TWO CASES OF SUPERNUMERARY URETERS OPENING INTRAVESICALLY*

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IN 1918 Mertz (1) extensively reviewed the subject of multiple ureters, having collected 300 cases from the literature and contributed 16 additional cases. Since that time Judd (2) has reported a case opening extravasically, and Wilhelmj (3) a case opening intravesically.

In 278 of the 318 cases the type of anomaly was stated, there being 63 of unilateral partial duplication, 9 of bilateral partial duplication, 141 of unilateral complete duplication, 50 of bilateral complete duplication, and 14 of bilateral duplication, being partial on one side and complete on the other. I wish to add two cases to the literature, one of unilateral complete duplication, and the other of bilateral duplication, complete on one side and partial on the other.

EMBRYOLOGY

About the end of the first, or the beginning of the second month of embryonic life, a diverticulum arises from the posterior wall of the lower end of the Wolffian duct, close to where the latter opens into the cloaca. This diverticulum or renal bud grows dorsalward and forward along the posterior abdominal wall, where its blind extremity expands and divides normally into two branches, which grow into the blastoma of the kidney. Each branch then divides, and by continued subdivision forms the straight and ureteric tubules, and calyces of the kidney. The proximal portion of the diverticulum becomes the ureter.

The kidney reaches its normal height about the end of the second month, and then receives its blood supply, the vascularization coming directly from the aorta, and occasionally from the suprarenal artery. The vessels seem to choose the nearest route to the hilum, the branch from the suprarenal sometimes entering the upper pole.

The ureter grows away from the Wolffian duct, and empties higher and more laterally. It assumes its relatively normal position in the same stage in which the kidney reaches its normal height, or about the end of the second month. The two processes are, however, quite distinct, the change in the position of the kidney being due to the development of the distal part of the bud and its surroundings, while the change in the position of the ureter is related to the growth of the lower end of the Wolffian duct.

Pohlman (4) has tabulated the steps in the development of the tract as follows:

1. The renal bud arises dorsally on the Wolffian duct after the duct has reached the cloaca, and at a short distance from its entrance.

2. The two buds grow dorsally, lying close together, each being capped with a mass of renal mesenchyme.

3. Each bud divides into an upper and a lower sprout at some distance from the Wolffian ducts.

4. The kidney wanders up from a position in front of the second sacral vertebra, rotates at the mid-lumbar region, and finally reaches its normal height about the end of the second month.

5. It becomes vascularized after it has reached its normal height.

6. The ureter changes its position on the Wolffian duct from dorsal to lateral, and comes to empty distinct from it.

7. The ureter loses its relation to the duct entirely, and opens higher and more laterally.

Should the renal bud divide too early, or the division extend into the ureter segment, that splitting found in the ureter itself would remain a permanent one, and any variation might exist as a result, from an exaggeration of the usual upper and lower pelves to an incomplete reduplication of the ureter. An incomplete double ureter may be defined as two ureters with a common orifice.

Should the splitting of the renal bud be so complete that it is affected by the shortening of the segment of the Wolffian duct lying between it and the cloaca, it is possible for each division to ac-
quire a distinct orifice in the duct. This would result in complete double ureter.

Pohlman has stated rules for double ureters, which hold true with relatively few exceptions. They are as follows:

In incomplete double ureters the ureter from the upper pelvis lies ventral to the one from the lower pelvis, and there is a common orifice into the bladder. In complete double ureters the proximal relations are the same, but as they approach the bladder, the ureter from the lower pelvis swings lateral to the one from the upper pelvis, and empties into the bladder lateral and slightly above it.

Case Reports

Case 1.—E. H., aged four months, admitted with a history of vomiting after feedings since birth; vomiting sometimes projectile in character.

The mother had observed that the urine was always scanty in amount. Weight on admission 7 pounds, 6 ounces.

Physical examination revealed a poorly developed, very poorly nourished infant in a state of collapse. The skin was markedly dehydrated, the abdomen lax and scaphoid with visible peristaltic waves; no pyloric tumour felt. The anterior fontanelle measured 3 x 4 fingers, with well-marked cranial tubers, and slight epiphyseal enlargement. The external genitalia were normal.

