of animals which, when punctured, produced polyuria. Lesions of the organs of the abdomen may be associated with an excessive flow of urine, which, however, should not be regarded as true diabetes insipidus. Dickenson mentions its occurrence in abdominal tumors; Ralfe, in abdominal aneurism. I have noted it in several cases of tuberculous peritonitis.

The nature of the disease is unknown. It is, doubtless, of nervous origin. The most reasonable view is that it results from a vaso-motor disturbance of the renal vessels, due either to local irritation, as in a case of abdominal tumor, or to central disturbance in cases of brain-lesion, or to functional irritation of the centre in the medulla, giving rise to continuous renal congestion.

Morbid Anatomy.—There are no constant anatomical lesions. The *kidneys* have been found enlarged and congested. The *bladder* has been found hypertrophied. Dilatation of the ureters and of the pelves of the kidneys has been present. Death has not infrequently resulted from chronic pulmonary disease. Very varied lesions have been met with in the nervous system.

Symptoms.—The disease may come on rapidly, as after a fright or an injury. More commonly it develops slowly. A copious secretion of urine, with increased thirst, are the prominent features of the disease. The amount of urine in the twenty-four hours may range from twenty to forty pints, or even more. The specific gravity is low, 1·001 to 1·005; the color is extremely pale and watery. The total solid constituents may not be reduced. The amount of urea has sometimes been found in excess. Abnormal ingredients are rare. Muscle sugar, inosite, has been occasionally found. Albumen is rare. Traces of sugar have been met with. Naturally, with the passage of such enormous quantities of urine, there is a proportionate thirst, and the only inconvenience of the disease is the necessity for frequent micturition and frequent drinking. The appetite is usually good, rarely excessive as in diabetes mellitus. The patients may be well nourished and healthy-looking. The disease in many instances does not appear to interfere in any way with the general health. The perspiration is naturally slight and the skin is harsh. The amount of saliva is small and the mouth usually dry. Cases have been described in which the tolerance of alcohol has been remarkable, and patients have been known to take a couple of pints of brandy, or a dozen or more bottles of wine, in the day.

The course of the disease depends entirely upon the nature of the primary trouble. Sometimes, with organic disease, either cerebral or abdominal, the general health is much impaired; the patient becomes thin, and rapidly loses strength. In the essential or idiopathic cases, good health may be maintained for an indefinite period, and the affection has been known to persist for fifty years. Death usually results from some intercurrent affection. Spontaneous cure may take place.

Diagnosis.—A low specific gravity and the absence of sugar in the urine distinguish the disease from diabetes mellitus. Hysterical polyuria may sometimes simulate it very closely. The amount of urine excreted may be enormous, and only the development of other hysterical manifestations may enable the diagnosis to be made. This condition is, however, always transitory.

In certain cases of chronic Bright's disease a very large amount of urine of low specific gravity may be passed, but the presence of albumen and of hyaline casts, and the existence of heightened arterial tension, stiff vessels, and hypertrophied left ventricle make the diagnosis easy.

Treatment.—The treatment is not satisfactory. No attempt should be made to reduce the amount of liquid. Opium is highly recommended, but is of doubtful service. The preparations of valerian may be tried; either the powdered root, beginning with five grains three times a day, and increasing until two drachms are taken in the day, or the valerianate of zinc, in fifteen-grain doses, gradually increased to thirty grains, three times a day. Ergot is recommended by DaCosta. Ergotin may be employed. Large doses are required. Antipyrin, the salicylates, arsenic, strychnine, turpentine, and the bromides have been recommended. The constant current may be used—one pole on the loins, the other on the nape of the neck.

IX. RICKETS.

Definition.—A disease of infants, characterized by impaired nutrition and alterations in the growing bones.

Glisson, the anatomist of the liver, described the disease accurately in the seventeenth century.

Etiology.—The disease exists in all parts of the world, but is particularly marked among the poor of the larger cities, who are badly housed and ill fed. It is much more common in Europe than in America. In the colored race it is frequently seen. It is a comparatively rare disease in Canada. In the larger cities of this continent it is frequently seen at the clinics, but in comparison with Vienna and London the contrast is very striking. In these cities from 50 to 80 per cent of all the children at the clinics present signs of rickets. Want of sunlight and impure air are important factors. A starchy diet, too much cows' milk, and the indiscriminate feeding, so common in the children of the poor, are important agents; but something is required beyond these, for children of healthy parents, who have an ample quantity of the proper food, may become rickety. It seems probable, however, that the combination of defective food and bad air plays the most important *rôle*. Prolonged lactation or suckling a child during pregnancy are accessory etiological factors.

There is no evidence that the disease is hereditary, but there is probably a form of fœtal rickets. It is doubtful, however, whether the changes met with in this are identical with the post-natal disease. In these babies,

which are generally still-born, the limbs are short, the curves of the bones are exaggerated, and at the junction of the epiphyses there is no proliferating zone of cartilage. This condition, which Parrot calls *achondroplasy*, is really more like a fœtal *cretinism*.

