THE CIBA COLLECTION OF MEDICAL ILLUSTRATIONS

VOLUME 3

DIGESTIVE SYSTEM

PART II

LOWER DIGESTIVE TRACT

FRANK H. NETTER, M.D.

THE CIBA COLLECTION OF MEDICAL ILLUSTRATIONS

VOLUME 3

A Compilation of Paintings on the
Normal and Pathologic Anatomy of the

DIGESTIVE SYSTEM

PART II

LOWER DIGESTIVE TRACT

Prepared by

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INTRODUCTION

The general outline for THE CIBA COLLECTION OF MEDICAL ILLUSTRATIONS calls for eight to ten volumes, each of which is designed to cover the anatomy, pathology and essential physiologic aspects of one of the various systems of the human organism. When planning the volume on the digestive system, it was decided that, because of its scope, the volume should be divided into three separate parts. Part I was to deal with the upper alimentary tract from the mouth through the duodenum; Part II, with the lower alimentary tract from the jejunum through the anal canal, the abdominal cavity and the fetal development of the gastro-intestinal pathway; Part III, with the liver, biliary tract and pancreas. For various reasons I did not undertake these sequentially but first prepared Part III and then Part I. When these two books were completed, I thought that the most difficult problems were behind me. Consequently, as I began this second part of Volume 3, I felt the relief of a long-distance swimmer who, having battled perseveringly against a strong current, senses the tide turning in his favor and believes that, despite his fatigue, the remainder of the course will be relatively easy. Imagine my chagrin to find, as I "paddled" furiously among the conflicting eddies of knowledge dealing with the lower digestive tract, that the "swimming" here was even more difficult than it had been in the upper alimentary canal.

Fortunately, however, in this portion of the course I had the support of a valiant "team", who had struggled with me through Part I of this volume. In the Introduction to that part, I wrote about the personal pleasure and scientific help received from my contacts with Professor G. A. G. Mitchell of Manchester, England; Dr. John Franklin Huber of Temple University, Philadelphia; Dr. Nicholas A. Michels of Jefferson Medical College, Philadelphia; Professor Gerhard Wolf-Heidegger of the University of Basle, Switzerland; and Dr. William H. Bachrach of the Veterans Administration Center, Los Angeles. In working on this book, my appreciation of these men and of the tremendous help they have given has been multiplied many times.

In addition, I have had the good fortune to make new associations which have proved equally enjoyable and advantageous. Notable in this respect was my collaboration with the São Paulo (Brazil) University Group — Dr. José Fernandes Pontes, his brother Dr. José Thiago Pontes, Dr. Mitja Polak, Dr. Daher E. Cutait and Dr. Virgilio Carvalho Pinto.

To Dr. Polak, in particular, I must express my sincerest appreciation, not only for his work in connection with those plates for which he was specifically the consultant, but also for his coordinating activities on behalf of the entire group. The spirit of cooperation among this group was exemplified by the way other members of the Faculty of Medicine of the University of São Paulo generously contributed of their time and knowledge. Specifically, I must mention Dr. Mario R. Montenegro of the Department of Pathology, Dr. Luis Rey of the Department of Parasitology, Dr. Fernando Teixeira Mendes of the Section of Hematology and Cytology, and Dr. Godofredo Elejalde, Bacteriologist.

From the Brazilian group I learned much about the diseases of the gastro-intestinal tract. I learned also to admire their knowledge and their sound and progressive medical thinking. In particular, I was gratified by their devotion to the project we had in hand and by the assiduity with which they pursued it.

Finally, I am grateful to Dr. Pontes and his associates for their efforts to acquaint me with the many wonderful cultural and social features of Brazil. In particular, its architecture and its music will remain among my most treasured memories.

In preparing those plates concerned with congenital anomalies and those demonstrating the anatomic complexity of the peritoneum, it became strikingly evident that, for a better understanding of these topics, a short review of the essential steps and phases in development would be indispensable. The next problem, naturally, was centered around the question as to how deeply we would have to go into detail to present a coherent narrative of the normal developmental processes and to clarify the deviations which lead to the most frequent congenital anomalies. Dr. E. S. Crelin, thanks to his many years of teaching experience and his acquaintance with the mentality of student and physician alike, knew exactly what and how much embryology we would need in order to provide the basic background for all the topics touching upon intestinal development and its anomalies. It was a rare pleasure to have Dr. Crelin as consultant, not only because of his interest in the task before us and the stimulation he conveys, but also because his critical attitude did not permit the omission of any important detail, in spite of the inevitable condensation.

As the specialty of proctology developed during the past few decades, it became important to obtain a more exact knowledge of the anatomy of the anorectal region. The older anatomic concepts did not suffice either for an understanding of the pathology of the region or for the development of improved operative techniques based on physiologic principles. This led to new investigations of the subject, undertaken by a number of men, largely spearheaded by the group at St. Mark's Hospital in London. In this country, Dr. Rudolph V. Gorsch of New York City was one of the pioneers in this work and is one of the leading students of the subject. His painstaking and meticulous studies were always carried out with an eye to practical application of the knowledge gained. It was through perusal of his publications, particularly of his classic book, *Proctologic Anatomy,* that I came to the conclusion that he was the man who could best help with this subject. This decision proved to be correct, and I enjoyed unraveling with him the most modern concepts of the anorectal regions, the perineopelvic spaces, the sphincters and the various related structures.

In the section dealing with the diseases of the small and large intestine, I encountered topics which, because of their almost totally surgical character, required special handling. For certain of these — volvulus, intussusception and the surgical aspects of ulcerative colitis — Dr. Cuthbert E. Dukes, the distinguished pathologist at St. Mark's Hospital in London, recommended to us the brilliant young surgeon, Dr. H. E. Lockhart-Mummery. His knowledge of the conditions on which we worked is all-encompassing, and his ability to restrict the discussion to its essentials was illuminating.

The enormous progress made in the handling of infants with serious congenital anomalies of the digestive tract similarly required the cooperation of a surgical expert. Dr. C. Everett Koop of The Children's Hospital in Philadelphia has made emergency surgery of the newborn his special field of endeavor. The benefit derived from discussing with him, and preparing under his guidance, the plates which appear at the beginning of Section XII was indeed remarkable, and I can only hope that his clarity in describing the pathophysiologic situations and the essential points of the surgical procedures is adequately reflected in the paintings.

