A.—CASES OF PERFORATING DUODENAL ULCER TREATED BY SUTURE.

CASE I.—May 23rd, 1901. Male, aged 41, temperate. No history of previous illness. Sudden pain in the epigastrium at 11 a.m. Applied for relief at 2 p.m. When he was first seen his pulse was 100; pain felt in right side; abdomen moving slightly and somewhat rigid. The symptoms were so ill-defined that he was thought to be suffering from pneumonia. At 12.30 midnight his pulse was 120 and his abdomen distended. I saw the patient for the first time at 2 a.m. and opened his abdomen. A punched-out ulcer was found just beyond the pylorus, from which clear fluid kept welling up.

Result.—Death five hours and a half after the operation. Post-mortem record: Imperfect closure of the perforation.

CASE II.—July 13th, 1901. Male, aged 47, a drunkard. No history of previous illness. Sudden, very severe abdominal pain at 9 a.m., but did not apply for relief until 8.30 p.m. When first seen his pulse was 104; the abdomen was moving during respiration. There was general tenderness, most marked in the epigastric region. Abdomen opened at 10.45 p.m. I found a circular and well-defined perforation at the back of the pylorus. Clear fluid was welling up through it.

Result.—Death eight days later. The patient felt so well until twenty-four hours before his death that he was constantly asking when he might get up. Post-mortem record: Subdiaphragmatic abscess; incomplete closure of the perforation. The ulceration had continued after the sutures had been inserted (? cancerous).

CASE III.—May 18th, 1902. Male, aged 37, temperate. Had complained for the last six months of stomach-ache half an hour after his meals. He had felt a sudden pain at twelve o'clock mid-day, on the right side beneath his ribs. The pain was so severe that he took a cab to the nearest hospital. On admission the pulse was 120, the abdomen was moving freely and equally. I operated at 6 p.m. There was no perforation in the stomach, but a small clean-cut ulcer in the duodenum, from which bile-stained fluid kept pouring out. The aperture was closed, and a counter-incision was made in the right lumbar region, through which a drainage tube was passed.

Result.—Death 35 hours after the rupture. Post-mortem record: The perforation was effectively closed, as it was water-tight.

CASE IV.—July 26th, 1902. Male, aged 26, temperate. Had suffered previous attacks of a similar pain, but none so severe as that which occurred suddenly at 7 p.m. He was admitted to the hospital at 3.50 a.m. on July 27th, but I did not see him until 10 a.m. on this day. His pulse was 112. There was pain over the right iliac region, which was full and tender. The abdomen, except over the tender area, was moving during respiration. I thought he was suffering from an acute inflammation of the appendix, but on opening the abdomen in the iliac region, the appendix was found to be normal. The stomach was intact, but a perforation was discovered on the anterior surface of the duodenum near the pylorus.

Result.—The patient left the hospital cured on September 12th, 1902. I have seen him on more than one occasion in the last year. He is at work, and says (October 20th, 1904) he is able to do his work but has to be careful in his diet.

B.—CASES OF NON-PERFORATING DUODENAL ULCER TREATED BY GASTRO-JEJUNOSTOMY.

CASE V.—Male, aged 31, temperate. Was admitted to the hospital with pain in the lower part of his abdomen and vomiting, which had made him an invalid for two months. He retched after food, and vomited at one time "a quart of dark blood, all in lumps," and at another time "a pint of blood." There was no melaena. On admission he had a point of maximum tenderness in each iliac fossa, and a tender point 1 in. to the right of the umbilicus, which varied somewhat in position. I diagnosed the existence of a duodenal ulcer, and performed a post-colic gastro-jejunostomy on May 19th, 1904.

Result.—The patient made a perfectly uneventful recovery, and left the hospital on June 17th, 1904. In November, 1904, he is improved but not well.

CASE VI.—Male, aged 69, temperate. Admitted to hospital, August 1st, 1902, for constant vomiting; a greatly dilated stomach, and a tumour as large as a Tangerine orange in the neighbourhood of the pylorus. The patient had lost much flesh. I performed a gastro-jejunostomy with McGraw's elastic ligature.

Result.—The patient recovered completely, with increase of weight and cure of dyspepsia. I saw him in July, 1904, a hale and hearty man of 71, just starting to Canada.

