ation of serial sections showed that one diverticulum had ruptured (figure). This may account for the interstitial emphysema seen histologically and may provide an explanation for the clinical features.

A 13 year old girl developed a precipitous asthmatic attack from which she died a few minutes after the onset of breathlessness. The histological findings of the lungs were again those of interstitial emphysema and numerous bronchial diverticula. Around most of the diverticula there was a pronounced inflammatory cell infiltrate, composed predominantly of eosinophils, plasma cells, and lymphocytes.

Dunnill has suggested that diverticula present in asthmatic airways represent the mouths of mucous gland ducts.[3] We propose that although they originate at the site of origin of these ducts, their evolution is comparable with that of diverticula of the intestine, gall bladder, and urinary bladder. The diverticula develop at points of least resistance in the muscular wall; in the colon this occurs at sites where vessels enter the muscle coat, but in the bronchial wall the weak point is likely to occur at the mouths of the mucous glands.

The features of these disease processes show a striking similarity in that they are all outpouchings of mucosa between muscle. In each, the lining epithelium may ulcerate, and pronounced inflammatory cell infiltration may occur in the mucosa, submucosa and peridiverticular tissues.

Consistent with diverticula at other sites, the cause of the bronchial diverticula is likely to be primarily mechanical, resulting from raised intraluminal pressure and changed smooth muscle contraction.[4] Furthermore, we propose that the bronchial diverticula are of clinical relevance, for not only may the associated inflammatory changes be important in the pathogenesis of airflow obstruction in asthma, but if a diverticulum ruptures, interstitial emphysema may result and complicate the exacerbation of asthma.

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**References**

1 Reid L. Pathological changes in asthma. In: *Asthma.* Clark TJH, Godfrey S, Eds. London: Chapman & Hall, 1977:79–95.
2 Dunnill MS. The morphology of the airways in bronchial asthma. In: *New directions in asthma.* Stein M, ed. Illinois: American College of Chest Physicians, 1975:213–21.
3 Dunnill MS. The pathology of asthma, with special reference to changes in the bronchial mucosa. *J Clin Pathol* 1960;**13**:27–33.
4 Almy TP. Diverticular disease of the colon—the new look. *Gastroenterology* 1965;**49**:109.

# Other correspondence

## Multiple slit membranes and proteinuria

I read with interest the article by Harrison, Jenkins and Dick,[1] which recorded the presence of multiple slit membranes having a "step ladder" appearance in renal biopsies before and after transplantation from a patient with focal and segmental glomerulosclerosis.

I have found identical multiple slit membranes in rat kidneys after induction of simple protein overload proteinuria.[2] One reason for their apparent rarity may relate to the fact that they seemed to occur only in glomeruli showing intermediate levels of structural damage and that they are best shown after enhancement of staining by the use of tannic acid. I am convinced that they are the same structures as those described by Ryan, Rodewald, and Karnowsky.[3]

As protein overload proteinuria is not immunologically mediated and unlikely to be a toxic or basement membrane charge effect,[4] it is highly debatable whether the slit membranes are in any way directly related to the initial causation of the proteinuria.

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**References**

1 Harrison DJ, Jenkins D, Dick J. An unusual interpodocyte cell junction and its appearance in a transplant graft kidney. *J Clin Pathol* 1988;**41**:155–7.
2 Rollason TP, Brewer DB. A scanning and transmission electron microscopic study of glomerular damage in the rat following heterologous serum albumiun overload. *J Pathol* 1981;**134**:39–56.
3 Ryan GB, Rodewald R, Karnowsky MJ. An ultrastructural study of the glomerular slit diaphragm in amino-nucleoside nephrosis. *Lab Invest* 1975;**33**:461–8.
4 Rollason TP, Brewer DB. A study of glomerular basement membrane anionic sites and glomerular visceral epithelial cell coat in protein overload proteinuria in the rat. *J Pathol* 1984;**142**:301–16.

## Immunological abnormalities in myelodysplastic syndrome

Economopaulos *et al*[1] and Multi *et al*[2] have both recently drawn attention to the occurrence of immunological abnormalities in patients with myelodysplastic syndromes (MDS). Among the abnormalities reported were hypogammaglobulinaemia, hypergammaglobulinaemia, monoclonal gammopathy, and tissue autoantibodies. A new finding by the Bournemouth group was of a positive direct antiglobulin test (DAT) in eight of 98 patients.

