OSTEOTOMY.

CHAPTER I.

THE RELATION BETWEEN RICKETS AND CERTAIN DEFORMITIES OF THE LOWER LIMBS.

MANY of the deformities of the lower limbs whose treatment is considered in this volume have their origin in rickets. It has therefore been thought best to devote a short chapter to this disease, and to point out its connection with the subject under consideration.

Those who are connected with our large dispensaries are well aware of the prevalence of rickets among the applicants for medical aid. Whether it is as common in this country as in certain parts of Europe is doubtful. It is not alone confined to the children of the middle and lower classes, but is met with among the offspring of the wealthy, not perhaps in its more advanced stages, yet sufficiently well marked to be easily recognized if its manifestations are carefully looked for. It is seen among children who have been brought up in the country as well as those who live in crowded cities, but to a much less extent. It is a disease that merits the careful attention not only of the surgeon, but of the general prac-

titioner, in order that its results, in deformities of the long bones and changes in the shape of the chest and pelvis, may be prevented. Rickets is not a disease of the bone alone, but is a constitutional affection, attacking the osseous structures in common with every other tissue of the body. It is essentially a disease of malnutrition. It may be congenital, but it usually first manifests itself in children from six months to three years of age. Bad air, improper food, and scanty clothing are its most prolific causes. Any child may become rickety, no matter how healthy it may have been at birth, if placed under any condition that interferes with its assimilative powers. It may be laid down as a rule that a healthy child, fed on good mother's milk, will never develop this disease. It is equally true that not every child who suffers from malnutrition will become rickety. Its beginning is insidious, with the ordinary symptoms of improper digestion. The little patient may be plump, but its muscles are flabby and its complexion pale and unhealthy; large veins are distinctly seen through the pasty-looking skin. The bowels may be loose or confined, more often capricious, a day or two relaxed, then followed by a period of constipation; the stools are white, curdy-looking, and extremely offensive; the food is often passed through the alimentary canal undigested. Accompanying this derangement of the digestive apparatus there is profuse sweating of the head, neck, and upper part of the chest, worse at night. The moisture will be seen standing in large drops upon the forehead, and often runs down the face, and at night the pillow is drenched with it. While the head and neck are thus

bathed in perspiration the abdomen and lower limbs are dry and hot. Another symptom is the desire of the child to keep cool at night. It constantly throws the clothes off from its feet and limbs, no matter how cold the temperature may be. The child soon loses its activity, and seems only happy when left alone. It will sit for hours almost motionless, is petulant, and cries on being moved. The desire to be let alone is due to tenderness, more or less marked, of the bones, so that any pressure on them is painful to the little one, and it dislikes to be handled.

Dentition in those affected with rickets is usually retarded, or, if the teeth have made their appearance, they soon become black and fall out, or are early attacked with caries. Jenner states that if the ninth month passes without the appearance of a tooth, the cause should be carefully inquired into, and will almost always be found in rickets. According to Eustace Smith, the symptoms of rickets seldom appear before the fourth, and usually not until the seventh month. Cases, however, occur in which the advent of the disease is delayed much longer. Jenner mentions a girl of nine years of age in whom the symptoms of rickets had just commenced.

Enlargement of the spleen, liver, and of the lymphatic glands in different portions of the body is a common accompaniment of this disorder. In some cases the patient is reduced to a skeleton, while in others, as mentioned before, it retains its plumpness.

While the symptoms mentioned above are manifesting themselves, changes are taking place in the bones, perhaps not more profound, yet more noticeable than in any other structure of the body. One

of the earliest of these is a beaded appearance at the sterno-costal junction and an enlargement of the epiphyses, especially those at the wrist-joint. If a child affected with rickets be carefully examined, a line of nodules will be felt, and often seen, marking the point of junction between the ribs and the sternum. This condition has been noticed in children suffering from rickets six weeks after birth. When this beading of the ribs is found, it is a positive proof of the existence of this disease. There are also certain alterations in the occipital bone, often found at a very early stage of the disease in young children. Macnamara states that they are almost as constant a condition in this disease as the abnormalities of the ribs, but they are not as easily detected. If the occipital bone be carefully examined in young infants affected with this disease, there will often be felt several small, round, or oval soft spots, situated within the sutural margins of the occipital and parietal bones. These spots are unossified portions of the structures from which the bone is produced. The number of these spots varies considerably. The occipital bone is often remarkably thin in cases of this disease, and the head has a peculiar elongated appearance, while the face in marked cases remains small. (Macnamara.)

