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DYSTOCIA FROM CONGENITAL CYSTIC KIDNEY OF THE FŒTUS [1]

With Report of a Case

From the Obstetrical Laboratory of the Johns Hopkins Hospital, Professor J. Whitridge Williams, Director

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ALTHOUGH congenital cystic kidney of the fœtus has long been recognized as a cause of dystocia, we find that there are comparatively few cases on record in the literature. Consequently we report the following case, in which was observed a twin pregnancy presenting the unique feature of cystic kidney and hydronephrosis of size sufficient to cause an obstacle to the birth of the first child.

Case. The patient was a young colored woman, who was brought to the Johns Hopkins Hospital in labor on January 26, 1899. She was 26 years old, and had previously had three spontaneous labors, the oldest child being six years of age, and the youngest three. All the children were perfectly formed, and there had been no complication in the labors or puerperia. She was just about at full term, and the pregnancy had been uneventful up to the last four months, during which time she had been suffering with more or less œdema of the lower extremities.

On examination, the pelvis was found to be of normal dimensions, but the uterus was very large, immensely distended, and reached up to within two fingerbreadths of the ensiform cartilage. The back of the child could be made out to the left, and the fœtal heart was heard on the left side, just below the umbilicus, and was 160 to the minute. On vaginal examination, the cervix was found completely dilated, the membranes ruptured, and the foot and knee, together with the cord, were projecting from the cervix. The cord showed a pulse of 162, corresponding closely to the rate made out on abdominal auscultation. Chloroform was at once administered, and both feet were brought down. It was seen that they both were clubbed, and showed six toes on the right foot, while there were seven on the left. As vigorous traction failed to accomplish descent, the fœtal body was palpated by passing a hand into the uterus, when it was at once found that the fœtal abdomen was of enormous size, and appeared to be filled with a cystic tumor mass, which prevented the body of the child from entering the maternal pelvis. Accordingly an incision some 6 or 8 cm. in length was made in the fœtal abdomen by Professor Williams, and a hard, more or less nodular tumor was easily palpated, removal of which was attempted by the fingers. This proved a very difficult operation, inasmuch at the hand soon became cramped, and it was necessary for two to work in turn, until some 470 grams of cystic tissue had been removed and the abdomen became somewhat collapsed. A soft cystic body now presented in the wound, and yielded on puncture about one pint of clear fluid, after which the child was extracted without great difficulty.

A second fœtus was now found to lie in breech presentation, and having ruptured the membranes, delivery was easily accomplished by extraction. This child proved to be a female, normally formed, and of the weight of 2,090 grams

The woman made a good recovery, and her temperature during the puerperium was never above 100°.

The Fœtus. The deformed fœtus was 42 cm. in length and 3,800 grams in weight. Projecting through the small posterior fontanel by an opening 2 x 2.5 cm. in diameter was a meningocele 4 x 3 x 2 cm. in size. Sixteen teeth were noted in the mouth, not ossified, but distinctly marked off, and cartilaginous in consistency. The right hand had a thumb and five fingers, and extending from the palmar surface of the little finger, an accessory finger was noted. The left hand had thumb and five well-developed fingers. Both feet were clubbed in the varus position and presented six toes on the right and seven on the left foot. The external genitals (male) were normal.

Autopsy At the autopsy no abnormalities were noted, save in the liver, kidneys, ureter, and bladder.

The liver weighed 150 grams and measured 12 x 8.5 x 3.5 cm. in its various diameters. On its under surface, near the left margin, were a number of small, clear, round cysts, the largest of which were 4 cm. in diameter. The left margin was markedly thinned, and contained apparently almost no liver substance, its place being taken by a number of tubular shaped cysts. The gall bladder and ducts appeared normal. On sections of the liver, the parenchymatous tissue showed an unusual amount of connective tissue bands, which appeared to correspond to the site of the biliary canals. Under the microscope this sheath appeared cellular, and contained an unusual quantity of ducts, some tortuous, and evidently of new formation, others irregularly dilated into cyst-like cavities. The epithelial lining of these corresponded in character to that of the larger cysts, which was columnar in type. Great numbers of round cells were seen occupying spaces between the hepatic cells. The picture presented accords with that usually noted in similar cases.

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The left kidney was the seat of the cystic growth. whose various pieces aggregated 1,190 grams in weight, Of this, 470 grams were removed at operation, while the larger mass remained in the abdomen and measured 16 x 13 x 6 cm., weighed 720 grams, and filled nearly the entire abdominal cavity. It was everywhere covered by peritoneum, save at the right and inferior surface, which was the site of the destructive operation. Beneath the peritoneal covering, myriads of cysts were noted, of bluish gray color. The ureter measured 1.5 mm. in diameter at the junction with the kidney and presented a patulous lumen. It was attached to the middle of the posterior portion of the tumor.

