for the next week, but then gradually developed mental symptoms and four weeks after operation she presented a clear picture of cerebral thrombosis with monoplegia. Died on Nov. 5. Necropsy showed bronchopneumonia, cloudy swelling of the myocardium, and cerebellar softening.

2. *Female, aged* 59.—Transferred from another unit on Oct. 25, 1948, with thyrotoxicosis and congestive heart failure. Found to be hypertensive, with a severe degree of pulmonary congestion and lumbar œdema. Treated along conservative lines initially, but her condition gradually deteriorated, and it was decided to carry out stage operations. On Nov. 1, the superior thyroid vessels were ligated under local anæsthesia. Condition continued to deteriorate and she died on Nov. 3. Post-mortem showed a large retrosternal goitre displacing the œsophagus, dilated right heart, hypertrophy of left ventricle, arteriosclerotic coronaries, fatty liver, and small arteriosclerotic kidneys. (A good case could be argued for not including this patient in the series.)

3. *Male, aged* 60.—A 16-st. man with a huge goitre present for over forty years. Recently, dyspnœa, hoarseness, and increasing pressure symptoms. B.P. 190/110. Thyroidectomy on June 2, 1948, when a nodular gland weighing 868 g. was removed. Did not regain consciousness and died five hours after operation. Necropsy revealed a grossly enlarged and fatty heart.

4. *Female, aged* 70.—Oct. 26, 1948, removal of a large bosselated goitre. Up on the sixth day. Died suddenly on the twelfth day. At post-mortem a large pulmonary embolus and advanced coronary disease was found.

5. *Female, aged* 36.—Thyroidectomy at another hospital in 1945. Recurrence three years later, with accompanying signs and symptoms of thyrotoxicosis. Had been subject to asthma since the age of 3. She had a small deformed chest with poor movement and air entry. On April 27, 1949, difficult thyroidectomy. She appeared to have no reserve whatever and gradually went downhill, dying on the fifth post-operative day. Post-mortem showed bronchopneumonia and congestive cardiac failure.

It is perhaps not out of place to conclude by adding that since the last-mentioned mortality, there have been some 900 thyroidectomies (non-malignant), with but 1 death.

SUMMARY

1. One thousand unselected consecutive thyroidectomies are reviewed.

2. The low morbidity and mortality rates vindicate this unit's tenet that, with occasional exceptions, surgery remains the treatment of choice of goitre. It is freely admitted that the advent of radio-active iodine may modify this view in relation to the toxic goitre. The elderly patient with myocardial damage, little cardiac reserve, and a goitre that would be difficult to remove is now being considered for treatment with I^{131}, provided there are no severe pressure symptoms which would make surgery obligatory. Another category which merits consideration for I^{131} therapy is the recurrent case in which operative difficulties are anticipated.

3. The operability rate is over 99 per cent.

4. Principles in technique are suggested which, it is submitted, will reduce the incidence of recurrences to negligible proportions.

5. Figures are produced which indicate a high incidence of cardiac damage in goitre.

My thanks are due to Mr. J. E. Piercy and Dr. Raymond Greene for advice and helpful criticism in the preparation of this paper, and for permission to use the information in the case papers. Finally, acknowledgement to my wife for help in the collection and analysis of the figures.

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CONGENITAL DEFECTS OF THE ANTERIOR ABDOMINAL WALL *

BY GEORGE M. WYBURN

THE proper development of the embryo depends on the interaction of two forces: (*a*) the genetic mechanism inherent in the fertilized ovum; and (*b*) the influence of environmental factors such as nutrition, protection, and a suitable hormonal medium. A fault in the initial genetic endowment, if not lethal, can be responsible for the perpetuation of hereditary defects. Congenital anomalies, on the other hand, are usually due to a flaw, localized in space and time, in some aspect of the ontogenetic pattern. The causal factor here is more probably some adverse environmental influence effective on a particular developmental process and cell group. Congenital anomalies can be classified broadly as resulting from (*a*) the localized arrest of normal development—these represent the persistence of an embryonic condition and can properly be termed congenital defects; (*b*) abnormal development of a particular region, for example, teratological formations and accessory structures.

