osteo-arthritis, and in our case, as in the vast majority of genuine cases reported, the focus of infection is in the lungs.

PATHOLOGY

In 1906, Alexander collected seventy-seven instances from the literature which he regarded as genuine cases of this disease, and he reviews the pathology of all the cases in which complete necropsies were performed (sixteen cases).

We quote Alexander's findings almost verbatim from Osler, and the description fits our case very perfectly:

Symmetrical deposits of new subperiosteal bone on the shafts of the long bones. Most frequently affected are the lower ends of the radius and ulna, the metacarpals and the first two rows of the phalanges; more rarely the lower end of the humerus and the upper ends of the radius and ulna. The new bone begins abruptly from 4 to 5 inches above the wrist joint and forms a sheath covering the lower ends as far as the epiphyseal line. The circumference of the bone is about equally affected, the thickest deposit being at the junction of the shaft with the lower extremity of the bone. The carpal bones are not affected, but the metacarpals are ensheathed and appear uniformly enlarged. The first two rows of the phalanges are similarly affected but less than the metacarpals. The terminal phalanges are apparently unaffected.

Fig. 9.—A sheath of new bone may be seen surrounding the shafts of the tibia and fibula. The cortex and medullary canal are apparently normal (March, 1919).

Similar bones of the lower extremity are affected, especially the lower tibia, which may be thickened by a layer of new bone one quarter of an inch thick. The phalanges of the toes are less affected than those of the fingers.

Deposits have been found on the iliac crests, on the clavicles and on the anterior surface of the patellae.

The symmetrical distribution is truly remarkable.

The cortex of the bone may show sclerosis and thickening, with diminution in the size of the medullary canal. The periosteum is very vascular and thick, and the main nutrient canals are large and many additional vessels enter the compact layer.

The bone changes are due to a chronic inflammation, causing new bone formation, also to atrophy and rarefaction and, to a much less degree, to osteophyte building.

The long bones do not bend. The skull is little, if any, affected.

In many cases there is excess of fluid in the joints, wrists, ankles, knees and fingers. In these affected joints the synovial membrane shows inflammatory changes, but there are no cartilaginous changes or lipping, no eburnation nor osteophyte building.

The bone changes are only part of the process, for there is much thickening of connective tissue, especially in the vicinity of the wrists, ankles and fingers.

Barker describes the affair as a slowly ossifying periostitis and osteitis, but he states that the joints are free.

Buck³ says that there is "an outside layer of dense, newly formed periosteal bone all along the shaft; this layer is striated and does not completely obscure the old cortical line. In pronounced cases all the bones in the body may be involved; the flat bones are thickened and there is marked kyphosis."

Fraenkel adds that it is "a true hypertrophy in consequence of periostitis, with new bone formation and at the same time a rarefaction of osseous tissue with dilatation of the natural interstices as well as increase of the medullary substance."

I make no attempt to harmonize the little discrepancies in the descriptions quoted above.

The thyroid and the pituitary glands are not altered.

TREATMENT

The treatment for the pains in the bones and joints is symptomatic. The only treatment which will logically have any effect on the progress of this rare disease must be directed toward the cure or improvement of the primary focus in the lungs.

CONCLUSIONS

The history, the clinical findings and the roentgenologic findings in our case all point quite conclusively to the diagnosis of hypertrophic pulmonary osteoarthropathy, by which we mean a subacute infectious osteo-arthritis, secondary to a septic focus in the lungs. However, we must acknowledge that the roentgenogram of the skull shows an abnormally small sella turcica, and the possibility of some change in the pituitary must be admitted.

Is it possible that our case, like the first case reported by Marie, may later develop into an irregular type of acromegaly?

CONGENITAL BILATERAL STRICTURE OF THE URETER*

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If we may judge from the literature on the subject, congenital stricture of the ureter is not rare; but a *bilateral* congenital stricture of the ureter is a rare defect, only eight cases, exclusive of the one here recorded, having been described. From the few cases recorded it would appear that these strictures are most often discovered at the extremes of life, most of them having become evident before the age of 5 years. Two cases have occurred in adults, one at the age of 25 years and the other at the age of 62.

No apparent reason for this congenital anomaly presents itself in studying the development of the urinary system. The ureters are primary derivatives of the bladder, growing cephalad to meet the metanephric tissue. Their lumina appear either as a result of central absorption of the epithelial outgrowth or are present primarily in the process of their evagination from the bladder¹. The failure of these processes to be completed would give rise to the obliteration of the lumen. Another explanation rests on an anatomic characteristic of the ureter in the embryo, namely, the presence of valves. In the case reported by Watson and Cun-

3. Bryant and Buck: American Practice of Surgery 1 and 3: 1906.

* From the Pathological Laboratory, Lincoln Hospital and Home, G. L. Rohdenberg, M.D., Director.

1. Huntington: Harvey Lectures, New York, 1906-1907, p. 222.

ningham,² it is probable that the valves became adherent at their margins or lost their elasticity and so caused the narrowing of the lumen.

