exudate, which was present in 30% of these streptococcal and 18% of the non-streptococcal sore throats. Using Bayes' theorem, as de Dombal and his group have taught us, more extensive information of this kind will enable us to make confident clinical diagnoses in sore throats. Bain, Morton, and I have published other illustrations of the palatal papules with a brief discussion of our views on the sign.[4]

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Bronchoalveolar mast cells in extrinsic asthma

SIR,—We would like to draw attention to the considerable discrepancy between the mast cell counts described by Dr Kevin C Flint and colleagues (5 October, p 923) and those described by others (table). The reasons for these substantial differences are unclear.

Mast cells are usually very easy to identify with the alcian blue stain, although mucus may also take up the blue dye and when attached to other cells can resemble mast cells. Macrophages also occasionally stain with alcian blue and this in turn could lead to overcounting. Staining overnight with alcian blue, which is the method used by Dr Flint and colleagues,[1] may aggravate this problem by causing these macrophages to stain more deeply. Alternatively, safranine when used with Carnoy's fixative is a relatively weak nuclear stain and it is possible that Dr Flint and others were undercounting the other lavage cells. In this respect the counting method described by Agius *et al*, and adapted by our group, which allows rapid and accurate counting of a large number of cells, may be more appropriate.

Support for the accuracy of our counts is offered by the close agreement between the mast cell histamine content reported by our group and the histamine content of dispersed lung cells (3·3 pg, range 2·5-10 pg),[3] where greater accuracy is possible. The mast cell histamine content calculated by Dr Flint and others is significantly lower, despite the fact that the relatively traumatic techniques used in the tissue dispersion method cause loss of histamine from mast cells during fractionation.[4] In addition, the authors do not explain why the mast cell counts that they first reported[1] are so much higher than in the control group in this paper (1·35% *v* 0·3%), although the patients in the two groups are very similar.

These differences in mast cell counts are of more than academic importance. The results of Dr Flint and colleagues lead them to support the concept of mast cells being the central triggering cells in asthma, whereas our results suggest that an increase in the number of mast cells in bronchoalveolar lavage fluid is a non-specific feature found in several lung diseases. Indeed, the only disease in which exceptionally high percentages of mast cells in lavage fluid have been described is extrinsic allergic alveolitis,[5] in which bronchospasm is not a major clinical feature.

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1 Flint KC, Leung KBP, Pearce FL, Hudspith BN, Brostoff J, Johnson NMcI. Human mast cells recovered by bronchoalveolar lavage: their morphology, histamine release and the effects of sodium cromoglycate. *Clin Sci* 1985;**68**:427-32.
2 Agius RM, Godfrey RC, Holgate ST. Mast cell and histamine content of human bronchoalveolar lavage. *Thorax* 1985;**40**:760-7.
3 Schulman ES, Kagey-Sobotka A, MacGlashan DW, *et al*. Heterogeneity of human mast cells. *J Immunol* 1983;**131**:1936-41.
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5 Haslam PL, Dewar A, Butchers P, Turner-Warwick M. Mast cells in bronchoalveolar lavage fluid from patients with extrinsic allergic alveolitis. *Am Rev Respir Dis* 1982;**125** (suppl): 51.
6 Tomioka M, Ida S, Shindoh Y, Ishihara T, Takishima T. Mast cells in bronchoalveolar lumen of patients with bronchial asthma. *Am Rev Respir Dis* 1984;**129**:1000-5.
7 Wardlaw AJ, Cromwell O, Celestino D, Fitzharris P, Collins JV, Kay AB. Bronchoalveolar lavage mast cells in asthma and other respiratory diseases. *Thorax* 1985;**40**:717.

The authors reply below.—ED, *BMJ*.

SIR,—The identification of mast cells has long been a problem.[1] Early reports contained many conflicting reports about their distribution. Many authors, for example, denied their existence in the gastrointestinal mucosa. This was found to be due to the fact that special fixation and staining techniques were required for their demonstration, and unless such procedures are adopted mast cells from specific sites may go unidentified. We therefore do not think that the differences in the percentage of mast cells in bronchoalveolar lavage between Professor Kay's group and our own are necessarily due to our overcounting. Historically it has been shown to be easier to miss mast cells than to overestimate their number.