Anomalies of the anterior abdominal wall are, for the most part, congenital defects due to some interruption in the sequence of events affecting one particular phase of its development, and their classification requires a knowledge of the embryology of the ventral body wall. In pre-somite embryos with commencing head and tail folds, the ventral aspect is occupied by the wide communication of the primitive gut with the yolk-sac and, more caudally, by the mesodermal tissue of the body stalk with the allantoic diverticulum and developing blood-vessels. These together form the 'belly stalk'. At this

* Being the Arris and Gale lecture delivered at the Royal College of Surgeons of England, on June 10, 1952.

stage the tail fold includes the primitive streak and what remains of the cloacal membrane (*Fig.* 638). At a later stage the ventral aspect is formed by the

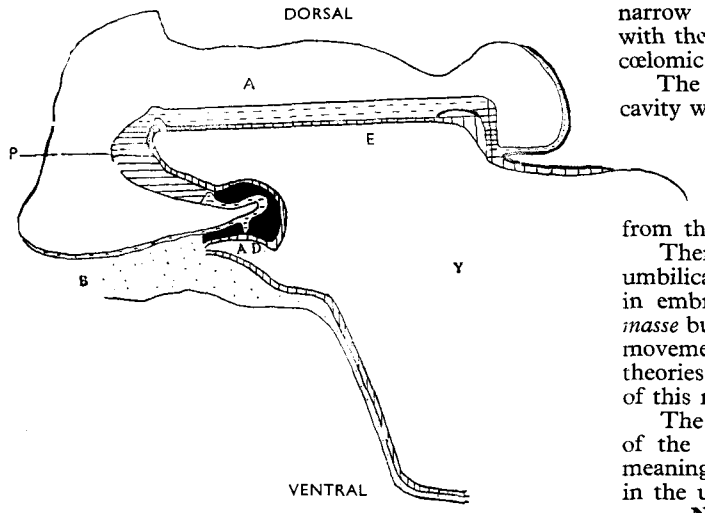

FIG. 638—Reconstruction of median sagittal section of 1·4-mm. embryo. (× 50.) A, Amniotic cavity; B, Body stalk; E, Embryonic endoderm; P, Primitive streak; Y, Yolk-sac; AD, Allantoic diverticulum.
(*Figs.* 638–644 and 647, 648 reproduced by kind permission from the '*Journal of Anatomy*'.)

ectodermal surface of the involuted pericardium, and caudal to this is the 'belly stalk' area encircled by the attachment of the amnion. This includes the still broad yolk-sac connexion and the body stalk proper with the allantois. In embryos of from 4 to 5 mm., the belly stalk is enclosed in a shallow funnel of amnion lined by thickened mesoderm. The yolk-sac connexion is now constricted to form the vitelline duct. The 'funnel' is part of and opens into the exocœlom (*Fig.* 639). A short length of umbilical cord is present in embryos of ± 7 mm. (*Fig.* 640). The tissue basis of the cord is the proliferated mesodermal lining of the enclosing amnion, and it contains the allantoic vessels, allantoic diverticulum, and part of the exocœlom left within the completed cord—the umbilical cord cœlom. This consists of a wide proximal portion which opens into the embryonic cœlom and a distal slit-like prolongation. About this time the narrowing vitelline duct becomes obliterated and finally severs its connexion with the embryonic gut. The embryonic root of the umbilical cord is sunk into the body of the embryo and as the loops of midgut develop in the ventral part of the still shallow body cavity of the embryo, they almost inevitably fall into the umbilical cord cœlom. With the growth and extension of the cord, the umbilical cord cœlom increases in size by the confluence of spaces in the cord mesenchyme until it attains its maximum size in embryos around 30 to 40 mm. By this time it contains most of the embryonic gut, which thus forms a physiological hernia (*Fig.* 641). This physiological hernia is a concomitant of the adaptive processes concerned in the evolution of the viviparous from the more primitive oviparous development of the fertilized egg.