Laboratory report: Urinalysis, acid in reaction, negative for albumin and sugar. Microscopic examination showed enormous numbers of pus cells, and many long chains of streptococci in an unstained smear. R.B.C. 6,000,000, W.B.C. 16,000, with 30 per cent. polymorphonuclears, and 70 per cent. lymphocytes. Haemoglobin 100 per cent., temperature 98.3° F. Diagnosis: 1, pyelitis; 2, rickets.

The infant was transfused, and given daily interstitial administration of 5 per cent. glucose solution, the pyelitis being treated with potassium citrate, grains 100 per diem. He succumbed four days following admission.

At autopsy both kidneys were in their normal positions. The right kidney showed remnants of foetal lobulation. Its greatest length was 5.2 centimetres, and greatest diameter 2.3 centimetres. On section the cut surface appeared to be normal. Two ureters emerged from the hilus, about 1 centimetre apart, from two distinct pelves, and followed a normal general course to the bladder, the ureter from the cephalic pole, 10.5 centimetres in length, lying anterior to the ureter from the caudal pole, 9.5 centimetres in length, and uniting with its fellow about 3 centimetres from the bladder. There was a single opening into the bladder in a normal position in relation to the trigone. Both ureters were slightly dilated, and contained some turbid urine.

The left kidney also showed remnants of foetal lobulation, but to a greater degree than the right kidney. Its greatest length was 6.4 centimetres, and its greatest diameter 3.6 centimetres. The amount of kidney tissue was very small, being only a few millimetres in thickness. Two distinct pelves were present, as on the right side, but here they were quite dilated, the lower to a much greater degree than the upper. Both ureters were markedly dilated and tortuous, and were filled with a yellowish turbid fluid. The ureter from the caudal pole, 19.0 centimetres in length, lay posterior to the ureter from the cephalic pole, and opened by an apparently normal orifice into the bladder, also in a normal position in the trigone.

The ureter from the upper pelvis, however, 21 centimetres in length, while at first anterior to its fellow, became more medial as it neared the
bladder, and penetrated the bladder wall at a point about 7 millimetres lower and more medial than the other ureter. It presented in the bladder cavity as a bean-shaped, cystiform swelling about 1 centimetre in diameter, with an opening in the form of a vertical slit, about 2 millimetres in length, situated on the lower part of the posterior wall. When the cyst was distended with fluid from above, it was observed that the slit-like orifice lay against the posterior bladder wall, and that the cyst partially blocked the opening of the other ureter on the same side. It also lay over the urethral orifice at the apex of the trigone.

The bladder contained about 15 cubic centimetres of yellowish turbid fluid, similar to that found in the left renal pelvis and ureters, and the bladder wall showed some congestion of the mucous membrane.

Haematoxylin and eosin sections of the right kidney revealed nothing abnormal. Sections of the left kidney showed both the cortex and medulla to be greatly thinned out, with a considerable increase in connective tissue, and infiltration with small round cells immediately underlying the pelvic epithelium. In this region, remnants of tubules could be made out. Some of the tubules were considerably dilated, being lined with flattened epithelium. A few of the glomeruli showed dilatation of the capsular space with fluid, the tuft being compressed to one side. The majority of the glomeruli, however, were normal. Diagnosis: hydrenephrosis, with beginning pyonephrosis.

Direct smears of the urine from the bladder showed large numbers of pus cells, and Gram positive cocci in chains; also a few Gram negative bacilli. On culture the predominating organism was a non-haemolytic streptococcus, with some colon bacilli.

Case 2.—F. W., male, aged 3½ months, with a complaint on admission of frequent green stools with mucus for two weeks. The infant had vomited considerably during the week previous to admission. The vomiting and diarrhoea were sequels to a bad feeding history with artificial foods high in carbohydrates, which need not be gone into detail here.

Physical examination revealed a poorly developed, badly nourished infant in a very toxic state. The eyes were sunken and the skin was markedly dehydrated. Chest and abdomen were negative.

