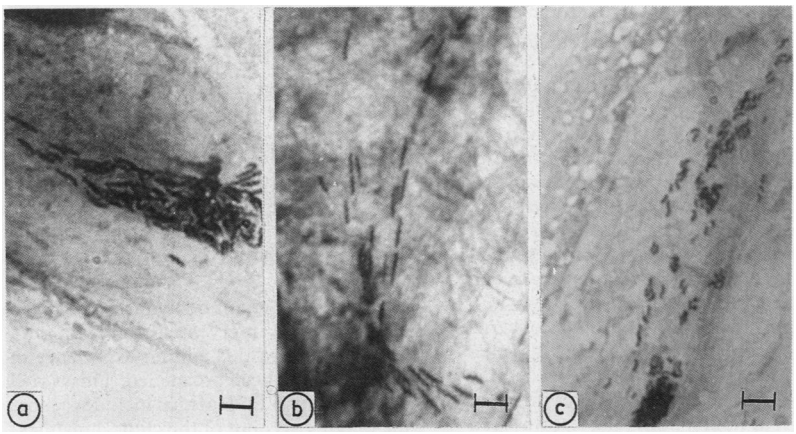

Gram stained smeared mucosa samples. (a and b) Tightly coiled spiral organisms in the two reported cases; (c) comparison of C pylori organisms. Bar = 5 μm.

Spiral shaped bacteria in gastric mucosa

McNulty *et al* reported a tightly spiralled organism morphologically different from *Campylobacter pylori* in the gastric mucosa of some patients with upper abdominal symptoms.[1] For this new spiral bacterium they suggested the name "*Gastrospirillum hominis*". We wish to report the observation of similar bacteria in two of the 210 patients who underwent gastroduodenal endoscopy for dyspepsia in Grosseto, Tuscany.

Five biopsy samples from each patient were taken from the gastric antrum and the edge of ulcers (when present), cultured on Columbia agar with 7% horse blood and the Skirrow mixture of chemotherapics, smeared and stained with the Gram stain and acridine orange, tested for the rapid urease activity, and examined histologically with haematoxylin and eosin stain.

In two cases in smeared mucosa samples we observed Gram negative, helical shaped organisms similar to those described by McNulty *et al*.[1] The bacteria were numerous in one case (figure, a), and scarce in the other (figure, b), had two to five spirals, ranged in length from 3 to 7 μm, and were morphologically easily distinguishable from *C pylori* (figure, c). Bacteria were also visible in smears stained with acridine orange. In one case the rapid urease test was positive within 15 minutes. No spiral organism grew after seven days of incubation in a microaerobic environment. Serology for *C pylori* (as investigated by an ELISA according to Kaldor *et al*[2]) was negative for both patients.

The first patient, a 40 year old man, was asymptomatic. He had undergone endoscopy for a gastric ulcer two years earlier. He was treated with ranitidine and antacids. The endoscopy was negative, but the histological examination showed a glandular gastric hyperplasia. The second patient, a 78 year old woman, had mild nausea, epigastric discomfort, moderate chronic gastritis and eosophageal leucoplakia. She was not receiving any treatment.

Lee *et al* have recently isolated a spiral-shaped bacterium from a cat's stomach which was morphologically similar to those seen by McNulty *et al*[1] and by us. The isolate was strongly urease, catalase, and oxidase positive. They also observed a tightly coiled spiral organism in the gastric biopsy specimens of two patients, and inferred that dogs (which also harbour spiral shaped micro-organisms in their stomach) and cats can transmit these bacteria to man.

The fact that these spiral shaped organisms have mostly been found associated with chronic gastritis may indicate that they represent a new group of primary pathogens for the gastric mucosa.

