LETTERS TO THE EDITOR

Pulmonary complications of HIV disease

We read with interest the review by Drs DG Mitchell and RF Miller (May 1992;47:381-90). They refer to a study published in 1991 that bacterial pneumonia occurred much less frequently than Pneumocystis carinii pneumonia in homosexual men in London over a four year period, in contrast to a high relative frequency in an American population that was published in 1990. In consecutive patients admitted with respiratory episodes in 1986-7 (33% of all admissions to the unit) Pneumocystis carinii pneumonia and five had bacterial infection (one had pneumonia and four had bronchitis, a diagnosis of pneumonia being made on radiographic grounds). The correct diagnosis was not always made, however, and inappropriate management may be fatal.

We wish to report the case of a 65 year old man with a 20 year history of rheumatoid arthritis who started taking methotrexate 10 mg weekly in March 1992, at which time his pulmonary function and chest radiograph were normal. He was admitted to hospital with increasingly severe hypotension, with consequent oliguria and acute renal failure. His oxygen saturation (SaO2) fell to 80% despite increasing concentrations of inhaled oxygen. Cardiac enzymes were normal, and dopamine was given through a central venous line—no aiv, antibiotics were not administered.

The possibility of methotrexate pneumonitis causing hypoxia and consequent myocardial depression was considered and he was treated with 500 mg of intravenous methylprednisolone over three successive days. Within 24 hours of his starting on this his SaO2 had risen to 95% and his blood pressure had become normal. Pulmonary function testing at this stage showed a pronounced restrictive defect with normal ventilation, and his saturations had improved and he was started on prednisolone 60 mg daily. He was discharged one week later. At review a month after discharge his renal function and chest radiograph were almost normal, and methotrexate therapy had been permanently discontinued and his pulmonary function is slowly improving with a reducing dose of oral prednisolone.

Pneumonitis due to methotrexate can closely mimic left ventricular dysfunction and failure to suspect it may lead to a fatal delay. Immunosuppressive doses of steroids may be required in severe cases, though infection should first be excluded. Upper lobe abnormality, although unusual, may be especially likely to cause diagnostic confusion.

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Acute pneumonitis associated with low dose methotrexate treatment for rheumatoid arthritis

We read with interest the paper by Dr MR Hargreaves and others (August 1992;47:628-33). With the increasing use of low dose methotrexate in the treatment of patients with rheumatoid arthritis, drug induced pneumonitis is likely to become increasingly recognised. The correct diagnosis is not always easily made, however, and inappropriate management may be fatal.

We wish to report the case of a 65 year old man with a 20 year history of rheumatoid arthritis who started taking methotrexate 10 mg weekly in March 1992, at which time his pulmonary function and chest radiograph were normal. He was admitted to hospital with increasingly severe hypotension, with consequent oliguria and acute renal failure. His oxygen saturation (SaO2) fell to 80% despite increasing concentrations of inhaled oxygen. Cardiac enzymes were normal, and dopamine was given through a central venous line—no aiv, antibiotics were not administered.

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Tuberculosis contact tracing: are the British Thoracic Society guidelines still appropriate?

The suggestion made by Dr SF Hussain and colleagues (December 1992;47:984-5) of a review of tuberculosis contact tracing recommendations is timely. The number of
cases diagnosed as a result of the contact tracing described in this paper is small, however, and it would be a pity if the possibility of late tuberculosis was discounted on the grounds that no such cases were found in South Glamorgan. We recently reported on a child who was found to have a tuberculous pleural effusion six months after being a close contact of a patient with smear positive tuberculosis.\(^1\) This child had been thought to be negative on being screened six weeks after the last contact. In children at least, longer follow up may be appropriate.

