Altounyan's discovery in 1958 that a derivative of Khellin would inhibit the asthmatic response of his own bronchi to inhaled allergen and the subsequent development of sodium cromoglycate (SCG) opened a new era in the understanding and management of allergic bronchial disease. The review of SCG presented in this issue (p.164) is the most comprehensive to have been made and it brings together clearly and concisely the extensive literature on this unique drug. By coincidence it appears at a time when another powerful therapeutic weapon — the corticosteroid aerosol — has been developed for the treatment of asthma (see Drugs 6: 84, 1973). The modes of action of SCG and corticosteroid aerosols are totally different, and yet in clinical use they can both overlap and complement each other.

Pharmacologists and immunologists have now been able to define the action of SCG with some precision. It is believed to stabilise tissue mast cells against degranulation caused by antibody-antigen reactions which take place on their surfaces, and thereby prevents the release of substances which induce the asthmatic reaction. Mast cell degranulation plays an important part in the pathogenesis of both asthma and rhinitis in atopic subjects who produce excess reaginic antibody of the IgE class against inhaled allergens such as house dust or pollen. It is in these patients that SCG might be expected to be of most benefit, and much clinical experience has confirmed this. SCG can be used in such patients in an entirely rational fashion, that is, as a prophylactic drug to prevent the effects of antigen challenge. For instance, in a person with multiple allergies it will probably be necessary to give regular treatment with SCG, whereas in a patient with allergy to a specific seasonal pollen, it may only be necessary to give SCG at the time of exposure to that particular allergen. A common symptom in atopic subjects is exercise-induced asthma, which often responds dramatically to SCG inhaled immediately before exercise. This has led to the suggestion that
exercise-induced asthma may be due to mechanical disruption of sensitised (?) unstable) mast cells caused by increased lung movements during exercise. It is important to remember that the prophylactic effect of SCG can be overwhelmed by heavy allergen challenge and also that in some patients shortening the interval between administration may lead to improved response [1].

In addition to patients with 'extrinsic' asthma, SCG may be of considerable benefit to many late onset ('intrinsic') asthmatics and also to certain patients with chronic bronchitis. In view of this response, it seems likely that mast cell degranulation plays a part in the pathogenesis of these diseases.

Although much is known about the mode of action of SCG, there are still no laboratory or clinical criteria which enable the clinician to predict with certainty which patients will respond to the drug. In both asthmatics and bronchitics the presence of sputum eosinophilia and strong evidence of an allergic component possibly has the best predictive value, and the occurrence of nocturnal attacks of cough and tightness should raise suspicion of an SCG-responsive element. Chronic bronchitis in a young person or in a non-smoker should also raise the same suspicion. In the absence of precise criteria, many feel that nearly all patients with chronic bronchitis, with or without airway obstruction, as well as those with asthma, should be given a short trial of SCG, for example 4 capsules daily for 1 month. This should be followed by a similar trial of corticosteroids in patients failing to respond satisfactorily to SCG (vide infra). A trial of SCG is also indicated in patients already receiving oral corticosteroids, in whom it may be possible to obtain a useful reduction of the steroid dose, and very occasionally to withdraw the corticosteroid completely. In these trials, careful assessment based mainly on subjective response supported by a symptom diary card and records of usage of other supportive therapy, is an essential, but often neglected requirement.

Unfortunately, there remain patients who respond poorly or not at all to SCG and who require corticosteroid therapy. It is in this group that the new aerosols have an important place. These aerosols contain corticosteroids of high topical potency (e.g. beclomethasone dipropionate and betamethasone 17-valerate) which have been used for several years in the treatment of inflammatory and eczematous skin conditions. Even though they are absorbed from the lung and gastro-intestinal tract after inhalation, they are so active that a local therapeutic effect can be achieved at doses too small to cause suppression of the hypothalamic-pituitary-adrenal axis and other systemic side-effects [2,3,4]. It is established that these aerosols can improve the majority of patients with asthma of both the atopic and late onset types; they may allow patients dependent on oral corticosteroids to reduce their oral dose significantly, or to withdraw them completely in a few cases. As with SCG, corticosteroid aerosols are not indicated in the treatment of status asthmaticus, as it is not possible to deliver an adequate dose by the inhaled route; in status asthmaticus systemic