abolition of airways obstruction usually takes about 7-10 days and your article was misleading when it referred to the maximum effect of an oral steroid being evident after 9-12 hours. You recommend that, because infection is a common precipitating cause of severe asthma, a broad-spectrum antibiotic should be given. Many studies^{2,4} have shown that asthma is commonly provoked by respiratory viruses and antibiotics can be of value only if there is secondary bacterial infection.

The most difficult problem in the emergency management of asthma is to assess its severity. Usually the patient is first seen by a GP and therefore the vital initial assessment has to be made on clinical grounds alone. It is unsafe to assume that an attack is only mild if the patient can converse with frequent pauses and it may be very severe long before he has become too dyspnoeic to speak at all.

Management does not end with the patient's recovery and the return of his ventilatory function to its optimum value. Every attack of severe asthma should be regarded as a failure in his previous medical care and a searching inquiry must be made into the circumstances leading up to it in order to avoid a repetition of these. The whole emphasis in the management of asthma should be upon teaching patients to treat themselves, using their drugs intelligently and effectively. Those taking an oral steroid should temporarily increase their maintenance dose on their own initiative whenever they have an exacerbation of asthma from whatever cause. Those taking a steroid aerosol must be given a reserve supply of oral steroid, which they should take in high dosage for a few days if they become unable to inhale their aerosol effectively and are thus deprived of any steroid protection.⁵

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- 1 Gregg I, *Respiration*, 1969, 26, suppl 123.
- 2 Lambert, H P, and Stern, H, *British Medical Journal*, 1972, 3, 323.
- 3 Minor, T E, et al, *Journal of the American Medical Association*, 1974, 227, 292.
- 4 Gregg, I, in *Royal College of Physicians of Edinburgh Symposium on Viral Diseases*, ed A T Proudfoot. RCPE Publication No 46, 1975.
- 5 Hodson, M E, et al, *American Review of Respiratory Diseases*, 1974, 110, 403.

SIR,—While subcutaneous adrenaline injection is admittedly easy to administer it could actually exacerbate bronchospasm in acute asthma. Because of beta-adrenergic blockade in severe asthma the alpha receptors may be even more markedly stimulated than the beta by adrenaline injection.¹ I do not agree, therefore, with Dr F Harris (1 November, p 288) that your leading article (11 October, p 65) was "remiss in not mentioning the role of subcutaneous adrenaline in the management of acute asthma."

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- 1 Sodeman, W A jun, and Sodeman, W A, *Pathologic Physiology*, 5th edn. Philadelphia, Saunders, 1974.

SIR,—In your leading article on the management of acute asthma (11 October, p 65)

you do not seem to stress that the treatment advised is essentially for adult cases. Furthermore, it appears to me that you have preferred to use the term "acute asthma" rather than "status asthmaticus." The word "asthma" still defies generally agreed medical definition. Status asthmaticus, however, is widely accepted as implying an acute attack of asthma resistant to the usual bronchodilators. Why then do you introduce a further term?

In contrast to adults, many children show moderate to severe hypoxaemia with hypercapnia¹ and slight to moderate metabolic (non-respiratory) acidosis. Oxygen therapy may, however, only worsen respiratory depression in children with hypercapnia. Viral respiratory tract infections may precipitate asthmatic attacks in children^{2,3} but there is little evidence to suggest that bacterial infection is associated with wheezing. Status asthmaticus is common in children, and certainly so in our area. I do not use antibiotics as a routine and find it difficult to accept your advice on the use of broad spectrum antibiotics.

Corticosteroids are no doubt the drugs of choice. These are unlikely to be required for more than a few days in most cases. Is there any evidence to suggest that oral potassium supplements must be given with such short courses of prednisolone?

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- 1 Downes, J J, Wood, D W, and Pitman, J C, *Pediatrics*, 1968, 42, 238.
- 2 Minor, T E, et al, *Journal of Pediatrics*, 1974, 85, 472.
- 3 McIntosh, K, et al, *Journal of Pediatrics*, 1973, 82, 578.