CASE VII.—Male, aged 48, temperate. Admitted to hospital on July 31st, 1904, complaining of intense abdominal pain relieved by vomiting. He had lost much flesh during the last few months. On admission his stomach was found to be dilated, there was evidence of slight portal obstruction, but no tumour could be felt in the region of the duodenum. I diagnosed the case as one of duodenal constriction, and performed a retro-colic gastro-jejunostomy on August 1st, 1904. On opening the abdomen the duodenum was found to be constricted to the size of a lead pencil, about an inch beyond the pylorus.

Result.—The patient made an uneventful recovery, and left the hospital on August 19th, 1904. In November he reports himself quite well, returned to his normal weight of 12 st., and walking 10 miles a day in comfort.

THE SPITTING NUISANCE IN BUENOS AIRES.—At a recent meeting of the Municipal Council of Buenos Aires a proposal was submitted by Dr. Conti that owners or tenants of houses and buildings who refused to allow tablets to be put up on the outside forbidding expectoration on the pavements should be fined. The proposal was referred to a committee.

SCIENTIFIC GRANTS COMMITTEE

OF THE

British Medical Association.

REPORT LXXXIX.

THE NATURE OF THE MALFORMATIONS OF THE RECTUM AND UROGENITAL PASSAGES.

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[THIS paper is a brief summary of the clinical side of a paper on the Development and Musculature of the Visceral Openings of the Hind End, accepted by the London University for the Degree of B.Sc., by Research, October, 1903.]

When the details of human embryology have merely an academic interest they are of little utility in a medical education, and serve no useful purpose in the object of all medical training—practical medicine and surgery.

To be of any value at all to the medical man, embryology must help him to understand how, and why, the adult organs become developed, it must furnish him a ready explanation of the abnormalities and imperfections of development with which he meets, and it should afford some guide to him in the treatment he must undertake for their relief. Regarded in this light, much that is taught of the development of the human body must be admitted to be of little practical value in medicine.

In many cases the teachings of embryology furnish no real explanation of the abnormalities that are met with clinically, and in very few do they afford any guide for the artificial repair of these abnormalities.

The generally accepted view of the development of the human hind end offers no explanation of some of the abnormalities which are commonly found clinically in this situation. The growth of septa and partition walls, which is so often invoked—and often wrongly invoked—as an explanation of various abnormal conditions, is founded on a misinterpretation of developmental changes, and, it would be urged, does not here occur, at any rate in the manner and to the extent that is usually described. It is therefore necessary to inquire if the present account of the development of the human hind end is a correct interpretation of the various stages that have been seen in the human embryo, if some stages have been overlooked, or some stages seen in other animals have been wrongly read into the early chapters of human development. That some stages are wrongly interpreted, and some are wrongly described, and that some abnormal conditions are capable of more practical and more clinically useful explanation, will I believe, be found from a further study of the development of the hind end of the human embryo.

In order to test the shortcomings of the present theory, and to estimate any advantages that some modification of it may possess, it is necessary to see, in outline at any rate, what are the problems that require solution. Most of the malformations of the rectum and urogenital passages that are commonly met with fall into a few well-recognized groups, and it is an outstanding fact in regard to these abnormalities that they are wonderfully constant in their anatomical arrangement. Their very constancy demands that their explanation should be definitely furnished by reference to their development. The visceral openings of the hind end are subject to two classes of malformations. In the first class a terminal septum closes the orifice, and in the second class there may be an actual absence of a portion of the tube which should normally open to the outside at one of the visceral orifices. In the first class come such abnormalities as imperforate anus, imperforate hymen, and urethral septum. In the second class come the more grave conditions of imperforate rectum, atresia vaginae, and atresia urethrae.

The extent of the deformity may not be limited to the actual developmental error, but may be increased and altered by secondary changes that this condition produces, and one such example of this more complex deformity is ectopia vesicae.

The anatomical condition of most of these abnormalities is simple; all those which are included in Class I are alike in possessing a terminal septum, and no discussion of their anatomical features is required. Some of those conditions that have been included in Class II, however, require some

further notice, and the common anatomical features of imperforate rectum will be considered in some detail, for it is a condition usually not well described.

When this malformation exists a very definite anatomical abnormality is found; there is an actual loss—or as we shall see a non-development—of a definite portion of the gut. The child may or may not possess a proctodeal depression, and, even if this depression does exist, no pervious gut is found at its bottom, and it is not until some depth has been reached in a dissection from the perineum that the lumen of the rectum is found. Now, it is the rule that this ending of the pervious gut takes place at a definite point just below the peritoneal reflection, in the neighbourhood of Houston's main fold, and it is also commonly found to end in a communication with the urogenital passages in the prostatic urethra in the male and in the vagina in the female.

