and treatment for 6 or 7 days will usually prove you correct.

Treatment is as a rule simple. The babies are usually bottle-fed and proprietary foods, condensed milk or cooked milk are used. Modified uncooked milk with orange or lemon juice should be given, one-half to one lemon or orange being used daily, with fresh beef juice. These children in my experience are greedy for the juice of both orange and beef. I usually give iron, etc., for the debility and anemia. In severe cases the child must be kept quiet on a frame, but I have never had to resort to this as yet.

I am aware that in what I have said I have considered the subject from the somewhat superficial point of view of a general practitioner, but my object is to make it clear what a very easy disease scrobutus is, as a rule, to recognize, and also how many cases we all have probably overlooked in the past, and called something else.

RHACHITIS.¹

BY ARTHUR R. CRANDELL, M.D., TAUNTON, MASS.

RHACHITIS is a chronic disease of nutrition.

It is essentially a disease of infancy, the symptoms usually appearing between the ages of 6 months and 2 years.

No race is free from its attack, but it is much more common among nationalities transported from warm to more temperate climates, as exemplified by its frequent occurrence in Negroes and Italians in this country.

Infants reared in the city are more prone to the disease than are the country-bred.

Etiology.—Various theories have been advanced to account for the origin of rhachitis, but they have been discarded, one by one, until it is now believed that this disease results from chronic malnutrition, often re-enforced by poor hygiene and unsanitary environment. In support of this view is adduced the fact that breast-fed infants almost never develop rhachitis, unless lactation be unduly prolonged, or the breast milk be poor in quality. Artificially-fed infants, on the other hand, frequently show evidences of rhachitis, especially when fed improperly. The diet in these cases is usually largely deficient in fats and proteids, with an excess of carbohydrates. This accounts for the common occurrence of rhachitis in children fed on proprietary foods, most of which contain low percentages of fats and proteids, with carbohydrates in improper proportion and often in improper form, as starch and cane sugar.

The influence of poor hygiene and unsanitary surroundings is shown by the frequency of rhachitis in city infants, compared with those of the country, especially in the city children living in crowded districts, where air and sunlight are insufficient and the mode of living unhealthful. How improper feeding and poor environment are

causative in the production of rhachitis is a subject for further speculation and study.

Gross pathology.—While all the organs of the body suffer from the effects of rhachitis, the essential lesions are in the bones. The grosser changes require a brief description.

The long bones ordinarily grow in length by the formation of bone in the cartilage between the epiphysis and the shaft; in thickness by the formation of bone beneath the periosteum; and at the same time the medullary canal grows in size by the absorption of the inner layers of the bone (Holt). In health the seat of greatest activity in growth is at the epiphyses; in rhachitis the most marked changes are found in the same locality. Thus in a rhachitic long bone the epiphyses are large and rounded, and the cartilage between the epiphysis and the shaft is wider and thicker than normal, while the process of ossification is delayed or may be entirely arrested. In the shaft of the bone the outer layers are thickened and soft, and the absorption of the medullary layers is more rapid than normal. The proportion of organic to inorganic matter is much increased, rendering the bones softer and more flexible. The long bones most affected are the ribs and the large bones of the extremities, most frequently those of the leg and forearm.

The flat bones are affected most markedly in the region of the centres of ossification, where a thickening occurs, giving rise to soft and spongy prominences, most often seen in the frontal and parietal region of the cranium.

As the disease subsides after some months, the epiphyses lessen in size, the prominences of the skull decrease, and there is a normal formation of bone. The parts affected often never regain their natural size, and are almost always harder than normal, owing to a process of condensation and contraction in the bony structure.

Early symptoms.—The pathognomonic symptom of rhachitis is the development of the bone lesions, but premonitory signs occur which lead to a suspicion of its oncoming. Perhaps the earliest of these is profuse sweating of the head accompanied by irritability and restlessness, particularly at night.

Delayed dentition is present in a large proportion of the cases, is a very suggestive symptom, and should always put the practitioner upon the watch for other rhachitic manifestations.

Enfeebled muscular action, again, is a suspicious condition; and delay in holding up the head, in sitting up or walking, is a frequent premonitory symptom. This apparent backwardness is often the most fruitful cause of anxiety to the parents, other signs remaining unnoticed, or thought to be of little importance.

A persistent disturbance of the bowels, either constipation or diarrhea, may also herald the approach of the disease.

LATER SYMPTOMS.

(1) *Osseous system.*—Of the bone lesions, one is first and constant, according to Morse,

¹ Read before the Massachusetts Medical Society, June 11, 1901, as a part of the general topic, "The Diseases of Nutrition of Infants."

and that is the "rosary" or beading at the junction of the ribs with their cartilages. This is not present in normal children, and its occurrence justifies the diagnosis of rhachitis. The beading is always greater on the inside than on the outside of the chest wall.