The mesoderm at the embryonic end of the umbilical cord commences to thicken in embryos of 16 mm., and thereafter forms a ring of dense connective tissue surrounding the now relatively narrow communication of umbilical cord cœlom with the embryonic cœlom (*Fig.* 642)—the so-called cœlomic portal.

The gut is taken into the embryonic abdominal cavity when the embryo is about 42 mm. (*Fig.* 643), after which there is a gradual obliteration of the umbilical cord cœlom by proliferation of adjacent mesoderm, and in embryos of 60 mm. the body cavity is sealed off from the umbilical cord cœlom (*Fig.* 644).

There is comparatively rapid passage of the umbilical loop of intestine into the abdominal cavity in embryos of ± 40 mm. It does not do so *en masse* but slides back in regular order in a continuous movement—the cæcum last. There are many theories and suggestions regarding the mechanism of this reduction—none of them wholly satisfactory.

The terms used to denote the congenital anomalies of the abdominal wall should have embryological meaning, and the following are suggested for defects in the umbilical region.

a. **Non-formation of the Umbilical Cord** (*Fig.* 645).—In this condition, the major portion of the abdominal wall is occupied by a large thin-walled sac about the size of a hen's egg, which contains most of the abdominal viscera. A stalk or cord arises from the apex of the sac. There is no neck to the sac, which communicates widely with the body cavity of the embryo. This is one variety of exomphalos with, however, the special characteristic of

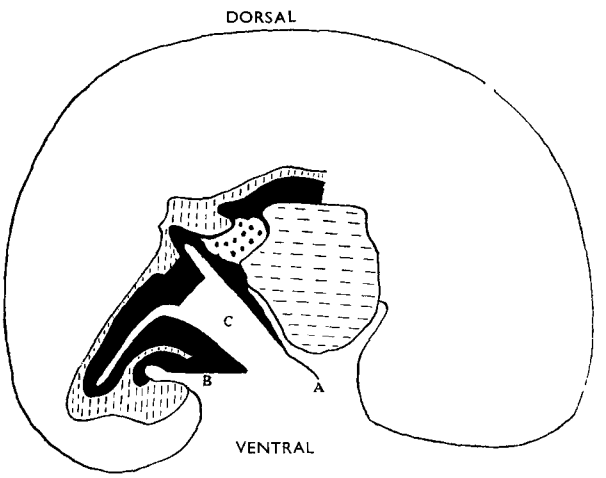

FIG. 639.—Reconstruction of a median sagittal section of 4·5-mm. embryo. (× 25.) Interrupted horizontal lines indicate pericardium; Large dots indicate liver. A, Amnion; B, Body stalk; C, Cœlom. The vitelline duct is not shown.

a supra- and infra-umbilical portion of the abdominal wall above and below the sac, and differing from cases of complete eventration in that there has been some attempt at closure of the belly stalk to form an umbilical cord. The defective development of the umbilical cord may well be related to the absence of the mesodermal condensation which is normally

present at the embryonic attachment of the cord. In consequence, there is no umbilical ring and the cœlomic portal expands *pari passu* with the exocœlom.

