# Thorax

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1 Anderson HR. Chronic lung disease in the Papua New Guinea Highlands. *Thorax* 1979;34:647–53.
2 Green AB, Brown CD. *Textbook of pulmonary disease*. 2nd ed. London: Silver Books, 1982:49.
3 Grey EF. Cystic fibrosis. In: Green AB, Brown CD, eds. *Textbook of pulmonary disease*. London: Silver Books, 1982:349–62.

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# LETTER TO THE EDITOR

## Bronchiectasis and homozygous ($P_1ZZ$) $\alpha_1$-antitrypsin deficiency

We read with interest the case report by Rodriguez-Cintron *et al* (April 1995;**50**: 424–5) of a man with bronchiectasis who also had $\alpha_1$-antitrypsin deficiency. They concluded that "bronchiectasis must be considered part of the spectrum ... that may be encountered in ... $\alpha_1$-antitrypsin deficiency" and the discussion attempts to establish that the biochemical defect has led to bronchiectasis. We would like to point out that a case report is at best an association and cannot be taken as evidence for a cause and effect.

The three case reports they quote were deemed inconclusive by other workers[1] for various reasons. The patient in the first report[2] had pertussis as a child which is a well known cause of bronchiectasis; the patient in the second report[3] had *Pseudomonas aeruginosa* in his sputum and a sweat chloride level of 77 mmol/l; and the third report had three cases – one again had pertussis as a child, another had bronchiectasis limited to the left base which mitigates against a systemic aetiology, while in the third case sweat levels of chloride were not estimated.[4]

The paper gives a false impression that the previous reports are a solid foundation on which the authors are building, produces a biased representation of the published literature and, lastly, fails to comment on whether a systemic condition such as $\alpha_1$-antitrypsin deficiency could virtually spare one lung.

The authors state that the true frequency of bronchiectasis in $\alpha_1$-antitrypsin deficient individuals remains to be determined. In the multicentre survey of deficient subjects conducted by the British Thoracic Society none of the 126 deficient patients or the 40 deficient relatives had clinical or radiological evidence of bronchiectasis.[5,6] Most had emphysema.

We surveyed all 35 cases of bilateral widespread bronchiectasis in our hospital (who did not have cystic fibrosis, tuberculosis, or any known cause for bronchiectasis) and none had $\alpha_1$-antitrypsin deficiency.[7] The relatively small number of cases reflects the rarity of bilateral bronchiectasis outside the spheres of cystic fibrosis, tuberculosis, or immunoglobulin deficiency.

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1 Barker AF. Alpha-1 antitrypsin deficiency presenting as bronchiectasis (letter). *Br J Dis Chest* 1986;**80**:97.
2 Longstretch GF, Weitzman SA, Browning RJ, Lieberman J. Bronchiectasis and homozygous alpha-1 antitrypsin deficiency. *Chest* 1975;**67**: 233–5.
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6 Hutchinson DCS, Tobu MJ, Cook PJL. Alpha-2-antitrypsin deficiency: Clinical and physiological features in heterozygotes of $P_1$ type SZ: a survey by the British Thoracic Association. *Br J Dis Chest* 1983;77:28–34.
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## Pulmonary diseases and HIV infection

In their review (March 1995;**50**:294–302) Mitchell and Miller rightly identify pneumothorax as a relatively common complication of AIDS. Although we appreciate the constraints of such reviews in presenting argument, we are concerned about the statement suggesting it advisable to avoid surgery for pneumothorax in HIV positive individuals.

In this setting pneumothorax is frequently recurrent, bilateral, and recalcitrant to conventional therapy. Tube thoracostomy (with or without chemical pleurodesis) remains the first line treatment. However, even with prolonged intercostal drainage there is a high failure rate. In patients without HIV infection failure of first line treatment and recurrence of pneumothorax are both indications for surgical intervention. To withhold such treatment in HIV positive individuals requires us to show that such treatment is contraindicated. We do not believe this to be the case.

Previous studies have shown that surgical intervention is both successful and well tolerated.[1-3] Subsequent postoperative stay is short[1,2] and the incidence of recurrent pneumothorax low.[1,3] These authors have therefore supported the role of surgery in this situation.

While most HIV infected individuals with pneumothorax have advanced disease and therefore shortened life expectancy, we do not think this in itself is a contraindication to surgery. Conversely, if surgery reduces the need for long periods in hospital, often debilitating in itself, short life expectancy may actually strengthen the argument.

Patient selection is, of course, critical; there will always be individuals who are not suitable surgical candidates. We believe, however, that surgery does have an important role. If surgery is avoided carte blanche we are potentially doing our patients a disservice.

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1 Gerein AN, Brumwell ML, Lawson LM, Chan NH, Montaner JS. Surgical management of pneumothorax in patients with acquired immunodeficiency syndrome. *Arch Surg* 1991; **126**:1272–7.
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# BOOK NOTICE

**Long-Term Oxygen Therapy.** Walter J O'Donohue, Jr. (Pp 416; $150.00). New York: Marcel Dekker, 1995. 0 8247 9499 0.

Two randomised controlled trials have shown that long term oxygen therapy (LTOT) improves survival in patients with chronic obstructive pulmonary disease (COPD). However, 15 years after publication of these results there are many questions still to be answered about the mechanisms of these improvements, the optimum prescription of LTOT, and the nature of oxygen technology. This monograph discusses the physiological basis for the use of oxygen therapy and the clinical applications in both adults and children with chronic hypoxaemia. The book is mainly concerned with patients with COPD as there is little information currently available on the outcome of LTOT in patients with other respiratory diseases.

The book starts with an excellent and personal chapter on the historical aspects of LTOT by Dr Tom Petty. The chapter has some entertaining anecdotes on the development of the service and is illustrated with photographs from LTOT centres throughout the world that have been visited by the author. There are chapters on the scientific basis and indications for oxygen therapy that are rather repetitive, with the same graph appearing in different chapters. A useful chapter on the neuropsychological aspects emphasises that LTOT has not yet been shown to improve the quality of life, even though in other chapters some authors add improvement in quality of life to their list of expected benefits from the use of LTOT. There are comprehensive chapters on the effect of oxygen on exercise and oxygen delivery systems, though the emphasis is on the technology currently available in North America. Travel for patients on LTOT poses particular problems and this is covered in considerable detail towards the end of the book.

The main value of this text is for reference purposes, and some of the major studies are described in some detail, which is useful for anyone entering the field of oxygen therapy. The book is well referenced and illustrated. Almost all the contributors are American and thus most of the practical information about oxygen delivery systems is only applicable to practice in North America. Despite over 50 years of research into oxygen therapy, provision of LTOT is still variable around the world and the future challenge is to identify factors that will lead to a definite improvement in quality of life. – JAW

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