When we came to "hernia", though it is, strictly speaking, a disease of the abdominal wall, the editor and I decided to devote a special section to it, because it is so important and so much of an entity. The consultant for this topic was Dr. Alfred H. Iason, a man who has studied the subject in all its phases, who has written voluminously concerning it, and who has had vast operative experience in the field. His monumental volume, *Hernia,* is widely known.

Intestinal obstruction and the "acute abdomen" proved to

be unique subjects because of the vastness of the fields they encompass. These two topics touch on almost every condition covered in this book, but what was needed was a cross-sectional view, a reclassification of the material in such a manner as to be helpful to the student and practitioner. For aid in these problems I called on a close friend, Dr. Samuel H. Klein of New York City. Because of his vast surgical experience and knowledge, his keen analytical mind and his understanding of the teaching approach, he was ideally suited for the task at hand. In order to get another point of view for the task of abridgment, Dr. Klein called in an associate, Dr. Arthur H. Aufses, Jr. Together, we worked out the plates which appear on pages 188 to 192.

Paroxysmal peritonitis is an entity of relatively recent recognition, and for the plate on this subject I fortunately was able to obtain the collaboration of Dr. Sheppard Siegal of New York City, who had much to do with the identification of this condition.

Gastro-intestinal physiologists and clinicians have for some time suspected that the ileocecal junction acts not purely as a flap valve but as a physiologic sphincter or pylorus. It remained for Dr. Liberato J. A. Di Dio of the University of Minas Gerais, Belo Horizonte, Brazil, to demonstrate a more appropriate concept of the structure of this valve and its function. Dr. Di Dio, who was in New York at the time I was working on this subject with Professor Wolf-Heidegger, most graciously explained to us his findings and showed us drawings and photographs of his dissections and also his remarkable motion picture of the function of the valve in vivo. The illustrations on page 52 are based on his material.

Our concept of the structure of the epithelial cells of the intestine has been greatly modified in recent years, and the help of an expert in this field was needed when it came to the making of the illustration on page 50. This subject is most important today because of the interest in absorption and malabsorption. I am therefore most grateful to Dr. S. L. Palay of the Laboratory of Neuro-anatomical Sciences, National Institutes of Health, Bethesda, Maryland, who has personally made most extensive electron microscopic studies of these cells and who graciously gave me of his time and his knowledge.

A number of illustrations in this volume were originally made in consultation with Dr. Jacob Buckstein of New York. They were first issued in individual brochures and later in THE CIBA COLLECTION OF MEDICAL ILLUSTRATIONS, published in 1948. These plates appear in Section X, Plates 1, 2, 3, 6, 7, 8, 25, 28 and 29; in Section XII, Plates 16, 18, 23, 30, 34, 44, 45, 46 and 49; in Section XIII, Plate 4; and in Section XIV, Plate 11. Some of them are reproduced here in their original form; others with modifications. I wish to thank Dr. Buckstein for his help with these plates.

Also from an older series of pictures stem Plates 9 to 14 in Section XIII, dealing with abdominal injuries. I am most grateful to Dr. Michael E. De Bakey, under whose guidance these pictures were developed in 1945, for his kindness in checking the correctness of both pictures and texts.

I should also like to express appreciation for the generous aid and advice given me by the following: Dr. Robert A. Nordyke, now of the Straub Clinic, Honolulu, Hawaii, for his aid in planning the illustration of the use of radio-isotopes in tests of absorption (Plate 22, Section XI); Dr. Robert J. Matthews of Van Nuys, California, for demonstrating to me the test for occult blood in the stool (Plate 24, Section XI); and Dr. Paul K. McKissock of Veterans Administration Center, Los Angeles, California, for his advice in connection with the sketch of reversal of an intestinal loop (Plates 1 and 2, Section XI).

For the Plates in Sections IX and X, dealing with the blood vessels of the abdominal wall and of the intestine, Dr. Michels and I were helped a great deal by Paul Kornblith, a medical student at Jefferson Medical College, and by Dr. Padmanabhan Siddharth of Madras Medical College, India, presently a teaching fellow at the Daniel Baugh Institute of Anatomy.

Throughout this project there has been to me one source of encouragement and stimulation, one fountainhead of counsel and advice, my very dear friend, the editor of these volumes, Dr. Ernst Oppenheimer, to whom I shall be forever grateful. I cannot here recount the multitudinous ways in which he has helped. Suffice it to say that his devotion to the work, his confidence in me and his tireless attention to organization and detail have been an inspiration.

FRANK H. NETTER, M.D.

* * * * *

The editor wishes to express his special gratitude to the consultants for their fine cooperation and for their meticulous care in writing the texts. The renewal of the pleasant relationships with those consultants who contributed also to Part I of this volume has been most enjoyable, and the ready understanding shown by those who encountered, for the first time, the various difficulties we face in producing these books has been deeply appreciated.

I wish to add my own expression of thanks to Dr. Mitja Polak for his successful efforts to make my task much easier by assuming the rôle of coordinator for the five São Paulo University contributors who, as a group, were responsible for the greatest number of plates.

We are deeply indebted to my good friend, Dr. Samuel R. M. Reynolds, Professor and Head of the Department of Anatomy of the University of Illinois School of Medicine, for supplying the very rare slides of the intestinal plexuses of the human being, which appear in the plate on page 78. To Dr. Jack Crane, formerly of the University of California Medical School, now Professor and Head of the Department of Pathology of the University of Oregon Medical School, we extend our thanks for making the three photomicrographs from the slides supplied by Dr. Reynolds. Our thanks go also to Dr. Leo Kaplan, Director of the Clinical and Anatomic Laboratories of Mount Sinai Hospital in Los Angeles, for the very difficult-to-obtain photomicrographs of human stools (page 108). Dr. Kaplan also kindly supplied us with the photomicrograph, on page 55, depicting the longitudinal section through the colon wall. The photomicrograph of the argentaffine cells (on the same page), as well as information regarding the function of these cells, we owe to Dr. Bernard J. Haverback, Chairman of Gastroenterology Service, University of Southern California School of Medicine.