In Curling's statistics of imperforate rectum 26 per cent. of the cases end in the urethra, and it is likely, as he himself points out, that since the opening is small, were the dissection more carefully performed the percentage would be much higher.

In 9 cases at the London Hospital 7 showed a communication between the termination of the pervious gut and the urethra. This condition of imperforate rectum is not in any way explained by the generally-accepted septal division of the cloaca, and is obviously one which by its frequent occurrence and its constancy should admit of easy explanation.

Fig. 1.—Diagram of the condition of imperforate anus, the type of malformation in which a septum blocks the visceral orifice. The constriction at the peritoneal reflection from the gut is Houston's main fold.

Fig. 2.—Diagram of the condition of imperforate rectum, the type of malformation in which there is failure of development of a portion of the tube. The cloacal opening of the hind gut persists, the post-allantoic gut having failed to develop.

The anatomical conditions presented by atresia vaginae and atresia urethrae require no explanation, and the deformity presented by ectopia vesicae will be considered later.

We may now turn to a brief consideration of the development of the rectum and the urogenital canal.

It must throughout be borne in mind that in the early stages of development the hind end is the site of a very rapid and very peculiar growth. The hind end of the embryo in its early stages is the body stalk—the body stalk in the later stages becomes a part of the umbilical cord; in fact, the umbilicus of the adult is the hind end of the embryo. In picturing the development of these parts, therefore, this very remarkable change must be constantly remembered.

Another predominating feature in the picture of the development of the human hind end is the condition of the allantois. Much that is misleading in human embryology is brought about by the grafting of stages seen in the well-studied development of the chick, into the knowledge gained from the study of the little-known stages of the human embryo. To adopt the condition of a fetal organ, such as the allantois, found in a creature, that carries out its physiology inside an egg-shell, into the life-history of the human embryo, whose early stages are spent so differently, and whose physiological requirements are so different, is only to cause confusion.

The allantois of the human embryo differs widely from the allantois of birds. The allantois of birds is an outgrowth of the hind gut; the allantois of the human embryo (of 12 somites) is the continuation and termination of the hind gut. It is impossible to say at this stage which portion of the tube posterior to the yolk sac should be called allantois and which should be called hind gut; it is impossible to determine where one begins and the other ends. This, then, is an important stage to be noted, and it is one that is entirely overlooked when the idea of a well-developed, well-defined,

ventrally-situated allantois is made the predominant one in the picture.

The hindermost end of the allantois is situated in the body stalk, and is the posterior termination of the hypoblastic canal. The body stalk, as the embryo grows, becomes ventral, the termination of the allantois becomes ventral with it, and so a U-shaped bend is formed in the posterior part of the tube, the dorsal limb, or hind gut, having the mesoblastic somites dorsal to it, the ventrally-bending limb, or allantois, having no mesoblastic somites accompanying it. The apex of the curve that is thus produced in the tube becomes now the site of a dilatation, so that there is produced a posterior chamber, into which opens (1) dorsally and anteriorly, the hind gut, and (2) ventrally and anteriorly, the allantois.

The stage now arrived at is, therefore, the well-known one in which there is a common chamber into which both gut and allantois open.

The terminal dilatation of the U-shaped bend becomes the cloaca, and in the transformations that bring about the change from the cloacal condition to the adult specialized condition some stages must certainly be reconsidered before an account of the development of the hind end will fit in with clinically-observed facts, or give a true picture of the various changes that take place in the embryo.

In reconsidering these transformations the rapid backward growth of the hind end must still be borne in mind. The previously-determined site of the umbilical termination of

Fig. 3.—Condition of hind gut, cloaca, and allantois in the human embryo of 12 somites. Shows the U-shaped bend of the posterior dilatation. The body stalk is still practically the hind end of the embryo.

Fig. 4.—The hind end of the embryo has grown back past the body stalk, and the post-allantoic gut is growing back with the growing hind end. The communication with the anterior portion of the terminal dilatation is still large.

the allantois, formerly the extreme hind end of the embryo, must be kept as a fixed point, and, in addition, a new landmark may be taken. In the stage in which the U-shaped bend occurs, the concavity of the curve marks the posterior limit of the body cavity; in the fetus the posterior limit of the body cavity becomes the recto-vesical pouch; this posterior limit of the body cavity may, therefore, serve as a further guide.

