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ALLERGY IN CHILDREN*

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THE importance of allergy in the field of medicine, particularly in pædiatrics, can easily be appreciated when one finds that approximately 7 to 10 per cent of our population suffer from some allergic condition. Since a large number of cases manifest themselves in the first decade of life, and respond more satisfactorily to treatment during this childhood period a general consideration of the subject should be of interest.

The diagnosis of the allergic condition is made perhaps more from a careful and complete medical and allergic history than from the other supplemental aids to diagnosis at our disposal. The taking of the allergic history, as suggested by Bray,[1] may be divided into predisposing factors, related to the onset of symptoms, and precipitating factors, specific and non-specific, which are related to the production of an attack.

A. **Predisposing factor** (related to onset): (1) Heredity: allergic predisposition has a strong tendency to occur in families. (2) Tissue trauma: severe illness before onset of symptoms, *viz.*: asthma; (*a*) pneumonia; (*b*) infectious disease, *e.g.*, pertussis, rubella.

B. **Precipitating factors** (related to production):

Specific: absorbed by: (1) *inhalation*: animals, hairs, feathers, dusts, pollens, moulds, fungi; (2) *ingestion*: foods, drugs; (3) *injection*: sera, drugs, bites, stings; (4) *infection*: bacterial allergy and worms; (5) *contact*: fabrics, chemical and physical agents. These are carried by the blood to the shock organ, with resultant allergic reaction.

Non-specific (catalysing factors): (1) dietary; (2) environmental; (3) toxic; (4) nasal; (5) psychic; (6) endocrine; (7) physical; (8) chemical; (9) mechanical. These lower the "allergic threshold" so that specific substances may act.

In infants and children, if careful inquiry is made, an allergic family history will be obtained in over 60 per cent of cases, which is more than 10 per cent above the general average for all ages. Allergic children, especially if very young, are also more likely to have a bilateral positive history of allergy.

In children the psychic factor plays no part unless the child receives too much sympathy from the parents, and develops wheezing respirations as a shelter complex. In adults with an allergic basis psychic factors may play a large part in recurring asthmatic attacks. Breathing can be accentuated by the emotions, *e.g.*, laughing, crying, fright, etc. An allergic adult, especially a female under emotional stress, can produce an attack of wheezing breathing with the hope of exciting sympathy. This, in my opinion, is the main reason why desensitization is not so successful in adults as in children.

Eczema is the commonest manifestation of allergy in infants. Most frequently it is confined to the face, and is very often associated with a cradle cap, but it may begin on the face and quickly become generalized. Ointments relieve the irritation, but they do not cure the condition. Eczema is usually self-limited, clearing up between the twelfth and fourteenth month. The last ointment to be applied, when the child is about to outgrow the condition, receives all the credit. I have never known weaning to cure an eczema. In the bottle-fed, if on a cow's milk formula, I always advise the use of evaporated milk, which in the very occasional case, due to the change in the protein molecule caused by heating, will clear the eczema.

In my opinion skin tests are of little value as far as obtaining a rapid cure by eliminating the positive food.

Elimination diets, especially for the child under two years, and for the most part for children over two years, are extremely difficult to carry out in the home, because of lack of

* Read before the Winnipeg Medical Society, November 17, 1939.

variety and the difficulty of obtaining co-operation from the mother. I have made very little use of the elimination diet in private practice. Restraint as carried out in the hospital is almost impossible in the home.

Flexural prurigo occasionally follows infantile eczema, and may persist into adult life. It is essentially chronic and very difficult to eradicate. Here again I am not impressed with results obtained from protein tests.

FOOD ALLERGY

During the pre-school age foods play a more important part than inhalants. After school age, with each year the inhalants play a more important part.

In infancy egg heads the list as a food producing allergic manifestations. With the introduction of egg into the infant's diet for the first time, I always warn the mother that if the baby breaks out in hives, vomits, or if loose stools develop, these symptoms are evidence of sensitivity to egg, and egg must at once be eliminated, and not tried again until 18 months.

A baby, one year old, suddenly developed a generalized urticaria. There was an allergic family history. Careful inquiry on my part failed to elicit the food at fault. The mother remembered that her brother could not eat potato. She eliminated potato from the child's diet, and the hives cleared up dramatically. She has since found out by her own experiment that she can give potato twice a week without reaction. I have instructed her to increase potato by a minimum amount with the hope of eventually desensitizing the baby.

