations per patient. Bowel sounds were heard 183 times in the first 15 s. Sounds were heard in the last 45 seconds but not in the first 15 s 26 times. Since absent bowel sounds are considered a sign of disease, this would have meant a 12% false-positive rate. On 14 occasions, bowel sounds were absent for the entire 15 s and not heard later. The false-positive rate varied from 7% in children aged 6–10 years to 20% if a nasogastric tube was present (although clamped during auscultation). There was no difference between morning and evening auscultation.

6 children were auscultated before and after major abdominal surgery. 1 patient with gastroschisis had no bowel sounds in either period. In the remaining 5 patients, bowel sounds were present during 1 min auscultation preoperatively and on each of the first 3 postoperative days in every patient.

Thus, abdominal auscultation does not correlate well with events in the first three days after a major intra-abdominal procedure. This fact, together with the unreliability of brief abdominal auscultation, calls into question the routine use of this physical sign.

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