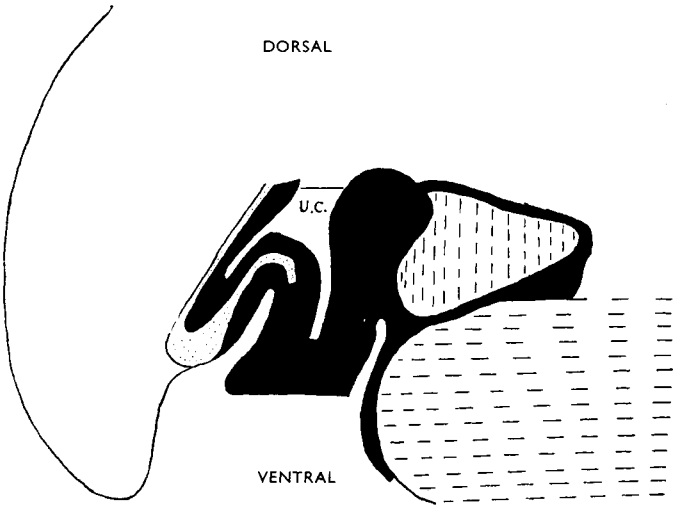

FIG. 640.—Reconstruction of median sagittal section of 7-mm. embryo. (× 25.) UC, Umbilical cord cœlom.

b. **Persistence of the Physiological Hernia** (*Fig.* 646).—Embryologically this is quite a different condition from the preceding one. There is a large sac which contains the major portion of the gut, including the cæcum and the appendix. This sac, however, communicates with the embryonic cœlom by a constricted umbilical ring. The wall of intestine is firmly adherent to the upper rim of the umbilical ring, and it is possible that this adhesion is a primary aetiological factor in some cases of persistent physiological hernia. The umbilical cord develops normally and the condition may have been brought about by a premature narrowing of the cœlomic portal due to a precocious proliferation of the fibrous tissue of the umbilical ring.

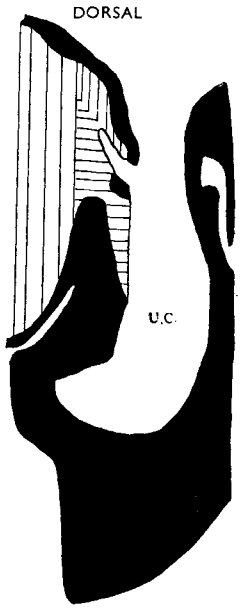

FIG. 642.—Reconstruction of median sagittal section of umbilical region of 16-mm. embryo. (× 13.) UC, Umbilical cord cœlom.

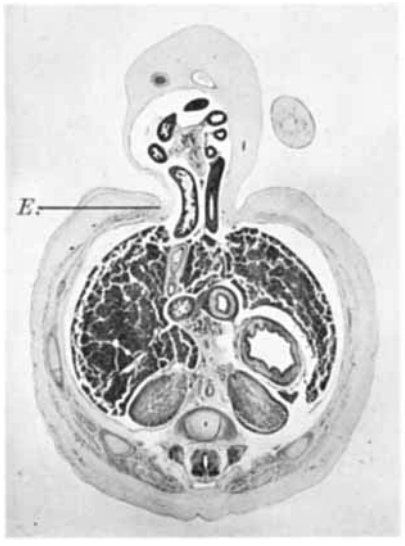

FIG. 641.—Photomicrograph of transverse section of 40-mm. embryo. (× 6.) E, Umbilical ring.

c. **Non-closure of the Umbilical Ring.**—The umbilical ring may remain patent, leaving a communication between the embryonic body cavity and a persistent umbilical cord cœlom, or a new peritoneal sac may be formed. In either case, the entrance into the sac of a loop of the bowel would result in hernia into the umbilical cord, which occurs after the reduction of the physiological hernia. This hernia may be symmetrical or asymmetrical, depending on the position of the allantoic stalk in

