

SUDDEN DEATH DUE TO ALLERGY TESTS*†

M. COLEMAN HARRIS, M.D., AND NORMAN SHURE, M.D.,
BEVERLY HILLS, CALIF.

THE occurrence of fatal anaphylactic reactions following the injection of foreign protein into the human circulation certainly is not recent in the history of medicine. The first formal report in medical literature according to Kojis¹ appeared in 1667 when the death of a mentally deranged patient after a second transfusion of calf's blood was described. Despite this report and other subsequent reports of similar fatalities, the mechanism by which death was produced in these cases was not understood until 1875 when it was demonstrated that animal serum actually hemolyzed human red blood corpuscles.

In 1894, in a paper presented before the International Congress on Hygiene and Dermography, Von Behring reported his discovery of antitoxin and its effectiveness in the treatment of diphtheria. Following this report, antitoxin came into general use. Its danger soon became apparent, and in 1896 simultaneous reports of deaths following its use appeared in American, British, and German medical literature. During more than fifty years of employment of foreign sera for the prevention and treatment of disease, dangerous and fatal reactions have been rare. In a report covering 350,000 injections, Park² estimated that one acute death occurred each 70,000 injections. Lamson,³ searching the literature for sudden deaths following the injection of foreign protein, was able to find a total of only 41 deaths from 1894 to 1910, and only 14 from 1910 to 1924.

Fatal reactions have followed not only the introduction of animal blood into humans and the use of antitoxin, but also the use of foreign proteins in clinical allergy. Fortunately, these have not been many, most of them occurring after the inadvertent use of an excessive dose of antigen for the treatment of an exceedingly sensitive patient. Deaths following the use of allergens in skin testing for determination of the cause of an allergic condition have been extremely rare. In 1922, Cooke⁴ reported the death of a 3-year-old asthmatic boy following an intracutaneous test with an extract of glue. In 1924, Lamson³ described the case of a 5-month-old child with chronic eczema who died 2 hours after a series of intradermal tests with extracts of milk, egg, oats, and rice. Baagöe,⁵ in 1928, had a similar experience with a patient after testing with egg white. The case of an adult male who died 5 minutes after being tested with an extract of buckwheat was reported by Lamson⁶ in 1929. Freedman,⁷ in 1935, reported a fatal case of anaphylactic shock in a 6-year-old boy following an intracutaneous test to determine sensitivity to horse serum. In 1940, Ferguson⁸ described a fatal serum reaction in another 6-year-old boy after an intracutaneous test with tetanus antitoxin. Among reports from the office of the Chief Medical Examiner of New York, in 1942, Vance and Strassmore⁹ published the case of a 4-year-old girl who died after intradermal tests with silk-

*From the Allergy Section, College of Medical Evangelists School of Medicine, Los Angeles, Calif.

†Read before the Sixth Annual Meeting of the American Academy of Allergy, Los Angeles, Calif., March 6-8, 1950.

worm, sheep's wool, and kapok. Hunt,¹⁰ in 1943, reported a case of sudden death from an intradermal injection of guinea pig hemoglobin. In 1945, Wiseman and McCarthy-Brough¹¹ reported the fatality of a 78-year-old woman following 17 intradermal tests consisting of 16 environmental allergens and one food. Swineford,¹² in 1946, discussed a case of fatal anaphylactic shock following intradermal tests with 56 food and 8 miscellaneous inhalant extracts. The most recent death following skin testing was reported by Blanton and Sutphin¹³ in February, 1949. They reported the case of a 57-year-old woman with asthma who died following a series of 56 intracutaneous tests with foods and animal epithelia.

In the cases cited above, no more than 4 autopsies were performed. In at least 5 cases^{3, 9, 11, 12} multiple tests were done and the exact antigen responsible for the fatality was never determined. In at least 2 cases,^{11, 13} other factors might have been responsible for the death. We are aware of 2 additional deaths which have not been reported formally. One was mentioned by Benson in discussing a paper presented by Bernton at the annual meeting of The American Society for the Study of Allergy held in St. Louis in May, 1939. Benson stated that he had caused the death of a 2-year-old child with allergic dermatitis by injecting the antigen of cottonseed meal in which a mistake had been made in the titer.¹⁵ The other case was mentioned by McGrath while testifying as an expert witness before a hearing on allergy to cottonseed held by the Federal Security Agency.¹⁴ McGrath testified that he had seen a 6-year-old child die before the physician, administering a skin test with cottonseed, could get the needle out of the child's arm. The above constitutes the available reports of fatal reactions following skin testing in allergy. It is probable that other deaths have occurred and have not been reported.