This case demonstrates that some children can tolerate a small amount of one food, but if given in large amounts there will often be an allergic reaction. Quantitative inquiry is important in any investigation as to food.

Another doctor referred homologous twin girls, aged 8, with an allergic rhinitis, chronic cough, occasionally associated with wheezing respirations. I had seen the twins at the age of two in consultation for a persistent papular urticaria. Here the sense of smell was of great help in arriving at the diagnosis. During the first visit the odour of onions in the office was plainly evident. With the second visit the same odour caused me to inquire of the mother whether the twins were fond of onions. She replied that no meal was complete without onions, and instead of taking an apple to bed "to keep the doctor away", they each insisted on a bunch of green onions in the summer and in the winter, a Spanish onion. Further inquiry revealed an absolute distaste for milk. Milk had been forced since infancy. Distaste for any food should be given consideration in allergic investigation. Scratch tests were negative. Intradermal tests were definitely positive for milk, onions, and house dust.

Papular urticaria.—Six years ago I saw a Jewish child of eight years who was visiting Winnipeg from Chicago. The mother had consulted many physicians because of a recurrence of a papular eruption. The eruption was very irritable, and from the scratch marks resembled scabies, and it had been so diagnosed many times, with the result that the child would be eliminated from attending school. I made the diagnosis of papular urticaria and advised skin tests. When I came to pork I said to the mother "There is no need to apply it". She replied, "Yes, we are not orthodox, she loves bacon". The child gave a marked reaction. Bacon was withdrawn and the urticaria has not returned.

Gastro-intestinal allergy.—This may produce symptoms of recurrent pain in the abdomen, associated with or without vomiting. Intermittent attacks of diarrhœa with mucus and blood may occur. As illustrative of this type I shall briefly report a case.

D.W., male, came under my observation at three months in February, 1933, with a mild facial eczema which at first appeared at the age of one month. He was nursed six weeks. The grandmother had eczema; the mother was unable to eat apples. At the age of 4½ months he developed diarrhœa, with mucus and blood in the stool. He responded to dietary management. At six months the diarrhœa recurred, and from this time on, on an average of one a month, he had an attack of diarrhœa. In spite of this he continued to gain fairly well. I tried every known dietary management, eliminating milk, substituting sobee, eliminating wheat and eggs, all without success. With every change in diet the diarrhœa would be controlled for a time never exceeding two months. At five years of age skin tests were applied and showed him sensitive to the following: milk, egg, cheese, sardine, and to a slighter degree, parsnip, beet, celery, corn, pea, tomato, peanut, salmon, orange and yeast.

Dryco was prescribed as a beverage and for cooking. The foods were eliminated. At six years he has been free from diarrhœa for one year, and weighs 46 pounds (normal). His disposition has completely changed. He is very active and very happy.

The cases I have quoted justify the old axiom: "What is one man's food is another man's poison". The slogan "A quart of milk a day for every child", is excellent commercial propaganda but not necessarily good advice, not only from an allergic standpoint, but also because many children cannot ingest a quart of milk a day and eat a mixed balanced diet. May I interject here that ½ pint of milk at 11 o'clock recess for the underweight child at school is the best deappetizer for the noonday meal so far devised by those interested in public health.

Allergic rhinitis and asthmatic bronchitis.—A child with the history of repeated head colds, constant nasal discharge, associated with or without a chronic cough, especially if the symptoms are as persistent in the summer as in the winter, and more especially if the tonsils and adenoids have been removed without improvement, indicates the following investigation: (1) a careful inquiry as to allergy in the family history; (2) inspection of the nasal mucous membranes. If pale and œdematous, a nasal smear should be examined for eosinophils. If

positive, the diagnosis is allergic rhinitis. If in addition there are associated bouts of sneezing, watery eyes, itchy nose and pharynx, with a definite month of onset, pollinosis or hay fever is the diagnosis. If the symptoms are associated with periodic attacks of audible wheezing respirations, with an annoying, dry, non-productive cough at onset, the attack subsiding within three or four days with a loose productive cough, temperature rarely above 101°, the diagnosis is asthmatic bronchitis.

The case histories of five patients are presented.

CASE 1

D.S., a girl, present age 10 years. I first saw this patient in April, 1929, at the age of three weeks, with a facial eczema and cradle cap. The father had vernal conjunctivitis. At 2 years she had a persistent watery discharge from her nose. At 4 years she was having frequent colds, both summer and winter; a constant nasal mucoid discharge, also vernal conjunctivitis. The mother dated the onset of the colds from an attack of whooping-cough a year before. The chest was clear. Eosinophilia was present in nasal smear. Protein tests were refused.

