LETTERS TO THE EDITOR

Laparoendoscopy for abdominal cancer

To the Editor: With growing interest in the application of laparoendoscopic techniques in abdominal surgery and the universal acceptance of endoscopic cholecystectomy, there is now work in progress both in the United States and in Europe to apply the technology to colorectal surgery. I wish to report a case where sigmoid colectomy was performed by means of a laparoendoscopic technique, which enabled a safe clearance for colon cancer and an early discharge from hospital.

An 80-year-old woman presented with an adenocarcinoma of the distal sigmoid colon. The site of the tumour was confirmed at laparoscopy, and the liver was checked for metastatic involvement. Laparoscopic instruments were used to prepare the proximal limb of the anastomosis. The mesentery of the sigmoid colon was divided and vessels were ligated with titanium clips. The inferior mesenteric artery was divided at the sacral promontory between ligatures that were tied using an extracorporeal knotting technique. The distal limb of the anastomosis was prepared by diathermy dissection and clip ligation of vessels in the rectal mesentery. A mirror image of the standard "battle" appendicectomy incision was made low in the left iliac fossa. The cancer and mobilised bowel were exteriorised and resected. An end-to-end anastomosis was created with the circular stapling device.

The patient passed wind and liquid stool on the second postoperative day and was rapidly mobilised with little abdominal pain. She was considered fit for discharge on the third day, but was kept in hospital for routine observation for a further 24 hours.

The pathology report confirmed the diagnosis of a moderately differentiated adenocarcinoma, Duke's stage B. The 10 nodes sampled in the sigmoid mesentery were all free of metastatic involvement.

There is obviously an important future for laparoendoscopic colon surgery. When intracorporeal stapling devices become available in this country later in the year it will be possible to perform similar operations by a totally closed technique, with extraction of the specimen through the rectal stump, depending of course on the size of the primary tumour.

I have attended operations in America where laparoscopic dissection of the rectum was made down to the level of the levator plate, with surprising accuracy and ease. Low anterior resection has not yet been performed, but it is certainly feasible, and the approach may also be useful for abdominal-perineal excision of the rectum.

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Detecting the cause of Lyme disease in Australia

To the Editor: Classical Lyme disease, a syndrome comprising erythema chronicum migrans (ECM) and various neurological, cardiac and rheumatological manifestations, is now recognised to be widespread throughout the Northern Hemisphere, where it is transmitted to humans through bites of a species of *Ixodes* tick that acquires the causal agent *Borrelia burgdorferi* during the developmental stages of its life cycle on wild animals.

Despite the absence from Australia of the primary Northern Hemisphere vector *I. dammini,* and its main vertebrate reservoir, the deer, since 1982 there have been sporadic reports of a syndrome like Lyme disease occurring in central coastal Australia.[1-3] A serological survey conducted at Westmead Hospital during 1988–1989 on 428 suspected cases of Lyme disease revealed 16 patients with low levels of IgG to *B. burgdorferi.*[4] In central coastal New South Wales there are anecdotal reports, from both physicians and farmers, of ECM and complications following tick bites. In the same area veterinarians occasionally recognise a Lyme disease syndrome in dogs and livestock, where the major presenting sign is shifting lameness. The accumulating evidence suggests a strong likelihood of a vector borne, zoonotic Lyme disease-like syndrome in Australia for which the incidence, prevalence and natural history will only become clear once its aetiology is established.

The starting point for our study, and for the preliminary results presented below, has been the notion that Australian Lyme disease is caused by a spirochaete similar, but not necessarily closely related antigenically, to *B. burgdorferi,* and that the spirochaete cycles through native fauna and domestic animals, transmitted by a tick with a wide range of hosts.

One of us (M C W) has cultured the gut contents of a large number of ticks (mostly adult female engorged *I. holocyclus,* but also a smaller number of both *I. holocyclus* nymphs and adult *Haemophysalis* spp.) in an attempt to detect likely Lyme disease spirochaetes. The ticks were collected from various areas in the Hunter Valley and Manning River districts of coastal New South Wales, many from pets and domestic livestock, some of which were clinically lame. Specimens were cultivated in BSK medium (a formulation developed for the isolation of *B. burgdorferi),*[5] and periodically examined by dark field microscopy for the presence of spirochaetes. To date, 70 of 167 specimens (42% of all ticks) were culture positive for Borrelia-like spirochaetes within eight weeks of inoculation into BSK media. The isolation rate was higher for *I. holocyclus* (44%) than for *Haemophysalis* spp. (35%). All initial isolates were in mixed culture, with Gram negative rods and sporulated bacilli predominating. So far systemic filtration and subcultivation has enabled us to purify nine isolates of tick-borne spirochaetes.

