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CORRESPONDENCE

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

Beta-blockers in asthma

SIR,—The reminder from Dr June M Raine and others (14 February, p 548) that beta-blocking drugs can precipitate serious asthma and death in vulnerable patients was useful. Although these complications are more likely to occur with non-selective drugs such as nadolol they can also occur with cardioselective drugs and no beta-blocking drug should be considered free of risk.

I wonder whether, in retrospect, a more aggressive approach with salbutamol should have been tried initially in the patient they describe. Beta-agonists and beta-blockers are competing for bronchial and other beta-receptors so it should be possible to overcome beta-blockade with large doses of beta-agonists. In normal subjects a 60-fold increase in the dose of salbutamol was necessary to achieve bronchodilatation after 80 mg propranolol.[1] A similar increase would be expected in asthmatic patients; so that doses of the order of 10 mg salbutamol every 10 minutes by inhalation and intravenously are probably needed, and should be given until bronchodilatation occurs—heart rate, electrocardiogram, and blood pressure permitting. Although the dose sounds alarmingly high, side effects should not occur since the other actions of salbutamol would also be blocked by the beta-blocking drug; and this has been our experience with normal subjects. Isoprenaline would reverse the cardiac effects of beta-blockade more effectively, but if given alone may reverse these and cause $beta_1$-stimulation before reversing bronchial beta-blockade. The relative amounts of salbutamol and isoprenaline to be given will depend on the clinician's assessment of whether bronchial or cardiac beta-blockade is contributing more to the patient's condition. In addition to salbutamol and steroids, inhaled ipratropium or atropine is worth trying and probably aminophylline, though its bronchodilator effect may be attenuated by propranolol[2] and its cardiovascular effects in the presence of propranolol may not be beneficial.

An alternative approach would be to try the effect of prostaglandin E_2 (PGE_2), which stimulates adenylate cyclase through a receptor separate from the beta-receptor and so should bypass the effects of beta-blockade, which it does in normal subjects.[3] This theoretically attractive approach has not, to my knowledge, been used in ill patients so would need to be tried cautiously, particularly as PGE_2 is rather irritant to inhale.

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[1] Gribbin HR, Baldwin CJ, Tattersfield AE. *Br J Clin Pharm* 1979;7:551-6.
[2] Mackay AD, Baldwin CJ, Tattersfield AE. *Thorax* 1980;35:239.
[3] Lewis RA, Seth RV, Tattersfield AE. *Clin Sci* 1981; 60:17P.

SIR,—Although the dangers of adrenoceptor beta-blocking drugs in asthma were pointed out nearly 20 years ago[1] these risks are still not widely enough appreciated as Dr June M Raine and her colleagues point out in their report (14 February, p 548) of a near-fatal attack of bronchospasm in an asthmatic given nadolol for hypertension.

Dr Raine suggests that if a beta-blocking drug is considered essential in an asthmatic patient treatment should preferably be started under medical observation with peak flow monitoring. I think that this is wise, but it may not always be enough. In an asthmatic subject the acute bronchoconstrictor response to a beta-blocking drug can vary substantially from time to time.[2] Hence even a small or absent response to one or more test doses does not guarantee that a severe bronchoconstrictor response to the same dose will not occur at a later date. Such a severe reaction may be especially likely if the patient is simultaneously exposed to other bronchoconstrictor influences, such as cold or exercise.[3]

In most instances the bronchoconstriction induced by beta-blockers in asthma promptly reverses, wholly or partly, with aerosol administration of a beta-stimulant bronchodilator drug. Hence before giving a beta-blocker to an asthmatic it is important to ensure that the patient has an adequate aerosol technique and that he actually carries an inhaler. The dose of bronchodilator (beta-agonist) can be double or treble the usual dose to overcome the antagonist.

Occasionally, as in the present case, the bronchoconstriction does not respond even to large doses of $beta_2$-stimulant drugs. A possible explanation for this might be that the bronchoconstriction is not a consequence of beta-blockade at all but results from some