bone and joint disease, and a few general statements of principle seem best to meet the need. The orthopedist has quite generally come to recognize that tuberculosis of bones and joints is due very largely to low general resistance of the individual. This being the case, we have two distinct features to observe in our treatment. One is to treat the part locally where the disease manifests itself, and the other is to see that everything is done that is possible to improve the general condition of the individual. The time was when the wards of our children's hospitals were filled with patients having hip or spine disease where the entire emphasis was put upon the treatment of the local condition. We have gone far beyond that point. The general treatment should receive at least equal attention.

It seems to me that we can pass over the local treatment by saying that in tuberculosis of the bones or joints, the things that the orthopedist aims to do are first to prevent deformity, or correct deformity if it has already occurred, and second, to put the part in the position of physiological rest—then allowing function in so far as the part will tolerate it. Anything that the individual can do with a diseased part (the individual, not someone else) we need have very little fear of doing harm. If the child can walk about once, the part is properly protected—let him walk about. If it is very acute, the child will instinctively not walk about. As fast as the child is able to use that part, it is to be encouraged. Use stimulates circulation, encourages bone repair and does the very thing we ought never to lose sight of—which is to stimulate the cells about the diseased portion, so that they will be stronger and able to control the disease rather than to weaken those parts by too great inactivity.

For the general condition the things which we are now doing have for their purpose the increasing, in so far as is possible, the general resistance and therefore the more perfect control of the local manifestation of the disease. This applies not only to tuberculosis, it applies to all sorts of infections,—the principles are practically the same. The thing that has helped us most in this connection and has given us something that we can speak about definitely because it is easily checked, has to do with the way the diaphragm is used. It seems to me that this is one of the most important single features of the physiology of the human being. If the diaphragm is down at the level of the twelfth rib and does not move—the circulation of the abdominal organs must suffer. Normal respiration involves the diaphragm and the normal circulation in the abdomen demands the same thing. Ideal respiration means the diaphragm high with slight movement of the chest—slow respiration and slow pulse. The diaphragm not only helps in the respiratory development, but it also pumps the blood from the abdomen and

the legs back to the heart. There is no other mechanism to accomplish this. With the diaphragm low, the circulation as well as the respiration will be interfered with and the general vitality will suffer.

Until you get the diaphragm so that it acts freely, you have not done all that you should do to improve the general resistance of the individual.

In trying to build up your individual to the best health—in trying to bring up their reserve so they can avoid tuberculosis—you have tangible things which you can check. Certainly there is more than just the microscope or the stethoscope in understanding these conditions.

Training for this physiological test is the sort of thing our preventoria should be doing. Get the children to stand properly. They will look better, but they will be more normal physiologically, and have greater resistance to tuberculosis or any other disease.

TUBERCULOSIS IN CHILDREN FROM THE STAND POINT OF THE INTERNIST.

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IN considering the subject of tuberculosis in children it is necessary to divide them into groups as this disease varies very much in its manifestation according to the age of the child. There are three distinct types of tuberculosis in children:—the general or infantile, the hilum or juvenile, and the apical or adult form.

The infantile form in its primary stage assumes the characteristics of an acute, infectious fever, and progresses rapidly to a fatal termination, as general miliary tuberculosis, meningitis or bronchopneumonia. This is the usual course when infection occurs before the age of two years. From this age on the cause of infection is gradually modified by the production of a steadily increasing immunity. The tissues gradually acquire the power to react and the bacilli become fixed in the lymph nodes or more rarely in the epiphyses of the long bones or in the bodies of the vertebrae. After children have reached the age of five they rarely develop the infantile type of tuberculosis.

From five to twelve the lymphoid type of tuberculosis largely predominates. This period is a golden interval of low mortality between the infantile form on the one hand and the adult pulmonary form on the other, both of which have very high death rate. This tendency to focalize in the lymph nodes is the outstanding characteristic of juvenile tuberculosis. This was very apparent a few years ago when cervical tuberculous adenitis was such a common affliction in the children that came to the surgical outpatient departments. It is not so today largely because of the activities of the throat specialists in removing adenoids and diseased tonsils. Also the dentists must be given some of the credit because of the progress of dental hygiene.