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**BOOK NOTICES**


This excellent book, the fifth in the "Fundamental and Clinical Cardiology" series, is directed towards cardiovascular physicians and research workers in this area. It is both detailed and comprehensive, but nevertheless remains perfectly readable. The opening chapters provide a clear background to the volume by explaining the ionic basis of cardiac action potentials and the electrophysiological effects of \(\beta\) adrenoceptor agonist and antagonist agents. The important concept of ventricular fibrillation threshold and its dependence on ischaemia and sympathetic activity is discussed, and forms a logical argument for the cardioprotective mechanism of \(\beta\) blockers. These ideas are expanded to form the rationale behind the use of \(\beta\) blockers for the treatment of arrhythmias in the setting of ischaemic heart disease and cardiac failure. Comparative clinical pharmacology and pharmaceutics of \(\beta\) antagonists are then discussed with reference to the additional properties of \(\beta\), selectivity and membrane stabilising and partial agonist activity. Most of the volume applies these fundamental concepts to the clinical setting and evaluates the role of \(\beta\) blockers in acute myocardial infarction and as primary antiarrhythmic agents. Interrelationships between catecholamines, myocardial ischaemia, and ventricular arrhythmias are emphasised, along with the beneficial effects of \(\beta\) blockade on risk of sudden death and the circadian rhythm of cardiovascular events. Conflicting effects of drugs on suppression of ventricular ectopy and the prevention of cardiac mortality are discussed with particular reference to the CAST study. The potential role of \(\beta\) blockers as primary antiarrhythmic agents is evaluated by discussing the clinical experience with conventional agents in this setting by comparison with the effect of class I agents and sotalol. The final chapter is a concise but comprehensive review of sotalol and its unique combination of \(\beta\) blocking and class III activity. Overall, the book is heavily clinically orientated, but throughout remains based on fundamental electrophysiological principles. It is an excellent overview of this area and is highly recommended.—NMW


This volume is one of a number comprising a series entitled "Fundamental and Clinical Cardiology". The stated aim of the editor was to bridge the gap between current concepts of the effects of disease on the cardiopulmonary system and the level of physiologic (sic) knowledge likely to be encountered in junior medical staff employed in what he terms the CCU (critical care unit). The 23 chapters (17 of which are contributed by the editor) are distributed between five sections, the logic for which is not immediately apparent. Thus part I, in dealing with mechanical concepts in cardiopulmonary physiology, includes a good deal of physics, much of which has limited clinical relevance. Part II includes detailed descriptions of different categories of pressure transducers, an electronic circuit diagram or two, and much complex mathematics relating to the principles of cardiac output measurement. The chapter dealing with regulation of peripheral blood flow covers the clinical implications of supply dependency of oxygen uptake, a hot potato among the critical care fraternity at present, in only a couple of pages and mentions little of the controversy surrounding this area, in which an understanding of basic physiology might influence clinical management in a major way. The pulmonary section was by far the best for my money, for the first time (in the chapter on respiratory muscle weakness) linking physiology to physical findings on examination and incorporating a well written contribution on gas exchange. Unfortunately for your reviewer, this represented an oasis of clinical relevance in an otherwise featureless sea of physiology, much of it explained in mathematical terms. In my experience, junior medical staff in the intensive care unit favour books that identify a clinical problem and indicate appropriate management options, each justified in physiological terms. By contrast, this book starts with the physiology and puts clinical medicine very firmly in second place.—TE

**NOTICES**

**British Society for Allergy and Clinical Immunology and VI Charles Blackley Symposium**

A joint meeting of the British Society for Allergy and Clinical Immunology (annual conference 1993) and the VI Charles Blackley symposium will be held from 2 to 4 August 1993 at the University of Nottingham. Details from BSACI conference secretariat, Congress House, 55 New Cavendish Street, London W1M 7RE (tel 071 486 0531; fax 071 933 7599).

**Symposium on scoliosis**

The ninth international Philip Zorab scoliosis symposium, on the theme of evaluating management (including cardiorespiratory aspects), will be held at Queen's College, Cambridge, from 15 to 17 September 1993. Details from the symposium secretariat, 42 Devonshire Road, Cambridge CB1 2BL (tel 0223 323437; fax 0223 460396).

**CORRECTION**

Portable liquid oxygen and exercise ability in severe respiratory disability

In the paper by R M Leach et al (October 1992;47:781-9) we regret an error on page 782, column 2, line 6 of the first full paragraph, which should read "endurance walk and a six minute walk were . . ."
Tuberculosis contact tracing: are the British Thoracic Society guidelines still appropriate?

Andrew Bush

Thorax 1993 48: 191-192
doi: 10.1136/thx.48.2.191-c

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