

FOOD ALLERGY: ACUTE VS. CHRONIC*†

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ALLERGIC reactions to foods are characteristically acute and transient. From the history the experienced physician can usually suspect the cause and readily reproduce the symptoms by clinical trial with the disguised food. The patient himself is often aware of the cause, particularly when attacks have been recurrent. Scratch tests are usually positive and avoidance of the offending food results in prompt relief within a few hours. This, then, is the typical pattern of food allergy.

However, syndromes such as perennial vasomotor rhinitis, perennial "intrinsic" asthma, chronic urticaria, atopic dermatitis, and chronic gastrointestinal disorders simulate clinically the acute allergic disorders due to extrinsic allergens but present disturbing variations such as confusing histories, negative skin tests, equivocal results following clinical trial with undisguised allergens, and most important of all, chronicity.

Over the years efforts have been made to include the atypical disorders within the allergic hypothesis, and many theories have been advanced to reconcile the obvious inconsistencies. Sincere attempts by many allergists to integrate these theories with clinical experience have been disappointing. Although the concept of acute allergic reactions to food is universally accepted, the hypothesis of chronic food allergy leaves much to be explained. It is most difficult to be certain that chronic or delayed manifestations can be induced by exposure to disguised foods.

Elimination diets³ and the leucopenic index,⁵ with various modifications, have been advocated as useful means to a solution of these problem cases. More recently, a quantitative increase in a constellation of events² such as sneezing, coughing, increasing fatigue, and muscular aching, has been recommended as a diagnostic criterion of chronic food allergy.

If the theory of chronic or "masked" food allergy is valid it seems likely that similar changes should be observable following the deliberate induction of an acute allergic reaction to food. Therefore, the following experiment was done:

L. Q., a 31-year-old man, an asthmatic, clinically and skin-sensitive to cottonseed, ingested cottonseed protein in gradually increasing amounts until asthma was induced. Serial blood counts were made at frequent intervals before, during, and after the attack. No significant variation in the circulating leucocytes was observed. Fig. 1 graphically summarizes the experiment.

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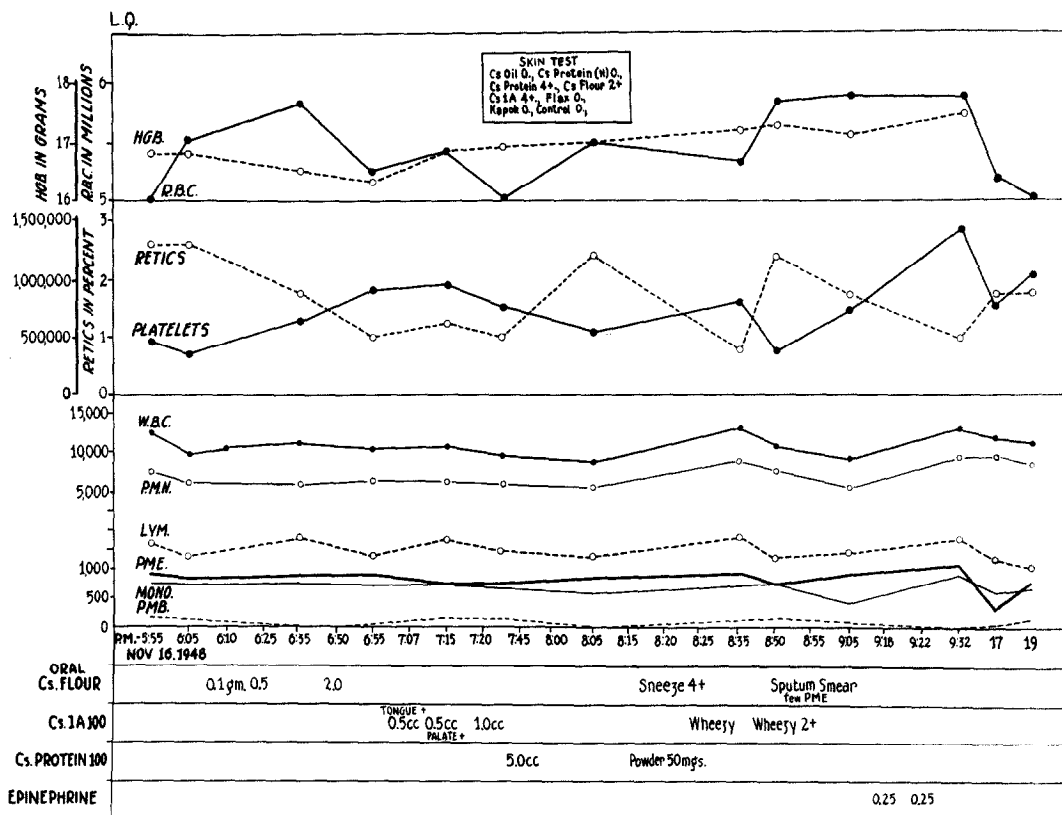