While these changes are going on, others of as marked a character are taking place in the ends and shaft of the long bones. The epiphyses become enlarged, and the shaft softened so that it is often bent, and the epiphyses may become twisted, according to the intensity of the disease and the force acting on the plastic bone. The condition of the bones varies with the stage and the intensity of the disease, and

whether the brunt of the changes falls upon the epiphyses or the diaphyses. At first the bones may be soft, so that they can be bent like cartilage; later they become very hard and deformed.

The pathological changes taking place in the bones are well described by Macnamara in his work "On Disease of the Bones and Joints." He says: "If a rickety bone be divided longitudinally during the first stage of rickets, the medulla filling the central canal and cancellated tissue will be found to be of a crimson color and jelly-like consistency, this soft medulla being especially abundant at the line of the junction between the diaphysis and epiphysis. The medulla of a child suffering from the first stage of rickets consists of a vast number of round cells, compound cells, and fat; the adenoid tissue and vessels are normal in appearance; in fact, *the elements characteristic of healthy medulla* are present in the bones of infants suffering from this disease, but there is an imperfect formation of the calcareous skeleton of the bones, and in its place we find an excess of medullary tissue. . . . The malady, so far as the bones are concerned, depends on the deficiency of earthy matter contained in this hyaline matrix. If a section made through the epiphysis into the diaphysis be examined during the first stage of the disease, there will be found at the line of ossification numerous villous-like processes of medulla, projecting, as it were, from the diaphysis into the epiphyseal cartilage. These processes, however, are not formed from the growth of the medulla of the diaphysis into the cartilage, but from a transformation of the cartilage-cells of

the epiphysis into medullary tissue at the normal line of ossification. The mass of the descendants of the cartilage-cell forming the processes referred to are incapable of producing healthy bone, in consequence of a deficiency of the bone-earth. So long as an infant is insufficiently supplied with, or is incapable of assimilating, elements necessary to the calcification of the cartilage-cells, it is impossible that healthy bone can be produced."

The changes in the shaft of the long bones, according to Virchow, "consist in the non-solidification of the fresh layers as they are formed, while the old layers of bone are consumed by normally progressive formation of medullary cavities. The periosteum is thickened and more adherent to the bone. Medullary spaces and vessels are met with where normally and properly not a single medullary cell and scarcely a single vessel ought to be found."

During the height of the disease, when the changes in the bones are marked, they can be bent by the least possible force, and their spongy portions may be easily cut with a knife. In rickets the ligaments are often altered in their structure, so that they may become easily elongated, and thus permit lateral motion in joints in which normally it does not exist. However profound the changes in the bones may have been, as soon as the child begins to masticate and is able to assimilate proper nutriment, the vast number of bone-cells rapidly take up the earthy salts from the blood, and very dense bone is speedily produced, so that in a short time the bone may become very hard. Again, the process of sclerosis may be much slower, depending

upon the power of the child to assimilate, and the quality of the food furnished. Rachitic changes in the osseous structure do not always take place in all the bones of the skeleton in equal degree, nor in every portion of an individual bone. Thus softening may be more marked at the epiphysis while in the shaft it is slight, or the shaft may show more advanced changes than the articular ends. The enlargement of the epiphyses is not an index of the changes in the shaft. The bone of one limb may be quite soft while that of the other is hard. I have met with this condition quite frequently in operating; one tibia has been found very easy to divide, while in the other section was difficult. The fibula has been found to be much harder than the tibia in the same limb.

Perhaps the existence of different deformities may be due in part to this irregularity in the portion of the bone on which the intensity of the rachitic process falls.

