STUDIES IN INTESTINAL OBSTRUCTION

WITH A REPORT OF FEEDING HETEROLOGOUS JEJUNAL AND ILEAC CELLS TO A HUMAN BEING *

JOHN WILLIAM DRAPER, M.D.
NEW YORK

To Roger we are indebted for the present conception of the cause of death in intestinal obstruction. He was undoubtedly the first to conceive of it as a true auto-intoxication.

In a paper read before the Johns Hopkins Medical Society seven years ago and based on a study of the effects of 400 duodenal obstructions produced in animals at the Surgical Research Laboratory at Columbia, I reported corroborative evidence, from a surgical aspect, of Roger's view. William Welch on opening the discussion of my paper said that, if corroborated, the facts presented would show that the duodenum had a function hitherto unknown and one evidently of the utmost importance in medicine. It is gratifying that this corroboration has just recently come from Dr. Welch's own laboratory at the hands of Whipple and his associates. By a series of skilfully devised experiments, these investigators have succeeded in casting much more light on this interesting problem than either Roger or I had been able to do, and the sum of our investigations taken in the aggregate seems now to offer incontrovertible proof that the cause of death is not bacterial in origin, but truly autotoxic from the cells of the epithelium of the intestine itself.

Perhaps no branch of medical research better illustrates than this one the advantages derived from having men with one object in view working at different centers of education toward the solution of a common problem. The different points of view ultimately arrange themselves on convergent lines so that when the answer is finally won it is evidently not the product of one man's aims or ambitions, but a homogeneous unit, resulting from the constructive work of several minds. For no single individual can expect to do much more with a modern problem than to advance it a single step. This problem also serves to demonstrate the rapid breaking down of the traditional barriers between medicine and surgery, forecasting the ultimate fusion of these old and arbitrary subdivisions into a new unit, the members of which will have a working knowledge of physiologic chemistry and its allied sciences in addition to requisite technical skill. Furthermore, it demonstrates that modern surgery, like physiology, does not consist in the manufacture and use of instruments of mechanical precision but in philosophical interpretation of natural phenomena.

The various old and groundless theories as to the cause of death in intestinal obstruction have one by one been disposed of, with the possible exception of the exact origin of the toxemia. Is it bacterial, or purely autctoxic from the cells of the intestinal epithelium itself? Were it not for the views of Fred Murphy, which result from his studies at the Washington University and which bear evidence favorable to the bacterial theory, one would be ready to accept the result of the painstaking and thorough researches at the Cornell laboratories of Hartwell and Hoguet who seem definitely to have excluded the anaerobes, and of Whipple and his associates at the Hopkins laboratories, who are disposed to rule out all bacteria as causative agents in the development of the toxemia. From the prosaic and common sense point of view of applied surgery, it is hard to understand why obstruction in the duodenal region should be many times as dangerous as obstruction in the colon. It has long been known that duodenal contents may be allowed to escape into the peritoneal cavity without danger, but that peritonitis is to be expected if any colonic contents escape.

No careful observer could watch the death of a series of duodenally obstructed dogs without noticing the close similarity between the ante-mortem symptoms of this condition and those seen after parathyroidectomy or long remain in doubt that the function of the liver was greatly impaired in each.

The accompanying tables are based on the study of the urine of duodenally obstructed dogs, both before and after a large dose of camphor had been given hypodermatically. The recovery of the paired glycuronic camphor has been made by the method devised by Tollens. I am indebted to Frederick W. Schlutz of the University of Minnesota for the privilege of presenting these results, which are a chronicle of his chemical studies made on dogs obstructed by me. In a previous communication we have jointly presented two similar studies, both of which appear to show more conclusively than the present ones that liver function was impaired. Negative findings, reported of carefully wrought work, are, however, just as valuable as positive findings, particularly in the case of a method which is as yet open to some question as to accuracy, and which is certainly open to criticism as regards the time taken in working it out. Dr. Schlutz is carrying on further studies of the accuracy of Tollens' method. As indicated in Table 1, the discrepancy between this report and our previous findings, in which the liver function, as indicated in terms of camphor-glycuronic was considerably lowered, may be due either to inaccuracies in the method itself or our application of it, or to the fact that we were unfortunate in having one of our dogs live but a very short time while the other one lived an unusually long time. Dr. Schlutz will make a