Whether slides are stained in alcian blue for four hours or overnight the final result is little different,[2] and counterstaining with safranin after alcian blue is the preferred method of identifying mucosal mast cells.[1] Mast cells have also been reported to make up 0·3-0·7% of bronchial lavages using toluidine blue, giving an identical calculated histamine content of 1·0 pg/mast cell.[3] The calculated histamine content of bronchoalveolar mast cells is indeed lower than that of dispersed human lung mast cells (mean 1·0 (SE 0·1) *v* 2·56 (0·3) pg/mast cell in parallel studies, unpublished observations), but we see no reason to assume that this should be identical in these two mast cell populations.

Similar differences in histamine content between mast cells from different sites have been reported in other species.[4] We have found a direct correlation, with the regression line passing through the origin, between the bronchoalveolar cell histamine content and the percentage of mast cells, suggesting accurate identification of the latter.[2] Similarly, the histamine content of the lavage increases in parallel with the number of mast cells in asthmatic subjects,[5] and more recently we have shown release of newly generated mediators (leukotriene C4 and prostaglandin D_2) in quantities appropriate to the mast cell content of bronchoalveolar lavage.[6] Bronchoalveolar lavage is itself a variable technique, and this problem is not exclusive to the mast cell. Lymphocytes make up 1-2% of control bronchoalveolar cells in some laboratories[7] and 18·6% in others.[8] Such differences are likely to be due to differences in the lavage technique and subsequent processing of lavage fluid.[9] More importantly, the differences in bronchoalveolar mast cell counts between different disease groups and controls are similar in our hands[5,10] and others.[11,12]

We agree that increases in the percentage of mast cells in bronchoalveolar lavage is not exclusive to asthma. In unselected patients with different diseases undergoing bronchoscopy to exclude bronchial carcinoma we reported a higher percentage of bronchoalveolar mast cells[2] than in our current control group. In 20 of the former group there was no endobronchial lesion, in seven bronchial carcinoma was confirmed, and four had evidence of past pulmonary tuberculosis. Our current control group was selected as having no evidence of lung disease either at the time of bronchoscopy or subsequently. We also found increased numbers of mast cells in lavage fluid in subjects with sarcoidosis.[10] We suggest that this is not a non-specific feature but is consistent with mast cell involvement in a variety of conditions, not just immediate hypersensitivity reactions. It emphasises the importance of the assessment of mast cell function and shows the inadequacy of simply counting cells. An increase in the number of mast cells with particular functional characteristics in one condition need not have the same effect as an increase in cells with different functional properties in another. High rates of spontaneous histamine release together with an antigen specific release of mediators so far appears to be characteristic of asthma.

The mast cell has a vast array of mediators at its disposal, and further work will probably show the differential release of these mediators in response to different pathological circumstances. Bronchoalveolar lavage provides the opportunity to investigate mast cell function in different pathological conditions, unlike the previous model—the human dispersed lung mast cell preparation—which is obtained almost invariably from patients with bronchial carcinoma. We look forward with interest to Professor Kay's further work.

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Summary of various workers' findings with mast cell counts

Author	Disease group	No of patients	% Mast cells (range)	Staining technique	No of cells counted	Calculated mean mast cell histamine content (pg)
Flint *et al*[1]	Ca lung and normal FOB	31	1·35 (0·5-3·0)	Carnoy's/ alcian blue	1000	1·0
Flint *et al* (5 October, p 923)	Asthma	10	1·40 (0·1-2·8)	Carnoy's/ alcian blue	1000	1·3
	Normal controls	14	0·3	—		
Tomioka *et al*[6]	Asthma	9	0·25 (0·3-0·6)	Toluidine blue+ immunohisto-chemistry	300	8·2
Agius *et al*[2]	Various lung diseases	97	0·22 (0·07-0·68)†	Toluidine blue+ immunohisto-chemistry	10000	3·7-10·9
Wardlaw *et al*[7] (and unpublished data)	Asthma*	11	0·19 (0·06-0·42)	Carnoy's/ alcian blue	5000	6·3
	Normal controls	8	0·03 (0-0·08)			
	Various lung diseases	44	0·17 (0·04-0·62)‡			

FOB = Fibreoptic bronchoscopy.
* Similar severity to the asthmatics studied by Flint *et al*.
† Excluding one count of 2·5% in a patient with allergic alveolitis.
‡ Excluding one count of 1·8% in a patient with unexplained shadowing on her chest radiograph.