The anterior fontanel normally should be closed by the 18th or 20th month, but in rhachitis usually remains widely open after that time; the posterior fontanel also occasionally remains open.

The formation of the frontal and parietal bosses previously mentioned gives the head a curiously square appearance and causes an actual increase in its circumference. The parietal eminences are more apt to be enlarged than are the frontal.

Craniotabes may occur. The other symptoms resulting from changes in the osseous system give rise to deformities more or less noticeable and permanent according to their severity.

The chest shows an increase in its anteroposterior diameter caused by the effect of atmospheric pressure upon the yielding ribs. This may be so marked as to form a distinct pigeon breast.

If the entrance of the air to the lungs be impeded by the presence of adenoids or enlarged tonsils, the sternal region may be depressed, causing the funnel chest.

There may be, and very often is, a lateral depression about the chest, corresponding to the insertion of the diaphragm. The contraction of this muscle pulls in the ribs at the point at which it is attached to them, and causes a furrow in the chest wall. This brings about a flaring of the lower costal region which is accentuated by the presence of the normally large liver of infancy.

An almost invariable symptom is the enlargement of the epiphyses of the long bones of the extremities, most marked at the wrists and ankles.

The diaphyses of the long bones also show changes, both bending and torsion, resulting from muscular pull and improper distribution of weight upon the softened bony structure. Bow legs are the commonest of these deformities, varying from the mild cases, where the condition is hardly more than an enlargement of the epiphyses at the ankle, up to the severer ones, where walking is almost impossible. Knock knee is frequent. Deformities of the forearm, especially an anterior bowing, are not uncommon. In fact all the long bones of the extremities may assume distortions of all grades of severity.

Other diseases involving changes in the bones of the extremities may here be differentiated.

In scurvy there is an apparent bony enlargement, but one really due to subperiosteal hemorrhage. Further, this occurs about the shaft and not in the epiphyses of the bone. The extreme tenderness accompanying scurvy, the subcutaneous hemorrhages, and the spongy and bleeding gums enable it to be differentiated. Immediate improvement on antiscorbutic diet clears up any doubt as to the diagnosis.

Syphilis is also frequently accompanied by bony changes in the extremities. Here again the

epiphysis is not affected, but the enlargement is at the junction of the epiphysis and diaphysis. It is a boggy infiltration over the bones and not an actual enlargement of the bone itself. It may be unilateral and may break down, conditions not found in rickets. The latter bone lesions of hereditary syphilis affect the shaft rather than the extremity of the bone.

In rhachitis there may be occasionally slight tenderness over the enlarged epiphyses. In my experience this has been very uncommon. If sufficient to attract attention and suggest a diagnosis of rheumatism, the absence of local heat and tenderness, the normal temperature and the presence of other rhachitic symptoms render the differential diagnosis simple.

(2) *Muscular system*.—Rhachitic children are usually fat and may appear at first glance to be well developed. The muscles, however, suffer from malnutrition, as do the other organs of the body. They are weak and flabby, and their action is enfeebled. The delay in holding up the head, in sitting up, and in walking, has already been mentioned.

Another very noticeable characteristic is the enlarged abdomen or "pot belly." This is due to weakness of the abdominal muscles, the condition being accentuated by the flaring of the lower costal margin and the atony of the muscular coats of the intestine which results in distension of the bowels. Percussion usually shows marked tympany of the whole abdomen. The absence of fever and free fluid differentiates any abdominal inflammatory condition, such as tubercular peritonitis, which might be suggested.

Weakness of the back muscles may be made manifest by the appearance of a rounded kyphosis involving the lower dorsal and lumbar regions. This is generally accompanied by abnormal flexibility of the spine and usually disappears on recumbency. The reverse may be true, and the persistency and rigidity of the kyphos may suggest caries to such an extent that recumbent treatment for a while is necessary for differentiation. The presence of other rhachitic symptoms is of considerable value in the diagnosis, and the absence of other symptoms of Pott's disease, such as increased patellar reflexes, psoas contraction and abdominal pain, makes the latter much less probable.

Weakness of the muscles of the extremities may be great enough to simulate paralysis, either cerebral spastic or anterior poliomyelitis. The former is ruled out by the absence of mental impairment, of increased reflexes and muscular spasm or contractures; the latter, by the normal surface temperature of rickets, the normal reflexes, the absence of atrophy of any particular region or extremity, and, in case of doubt, by the electrical reactions.

(3) *Ligaments*.—The ligaments show evidence of impaired nutrition in an undue laxity, especially in the lower extremities. Here there may be hyperextension of the knee-joints, but more characteristic are the weak ankles.