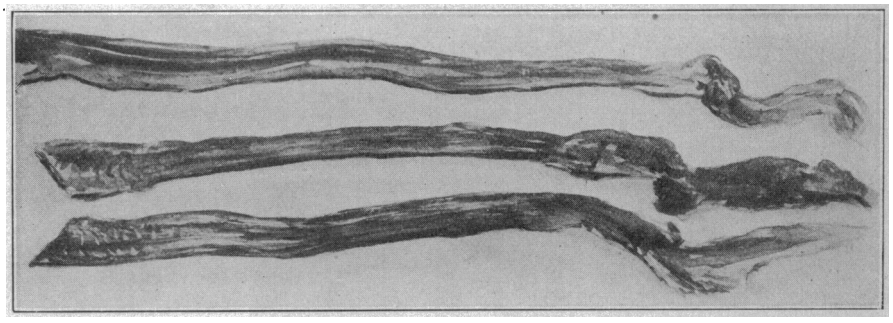

Fig. 1.—Above, the split colon of a normal dog; in the middle, the split colon of a dog salivated for four days by pilocarpin, and below the split colon of a dog dying from acute duodenal obstruction.

* From the Laboratory of Experimental Surgery, New York University.

* Read before the Section on Pathology and Physiology at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

further report which will clarify this interesting question.

When Hartwell and Hoguet advanced their hypothesis that water privation was the chief lethal factor after intestinal obstruction, at the suggestion and with the help of George Wallace we conducted some experiments to determine the exact water percentages in the tissue of dogs under various conditions. It was thought that salivation by pilocarpin, fasting, and duodenal obstruction might cause approximately the same water-loss to the tissues. Table 3 shows the results from six tissues, namely, liver, lung, heart, kidney, spleen and muscle. It is seen that within a few tenths of 1 per cent. the water-loss after pilocarpin, after fasting, and after death from duodenal obstruction was the same, namely, 10 per cent. In the case of the salivated dogs and the fasting dogs, euthanasia was practiced on the fourth and seventh day, respectively, long before there were any signs of disability. On the other hand, the duodenally obstructed dogs were allowed to die of their autotoxemia, so Table 3 gives the maximum water-loss for obstruction but not for salivation or for fasting.

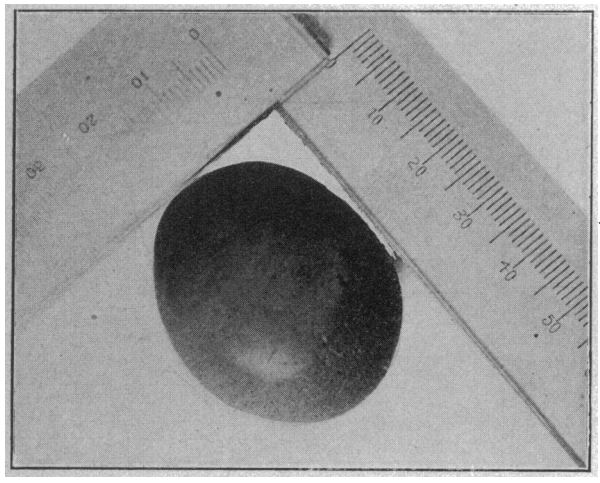

Fig. 2.—Acute jejunal obstruction in dog. Stone swallowed six months before operation, causing chronic stomach symptoms, but no obstruction until it slipped into the bowel. Note partial digestion of soft area by gastric juice. Recovery followed removal of pebble and feeding of dog with jejunal and ileac epithelium.

These studies appear to offer additional proof that in intestinal-obstruction toxemia, as in other toxemias, whether bacterial or truly autotoxic in origin, the solvent power of water on the toxins and its help in eliminating them from the body serve in many cases to prolong life, and, under certain conditions, to save it. Indeed, Wallace has shown that an animal may be given double the lethal dose of diphtheria toxin and yet live, if treated by abundant hypodermoclysis. But while valuable from a therapeutic point of view they cast no light whatever on the origin or the nature of the toxins. Furthermore, it has been the clinical experience of all surgeons that even an abundance of water will not serve to save the life of a human being in whom there had existed for any length of time complete duodenal obstruction.