N FIGURA
P GUGLIELMETTI
Istituto di Malattie Infettive, Università di Siena, I-53100 Siena
S QUARANTA
Servizio di Gastroenterologia, Divisione Medica 2a, Ospedale di Grosseto, I-58100 Grosseto, Italy

1 McNulty CAM, Dent JC, Curry A, *et al*. New spiral bacterium in gastric mucosa. *J Clin Pathol* 1989;**42**:585–91.
2 Kaldor J, Tee W, McCarty P, Watson J, Dwyer B. Immune response to Campylobacter pyloridis in patients with peptic ulceration. *Lancet* 1985;**i**:921.
3 Lee A, Hazel SL, O'Rourke JL, Kouprach S. Isolation of a spiral-shaped bacterium from the cat stomach. *Infect Immum* 1988;**56**: 2843–50.

MATTERS ARISING

Sclerosing cholangitis and hepatic microvesicular steatosis in cystic fibrosis and chronic pancreatitis

Liver disease is common in patients with cystic fibrosis, the characteristic histological feature being the presence of inspissated, eosinophilic, periodic acid Schiff positive diastase resistant concretions in small bile ducts and ductules. The precise nature of these concretions is uncertain and the factors responsible for liver disease in only a few patients with cystic fibrosis have not been clearly defined.[1] Two recent studies, however, reported that bile duct lesions may be a factor in the development of intrahepatic disease. Gaskin *et al* found evidence of biliary tract obstruction with stenosis of the distal common bile duct in 36 patients with sclerosing cholangitis in two of 50 patients with cystic fibrosis and hepatic disease[2]; a normal biliary tract was found in 31 control patients with no disease of liver disease. Strandwick *et al* reported that four of 102 patients with cystic fibrosis had ERCP evidence of sclerosing cholangitis.[3] The implications of these findings are that surgical intervention may prevent the onset or progression of the liver disease in cystic fibrosis; the cholangiolar dilatation may be due to bile duct obstruction, the inspissated concretions being a manifestation of the generalised abnormality of exocrine secretion which characterises mucoviscidosis.

RNM MACSWEEN
University Department of Pathology, Western Infirmary, Glasgow G11 6NT

1 Gaskin KJ, Waters DLM, Howman-Giles R, *et al*. Liver disease and common bile duct stenosis in cystic fibrosis. *N Engl J Med* 88;**318**:340–6.
2 Strandvik B, Hjelte L, Gabrielsson N, Glaumann H. Sclerosing cholangitis in cystic fibrosis. *Scand J Gastroenterol* 1988;**23**:121–4.
3 Hultcrantz R, Mengarelli S, Strandvick B. Morphological findings in the liver of children with cystic fibrosis: a light and electron-microscopic study. *Hepatology* 1986;**6**:881–9.

Intestinal microsporidiosis in AIDS

We read with interest the letter by Lucas *et al* regarding the diagnosis of intestinal microsporidiosis in patients with AIDS.[1] Although we agree that under ideal conditions appropriately stained microsporidia can be visualised at light microscopy, we are not of the opinion that this can be an absolutely confident diagnosis and feel that electron microscopical examination is essential, particularly in view of the paucity of experience in diagnosing these organisms.

Lucas *et al* state that, "cases of intestinal microsporidia may therefore be confidently identified by light microscopic examination, supplemented by electron microscopy," but in two of their three cases, which were from dewaxed material reprocessed for electron microscopic examination, only "probable spores" were seen. The fact is that in only one case have they "confidently" diagnosed microsporidia, and this was by electron microscopy, having already suspected the presence of microspondia by light microscopy. This may seem to be somewhat pedantic but confidence levels among histopathologists are extremely variable.

It is interesting that the microsporidia found by Lucas *et al* was consistent with *Enterocytozoon bieneusi*.[2] Since the publication of our case[3] the organism has also been identified as *Enterocytozoon bieneusi* by Professor E U Canning.

We feel that until diagnostic expertise is widely available in this field, it should be routine procedure to submit a part of the tissue from cases of AIDS immediately following biopsy for electron microscopy, so that definitive diagnoses can be made if such parasitic infection is suspected by light microscopy. Only then can we further our knowledge about this little known group of human infections.