Many other persons contributed generously of their time and energy to make this volume possible. In particular, I would like to mention Mrs. L. A. Oppenheim and her associates on the CIBA staff in Summit, N. J., and our literary consultants, Wallace and Anne Clark of Buttzville, N. J. Many thanks go also to Harold B. Davison of Embassy Photo Engraving Co., Inc., and to the staff of Colorpress for their devotion and effective support of our desire to improve, with each new book, the high standard we set for ourselves when we embarked on this project.

Finally, to Dr. Hans H. Zinsser, Assistant Clinical Professor of Surgery at the College of Physicians and Surgeons, Columbia University, I would like to express my appreciation and thanks for his cooperation and help in his capacity of associate editor.

E. OPPENHEIMER, M.D.

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Section VIII

DEVELOPMENT OF THE DIGESTIVE TRACT

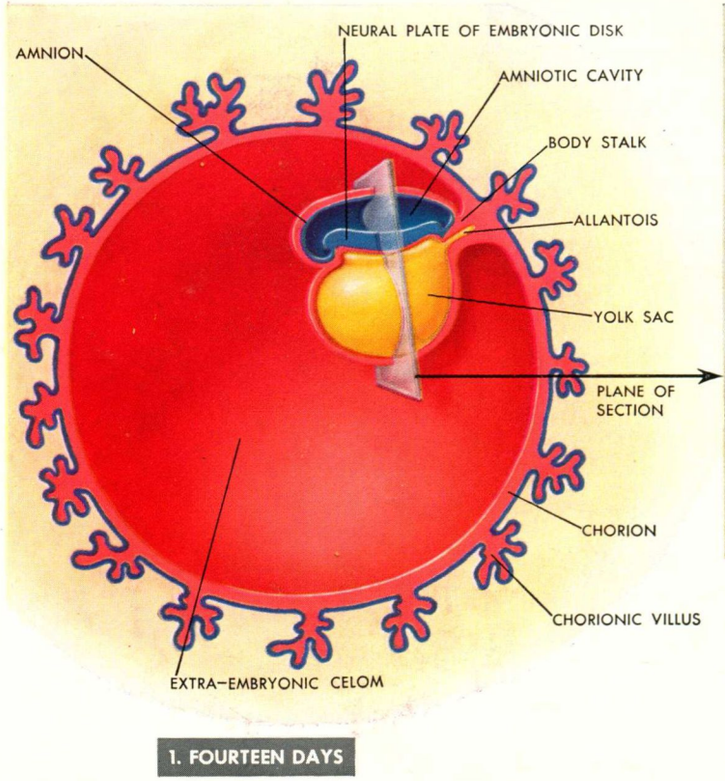
by

FRANK H. NETTER, M.D.

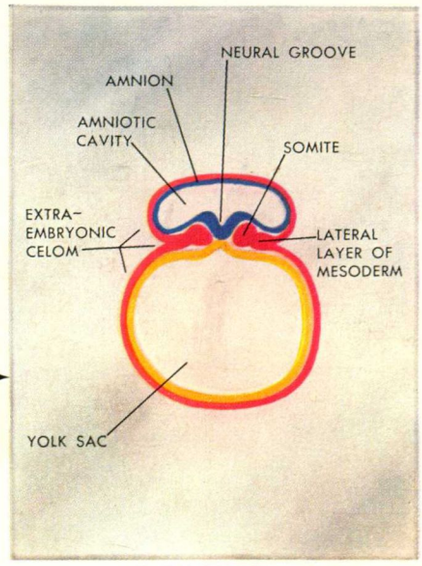
in collaboration with

E. S. CRELIN, Ph.D.

DEVELOPMENT OF GASTRO-INTESTINAL TRACT



1. FOURTEEN DAYS



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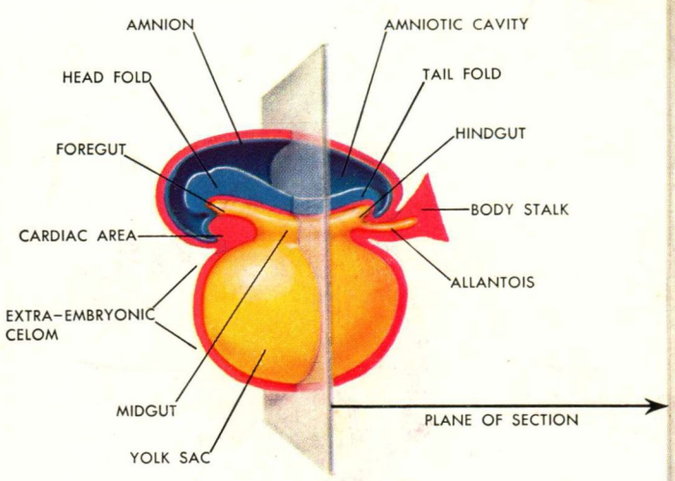

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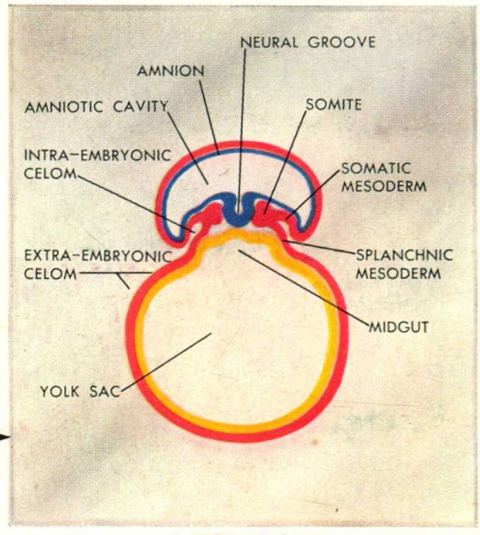

