LETTERS TO THE EDITOR

Vocal cord dysfunction and wheezing

We read with interest the editorial by Drs J Goldman and M Muers (June 1991;46: 401–4). Although we agree with most of the review, we would disagree with their comments on hypoxaemia. They state that absence of hypoxaemia in vocal cord dysfunction helps to differentiate this condition from acute asthma.

We have seen two cases ourselves[1] where hypoxaemia is a feature of vocal cord dysfunction. In one case the hypoxaemia (arterial oxygen tension Pao_2) 79 mm Hg with 100% inspired oxygen) and the induced "wheeze" were immediately relieved by intubation without recourse to assisted ventilation. On another occasion a similar effect was produced by the administration of a small dose of anaesthetic.

Nolan *et al* 1989[2] report four patients, three of whom had demonstrable hypoxaemia during an acute episode (Pao_2 8·45, 5·18, and 6·1 kPa respectively). Appelblatt *et al* 1981[3] report three patients who had hypoxaemia during an acute attack (Pao_2 50, 52, and 44 mm Hg). Finally, in the report by Christopher *et al* 1983[4] cited in the editorial the alveolar-arterial oxygen tension gradient was reported as normal in all five patients yet two young and apparently otherwise fit subjects had a Pao_2 below 70 mm Hg.

We conclude that the comments regarding hypoxaemia are inaccurate and may lead to inappropriate management. As the authors point out, failure to make the correct diagnosis could result in potentially harmful iatrogenic complications.

Thus in our experience and that of other authors the presence of abnormal blood gases does not exclude a diagnosis of vocal cord dysfunction. We would however support the second summary conclusion, that any acute attack should be treated as asthma unless or until there is objective evidence to the contrary.

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1 Niven RMcL, Roberts T, Pickering CAC, Webb AK. Functional upper airways obstruction presenting as asthma. *Respir Med* (in press).
2 Nolan NT, Gibney N, Brennan N, FitzGerald MX. Paradoxical vocal cord motion syndrome in asthma. *Thorax* 1985;40:689.
3 Appelblatt NH, Baker SR. Functional upper airway obstruction, a new syndrome. *Arch Otolaryngol* 1981;107:305–7.
4 Christopher KL, Wood RP, Eckert RC, Blager FB, Raney RA, Souhrada JF. Vocal-cord dysfunction presenting as asthma. *N Engl J Med* 1983;308:1566–70.

AUTHORS' REPLY We wish to thank Drs Niven and Pickering for their interest in our paper. We consider that our views on patients with vocal cord dysfunction and wheezing are in fact quite similar. There is no doubt that hypoxaemia can occur in association with this syndrome, but we believe this to be the exception and not the rule. We did in fact state that hypoxaemia had occurred in the patients of Appelblatt *et al* (their reference 3). We still believe that the absence of hypoxaemia is usually helpful in differentiating vocal cord dysfunction from acute severe asthma. The presence of hypoxaemia does not, however, *exclude* vocal cord dysfunction. When faced with a wheezing hypoxaemic patient we feel it safest to recommend treatment for asthma, though this policy will very occasionally lead to unnecessary treatment. Physicians attending patients in whom they have diagnosed vocal cord dysfunction and who subsequently present with hypoxaemia may be confident enough to "administer a small dose of anaesthetic" but we believe that this advice cannot be given in an editorial to those less familiar with the condition. We stand by our statement that hypoxaemia is a useful indication that asthma is *likely* to be genuine, although perhaps "in nearly all patients" might be added in anticipation of Dr Niven and colleague's case report.

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Intercostal arteriovenous fistula due to pleural biopsy

Dr J-H Lai *et al* report the development of a traumatic arteriovenous fistula after closed pleural biopsy (December 1990;45:976–8). In 1960 J Elkington and I reported a suspected arteriovenous aneurysm of intercostal vessels following simple pleural aspiration in a hypertensive young woman (*Thorax* 1960;15:266–7). But when the pleural cavity was later exposed during a Smithwick operation for sympathectomy the murmur was found to be due to a small traumatic arterial aneurysm only. Clearly the cutting edge of an aspirating needle is sufficient to induce such a lesion if it is inappropriately directed.

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AUTHORS' REPLY Abnormal communications between arteries and veins may arise from rupture of an arterial aneurysm into the adjacent vein, developmental defects, inflammatory necrosis of adjacent vessels, and penetrating injuries that pierce the wall of artery and vein.[1] The Cope needle used for pleural biopsy and the common needle used for aspiration may all cause this if they are inappropriately introduced. An arteriovenous fistula due to rupture of an arterial aneurysm into the adjacent vein may take longer and depend on different mechanisms from those of fistulas due to penetrating injuries through the artery and vein. In our case we clearly showed the intercostal vein in the early arterial phase as well as a pseudoaneurysm formation. Isolated arterial aneurysm can not therefore be the cause. Nevertheless, we agree that the cutting edge of an aspirating needle inappropriately directed is sufficient to induce such a lesion. When we presented our case to our Chest Medicine Society no one had had a similar experience. Thus although traumatic arterial aneurysm or arteriovenous aneurysm may occur after invasive procedures they appear to be rare.

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1 Cotran RS, Kumar V, Robbins SL. *Robbins' Pathologic basis of disease*. Philadelphia: 4th ed. Saunders, 1989:556.

BOOK NOTICE

Clearing the Air. A Guide for Action on Tobacco. M Raw, P White, A McNeill. (Pp 151; £6.95.) London: British Medical Association for the World Health Organisation Regional Office for Europe, 1990. ISBN 0 7279 0287 3.

In the foreword to this wonderful book we are reminded that "Tobacco kills worldwide more than 2.5 million people prematurely every year. This is an appalling, almost unimaginable toll of suffering and death, representing a waste of human resources which ought to be unacceptable in a civilised society." The authors of this book then seek to enthuse, inform, and encourage their readers in the struggle against tobacco. They are remarkably successful in this aim. Most of the book is taken up by 10 "case studies" of initiatives, policies, and events that have shaped recent smoking control history. The topics include the enormously influential compilation of statistics of smoking related diseases in the United Kingdom called *The Big Kill*, with the numbers of affected individuals broken down according to individual health authorities and parliamentary constituencies, and the shameful episode of Shoal Bandits (when the Government subsidised a new tobacco factory in Scotland, built by US Tobacco to manufacture oral tobacco snuff). Some of the case histories are an inspiration—for example, the Victorian Tobacco Act—and others rather depressing—for example, the voluntary agreement between the tobacco industry and the Government. Throughout this book there is much good advice on how things can be achieved, and the names and contact addresses of all of the key individuals and organisations are listed. There is also an enormously useful book list. This book is written with an authority that reflects the direct experience and commitment of the authors working in the field of tobacco control. In my view it should be required reading for anyone interested in combating smoking.—JM

CORRECTIONS

Carboxyhaemoglobin in women exposed to different cooking fuels
In the paper by Dr D Behera and others (May 1991;46:344–6), on page 344 in line 13 of the abstract, 7·52% should be 7·49%, and on page 345 in line 10 of "Results" ($p = 0·33$) should appear after "users."

Prevalence of asthma among 12 year old children in New Zealand and South Wales: a comparative survey
In the paper by Dr DMJ Barry and others (June 1991;46:405–9) on page 407, column 1, line 17, 3% should be 31%.