LJ McWILLIAM
A CURRY
University Department of Histopathology University Hospital of South Manchester Withington, Manchester

1 Diagnosis of intestinal microspondiosis in patients with AIDS. Lucas SB, Papadaki L, Conlon C, Sewinkambo N, Goodgame R, Serwadda D. *J Clin Pathol* 1989;**42**:885–7.
2 Desportes I, Le Charpentier Y, Galian A, *et al*. Occurrence of a new microsporidan: Enterocytozoon bieneusi ng, nsp, in the Enterocytes of a human patient with AIDS. *J Protozool* 1985;**32**:250–4.
3 Curry A, McWilliam LJ, Haboubi NY, Mandal BK. Microsporidiosis in a British patient with AIDS. *J Clin Pathol* 1988;**41**:477–8.

Dr Lucas comments:

I welcome the comments of Drs McWilliam and Curry on the problems of diagnosing microsporidiosis in small intestinal biopsy specimens. Of course, the final arbiter is properly prepared electron microscopy. The "probable spores" in our cases were "probable" on electron microscopic examination of very limited quantities of material originally taken to paraffin wax, then reprocessed. The important point is that the spores seen by light microscopy of smears stained with haematoxylin and eosin in the case of microsporidosis definitely confirmed by electron microscopy were identical with those seen in stained sections of our other four cases (our figs 2 and 3 showed different patients). Thus I feel confident in making the diagnosis when these characteristic intracellular bodies are seen.

If they are not seen by light microscopical examination the diagnosis of microsporidiosis cannot be excluded. Sampling is one reason. Also, in my experience only the spore and spore-forming stages of the life cycle of intestinal microsporidia can be seen on standard haematoxylin and eosin staining; the meront stages (which may be more numerous than spores) are essentially invisible, only being identified by electron microscopy.

Finally, a large series of small intestinal biopsy specimens from American homosexual HIV positive patients was reported at a recent AIDS conference. Of 71 biopsy specimens from 67 patients, 22 showed microsporidia by transmission electron microscopy. Seventeen of these 22 positive cases were also identified on semithin sections. Tantalisingly, the authors say, "in retrospect, parasites were also visible by light microscopy in many of the standard haematoxylin and eosin stained sections". We eagerly await the definitive publication.

I think that pathologists can spot these protozoa in many instances in ordinary sections, but as Drs McWilliam and Curry say, we certainly need more experience of this parasite, with confirmation by electron microscopy.

Peliosis thymomis

Peliosis thymomis is probably a misnomer.[1] The latin translation of peliosis of a thymoma is peliosis thymomatis.

E CALLEWAERT
Algemeen Ziekenhuis St Lucas—St Jozef 8320 Brugge 4, Belgium

1 Peliosis thymomis: association with tuberculosis. *J Clin Pathol* 1989;**42**:331.

Rapid urease tests for Campylobacter pylori

We read the reply of Vaira, Holton, and Salmon to the points raised by us in our paper.[1] The authors state that, "the results of 2% urea test, 6% urea test (CP test), and CLO test were done at five, 10, and 20 minutes. The results at one, three, and 24 hours are also given in our letter". Vaira *et al* have reported the results with these three tests (table 2) at five, 10, and 20 minutes, one hour, two hours, and 24 hours in an in vitro urease test of *C pylori*, *Proteus*, and *Klebsiella* strains, and not an in vivo study using mucosal biopsy specimens.[2] As we pointed out, in the in vivo study Vaira *et al* read the results of the CLO test at 20 minutes, 90 minutes, and 24 hours, results of the 2% RUT at three hours, four hours, and six hours, results of the CP test at 15 minutes, 20 minutes, and two hours. In their subsequent letter the authors reported the sensitivity and specificity of various tests, but comparison of sensitivity and specificity of various tests at different time intervals was not described either in the tables or in the text.[2]

The original letter of the authors[3] concerns the four hour urease test which uses 2% urea broth and incubation at 37°C. The text of this letter gave the sensitivity and specificity of the test at four hours, but as the test was read at a fixed interval of four hours, the authors' point that they gave the sensitivity and specificity of tests at different times in the text of their original letter does not hold true.

We had raised these basic points to arrive at an understanding of whether the results with different types of rapid urease tests with different media should be read at different time intervals or at the same fixed interval. The point remains unanswered.