4. SECTION OF 3.

KEY
ENTODERM
MESODERM
ECTODERM

F. Netter M.D.
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The flat entodermal roof of the yolk sac underlying the embryonic disk [1, 2]* becomes incorporated within the human embryo in the form of a tube, the primitive gut, as the embryonic disk folds into a cylindrical embryonic body. The cranial end of the yolk sac roof invaginates into the developing embryonic *head fold* to become the *foregut* [3]. Then the caudal end of the yolk sac roof invaginates into the developing *tail fold* to become the *hindgut*. Another tubular diverticulum of the caudal end of the yolk sac roof, the *allantois*, originally invaginates into the *body stalk* before the hindgut develops [1]. It is drawn into the tail fold along with the hindgut to become a hindgut diverticulum [3, 5]. Within the body of the embryo, the roof of the yolk sac intervening between the fore- and hindguts, the *midgut*, originally has a wide communication with the extra-embryonic portion of the yolk sac [3, 4]. Along the periphery of this midgut and yolk sac communication, the body of the embryo becomes bounded by definite folds, which increase in depth and undercut the embryo, gradually to decrease the size of the midgut and yolk sac communication [6, 7]. Before the communication is ultimately lost, it is reduced to a long, slender tube, the *yolk stalk* [8], passing from the tubular midgut into the umbilical cord [11-13]. The approximation of the body folds forms the ventral body wall and is associated with the formation of the umbilical cord [8, 11, 12].

The blind cranial end of the foregut forms the inner entodermal layer of the *buccopharyngeal membrane*, the outer layer of which is the ectodermal floor of a surface depression in the oral region, the *stomodeum* [5]. Disintegration of this membrane establishes the cranial gut opening [8]. The blind caudal end of the hindgut forms the inner entodermal layer of the cloacal membrane, the outer layer of which is the ectodermal floor of a surface depression in the anal region, the *proctodeum* [5, 8, 11]. Disintegration of the cloacal membrane establishes the caudal gut opening [13].

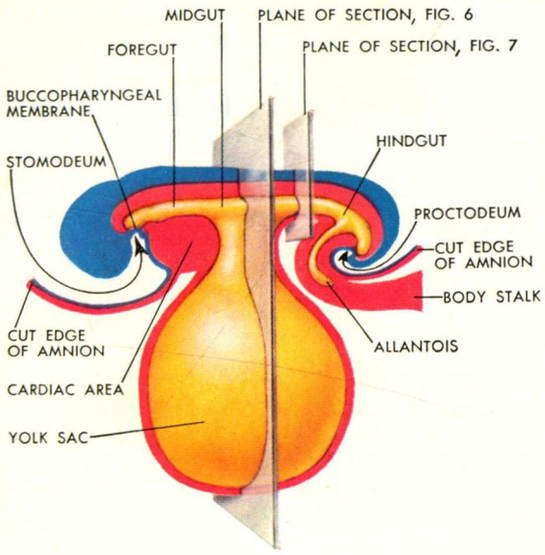
The gut entoderm gives rise to the mucosal lining (and the secretory cells of the glands derived from it) of various structures: *foregut* — pharynx, respiratory tract, esophagus, stomach, first part and upper half of the second part of the duodenum; *midgut* — lower half of the second part and the third and fourth parts of the duodenum, jejunum, ileum, cecum, appendix, ascending colon, right and middle thirds of the transverse colon; *hindgut* — left third of the transverse colon, descending and sigmoid colon, rectum, upper part of the anal canal and a greater part of the urogenital system from its allantoic diverticulum.

Before the primitive gut develops into a tube, a flat layer of mesoderm, continuous with the somites located on each side of the midsagittal plane of the embryonic disk, intervenes between the disk ectoderm and the entodermal yolk sac roof [2]. A split occurs in each of these lateral mesodermal layers throughout their length within the boundaries of the embryonic disk to produce a slitlike cavity, the *intra-embryonic celom* [4]. It communicates with the relatively large chorionic cavity, the *extra-embryonic celom*, beyond the boundaries of the embryonic disk [1, 2, 4]. The intra-embryonic celom becomes the pericardial, pleural and peritoneal cavities. The dorsal sheets of intra-embryonic mesoderm, the somatic (parietal) mesoderm, resulting from the split in the lateral mesodermal layers to form the intra-embryonic celom, become closely associated with the disk ectoderm and ultimately give rise to the parietal peritoneum of the abdominal cavity [4, 6, 7, 9, 10]. The ventral sheets of the intra-embryonic mesoderm, the splanchnic mesoderm, resulting from the split in the lateral mesodermal layers, become closely associated with the primitive gut. They give rise to the musculature of the gut, its serosal covering (visceral peritoneum) and its primary ventral and dorsal mesenteries.

When the body folds at the periphery of the midgut and yolk sac communication completely undercut the embryo and form the ventral abdominal wall and umbilical ring, the communication between the intra-

(*Continued on page 3*)

*Numbers in brackets refer to the individual pictures in the six following plates.



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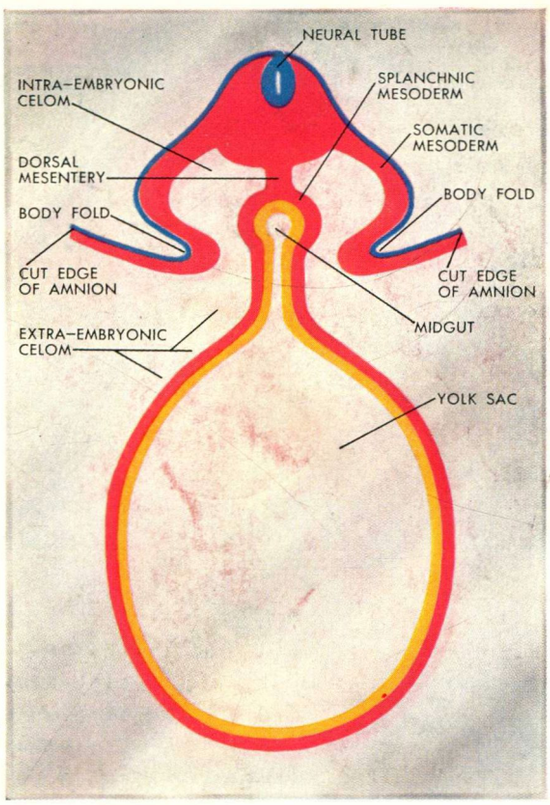