The exterior of the kidney was divided into a number of small, irregularly shaped areas, some of which were distinctly cystic. The capsule peeled without difficulty. On section, the tumor was semi-solid, and was found to be composed of myriads of cysts, which ranged in size from several centimeters in diameter to one so small as to be perceived with difficulty. The fluid obtained from the larger cysts was of clear serous character. The microscopical appearance is considered on page 636.

The right kidney was represented by a collapsed sac 13 x 7 x 4 cm. Its exterior was generally smooth, while the interior was lined by a smooth, glistening membrane, under which were a number of corrugations which gave a honeycomb appearance to the whole. The wall of the sac measured but 2 mm. in thickness. The urinary bladder was empty, and a few centimeters above the bladder the right ureter was "kinked" and stenosed. The placenta measured 16 x 14 x 2.5 cm. and weighed 470 grams. Comparatively few small white infarcts were noted. On microscopical sections, the villi were found swollen and œdematous in appearance, and the connective tissue core resembled that of a four months' placenta in its cellular consistency. Otherwise the examination was negative, as was that of the umbilical cord.

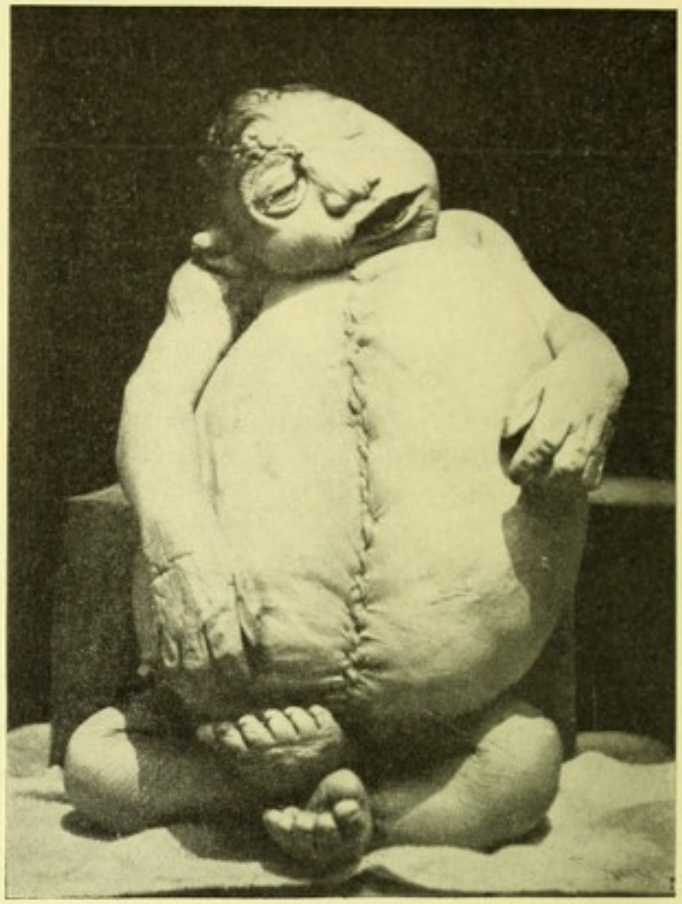

Fig. 1. Photograph of case.

In 1725 there was reported to the Royal Society of Paris the first cystic kidney of which I can find record, although Forbes states that the condition was known to Willis at an earlier period. It would appear, however, that these cases were probably simple retention cysts, and that the congenital cystic kidney was not brought before the medical world for discussion until 1790. The majority of authors cite Osiander's case in 1821 as the first recorded instance in which the congenital cystic kidney of the fœtus caused dystocia, although we find that Merriman in 1810 described a case in which delivery was impeded by cystic kidneys and a fœtal bladder tumor which contained some 240 c.c. of fluid at time of death, eight hours after birth. Following these observations, other cases were recorded by Mansa and Jacobson (1828), Oesterlen (1840), Cormack (1845), and Bouchacourt (1845), who, according to Budin, also assembled the literature of that time, and since then by other observers, the number of which has been variously stated by different authors. Thus Nieberding, in 1887, claims that dystocia has been known in twenty cases in the literature, although in his original report he cites but fifteen. Budin and Demelin, in 1900, state there are some fifty casse, but give no bibliography, and we are inclined to believe from our review of the literature that this latter figure is possibly too large, and includes, in all probability, cases in which there was no dystocia. We naturally, however, should expect much confusion in various summaries, inasmuch as cystic kidneys are frequently seen in combination with other deformities of the abdomen, such as cysts of the liver, dilatation of the ureters or bladder, ascites etc.; and where a combination of these exist, it may be impossible to state with certainty as to the relative importance of the individual factors in the obstruction. While reviewing the literature we have found thirty-eight cases in which it appeared that dystocia

was due to cystic kidney; yet, if we exclude the cases in which destructive operations were not necessary, the list is reduced to nearly a third.