DK BHASIN
A AYYAGIRI
V SINGH
SK MEHTA
Departments of Gastroenterology and Microbiology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

1 Bhasin D, Yachha S, Ayyagiri A, *et al*. How specific is rapid urease test for diagnosing C pylori? *J Clin Pathol* 1989;**42**:671.
2 Vaira D, Holton J, Cairns S, *et al*. Urease tests for Campylobacter pylori: care in interpretation. *J Clin Pathol* 1988;**41**:812–3.
3 Vaira D, Holton J, Cairns S, Falzon M, Salmon P. Four hour rapid urease test (RUT) for detecting Campylobacter pylori: is it reliable enough to start treatment? *J Clin Pathol* 1988;**41**:355–6.

A five minute stain for Campylobacter in tissue

Gray *et al* described a 30 minute stain for *Campylobacter* using 2% Giemsa without differentiation.[1] We have also found this method successful. By chance we also used stronger stain with shorter times of staining. The results were better because there was less background staining.

Our technique is to stain with 20% Giemsa for five minutes, then blot dry on filter paper, and very quickly dehydrate in one jar of absolute alcohol and transfer to Xylol before any loss of stain has occurred into the alcohol. After five minutes the staining of the *Campylobacter* is as heavy as that after longer staining. An advantage of the shorter staining time is that the background mucin stains less intensely, as do the gastric glands. As a result *Campylobacter* can be seen deep in the mucin of the gland crypts as well as in the mucin on the surface. The 20% Giemsa has a bench life of a month if kept at room temperature.

CR ROBINSON
Institute of Pathology Soroha Hospital, Beer Shera, Israel

1 Gray SF, Wyatt JI, Rathbone BJ. Simplified technique for identifying Campylobacter pyloridis. *J Clin Pathol* 1986;**39**:1279–80.

BOOK REVIEWS

A Colour Atlas of AIDS and HIV Disease. 2nd ed. CF Farthing, S Brown, RCD Staughton. (Pp 115; £16.) Wolfe Medical Publications. 1988. ISBN 0-7234-0995-1.

The first edition of this book was produced in 1986, and in the intervening two years until the availability of this edition experience of HIV disease has been broadened. This is well reflected in this edition which includes a more wide ranging account of the clinical manifestations of HIV disease. The appendices have also been expanded to cover important topics including the full Centers for Disease Control definition of AIDS and advice for people who are HIV antibody positive.

As with the first edition, the pictures are all of high quality, which, when coupled with the concise text, make this book highly recommended reading for all health care personnel who are interested in, or more especially involved with, the care of people with HIV disease and AIDS.

P GRINT

Disorders of the Spleen. Major Problems in Pathology. Vol. 20. Barbara C Wolf and Richard S Neiman. (Pp 211; £37·50.) WB Saunders. 1989. ISBN 0-7216-2503-7.

Most pathologists become decidedly uneasy when called on to interpret the histology of the spleen, not least because definitive accounts of splenic pathology are few and far between. This book goes a long way towards remedying this deficit. The information is presented in a precise and well ordered fashion and in most areas is admirably up to date and comprehensive: it is also supplemented by many helpful diagrams, tables, and references. Indeed it is difficult to find omissions, although more data regarding the hairy cell leukaemia variants—possibly the most common primary splenic neoplasms—would have been useful. It must also be said that not all of the photomicrographs (mainly in black and white) are wholly successful and some would have benefited from higher magnification.

All the same these are minor complaints and most pathologists, haematological or otherwise, will welcome this most creditable account of splenic pathology and might at last begin to make some sense out of an organ which hitherto has jealously guarded its mysteries.

F D LEE

Recent Advances in Clinical Pharmacology and Toxicology. No. 4. Ed P Turner, GN Volans. (Pp 276; $45.) Churchill Livingstone. 1989. ISBN 0-443-03297-5.

Five years have elapsed since publication of the last *Recent Advances in Clinical Pharmacology*, and in producing the latest volume the editors have taken the initiative to broaden its scope to include recent advances in the field of "clinical toxicology". Thus a very wide and often puzzling array of subject areas are covered in the present volume. For example, the first of the 13 essays is an