Fig. 1.

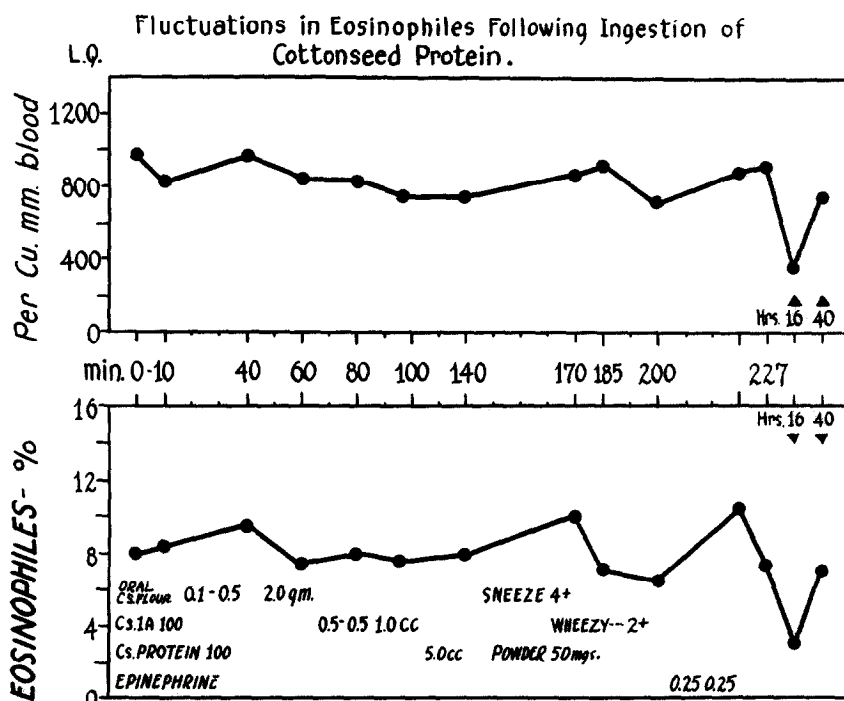


Fig. 2.

Fig. 2 has been prepared on a scale to exaggerate eosinophile fluctuations. With the onset of wheezing (185 minutes) there was a slight decrease in eosinophiles; then a slight increase (227 minutes) after the injection of epinephrine 1:1000, 0.25 c.c. $\times$ 2 for the control of asthma. These variations were not of sufficient magnitude to be considered of diagnostic significance. The patient had received immunizing inoculations of cottonseed protein extract for several years and had developed considerable tolerance. In fact, it was quite