We may obtain many items of interest from a review of these cases. The girth of the fœtal abdomen which causes dystocia is usually 45 cm. or more, and the kidneys may attain tremendous size. Not infrequently they have weighed 1,000 grams, while the maximum, according to Winckel, was noted in the case of Andrae, in which they attained the weight of 2,250 grams. The disease is usually bilateral, and the fœtus is the frequent subject of other deformities. Among these we find hydrocephalus, hemicephalus, encephalocele, and meningocele, harelip and cleft palate, amniotic adhesions and constrictions, abdominal fissure, peromelia, club-feet, polidactyla; anomalies of the circulatory organs, such as congenital absence of the aortic valves, of the ductus Botalli, defects in the ventricular septum, cysts of the liver; various deformities of the genito-urinary system, such as hypospadias, rudimentary external genitalia, uterus bicornis and septus, vaginal septa, absence of a tube or ovary, stricture of the urethra or ureter, absence or deformity of the urinary bladder and ureter, hydronephrosis, absence of kidney pelvis, etc., while hydramnion and oligohydramnion have both been noted. As a rule, labor has been premature, but the maternal abdomen has been distended to a marked degree. Many of the cases were in primipara, and the subsequent history shows that the condition is apt to recur in later pregnancies, which point has long been emphasized by others.

The fœtus frequently presents in breech, although, unfortunately, data as to the presentation and position is often lacking. In 27 cases in which it is given we find: Vertex, 15; breech, 11; face, 1.

As would be expected, the greatest difficulty has been experienced with the vertex cases, as much time has been lost in determining the cause of the dystocia. Generally, the head has been born spontaneously, although forceps were used in several instances. Some few cases should be mentioned to give a proper appreciation of the difficulties experienced. Thus in Mansa's case, the midwife, before sending for help, pulled off the head in her effort to overcome the obstruction, and Mansa, in turn, pulled off both arms with the blunt hook, and finally was obliged to cut through the fœtal thorax and into the abdomen before accomplishing delivery. Likewise, Hoering decapitated to obtain room to cut away the chest, and Wilson cut away the head and arms and part of the chest without being able to dislodge the kidneys, which were finally forced out into the uterine cavity in the extraction of the trunk following version. Bruckner and his colleague spent the entire day in attempting delivery, and performed, in turn, decapitation, amputation of the upper extremities, thoracotomy, evisceration, version, and extraction, while Voss consumed three hours in effecting delivery in his case. Embryotomy was likewise necessary in the cases of Wolff, Witzel, Porak and Couvelaire, and Porak, which presented by vertex.

The cause of the dystocia was more readily ascertained in the breech cases, and Nieberding alone appears to have attempted an extensive destructive operation. The fœtus was dead before the case was brought to the hospital, and Nieberding, suspecting ascites, punctured the abdomen without result, cut away the fœtal legs and pelvis, removing the kidneys without further difficulty. In the cases of Singer and Uhde, simple evisceration alone was necessary.

It is remarkable that in these cases, which were mostly observed before the days of antisepsis, there was no maternal mortality; nor yet in the series of dystocia from hydronephrosis and allied conditions collected by Magenau, save in one instance, in which the uterus ruptured before delivery. There are some few cases in which the obstruction was not serious enough to necessitate evisceration, in which the newly born fœtus made effort to breathe. Death, however, speedily supervened, due, in all probability, to the encroachment of the tumor upon the thorax and consequent limitation of the movement of the diaphragm.

