a very useful review of current methods of gastrointestinal investigation, and an academic consideration of principles underlying antibiotic therapy. Present ethical preoccupations are given a scholarly airing, and the list is completed with a discussion of factitious illness for whose reader recognition the editor deserves much credit.

My only serious criticism is the considerable number of printing errors that sometimes potentially obscure meaning such as 'pinocytic raculose' (p. 63) 'antiography' (p. 93), 'arbitrary initiation right for heaven' (p. 121)—among others. There is no prize for the correct solution! The price of the book could have set a little aside for another proof reading but these puzzles may yet further attract readers to an obligatory paediatric up-date.

C. G. H. NEWMAN,
Westminster Hospital,
London SW1.

Textbook of Pulmonary Diseases

Edited by GERALD L. BAUM and EMANUEL WOLINSKY. 3rd edn. Pp. xii + 1368, illustrated. Little, Brown, Boston, Totonto, 1983. £60.00.

This book, the first edition of which was produced in 1964, is regarded by the editors as a valuable source-book. In this particular aim I think it succeeds. Almost 70 authors, the majority from North America, attempt to cover the whole of pulmonary medicine and the

book has well-reproduced radiographs with a good index and over 5000 references (although few are later than 1980). A new and informative initial section has been added covering the immunology, physiology and cell biology of the normal lung with an unusual but important section on basic concepts in pulmonary function testing.

The body of the book is, however, extremely variable in quality. There are many good sections including those, for example, on diagnostic procedures, preoperative assessment, and pulmonary infections with a thankfully balanced account of chemotherapy in lung cancer. Unfortunately there is a woefully inadequate section on asthma which mentions neither nocturnal asthma nor the use of the peak flow meter and the chapter on computers in pulmonary medicine could not conceivably have been detailed enough to be of practical help. Indeed it is in the translation of the theoretical and experimental background to practical details of patient diagnosis and management that I found the book disappointing. Its highly compartmentalised disease-oriented approach affords little discussion of problems of differential diagnosis either at the level of clinical presentation or of chest radiographic appearances. I could not, for example, find a discussion of the common problem of widespread nodular shadowing.

Thus, although the book contains much valuable information and several excellent individual contributions, I cannot recommend it, particularly at £60, for the shelves of most medical libraries in this country.

I. D. A. JOHNSTON,
Whittington Hospital,
London N19.

Letter to the Editor

July 30th, 1984

Persistent akathisia associated with early dyskinesia

Sir, I read with interest the report by Barnes and Braude (1984) in which the possibility that akathisia may be a forerunner to tardive dyskinesia (TD) was raised. I have recently encountered a case of akathisia that developed into TD following the administration of clomipramine.

Eight months before presentation this 33-year-old woman received clomipramine (50 mg daily) for endogenous depression. Four months before presentation she suddenly stopped the medication with no adverse effects. Approximately 3 months later she became depressed, irritable and suicidal and was given once again clomipramine (25 mg daily). Twenty-four hours later she developed extreme restlessness with constant urge to move. Examination was unrevealing except for fine tremor of the hands. One week later she presented with facial-buccal dyskinesia that included tongue protrusion, sucking movements of the lips and chewing mandibular movements. Abnormal movements were also noticed in the neck and shoulder muscles. Clomipramine was immediately discontinued with the result that the abnormal movements gradually abated within 36 hr.

In this case akathisia preceded the development of TD. The former could be considered a dose-dependent idiosyncratic reaction to clomipramine, or else the result of altered receptor sensitivity in the meso-cortical dopaminergic system (Marsden and Jenner, 1980). TD could have been caused by dopamine-receptor supersensitivity in the nigro-striatal system (Marsden and Jenner, 1980). The development of TD after akathisia may therefore be a result of different susceptibility of dopamine receptors in the behavioural (mesocortical) and motor (nigro-striatal) dopamine system. The report by Barnes and Braude (1984) as well as the present one suggest that both conditions are interlinked. Both represent the clinical manifestation of dopaminergic involvement along the mesolimbic and nigro-striatal systems.

Yours faithfully,
R. SANDYK

Department of Pharmacology, Medical School,
University of the Witwatersrand, Johannesburg 2193,
South Africa

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