

A PHYSICIAN'S VIEW OF ALLERGY

By

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There is no rational concept of allergy and atopy, and allergists are still on the defensive after thirty years. Internists rightly demand that the allergy of the atopic minority shall be integrated with the physiological pathology common to all. Until this is done the conscientious physician-allergist cannot be at ease in his practice, and the fascinating problem of what allergy is be solved.

I have sought an answer in the clinical field and I present here certain concepts which may help. They satisfy me at general level, and most criticism at clinical detail level is answerable.

My reference is to atopic and principally respiratory allergy; and I define an allergic as an individual in whom a positive skin test to foreign, non-multiplying, and normally non-toxic protein is demonstrable.

The lesion produced in this skin test is the histamine wheal. It is the basic lesion of all atopic disease. It comprises vasodilatation, fluid exudation and specific cellular infiltration, and is a response to a special kind of irritant. Septic inflammation has these same fundamental characters.

The allergic lesion functionally destroys antigen and prevents its further ingress into the body; these are also the functions of antimicrobial inflammation. It is postulated still, I think, that there is a chemotaxis which determines the occurrence of septic inflammation; histamine is the known chemotaxin of allergic lesions.

There is so much parallelism between the two processes that

it is logical to infer that the allergic lesion is an inflammatory one of a special kind. But if this is so then why is it only found in atopic individuals and very rarely in other forms of life than man, and why cannot such inflammatory response be adduced in those non-atopic creatures?

Atopy, or capacity for allergy, is actually found in about 25 % of people and a family history is readily elicited in 80 % of cases of allergic disease. 25 % is the Mendelian recessive number. It is a tenable hypothesis that this capacity for allergy is a physiologic norm in genetically determined persons, the gene having arisen as a permanent variant late in the evolution of homo sapiens.

As a protective mechanism the atopic response is a very imperfect one. Repeated exposure is necessary for its appearance and the development of immunity is a very slow process; on the way to the immunity the local response in about one-third of atopics rises to such a degree as to cause sufficient swelling as to produce obstruction and thereby symptoms and disease.

The majority of allergics never suffer from allergic disease. Many apparently normal people have an allergic nasal mucosa and not only never have symptoms, but in later years have normal mucosae. Many people have years of hay fever and then it disappears without treatment or change of environment; three fourths of asthmatic children grow out of their disease. The milk-egg-cereal eczema of infants spontaneously disappears at the age of two. These are all evidences of a slowly developing immunity to specific irritants.

Lactalbumin is the first foreign protein which the baby meets, it is the first of life's allergens. Milk substitution benefits 20 % of asthmatic infants, 5 % of preschool age, 2 % of school age and about .5 % of asthmatic adults. Positive skin test to milk occurs in about the same ratio but the age incidence is many years later. Clinical immunity is gained with passage of time and repetition of exposure, but perfection of antibody and total immunity take many years more.

Allergic disease in its pure form has a clear story of gradual

increase over years, a plateau period of severity, and a gradual wane. This is characteristically seen in juvenile asthmatic bronchitis and in pollinosis. Pollinosis patients are known to have positive skin tests for years before they complain, and these tests persist for years after the disease has disappeared: eventually they too disappear.

Certain recognized characters of allergic disease must be considered; episodicity, psychosomatism, infection, response to steroid.

Juvenile asthmatic bronchitis is a disease characterized by the regular recurrence of colds which gradually increase in severity, until they come to be associated with bronchitis, then with fever and bronchospasm. After a series of attacks on this level the severity wanes until the child is left with allergic rhinitis, spasmodic asthma, both, or, and usually, no disease at all. This pattern clearly illustrates the gradual wax and wane of the allergic response.

Juvenile asthmatic bronchitis is a disease of patterns in both group and individual. In the group, attacks are as like as those of any infectious disease. In the individual successive attacks are as like as those of migraine. Two days dispositional change, two days coryza, three days fever and bronchospasm, fourteen days of gradual resolution. The whole attack lasts three weeks which is the usual duration of the whole attack of episodic asthma. Between the attacks there is complete freedom from any signs of the disease. Periodicity, if sought, is establishable in 80 % of cases; only one-fourth are accompanied by purulent secretions and the introduction of antibiotics has made no difference to the course of the attack or the disease. These children get colds without having asthma and many of the colds which are followed by asthma are not caught from other people. The fever and the raised ESR are evidences of inflammation not infection. Two doses of ACTH in the incubation period will abort the attacks in most cases.

This disease presents the picture of a specific toxæmia which repeats itself rhythmically as no other infection does, or of a rhythmically recurrent metabolic change during which the

patient responds to some constant irritant with a patterned inflammatory response. The evidence is I think all in favour of the latter.

This pattern of phasic reactivity and refractoriness is, I think, recognized as being characteristic of the atopic response. Such a rhythm must be due to a metabolic tide of some sort and such tides must be hormonal in origin. This theory is borne out by the effect of ACTH in the prodromal period of juvenile asthmatic bronchitis. There is apparent reversal of the reactionary status. ACTH would surely facilitate and ultimately increase bacterial or viral inflammation due to infection. What then possibly is this hormonal origin? It is important to know, for if it be acceptable that allergy is a variant on normal inflammation then it must also be acceptable that whatever controls liability to allergic response must also have an effect in infective response. ACTH mediates the hormonal nexus, cortisone simulates the response produced by ACTH. The time relation of response is such that the effect of ACTH cannot be by cortisone production. The only neurosurgery which is apparently effective in asthma is stellectomy, a paradoxical result unless it is obtained by abolition of stimuli to some structure within the cephalic area. Can this be another function of the pituitary?

The psychosomatic element in asthma is essentially an emotional one, thalamic rather than cortical. It is a response to fear. This may be through hormonal activity via the autonomic system, or may be purely neurogenic. Obstructed breathing generates instinctive acute fear, repeated attacks of it both acute and chronic fear. Fear stimulates the vagus and there is provocation to bronchospasm and oedema. Recurrent attacks of acute asthma with associated fear canalize the nerve path and create the vagotonic lung, so that, after much asthma due to allergy, the condition is provokable by other stimulants to bronchial constriction, such as the expiratory efforts of coughing and laughing, and as a conditioned reflex through basal and cortical association tracts. The asthma then becomes a habit spasm unresponsive to allergic methods.

Fear is contagious from mother to child—more so in the mother-dependent child. And so the mother's acute fear and chronic anxiety concerning the child's asthma increase both the asthma and the liability to it. Hence, the value of parental education and confidence in all cases, and of parentectomy in many.

These conclusions are briefly stated, arguments are omitted and further conclusions unsaid on account of time, and the whole oversimplified. The essential concept is that the allergic response is no mystery but is a normal physiological response in a genetically determined individual, and that if this be acceptable and can be reasonably proven, there need be no further conflict between the internists and the allergists; and quite importantly the application of the knowledge we have of allergic inflammation to the study of other inflammatory processes may throw a considerable light on fundamental pathology. I would commend consideration of these concepts to you.

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