In the ureter there are three sites of predilection where the lumen may be obliterated: at the junction of the ureter with the renal pelvis, at the brim of the bony pelvis, and at the vesical end. When the obstruction occurs at the vesical end, obliteration may occur just outside the bladder, or in the muscular coat, or at the bladder opening of the ureter. With obstruction just outside the bladder, there is a cystic dilatation extending up a variable distance from the bladder while the inner surface of the viscus is negative. With the stricture in the middle coat, there is no obvious abnormality, except perhaps a dimpling or a moderate pouting at the site of the ureteral opening. When the obliteration occurs at the ureteral orifice, there is a cystic outpouching from the ureteral opening which may vary daily in size, as in Buerger's case,³ or enlarge so that it may present at the external urethral meatus as in the cases described by Bostrom.⁴

As is to be expected, the narrowing of the lumen produces a back pressure which results in structural and physiologic changes in the tract behind the stricture. The extent of these changes is dependent on the degree of the atresia. If obstruction is incomplete, there is a dilatation of the lumen of the ureter accompanied by a hypertrophy of the wall. The renal pelvis becomes dilated, and atrophy of the pyramids and renal parenchyma follows. If the atresia is complete, the result is either an atrophy of disuse or a hydronephrosis, with or without a pyogenic infection. The fluid in the hydronephrotic cases, as Sollmann, Williams and Briggs⁵ have shown, possesses the characteristics of a transudate and not that of urine.

The cases occurring in the literature are, briefly abstracted, as follows:

REPORT OF CASES

CASE 1.—A man, aged 25, was operated on at the age of 14 for a right-sided hydronephrosis and at the age of 25 for a left-sided pyonephrosis (Wipple⁶).

CASE 2.—A bilateral congenital atresia occurred in an 8 month old fetus at the vesical opening of the ureters with beginning double hydronephrosis (Welz⁷).

CASE 3.—The age of the patient is unknown, but he presented a marked degree of bilateral hydronephrosis due to unyielding ureteral valves (Watson and Cunningham⁸).

CASE 4.—A boy who died thirty hours after birth presented a right ureter obliterated 5 cm. from the kidney and the left ureter obliterated 2 cm. above the bladder (Mutach⁹).

CASE 5.—A man, aged 62, presented a bilateral stricture at the vesical openings indicated by minute depressions at the tip of protrusions appearing at the normal ureteral openings in the bladder. This was probably the type in which the stricture was situated in the ureter as it passed through the muscular coat of the bladder (Burchard⁹).

CASE 6.—A boy who died one hour after birth from respiratory difficulties presented the ureter patent on the right side for a distance of 3 cm. from the bladder, the left ureter being patent for 2 cm. This author states that similar cases have been reported by Hoenes and Ahlfeld (Dycherhoff¹⁰).

AUTHOR'S CASE

A boy, aged 1 month, was brought to the hospital with the statement that he had been restless and vomiting since birth. Birth had been normal. Physical examination revealed a marked state of malnutrition. The abdomen was much distended, the superficial veins being prominent on both sides. The child was moderately jaundiced. The temperature varied from 99 to 100 F. After three days in the hospital the infant died.

At the necropsy, aside from the jaundice of the skin and organs, only two abnormalities were found: the first of these was that of the gallbladder. The viscus was distended with a colorless mucoid substance, and the cystic duct was completely angulated on itself so that the two limbs were parallel. They were held in this position by a vascular band containing the cystic artery. On cutting this band the obstruction was released and the contents of the gallbladder could be expressed into the duodenum.

The second abnormality was in the urinary system: Both kidneys were enlarged, the pelvis and calices were dilated.

Congenital bilateral stenosis of ureter followed by hydronephrosis. Dilatation of calices, abscess pockets in kidney and diameter of ureter, as shown in cross section at lower pole of one kidney, should be noted.

and the pyramids were flattened. The left kidney presented several abscess pockets, the largest of which was 5 cm. in diameter. Both ureters were markedly dilated, measuring almost 1 cm. in diameter, the ureteral walls being 0.25 cm. thick. A probe could be passed through their entire course except at the vesical end where they permitted the passage of only a horsehair. The urethra was patent and the bladder showed no abnormality. The accompanying illustration shows the condition better than words can describe it.

10. Dycherhoff: Inaug. Diss., Kiel, 1900.

Institution to Promote Cleanliness and Hygiene Among Schoolchildren.—The women of the Comité femenino de Higiene Popular of Spain have fitted up a building at Madrid equipped with all that is necessary for baths, etc., for schoolchildren, especially to "hygienize" the children as they are to be sent out of town to the seashore sanatoriums, school colonies and for other outings. The building has been placed at the disposal of the city authorities, and the work of caring for the children is done by the women of the league or the public health personnel. The children have their hair cut, the head disinfected, the mouth put in order and the teeth attended to by a woman dentist. There are douches and other bathing facilities and the institution is open for work every week-day, from 8 a. m. to 8 p. m., everything gratuitous. It is hoped to install more of these Casas de Higiene Benéfica Infantil in other crowded parts of the city.

2. Watson and Cunningham, cited by Eisendrath: Surg., Gynec. & Obst. 12: 533 (June) 1911.

3. Buerger, cited by Bottomley, Ann. Surg. 52: 597 (Nov.) 1910.

4. Bostrom, cited by Schwarz: Beitr. z. klin. Chir. 15: 159 (Nov.) 1895.

5. Sollman, Williams and Briggs: J. Exper. M. 9: 71 (Jan.) 1907.

6. Wipple, cited by Welz: Inaug. diss., München, 1903.

7. Welz: Inaug. Diss., München, 1903.

8. Mutach, cited by Welz (Footnote 7).

9. Burchard: Centralbl. f. allg. Path. u. Path. Anat. 7: 129 (Feb.) 1896.