Various rules have been laid down to determine the differential intrauterine diagnosis, but, in general, it has not been made until puncture of the abdomen fails to reduce its bulk or the definite cystic masses are felt in the abdominal cavity; frequently the uterus is so firmly contracted by the time dystocia is recognized,

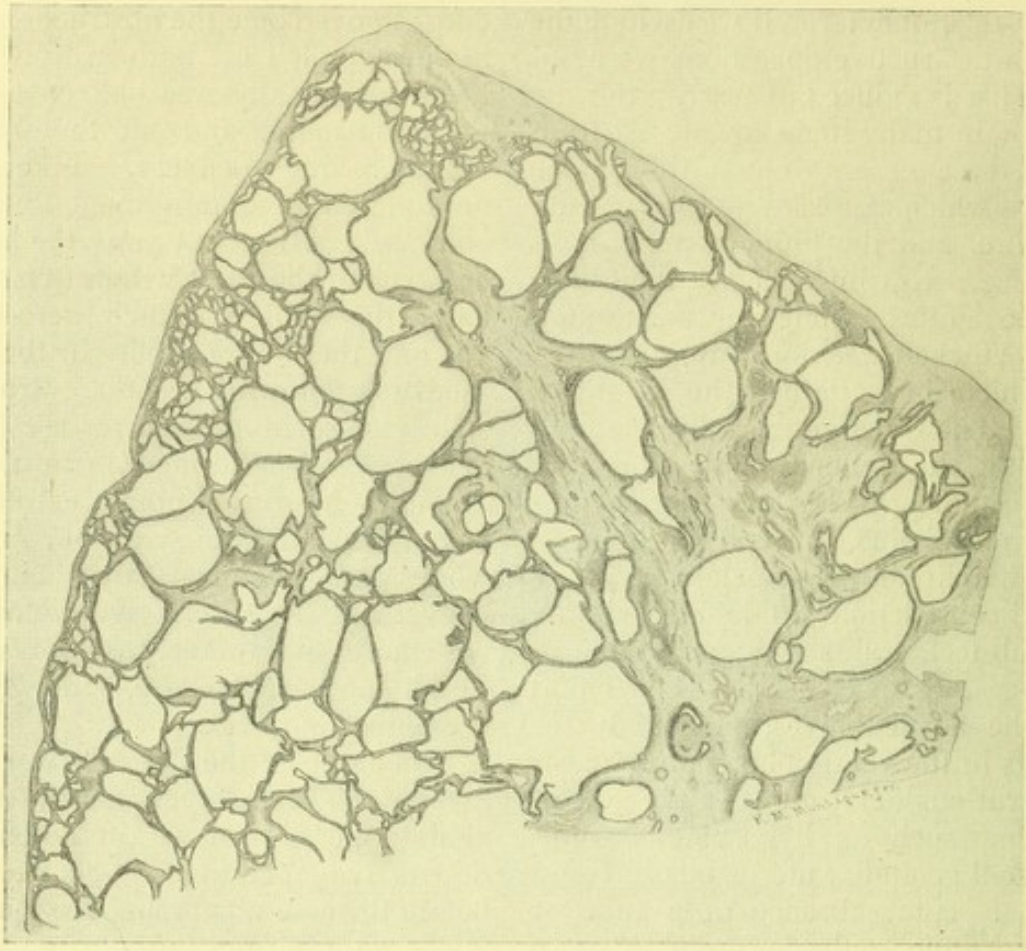

Fig. 2. Drawing of section of congenital cystic kidney, near cortex.

that careful palpation of the fœtus may be impossible. Suspicion should always be aroused by dystocia from overdistention of the fœtal abdomen associated with external deformities.

Many theories have been advanced to explain the ætiology of this disease, so many, in fact, that it has been questioned whether we have been dealing with a single pathological process. And yet, while the views advanced differ much in various details, there is enough similarity in the principles presented to admit of classification. Four theories are now recognized:

1. That the cysts result from obstruction and retention of secretion;
2. From errors in development;
3. New growth;
4. That the condition is of the character of both new growth and error in development.

1. The obstruction theory is the oldest of these, and assumes that the cysts are dilated uriniferous tubules and Bowman's capsules. It was first believed that the obstruction was in the lumen of the tubules. Thus Rokitansky, Förster, Frerichs, and formerly Virchow, ascribed it to infarction by various salts, as carbonates, phosphates, and urates; Ruyssenaers, Frerichs, and Albers suggested fibrinous exudate; Johnson, Erichsen, and others, impaction of desquamated epithelium and colloid substances; while Klein, Rosenstein, and for a time Brigidi and Severi, considered that the lesion might be nearer to the cortex, and attributed it to hemorrhage from the glomerular tufts with obstruction of the outlet and dilatation of the capsules. All of these views have been abandoned.