In the commonly-accepted account of the development of the adult specialized condition of the visceral openings of the hind end, the cloacal cavity becomes subdivided by the growth of the septa, and the wide opening becomes thus split into the secondary channels that we find in the fully-developed condition.

The facts of embryology are only gathered by arresting the process of development at various stages, and putting forward the most probable explanation of the means by which the various conditions seen at these different stages were changed the one into the other.

In the process by which one cavity becomes subdivided into two or more secondary cavities, a change which appears to be due to the active growth of a septum or septa may, in reality, be due to an active growth of the cavities themselves, and a passive relative diminution of the openings that connect them. It is probable that in various organs this active growth of cavities and passive growth of septa is the more probable explanation of the process by which the fully developed condition is arrived at; in the formation of the rectum there is, I think, no doubt of it.

The cloaca has, in the stage to which it has been described, two openings in it: the cloacal opening of the hind gut dorsally, the cloacal opening of the allantois ventrally. The cloacal opening of the hind gut has normally, however, a temporary existence only, its connexion with the cloaca becomes lost by the relative diminution in the size of its cloacal opening, and also probably by the lateral infolding of

the cloacal wall that Kiebel has described, and which is seen in a slight degree in the embryo of 12 somites. Meanwhile, the hind end is continuing its backward growth, the growth of the dorsal surface being more rapid than that of the ventral surface. With this growth the hind gut keeps pace, and it buds backwards past its cloacal opening, past its old termination in the allantois, and forms the portion of the hind gut distal to the allantois, which portion I propose to call the post-allantoic gut. It will be seen that this portion of the gut is new. It is the new backgrowth of the hind gut in the rapidly growing hind end of the embryo; and in those animals in which the posterior part of the vertebral column becomes prolonged it of course reaches a very considerable length. The cloacal opening of the hind gut is now normally lost; originally a small opening in the embryo of 12 somites (which is about 2 mm. in total length), the rapid growth of the hind gut, the post-allantoic gut, and of the allantois itself, together with the lateral infolding of the wall described by Kiebel, serve to close the opening of the hind gut into the cloaca.

The hind gut and its continuation—the post-allantoic gut—are now without any communication with the exterior, and when this communication is made, it is at the site of the permanent anus, and is formed by the proctodeum meeting ventrally the post-allantoic gut. The permanent anus is therefore a new opening, forming a communication with the

Fig. 5.—The hind gut has still its cloacal opening. The post-allantoic gut has continued its backward growth. The opening of the Mullerian ducts on the concavity of the U-shaped bend is shown.

Fig. 6.—The hind gut has lost its cloacal opening. The post-allantoic gut has continued its growth, and is about to meet the proctodeal depression.

post-allantoic gut, and is wholly distinct from the cloacal opening of the hind gut. The portion of the post-allantoic gut distal to the ventrally invaginating proctodeum forms the post-anal gut. The posterior portion of the large intestine in the adult therefore contains three embryonic portions: (1) the hind gut, (2) the post-allantoic gut, and (3) the proctodeum. The limitations of these separate embryonic portions must now be determined.

The second landmark taken—that is, the posterior limit of the body cavity or recto-vesical pouch—serves to mark roughly the site of the junction of hind gut and allantois, and subsequently the junction of the hind gut and terminal dilatation or cloaca. The reflection of peritoneum from the anterior surface of the rectum will therefore serve to mark, within at any rate fairly narrow limits, the site of the former cloacal opening of the hind gut, and the site of the origin of the post-allantoic gut. The posterior limit of the post-allantoic gut is the recess formed by the anal sinuses, the proctodeum invaginating the post-allantoic gut in such a manner that its anterior limit becomes the free edge of the anal valves, and, since the proctodeum invaginates the post-allantoic gut from a somewhat ventral position, the anal sinuses on the posterior wall of the gut are deeper than those on the anterior wall (see Fig. 8).

Having thus briefly reviewed the stages of the development of the adult rectum, and having determined the various parts which enter into its formation and fixed their limits, it is necessary to study the abnormal conditions that are found clinically and to make some attempt at their explanation. The conditions that require consideration are these: (1) Imperforate rectum and its varieties. (2) Imperforate anus and its varieties. Imperforation of the rectum is a condition the treatment of which is difficult and unsatisfactory, and part of the difficulty no doubt arises from the generally present idea that if the gut is not found ending at the proctodeal depression, then it may end anywhere. As a matter of fact

the ending of the gut is, in the great majority of cases, situated at a definite point, and this for the reason that in imperforate rectum a definite embryological portion of the gut is missing—the missing portion being the post-allantoic gut.

