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Allergic bronchitis

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ALLERGIC BRONCHITIS

Allergy, affecting as it does in a slight or marked degree about fifteen per cent of all persons, must have been observed and recognized as a disease entity by those who treated illness even in prehistoric times and recognized by witches who cured disease with songs and dances. Hippocrates, 460 B.C., observed the effect of cold and moisture on the asthmatic and its autumnal seasonal incidence and expressed the opinion that "those who from asthma or cough have the spine incurvated before puberty generally die". Aurelianus, 5th century, stated that asthma was incident in the old and the young, attacking patients after deep colds. Cases of allergic coryza were described in some detail by Botallus in 1565, by Schneider in 1662 and by Beningerus in 1673 under such terms as "rose catarrh" and "catarrhus aestivalis". Bostock, in 1819, the "pioneer" of hay fever, assigned the cause of asthma to pulmonary trauma because a large proportion (as much as 80 per cent) of cases of asthma in the young dates from one or the other of whooping cough, bronchitis or measles. He described its seasonal appearance and made the observations that sometimes it was cleared up by taking a journey and that if the patients kept inside and avoided exercise, their symptoms would not be so severe. Blackley, in 1856, described the condition in such detail that few, until the last few years, added materially to the facts which he disclosed. He produced symptoms in himself, a sensitive person, by the application of pollen and pollen solutions to the mucous membranes of the eyes, nose and mouth. He performed skin tests by the appli-
ocation of dry pollen to scarified areas on the skin, which caused extreme local itching and swelling of the tissues, lasting about four days. He also studied pollen plates and noticed a close relationship between the number of pollen granules in the air and the severity of clinical symptoms.

With the discovery of animal anaphylaxis by Richet, in 1893, we were given basis for the remarkable advances of the last ten years in our understanding of allergy. Meltzer, in 1910, suggested that the bronchial constriction of asthma was a phenomenon of anaphylaxis analogous to that described in animals dying from anaphylactic shock. Koessler, in 1913, reported a case of asthma caused by hypersusceptibility to hens egg and since then development has proceeded rapidly (23) (24) (26).

Cooke defines allergy as the reaction occurring in individuals, naturally hypersensitive, on the absorption of a specific substance. (1). Kolmer defines it as a state of altered reactivity of the body cells (9). Duke defines it as an altered reactivity caused by specific hypersensitiveness to the action of physical agents (25).

Asthma, hay fever, and serum sickness have been recognized as manifestations of allergic sensitivity for a number of years. During the last two decades urticaria, angioneurotic edema, certain types of headache resembling migraine, eczema and true allergic bronchitis have been recognized as manifestations of hypersensitiveness to an allergen.

Allergic bronchitis was not diagnosed as a clinical entity until 1925, when Cooke in Tice's Practice of Medicine (1) suggested that a cough complicating allergic coryza and difficult
to differentiate from pertussis might be on an allergic basis. He described the cough as violent and paroxysmal, frequently resulting in vomiting and associated with a marked temporary enlargement of the lingual tonsil.

It may be well at this point to discuss the causes of bronchitis so that an allergic manifestation may be differentiated. Detweiler lists the following as causes (13):

1. Focal infection, particularly upper respiratory.
2. Protein asthma.
3. Arteriosclerosis, usually associated with emphysema.
4. Mitral and cardiac disease.
5. Occupation.
6. Climate.
7. Tumors or aneurysms pressing on a bronchus.
8. Bronchiectasis.
10. Increased susceptibility of bronchial mucosa to infection.

In connection with focal infection, infection about the gingival margins, tonsils and accessory nasal sinuses is of prime importance. Protein asthma may cause a chronic bronchitis by long continued irritation and spasm of the bronchial musculature with turgescence of the mucous membrane, which eventually lowers the resistance of the bronchial tree and allows infection to take place. Arteriosclerosis of the pulmonary vessels lowers resistance and allows infection to take place by reason of circulatory change. In the same way, mitral and cardiac disease and cardiac asthma cause bronchitis. Occupations which expose the patient to irritation from fumes and dust, wet and extremes of temperature predispose to bronchitis. Retained secretion
and altered circulation of a bronchus distal to a tumor or aneurysm pressing upon it usually results in a local infection. A bronchiectatic cavity serves constantly to reinfect the remainder of the bronchial tree. Foreign bodies in a bronchus are often overlooked when there is a lack of history. Roentgen ray and bronchioscopic examination will rule this possibility out in the majority of cases. In cases where all of these factors can be ruled out, the bronchitis must be due to a lowered resistance of the bronchial mucous membrane and it is in this type of case that perhaps allergy plays a large part.