Laboratory report: Urinalysis, acid in reaction, a trace of albumen, negative for sugar. Microscopic examination showed a few hyaline casts. R.B.C. 4,200,000, W.B.C. 28,500, with 40 per cent. polymorphonuclears and 60 per cent lymphocytes. Haemoglobin 70 per cent., temperature 98.0°F. Diagnosis—1, Acute intestinal intoxication; 2, Fermentative diarrhoea.

![Case 2.—Probes are shown placed in the orifices of the two right ureters, of the left ureter, and in the urethral orifice.](image)

The feedings were corrected, the baby was transfused, and 5 per cent. glucose solution was administered daily, both intravenously and interstitially. The child seemed to improve, but developed double otitis media ten days after admission, following which his general condition became worse, and he suffered a collapse and died four days later. Five days before death the urine was negative for albumen, and showed no microscopic changes.

At autopsy both kidneys were found to occupy their normal positions. The left kidney, 5.3 centimetres in length, and 2.8 centimetres in diameter, showed nothing abnormal outside of being somewhat congested. From a single pelvis arose a single ureter, 9.5 centimetres in length, which pursued a normal course to the bladder, and emptied by a normal orifice in the trigone.

The right kidney, 5.8 centimetres in length and 3.0 centimetres in diameter, also showed a slight
amount of congestion. Two distinct pelves gave rise to two ureters about 1 centimetre apart at their origins. The ureter from the cephalic pole, 9.5 centimetres in length, lay anterior to its fellow, 8.5 centimetres in length, and after following a normal course to the bladder opened by an orifice about 1 millimetre medial and anterior to the orifice of the ureter from the caudal pole. Both ureters were patent throughout their course and showed no apparent dilatation.

The bladder contained about 15 cubic centimetres of clear, amber coloured urine, and its walls showed nothing abnormal.

Hematoxylin and eosin sections of the kidneys showed only a slight degree of nephrosis. No glomerular involvement was found.

**Remarks**

Thus it can be seen that Pohlman's rule applies also to these two cases, that for incomplete double ureters to the right ureters in Case 1, and that for complete double ureters to the left ureters in Case 1, and the right ureters in Case 2.

Bottomley (5) mentions seventeen cases of ureteral obstruction with cystiform protrusions of the ureter into the bladder, seven of which were associated with multiple ureters, while in thirteen of the cases of ureteral duplication collected by Mertz, a similar condition was present. Johnson's (6) case is specially interesting in that the cyst presented at the vulva. He writes as follows: "The vesical orifice of the right ureter is normal in size and position. From the corresponding point on the left side, a raised column 5 millimetres across, covered by the mucous membrane of the bladder, extends downwards, gradually increasing in diameter, and ending in a bulbous enlargement which, with the parts in their natural position, occupied the whole length of the dilated urethral canal, and protruded externally between the labia. On its posterior surface the bulbous enlargement is attached to the floor of the urethra nearly as far as its orifice. On its right side the enlargement presents a long rent, freely opening the hollow interior, from which the probe can be passed up into the ureter."

In Bottomley's case there was a history of 2½ months' illness from vomiting, diarrhea and loss of weight as symptoms in a 5½ months infant. His findings were similar to those in Case 1, with the exceptions that in his case the ureters arose from a common pelvis, and the cyst had no opening either into the bladder or into the other ureter, while here there were two distinct pelves and the cyst possessed an opening into the bladder.

According to Mertz there is no pathological process of the kidney, ureter or bladder dependent directly upon the existence of a double ureter nor does he conclude from his study on the subject that there is one type of disease more prevalent in patients with double ureters than in those with no such anomaly, although he quotes one writer as saying that kidneys with double pelves and ureters are frequently affected with calculi. Case 2 demonstrates that the anomaly can exist without giving rise to disease. The hydronephrosis and pyonephrosis present in Case 1 did not depend directly on the existence of double ureters, for such processes are present in a much greater number of patients possessing single ureters.

In the 276 cases of Mertz, disease was stated to be present in 85, or 30 per cent. In 76 of the diseased cases the pathological condition was stated, and of these there were 31 of hydronephrosis and 18 of pyonephrosis.