Epidemiological aspects of choriocarcinoma

SIR,—Your leading article (13 September, p 606) on the epidemiological aspects of choriocarcinoma very generously credited my colleagues and me with having shown that choriocarcinoma was more common in Indians and Eurasians than in Chinese and Malays in Singapore. Our paper¹ did not in any way discuss choriocarcinoma in Singapore: it was on the epidemiology of hydatidiform mole in Singapore. While the incidence of hydatidiform mole in Chinese and Malays was 1 in 811 (151 out of 1 427 000 pregnancies) and 1 in 879 viable pregnancies (39 out of 276 000 pregnancies) respectively, the incidence in Indians and Eurasians was 1 in 680 (20 out of 156 000 pregnancies) and 1 in 310 viable pregnancies 3 out of 53 800 pregnancies) respectively. The higher incidence of hydatidiform mole in Eurasians is thus more apparent than real. These differences in incidence of hydatidiform mole do not apply to choriocarcinoma, which is a different condition.

In contrast to the assertion in your leading article that choriocarcinoma was more common in Indians and Eurasians than in Chinese and Malays in Singapore, studies carried out on choriocarcinoma arising from different forms of antecedent pregnancy showed that choriocarcinoma was more common in Malays than in Chinese.^{2,3} In fact, the incidence of choriocarcinoma in Indians and Eurasians was the lowest, but this was more apparent than real as the numbers in these two ethnic groups were small.³

A study in London showed that the risk of choriocarcinoma was higher in women with blood group A whose partner was blood group O than those whose partner was blood group A.⁴ At the same time a study in Singapore on both hydatidiform mole and choriocarcinoma showed that hydatidiform mole had no special selection for any particular blood group but choriocarcinoma was significantly more common in patients with blood group A and that choriocarcinoma patients with blood group AB responded poorly to chemotherapy.⁵ The role of HLA antigen in trophoblastic disease has not been clearly established. In 52 patients with hydatidiform mole HLA antigen typing in both the patients and their spouses showed no obvious histoincompatibility. However, there was a preponderance of HLA-9 antigen in these patients with hydatidiform mole.⁶ When the HLA antigen types for different parts of the world were examined it emerged that HLA-9 was more common in those parts of the world such as the Far East, Mexico, and Greenland where molar pregnancies are more frequent than in other parts of the world. This would suggest some sort of genetic or immunological factor towards risk of developing hydatidiform mole. The full significance of the above observation of HLA-9 and molar pregnancies remains to be explained and further investigations along this line are likely to be fruitful.

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- 1 Teoh, E S, Dawood, M Y, and Ratnam, S S, *American Journal of Obstetrics and Gynecology*, 1971, 110, 415.
- 2 Teoh, E S, Ratnam, S S, and Dawood, M Y, *Acta Obstetrica et Gynecologica Scandinavica*, 1971, 50, 247.
- 3 Teoh, E S, Dawood, M Y, and Ratnam, S S, *Obstetrics and Gynecology*, 1972, 40, 519.
- 4 Bagshawe, K D, et al, *Lancet*, 1971, 1, 553.
- 5 Dawood, M Y, Teoh, E S, and Ratnam, S S, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1971, 78, 918.
- 6 Dawood, M Y, Joysey, V, and Loke, Y K, unpublished data.

** We thank Dr Dawood for pointing out our mistake. The London and Singapore series he refers to agreed about the lack of effect of blood groups on the occurrence of hydatidiform mole and about the adverse effect of blood group AB on choriocarcinoma.—ED, BMJ.

Medical training in developing countries

SIR,—May I comment on the important article by Dr B Senewiratne and his colleagues (4 October, p 27)? My original support for the idea of altering the medical course in order to stem the flood of emigration and serve the needs of the developing country better (26 April, p 190) concerned the emphasis of the course and not its basic structure. Medical education within Britain itself is in a state of flux, as the article by Dr J S P Jones on the Nottingham medical school (4 October, p 29) shows; should then the teaching in developing countries be based on a probably outdated structure? The feeling even in Britain is more and more that the student must get out of the hospital and into the community; surely this is much more the case in the developing world.