3 Patterson R, McKenna JM, Suszko IM, *et al.* Living histamine containing cells from the bronchial lumen of humans. Description and comparison of histamine content with cells of rhesus monkeys. *J Clin Invest* 1977;**59**:217-25.
4 Enerback L, Wingren U. Histamine content of peritoneal and tissue mast cells of growing rats. *Histochemistry* 1980;**66**: 113-24.
5 Flint KC, Leung KBP, Hudspith BN, Brostoff J, Pearce FL, Johnson NMcI. Bronchoalveolar mast cells in extrinsic asthma: a mechanism for the initiation of antigen specific bronchoconstriction. *Br Med J* 1985;**291**:923-6.
6 Flint KC, Hudspith BN, Leung KBP, *et al.* IgE-dependent release of leukotriene C4 and prostaglandin D2 from human bronchoalveolar cells. *Thorax* 1985;**40**:716.
7 Law D, Jackson L, Fulmer J. Bronchoalveolar lavage analysis in the interstitial disease. *Am Rev Resp Dis* 1982;**125**:105A.
8 Goddard P, Clot J, Jonquè T, Bousquet J, Michel F. Lymphocyte subpopulations in bronchoalveolar lavages of patients with sarcoidosis and hypersensitivity pneumonitis. *Chest* 1981;**80**:447-52.
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10 Flint KC, Hudspith BN, Leung KBP, Pearce FL, Brostoff J, Johnson NMcI. Hyperresponsiveness of bronchoalveolar mast cells in sarcoidosis. *Thorax* 1985;**40**:220.
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The spirit of Griffiths

SIR,—In the report of the Central Committee for Hospital Medical Services (12 October, p 1065) there was a report that as unit general manager to the University Hospital of Wales, Cardiff, I have a five notional half day appointment for management. To avoid confusion I should point out that while this is true, I have assumed a whole time commitment to this major post in a hospital of 860 beds combining a medical and dental school. My managerial sessions are in the afternoon, but it is not unusual for them to last from 2 pm until 8 pm, while at weekends I visit other areas of the hospital as well as my own ward. I hope that this time consuming commitment will reduce as I become more familiar with the tasks and as new management arrangements are introduced.

I write this letter to emphasise the need for a whole time commitment even though contractually it may be only part time. It is early days but I would like to say that my initial three months' experience has been enjoyable and that I believe I made the right decision to apply. I am more than ever convinced of the importance of clinicians becoming involved in these new management arrangements and of the profound effect the Griffiths reorganisation will have for everybody working in the hospital. The retention of a limited clinical role for the medical unit general manager is important, not only for his or her career but also so that he is reminded of the difficulties remaining in clinical practice in NHS hospitals.

Many clinicians have not applied for these general manager posts because of job descriptions apparently requiring a full time involvement as well as commitment. I urge interested clinicians to apply and be prepared to argue the case for a part time contract based on a new managerial infrastructure which will enable them to assume a corporate responsibility. NHS hospitals cry out for leadership, and many consultants are ideally suited to give it.

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Paying for others' mistakes?

SIR,—I read Dr Robert Wilkins's letter (12 October, p 1051) with much sympathy. Microbiologists comprise another group of doctors rarely cited in reports of litigation. The protection societies' policy of applying the same subscription rate to such groups as to workers in the major clinical specialties is particularly unfortunate. Not only are such practitioners rarely the subject of law suits but, for the same reason that they figure less prominently in the public eye, they are less likely to receive the same level of income as workers in the clinical specialties. They are, therefore, effectively subsidising their higher paid colleagues.

There is no specific requirement for a medical practitioner to belong to any of the three protection societies, and evidence of adequate indemnity provided by any other body would be acceptable to health authorities. Perhaps if a sufficient number of interested practitioners could be coordinated then an independent organisation might become interested in providing insurance at rates reflecting the relative risks.

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SIR,—One must clearly respect the evolution of medical defence societies over the past 100 years (19 October, p 1071), and indeed I have no personal wish to seek subscriptions from the ruthless world of commercial insurance.

However, with escalating yearly subscription costs, the time has surely come for the societies to acknowledge that the profession is heterogeneous in its practices and accompanying medicolegal risks. The generous subscription discounts given to junior doctors in training positions are fully justified. But low risk non-training grade senior doctors should not subsidise those branches of the profession exposed to higher risk. Although the societies argue that such a policy of differential scales would not be financially feasibly, I understand the matter is kept constantly under review. In 1985 consultants running Minis and Rolls-Royces quite correctly pay different car insurance premiums. The allocation of different consultants into two or three different groups depending on risk would appear highly desirable.