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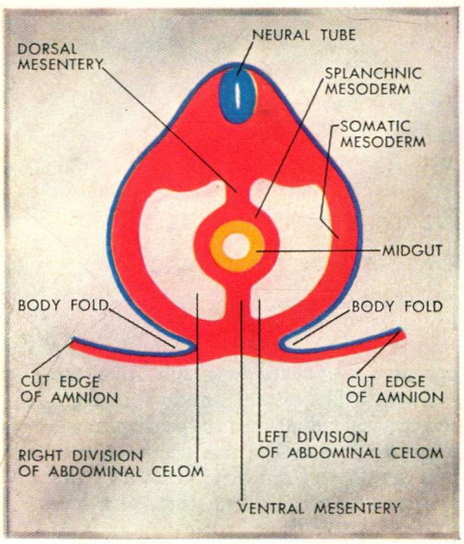

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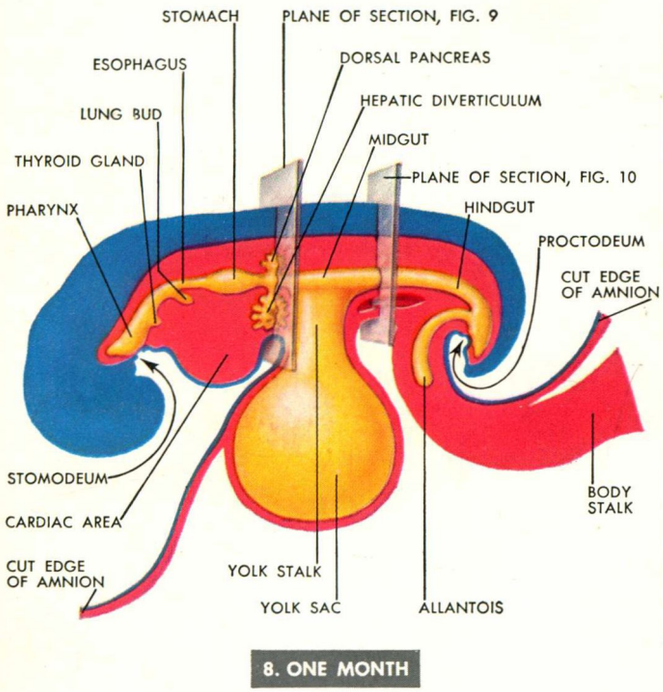
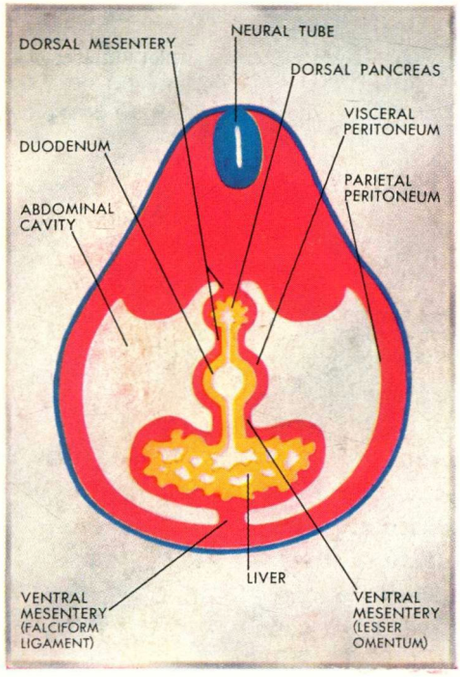
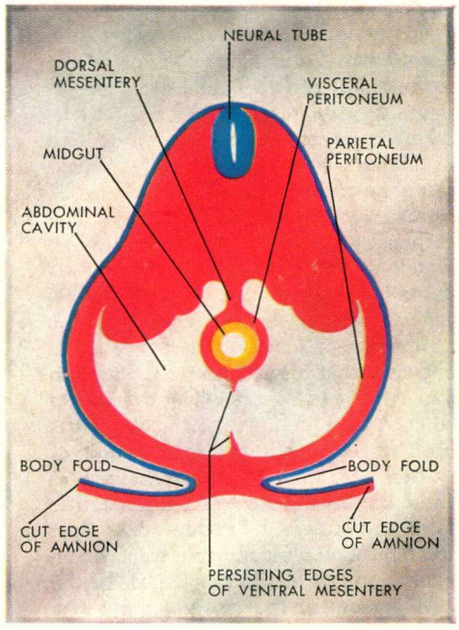


8. ONE MONTH



9. SECTION OF 8. (ANTERIOR)



10. SECTION OF 8. (POSTERIOR)

Development of Gastro-intestinal Tract

(*Continued from page 2*)

and extra-embryonic celoms is greatly reduced in size as the extra-embryonic celom becomes a tubular cavity within the umbilical cord [12]. Faulty closure of the body folds to form the umbilical ring can result in most of the abdominal viscera developing outside of the body cavity in a transparent sac of amnion. The sac is directly attached to the placenta, with only a portion or no true ventral abdominal wall present. This is one type of omphalocele (see page 125), also known as eventration of the abdominal viscera or abdominal hernia. During the closure of the body folds, the two layers of splanchnic mesoderm approach each other and come into direct contact at the midline of the embryonic body. In so doing, they enclose the now tubular gut and form the dorsal and ventral divisions of the primary mesentery, which suspends the gut from the dorsal and ventral body walls [4, 6, 7, 9, 10]. This mesentery completely separates the celomic cavity into right and left divisions in the abdominal area. However, each of the two abdominal divisions of the cavity at this developmental stage extends as a pleural canal, one on each side of the esophagus dorsal to the transverse septum, to become continuous with the single pericardial celom surrounding the developing heart [11]. The pericardial celom is later subdivided into the pleural and pericardial cavities as the lungs develop.