From what has been said with regard to the limits of this portion of the gut, it will be seen that the termination of the pervious gut should be somewhere near the peritoneal reflection from its anterior surface—this is, in the region of Houston's main fold—and here it usually occurs. Again, from what has been said as to the cloacal opening of the hind gut, it will be seen that should this opening persist (and it frequently does persist in these cases) it should be situated at a definite point—that is, the peritoneal reflection to the allantois—and here in the region of the prostatic urethra it usually occurs in the male. The constancy of the opening between the imperforate rectum and the urinary passages in the neighbourhood of the prostatic urethra, is very striking in the male. In the female the opening is into the vagina, and the level of its opening is frequently just below the cervix; it may, however, be lower.

The alteration in the level of the opening in the female, admits of easy explanation, which will be considered when the development of the vagina is discussed. In some cases in which the rectum is imperforate—that is, in which the post-allantoic gut has not grown back in the growing hind

Fig. 7.—The urogenital sinus and post-allantoic gut have both opened to the outside. The urogenital sinus opening of the Mullerian ducts is still retained.

Fig. 8.—The Mullerian ducts have lost their urogenital sinus opening, and the new solid vagina has grown down. This solid vagina later becomes canalized. It is this stage that persists as atresia vaginae.

end—the cloacal opening does not persist. In these cases, however, the old connexion of the hind gut and allantois will be found on dissection as a fibrous cord binding the rectum to the prostatic urethra in the male, or the posterior vaginal wall in the female.

It will therefore be seen that in those cases in which the gut does not end at the proctodeal depression, its disposition within the child's pelvis is, as a rule, such a one as admits of deliberate and ordered search, via the perineum, with a good chance of success. And further, as the cloacal opening of the hind gut is distal to the posterior limit of the body coelom, the pervious gut will be found posterior to peritoneal cavity. Imperforate rectum admitted of no explanation by a reasonable interpretation of a septal subdivision of a wide open cloaca; nor did that theory in any way account for the communications that are so commonly found between the rectum and the urethra and vagina (Fig. 6).

On the other hand, the explanation of imperforate anus on the present lines differs in no way from the generally-accepted account, with the exception that the varying thicknesses of anal septum which are found in practice are perhaps easier to explain when it is known that both cavities—the post anal gut and the proctodeum—are actively growing.

This modification of the description of the development of the human rectum must of course necessitate some alteration in the account of the processes by which the other visceral openings are formed. It therefore becomes necessary to consider the development of the urinary and genital passages, and to see what explanation it will afford of the many abnormalities that are met with clinically. That the clinical observations fall in line with the changes to be described in the development of the other visceral orifices, which this modification of the formation and differentiation of the cloaca demands, is some argument for their utility. Since it is seen that there is in the adult rectum no real part of the

cloaca, it becomes necessary to inquire what becomes of the remains of this embryonic cavity in the adult. The hind gut, as has been seen, early loses its connexion with the cloaca, but the other cloacal orifice—that is, the allantoic opening—persists; and at this stage the allantois, now enlarged, opens by its original opening into the cloacal chamber. This chamber elongates with the growth of the hind gut end, and now represents that passage, the urogenital sinus, which is usually described as being the anterior part of the cloaca, split off from the posterior part by the down growth of the septum. Since, as we have seen, there is no growth of the septum—but a growth of a cavity (the post-allantoic gut)—it is obvious that the urogenital sinus is the true remnant of the cloaca—the cloaca of the embryo becomes the urethra (Fig. 7).

In the generally-accepted account of the development and fate of the urogenital sinus a further septal growth is called in, in the female, to split the single chamber into two chambers—the urethral and vaginal.

The common abnormalities of the vagina receive no explanation from embryology if this septal downgrowth be accepted as the means by which the urethra and vagina are formed. No true conception of the formation of the channels is gained from this theory, and no hint is given of the pathology of many conditions of urethra and vagina that are capable of rational and helpful explanation. Now there is not the slightest doubt that no septum grows down to separate the vagina from the urethra in the human embryo. The Mullerian ducts open on the concavity of the U-shape bend of the terminal dilatation (cloaca), the opening being situated between the cloacal opening of the hind gut behind and the cloacal opening of the allantois in front (see Fig. 5).