We have again subjected to careful microscopic study the liver, heart and kidneys of a large number of obstructed animals and find that except for capillary dilatation they appear normal. In this connection as well as in the conduction of the feeding experiments, I acknowledge the efficient help of Dr. Eisberg. These microscopic findings are simply corroborative of previous reports made by myself and others. The beginning and end of the alimentary canal, however, on its epithelial surface, shows this capillary dilatation so markedly as to attract the eye on gross examination. I have shown a colored photograph of a stomach removed from a duodenally obstructed dog at the Mayo Clinic. I have shown and called attention to this before, without knowing its importance or significance; and because of its bearing on certain data which I shall present later, I am glad to say that the observation is corroborated by Whipple. In our necropsy experience the small intestine has shown little if any, gross change, but the colon is almost invariably affected and in a very characteristic manner. I understand from Wallace that the same condition obtained after a lethal dose of diphtheria toxin had been given.

TABLE 1.—INTESTINAL OBSTRUCTION, DOG 201: LIVER FUNCTION IN TERMS OF GLYCURONIC CAMPHOR (TOLLENS), SCHLUTZ-DRAPER SERIES

Date of Experiment	Length of Period, Hrs.	Vol. of Urine, c.c.	Normal Amt. of Glycuronic Acid	Amt. actually Passed as Combined Camphor Glyc. Acid	Increase Over Normal in Glycur.	Remarks
1-27-28	24	130	0.1794	.....	.....	2 gm. of camphor.
1-28-29	24	122	0.1092	.....	.....	A Theoret. total of 452 gm. Comb. camph. glyc. acid. 37.02 per cent. combined before, 5.64 per cent. obstruction.
	Camphor 2 gm. hypod. 3 p. m. ½ 9-1912					
1-29-30	24	147	0.1470	0.4428	0.2958	1 c.c. of normal undiluted urine = 0.0010 gm. glyc. acid.
1-30-31	24	215	0.2150	1.4094	1.1944	1 c.c. of normal diluted urine = 0.00028 gm. glyc. acid.
1-30-11-1	25	227	0.2270	0.4104	0.1834	
	Intestinal obstruction, 3 p. m. 7-1912; 24 hrs. later 2 gm. camphor hypodermically.					
11-1-2	34	900	0.9200	1.1772.	0.2572	

Discrepancy with results previously reported may be due to short postoperative life or to inaccuracy of Tollens' method.

Thus it appears increasingly probable that the toxins of intestinal obstruction are eliminated both from the stomach and from the colon. When we recall the method of elimination by the stomach of morphin, its reabsorption by that organ and the value of frequent stomach-washings in the treatment of morphin poisoning, the post-mortem picture of intestinal obstruction is not so surprising, and the value of lavage and epithelial cell feeding in complete and incomplete obstruction as well as in acute dilatation of the stomach is more easily understood. It strongly suggests that most cases of postoperative dilatation are due to an adynamic duodenum, just as hydronephrotic dilatation of the kidney is often caused by physiologic ureteric obstruction. An organ dilates first at the point of obstruction to its outlet, be the obstruction mechanical or physiologic.

The capillary evidence of stomach elimination affords a working hypothesis on which we may

explain the interesting results of feeding to duodenally obstructed animals the epithelial cells from the ileum and jejunum of other animals. Table 4 shows the result, as regards the length of life, to be quite definite, the fed dogs living nearly twice as long as the controls. This series agrees with a similar series reported by me from the Mayo Clinic. It is not to