The *transverse septum* is a shelf of somatic mesoderm extending from the ventral body wall, which partitions off the pericardial region from the abdominal region [11]. It becomes the *ventral part of the diaphragm* [16]. The pleural canals become closed by folds of somatic mesoderm, the pleuroperitoneal membranes, arising from the posterolateral body wall on each side. They extend toward the midline of the body, being continuous during this process with the dorsal border of the transverse septum, to meet and *fuse with the midline visceral mesoderm* in which the *esophagus* is *embedded* to complete the formation of the diaphragm [12, 13, 16]. Later, the musculature of the diaphragm devel-

(*Continued on page 4*)

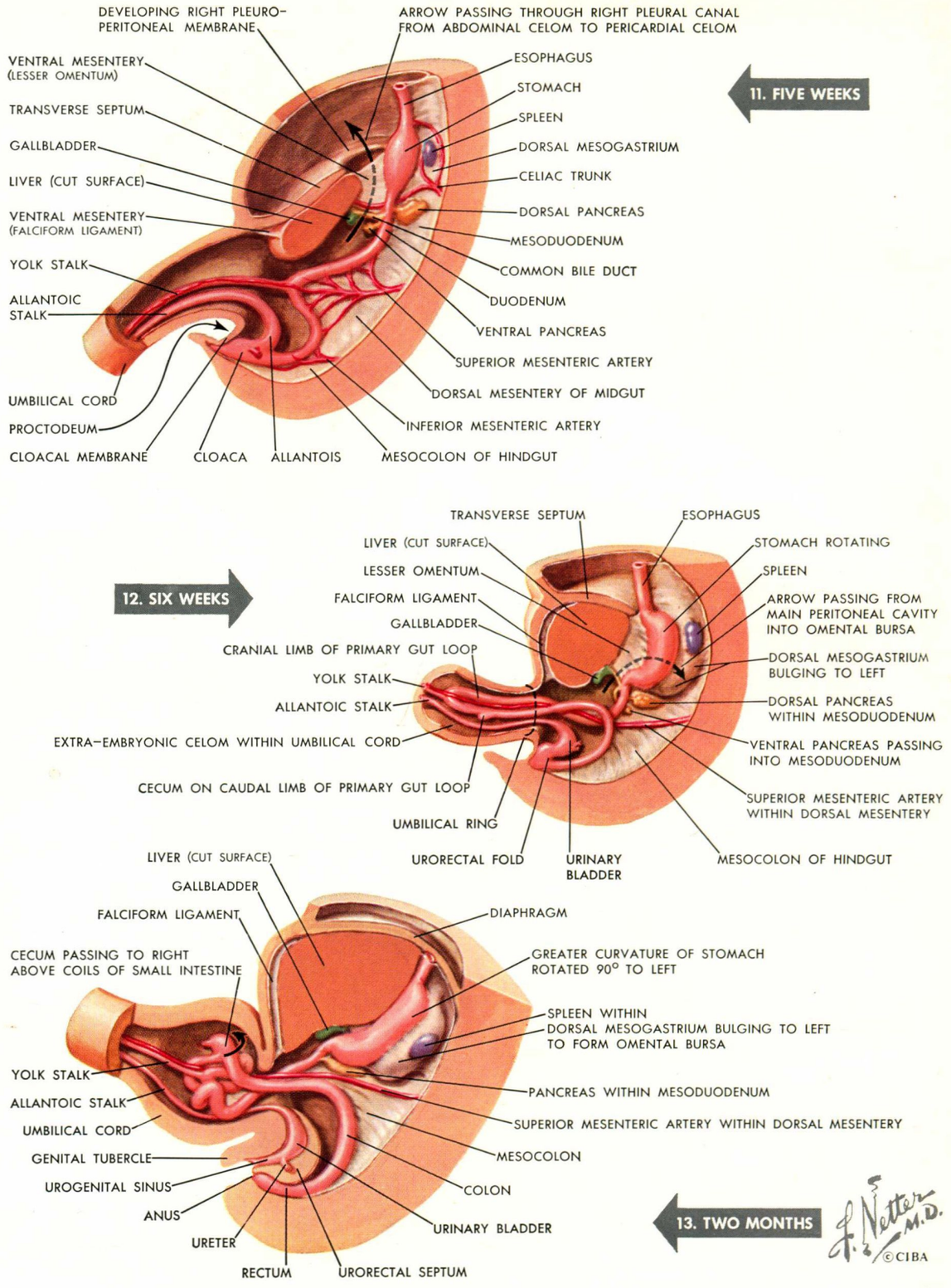
DEVELOPMENT OF GASTRO-INTESTINAL TRACT

(*Continued from page 3*)

ops as a secondary ingrowth from the body wall. The phrenic innervation from the cervical spinal cord to the diaphragm originates when the transverse septum first develops at the cervical level of the embryo. As the septum shifts to a low thoracic level, the phrenic nerves keep pace with it by elongating. The commonest developmental abnormality of the diaphragm is a faulty growth of the left pleuroperitoneal membrane, resulting in an opening through which abdominal viscera tend to herniate into the left pleural cavity (see page 123).

The portion of the foregut caudal to the origin of the tracheal outgrowth (lung bud) becomes narrowed to form the *esophagus*. The primary dorsal mesentery suspending the esophagus to the dorsal body wall never develops to the extent that it does along the gut caudal to the esophagus [8]. Therefore, the mesoderm of the esophageal portion of the dorsal mesentery remains relatively extensive in amount, by never becoming thinned into a membrane, and contributes to the formation of the mediastinum [16]. The esophagus is at first a short tube. Immediately caudal to it, the gut undergoes a dilatation, the *stomach* [8]. Originally, the stomach is situated cranial to the transverse septum. It then passes to an abdominal position caudal to the septum and, as it does, the esophagus elongates [8, 11-13]. The lumen of the esophagus becomes occluded during a period of extensive growth of its epithelium as the tube elongates but the lumen is later re-established. Failure of proper recanalization of its lumen results in congenital atresia of the esophagus. Faulty development of the tracheal outgrowth leads to the commonest anomaly of the trachea, in which an abnormal opening between the trachea and esophagus occurs below the level of the larynx. This is known as a tracheo-esophageal fistula. Atresia of the esophagus ranks second to imperforate anus as a cause of obstruction of the alimentary canal in newborn infants and is usually accompanied by a tracheo-esophageal fistula (see CIBA COLLECTION, Vol. 3/I, page 138).