Many have referred the cause of obstruction to inflammation of the kidney, which theory was first advanced by Ruyssenaers in 1844. This view was elaborated by Virchow, who, having abandoned the theory that a uric acid diathesis in the mother, with consequent uric acid infarcts in the fœtal kidney, was the cause of stenosis, promulgated in 1855 the doctrine that has been adopted by the majority of the advo-

cates of the obstruction theory. Virchow now claimed that the disease resulted from intrauterine inflammation of the papilla, and that the subsequent contraction of the newly formed interstitial tissue caused papillary atresia, so that communication no longer existed between the uriniferous tubules and the calyces of the kidney. Moreover, he concluded that the cysts might lose their connection with the tubules from which they started, during the progress of the disease. He adhered to this theory in later life, and made it hold good even for the cases in which there was obstruction in the ureters.

Thorn, in 1882, carried the idea still further, and, in a case observed in an adult, showed that there had been extension of the inflammation from the kidney pelvis to the papillæ, resulting in atresia of the uriniferous tubules. Leichenstern, in 1884, also noted a similar condition in the cystic kidneys of an adult, which he designated nephropapillitis fibrosa. He assumed that the inflammation originated in the walls of the arteriæ rectæ, and then passed out to the papillary ducts, causing stenosis and retention cysts. Durlach, in 1885, reports a case in an infant of six months. The kidneys were large and cystic, and the pelvis was the seat of fibrous pyelitis; bands of connective tissue passed to the kidney surface through the columns of Bertini, excess of connective tissue was also noted in the lateral portions of the pyramids, and above this retention cysts. Arnold, in 1890, reported a case in a syphilitic fœtus, and demonstrated a nephropapillitis as the ætiological factor, holding that in the extension of the disease the tubules became constricted, with consequent alteration of the normal secretions. Among others who have favored this theory of intrauterine inflammation may be mentioned Stöhr, Mackenzie, Smith, Schultz, and Bockenheimer.

Others have taken the stand that a general interstitial new growth or cirrhosis has been the cause of the constriction of the tubules, and see in the congenital cystic kidney lesions analogous to the cysts in chronic nephritis. Sabourin was an enthusiastic advocate of this view, and Cornil and Brault have described various conditions of the tubular epithelium, secondary to the retention and resulting from a primary kidney sclerosis. Ebstein, Palm, and Ruckert have reported cases in which the fœtal kidneys shrunk to the size of a bean. Dunger notes a similar case in which the lesion was unilateral.

The obstruction theory has not proven satisfactory to all observers. Some, after reviewing cases presented in support of this view, have adduced them as evidence for other theories. Thus, Mutach observed a case presenting the same features as one which Virchow thus described: "The ureters on both sides were open only to the level of the lower rim of the kidney, where they gradually became narrower and the lumen stopped entirely, while the wall became merged in the connective tissue which filled the region of the hilum." "Atresia of the kidney pelvis therefore certainly existed on both sides." Although Mutach was able to see no opening in his case, he found he could inject alcohol from the ureter into the tubules, and by cutting serial sections demonstrated communication to the papillary zone. He stated, therefore, that with the laboratory methods of recent time, Virchow might have found that the atresia was not absolute. Others have called attention to the fact that as yet there has been no satisfactory explanation of a peculiar localized inflammation of the papilla of each kidney (for the lesion is commonly bilateral) associated with defects admitted to be the result of error of development of the kidney, such as absence of the pelvis of the kidney, branching of the ureters, etc.

2. Many have taken the ground that the great frequency of deformities resulting from errors of development in cases presenting congenital cysts of the kidney is of more than accidental relation. The probability of this view has been strengthened by the complexity of the process of development of the genito-urinary tract. Koster, in 1866, claimed that atresia of the papilla was more likely due to a primary defect of development than to an intrauterine inflammation, and called attention to the frequency of congenital defects of the kidneys, as absence of the kidney pelvis or ureters, in cases of cyst kidneys. He evolved his theory on the basis of Kupffer's conception of the development of the kidney; i.e., that the tubules develop independently of the pelvis, and fuse at a later period. Failure so to unite would cause this lesion. Koster advocated this

theory only for the cases which presented no kidney pelvis.