The lack of a uniform terminology has hampered investigation of cough due to hypersensitiveness. The term "asthmatic bronchitis" has been used for any cough accompanied by wheezing and dyspnea, whether allergic or not. Packemann (3) suggested that this term be restricted to emphysematous bronchitis which follows asthma of long standing. Peshkin introduced the term "para-asthma", indicating a non-hypersensitiveness in contrast to asthmatic or hypersensitiveness.

Waldott (7) (5) has suggested the following classification of bronchitis:
1. Para-asthmatic bronchitis.
2. Asthmatic bronchitis.
   A. Allergic bronchitis.
   B. Intercurrent infectious bronchitis.
   C. Post-asthmatic bronchitis.

"Para-asthmatic bronchitis" is used in the same sense as Peshkin introduced the term, i.e. indicating a non-hypersensitiveness and "asthmatic bronchitis" indicating a hypersensitiveness.
Allergic bronchitis can be considered a disease entity just as any of the other allergic diseases. It is characterized by a sudden onset, spasmodic dry cough, brought on by contact with a sensitizing substance, and the symptoms of which are relieved by the administration of adrenalin. If sputum is obtained and examined for cell content it will show numerous eosinophilic cells and few if any leucocytes. Roentgen ray examination may show the hilum glands considerably enlarged.

Intercurrent infectious bronchitis is the common cold of the asthmatic patient. It is often difficult to differentiate between infectious colds and true allergic bronchitis. Examination usually shows upper respiratory pathology to be the source of the cough. Sputum examination shows a large number of bacteria of a type usually found in the respiratory tract. Eosinophils are rarely found. Ordinary expectorants are useful in the treatment but adrenalin is of no avail. Correction of nasal pathology and a series of injections with autogenous vaccines are beneficial.

Post-asthmatic bronchitis is characterized by a more or less continuous cough producing permanent changes in the chest and lungs. The sputum may be abundant and contains Charcot-Leyden crystals and eosinophils. Adrenalin has no effect on the course of the disease. In certain types of these cases bronchoscopic withdrawal of sputum and dilation of constricted bronchioles is of decided help.

Colmes and Rackemann (2) have described these three types of cases and suggested that the variation in the clinical picture denotes only the different degrees in the amount of
secondary bronchial infection. They presented several cases and came to the conclusion that when hypersensitiveness forms the background of the disease, amelioration of the symptoms may follow the removal of the offending substance, even in the advanced stages of the disease.

Allergic bronchitis is closely related to other pathologic conditions, especially coryza, croup, asthma and upper respiratory pathology (3) (4) (6) (9) (11) (14) (15) (24).

Bivings (9) states that coryza, croup and bronchitis, particularly when chronic or frequently recurrent, are, more oftenly than is commonly believed, symptoms of an underlying allergic state. He believes that allergy should not be considered until all other factors are ruled out, unless the condition exists in a patient having a definite family and personal history of allergy.

Perhaps allergic bronchitis is more closely associated with asthma than with any other disease. In van Leeuwen's series (6) thirty per cent had suffered from bronchitis prior to the onset of the asthmatic attack. Kahn (4) has made the observation that initial pollen asthma in children is usually or almost invariably preceded by a history of cough or bronchitis of months or years duration. He has observed the condition in children whose parents were under treatment for hay fever or asthma. The sudden onset, recurrence or increase of the symptoms during pollenination seasons is the only clue in the history. The condition can be recognized by the accompanying vasomotor rhinitis and proved by immediate relief of symptoms upon administration of adrenalin and its abeyance under pollen-free environment, after usual bronchitis treatments have
failed. Peshkin (14), in a series of one hundred asthmatic children, found that 33 per cent showed a history of recurrent bronchitis preceding the onset of the asthma.

There has been much controversy concerning the relationship of allergic diseases and upper respiratory pathology. Most authorities are now agreed that both conditions may exist and better results are obtained when the allergist and rhinologist cooperate. To quote Cassady (15), "It is very debatable whether asthma and bronchitis are ever other than allergic diseases, but granting that they are, the secondary invasion of the sinuses by bacteria continues the irritation after the allergic offender is no longer active. In the acute stages epinephrine may help to relieve the symptoms. In the chronic state when permanent pathologic changes have occurred, such as hyperplasia, polyps and purulent inflammations remain, the tendency to recurring colds, focal infection from poor ventilation and drainage are important trigger factors in setting off more allergic phenomena which result in a continuation of the disease with its resultant coughing."