All writers on this disease divide it into a preliminary stage, one of softening and one of sclerosis. It is in the latter that the bone-cells take up from the blood the earthy matter that gives bone its hardness. It will be evident that this stage can not begin until the child is able to digest and assimilate appropriate food. No rule can be laid down as to the length of time that the soft stage of rickets lasts, nor the rapidity with which the hardening may advance. Each case must be judged by itself. The general health, ruddy appearance, and firm condition of the muscles are the best guide. It may take many years in children of low vital powers for

the bones to become firm, while in others it may be accomplished in a few months.

There is a condition described by some observers (Barlow, Page[1]), as "acute rickets." It occurs in children under two years of age. There is profuse sweating about the head, changes in the epiphyses, and other symptoms usually found in rickets, together with swelling and great tenderness of the lower extremities, due, it was supposed, to an effusion of blood under the periosteum and between the deeper muscular layers. Barlow considers it a combination of rickets and scurvy. By change of diet, fresh air, and compression of the limbs, recovery may take place rapidly.

The age beyond which rickets is not developed is uncertain, but probably in the vast majority of cases not after the fifth year. Some writers consider that this disease may be developed between the twelfth and twentieth years; that is, during the period of rapid growth. Macewen is an advocate of this late appearance of rickets. But it is denied by the majority of writers. In the chapter on genu valgum the cases of Macewen are given. I have never met with a case, and I think if they ever occur it must be exceptional. Deformities about the knee-joint are sometimes developed in persons from twelve to twenty years of age, but I think that their cause can be explained without attributing them to rickets. In the cases that I have had the opportunity to examine there was absolutely no symptom of rickets except the bending of the bone at the epiphyseal line. Most of the deformi-

[1] "Brit. Med. Jour.," March 31, 1883, p. 619.

ties of the lower limb are developed during the period of rapid growth. They are met with first in infantile life, when all the nutritive processes are at their height, and the child rapidly increases in weight and stature. This period, as a rule, does not extend beyond the seventh year. Then comes a time, extending from the seventh to the twelfth year, during which growth is much slower and deformities are seldom developed. From the twelfth to the twentieth year is another period of growth and development during which the long bones rapidly increase in length by the deposit of osseous material at their extremities, and which is finally completed by the consolidation of the epiphyses and diaphyses. In this period, again, certain deformities, especially about the knee-joint, are developed in those who are compelled to labor hard and undergo fatigue.

During the first period bending of the shaft of the long bones, with the consequent deformities, are common, while in the last period deformities having their origin near the joints are met with, and curvatures of the shaft of the bones are seldom if ever seen.

The connection between rickets and deformities of the bone is one of cause and effect. I do not think that the muscles exert an active influence, but that position and weight are the cause of the abnormal shape of the bones.

In this very imperfect review of the symptoms of rickets, as it affects the bones of the extremities, nothing new is claimed, the object being simply to call attention to this most prolific cause of deformities.

While the bones are soft, any abnormal change in

their shape can, and should, be corrected by appropriate apparatus. But after sclerosis has taken place, or even is well advanced, orthopedic appliances will not correct. I am not a believer in the spontaneous cure of bending of the long bones. We often hear the advice given to mothers by members of the profession not to submit these cases to treatment; that the child will "outgrow" the malposition; and I wish to enter a protest against such advice, as it will only lead to disappointment.

CHAPTER II.

OSTEOTOMY.

"OSTEOTOMY," says Macewen, "in its broadest acceptation, may be defined as a section of bone. It has, however, been regarded in a much more restricted sense, the term being applied to such divisions of bone as have been proposed and undertaken for the relief of deformity, for the rectification of badly united fractures, and for the straightening of limbs affected with osseous anchylosis, which are fixed in a bad position." ("Osteotomy," p. 37.)

Section of the long bones for deformity had been proposed by many early writers on surgery, yet it does not appear to have been put in practice until 1815, when Le Mercier made a section of the tibia with a saw for a badly united fracture of that bone; and in the following year Wasserführ practiced the same operation upon the femur. Barton, in 1826, performed an osteotomy just below the trochanter major for anchylosis with flexion of the thigh, through an open wound, the division being made with the saw. In 1834 Clémot removed a wedge-shaped piece of bone for the correction of an angular deformity of the femur. Portal, Ashley Cooper, Warren, of Boston, and others, performed similar operations. All sections of bones prior to 1852 were performed through