In Case 1 the obstruction resulting in the hydronephrosis and secondary pyonephrosis may have been due to either of two factors. It may have been caused by pressure of one ureter against the other when they crossed, for it has been observed that stasis is produced in such a case, especially when the ureters are closely bound together. Or it may have been brought about by the cystiform swelling acting as a valve when distended. This was tried out experimentally, it being found that when the ureter with the cystoform ending was filled with fluid at the pelvis with a syringe, no great distension took place at first, the fluid passing readily into the cyst. But when this was filled the ureter dilated, and more pressure was required to force the fluid from the orifice of the cyst. When the other ureter was filled with fluid, while the first was still distended, it was found that resistance to outflow from its orifice was also present. On the right side, fluid passed through both ureters with no difficulty.

Thus the cystiform swelling when distended with urine was responsible for the stasis in both ureters. Its own orifice was pressed against the posterior bladder wall, thus obstructing any outflow from its interior, and also it covered up the opening of the other ureter and blocked the urethral orifice. The latter would result in distension of the bladder, with increased intravesical pressure, which in turn would cause the cyst to increase the existing blockage of the left ureters, and would also cause some obstruction in any
outflow from the right side. This latter explains the cause of the slight dilatation of the right ureters.

These two cases were not suspected until autopsy, but as multiple ureters produce no pathological processes directly dependent on their existence, they produce no symptoms and hence cannot be diagnosed by this means. The single ureter with an ectopic orifice produces the same symptoms as an abberant supernumerary ureter with a similar orifice. Cystoscopical examination might have revealed the condition in these cases, but as yet it is a procedure not carried out very extensively with infants.

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**References**


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**Prevention of Simple Goitre in Man.**—The ultimate cause of simple goitre is totally unknown, notwithstanding a relatively large amount of study. The immediate cause is a lack of iodine. The enlargement, therefore, is a symptom and may result from any factor which increases the iodine needs of the organism, as in certain types of infection, or which interferes with the normal utilization of iodine; or it may result from actual experimental deprivation of iodine. After consideration of all the various substances, agents and theories that have been put forward as having a role in the etiology of goitre, David Marine, New York, and O. P. Kimball, Cleveland (Jour. A. M. A., Oct. 1, 1921) state that at present we must fall back on the view that thyroid hyperplasia (goitre) is a compensatory reaction arising in the course of a metabolic disturbance and immediately depending on a relative or an absolute deficiency of iodine. No accomplishment in preventive medicine has a firmer physiologic and chemical foundation than that underlying goitre prevention. As the work of prevention is based on certain of these facts, the more important are reviewed by the authors. A milligram of iodine, given at weekly intervals, has been found sufficient to prevent thyroid hyperplasia in pups. If the iodine store in the thyroid is maintained above 0.1 per cent., no hyperplastic changes, and therefore no goitre, can develop. The method as applied to man consists in the administration of 2 gm. of sodium iodide in 0.2 gm. doses, distributed over a period of two weeks, and repeated each autumn and spring. This amount of iodine is excessive, and far beyond the needs of the individual or of the ability of the thyroid to utilize and store it. One gram distributed over a longer period would be better. The form or mode of administration of iodine is of little consequence. The important thing is that iodine for thyroid effects should be given in exceedingly small amounts, and it is believed that most of the untoward effects recorded are due to the excessive doses employed, or more concretely, to the abuse of iodine. The results of their two and one half years' observations on school girls in Akron are as follows: Of 2,190 pupils taking 2 gm. of sodium iodide twice yearly, only five have developed enlargement of the thyroid; while of 2,305 pupils not taking the prophylactic, 495 have developed thyroid enlargement. Of 1,182 pupils with thyroid enlargement at the first examination who took the prophylactic 773 thyroids have decreased in size; while of 1,048 pupils with thyroid enlargement at the first examination who did not take the prophylactic, 145 thyroids have decreased in size. These figures demonstrate in a striking manner both the preventive and the curative effects. The dangers of giving iodine, in the amounts indicated, to children and adolescents are negligible.