I did not see her again until August 5, 1938, aged 8½ years. At 7 years the tonsils had been removed without improvement. In July, 1938, she was seen by a sanatorium superintendent who made a diagnosis of chronic septic infection at the base of the left lung, and referred the case to me.

She was 20 pounds underweight. The physical findings were confirmed, but eosinophilia was present in the nasal smear; nasal mucosal swelling of the allergic type; flexural prurigo at the flexures of the elbows. The signs in the chest cleared up in two weeks with inhalations and postural drainage; cough and nasal discharge persisted.

Protein scratch tests showed questionable sensitivity to a number of grass pollens (vernal conjunctivitis). Intradermal tests indicated multiple sensitivity with marked reactions to autogenous house dust and dog. The positive foods were eliminated from the diet but the family refused to do away with the dog. Desensitization had been refused. There has been no marked improvement in the condition.

CASE 2

E.F., female, aged 10 years, was admitted to the Children's Hospital out-patient department on April 11, 1939, for the investigation of recurring asthmatic attacks during the previous five years, following whooping cough. Between attacks she was constantly troubled with a running nose, sneezing and inflamed eyes.

The mother had bronchitis in England, but has been well since coming to Canada. A younger brother had infantile eczema. The nasal smear showed 16 per cent eosinophils. Blood smear showed 17 per cent eosinophils, otherwise normal. X-ray of the chest was negative.

She was admitted to the hospital on April 22nd for investigation. Scratch tests were all negative. Intradermals gave cat hair +++, horse hair ++++, house dust +++, rabbit, squirrel and cattle hair +, autogenous house dust (1:10,000) +++.

Treatment.—Two cats were removed from the home. Old horsehair furniture was also removed. The mattress and pillows were covered with rubberized sheeting. On May 2, 1939, desensitization with autogenous house dust was begun, and continued twice weekly until June 13th, and then weekly, and from September 26th a maintenance dose of 1 c.c. of 1/100 house dust was given weekly.

From April 22nd to June 6th the patient gained 12 pounds while in hospital. On June 18th she reported that she had had a mild spell of wheezing at night. She has had no attacks since that date although she has had several colds, unaccompanied by asthma.

CASE 3

M.W., a girl, aged 7½ years. Uncle has had asthma since infancy. The child was first seen at 6 months with eczema and urticaria. Mother thought that the onset was coincident with the addition of cream of wheat to the diet. Wheat cereals were removed from diet; an evaporated milk formula was given. The urticaria disappeared; eczema remained until 14 months. None since.

She was not seen again till 5½ years old (1937). At 3½ she had had a running nose and cough from April to September. At 4 the condition was diagnosed as whooping-cough and she was given injections without improvement. At 4½, again a running nose and cough but not so bad as the year before. At 5 she was treated by a nose and throat specialist by nasal packs and vaccine. At 5½ she had a constant cough, worse at night, and a persistent nasal discharge interfering with sleep. She was spending a week out of every month in bed. The nasal smear showed 30 per cent eosinophils. Diagnosis: allergic rhinitis and bronchial asthma. The following reactions occurred to proteins applied by the scratch test: chicken feathers, camel hair, grapefruit and pineapple, +; autogenous house dust, wheat proteose, +++; horse dander, apple, ++++; she also reacted to all common Manitoba pollens.

The foods were eliminated from the diet. A treatment set was made and desensitization carried out from August 27, 1937, to November 30, 1937, and she was given a maintenance dose monthly of 0.5 c.c. of a 1:10 dilution.

On Hallowe'en, 1937, she indulged in apples and peanuts and had an attack of asthma. Other than this she was free from symptoms throughout the summer.

CASE 4

S.C., female, aged 11 years, on September 8, 1939, was admitted to the out-patient department with a history of recurring asthmatic attacks since 1933, with no seasonal relation. Her dietary history suggested sensitivity to eggs and milk. She was placed on an egg and milk free diet without any improvement. On October 17th she was admitted to the Children's Hospital for investigation. The only family history of allergy was that her sister was sensitive to milk. A blood smear showed 20 per cent eosinophils, otherwise negative. The nasal smear was negative.