TABLE 2.—INTESTINAL OBSTRUCTION, DOG 202: LIVER FUNCTION IN TERMS OF GLYCURONIC CAMPHOR (TOLLENS), SCHLUTZ-DRAPER SERIES

Date of Experiment	Length of Period, Hrs.	Vol. of Urine, c.c.	Normal Amt. of Glycuronic Acid	Amt. actually Passed as Combined Camphor Glyc. Acid	Increase Over Normal in Glycur.	Remarks
1-27-28	24	116	0.1458	.....	.....	1.7 gm. camphor = a theoret. total of 3.842 gm.=comb. camph. and glyc. acid. 0.2 gm. camphor = 452 gm. comb. camphor and glyc. acid.
1-28-29	24	150	0.1548	.....	.....	
	1.7 gm. camphor in oil subcut.					
1-29-30	24	83	0.0913	1.2294	1.1381	Before obstruc. 44.56 per cent. of camphor comb. with glyc. acid.
1-30-31	24	100	0.1100	0.3582	0.2482	After obstruc. 47.64 per cent. were comb., but practically all in the first 24 hrs. after that markedly dim. function.
1-31-2-1	24	130	0.1430	0.2880	0.1450	1 c.c. of normal undiluted urine = 0.0011 gm. glyc. acid.
	Intestinal obstruction 4:30 p. m. 2/1/12					
2-1-2	24	55	0.0615	0.2424	0.1809	
	Camphor 2 gm. subcut. 2/2/12, 2 p. m.					
2-2-3	24	366	0.4026	2.3100	1.9074	
2-3-4	24	109	0.1199	0.2310	0.1111	
2-4-5	24	246	0.2706	0.2906	0.0200	
2-5-6	24	134	0.1474	0.2208	0.0724	
2-6-7	24	85	0.0935	0.1140	0.0205	
2-7-8	24	60	0.0660	0.0888	0.0228	

Postoperative life unusually long may indicate high liver resistance or inaccuracy in Tollens' method.

be forgotten that scrapings from the frog's duodenum will stimulate pancreatic secretion in the higher vertebrates. Intercellular reactions must be among the most ancient and most fixed of our functions, and the feeding of intestinal cells may prolong life by reestablishing certain vital intercellular reactions which the obstruction had interrupted. Although knowing empirically that if jejunal and ileac epithelium were placed in the stomach of a duodenally obstructed dog, that dog would outlive one either not fed on cells at all or fed on cells of organs other than the small intestine. I could not previously understand this but it now seems probable that since the toxins are undoubtedly thrown out into the stomach they are directly rendered harmless by the heterologous cells from the small bowel derived from a healthy animal and are thus prevented from reentering the body. This explains the immediate benefits derived in the treatment of acute dilatation of the stomach and since this condition is undoubtedly due to physiologic duodenal obstruction, it seems probable that the feedings of these epithelial cells may prove to be just as useful in the treatment of dilatation as in the postoperative treatment of obstruction.

The study of Table 4, constructed from the last series, shows that it differs from the former series in that the pulse-rate of the different groups has been unaltered. A reasonable explanation of this is that the pulse was taken in the recent series at the femoral, whereas in the series reported from Rochester it was in each case taken at the apex. The femoral count is certain to be much lower when the heart is running high because many of the beats are not transmitted to the finger. While this series for the foregoing reason shows little change in the pulse-rate, it does show a much greater increase in the length of life, and this certainly is the best index of the effect of any treatment.

A glance at Table 4 shows that of the dogs fed with epithelium only two died before the hundredth hour, whereas of the controls only three lived beyond the hundredth hour. It is interesting to note that one of these lived 239 hours, showing that occasionally one finds an animal naturally resistant to duodenal obstruction. In the average this is balanced in the fed series by one which lived 284 hours, so that the general average remains unchanged and it is quite safe to say that the increase of life, which in this series is nearly doubled, is due entirely to the detoxicating effect of the epithelium.

In order to show that fed jejunal and ileac epithelium exercise some special detoxicating power not yet understood but definitely recognizable, we fed a control series of dogs on the emulsified cells of liver, spleen, pancreas and muscle-tissue. These animals lived a few hours longer than the not-fed controls, but it is evident that these cells had either no detoxicating action, or a very feeble one, compared with the intestinal epithelium. If we prove to have at hand

TABLE 3.—WATER PERCENTAGES OF SALIVATED AND DUODENALLY OBSTRUCTED DOGS ACCORDING TO TISSUE; SALIVATED SERIES, EUTHANASIA FOURTH DAY; FASTING, EUTHANASIA SEVENTH DAY; OBSTRUCTION SERIES, AUTOTOXIC DEATH ON FOURTH DAY (AVERAGE)