We have studied 37 patients with various types of MDS presenting to us between July 1985 and October 1987. Eight (21%) had a positive DAT (six IgG, one $C_3$, one IgG plus $C_3$). Three of the eight had refractory anaemia (RA), three chronic myelomonocytic leukaemia (CMML), and two RA with excess of blasts (RAEB).

The high prevalence of positive DATs in MDS is not easily explained. A general increase in immunoglobulin production has been described in CMML,[3,4] possibly caused by the release of B cell growth factors from activated monocytes. Alternatively, the Bournemouth group[5] have suggested that an initial oncogenic event selects a clone of stem cells which retains the capacity to differentiate into both myeloid and lymphoid cells, both lineages being marked by functional abnormalities. Whatever the pathophysiological basis of this finding, a positive DAT is of considerable practical importance in a group of patients requiring frequent blood transfusion.

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**References**

1 Economopaulos T, Economidou J, Giannopoulos G, *et al.* Immune abnormalities in myelodysplastic syndromes. *J Clin Pathol* 1985;**38**:908–11.
2 Mufti GJ, Figes Ann, Hamblin TJ, Oscier DG, Copplestone JA. Immunological abnormalities in myelodysplastic syndromes. I. Serum immunoglobulins and autoantibodies. *Br J Haematol* 1986;**63**:143–7.
3 Solal C, Desaint P, Hererq B, *et al.* Chronic myelomonocytic leukaemia according to FAB classification: Analysis of 35 cases. *Blood* 1984;**63**:634–8.
4 Bernard DL, Burns GF, Gordon J, *et al.* Chronic myelomonocytic leukaemia with paraprotein but no detectable plasmacytosis. *Cancer* 1979;**44**:927–36.

5 Copplestone JA, Mufti GJ, Hamblin TJ, Oscier DG. Immunological abnormalities in myelodysplastic syndromes. II. Coexistent lymphoid and plasma cell neoplasms: a report of 20 cases unrelated to chemotherapy. *Br J Haematol* 1980;**63**:149–59.

### Laboratory diagnosis of *Branhamella catarrhalis*

Ahmad *et al* have highlighted the problem of distinguishing *Branhamella catarrhalis* from the *Neisseria* genus[1] but their identification scheme of up to nine characterisation tests may not find favour in a busy diagnostic laboratory.

To rely on the failure of *B catarrhalis* to grow either anaerobically or on selective media is to place much emphasis on a negative test. Furthermore, it is recognised that *B catarrhalis* may grow on modified Thayer-Martin (MTM) medium, and may fail to grow on nutrient agar incubated at 22°C.[2] The lack of pigmentation of *B catarrhalis* is likely to be a more useful characteristic than its positive superoxol test. *N perflora, N pharyngis,* and *N mucosa*—all superoxol negative—are pigmented as are the common superoxol positive non-pathogenic *Neisseria*.[3]

The demonstration of the ability of *B catarrhalis* to liberate high concentrations of butyric acid from tributyrin is a useful diagnostic test[3,4]: it can be carried out using commercial discs impregnated with tributyrin and phenol red (A/S Rosco). A positive reaction is indicated by a yellow colour after six to 12 hours' incubation at 37°C. If the indicator remains red after 24 hours the test is negative. *B caviae, B ovis,* and *B cuniculi* produce moderate amounts of butyric acid but are not encountered unless the specimen is of animal origin. All *Neisseria* strains produce negative results.

The colonial appearance of *B catarrhalis* will distinguish it from the pathogenic neisseriae *N meningitidis* and *N gonorrhoeae*. The following minimal criteria will differentiate *B catarrhalis* from non-pathogenic *Neisseria* species: lack of pigmentation, failure to produce acid from glucose, nitrate reduction, tributyrin and Dnase activity.

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**References**

1 Ahmad F, Young H, Mcleod DT, Croughan MJ, Calder MA. Characterisation of Branhamella catarrhalis and differentiation from Neisseria species in a diagnostic laboratory. *J Clin Pathol* 1987;**40**:1369–73.
2 Doern GV, Morse SA. Branhamella (Neisseria) catarrhalis: criteria for laboratory identification. *J Clin Microbiol* 1980;**11**:193–5.
3 Wilson G, Wilkinson AE. Neisseria, Branhamella, and Moraxella. In: Parker MT, ed. *Topley and Wilson's principles of bacteriology, virology and immunity. Vol 2* Systematic bacteriology. 7th ed. London: E Arnold, 1983:162–7.
4 Riou JY, Buissiere J, Guibourdenche M, Brault G, Carlier JP. Hydrolyse de la tributyrine par les Neisseria et les Branhamella. *Ann Microbiol* 1981;**132A**:159–69.

