tumour cells. No additional metastases were identified in a thorough search. Such an occurrence may be coincidental but it makes one wonder whether intra-operative seeding of tumour cells should not be a real concern of the surgeon, and what can be done about it?

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Carcinoma of the splenic flexure – a case for extended right hemicolectomy?

Sir,

We have recently managed a patient with a splenic flexure colonic carcinoma, who had an unusual distribution of lymph node metastases. This questions the validity of transverse colectomy for this condition.

The classical treatment of resectable colonic carcinoma has been excision in continuity with its vascular supply in an attempt at reducing local recurrence by removing involved or potentially involved lymph nodes. This is based on Jamieson and Dobson's work¹ which suggested that the lymphatic drainage of the colon followed its main blood supply. However, blood supply to the splenic flexure has been shown to be somewhat variable. Griffiths² demonstrated that it was supplied by the inferior mesenteric artery via the left colic in 89% of cases and by the superior mesenteric artery via the middle colic in 11%. He also noted that the middle colic was absent in 22% of cases. These findings were confirmed by Sierocinski³ who found in 100 post-mortem dissections that the middle colic vessel supplied the splenic flexure in only 19. Where the middle colic is absent, the ileocolic supplied the transverse colon and the left colic supplied the splenic flexure.

Goligher⁴ has advocated transverse colectomy for splenic flexure carcinoma, ligating both middle colic at its origin and the ascending branch of the left colic to remove those nodes most likely to be involved.

We report the case of an 83 year old female admitted as an emergency with vomiting and abdominal pain. Plain abdominal X-rays and a gastrografen enema revealed a stenotic lesion at the splenic flexure with complete obstruction. At laparotomy a splenic flexure carcinoma was found and an extended right hemicolectomy performed with an end to end ileocolic anastomosis. The patient made an uneventful recovery and was discharged home.

Histological examination of the specimen showed a moderately differentiated adenocarcinoma of the splenic flexure with one of eight local lymph nodes involved (Duke's stage C Jass Class IV). However, one node at the ileo-colic junction showed adenocarcinomatous deposits.

Aldridge⁵ has shown that patients with a splenic flexure carcinoma have a reduction in 5 year survival rate even when variables such as age, sex, Duke's stage and tumour differentiation were excluded. Three possibilities have been suggested to explain this. Firstly, that the carcinoma may be biologically different, and, secondly, that the host reaction may differ, but these seem unlikely. Lastly, the method of surgical management of the lymph nodes may be inadequate suggesting that an extended right hemicolectomy would be more appropriate. The higher

local recurrence rate of tumours of the splenic flexure would seem to support this view.

We suggest, therefore, that an extended right hemicolectomy should be performed for lesions of the splenic flexure. This avoids a colo-anastomosis, and problems due to inadequate bowel preparation and obstruction. It may also remove involved lymph nodes in cases where lymphatic drainage does not follow the classical pattern.

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Intracranial tuberculoma: paradoxical expansion during medical treatment

Sir,

Intracranial tuberculomas are only rarely encountered in developed countries. We report a patient whose disease was difficult to diagnose. His clinical situation deteriorated in spite of appropriate administration of antibiotic treatment, and only corticosteroid therapy achieved symptomatic improvement.

A previously healthy 61 year old male began to suffer from nocturnal headaches and rhythmic, clonic contractions of his left lower limb. Blood analysis (including ESR and chest X-ray) was normal. Computerized tomographic (CT) scan showed a hypodense right, parasagittal lesion, with surrounding oedema that enhanced with contrast near the falx, could be seen. A magnetic resonance imaging scan showed a right parasagittal, tumour-like lesion. Corticosteroid treatment was initiated, and symptoms promptly disappeared.

Five cerebral biopsies under stereotactic guide were necessary before noncaseating epithelioid granulomas were found. Appropriate Ziehl–Neelsen staining identified acid-fast bacilli. Daily parenteral treatment with rifampicin, isoniazid, ethambutol, pyrazinamide and dexamethasone was then administered. After six weeks of successful treatment and coinciding with gradual corticosteroid withdrawal, a sudden clinical deterioration including generalized seizures and loss of left lower limb strength was noted. A CT scan showed enlargement of the previous lesion as well as surrounding oedema. Corticosteroid treatment at the initial dose was restored, with the patient improving markedly.