Tissue	Normal					Salivated (Pilocarpin)			Fasting 7 Days	Duodenally Obstructed					
	Dog	211	193	236	Aver.	205		Aver.	204	207	206				Aver.
Liver..	73.6	72.0	70.0	79.2	73.7	74.8	60.0	67.4	62.6	59.0	60.6	69.0	68.0	71.0	65.5
Lung..	....	80.4	68.2	84.3	77.6	71.2	79	75.1	76.8	72.8	66.8	....	....	....	69.8
Heart..	77.0	77.0	84.0	75.0	78.2	73.0	63.0	68.0	74.8	74.4	62.0	55.0	75.0	77.0	68.7
Kidney.	....	78.7	80.0	74.5	77.4	75.2	....	75.2	70.9	67.2	59.3	....	74	78	69.6
Spleen.	....	76.3	77.5	74.1	75.9	....	51.0	51.0	....	64.5	....	....	74.0	79.0	72.5
Muscle.	75.0	74.0	75.1	72.7	74.2	44.2	74.0	59.1	45.6	42.8	40.0	65.0	69.2	....	54.2
Average	....	....	....	....	76.1	....	....	66.0	66.1	....	....	....	....	....	66.7

Salivation, fasting and duodenal obstruction each cause 10 per cent. water loss, showing this factor to be negligible in death by obstruction.

some less cumbersome method than Tollens' for determining liver function, it would be interesting to use it as an index of the effect of feeding small intestine epithelium.

I have utilized jejunal and ileac epithelium clinically in two instances. First in a valuable female dog which had been imported from England about six months before, and had had "chronic stomach trouble" ever since her arrival. She, however, whelped a large litter of pups about six weeks before I saw her, without difficulty. She was a large poodle, weighing about 40 pounds. She had had complete intestinal obstruction for five days and she presented all the clinical symptoms which I have described, notable tachycardia, extreme nervousness, and great weakness in the hind extremities. On opening her, with the consent and on the advice of Dr. Blair, I removed from the oral portion of the small intestine and at a point not exactly determined, the pebble which is shown in the illustration. Her condition was very grave, the pulse running from 180 up and showing great irregularity at the apex. She was immediately put on small intestine epithelium derived from two dogs of a different breed. No one is justified in drawing any deductions from one case, but from a long experience with the symptoms of duodenally obstructed dogs, I should not under ordinary conditions have expected her to recover. The symptoms, however, gradually subsided and she lived.

TABLE 4.—FEEDING HOMOLOGOUS CELLS TO DUODENALLY OBSTRUCTED DOGS

Fed Jejunal and Ileac Epithelium			Fed Emulsified Liver, Spleen, Kidney, Pancreas and Muscle Tissue			Not Fed (Controls)		
Dog No.	Hours Lived After Obst.	Average Pulse	Dog No.	Hours Lived After Obst.	Average Pulse	Dog No.	Hours Lived After Obst.	Average Pulse
200	197	157	49	72	156	185	83	149
202	114	147	53	115	140	186	239	109
204	171	161	56	75	149	188	66	133
33	166	200	57	71	179	189	50	167
34	94	136	58	147	155	191	126	174
38	284	182	63	74	171	192	53	134
48	72	144	64	52	163	193	108	170
44	191	140	65	47	140	196	68	154
45	101	150	62	144	181	197	72	141
46	258	143	59	247	140	198	20	172
	1648	1560		1044	1574		885	1563
	164.8*	156†		104.4‡	157.4¶		88.5§	156.3‖

* Average hours of life when fed epithelium.
† Average pulse when fed intestinal epithelium.
‡ Average hours of life when fed emulsion of organs.
¶ Average pulse when fed emulsion of organs.
§ Average hours of life of control.
‖ Average pulse of control (at femoral).

The second instance in which I have used the epithelium therapeutically was in the case of a man referred to me by my colleague Jerome Lynch. The patient had had definite symptoms of obstruction for ten days, and when the abdomen was opened, an annular cancer was found which closed the terminal ileum. Not knowing whether heterologous epithelium would have any detoxicating effect, but remembering that the duodenal epithelium of frogs is capable of stimulating pancreatic secretion in the higher vertebrates, and realizing that this patient was in a desperate condition, Dr. Lynch and I decided to feed him the emulsion from a dog. Hourly doses were given until the entire epithelium from the two animals had been taken. The patient objected much more to some magnesia which we ordered, than to the dose of epithelium, which he described as "not half bad." The pulse improved and the patient lived. These cases prove nothing; they simply show that heterologous cells may be given without discomfort or harm.