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Points

Animal research

Dr L C COOPER (Boreham Wood, Herts) writes: I refer to comments made by Dr J Christie Brown and others (12 October, p 1045). While it should be obvious to any scientist or clinician that research on animals must continue, wholesale support of the government White Paper *Scientific Procedures on Living Animals* is quite another matter. The medical profession—indeed the scientific community as a whole—should beware of lending uncritical support to proposed legislation which makes no distinction between essential experiments and those done for less honourable motives, such as weapons research and the testing of smoking materials and cosmetics.

High dose phosphate binders: calcium carbonate versus aluminium hydroxide

Dr JACOB ZATUCHNI (Episcopal Hospital, Philadelphia, PA19125-1098, USA) writes: Dr R H K Mak and others (7 September, p 623) report that calcium carbonate is as effective as aluminium hydroxide as a phosphate binder. Although they indicate few and easily reversible side effects, there is concern about both aluminium toxicity and hypercalcaemia. The purpose of this brief note is to call attention to the possible use of sucralfate, a basic aluminium salt of sucrose sulphate used in the treatment of duodenal ulcer, which has been reported to be valuable in lowering serum phosphorus concentrations in uraemia.[1,2] The dose is 1 g tablet four times a day. The drug is well tolerated and has few or no adverse effects and no contraindications. Serum calcium concentration is not affected and aluminium concentrations are not significantly raised, if at all.[1,3] Although somewhat costlier, the difference is not that great considering the quantity of other drugs necessary for phosphate binding.

1 Leung ACT, Henderson IS, Halls DJ, Dobbie JW. Aluminium hydroxide versus sucralfate as a phosphate binder in uraemia. *Br Med J* 1983;**286**:1379-81.
2 Sherman RA, Hwang ER, Walker JA, Eisinger RP. Reduction in serum phosphorus due to sucralfate. *Am J Gastroenterol* 1983;**78**:210-1.
3 Kinoshita H, Kumaki K, Nakano H, *et al.* Plasma aluminium levels of patients on long term sucralfate therapy. *Res Commun Chem Pathol Pharmacol* 1982;**35**:515-8.

Timing of lumbar puncture in severe childhood meningitis

Dr P J METCALF (Haig Clinic, Lethbridge, Alberta T1J 0Z2, Canada) writes: I am extremely concerned that any paediatrician would promulgate the advice that a lumbar puncture is not necessary in the diagnosis of meningitis (7 September, p 651; 28 September, p 898; 19 October, p 1124). A lumbar puncture is an essential part of allowing the appropriate diagnosis to be made. As was pointed out by some of the specialists whose opinions were sought, the presence of a rash does not always indicate that the organism is *Neisseria meningitidis*. Furthermore, although most of the organisms which cause paediatric meningitis are susceptible to chloramphenicol, the emergence of resistant strains, particularly of *Haemophilus influenzae*, makes it mandatory that antibiotic sensitivities are performed on all patients so that appropriate antibiotic therapy can be started as soon as possible. . . .

Therapeutic diets

Ms ANNIE S ANDERSON and others (Aberdeen Royal Infirmary, Aberdeen AB9 2ZB) write: It is encouraging to see the *BMJ* devoting so much space to Professor Stewart Truswell's clearly presented articles on nutrition and diet. Professor Truswell spends some time describing techniques for advising patients on therapeutic diets (21 Sept, p 807). However, he fails to emphasise that there already exists a highly trained body of skill in these matters. Surely, the most appropriate person to work out "the most comfortable way . . . to incorporate the doctor's prescription into the family's food pattern" and to advise the medical profession is the dietitian. . . .

Vitamins for vegetarians

Mr ALAN LONG (The Vegetarian Society, London W8 6LA) writes: Although Professor A Stewart Truswell's ABC of Nutrition takes account of the increasing interest in vegetarianism, doctors may need extra advice over vitamins B-12 and D. British vegetarians and vegans can obtain vitamin B-12 from various non-animal foods in their diet (including an estimable low salt yeast extract and fermented foods of oriental origin). We distribute a list of such sources. Until recently foods have been supplemented with vitamin D_2, which is derived from yeast. Recently vitamin D_3 has supplanted such "vitamin D." It derives from wool grease, some of which may be a slaughterhouse byproduct. Vegetarians and vegans (as well as religious groups such as orthodox Jews and Rastafarians) may therefore abstain from foods and supplements because they contain vitamin D_3 or unspecified vitamin D. We are pursuing these matters with manufacturers and labelling authorities. Meanwhile, soya milk and margarine fortified with vitamin D_2 remain available. Soya based baby foods that would be otherwise suitable contain vitamin D_3. Plant sources of vitamin D (such as shi-itake mushrooms) cannot yet be counted important sources in British diets.