BRONCHIAL GLAND TUBERCULOSIS.*

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We are told often enough that if we would educate a child properly we should begin with its grandmother, and it begins to seem equally probable that if we are to prevent the tuberculosis of the grandmother we must begin with the child.

Bushnell thinks that the child's future is decided before it is five years old, and it is altogether probable that when tuberculosis develops it begins in the bronchial glands. This beginning does not seem to depend upon exposure to infection, nor inheritance, nor bad housing, nor any other known factor, because it occurs under ideal conditions of health and sanitation and perfectly regardless of inheritance or exposure. In an exceedingly healthy hill district, where there had not been a death from tuberculosis in ten years, Hillenberg found that from 25 to 50% of the children gave positive reactions to tuberculin.

A large percentage of our school children give these same positive reactions, and in the majority of cases no tuberculous foci are found. It is thought that the residence of tubercle bacilli in the bronchial glands accounted for these reactions and for the absence of demonstrable foci of infection. This presence of tubercle bacilli does not necessarily mean the development of tuberculous processes. It has been shown by autopsies that the bronchial glands are exceedingly often infected, but that there are times when the tissues are simply swarming with tubercle bacilli and yet there is not the slightest trace of structural changes even under the microscope. This harmless symbiosis may change to the antagonistic symbiosis of von Behring with hyperplasia of the endothelial lining of the lymph passages, but no suggestion of tubercle formation; this lymphoid latency of Bartels is often the end reaction, but cellular reproduc-

tion may be so stimulated as to form neoplasms called tubercles with all the possibilities of necrosis and caseation.

During the first few months of infancy manifest tuberculosis of the bronchial glands is acute, violent and usually fatal. With added years this fulminating character is progressively lost and replaced more and more each year by a hideously subtle, insidious latency. Each year brings less danger for the child and the present and more for the adult and the future because chronicity means the possibility of the extension of tuberculous processes from the bronchial glands to the lungs. With each year this extension is more certain, so that after puberty tuberculosis of the bronchial glands alone is unknown; it disappears as a clinical entity and is replaced by its successor, tuberculosis of the lungs. From birth to puberty bronchial gland tuberculosis may dominate the clinical picture, but after puberty we must admit pulmonary tuberculosis. Given sufficient time, the tuberculous processes in the bronchial glands of children may extend into the lungs and create the pulmonary tuberculosis of adults.

A thorough comprehension of the tuberculous processes in the bronchial glands of children with all their possibilities of danger for adults teaches us the importance of discovering and arresting them at the very start. When we say that this boy eight years old has tuberculosis of the bronchial glands we do not mean that he will die nor even that he is in very great danger, but we do mean that the future of the wage earner, of the parent, and the health of all the years to come is threatened. It is because the tuberculosis of bronchial glands may have to be paid for like the merry madness of the hour by the long penalty of coming time that we insist upon the importance of its early recognition and arrest. This is precisely where we are weak in our present methods of diagnosis. We expect symptoms or physical signs or tubercle bacilli in the sputum to warn us of the danger and these may not exist. Let us consider a typical case of absence of all warnings. Only a few weeks ago a man came to us who had never been sick a day in his life; his home physician was puzzled because a slight irritation in the

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throat would not respond to treatment and so had the sputum examined and tubercle bacilli were found. The radiograph showed dense infiltration of the entire left lung and yet there had never been any cough or temperature or loss of appetite or weight or strength. Not a single symptom of any kind and the loss of the left lung not discovered until it was complete. This is the case of a man of forty built like an athlete, living a most temperate life, from a splendid family with no history of tuberculosis, with plenty of money and no overwork; with not one single predisposing factor, and yet the left lung was completely destroyed without his knowledge. When was the digging of this pit begun, and where and how? Years ago, in earliest childhood, in the bronchial glands. Long before there was any reason for expecting danger, when only the X-ray could have revealed it. It is all too plain now and too easily read—this story of how the bronchial glands enlarged and furnished perpetual supplies of infection for the extension of tuberculous processes through the lungs until the devastation was complete. When and how should this disaster have been prevented? Years ago when, as a romping, healthy boy he offered every promise of a successful life. Talented, well born, well equipped in every way, his future seemed absolutely assured until just at the very height of well deserved and brilliant success he fell with a crash, broken and gone. All this might have been prevented and his life abundantly protected if he had been carefully and regularly examined by an expert well informed and on his guard against the dangers peculiar to good health because of his knowledge that good health is the very cloak behind which the development of tuberculous processes are most effectually concealed. The tuberculosis expert knows full well that healthy school children are in far greater danger from the unsuspected extension of tuberculous processes than the sick ones are because bad health stimulates examinations and good health prevents them.