Scratch tests indicated sensitivity to cat hair, +++; horse hair, ++; cattle hair and house dust, +; beef, +++; whole milk, +++; mutton, ++; pork, +; cheese, +. On October 20th I caused the child to drink 8 ounces of milk. Within 20 minutes her face became flushed and she complained of headache and nausea. The symptoms passed off in approximately one hour. On October 21st beef was given with no apparent reaction. She was discharged on October 27th from the hospital with instructions to return to the out-patient department for desensitization, but to date she has not returned. Forty-eight hours after return to home she had a severe attack of asthma.

CASE 5

R.P., male, aged 10 years, was admitted to the Children's Hospital on April 14, 1939, with the complaint of a dry, non-productive cough for 8 weeks, recurring asthmatic attacks for the past six years. Since the onset of the cough he had had recurring bouts of fever. He developed eczema at eleven months

of age, which persisted intermittently until one year ago.

His father had a chronic cough, otherwise the family history as to allergy is negative. The boy dislikes fish, and for a time the asthma seemed to improve with the elimination of fish from his diet and the removal of pets and flowers from the house.

Two days after admission he developed a temperature with an evening rise to 102°. This persisted for one week. Physical examination of the chest was negative. Scratch tests indicated multiple sensitivity to animals, house-dust, pollens, and a great many foods.

Treatment consisted in: (1) desensitization for pollens; (2) desensitization for inhalants, cat, dog, horse and rabbit (the mother had two fur coats of rabbit origin); (3) elimination diet, excluding all foods which showed positive reactions; (4) breathing exercises; (5) dust-free room.

Discharged from hospital June 17, 1939; weight 52 pounds, a gain of 8 pounds during hospitalization. *Result.*—September 12, 1939, steady improvement during desensitization; he had slight allergic eczema on arms and legs in September.

I desire to thank Dr. V. H. Patriarche, Assistant Radiologist to the Children's Hospital for her co-operation in preparing and demonstrating x-ray films illustrating allergic cloudy antrums and allergic basal disease in the lungs in cases one, three and five.

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SONNE DYSENTERY IN AND AROUND VANCOUVER*

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WE are not accustomed to think of bacterial dysentery as a potent cause of ill health in a country with the temperate climate of our own. Reports[1, 2] from England and Scotland reveal that its recognition has increased greatly in these countries in recent years. My own experience in England and Vancouver has impressed me with the prevalence of this type of infection in the northern countries. Because of its usual mild symptomatology in these countries the high incidence of the disease is often overlooked, and, so, many experienced practitioners are unfamiliar with its true nature. Because it is unrecognized in its mild form it will occasionally spread as an epidemic amongst children and cause some deaths.

While a variety of types of dysentery bacilli have been recovered from the stools in sporadic cases and epidemics, it appears from recent statistics that the Sonne or Duval organism is the commonest offender in temperate countries. In fact, one authority[3] states that sporadic bacillary dysentery in the United States is caused more frequently by this organism than by any other of the dysentery group. On the other hand, Gibbons,[4] in a study of the sera agglutination titre of the general population of British Columbia and of mental hospital patients, found that 2.9 per cent of the former and 11 per cent of the latter showed agglutinins to the Flexner type of *B. dysenteriæ,* whereas none of the sera showed significant titres to the heat-killed antigen of *B. dysenteriæ,* Sonne. The same author pointed out that the infant mortality in British Columbia due to diarrhœa and gastro-enteritis was consistently lower than in any of the other Canadian provinces. He suggests from this and from the fact that the Sonne organism was seldom reported in stool cultures prior to 1936 that the incidence of Sonne dysentery was negligible in British Columbia. This is of interest as regards the geographic distribution of disease, because reported surveys from the Atlantic States and California recognize the prevalence of the disease in these districts.[6, 7, 8]

During the past few years, however, the number of stool cultures positive to *B. dysenteriæ* Sonne reported from the Provincial Board of Health Laboratories has risen sharply, and there has been at least one serious outbreak of the disease in one of the children's wards in Vancouver. It would seem from these facts that the disease *is* present in our community. Moreover, its recognition is apparently increasing, due possibly to more frequent use of the laboratory facilities for bacteriological examination of stool specimens. Certainly it would seem that its presence here is greater than Gibbon's figures of 1936 suggest. For example, in the university area alone (a population of a few thousand) in the past year four sporadic cases were discovered in the only four cases from which stool specimens were collected; there may have been many others. Then, also, in my own practice I have been able to collect 10 sporadic cases in the past year.

With the possibility in mind that still further use of the diagnostic facilities may reveal many

* A paper delivered before the Pædiatric Section of the Vancouver Medical Association.