The site of the opening explains their occasional malformation that is presented in such cases as those described by Evanson and Maunsell and others, in which the uterus opens into the rectum. Again, since this is the site of their opening in the embryo, and since in the male they retain this embryonic position, it is not surprising that when the post-allantoic gut fails to develop and the hind gut retains its cloacal—or, as we have now seen, its urethral—opening, this opening should be found in the immediate neighbourhood of the remnants of the Mullerian ducts—that is, in the prostatic urethra (see Fig. 1).

This embryological site of the opening of the Mullerian ducts is not, however, retained by the female for long, and it is necessary to follow the steps by which the adult female condition is arrived at.

The cloacal opening of the hind gut is lost, the post-allantoic gut has grown back, and now the cloaca is the urogenital sinus. Into this sinus open, in front, the bladder; behind, the Mullerian ducts; both openings being situated at the upper end of the sinus. The Mullerian ducts now migrate towards the posterior surface of the body, and this migration is the most striking embryological process in the whole of the transformation of the hind end.

It is customary to describe and picture the fetal vagina as an open tube having very much the characters of the vagina of the adult—that is to say, it is a canal separated by a septum in front from another canal. This idea of a wide open vagina, formed by the growth of a discrete septum, is the cause of the failure of embryology to explain the most common abnormalities, and is also the cause of ascribing to infantile fevers and septic conditions of childhood the production of abnormalities, which really admit of simple and clinically useful explanation by a study of the development of the part.

The vagina is for a great part of fetal life a solid rod, and not an open canal at all. The Mullerian opening into the urogenital sinus becomes early lost, and its communication with the exterior is only made again late in fetal life.

The whole subject of the development of the vagina is exceedingly complex, and it would be better to reserve for some future time any discussion as to the precise morphological elements that take part in its strange growth. The developmental process involved is, however, clear: Early in the history of the embryo the Mullerian ducts open into the urogenital sinus at its upper part; late in its history they open at the hind end by the vagina, and for a considerable interval they have no opening at all—the old one being lost and the new one not yet formed. No septal division is employed in this change, but as the hind gut, when its cloacal opening is lost, re-establishes communication with the exterior by a new

downgrowth, so the Mullerian ducts, when their cloacal opening becomes obliterated, tunnel a new passage to the hind end.

The active agents in this strange growth are two epithelial masses that have been described by Berry Hart as the Wolffian bulbs, but to give this name to them is to give a definite idea as to their origin, and this seems to be by no means clear.

The active migration of the Mullerian opening is not an unparalleled fact in the animal kingdom, as Dr. Arthur Keith has pointed out to me; for James Hill has shown that in *peramele* the median vagina is formed at delivery by an active rupture of the Mullerian ducts into the urogenital sinus.

The actual significance of this extraordinary process does not, however, concern us here; but the fact that the vagina is formed as a solid structure, that becomes canalized only late in development, is of extreme practical importance. Without going further into the development of the vagina, it will be seen that such statements as "congenital atresia in the sense of a *vitium primae formationis* does not occur. The embryological development of the vagina furnishes no explanation to transverse vaginal septa" (Henroten and Harris), are not correct when the development of the vagina is rightly understood.

The active downgrowth of the vagina will now serve to explain the varied levels at which, in imperforate rectum, the cloacal opening of the hind gut is found, and it will also serve to explain why—as is the usual case—it is a larger opening than is the persistent cloacal opening of the hind gut in the male.

One word may here be said as to the development of the hymen. The vagina is an active downgrowth like the post-allantoic gut, and the amount of tissue that is left solid between the downgrowth and the cavity, towards which it grows, is the hymen; it is not formed by Berry Hart's "Wolffian bulbs," but is the tissue these bulbs do not penetrate. Its varying thicknesses can, therefore, be easily understood (Fig. 8).

It will be seen from this account that the urethra of the adult is the real representative of the cloaca; but some additional points in its development must be noted, inasmuch as they have a clinical interest.

The female urethra is the cloacal remnant in its simplest form, but it does not arrive at its adult condition by any means in the way that it is usually supposed to do, by remaining throughout fetal life as a simple tube. The wonderful growth that brings about the migration of the Mullerian ducts has its effects also on the urethra. The human urethra in the female is, for a time at any rate, obliterated, more or less completely, by the proliferation of the vaginal bulbs.