Shattock, in 1886, concluded that the cysts were retention cysts derived from portions of the Wolffian body which were included within the kidney, and called attention to the similarity of origin of cysts of the ovary and testicle. He supported this theory on the ground that the kidneys are developed in the following manner: The Wolffian body and kidneys are both developed from the intermediary cell mass of mesoblastic tissue which lies between the dorsal plate and the mesoblast of the somatopleure. This intermediate cell mass becomes divided into three portions: 1. Anterior, forming the pronephron, or head of the kidney. It is rudimentary, and disappears very early in the human embryo. 2. Middle, is the mesonephron, or Wolffian body, from which develop the generative glands. 3. Posterior, or metanephron, gives rise to the kidney. The ureter develops from the Wolffian body and becomes invaginated into the kidney to form the pelvis and calyces, while the glomerulæ develop from mesoblastic tissue connected with the aorta. 'It may not be beside the question of want of demarcation between the meta and mesonephros," he says, "to refer to the doubling or trebling of the ureter, which is not rarely seen in man. One may regard the variation as a reversion to the lower type. In amphibia the mesonephron consists of an anterior sexual and a posterior non-sexual or urinary part; the collecting tubules of the latter, in the female at least, enter the Wolffian duct directly, so that there are as many ureters as there are segments of the mesonephric kidney. The connection of the two ureters with the Wolffian duct has subsequently been shifted from its side, as is normally the single metanephric ureter, which, in mammals, also primitively opens into the same duct. If this view is correct, it must be assumed that the portions of the kidney in connection with the superior of the ureters represents a persistent segment of the mesonephros, or permanent kidney, with which it retains its primitive continuity."

Mutach studied the kidneys of normal fœtuses between 15 and 30 cm. in length, and found that interstitial tissue was present in exceedingly large quantities in the apex of the kidneys

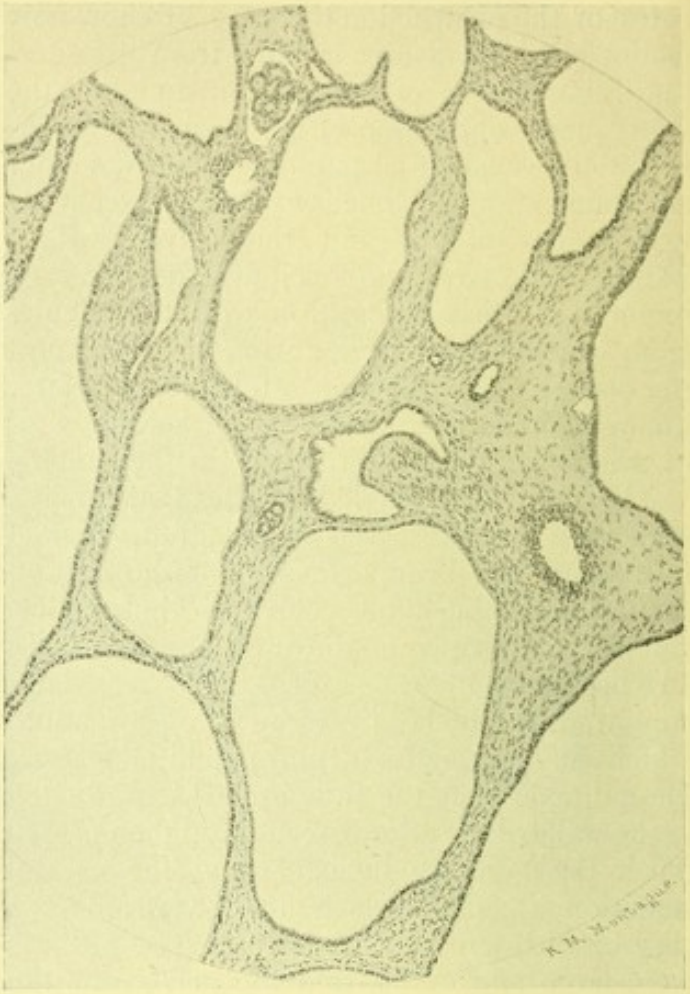

Fig. 3. Drawing. Intracystic papillary formation. +

in the younger specimens, but was less marked in the older kidneys. In both of his cases of congenital cystic kidney there was a similar increase of connective tissue of embryonic type about the papilla, which he thought could be attributed more fairly to a persistence of an embryonic condition than to an intrauterine inflammation of such limited extent, especially as a typical error of kidney development was observed in the second case, in which there was no pelvis, and the ureter was attached to the kidney by means of a number of minute tubules. Several islands of hyaline cartilage were also noted in the stroma of this case.

Hildebrand modified the view of Koster to meet the requirements of the modern dualistic theory of the development of the kidney, which assumes that both the glomeruli and convoluted tubules arise from one "anlage" and are joined secondarily by the collecting tubules which develop from the kidney pelvis. If the two systems of tubules fail to join, retention

cysts would form in the convoluted tubules. Springer, in 1897, and later Ribbert, in 1899, also make use of this conception, but emphasize as the causal factor a wild proliferation of the cells of the tubules which fail to join, rather than a retention of secretion. This proliferative tendency was so marked in Ribbert's case as to merit the description "tumor-like," which tendency has also been observed by Meyer and Busse. On the other hand, Bard and Lemoine have arrived at the conclusion in their case, that the ætiological factor was not a hyperplasia of the tubular epithelium, but rather a marked weakness of the walls of the tubules, especially of the basement membrane, which dilate under pressure of the normally secreted fluid. Among others who explain the ætiology of their cases on the basis of error of development may be mentioned Hanau, Mirabeau, Brouha, and Bland-Sutton, who did much to popularize the view of Shattock.