Flat foot due to muscular and ligamentous weakness is common, a deformity which is increased by the improper weight distribution resulting from bow legs and knock knees.

(4) *Respiratory organs.*—There are no constant changes in the respiratory system. If the thoracic deformities are marked, especially depressions in the chest wall, the underlying portion of the lungs may be collapsed, with emphysema of the region just anterior to the atelectatic area. The bronchial mucous membrane shows an increased susceptibility to disease, and the respiratory muscles are of diminished power as the result of poor nutrition. These conditions render bronchitis common, especially in the severer cases, and bronchopneumonia a frequent and dangerous complication.

(5) *Gastro-enteric tract.*—The intestinal mucous membrane, like the bronchial, is more vulnerable, and gastro-enteric disturbances are therefore common. These may be of all forms and of all grades of severity. There is nothing about them that is characteristic. The atony of the muscular walls of the intestine results in diminished peristalsis, often accompanied by putrefactive changes in the intestinal contents. Thus arises the distension of the bowels already referred to. Either constipation or diarrhea is usually present, and a normal regularity of the bowels is uncommon. These conditions are fostered by the improper diet of the rachitic infant. The liver is not very often increased in size, although its downward displacement by the contraction of the chest often causes it to appear abnormally large.

(6) *Spleen.*—The spleen is enlarged in a small proportion of cases, the enlargement being due to simple hyperplasia. There seems to be no relation between the size of the spleen and the severity of the particular case, or the condition of the blood. There is a return to normal size with subsidence of the disease.

(7) *Blood.*—The impaired nutrition in rachitis is accompanied by an anemia which has no special characteristics. The more marked the rachitis, the more severe the anemia is apt to be. The hemoglobin is usually relatively low. There may be a leucocytosis.

(8) *Nervous system.*—The nervous system of the normal infant is unstable. This condition is aggravated in rachitis through the defective nutrition of the nervous centres, rendering nervous manifestations early and common. Restlessness at night and irritability have already been noticed. Laryngismus stridulus and tetany frequently occur. Holt states that, as a predisposing cause of convulsions in infancy, rachitis takes the first place. These are due to the quick response of the unduly susceptible nerve centres to relatively slight stimuli.

(9) *Glandular system.*—General glandular enlargement is not infrequent, but is not especially characteristic. Enlargement of the tonsils and the occurrence of adenoids are not uncommon. Their operation in producing thoracic deformity has

been mentioned. Their presence gives an added susceptibility to pulmonary complications.

Diagnosis.—The premonitory signs of rachitis, as head-sweating, irritability, restlessness at night, prolonged gastro-enteric disturbances and delayed dentition have been mentioned. These symptoms should excite suspicion. The detection of the rosary and the enlarged epiphyses renders the diagnosis certain. The clinical picture is completed by the appearance of the prominent cranial bosses, the delayed closure of the anterior fontanelle, the various thoracic deformities, the pot belly, deficient muscular action, and, as the disease progresses, by the changes in contour of the extremities. The various diseases which might obscure the diagnosis have already been considered.

Prognosis.—The prognosis of uncomplicated rachitis is good. The disease untreated tends to a spontaneous recovery after a duration of several months. With the use of a more general diet after the first year or year and a half, and as the food elements are supplied in better proportion, there is a gradual improvement in condition. The affected organs slowly regain their normal state, unless the lesions have been severe, under which circumstances there may be a varying persistence of the several deformities, even to permanency.

Pulmonary, gastro-enteric and nervous complications are frequent, and render the prognosis grave. Any acute disease in a rachitic infant assumes a severe type and is increased in danger.

Treatment.—Cheadle says the occurrence of rachitis is in most cases a grave reflection upon those responsible for the child's diet. Prophylaxis is, therefore, the most efficient treatment. This comprehends painstaking and accurate feeding, careful hygiene and supervision of methods of living; if, however, prophylaxis has been neglected, then active treatment must be carried out.

Since rachitis is due largely to the deficiency of fats and proteids in the food, a proper percentage of each must be supplied, and improper elements in the diet must be eliminated. Regular feeding must be instituted, and the infant kept under observation, that changes in the food may be made when necessary.

Proper amounts of light and air must be secured. City living should be exchanged for life in the country, or better still, life at the seashore. If poverty prevents this, the child may be taken on daily trips to spots where light and air are more abundant. Bathing and massage favor bodily nutrition.

Drugs.—The drug treatment of rachitis is of minor importance. There is no specific for its cure, phosphorus, once recommended so highly, having been practically discarded. While milk and cream afford fat in the best form, it is sometimes expedient to administer it as cod-liver oil in assimilable doses. The anemia is to be treated by the use of iron.