The case we are about to present is significant inasmuch as we were able (a) to verify the cause of death by autopsy, (b) to identify the allergen responsible for the death, and (c) to prove the presence of a circulating antibody corresponding with the responsible antigen.

CASE REPORT

A 25-year-old white married woman, complaining of asthma and hay fever of 20 years' duration, was admitted to the allergy clinic of the White Memorial Hospital. From the age of 5 until 18, she had received injections of house dust and geographically local pollens with satisfactory results. For 6 years thereafter, she was symptom-free and required no treatment. In the year prior to her admission to the clinic, however, there was a recurrence of stuffiness of the nose and mild paroxysms of wheezing dyspnea.

Physical examination disclosed a pale, boggy nasal mucous membrane with a moderate amount of serous secretion. The heart was not enlarged, no murmurs were heard, and the rate and rhythm were normal. Scattered moist râles were distinguished in both pulmonary bases posteriorly. Otherwise, the physical examination was essentially negative. A provisional diagnosis of (1) allergic rhinitis and (2) bronchial asthma was made with a notation that both allergy and infection probably were contributing factors. Intradermal tests were performed with routine testing strengths of local pollen extracts. Mild reactions, 1 and 2 plus (based on a grading of 1 to 4 plus) were obtained to *Cynodon dactylon* (Bermuda grass), *Poa annua* (annual June grass), and *Lolium perenne* (perennial rye grass). An iodide mixture was prescribed and the patient was asked to return the following week for further skin tests.

At the second visit, one week later, the patient reported that she had not experienced any attacks of asthma during the period and was feeling better. Because of the mild reactions to the potent pollen intradermal tests administered previously, skin testing was resumed with extracts of miscellaneous environmental substances. Within one minute after these intradermal tests had been completed, the patient developed severe dyspnea. A tourniquet was applied immediately above the skin test sites and 0.3 c.c. of 1:1000 solution of epinephrine hydrochloride was injected above the tourniquet. The patient was not relieved, and another injection of 0.3 c.c. of epinephrine was given. She still failed to respond and rapidly became cyanotic and unconscious. She was removed to another room on a stretcher and additional epinephrine as well as aminophyllin was administered intravenously. Respiration became increasingly difficult and a gentle flow of oxygen by nasal catheter was instituted. The patient rapidly became moribund. As a last resort, a tracheotomy was performed because of the possible presence of laryngeal edema. However, despite the administration of oxygen through the tracheotomy opening, artificial respiration, and further cardiac and respiratory stimulation, including epinephrine intracardially, the patient expired within 10 minutes after the onset of the initial dyspnea.

AUTOPSY*

Gross Description

External Examination.—The unembalmed body is that of a well-developed, well-nourished, 25-year-old white female. A tracheotomy wound is present in the neck. Her neck and face are quite cyanotic as are the hands and nail beds. There is a subcutaneous emphysema around the neck and upper chest.

Neck.—There is a 4 cm. midline and longitudinal incision in the neck below the thyroid cartilage for the purpose of a tracheotomy. There is some subcutaneous emphysema around this area. The neck vessels appear to be engorged. There is no edema of the epiglottis, glottis, or vocal cords. The tracheotomy is on the right lateral side of the trachea.

Cardiovascular System.—The pericardium has no lesions and on opening contains 10 c.c. serosanguinous fluid. The right auricular appendage of the heart, the right side of the heart, and the superior vena cava are all dilated. The left auricular appendage is small and nubbin-like. The left ventricle is hard, contracted, and small. The heart weighs 225 grams. The valves measure: tricuspid, 11 cm.; mitral, 9 cm.; aortic, 6 cm.; pulmonic, 6 cm. The right ventricular wall is 5 mm. thick and the left ventricular wall 11 mm. The coronary arteries are traced and there is no narrowing; however, the arteries are quite small. The myocardium, on cross section, shows no sign of any scarring or recent infarct. The aorta is perfectly smooth throughout its extent. The blood throughout the body is not clotted and a sample of blood is taken for serum: this does not clot over a period of 24 hours.

Respiratory System.—There are no pleural adhesions. The lungs are fully expanded when the chest is open and are pink with very little anthracotic pigmentation over the surface. The lungs appear billowy and float very high in water; on picking up the lungs, they give the sensation of being very light. The right lung weighs 300 grams, the left, 250 grams. On cutting across the lung most of it is extremely dry and it is difficult to express the air from the alveoli. In the lower lobe there is a very slight amount of pulmonary edema. On opening the bronchi there is a small amount of mucosanguinous secretion. There are no thrombi in the pulmonary vessels. The tracheobronchial lymph nodes are slightly enlarged and appear normal on sectioning.

