

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 Pao₂ 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.¹

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 Winterbauer² 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.³ 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^{1,2} 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-

ing the tumour to be composed predominantly of cartilage but with epithelial structures, fat, and fibromuscular tissues. Eight years later a repeat chest radiograph showed an opacity in a similar position and at subsequent exploration a further nodule was removed from the area of scar tissue at the site of previous surgery. This again was confirmed histologically as being a chondromatous hamartoma.

The appearance of an opacity at the site of a previous hamartoma should not, however, be assumed to be a recurrence as there must be a possibility of scar carcinoma.

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- 1 Bateson EM. Cartilage-containing tumours of the lung. Relationship between the purely cartilaginous type (chondroma) and the mixed type (so called hamartoma): an unusual case of multiple tumours. *Thorax* 1967;22:256–9.
- 2 Oldham HN, Young WG, Sealy WC. Hamartoma of the lung. *J Thorac Cardiovasc Surg* 1967;53:735–42.

Book notices

Essentials of Respiratory Medicine. JF Cade, MCF Pain. (Pp 185; £9.95.) Oxford: Blackwell, 1988. ISBN 0-632-01913-1.

This book aims to be suitable for undergraduate students, general physicians, respiratory nurses, and technicians. It is in two broad sections, the first 80 pages dealing with structure, function, diagnosis, and treatment, and the second 80 dealing with specific diseases. The introductory chapters cover old ground and new developments in physiology and diagnostic techniques. There are some familiar illustrations redrawn from other books. Overall, the book covers the essentials of respiratory disease as well as it is possible to do in a text of this length. The most common conditions get reasonable coverage and rarer conditions have a mention. The earlier chapters integrate science with clinical medicine. In some respects the authors fail to provide the practical help that we might expect from such a book. In the chapter on symptoms and signs, some terms, such as “mediastinal crunch,” are introduced with no description of their features. There is no overview of the way the physical signs fit together in different diseases. Again, in the respiratory function chapter a list of normal values is given with no account of variability, and few indications are given for the various tests. To some extent the same criticism is applicable to the clinical chapters. It would be difficult to extract from the chapter on asthma a way of managing acute, severe disease. There are no particular guidelines about severity or practical help with the drugs used for management. On the other hand, there is a detailed list of all the drugs available and their doses, which—given the developments in the pharmaceutical industry—will inevitably become out of date fairly quickly. Perhaps the balance should be adjusted in these chapters. The text is clearly laid out and easy to follow. There are informative tables and line drawings. Overall, this is a useful introductory

text, which will be suitable for undergraduate students and paramedical workers but will not give the practical help that postgraduates would require. It comes at a reasonable price but enters a competitive market of introductory texts on thoracic medicine.—PJR

Respiratory Intensive Care. K F MacDonnell, P J Fahey, M S Segal. (Pp 478; £50.) Boston: Little, Brown and Company, 1987.

This book is American and has 37 contributors, of whom 26 are from Saint Elizabeth's Hospital, Boston. The book is divided into three sections, dealing with physiological principles and techniques of respiratory intensive care, disease states and their management in the respiratory care unit, and nursing and legal implications of the respiratory intensive care unit. The appendix includes some useful data on pulmonary function tests. The first section has an excellent chapter on oxygenation, emphasising the importance of maintaining adequate oxygen delivery ($12\text{--}15\text{ ml O}_2\text{ min}^{-1}\text{ kg}^{-1}$), an unusual and interesting chapter on the assessment and clinical approach to carbon dioxide disorders in critically ill patients, and a chapter on nutritional support in the intensive care unit. Despite popular belief in the efficacy of nutritional support, investigators have not yet been able to show, in a prospective randomised double blind manner, that it leads to any reduction in morbidity or mortality. None the less, the chapter gives helpful guidelines on intravenous and enteral nutrition. The author emphasises the importance of careful observation of the arterial carbon dioxide tension and the relation of carbon dioxide production to glucose oxidation and the rate of glucose infusion: an infusion rate of more than $5\text{ mg kg}^{-1}\text{ min}^{-1}$ produces a rapid rise in the respiratory quotient and excessive production of carbon dioxide, which in the patient with respiratory failure may lead to carbon dioxide narcosis. A chapter exclusively on muscle relaxants seems inappropriate—there is insufficient emphasis on the importance of sedation and analgesia, the correct combination of which produces a neuroleptic state and eliminates the requirement for muscle relaxants. Clearly, when it is essential that the patient should not breathe against the ventilator relaxants may be necessary; but if they are used it is important that the patient is well sedated and free of anxiety and pain. A well written and descriptive chapter on mechanical ventilation has clearly come from someone who has had a great deal of practical experience. In the second section the chapter on bronchial asthma is good, but fails to mention the hazards of losing cardiac output when mechanical ventilation is started and the importance of excluding bronchospasm related to left sided heart failure. The chapters on the adult respiratory distress syndrome and aspiration syndrome are recommended reading. The short third section includes a chapter on nursing aspects of respiratory intensive care, and another on legal considerations. This section is not particularly pertinent to medicine as practised in Britain. This book is useful as a reference manual, and would be valuable for any chest physician wishing to gain further insight into management of pulmonary disease in an intensive care unit. The drugs and ventilators used in Britain often differ from those in the United States, but this does not detract from the value of the book.—GCH