Different operative procedures were used in each. In the first, the continuity of the bowel was reestablished and most of the pent-up products of obstruction were allowed to flow to the aboral portion of the intestine. In the second, owing to the patient's condition, it was found necessary to do an ileostomy, and all the obstruction products were drained to the surface. As I have already stated, it is by no means proved that because such products are toxic to other individuals, they are necessarily toxic to their host.

CONCLUSIONS

The power of the liver to pair camphor and glycuronic acid is probably seriously impaired after duodenal obstruction. This can be studied by the method of Tollens, which, however, is cumbersome and may be faulty. Such decreased power of camphor-pairing is presumably an evidence of impaired liver function. This, however, is not reflected in the histologic appearance, either grossly or microscopically.

The decrease in the water-content of the tissues in duodenal obstruction is about the same as obtains after salivation by pilocarpin for four days or after fasting for seven. As this decrease produced no visible change in either case before euthanasia, it is reasonable to believe that it produces none in intestinal obstruction. The loss is 10 per cent.

The toxemia in duodenal obstruction undoubtedly arises from an interference with cellular reactions of the intestinal epithelium. The toxins undoubtedly are at least in part eliminated from the stomach and colon. If small-intestine epithelial cells of healthy animals are placed in the stomach of duodenally obstructed animals, such animals have lived nearly twice as long as not-fed control animals. This evidence is strongly opposed to the bacterial theory of origin of the toxins.

In addition to the placing of jejunal and ileac epithelium in the stomach of postoperative obstruction cases, an emulsion of them should also probably be used in colonic irrigations for the same indication and purpose.

104 East Fortieth Street.

ABSTRACT OF DISCUSSION

DR. JOSEPH COLT BLOODGOOD, Baltimore: Dr. Draper's paper seven years ago made me think clinically about the duodenum in relation to some things we had observed in intestinal obstruction. In the beginning of surgery, we were dominated by anatomy, and then followed pathology. At present surgery does not look for much help to anatomy, except in certain regions, or even to pathology. We have about reached the limit of the microscope, but we do look for help to experimental physiology in the solving of problems which will be met on the operating-table. Perhaps, later, we will look to experimental chemistry. Surgeons, as soon as they conquered technic in surgery of the stomach, found that their patients did not die of peritonitis. As soon as they used anesthesia, the majority of their patients didn't die of shock and later of anemia, but up to the present there is one complication which has always bothered. If they do a posterior gastro-enterostomy a number of their patients vomit

the contents of the duodenum, because of some fault in the technic. Now its contents are washed out with the stomach-tube. These patients in the beginning died because we didn't know how to use the stomach-tube. There was no place for the duodenal contents to flow. They couldn't pass up into the stomach and couldn't come through the anastomosis because it was kinked, and these patients died within from three to five days. When I heard Dr. Draper's paper I couldn't understand some of the mortality after operations of that kind, either of my own or others. Patients going well on the operating-table, apparently absolutely comfortable for twenty-four or thirty-six hours, suddenly going to pieces, with no pumping, no distension or rapid pulsing that show in intestinal obstruction. That is a definite case of death from acute dilatation of the duodenum as a definite postoperative complication. If there is a patent pylorus we can relieve that with a stomach-pump when the pylorus is closed by section. Some surgeons are now advocating that posterior gastro-enterostomy is without an element of danger, and will not be followed by complications in two or three days. The operation has led to chronic dilatation of the duodenum, a terminal condition which is responsible for the ill-health of a group of patients. In the last three years I have recognized and operated on six. In the previous three years I didn't recognize three. The previous three died. The subsequent six have recovered, and the cause of the dilatation I have found has been a congenital defect in the mesentery to the ileum from the appendix and the giant cecum descending into the pelvis, and on account of the short mesentery of the ileum lying directly on the base of the mesentery section, which kinks the small intestine as it passes through to a junction with the duodenum, which produces a chronic dilatation with its symptoms. These patients have been relieved by removing the colon and taking away that heavy part and exposing the transverse colon so that it will do its work and be allowed to expand. This is a demonstration of what experimental physiology can do for practical surgery.