Digestive System.—The peritoneum is perfectly clear and shiny. The esophagus and stomach show no lesions. In the upper part of the stomach there is a submucosal blushing which is present in the cardiac end only. However, there is no actual injection of the vessels. No lesions of the intestines are encountered except for some injection of the mucosa at several sites. The pancreas weighs 100 grams and is normal in appearance on sectioning. The vessels to the viscera are fairly widely dilated.

*Performed by Ethel R. Nelson, M.D.

Hepatic System.—The liver is brownish-red and weighs 1,350 grams. The lower border is at the costal margin. The external surface is perfectly smooth. On cut surface the normal lobular markings can be made out. It is firm in consistency and rather brownish-red. The gall bladder is of normal size and contains bile. The bile duct is patent and the portal vein has no thrombi.

Spleen and Lymphatic System.—The spleen is rather deep purple, firm in consistency, and weighs 22 grams. On cut surface it is deep purple and congested. The malpighian bodies stand out as white dots against a purple background. An accessory spleen, 2 cm. in diameter, is found at the lower pole of the spleen. The lymph nodes of the mesentery, retro-aortic, and left axillary areas are slightly hypertrophied. However, on sectioning they are not unusual in appearance. The splenic vein is dilated but contains no thrombi.

Urinary Tract.—The kidneys are small, the right kidney weighing 125 grams and the left kidney, 110 grams. They are normal in external appearance. The capsules strip with ease leaving a few slightly indented scars on the surface. On cut surface the cortex is 4-5 mm. thick. The tissue is deep purple, both the cortex and medullary portion. The ureters are patent and there are no lesions of the pelvis or urinary bladder.

Genital System.—The external genitalia are normal. The cervix has a mucinous plug extruding from it and there is chronic cervicitis. The uterus, tubes, and ovaries are within normal limits.

Endocrine System.—The pituitary is not examined. The thyroid is small and normal in appearance. Parathyroids are not examined. Thymus is small. Suprarenal glands are small and rather firm when cut across, having yellow cortex and white medulla.

Skeletal and Muscular Systems.—Normal.

Microscopic Description

Lung.—There is a marked hypertrophy of the smooth muscles surrounding the bronchioles with numerous round cells, mostly lymphocytes, a few eosinophiles, monocytes, and plasma cells. The mucous glands are rather prominent and the bronchioles are filled with mucus, red blood cells, and scattered eosinophiles. The epithelial cells of the bronchioles are tall, pseudostratified, with large mucous droplets. The alveoli surrounding the bronchioles are widely dilated and empty. Some of the walls are broken. There are a few areas of red blood cell infiltration into the alveoli.

Lymph Node.—Occasional germinal centers are seen. The stroma is not very cellular. There is increased fibrous tissue content.

Thymus.—Lobulated gland, stromal cells not too densely arranged. There is some fibrous infiltration and Hassells' corpuscles are present. Normal thymus for this age group.

Adrenal.—No significant lesions.

Liver.—The liver cells are normal in appearance and in normal trabecular arrangement. The intercellular bile capillaries appear normal, are not distended, and contain no abnormal cells. The periportal connective tissue is not increased. No significant lesions.

Pancreas.—Very glandular with little fibrous tissue. Islets present. No significant lesions.

Stomach.—Parietal cells very prominent and abundant mucosa. Muscle layers normal appearing. No significant lesions.

Bone Marrow.—No significant lesions.

Spleen.—Capsule not thickened. Sinusoids widely dilated and filled with red blood cells. Malpighian corpuscles present. No significant lesions.

Bronchial Lymph Node.—Prominent germinal centers. Anthracotic pigment. Coagulated protein in blood vessels. No significant lesions.

Ovary.—Several corpora albicans. Recent corpora lutea. Several follicles. Normal stroma. No significant lesions.

Kidney.—Capsule not thickened. Vessels dilated. Occasional hyaline casts in the tubules. No significant lesions.

Breast.—Hyperplasia of ducts of one nodule. No significant lesions.