*Dr Ahmad comments:*

Dr Cooke has perhaps missed the point of our paper. We set out to discover the simplest way of accurately identifying *B catarrhalis* and differentiating it from other *Neisseria* species. Of course, there are many other tests that can be applied to achieve the same purpose. The main tests used in our study, in addition to usual microscopy and carbohydrate fermentation tests, were superoxol and Dnase activity. The growth on various commonly used culture media and under different conditions is recommended for difficult strains not identifiable by the routine techniques.

The colonial and microscopic appearance of *B catarrhalis* is very familiar to all experienced workers in respiratory bacteriology. One would not rely on this criterion alone. Similarly, pigmentation and even rough and smooth nature of colonies in the family Neisseriacae is variable. Tributyrin hydrolysis is a valuable test, has recently been made commercially available, and could be used if so desired as an additional test. Nitrate reduction is yet another available test but, when using Dnase and superoxol tests, we found both to be unnecessary.

### Vero toxin producing *E coli* in haemmorrhagic colitis

Walker, Upson, and Warren tested 80 faecal samples from patients with bloody diarrhoea or microscopic evidence of red blood cells and found four with *Escherichia coli* 0157 and 22 with *Campylobacter, Salmonella,* or *Shigella*.[1] We have also looked for Vero toxin producing *E coli* in patients with haemorrhagic colitis. Freshly passed stools from 40 patients recently admitted to the Northwick Park Lister Infectious Disease Unit were quantitatively cultured on both selective and non-selective agar. Ten colonies of each type of coliform were subcultured for identification and further testing, including subculture from both the higher and lower dilutions inoculated. Fresh rectal biopsy tissue from 17 patients was homogenised and cultured in a similar way.

Fourteen patients yielded campylobacters, salmonella, or *Shigella*; one specimen contained *Entamoeba histolytica* trophozoites. No *E coli* were isolated from seven patients. *E coli* from the remaining 18 patients were sorbitol fementors. Six biopsy cultures yielded sorbitol positive *E coli* and no biopsy yielded organisms not also found in the stool. These *E coli* strains were inoculated into syncase/glucose broth and tested for Vero toxin production.[2] A Vero toxin positive control strain obtained from Dr Sylvia Scotland (Colindale) and a Vero toxin positive strain isolated from a patient with haemolytic uraemic syndrome by Dr SP Borriello (CRC) were tested in tandem. Both positive control strains were positive on assay in Vero cells, but none of the *E coli* strains from patients with haemorrhagic colitis was.

Bloody diarrhoea without routinely recognised microbial or viral pathogens is a common presentation to an infectious disease unit.[3,4] Vero toxin producing *E coli* are clearly responsible for some cases of haemorrhagic colitis in the United Kingdom. Our results, which agree with those of Walker *et al*, show that these organisms do not appear to be responsible for the common, sporadic, form of haemorrhagic colitis seen in the community.

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**References**

1 Walker CW, Upson R, Warren RE. Haemorrhagic colitis: detection of Vero toxin producing *Escherichia coli* 0157 in a clinical microbiology laboratory. *J Clin Pathol* 1988;**41**:80–4.
2 Willshaw GA, Smith HR, Scotland SM, Rowe B. Cloning of genes determining the production of vero cytotoxin by *Escherichia coli*. *J Gen Microbiol* 1985;**131**:3047–53.
3 Jewkes J, Larson HE, Price AB, Sanderson PJ, Davies HA. Aetiology of acute diarrhoea in adults. *Gut* 1981;**22**:388–92.
4 Mandal BK, Schofield PF, Morson BC. A clinico pathological study of acute colitis: the dilemma of transient colitis syndrome. *Scand J Gastroenterol* 1982;**17**:865–9.

# Book review

**Steroid Hormone Receptors. Their Intracellular Localisation.** Ellis Horwood Series in Biomedicine. Ed CR Clark. (Pp 277; DM 125.) VCH. 1987. ISBN 0-89573-498-2.

This multiauthored book examines the ques-