ROVSING'S OPERATION FOR CONGENITAL CYSTIC KIDNEY*

F. B. LUND, M.D.
BOSTON

My first acquaintance with congenital cystic kidney was gained in the dissecting-room of the Boston City Hospital, where I saw several necropsies on patients of middle and adult life who had come into the hospital in convulsions and died of uremia. In some of them, the enlargement of the kidneys had been noticed before death; in others, not, but at the necropsy the kidneys were found very much enlarged and studded with numerous cysts ranging in size from a pea to an orange. These cysts were yellow, green, red and brown, and the effect was very beautiful, making a pathologic specimen truly remarkable and imposing in appearance. These necropsy cases were bilateral, one kidney being considerably more enlarged than the other.

Cases in which gradual enlargement of a polycystic character takes place in middle life, and in which the patients present themselves to the surgeon on account of dragging pain and the presence of a tumor in the loin, or possibly on account of the mobility of the enlarged kidney, may suggest malignant disease of the kidney or hydronephrosis; and it is frequently only when they are cut down on that their true nature is revealed and the importance of a knowledge of their nature and treatment becomes evident. Such kidneys have not infrequently been removed, with the result that the patients have died of uremia, and the necropsy has revealed the presence of cystic disease of the other kidney, usually less advanced, so that a tumor was not felt before operation. A knowledge of the fact that this disease is usually bilateral might have saved the patient's life and the surgeon's discomfiture.

Congenital cystic kidney is a disease which frequently runs its course without a diagnosis being made during life, death occurring from uremia or cerebral hemorrhage due to kidney insufficiency. The real cause of the insufficiency is first discovered at the necropsy, when the enlarged cystic kidneys, not felt during life, are found by the pathologist. In fact, the course of the disease may be so latent that a patient in whom pathologic changes have not been suspected may be stricken with uremia or cerebral hemorrhage.

The term "congenital cystic kidney" does not include hydronephrosis, which is a dilatation primarily of the pelvis and sinus of the kidney from retention due to obstruction in the pelvis itself or the ureter, and does not concern the urinary tubules at all. It does not include cystic dilatation of the tubules due to contraction of the interstitial tissue, which sometimes accompanies a chronic interstitial nephritis. Here the kidney cortex becomes studded with little cysts, seldom larger than a pea, and not, as in the true congenital cystic kidney, greatly enlarged and varying in size. The cysts in interstitial nephritis play only a subordinate rôle and are secondary to scar contraction. Neither does the subject under discussion include the single or occasionally multiple cortical cysts which sometimes result from the absorption of embolic infarcts. In the true cystic kidney the organ may be enormously enlarged; for example, the cases in which they present an obstacle to parturition or in adults occupy half or two-thirds of the abdominal cavity. The whole organ, both cortex and medulla, is studded with cysts of various sizes, from that of a pin-head to that of a mandarin orange. The surface is knobby with cysts of different size and color. The walls of the cysts are thin and vascular, and the larger ones show folds on their inner surface, which are the remains of the walls of the smaller cysts which have coalesced to form them. The fluid consists of urine, since in it are found urinary constituents, such as uric acid and hippuric acid, calcic oxalate, cystin, leucin, and tyrosin; but in the larger cysts, this fluid is considerably diluted (probably by serum which has transuded through the walls), is albuminous, and contains blood, pigment, fat and cholesterin, in varying quantities, so that the color of the cysts may be red, brown or yellow, and the fluid in them more or less cloudy. Between the cysts are often found more or less extensive areas of normal renal parenchyma; other cysts are separated merely by fibrous tissue. In some cases with large amounts of parenchyma, we cannot distinguish between cortex and medulla.

The efferent passages which protrude into the calices or pelvis frequently show no changes, but in some specimens are shrunken and narrowed, and may be almost obliterated. Thin-walled cysts may sometimes be seen projecting into the pelvis, just as they do on the outer surface of the kidney. Microscopically, each cyst is lined with a single layer of flat epithelial cells, which in the smaller cysts correspond closely to a tunica propria. In the larger cysts this is not found, either because they have no proper wall, or because

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