A second class of cases is equally dangerous and equally benefited by a comprehension of the nature of bronchial gland tuberculosis,—cases with suspicious symptoms not confirmed by physical signs in

the lungs or tubercle bacilli in the sputum.

A mother takes her child to the good old family physician and friend because she is worried by a cough. The child seems well enough; is strong and vigorous; eats well, sleeps well, is up to the standard of weight. The kind old doctor neglects nothing; the throat, the tonsils, the larynx and lungs are all carefully and competently examined and nothing found to explain the cough. With a sigh of relief he straightens up, pats the worried mother on the back, kisses the pretty little girl and tells them to run along home; that there is absolutely nothing the matter because nothing can be found in the lungs. The child eventually marries, has a child of her own, still coughs a little, still is told that there is nothing in the lungs. Finally, after the birth of the second child, the right lung is found involved down to the third rib in front and seventh behind. Is this a sudden development? Has she recently "contracted" tuberculosis? Do we explain the unconscious and rapid production of so much loss by the virulency of the strain of tubercle bacilli? Suppose at the age of five, or whenever the slight cough began, the doctor had said: "A cough, eh? We must see about this." After all the negative findings, suppose an X-ray had been made and the cough explained by the sight of the enlarged glands pressing upon the trachea and bronchi? Bronchial gland tuberculosis with negative findings in the lungs and sputum would then have been the diagnosis.

Or suppose there is not even a cough; that the child is just not quite right; is not strong enough, somewhat listless and lacking in vigor. The mother's instinct alarms her; a careful examination is made; nothing is found. The mother's instinct warns her that all is not well; she tries to increase the child's strength by careful feeding and life in the country; always there is that haunting fear that the doctors may be mistaken: at twenty-two tuberculosis is discovered and now, after seventeen years of delay, physical signs appear. For seventeen years the tuberculous processes in the bronchial glands were overlooked because their importance was not appreciated.

A young man spits a little blood and rushes to his father's friend, the famous

surgeon. "It's nothing, my boy; just a little blood vessel in the throat or stomach; you probably have malaria. There is nothing in the lungs; absolutely nothing to worry about; go on back to work." A few days later a little more blood is raised; the sputum is negative and so are the lungs; another physician and still another guarantee freedom from tuberculosis because of negative findings. If an X-ray had been taken they would have seen the enlarged glands and remembered that hemoptysis all the way up to immediate death by violent hemorrhage is caused by the pressure upon the vessels by bronchial glands. A study of the tuberculosis of bronchial glands teaches us why physical signs are and must be absent in the lungs and tubercle bacilli absent from the sputum and why these negative findings, together with the absence of all symptom complexes, may have absolutely no diagnostic value.

When the bronchial glands enlarge in infancy, the cartilages of the trachea are soft and yielding, and easily pushed in by pressure; this stenosis makes it hard for the air to get out of the lungs. The air enters the lungs easily enough when the chest wall is pulled out and there is an increase of space, but when the wall shrinks back and the bronchi are pressed upon, the stenosis is aggravated and becomes manifested by violent expulsive coughing efforts to overcome it and expiratory dyspnea or stridor. The cough is explosive, highly metallic and resonant, clinking like glass and so pathognomonic that, once heard, it is never forgotten and furnishes a reliable diagnosis without even seeing the child. After the second year the cough changes; it may be paroxysmal like croup or spasmodic like whooping cough; more often, it is like bronchitis and may be only a hemming or clearing of the throat, so slight as to escape attention. Many cases of suddenly fatal pneumonias, without warning of their seriousness, may owe their unexpected fatal end to the underlying condition of the lung which has never been suspected. In these cases there is often a history of a cough since childhood, too slight to be bothered with. It would be worth while to take an X-ray of every child with bronchitic symptoms to see how often the bronchial condition is due to pressure by

enlarged bronchial glands upon the bronchi, causing stasis of the bronchial circulation, a backing up of serous effusions and accumulation of mucus.

The expiratory dyspnea is wet, the rales can be heard at a distance and felt as if a column of fluid were moved up and down by inspiration and expiration. Suffocative, capillary bronchitis may be simulated as well as all kinds of winter or other colds and phases of chronic, frequently recurring bronchitis.

Difficult expiration may create asthma only with difficulty differentiated from true bronchial asthma and the high intra-alveolar pressure so distends the infundibula that emphysema results. The strain on the larger bronchi causes bronchiectasis with accumulation of edematous infiltrations which are expectorated in large quantities, rich in leucocytes, but not containing tubercle bacilli.