Rackemann (3) reported a series of 1074 asthmatic patients in which 40 were found to have reflex asthma due to pathology of the nose, sinuses and throat. Copper (19) reported a series of 31 cases of chronic undiagnosed cough, six of which had a sinusitis. Three of these had a dry cough similar to pertussis and roentgen ray examination showed enlarged hilar glands.

Piusss (16), in 1925, found that about half of his patients had one or more unsuccessful nose or throat operations and in 1927 (17), he advocated surgical treatment to remove
the sequelae of allergic disease and in certain instances to remove foci of infection. Cassady (15) reported a series of 56 cases of chronic bronchitis which were operated to correct nose and throat pathology, 26 showed improvement, 7, subsidence of symptoms and the remaining were not followed.

Allergic individuals are frequently susceptible to upper respiratory infections which are often held solely responsible for asthmatic attacks. It can not always be determined whether a bronchitis is an infection or an allergic phenomenon or both. It is only after such patients have been studied very carefully that the relation between the existing allergic state and infection becomes clear. In the acute stages if there is no temperature, it is more apt to be allergic (14).

Calmes (18) presented a case of chronic cough with no other symptom. The patient was found to be sensitive to orris and freedom from cough was obtained by treating her with the extract. Voorsanger (8) in a study of 200 cases of chronic cough placed 38 per cent, in which the only positive findings were peribronchial thickening and enlarged bronchial root glands, in an undiagnosed group. Waldbott has reported nine cases of allergic bronchitis (7); Rowe reported a typical cases due to wheat and milk (7). Peshkin observed the occurrence of allergic bronchitis independently of an asthmatic state and that it is more readily demonstrated in children (7). In the same discussion Kahn cites a case in an adult.

Persistent cough has been produced by pollen extract overdosage in desensitizing children suffering from perennial hay fever who had neither previous bronchitis nor cough, in one case four attacks. The cough in these instances bore a
considerable resemblance to pertussis (4). Waldbott (20) has produced similar cough by the use of small doses of roentgen ray over the spleen in the treatment of asthma.

Wilmer, Cobe and Lee (22) have reported several cases of post-operative atelectasis occurring in allergic individuals. One of these had been extremely susceptible to attacks of bronchitis for six years and had been susceptible in a somewhat lesser degree as long as he could remember. He also had a vasomotor rhinitis during the summer months. He was found sensitive to timothy, June grass and house dust. Since the adoption of a routine pre-operative treatment of .3 gram peptone and 2 c.c. of normal saline fourteen hours before operation they have not had any post-operative lung conditions.

Case 1 (26). Mrs. AE, age 55, had pertussis when twelve years old and since that time has had a chronic cough, more or less constant, worse from the latter part of August until frost and aggravated by cold infection. The cough has always been dry, sputum never being raised. For the past eight years she has been subject to attacks during which she had the sensation of not being able to breathe, accompanied with very little wheezing. There was never any hemoptysis, night sweating nor chills. Temperature was normal. There was a rhinorrhea accompanied by some sneezing during the latter part of the month of August, continuing until frost. Bowels functioned well but she was subject to flatus. She had spasms of vomiting and her cough was worse after eating the following foods: chocolate, eggs, pork, cheese and pepper. Beets, cabbage, fried foods and white bread caused her to have gas. No urinary disturbances with the exception of a
nocturia, 2-3 times which she believed was caused by drinking two or three glasses of water before retiring. Has not menstruated for 23 years. Weight gain of 47 lbs. in the last twenty years. None of the medicines prescribed for her gave any relief.

The following points of interest were noted in her past and family history: For the past number of years she has had hay fever from about August 26 until frost. In 1927, she had influenza, complicated by an otitis media, necessitating puncture of both drums. Measles and chicken pox in early childhood, typhoid fever at the age of eight, pertussis at age of twelve, scarlet fever twenty years ago. Father died of old age, mother of carcinoma of the breast, two brothers died in infancy, no sisters, no children. No history of allergic manifestations on her fathers or mother's side that the patient knew of with the exception of a cousin who has a chronic cough.