Deformities.—Many deformities may be prevented if their possible occurrence is borne in mind. Creeping, walking and sitting up may cause

bending of the softened bones, and should be allowed guardedly until the bones begin to harden. Manipulation will do much to reduce the lesser deformities of the extremities while the bones are flexible. Deformities that are severe, and remain in spite of all measures undertaken, require orthopedic treatment. Something may be done for the thoracic changes by various exercises, while the distorted bones of the extremities may be corrected by osteotomy at the proper time. The use of apparatus in cases of bow legs and knock knees results commonly in annoying failure, and is an unnecessary infliction upon the patient. Osteotomy, when the bones have regained their normal hardness, but not before, is much to be preferred.

Medical Progress.

REPORT ON PATHOLOGY.

BY JAMES H. WRIGHT, M.D., BOSTON,

Director of the Clinico-Pathological Laboratory, Massachusetts General Hospital.

THE "JOURNAL OF MEDICAL RESEARCH."

It is a gratifying sign of the progress of pathology in North America that the *Journal of the Boston Society of Medical Sciences* has been enabled to expand into the new *Journal of Medical Research*.

The initial number contains the papers read at the first meeting of the Association of American Pathologists and Bacteriologists.

This collection of papers compares favorably with any volume of pathological transactions with which we are acquainted, and its appearance is a most gratifying indication of the increasing activity of American medical men in scientific medicine.

The new journal is very tastefully printed and is well provided with illustrations in the best form. It reflects great credit upon Dr. Harold C. Ernst, its able editor.

YELLOW FEVER AND MOSQUITOES.

The brilliant work on this subject by Drs. Walter Reed, James Carroll and A. Agramonte¹ in Cuba has been so thoroughly discussed in this and other journals that it is superfluous to report the results of their work here.

BOVINE AND HUMAN TUBERCULOSIS.

The important statement made by Professor Koch at the British Congress on Tuberculosis, that he does not believe that the bacillus of bovine tuberculosis is capable of producing tuberculosis in man, has been received with considerable astonishment by medical men.

This subject has recently been discussed thoroughly in the JOURNAL so that it is unnecessary to enter into it here. The position taken by the JOURNAL in its editorial of Aug. 1, 1901, probably

¹ Transactions of the Association of American Physicians, 1901, vol. xvi.

represents the best opinion among pathologists on this question.

THE SOURCE OF THE LEUCOCYTES IN LEUCOCYTOSIS.

The view, that polynuclear leucocytes are formed in the bone marrow, receives additional confirmation in two recent publications. Each of the observers has produced experimental leucocytosis in rabbits and studied the changes in the bone marrow.

Robert Muir² produced leucocytosis by means of inoculations with the staphylococcus pyogenes aureus.

H. Rubinstein³ used streptococcus cultures, turpentine and other substances which are capable of producing a marked leucocytosis.

Each of these observers found, associated with leucocytosis, increase in the number of certain cells in the bone marrow, which cells they regarded as the mother cells of the polynuclear leucocytes in the circulating blood. It is not apparent from their papers that they are in agreement with one another as to the characters of these cells, which they find increased in number, although probably each has observed an increase in the same kind of cells.

THE PROSTATE AND GENERAL INFECTION.

H. Ullmann⁴ reports 4 cases of crypto-genetic septicopyemia, in each of which it was found at the autopsy that the source of infection was a suppurative process in the prostate, presumably a more or less remote result of gonorrhea.

In the light of these observations the author points out the importance of examining the prostate in all cases of obscure septicopyemia, even if there are neither signs nor history of gonorrhea.

THE ELASTIC TISSUE OF THE LUNG IN CHRONIC PASSIVE CONGESTION.

R. M. Pearce⁵ has investigated the question of the increase of elastic tissue in the lung in chronic passive congestion, making use of Weigert's selective stain for elastic tissue. The result of his work he summarizes as follows:

"In chronic passive congestion the increase in density of the lungs is due, in large part, to the newly-formed elastic tissue. This tissue is found to be increased in the finer structures of the lung in all cases of the disease, and in marked cases an increase is also observed in the pleura, intrapulmonary septa, blood vessels and bronchi. The increase is progressive, depending on the age and degree of the congestion, and it apparently indicates an effort to strengthen the walls of the air passages, supporting the over-filled capillaries, and preventing the collapse of the air cells."

INTESTINAL PARASITES AND APPENDICITIS.

El. Metchnikoff⁶ believes that a large majority of the cases of appendicitis are due to intestinal

² The Journal of Pathology and Bacteriology, February, 1901.

³ Zeit. f. Klin. Med., Bd. XLII, S. 161.

⁴ Deutsch. Arch. f. Klin. Med., Bd. LIX, S. 309.

⁵ University of Pennsylvania Medical Bulletin, vol. xiv, p. 228.

⁶ Acad. de Méd., séance du 12 Mars, 1901; Semaine Médicale, March 13, 1901.