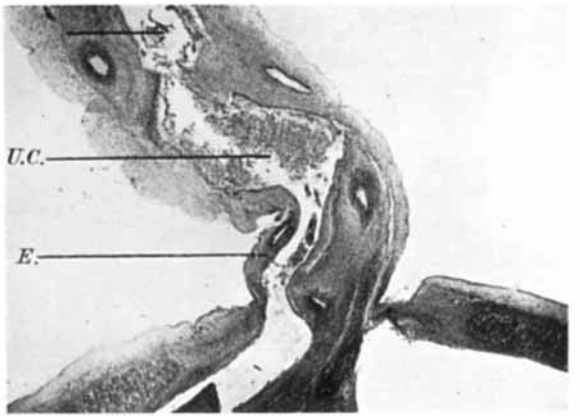

FIG. 643.—Photomicrograph of section of umbilical cord of 42-mm. embryo. (× 75.) E, Umbilical ring; UC, Umbilical cord cœlom.

the cord. Clinically, this is the commonest type of umbilical hernia and constitutes the simple umbilical hernia of Schwalbe and the post-natal

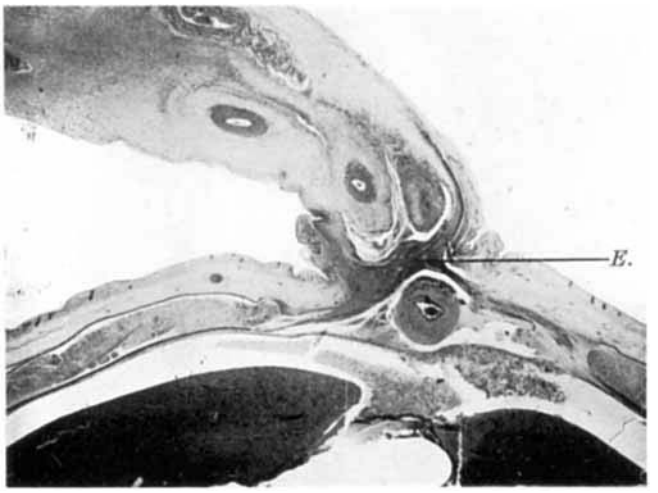

FIG. 644.—Photomicrograph of ventral body wall of 60-mm. embryo. (× 6.) E, Umbilical ring.

umbilical hernia of Fraser. It has also been called 'congenital hernia into the cord' and 'infantile hernia'.

d. **Defects of the Parietes in the Umbilical Region.**—There may be defects in the abdominal wall affecting the linea alba immediately above or below, or to one or other side of, a normal umbilicus. These are due to a defect somewhere in the umbilical ring of compact mesoderm. This leaves a potential hernial site which commonly results in a small asymmetrical hernia to one or other side of an apparently normal umbilical cord. The term 'amniotic hernia', which is in common use to describe such defects, should be discarded and replaced by 'congenital parietal hernia'.

The most significant defects of the infra-umbilical abdominal wall are associated with some degree of ectopia vesicæ or epispadias, the latter merely a minor degree of the former. In early embryos of the pre-somite stage, the cloacal membrane is relatively extensive, reaching from the cloaca up to and along the body stalk, where there is an area of contact between the amniotic ectoderm and the allantoic endoderm (*Fig.* 647). This allantoic cloacal membrane is a human characteristic. Coinciding with the appearance of the first somites, there is a piecemeal obliteration of the allantoic cloacal membrane and, in embryos of 2 to 3 mm., the membrane is limited to the cloaca proper. The obliteration is accomplished by the interpolation of mesoderm between the ectoderm and the endoderm. This mesoderm is derived from wing-like extensions from the hind end of the primitive streak which encircle the cloaca, press in towards the midline, and fuse with the mesoderm of the caudal aspect of the body stalk (*Fig.* 648). This tissue is also the mesodermal basis of the infra-umbilical portion of the abdominal wall, the genital tubercle, the external genitals, the symphysis pubis, and musculature of the bladder (*Fig.* 649). Defective development of this particular mesoderm would therefore leave the allantoic cloacal membrane occupying the area

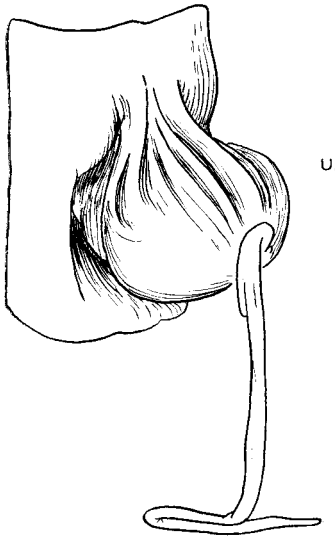

FIG. 645. — Hunterian pathological specimen showing non-formation of umbilical cord.