If the glands press upon the larger vessels the return of the blood may be impeded; the nasal veins distended, there is facial cyanosis, facial edema, and even edema of the hands and feet. Worst of all, a basis for meningeal involvement is created which all too often follows the circulatory type of bronchial gland tuberculosis. If the esophagus is eroded, evil smelling, crumbling masses are spit up; through the trachea portions of the glands are coughed up; masses of pus and caseous materials, from the bronchi or blood from a large vessel, may prove rapidly fatal.

The constitutional symptoms are too varied to be of much diagnostic value. They belong to tuberculosis as a whole and to neurasthenic, cardiac, gastric, arthritic, pelvic, intestinal and all other disorders so that it might be said that all symptoms lead to tuberculosis. On the other hand, there may be no symptoms at all, because the enlarged glands do not cause irritating processes in the surrounding tissues and give no indications of pressure. Constitutional symptoms depend upon the destruction of tubercle bacilli. If there is no disintegration of the bodies of tubercle bacilli, there is no liberation of toxins and no absorption and no toxemia. What is the danger in these concealed, latent, occult cases?

The danger is that some day these tuberculous processes may extend into the lungs and we never know it. Some of the

bronchial glands lie directly upon the lungs, so close that they amalgamate by tuberculous processes, and it is impossible to determine where the bronchial glands end and the lung begins. Any acute infection, measles, influenza, typhoid, even a "cold," may fill these glands with distended vessels and the backward wash of the lymph carry tubercle bacilli into the adjacent districts. Through the lymph channels surrounding the bronchi and their vessels they are inertly conveyed and deposited out like so many grains of sand. There is no irritation, no inflammation, no tuberculous attack and defense; in utter sluggishness they drift through the interstitial tissues, settling down here and there like so many barnacles, clogging, obstructing, but purely mechanical. In this way the hilum of the lung is first involved; then the main bronchi are followed up into the apex, out into the axilla and down into the base. Our bronchial gland tuberculosis has disappeared as a clinical entity and been replaced by tuberculosis of the lungs, but always these glands remain chances of inoculation from which tertiary tuberculosis, like tertiary syphilis, may at any time develop, be it five, ten or thirty years from now. Jordan has shown how these extensions follow the bronchi as peri-bronchial and peri-vascular infiltrations; as long as they are purely interstitial and dry they cause no alterations in breath sounds or resonance; the larger bronchi are filled with air; their lining is not roughened; there need be no suggestions of inflammatory reactions. Gradually the terminal bronchi are involved and these become tied down so that the air does not enter. The inspiratory murmur is smooth as far as it goes, but it stops abruptly when it reaches the obliterated infundibula; shortening of the length of inspiration, and an abnormally long pause after the stethoscope says the entrance of air has ceased and before expirations begin, are the first changes, preceding suppressed breath sounds. These suppressed breath sounds are merely relative at first: it requires atelectatic areas to complete them. Before atelectasis comes there is the slow, difficult, uneven expansion of the alveoli; you can hear how hard it is for the alveoli to be distended by the insufficiently entering air. These dry, interstitial infiltrations

are the basis of fibroid phthisis and 50% of all cases of pulmonary tuberculosis are of this type, according to Jordan.

If these infiltrations enter the infundibula and leave the interstitial tissues, a lobular area of infiltration constitutes the broncho-pneumonic type of involvement. This type enters the lumen of the bronchus, irritates, congests; there is hyperemia and serous effusions. This edematous infiltration collects, the cells of the alveoli fall in, leucocytes accumulate and we have the typical clinical and physical signs of broncho-pneumonic infiltrations. This type furnishes us with plenty of information as to its presence; the dry, purely interstitial fibroid processes do not yield signs until their nature is changed by necrosis or location. If the processes from the bronchial glands extend out over the periphery of the lung close up under the pleura, it is surprising how dense an infiltration all over the lower lobe of a lung, as shown by the X-ray, may be concealed under relatively normal breath signs and resonance. The frequency of these extensions may come to be associated with the diagnostic significance of idiopathic pleuritis and serous pleuritis. It certainly seems as if these sub-pleural extensions from tuberculous bronchial glands might well be the cause of these pleuritis.

This paper is one of a Symposium on Tuberculosis. The remainder of the papers, together with the discussions of all the papers, will appear in the August issue.

THE RECOGNITION AND TREATMENT OF OCCULT TUBERCULOSIS.*

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The diversified symptoms which may be produced, either directly or indirectly, by an infection with tubercle bacilli, frequently present problems of great difficulty and impose a strain upon the diagnostic acumen of the physician, which is, in many cases, relieved only by accident.

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