Only comparatively recently, however, has the rôle of the tracheo-bronchial or hilum glands been given serious consideration in relation to the tuberculosis problem. As the lymphatics of the throat drain into the cervical glands, so the lymphatics of the lungs converge at the hilum and drain the entire pulmonary area into the tracheo-bronchial lymph nodes. There is this important difference however; the cervical glands only drain a small region, the tracheo-bronchial lymph nodes become the converging point of all bacilli that reach the lymphatics of the lungs, either by inhalation or through the pulmonary circulation that may take up infection from any part of the vascular system. There is, therefore, little chance for this group of glands in any individual to escape infection as long as tuberculosis is the universal disease that it is today. Fortunately the bacilli are so few in number or they are low in virulence or the individual's immunity is sufficient to prevent active disease in 70% of the people.

Pathologists are not yet in agreement as to whether the first lesion occurs in the lungs or in the tracheo-bronchial nodes. Krause has demonstrated that animals inoculated with tubercle bacilli exhibit foci of tuberculosis in the tracheo-bronchial nodes even when the lungs are free from disease. Furthermore, we do know that whenever the lungs show a tuberculous lesion that the tracheo-bronchial nodes are always involved. Most important of all to us is the fact that tuberculosis can be demonstrated in the glands of the hilum before any pulmonary lesion is large enough to be recognized by any method of examination now available.

Apical lesions or the adult type of tuberculosis rarely occur before the age of twelve years. After this age it is met with increasing frequency until the sixteenth year, when it becomes the usual type. The juvenile form after this age is rarely found as active disease. The healed hilum lesions are, however, often seen by x-ray or post-mortem in adults.

It is most important to recognize hilum tuberculosis when it is confined to the lymph nodes because this tissue has a tendency to keep the focus circumscribed. The resistance to tuberculosis between the ages of five to fifteen is greater relatively than at any other age period. Arrest or cure can reasonably be expected if proper treatment is carried out at this time.

There is a transition period due to the developing immunity between the ages of two to five, when either the infantile or juvenile type of tuberculosis may prevail. It is a dangerous period in an infected child's life. The disease is masked more completely than it is at later years. The child is usually well developed and nourished and of good color. Localizing symptoms may be delayed and there ensues an anxious period when the physician is puzzled to know

the cause of the occasional reoccurring febrile attack. Is it due to tuberculosis, and if so will the tubercle focalize in the head of a long bone, in the vertebral column, in the peritoneum, in the intestines or in some group of lymphatic glands? Those are some of the questions that a clinician has to answer.

Again we have another dangerous transition period after the child passes the age of twelve. Something as yet unknown to us occurs during the age of adolescence that influences the course of tuberculosis. The cases one sees of involvement restricted to the region of the hilum become fewer and the number of apical or adult type of tuberculosis increases as the patients approach the age of sixteen. It occasionally happens, but it is extremely rare to find a case of active hilum tuberculosis in an adult without apical lesions.

Juvenile tuberculosis involves the lymphatic glands at the hilum and the adjacent peribronchial tissue in its early stages. If the disease progresses the deep parenchymatous tissue between the trunks of the bronchial tree becomes invaded by infiltration. There may be peribronchial thickening with beading along the trunks that extend into the second and third interspaces.

Adult pulmonary tuberculosis involves the apices and the peripheral parenchymatous tissue. If one bears in mind this difference in the pathology of the two types of disease it will aid materially in diagnosis. The physical signs are different because in one the focus is superficial and in the other it is deep. Furthermore, in one the alveolar structure of the lung is involved and in the other the lymphatic tissue. Auscultation therefore does not give any positive information in early tracheo-bronchial tuberculosis. As the disease progresses and invades the deep parenchyma and peribronchial tissue changes in the breath sounds are sometimes heard in the infraclavicular triangle and along the vertebral border of the scapulae. Râles are not heard in hilum tuberculosis; the structures in which râles are produced, namely, the smaller bronchi and alveoli, are not involved until the pulmonary or adult type of tuberculosis is developed. If râles are numerous in any part of one or both lungs of a child from five to twelve they are probably due to some non-tuberculous infection, such as bronchitis, asthma, bronchopneumonia or abscess. If the râles are due to pulmonary tuberculosis, it is a case in the advanced stage, which fortunately rarely occurs in children of this age. One should not expect to elicit râles in early juvenile tuberculosis; when heard they should be regarded as negative evidence and should be considered due to non-tuberculous causes unless enough positive signs and symptoms of tuberculosis are present to justify such a diagnosis.