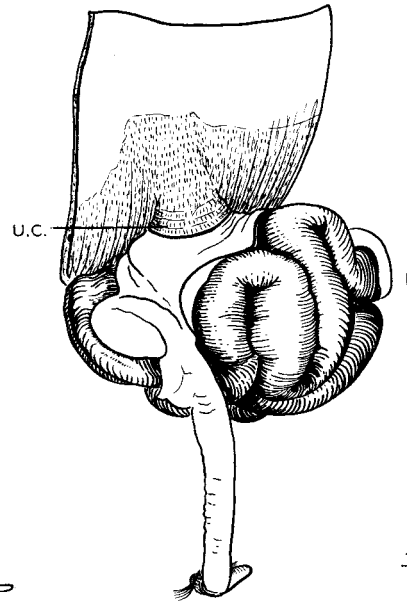

FIG. 646.—Hunterian pathological specimen showing persistence of physiological hernia and presence of umbilical ring (UC).

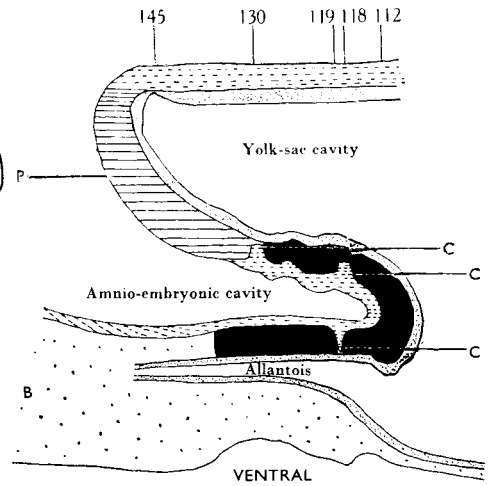

FIG. 647.—Reconstruction of median sagittal section of tail fold of 1·4-mm. embryo. (× 100.) B, Body stalk; C, Cloacal membrane; P, Primitive streak.

between the attachment of the body stalk and the cloaca (*Fig.* 650). Any persistent contact of embryonic ectoderm and endoderm inevitably breaks down and, with the absorption of this allantoic cloacal membrane which normally occurs about the second month, the condition of extroversion of the bladder would be present. The mesodermal septum, dividing the cloaca into the ventral and dorsal parts, reaches the surface at the perineum, so that the extroversion is limited to the ventral cloaca and the anal region can develop normally. There is some evidence that extroversion of the bladder is a condition peculiar to man.

These congenital abnormalities of the anterior abdominal wall are all associated with a defect of the midline mesoderm. This midline mesoderm is morphologically independent of the myotomic downgrowths from the paraxial mesoderm and is derived from the marginal mesoderm of the embryonic disk, where there is a fusion of extra-embryonic and primitive streak mesoderm. There is some evidence from comparative embryology that the extra-embryonic contribution to the marginal mesoderm attains its maximum in man in association with the precocious development of the allantois, and this shift from the established pattern of development may be correlated with the greater frequency of congenital defects of this region in man.

Explanations of congenital defects in terms of purely mechanical factors—amniotic bands, abnormal intra-uterine positions, etc.—are now discredited.