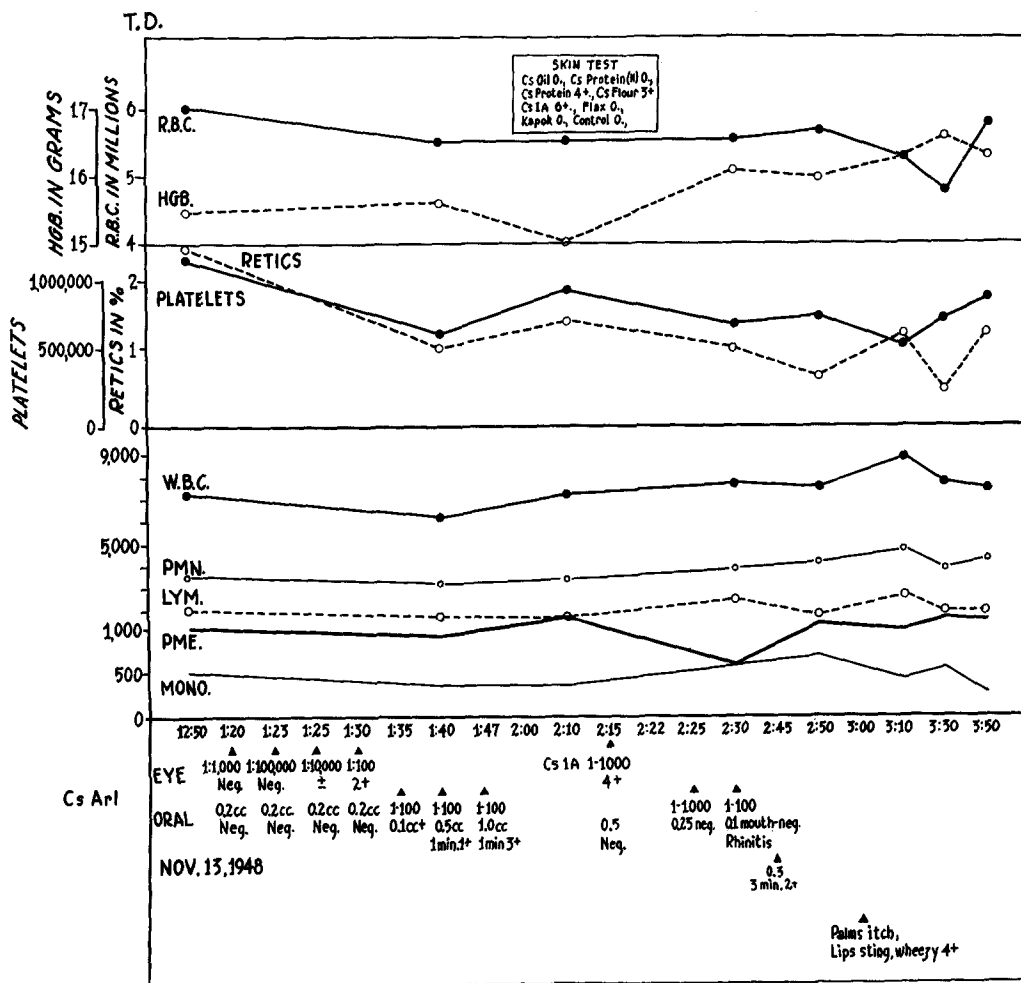


Fig. 3.

a surprise to observe the amount of cottonseed protein he could ingest in aqueous solution. Finally a 50 mg. vial of powder was emptied onto his tongue. Immediately he began to sneeze and within a few minutes was definitely asthmatic. From the rapidity of the reaction it was assumed the powder had been inhaled. So actually, this attack was not induced by ingestion but rather by inhalation of the allergen. Of particular interest, however

was the count taken 14 hours after the onset of asthma showing a decrease of over 50 per cent eosinophiles, due probably to the epinephrine effect. At the next count made 48 hours later, there was a return to the base line.

A similar experiment was repeated in the case of T. D., a 34-year-old man, also a cottonseed-sensitive asthmatic; the results are summarized in Fig. 3. Asthma was induced using liquid preparations of the allergen so that there was no chance for inhalation. Here again, there was insufficient fluctuation in the total leucocytes or in the eosinophiles to be considered diagnostically significant. Fig. 4 gives a high-power magnification of the eosinophile curve.