In the abdominal area the gastro-intestinal tract is at first suspended between the ventral and dorsal body walls by the primary *ventral* and *dorsal mesenteries* [7, 9]. The more caudal part of the *ventral mesentery disintegrates* very early after its formation, bringing the right and left divisions of the abdominal celom into confluence to establish the unpaired condition of the peritoneal cavity [8, 10]. The *liver* develops within the persisting cranial portion of the ventral mesentery.



The portion of the ventral mesentery between the developing liver and the ventral body wall becomes the *falciform ligament* [8, 9, 11-13]. When the single umbilical vein is formed, it passes within the caudal free edge of the falciform ligament [18, 19]. After birth, the vein becomes a fibrous cord, the ligamentum teres [20]. The portion of the *persisting ventral mesentery between the liver,* the developing stomach and the upper *first part of the duodenum* becomes the *lesser omentum* [9, 11, 12, 17]. In contrast, almost the entire *primary dorsal mesentery* persists. It not only supports the gut in the abdominal cavity but serves as a pathway through which vessels and nerves pass to reach the gut from their main trunks along the dorsal body wall. When the subdivisions of the gastro-intestinal tract are first established, the portion of the continuous dorsal mesentery attaching to each subdivision is regionally named: *i.e.,* stomach — *dorsal mesogastrium;* duodenum — *mesoduodenum;* jejunum and ileum — mesentery; and colon — *mesocolon* [11].

During the early stages of the formation of the duodenum, a small hepatic diverticulum (see also CIBA COLLECTION, Vol. 3/III, page 2) arises from a thickened ventral area of duodenal entoderm, which is destined to form the parenchymatous liver tissue, the secretory liver tubules, together with their duct system, and the gallbladder. The young hepatic tubules grow out from the diverticulum, extending between the two layers of the ventral mesentery toward the transverse septum [8, 9]. When the liver grows, the surrounding layers of the ventral mesentery become its serosal lining, which does not completely encapsulate it. The reflections of the serosal lining off the liver onto the transverse septum constitute

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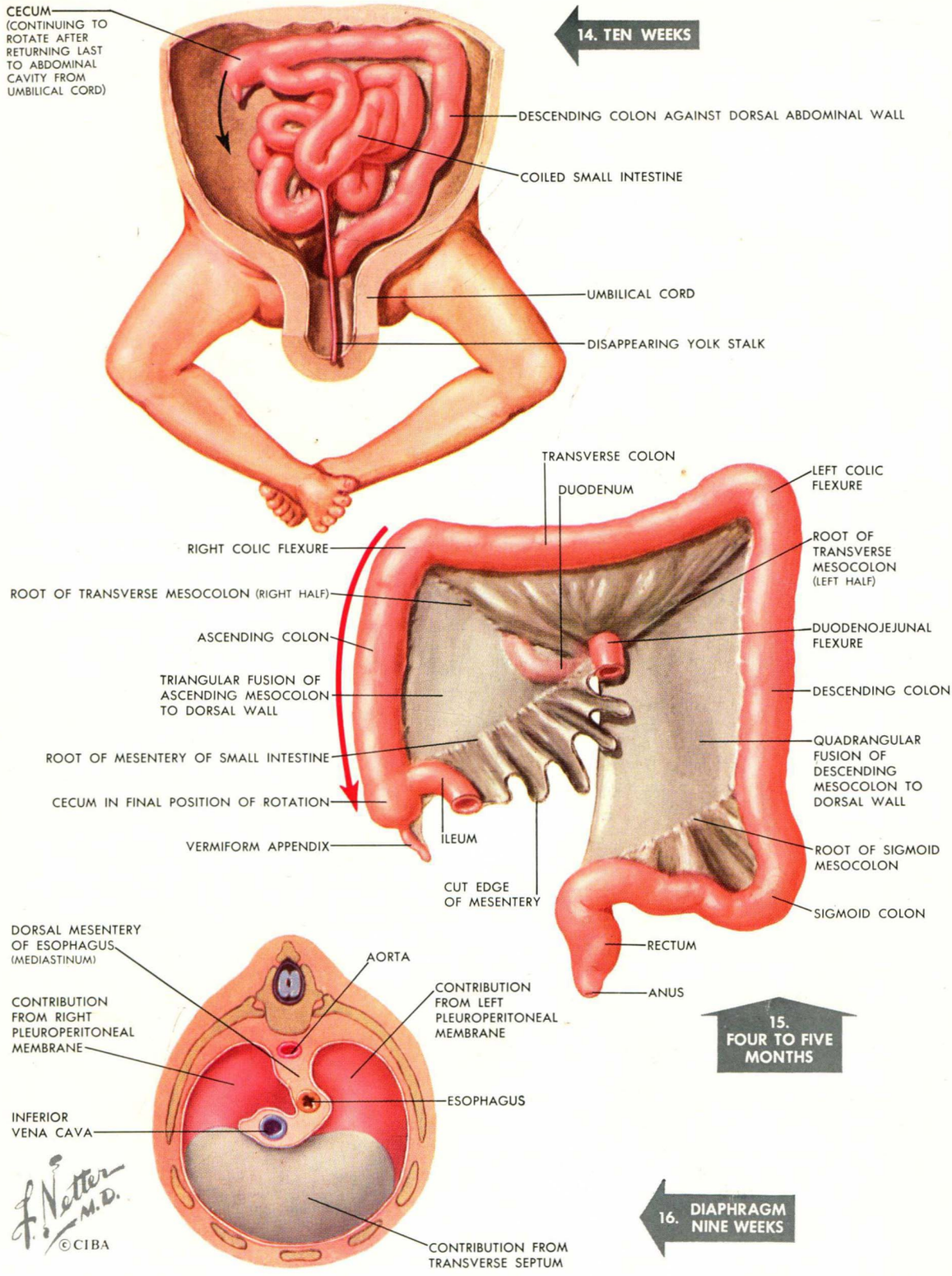
the coronary ligament. The area between these reflections, where the liver is in contact with the transverse septum without a serous lining intervening, constitutes the liver bare area [18-20]. Coronary ligament is an appropriate term early in development when the bare area is the shape of a crown; however, it later becomes more crescentic. The lateral extensions of the then-known anterior and posterior layers of the coronary ligament constitute the right and left triangular ligaments (see Ciba Collection, Vol. 3/III, page 5). The anterior layer of the coronary ligament is continuous with the falciform ligament.