Summary of Findings:

1. Bronchial asthma.
2. Bronchial edema.
3. Emphysema.
4. Accessory spleen.

ALLERGY STUDIES

The patient died showing no skin reactions to the tests performed. There was no evidence of accidental intravenous penetration. In order to determine which antigen or antigens were responsible for the anaphylactic shock, passive transfer studies were initiated. Serum secured from the patient at the time of autopsy was injected intracutaneously into a series of skin sites of a non-allergic recipient. Additional sites, for purposes of control, were injected on the opposite arm with the serum from an individual previously determined nonallergic. Twenty-four hours later all these sites were tested with the identical allergens used in testing the deceased patient, using the same vials from the testing tray. For additional control, the same tests were repeated, at the same time on normal nonsensitized areas of the recipient's skin. Sensitized sites were also tested with the diluent used to dilute the testing allergens.

TABLE I. RESULTS OF PASSIVE TRANSFER TEST

TEST	SITES		
	A SENSITIZED WITH PATIENT'S SERUM	B SENSITIZED WITH NORMAL SERUM	C NONSENSITIZED
1. Cat hair	negative	negative	negative
2. Chicken feathers	negative	negative	negative
3. Cottonseed	markedly positive (4 plus)	negative	negative
4. Dog hair	negative	negative	negative
5. Duck feathers	negative	negative	negative
6. Goose feathers	negative	negative	negative
7. Horse serum	negative	negative	negative
8. Horse dander	negative	negative	negative
9. House dust	slight (1 plus)	negative	negative
10. Kapok	negative	negative	negative
11. Orrisroot	slight (1 plus)	negative	negative
12. Pyrethrum	doubtful (plus-minus)	negative	negative
13. Rabbit hair	negative	negative	negative
14. Sheep wool	negative	negative	negative
15. Silk	negative	negative	negative
16. Tobacco	negative	negative	negative
17. Diluent (used to dilute allergens)	negative	negative	negative

The following results were obtained: There were no reactions in any of the control sites; the sites sensitized with the patient's serum disclosed a doubtful (plus-minus) reaction to the extract of pyrethrum, and a slight (1 plus) reaction to the extracts of house dust and orrisroot; there was a

marked (4 plus) reaction to the extract of cottonseed. This reaction was very striking: the wheal measured 2.5 cm. in average diameter and had numerous pseudopodia extending from the periphery. The surrounding erythema was 5.0 cm. in diameter. Reactions to the extracts of cat hair, dog hair, rabbit hair, tobacco, horse serum, wool, chicken feathers, duck feathers, goose feathers, horse dander, kapok, and silk were negative. (Table I.) These studies indicate that the serum of the patient contained the antibodies of cottonseed, house dust, orrisroot and, perhaps, pyrethrum.

In order to determine quantitatively the concentration of antibodies present, serial dilutions of the patient's serum were made with buffered saline solution for use in passive transfer dilution studies. The serum was diluted 1:10, 1:20, 1:40, 1:80, and several sites on the skin of another nonallergic recipient were sensitized with each. These sites were then tested with the extracts of cottonseed, house dust, orrisroot, and pyrethrum. The cottonseed antigen continued to exhibit a reaction through the 1:80 dilution (Table II), whereas pyrethrum was negative in the 1:10 site, and dust and orrisroot were doubtful in the 1:20 sites becoming negative in the 1:40 sites. Because of the violence of the reaction to cottonseed, it is logical to assume that this antigen was responsible for the death.

TABLE II. PASSIVE TRANSFER OF ALLERGENS TO PREPARED SITES

DILUTION	ANTIGEN			
	COTTONSEED	DUST	PYRETHRUM	ORRISROOT
1:10	marked (4 plus)	slight (1 plus)	negative	slight (1 plus)
1:20	marked (4 plus)	doubtful (plus-minus)	negative	doubtful (plus-minus)
1:40	marked (3 plus)	negative	negative	negative
1:80	moderate (2 plus)	negative	negative	negative

DISCUSSION

For a long time, cottonseed has been recognized as a potent allergen. Many allergists have noted marked reactions following its use in skin testing allergic patients, but we are aware of only 2 deaths prior to this one.^{14, 15}

The cottonseed extract used in testing our patient was of routine testing strength and in use in our clinic for over 10 years. The vial of extract from which the fatal dose was removed had been on the testing tray for several weeks, and samples of it had been used for testing other patients with no constitutional reactions resulting.