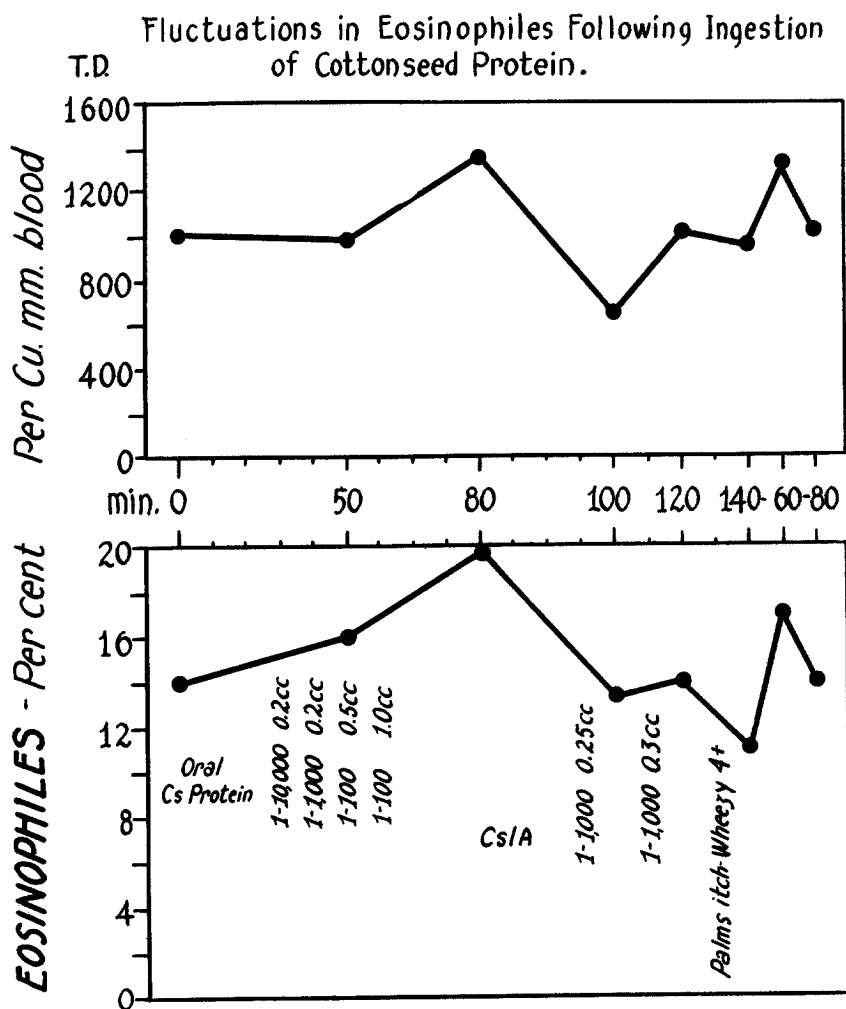


Fig. 4.

Unfortunately this man was not asked to return on subsequent days for further counts. At the time these studies were made, interest centered in the immediate effects of the acute allergic reaction to the allergens rather than the delayed effects of epinephrine described by Thorn.⁴

In addition to blood counts, clinical observations were made; no increase in sneezing or coughing was observed nor was there any mention by the patient of fatigue or muscular aching during the 3-hour period previous to the development of wheezing. Patients had not been warned to be on the lookout for these symptoms; the observer merely made notes of what he saw.

Results of similar studies of 2 patients,¹ where asthma was induced by subcutaneous administration of cottonseed protein, conform with those reported here. There was a surprising stability of the eosinophiles in spite of severe constitutional reactions. The total leucocytes increased sharply immediately following necessary injections of epinephrine but did not fluctuate when the drug could be withheld.

CONCLUSIONS

1. Serial blood studies and observations of minor symptoms appear to be of little aid in anticipating the onset of asthma deliberately induced by the ingestion of cottonseed protein.

2. The circulating eosinophiles did not fluctuate significantly before, during, or immediately after the constitutional reaction.

3. The validity of the theory of chronic or "masked" food allergy is questioned.

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