Morphologically our isolates are indistinguishable from our reference strain of *B. burgdorferi* (B31); they are large, coiled motile bacteria with an irregular rotational movement. All have fastidious growth requirements, growing only in BSK.

Preliminary serological characterisation, using both monoclonal and polyclonal sera prepared against *B. burgdorferi,* indicates that at least four of our pure isolates share antigenic epitopes with classical *B. burgdorferi,* as detected by ELISA, immunofluorescence and western blotting. Blot analysis indicates that at least three strains have a 31 000 molecular weight structural protein that reacts antigenically with monoclonal antiserum to *B. burgdorferi* surface antigen, outer surface protein A (OspA).

These findings indicate that some species of tick often responsible for human and animal tick bites in this country commonly harbour *Borrelia* spp. spirochaetes. On structural and antigenic grounds these microbes are likely to be the aetiological agents of Lyme disease in Australia.

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Caffeine and disease

To the Editor: While debate continues about caffeine ingestion and cardiovascular disease,[1-4] enquiries about caffeine ingestion are of practical importance in general and psychiatric practice.

Caffeine is available from a number of sources including coffee, soft drinks, chocolate and cocoa, and is an ingredient of certain medications including the anti-migrainous preparations Cafergot, Cafergot P, Ergodryl and Migral. It is used also in Drixine cough suppressant, Travacalm, No Doz and No Doz plus.

It is recognised in the *Diagnostic and statistical manual of mental disorders III revised* (DSM-IIIR) as a diagnostic category under the heading of "Caffeine induced organic mental disorder".[5] Section 305 refers to the essential features of this disorder as restlessness, nervousness, excitement, insomnia, flushed face, diuresis and gastrointestinal complaints. Caffeine is an important often unrecognised cause of vague, sometimes severe, abdominal pain.

Reference is made to symptoms appearing in certain people following the ingestion of as little as 250 mg caffeine per day; increasing symptoms are inevitable between 500 and 1000 mg per day; and significant disturbance follows consumption of more than 1000 mg per day, commonly presenting as anxiety disturbance — which the patient usually does not associate with caffeine ingestion.

Approximate values for caffeine per drink include 100 mg of caffeine for percolated coffee, 150 mg for fresh drip coffee, and 50 mg per cup of tea or serve of soft drink. Both drink and sources such as medications containing caffeine must be considered.

DSM–IIIR refers to differential diagnoses in regard to caffeine induced organic mental disorder as major episodes, panic disorder and generalised anxiety disorder.

In my practice I have not seen a psychotic disorder arising out of caffeine ingestion from beverages alone but I have seen an acute organically induced psychosis secondary to the ingestion of large quantities of No-Doz (which contains 100 mg of caffeine per tablet).

In most cases the differential diagnosis is between caffeine intoxication, panic disorder and generalised anxiety. A simple question about caffeine consumption will prevent the practitioner from falling into the trap of prescribing benzodiazepines for the treatment of anxiety when the appropriate management is reduction of caffeine intake.

This may be achieved by reducing the ingestion of coffee by substituting decaffeinated coffee, which for practical purposes may be regarded as containing no caffeine, and by restricting caffeine intake in soft drinks by advice relating to use of non-caffeine containing beverages.

Children may develop caffeine intoxication from soft drinks.

Sudden cessation of all caffeine products produces obvious withdrawal syndromes, described by Joseph Greden, Professor of Psychiatry at the University of Michigan, as comprising headaches, irritability, nervousness, lethargy and yawning.[6] While Professor Greden advises an abrupt cessation of all caffeine products to produce symptoms of withdrawal and convince the patient of the effect that caffeine is having, in my practice it appears to be equally effective to substitute non-caffeine containing products. Patients then become aware of progressive improvement in their state of health.

A final decision has to be made as to whether caffeine containing products (which include coffee, commercial beverages and No Doz) should be excluded. As a general rule, caffeine ingestion sh