

performed. Their conclusion that "reduced gonadotrophin secretion was not found in our patients with chronic obstructive lung disease" must therefore be interpreted with caution. It is likely that the source of the gonadotrophin abnormality in patients with chronic obstructive lung disease results from a variation in the amplitude or frequency (or both) of gonadotrophin releasing hormone release, so that single serum follicle stimulating hormone and luteinising hormone results are of limited value.

With regard to other endocrine studies in chronic obstructive lung disease, Dr Gow and her colleagues confirm our findings of occasional pituitary suppression of thyroid stimulating hormone release and occasional elevation of prolactin. As in our studies, these were not consistent findings and did not correlate with Pao<sub>2</sub> levels. This is not to say, however, that hypoxia is not a contributory factor in individual patients.

We believe that the findings of Dr Gow and colleagues are in keeping with our own and there is nothing in the paper to contradict our view that hypoxia may be a suppressive factor in hormone production, especially testosterone, in chronic obstructive lung disease.

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1 Semple P d'A. *Clinical, endocrine and metabolic studies in medical conditions characterised by hypoxia*. MD thesis, University of Glasgow, 1984.

**AUTHORS' REPLY** We agree with most of the points made by Dr Semple and others. Hypoxia may well be a factor in suppression of hormone production, particularly testosterone, in chronic obstructive lung disease in patients aged under 70 years. Our study, however, was carried out in an unselected group of inpatients admitted to a respiratory unit. Their average age was 73 (range 57–83) years, which we think is representative of most patients with chronic obstructive lung disease.

Our findings suggest that in such "elderly patients" the general effects of age and illness may be more important than direct effects of hypoxia on endocrine function.

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**Sarcoidosis possibly predisposing to disseminated histoplasmosis**

SIR,—Dr J G Tebib and colleagues (January 1988;43:73–4) report disseminated histoplasmosis in a patient with endo-

thoracic sarcoidosis. The diagnosis of sarcoidosis was based on radiographic and clinical evidence, histology of the lymph node biopsy specimens, lymphocytosis in alveolar lavage fluid, and raised serum concentrations of angiotensin converting enzyme (ACE). It should be noted, however, that none of these findings is pathognomonic and serum ACE levels especially may be increased with histoplasmosis.<sup>1</sup>

This observation also brings up the problem of superinfection in sarcoidosis, where cellular immunity is decreased. Among the numerous publications on this subject, the paper by Winterbauer<sup>2</sup> looked at 122 patients with sarcoidosis and concluded that only superinfection with aspergillosis appears to be more frequent in patients with sarcoidosis. This paper summarised 26 studies dealing with the association of sarcoidosis and fungal infections. In only 14 of these was there a causal association. In fact, the diagnosis of sarcoidosis may be proposed too frequently, especially in cases of fungal infection. This is especially true when the delay between the diagnosis of sarcoidosis and the fungal infection is less than one year.

Corticosteroid treatment in a patient with sarcoidosis is a major cause of immunodepression, as noted in a report of a parasitic infection in patient treated with corticosteroids for sarcoidosis.<sup>3</sup> The paper by Dr Tebib and his colleagues is of interest because treatment with corticosteroids was not started until late in the course of the disease and, although it most likely contributed to the acute presentation of the disease, it cannot be considered to have been a major causative factor in the immunodepression. Another interesting aspect of this article is that, along with the increased frequency of opportunist infections, certain "exotic" diseases are becoming cosmopolitan.

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- 1 Jay SJ, Ryder KW, Kiblawi SO. Serum angiotensin converting enzyme activity in patients with acute histoplasmosis. *Am Rev Respir Dis* 1981;123:106.
- 2 Winterbauer RH, Kramer KG. The infectious complications of sarcoidosis. A current perspective. *Arch Intern Med* 1976;136:1356–62.
- 3 Mulliez PH, Dabouz R, Demory JL, Darras A, Crinquette J. Une observation de leishmaniose chez un malade traité par corticoïdes pour sarcoïdose. *Méd Mal Infect* 1987;17:412–3.

### Mesenchymoma of the lung

SIR,—We read with interest an article by Dr JMM van den Bosch and colleagues (October 1987;42:790–3), in particular the short report of two recurrent tumours. The literature survey shows that this is a highly unusual occurrence<sup>1,2</sup> and where they have been noted they were usually associated with multiple hamartomas.

We should like to report another case of recurrence. In 1979 a 57 year old man was found to have a rounded opacity in the right upper lobe on routine chest radiography. At thoracotomy a 1 cm hamartoma was enucleated and histological examination confirmed the clinical diagnosis, show-