3. The theory that the cystic kidney is a neoplasm is usually thought to be of recent origin, yet we find that as early as 1875 Sturm expressed the view that the cysts originated from the epithelium of the convoluted tubules, which, as a result of the dilatation, hypertrophied, and later developed into bud-like processes of an adenomatous nature. In the following year, Michalowicz called attention to similarity of the appearance of cystic kidneys and cystic disease of the ovary, testicles, and mammary glands. Brigidi and Severi, in 1880, stated that the condition was definitely a new growth. They recognized an active proliferation of the epithelium of the tubules, and described the lesion as a cystoma of the kidney. Chotinsky, in 1882, also observed an active proliferation of the epithelium. Papillary outgrowths were frequently seen, similar to those in the cysts of the senile mammary gland. Many of the smaller cysts were filled with epithelium, and there was evidence of new formation of the tubules. Moreover, he found that the membrana propria was irregularly hypertrophied, and concluded that the primary factor was a hyperplasia of the tubules and of the stroma, as he could find no evidence that the glomeruli were concerned in the process. Philippson, although he thought that Virchow's doctrine might hold good in the fœtal lesions, described a proliferation of epithelium and of the membrana propria in adult specimens which resembled cystoma, and, although he drew no definite conclusions, stated that this proliferation must be considered as well as any obstruction in the tubules. Hommey, in 1887, also recognized the proliferation of epithelium which proceeded to cyst formation, and although he was unable to account for this condition, held, in contrast to Sabourin, that the proliferation of the epithelium was secondary. Lejars, in 1888, considered that the great majority of cyst kidneys were of the type of an "epithelioma mucoide," together with the cysts of ovary and testicle. Nauwerck and Hufschmid, in 1893, showed that the epithelium of the urinary tubules proliferates, fills the tubule, and causes its dilatation. The cysts arise from the colloid transformation of the epithelium and the transudate from the walls. In addition to the inward growth of the epithelium, which they regard as the essential point in the formation of the cysts, there occurs less frequently a progression of solid epithelial columns into the stroma. They definitely place the lesions in the group of adenocystomata. Von Kahlden, in the same year, also supported this conception, but emphasized further, that there was a coordinate development of the stroma analogous to that in adenofibromata. In 1894, he described the cystic kidney of a fœtus in which the histological picture strongly resembled the intracanalicular myxofibromata so frequently noted in the mammary glands. Since then there are many supporters of the theory that the condition is a cystoma, among whom may be mentioned, Mutach in his second case, Hansemann, Claude, Albert, Hein, and Schmitz.

4. If one accept the theory of Cohnheim that certain tumors arise from the proliferation of persisting centers of embryonic tissue, i. e., an error in development, it will be seen that there may be no sharp line of distinction between certain theories in groups 3 and 4, and several observers have taken a position midway between these theories. Thus Couvelaire, in 1899, objects to the consideration of the lesion as a tumor of the cystoma type. He holds that the process is essentially a general proliferation of the epithelium of the urinary

assages in the kidney, together with a correlated reaction of the connective tissue, in hich the cells lose their specific character; nd inasmuch as this change takes place in the eveloping organ, the lesion is to be considered s a malformation. Börst also considers it as n error of development, in the sense that it is ue to a disturbance of equilibrium between the rowth of epithelium and connecting tissue. e sees in the cysts of the kidney and liver a ailure of the epithelium to develop in the ormal manner, and as a result of its increased ower of growth there is invasion of the conective tissue. However, he retains the lesion nder the cystic adenofibromata. Bunting ates that Luzzatto concludes that the pathoenesis of the cystic kidney is not uniform; at in the shrunken cystic kidneys the lesion of an interstitial nephritis of fœtal origin, ut that in the more common type it is to be garded either as a malformation or a tumor congenital origin, between which he is nable to draw a sharp line. Still, he is inclined include four congenital cases of his study nder fibrocystadenomata, and a fifth under alformations, due to an excessive developent of connective tissue in the organs. Dunger lso recognizes an inflammatory type, but elieves that the largest number of cases are but bildungs anomalie." He says in some intances there arises a proliferation of tissue onsequent to the error of development, and e result is termed an adenocystoma, but we hould constantly bear in mind that the cause an error of development, and the adenoystoma merely represents the histological icture. Quite recently Bunting has had the pportunity of examining two fœtal cases in the arly stages of the disease, and concludes that e position of the lesion in the pathological lassification depends upon the definition of rms. "If it be a neoplasm, it differs from e conditions usually included under that rm." "If it be a malformation, it is one in e sense of . . . a disproportionate activity in e growth of the epithelium."

