Correspondence

High prevalence of familial sarcoidosis in an Irish population

SIR,—Dr NJ Brennan and others ended the discussion of their interesting paper on familial sarcoidosis (January 1984, p 14) by suggesting that a study of familial sarcoidosis combined with HLA marker studies might help to delineate the relative contributions of genetic and environmental factors in the pathogenesis of the disease. HLA specificities were estimated in 14 families where more than one member had sarcoidosis.[1] The haplotype segregation in the affected relatives of propositi was very similar both to predicted distributions and to the distribution in unaffected relatives. The study was done before DR specificities had been properly defined but, since this was a family study, haplotypes were identified and the haplotype A1/B8 is in very close linkage disequilibrium with an antigen, DR3, which is of particular interest. HLA family studies have a number of advantages over investigations of populations of unrelated individuals having disease and the negative results of this investigation are the best evidence so far available against an association between HLA and the development of sarcoidosis. However, the paper by Dr J Gardner and others (January 1984, p 19) showing an HLA association with good prognosis provides further evidence in support of two previous independent studies.[2,3] It seems that even if HLA does not influence the development of sarcoidosis it does modify the way that the disease is expressed once it has developed.

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[1] Turton CWG, Morris L, Lawler SD, Turner-Warwick M. HLA in familial sarcoidosis. In: Jones-Williams W, Davies BH, eds. *Proceedings of the Eighth International Conference on Sarcoidosis and Other Granulomatous Diseases.* Cardiff: Alpha Omega, 1980:195–200.

[2] Smith MJ, Turton CWG, Mitchell DN, Turner-Warwick M, Morris LM, Lawler SD. Association of HLA B8 with spontaneous resolution in sarcoidosis. *Thorax* 1981;**36**:296–8.

[3] Neville E, James DG, Brewerton DA, James DCO, Cockburn C, Fenichal B. HLA antigens and clinical features of sarcoidosis. In: Williams WJ, Davies BH, eds. *Proceedings of the Eighth International Conference on Sarcoidosis and Other Granulomatous Diseases.* Cardiff: Alpha Omega, 1980:201–5.

** This letter was sent to the authors, and Professor Fitz-Gerald replies below.

SIR—Dr Turton is correct in pointing out our omission of a reference to a valuable HLA study in familial sarcoidosis with which he was associated. We were aware of this work and included the reference in earlier drafts but unaccountable omitted it from our final submission.

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Definitions of emphysema, chronic bronchitis, asthma, and airflow obstruction

SIR,—We read the excellent editorial by Drs CM Fletcher and NB Pride (February 1984, p 81) with pleasure. We would, however, disagree with one term that they suggest. They recommend that the term chronic airflow obstruction or limitation should be used for persistent airflow obstruction. We wholeheartedly agree with the term chronic airflow obstruction but object that chronic airflow limitation is ambiguous. The latter term does not exclude airflow limitation as the result of reduced driving force as in advanced restrictive lung disease, where the decreased lung volume is the principal cause of reduced flow. In chronic obstructive pulmonary disease, however, there is obstruction to flow, whether as a result of airways collapse or of narrowing. If there is ever a need for pedantic exactitude it must be in precision of definition, so that we may achieve a generally agreed and unequivocal clinicopathological system of classification.

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** This letter was sent to the authors, and Dr Pride replies below.

SIR,—We agree that maximum airflow can be reduced when there is respiratory muscle weakness or severe loss of lung volume, and this is a disadvantage of the term airflow limitation.

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Retrieval of left atrial catheters with a mediastinoscope

SIR,—Continuous monitoring of the left atrial pressure is widely used both in the operating room and in the intensive care unit in the management of patients undergoing open heart surgery. The catheter is usually fixed, at its entrance either to the left atrium or to the pericardium, to prevent its displacement during the operation. Occasional difficulty is encountered when removing the left atrial pressure line if the retaining suture has not been divided before the chest is closed or if the line fragments when an attempt is made to remove it. In either situation its recovery is essential because a retained fragment can cause a recurrent bacteraemia,[1] initiate thrombus formation, or cause immobilisation of the disc prosthesis.[2]

Since Carlens introduced the mediastinoscope in 1959, it has been found to have several diagnostic and therapeutic uses.[3] We have employed it successfully to retrieve a left atrial pressure monitoring catheter without reopening the chest in two patients. In one patient who had his mitral valve replaced the temporary anchoring stitch for the left atrial catheter was not cut before the chest was closed. The