Physical examination, 1929, showed the following points of interest: reddened throat, tonsils small, hyperresonant lungs with low pitched respiratory sounds, eyes, neck, abdomen and extremities normal. Pulse 85, temperature normal, urinalysis negative, blood count and differential not remarkable with the exception of an eosinophilia of six percent. X-rays of chest and sinuses negative as to pathology (taken before coming to Dr. MacQuiddy).

She was tested with the more common foods, pollens commonly found in this locality and animal emanations and was found sensitive to pigweed (4 plus) and cat hair (3 plus). She was desensitized to pigweed and was free from her cough
for the season. Whenever she eats chocolate she is very nauseated and has some cough following. She has been comparatively free from symptoms.
Case 2. Mrs. LE, age 43 came to the University Dispensary Dec. 4, 1930, complaining of a cough that she had had for about thirty years. This cough was worse in the morning and was somewhat relieved by coughing up a little mucous phlegm. The cough was aggravated by colds and was worse during the winter months. She had been diagnosed as tuberculous in spite of the fact that tubercle bacilli had never been found. The cough was constant and never was accompanied by wheezing. There had never been any hemoptysis but she had been subject to night sweats. She had been constipated and was subject to gas after eating. Her best weight had been 105 lbs. and on entrance to the clinic was 78 lbs. Nocturia 2-3 times, no burning nor tenesmus.

There is no family history of allergy that the patient was aware of. She had measles and small pox in childhood, has been subject to attacks of bronchitis. She had an appendectomy in 1902, salpingectomy in 1910 and a ventral hernia repair in 1925.

Physical examination showed an emaciated woman of about the stated age. Head and ears normal, pupils irregular but react to light and accommodation; nose dry, no polyps nor enlarged turbinates; tonsils removed; pyorrhea 2 plus. Left supraclavicular fossa not as prominent as the right, lungs resonant throughout and rales heard all over the chest. Heart and abdomen normal. Urinalysis negative, temperature normal, blood count showed an eosinophilia of 4 per cent, Wassermann 4 plus.

The more common tests were run and she was found sensitive
spinach (3 plus), soy beans (3 plus) and orris root (4 plus). The tests were performed with the scratch method, using dry proteins and decinormal sodium hydroxide as diluent and read within half an hour.

The patient was in the County Hospital at the time and all contact with orris root was avoided and anti-lustic treatment started. Her cough was relieved and she showed a weight gain of eight pounds during the remainder of her stay in the hospital.
Comment. In determining whether or not a patient is allergic or not there must be a chronological series of events:

1. Family or personal history of allergy; not always possible to obtain but its presence should put one on guard.
2. Specific allergen must be found if possible.
3. Relief of symptoms must be experienced upon withdrawal of allergen.
4. Return of symptoms if brought in contact with the specific allergen.

Hypersensitivity to foreign substances, when it finds expression in the bronchial tree manifests itself ordinarily in form of bronchospasm or bronchial asthma. Cough usually appears toward the end of the attack when there is a reflex in response to secretion of mucoid, tenacious substance into the bronchial tubes. The cough in these instances is a reflex in response to irritation of nerve endings in mucosa of respiratory tract. When associated with asthma the severity of the cough is not determined by the degree of bronchospasm.

The striking feature about these cases lies in the fact that cough, without the presence of bronchospasm or any other factor that would be likely to cause irritation, was a true manifestation of hypersensitivity as was proven by relief of symptoms upon removal of specific allergen.

Rackemann (21) has observed that the time from first exposure to an allergen and appearance of first symptoms varies from six months to six years. If the allergen alone were sufficient to cause hypersensitization in a potentially
hypersensitive individual, the time interval should be the same in all cases between exposure and onset of symptoms. Fatigue, overwork, exposure or acute infectious disease are so often mentioned as preceding the onset of symptoms that they may be considered as essential factors in lowering the individual's threshold of resistance.

True allergic bronchitis occurs more frequently than is often believed. It is important to recognize it among similar conditions so one might be able to detect bronchial asthma in earliest stages. Being aware of its resemblance to hilum tuberculosis and pertussis, one may guard against misdiagnosing these conditions.

Allergic bronchitis occurs in hypersensitive individuals and differs from infectious bronchitis. It is characterized by sudden onset, normal temperature, dry cough and response to ephedrine and adrenalin. It is elicited by substances which are known to cause asthmatic attacks. It may be found as the only sign of an allergic condition.
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