Rickets affects male and female children equally. It is a disease of the first and second years of life, rarely beginning before the sixth month. Jenner has described a late rickets, in which form the disease may not appear until the ninth or even until the twelfth year. It has been held that rickets is only a manifestation of congenital syphilis (Parrot), but this is certainly not correct. Syphilitic bones rarely, if ever, present the spongy tissue peculiar to rickets, and rachitic bones never show the multiple osteophytes of syphilis. It has been regarded as an effect of malaria.

Morbid Anatomy.—The bones show the most important changes, particularly the ends of the long bones and the ribs. Between the shaft and epiphyses a slight bulging is apparent, and on section the zone of proliferation, which normally is represented by two narrow bands, is greatly thickened, bluish in color, more irregular in outline, and very much softer. The width of this cushion of cartilage varies from five to fifteen millimetres. The line of ossification is also irregular and more spongy and vascular than normal. The periosteum strips off very readily from the shaft, and beneath it there may be a spongioid tissue not unlike decalcified bone. The practical outcome of these changes is a delay in, and imperfect performance of, the ossification, so that the bone has neither the natural rate of growth nor the normal firmness. In the cranium there may be large areas, particularly in the parieto-occipital region, in which the ossification is delayed, producing the so-called cranio-tabes, so that the bone yields readily to pressure with the finger. There are localized depressed spots of atrophy, which, on pressure, give the so-called "parchment crackling." Flat hyperostoses develop from the outer table, particularly on the frontal and parietal bones, and produce the characteristic broad forehead with prominent frontal eminences, a condition sometimes mistaken for hydrocephalus.

The chemical analysis of rickety bones shows a marked diminution in the calcareous salts, which may be as low as from 25 to 35 per cent.

The liver and spleen are usually enlarged, and sometimes the mesenteric glands. As Gee suggests, these conditions probably result from the general state of the health associated with rickets. It is interesting to note that Beneke describes a relative increase in the size of the arteries in rickets.

Kassowitz, who may be considered the leading authority on the anatomy of rickets, regards the hyperæmia of the periosteum, the marrow, the cartilage, and of the bone itself as the primary lesion, out of which all the others develop. This disturbs the normal development of the growing bone, and excites changes in the bone already formed. The cartilage cells in consequence proliferate, the matrix is softer, and the bone which is

formed from this unhealthy cartilage is lacking in firmness and solidity. In the bone already formed this excessive vascularity favors the normal processes of absorption, so that the relation between removal and deposition is disturbed, absorption taking place more rapidly. The new material is poor in lime salts. Kassowitz seems to have proved experimentally that hyperæmia of bone results in defective deposition of lime salts. Barlow and Bury * have given an elaborate analysis of the changes described by this author. It is interesting to note that Glisson attributed rickets to disturbed nutrition by arterial blood, and believed the changes in the long bones to be due to excessive vascularity.

Symptoms.—The disease comes on insidiously about the period of dentition, before the child begins to walk. In many cases digestive disturbances precede the appearance of the characteristic lesions, and the nutrition of the child is markedly impaired. There is usually slight fever, the child is irritable and restless, and sleeps badly. If the child has already walked, it shows a marked disinclination to do so, and seems feeble and unsteady in its gait. Sir William Jenner has called attention to three general symptoms which are present in many cases of rickets. There is first a diffuse soreness of the body, so that the child cries when an attempt is made to move it, and prefers to keep perfectly still. This tenderness is often a marked and suggestive symptom. Associated with this are slight fever and a tendency at night to throw off the bedclothes. This may be partly due to the fact that the general sensitiveness is such that even their weight may be distressing. And, third, there is such profuse sweating, particularly about the head and neck, that in the morning the pillow is found soaked with perspiration.

The tissues become soft and flabby; the skin is pale; and from a healthy, plump condition, the child becomes puny and feeble. It is in this stage of the disease that we sometimes find such a degree of disability in the muscles, particularly of the legs, that paralysis may be suspected. This so-called pseudo-paresis of rickets results in part from the flabby, weak condition of the legs and in part from the pain associated with the movements. Such cases are by no means uncommon, but they are readily distinguished from infantile paralysis. Coincident with, or following closely upon, the general symptoms the characteristic skeletal lesions are observed. Among the first of these to appear are the changes in the ribs, at the junction of the bone with the cartilage, forming the so-called rickety rosary. When the child is thin these nodules may be distinctly seen, and in any case can be easily made out by touch. They very rarely appear before the third month. They may increase in size up to the second year, and are rarely seen after the fifth year. The thorax undergoes important changes. Just outside the junction of the cartilages with the ribs there is an oblique, shallow depression extending downward and out-

* Cyclopædia of the Diseases of Children, vol. ii.