In serial transverse sections and in longitudinal sections of the urethra and vagina at this stage a curious condition is seen. The urethra becomes occluded, and appears to be regenerated by active epithelium in very much the same way that the vagina is. The vaginal rods give off, in addition, lateral buds similar to, though smaller than, the vaginal bulbs; and so in serial sections many islands of epithelial cells are seen stranded in mesoblastic tissue, and entirely isolated from vagina or urethra. That these active epithelial inclusions may be the starting-point of vaginal and urethral cysts seems not at all unlikely.

The male urethra as far as the bulb is the analogue of the entire female urethra, for it, too, is the cloacal remnant. The penile urethra is formed by closure of the genital folds and by central desquamation of the invading plug of cells, as Dr. Berry Hart has demonstrated, and it is for some time during development also blocked.

A word may be here said with regard to *ectopia vesicae*. It has been seen that during embryonic and fetal life the urethra in both sexes has a stage in which it is imperforate, and this stage may persist as a malformation. The pathology of *ectopia* is, it is here maintained, simply the bursting of the bladder and urethra from the internal pressure. Not one of the various theories put forward to explain the formation of this condition is satisfactory. The theories of Bartels, Perls, Ahlfeld, Roglans, and Reichel may be put on one side, and there remain (1) the theory that it is due to rupture of the cloacal membrane, and (2) the theory that it is due to failure of the two sides of the primitive streak to meet in the middle line. Neither of these two last theories will fit in with the common features of the abnormality. If the cloacal membrane or the primitive streak be the site of the

defect, more abnormality than that constantly found should be present. There is often a scrotum. This could not occur if the primitive streak or the cloacal membrane were open. There is always a perineum, often a perfect anus, and usually a normal vagina; none of these should be present should these theories be true, for by the curious backgrowth of the hind end the primitive streak in the adult is represented by the line from a point just below the umbilicus to a point behind the anus, and all structures on this line should be cleft. The reasons why this condition is here considered to be a burst urinary canal are as follows: Urine is secreted by the fetus in utero, and when the urethra is imperforate (when the normal imperforation persists) all stages of distended bladder have been found. The stage of distended bladder, of which there are many cases on record, being to ectopia vesicae as hydrocephaly is to anencephaly, or as syringomyelocoele is to spina bifida. In this connexion the case of a woman in the London Hospital Maternity Charity may be mentioned, who at three successive labours gave birth to fetuses, of which the first suffered from anencephaly, the second from spina bifida, and the third from ectopia vesicae, the cause here being probably an abnormality of fetal secretion.

Again, when there is absence of kidneys or ureters, or there is an umbilical fistula, fetuses have been found with persistent urethral occlusion, and in well-recorded cases of ectopia vesicae an actual occlusion of the cleft urethra is reported. Signs of the distension stage are seen in cases of ectopia vesicae in the condition of the pelvis, which is exactly that which would be produced by internal force driving the symphysis pubis asunder, and splaying out the entire pelvis. There is no loss of middle-line development, but the two sides of the pelvis have been pushed aside from within, and the recti abdominis forced aside with them.

The view here briefly put forward as to the development of the visceral openings of the human hind end differs in many ways from the view that is generally held; but it is maintained that when the idea that these canals are new downgrowths, and not smaller subdivisions of wide open cavities, is rightly considered, it gives a truer picture of what takes place, and a better explanation of the abnormalities that are found in practice.

A stage of imperforation normally occurs in the developmental history of all the posterior vesical orifices; in all of these orifices the imperforation stage may persist as an abnormality. It is not uncommon for this condition to affect more than one orifice in one of these cases.

When the imperforation persists in the different canals varying results are produced. The persistence of the block in the urethra is the rarest condition, as a fetal use is made of the canal, for it is recognized¹ that the kidneys are active during fetal life. When this block persists various abnormalities are met with—enormously distended bladder, umbilical fistula, epispadias, or ectopia vesicae—results caused by the different internal strains produced, and the different yielding points in the urinary canal; or, again, the kidneys may not develop and there may be no distension, but the block remains.

In the rectum the terminal block is more common, as the canal is not normally used till at, or shortly after, birth, and when it persists imperforate anus is produced.

In the vagina the persistence of the terminal block is normal, as the canal is of no functional use till later life, and here its persistence produces the hymen, or its abnormal persistence the imperforate hymen.