Study of the cases in the literature indicates at there is great difference in the pathological ictures presented, and it seems quite probable at all cases of the disease could not be brought nder one ætiological classification. Unfortunately, experimental pathology offers us no solution of the problem, inasmuch as the disease has not yet been reproduced experimentally. Pettersson recently attempted it by ligatures passed through the papillæ of guinea-pigs. His results, however, are similar to those of others; i. e., no cyst formation, but dilatation of all tubules, together with atrophy of the organ, sometimes destruction of the collecting tubules and the papillary connective tissue. Tollens also opened the kidneys of rabbits and cauterized the papillæ in order to cause stenosis of the ducts. After intervals of 4, 8½, and 12 weeks, the animals were killed and the kidneys examined. The first specimens showed kidneys of twice normal size, with minute cysts formed by the dilated tubules. The papillary ducts were closed by scar tissue, and there was considerable proliferation of the connective tissue as well as of the epithelium of the tubules. The later specimens showed atrophy of the gland, proliferation of interstitial tissue, and, occasionally, dilated tubules, and it is the general belief that an experimental constriction of the draining passages of glandular organs causes an atrophy of the gland, rather than a cystic change.

Kidney tissue from our case was fixed in various fluids, alcohol, Müller's and Zenker's fluids being used, and a series of serial sections was cut from different portions of the tumor. To the naked eye these presented similar pathological pictures, the chief difference being the size of the cysts and the thickness of the septa. No normal kidney tissue was anywhere seen, and the entire section gave the appearance of a piece of lace-work. As a rule, the cysts present a more or less rounded appearance, although they are frequently compressed into irregular outlines. Usually the cyst spaces are clear. The lining membrane is generally of a single layer of cuboidal epithelium with large rounded darkly staining nuclei. The connective tissue wall is many times as thick as the epithelial lining, and is composed of fairly cellular connective tissue, which frequently gives the appearance of a greatly thickened basement membrane. It is usually of even thickness. This, in turn, is supported by underlying tissue, containing comparatively few nucleated cells, and, in a

measure, it suggests the structure of the connective tissue of early chorionic villi. Some extravasation of blood is noted on the looser tissue spaces, yet, in general, there are few blood-vessels, and these present as normal.

Here and there tubules may be seen between the larger cysts, sometimes lying in the supporting connective tissue core of the cysts themselves. The epithelium is dark-staining and of columnar type. Occasionally the nuclei show evidence of proliferation, and in rarer instances the epithelium completely fills the lumen of tubules which seem to show some evidence of dilatation. Few glomeruli are seen, and when present they show evidence of dilatation with thickening of the capsule sheath. Projecting into the cysts, papillary growths are frequently seen. In general, they are composed of tissue presenting the general characteristics of embryonic cells. Frequently they contain in their substance tortuous canals lined by deeply staining columnar epithelium, which often presents evidences of proliferation. The epithelial coverring of the intracystic growths is usually of single layer. Here, too, may be seen mitotic figures. No downgrowth of epithelium into the connective tissue was noted, although on all sections we can see columns of epithelium extending into smaller cysts. Rarely do we observe connection between the larger cysts. When present, these are short narrow canals lined by cuboidal cells.

When we attempt to determine the ætiology of our case we are naturally confronted with the question, Was there common cause for the hydronephrosis, cystic kidney, and the other deformities? It is, of course, impossible to answer this with certainty, yet it would appear from the uniform results of the experimental work of Aufrecht, Strauss and Germont, Holste, and others, on the cause of hydronephrosis, that this lesion is the result of the stenosed ureter. Orth, however, voices the opinion of the majority when he states that the cause of cystic kidneys must be sought above the kidney pelvis. Our sections give no support to the view that the lesion is the result of previous inflammation, nor yet of error of development, in the sense of failure of the tubules to fuse, which could be proven only in a kidney retaining some normal structure. The histological picture resembles that described by the advocates of the neoplastic theory: irregular growth of epithelium and connective tissue, the formation of new tubules, the presence of intracystic papillomata. These merit the term of adenocystoma.

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