Percussion, however, is of great value in determining consolidation at the root of the lung. One must develop a careful technic to be of serv-

ice in revealing changes in the deeper structures of the lung. The child to be examined should be standing with shoulders slightly sloping forward with all muscles of the shoulder girdle in complete relaxation. Light percussion must be used with dependence for values placed more upon the tactile sense than upon the sound produced. It is always better to begin from below and progress upward. The reason for this is that the bases are seldom diseased and one can in this way perceive the normal resonance first for a standard and thus more easily detect any abnormal changes in the upper part of the lung. It should also be borne in mind that in children with active tracheo-bronchial nodes disease there is a reflex condition that tends to produce a chronic spasm of the muscles covering the upper part of the thorax posteriorly. This may be sufficient to change the percussion note enough to simulate dullness due to tuberculous infiltration of the apices. As a check, however, upon such an erroneous interpretation, one finds in such cases that the resonance is normal over the apices anteriorly and râles are not present.

SYMPTOMS.

In making a diagnosis in children great significance must be placed on symptoms which are an evidence of tuberculin toxemia. In the order of importance these symptoms are fatigue or weakness. This is the first and most frequent of symptoms of onset. It is exhibited often as a change of disposition. A sunny, happy child becomes nervous and fretful, or an active, energetic one becomes languid and listless. Games no longer interest them and school work that was formerly enjoyed becomes a hardship. Light mental or physical efforts tire. Lack of appetite, failure to gain, or—more significant—loss of weight; tendency to profuse perspiration; fever. In hilum tuberculosis the toxemia does not produce continuous fever. It occurs only after unusual exercise or fatigue from any cause. It is transitory. An afternoon temperature that does not go above 99.6 may be considered within the normal limits. A temperature of 101 or more is usually due to some non-tuberculous cause. It should be remembered that tuberculous disease of the lymph nodes or pulmonary tissues when not accompanied with pyogenic infection does not produce a marked rise in temperature. A purely tuberculous disease produces only a low grade of fever and then only when an increased amount of tuberculin is absorbed, as a tuberculin tolerance is gradually acquired. We must not, therefore, consider the absence of fever to contraindicate juvenile tuberculosis. Febrile attacks are infrequent, long fever-free periods are the rule.

A child's pulse is so erratic and normally so much higher than in adults that it is of no value in early diagnosis.

TUBERCULIN TEST.

We have found the intradermal tuberculin test so much more reliable than the Von Pirquet cutaneous method that we are using it exclusively. Absorption is certain and the dose accurate in each case. We give .01 m.g. of tuberculin intracutaneously for the first dose. If there is no reaction a second dose of 0.1 m.g. is given.

PREVIOUS HISTORY.

The exposure of a child to tuberculosis in the home, if long continued, is evidence of great weight in so far as it indicates the probability of massive and frequent infection. Such a child should be provided with excellent hygienic surroundings and care, even if there are no symptoms of disease. The lack of family history, however, as regards exposure to tuberculosis in the home, is of little value and should have no negative weight in diagnosis if there are definite signs and symptoms of disease. Out of a series of 200 children in our care 34% gave no history of intimate contact with any case of tuberculosis in the home.

DIAGNOSIS.

The points upon which a diagnosis of juvenile tuberculosis must be based are these:—

1. History of frequent colds or predisposing causes such as measles, whooping cough, and influenza. These diseases lessen the resistance to infection and tuberculous disease frequently follows in their wake. Also the frequent association of phlyctenular disease and scrofuloderma with hilum tuberculosis should always be kept in mind.
2. Symptoms of tuberculin toxæmia, viz.—fatigue, malnutrition, anorexia, febrile attacks.
3. Exposure to tuberculosis in the home.
4. Positive cutaneous tuberculin test.
5. Evidence of enlarged tracheo-bronchial lymph nodes and infiltration or thickening of the adjacent tissues as demonstrated by physical signs and confirmed by roentgenogram.

In conclusion I wish to emphasize the point that juvenile tuberculosis is a manifestation of primary tuberculosis, apparent only in the lymph nodes and deeper tissues at the root of the lung. The adult type is a manifestation of tuberculous disease involving the superficial parenchymatous and peribronchial tissues of the lungs. The tissues involved are radically different in structure. The way in which they react to disease is distinctive. Therefore when we examine the chest of a child we should not expect to find râles and other signs of pulmonary tuberculosis. We should look carefully for evidence of bronchial adenitis. When we find enlargement of the bronchial lymph nodes with thickening of adjacent tissues, accompanied by symptoms of a tuberculin toxemia, together with a positive intracutaneous test, a diagnosis of hilum tuberculosis should be made.