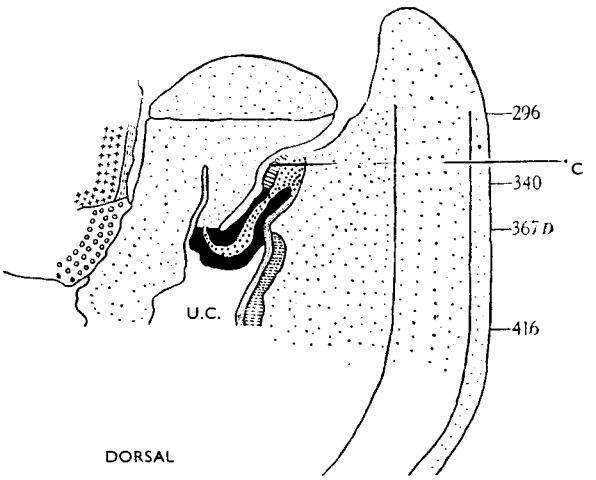

FIG. 648.—Reconstruction of caudal end of 7-mm. embryo. (× 50.) C, Cloacal membrane; UC, Umbilical cord cœlom.

SUMMARY

1. The congenital anomalies of the anterior abdominal wall are classified according to their embryology.

2. Structurally these anomalies could result from defects of the midline mesoderm.

3. In man, the midline mesoderm has significant contributions from the extra-embryonic mesoderm in association with the precocious development of the human allantois.

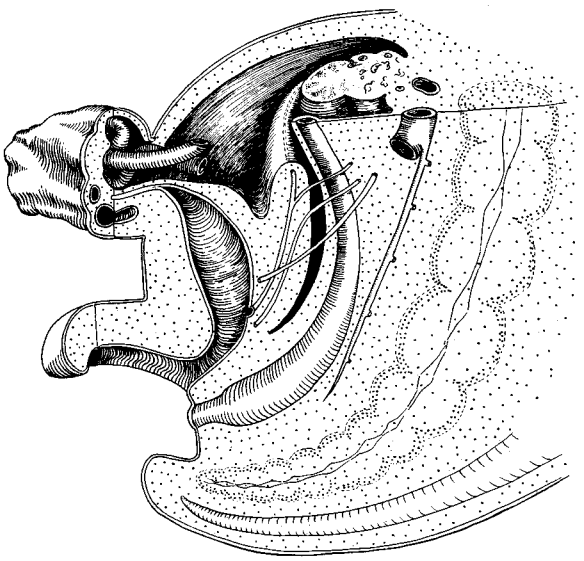

FIG. 649.—Reconstruction of caudal region of normal embryo. (*After Patten and Barry, Amer. J. Anat.*, 1952, **90**, No. 1.)

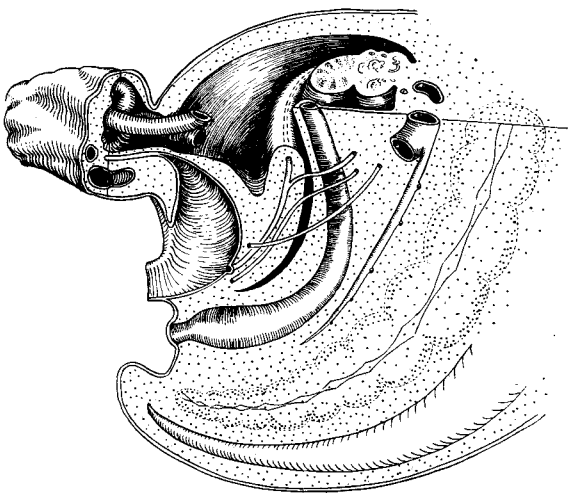

FIG. 650.—Reconstruction of caudal region of embryo showing extroversion of the bladder. (*After Patten and Barry.*)

There is considerable experimental evidence available to show that such environmental factors as nutritional deficiencies, virus infections, hormonal disturbances, may operate at specific times in the gestation period, and thereby interfere with a particular phase of a regional pattern to produce localized defects.

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