The necessity for routine use of cottonseed extract for allergy tests has been questioned. There is sufficient evidence that cottonseed oil as commonly employed in commercial shortening, for food packing, and in salad oils does not contain the water-soluble cottonseed protein. Figley¹⁶ recently reviewed the literature, cited cases of his own, and quoted excerpts of expert testimony before a public hearing of the Federal Security Agency called for the purpose of establishing standards for salad dressing. As a result of this hearing, it was established that allergic sensitivity to the water-soluble protein does not

indicate allergy to the refined oils expressed from this seed. In addition, it was pointed out by expert chemists' testimony that the methods of processing edible vegetable oils renders highly unlikely their retention of any allergic activity.

It is true that an individual, if engaged in farming, may come in contact with cottonseed meal as an ingredient of feeds for livestock, or as a component of commercial fertilizer. Occasionally, an individual may come in contact with cottonseed meal or flour in a small variety of bakery products such as doughnuts, cookies, and fig bars. A diabetic on a decreased carbohydrate diet conceivably may substitute cottonseed meal for wheat. Cotton linters, used in the manufacture of mattresses, are refined crudely and, in some instances, may contain the whole seed.

These contacts, however, are limited, the major contact with cottonseed being with the prepared oil—now considered nonallergenic.

The outstanding pathological finding in this case was marked pulmonary emphysema. The lungs did not deflate on opening the thoracic cage, and great difficulty was encountered in trying to express the air from the lungs. Death was apparently due to asphyxia as a result of generalized bronchospasm. Other findings in the lungs, such as hypertrophy of the smooth muscles surrounding the bronchioles and plugging of the bronchioles, could be attributed to chronic bronchial asthma. The markedly increased coagulation time of the blood, as noted in this case, has been described as a constant finding in experimental anaphylaxis in animals. The generalized venous stasis, dilatation of the right side and contraction of the left side of the heart were apparently secondary to the pulmonary pathology and verifies experimental work on the pathological physiology of anaphylactic fatalities in guinea pigs. In such cases there is chiefly an overdistention of the lungs which remain in this state so that little or no air enters despite violent respiratory effort. A drop in the peripheral blood pressure occurs with a corresponding rise in pulmonary arterial pressure. The force of cardiac contractions progressively fails with ultimate cessation while the right chambers remain in diastole.

The train of events just described, the "guinea pig type" of anaphylactic reaction, is contrasted with the "dog type," in which the liver is the main point of involvement and the "rabbit type," in which the lungs are not overdistended though there is a characteristic distention of the right side of the heart due to spasm of the arterial side of the pulmonary circulation. In a survey of 24 necropsies among the 61 anaphylactic deaths found in the literature, Kojis¹ summarized the causes of death as follows: 3 showed nothing; 6 had an enlarged thymus; 1 had subacute nephritis; 1 demonstrated a dilated left ventricle, congested kidneys, and a pulpy spleen; 7 died in the "guinea pig type" of reaction; 3 in the "dog type"; 1 in the "rabbit type"; 1 in the combination of "guinea pig type" and "dog type" combination.

One of the tenets of anaphylaxis is that different animal species react with entirely different symptoms and pathological findings, but with each animal species the symptoms and findings are the same, regardless of the

antigen used to provoke the anaphylaxis. This has certainly been true of the guinea pig, the rabbit, and the dog and, to a great extent, of other animals not refractory to anaphylaxis. Apparently this is not entirely true of the human species. The few cases of fatal anaphylaxis in humans on record seem to indicate that all types of anaphylaxis have occurred, though the "guinea pig type" seems to be preponderant. Undoubtedly, if man could be used as an experimental animal and fatal anaphylaxis produced at will, one type would be overwhelmingly predominant.

Unfortunately, we were not permitted to do an autopsy on the brain, and as a result such conditions as ruptured cerebral aneurysm and pontine hemorrhage were not ruled out definitely as possible causes of death. However, this possibility is rather remote. In view of the circumstances preceding death and the other findings at necropsy, it need not be considered. Sudden death is often attributed to an enlarged thymus gland, and in Kojis' report¹ of 24 cases of fatal anaphylactic death, the thymus was indicated in 6. Our case had a persistent thymus gland, normal for this age group.

This case fulfills the criteria for the diagnosis of fatal anaphylactic shock for the following reasons: (1) The symptoms and circumstances preceding death were characteristic of an allergic reaction. (2) The autopsy findings revealed no other explanation for the cause of death. (3) The autopsy findings were typical of those described in fatal anaphylactic reaction. (4) A specific circulating antibody corresponding with the antigen injected was demonstrated in the serum of the patient by the technique of passive transfer. (5) The responsible antigen was found to be harmless to normal individuals. (6) Possibility of contamination of the injected material was ruled out by its use on normal individuals.