These conditions explain one set of clinically well-recognized abnormalities; the failure of the downgrowing portion to be developed at all explains another set. Atresia vaginae is the consequence of the failure of development of the vaginal downgrowth to occur, or to become canalized as has been described; imperforate rectum is the consequence of the failure of the development of the post-allantoic gut.

It is by no means uncommon to find these abnormalities combined—atresia vaginae, imperforate rectum, and ectopia vaginae—in different combinations; and it is claimed as an argument in favour of the account given of the pathology of ectopia vesicae that it should occur in cases where, far from the visceral orifices being too widely open, they are abnormally closed.

[Towards the expenses of this investigation a grant has been received from the British Medical Association.]

REFERENCE.

¹ Ballantyne, *Antenatal Pathology*

THE GERM CELL THEORY OF CANCER.*

By ALBERT S. GRÜNBAUM, M.A., M.D., F.R.C.P.,

Professor of Pathology, University of Leeds; formerly Director of the Liverpool Cancer Research.

THE explanation of the genesis of malignant growths has been brought nearer to us by two notable investigations. They are (1) the proof of the morphological continuity of the germ cells; and (2) the discovery of heterotype mitosis in malignant tumours. They corroborate and fortify each other in a remarkable way.

Until recently it was always taught that the sexual cells arose in the embryo from a layer of peritoneal epithelium, known, it is true, as the germinal layer, but not otherwise distinguished in origin from the remaining cells of the embryo. It is to the credit of Dr. James Beard to have definitely disproved this mode of formation, and to have clearly shown the morphological continuity of the germ cells. He has combined with this the suggestive hypothesis of an antithetic alternation of generations also in man.

Put very briefly this may be stated as follows: After the union of the sperm and the egg there is formed by the subdivision of the united cell what corresponds to a larva, or trophoblast; in man the chorion. From one of its cells there is formed, as it were, a number of spores. One of these spores ultimately becomes the embryo, which includes within itself the remaining spores to become its sexual cells, which are thus not formed from the embryo, but are handed down to it, and for which it merely forms a home.

In passing to their proper position in the embryo these germ cells may go astray, and may in some animals be demonstrated for a time in all sorts of abnormal positions. I believe that Dr. Beard was also the first to suggest that the aberrant germ cell might be the origin of a cancerous growth, although he now prefers to consider it an identical twin, namely, an embryo developed, or rather undeveloped, from the same germ cell. I prefer the former idea.

But what proof is there of any connexion between a germ cell and a malignant growth? This has been supplied by the discovery of Professor J. B. Farmer with Messrs. Moore and Walker. They found in malignant tumours a form of nuclear division which normally occurs only in the sexual cells, amongst the spermatozoa and ova. One of the distinguishing features of this heterotype mitosis is the formation by the dividing cells of only half the normal number of chromosomes. Both this and the other characteristics were observed in the cells of malignant growths; there must therefore be something in common between the germ cell and the tumour cell.

In innocent tumours they were unable to find this form of nuclear division, and considered its absence an essential distinction between the two kinds of growth. In my opinion it corroborates a view which I have long held, that the distinction between innocent and malignant tumours is not fundamental. We know that clinically one form may pass into the other.

The heterotype mitosis does not supervene at once in the evolution of the sexual cells. In the testis it is not before the third division, and in the ovary when the membrana granulosa begins to proliferate, and the discus proligerus is formed; definite proof of their non-somatic origin. In the same way, heterotype mitosis may not occur at first in tumours, and during this stage they are not malignant, but when it supervenes they become so. Why this change should happen is another matter; but it is no more mysterious in the case of a tumour than in that of the testis or ovary.

Given the cell or cells of the potential growth, it seems not improbable that the toxin of a parasite, the short stimulus of a trauma, the long-continued stimulus of chronic irritation, or the chemical conditions of disordered metabolism, might suffice to let loose their energies, and thus one or all the alleged causes in turn have their share.

The signpost of further investigation of the problem points along the road of embryological and cytological inquiry.

* Abstract of a Paper read before the Leeds and West Riding Medico-Chirurgical Society on November 18th, 1904.

DR. E. P. DICKIN was on December 5th elected Deputy Mayor of the Cinque Port Liberty of Brightlingsea for the ensuing year, *vice* Mr. J. Bateman, J.P., D.L.

In the estimates for 1905 recently submitted to the German Reichstag there is an item of £10,000 for the prevention of typhoid fever.