Allergy studies with the serum of the deceased patient indict the cottonseed antigen as the cause of the fatal reaction. The comparative safety of the cottonseed allergen, as evidenced by its routine use for intradermal testing in this clinic for a period of many years without severe reaction, verifies the belief that fatal or constitutional reactions are not solely due to the nature of the antigen but rather to exquisite sensitivity on the part of the individual. That this exquisite sensitiveness in any one patient is usually limited to one or several antigens is proved in this case by the fact that this patient showed very minor reactions to pollen allergens, which usually give more persistent and marked skin reactions. Since one cannot predict which antigen or antigens to which a patient may be exquisitely sensitive, and reactions to other allergens are no criteria, it would appear that the safest available precaution against severe and fatal reactions would be to precede each intradermal allergy test with the much less sensitive scratch test, even though fatalities have also occurred following scratch tests.¹⁷

SUMMARY

1. A case of fatal anaphylactic shock following intradermal tests for allergy is reported.

2. The pathological anatomy is described, consisting primarily of acute pulmonary emphysema of the so-called "guinea pig type" of anaphylactic reaction.

3. Allergy tests are described which, by passive transfer and dilution studies, indict the cottonseed allergen as the cause of the fatal reaction.

4. The importance of cottonseed allergen is discussed and its absence in commercial cottonseed oils and salad dressings is pointed out.

5. The cases of anaphylactic deaths in the medical literature are reviewed.

6. The advisability of preceding intradermal tests with scratch tests is advocated.

REFERENCES

1. Kojis, F. G.: Serum Sickness and Anaphylaxis, *Am. J. Dis. Child.* **64**: 93, 1942.
2. Park, W. H.: The Use of Antitoxin in the Treatment of Diphtheria, *J. A. M. A.* **76**: 109, 1921.
3. Lamson, R. W.: Sudden Death Associated With the Injection of Foreign Substances, *J. A. M. A.* **82**: 1091, 1924.
4. Cooke, R. A.: Studies in Specific Hypersensitiveness. III. On Constitutional Reactions: The Dangers of the Diagnostic Cutaneous Test and Therapeutic Injection of Allergens, *J. Immunol.* **7**: 119, 1922.
5. Baagöe, K. H.: Case of Allergic Death Due to Intracutaneous Injection of 10 c.c. of White of Hen's Egg, *Ugesk. f. læger.* **90**: 550, 1928.
6. Lamson, R. W.: So-Called Fatal Anaphylaxis in Man, *J. A. M. A.* **93**: 1775, 1929.
7. Freedman, H. J.: Acute Anaphylactic Shock Following Intracutaneous Test for Sensitivity to Horse Serum, *New England J. Med.* **212**: 10, 1935.
8. Ferguson, R.: A Case of Fatal Serum Reaction, *Canad. M. A. J.* **43**: 469, 1940.
9. Vance, B. M., and Strassmore, G.: Sudden Death Following the Injection of Foreign Protein, *Arch. Path.* **34**: 849, 1942.
10. Hunt, E. L.: Death from Allergic Shock, *New Eng. J. Med.* **228**: 502, 1943.
11. Wiseman, J. R., and McCarthy-Brough, M. P.: Skin Sensitivity in the Aged—Fatality Following Intradermal Tests, *J. ALLERGY* **16**: 250, 1945.
12. Swineford, O.: Anaphylactic Shock from Skin Testing, *J. ALLERGY* **17**: 24, 1946.
13. Blanton, W. B., and Sutphin, A. K.: Death During Skin Testing, *Am. J. M. Sc.* **217**: 169, 1949.
14. Allergy to Cottonseed and Other Oilseeds and Their Derivatives. Excerpts from Testimony Before the Administrator, Federal Security Agency in the Matter of Fixing and Establishing Definitions and Standards for Identity for Mayonnaise, French Dressing and Related Salad Dressings (Docket FDC-51). Public hearing held at Washington, D. C., November 18, 1947, and January 6 to 8, 1948. Published by National Cottonseed Products Association, Inc., Memphis, Tenn., 1948.
15. Benson, R. L.: Discussion of Bernton, H. S., Spies, T. R., and Stevens, H.: Significance of Cottonseed Sensitiveness, *J. ALLERGY* **11**: 138, 1939.
16. Figley, K. D.: Sensitivity to Edible Vegetable Oils, *J. ALLERGY* **20**: 198, 1949.
17. Vaughan, W. T.: Practice of Allergy, St. Louis, 1939, The C. V. Mosby Company, p. 171.