The caudal portion of the original hepatic diverticulum becomes demarcated early from the main hepatic mass and gives rise to the gallbladder and cystic duct. The single hollow stalk of attachment to the duodenum of the developing hepatic ducts and cystic duct elongates to form the common bile duct, which lies in the free caudal edge of the persisting portion of the ventral mesentery (lesser omentum) [11, 12, 17].

When the caudal portion of the ventral mesentery disintegrates, the future intestines are represented by the part of the primitive gut tube extending from the stomach to the proctodeum. It lies in the midsagittal body plane parallel to the dorsal body wall, to which it is suspended by the dorsal mesentery [11]. The first departure from this condition is a rapid elongation of the gut, which results in the formation of a horizontal U-shaped or hairpin-shaped loop that extends into the proximal portion of the umbilical cord [12]. The connection of the yolk stalk with the gut lies at the bend of this primary gut loop. The part of the gut between the stomach and the yolk stalk is the cranial limb of the loop, and that part between the yolk stalk and future anus is the caudal limb. The yolk stalk connection is just proximal to the point of transition between the small and large intestine. Therefore, the cranial limb of the gut loop is involved in forming the 18 to 20 ft. of small intestine comprised of the duodenum, jejunum and upper part of the ileum. The caudal limb forms the terminal 2 to 3 ft. of the ileum and all of the large intestine. When the primary gut loop develops, a small local dilatation in its caudal limb appears, the *cecum* [12]. This cecal dilatation enlarges as a whole, but, in time, its distal end begins to lag behind the rest of it, so that its distal end appears as an extension of a smaller diameter. This slender extremity becomes the vermiform appendix [13-15].



The part of the gut which is to become the large intestine is, at first, of a smaller diameter than the part which becomes the small intestine. Later in development, the large intestine acquires a greater diameter.

The positional changes which bring about the adult relationships of the intestine are initiated by the occurrence of a twist or *rotation* in the horizontal *primary gut loop situated in the umbilical cord*. The rotation is in a counterclockwise direction when one views the ventral aspect of the embryo [13]. The central axis of the twist is the *superior mesenteric artery* passing through the dorsal mesentery directly from the dorsal body wall to the yolk stalk [11-13]. As the cecal dilatation begins to rotate in a counterclockwise direction, it passes above the original cranial limb or original right half of the horizontal primary loop. Below the cecum, which has started to rotate, the original cranial limb of the gut loop rapidly increases in length and undergoes *coiling*. Since this coiling of the small intestine begins while the partially rotated gut loop is still in the umbilical cord, it produces the normal umbilical hernia of development. When the abdomen has enlarged sufficiently to accommodate the entire intestinal tract, the protruding part of the tract passes back through the umbilical ring into the abdominal cavity, and the *yolk stalk* begins to *disappear* [14]. In this retraction the coils of small intestine tend to slip into the abdominal cavity ahead of the protruding part of the colon. In so doing, they crowd the lower part of the colon, which had always remained within the abdominal cavity, to the left. This establishes the *descending colon* in its adult position *close against the left side of the dorsal body wall*. When the upper part of the colon, which protruded into the umbilical cord, is

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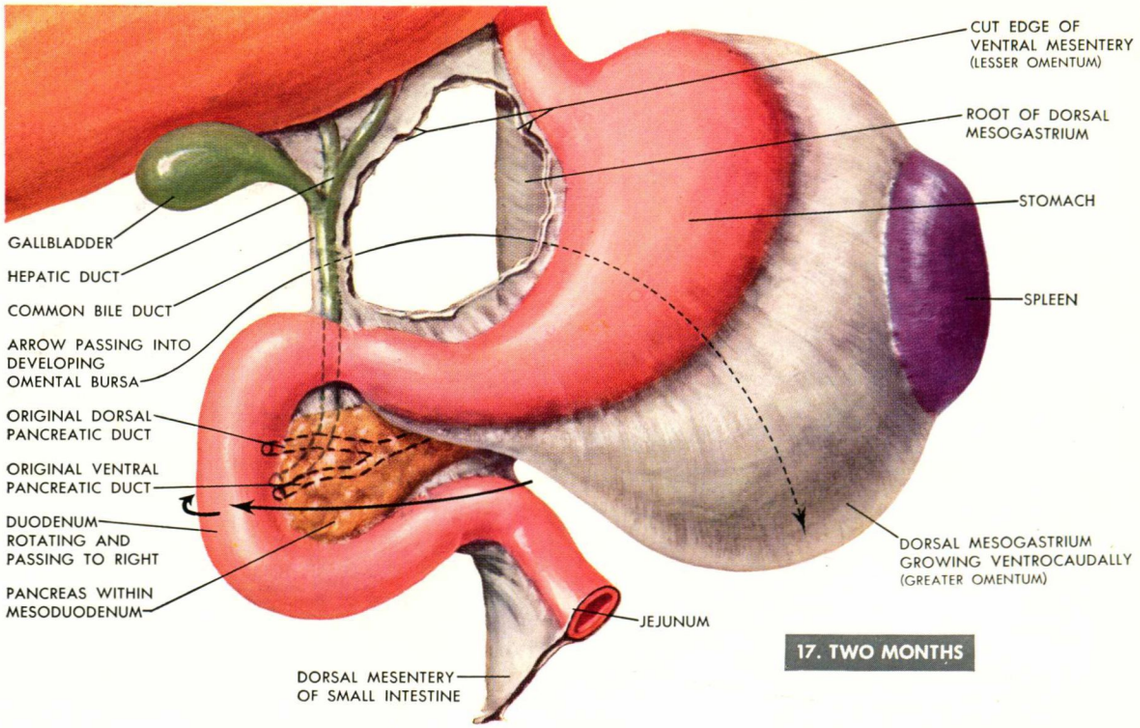
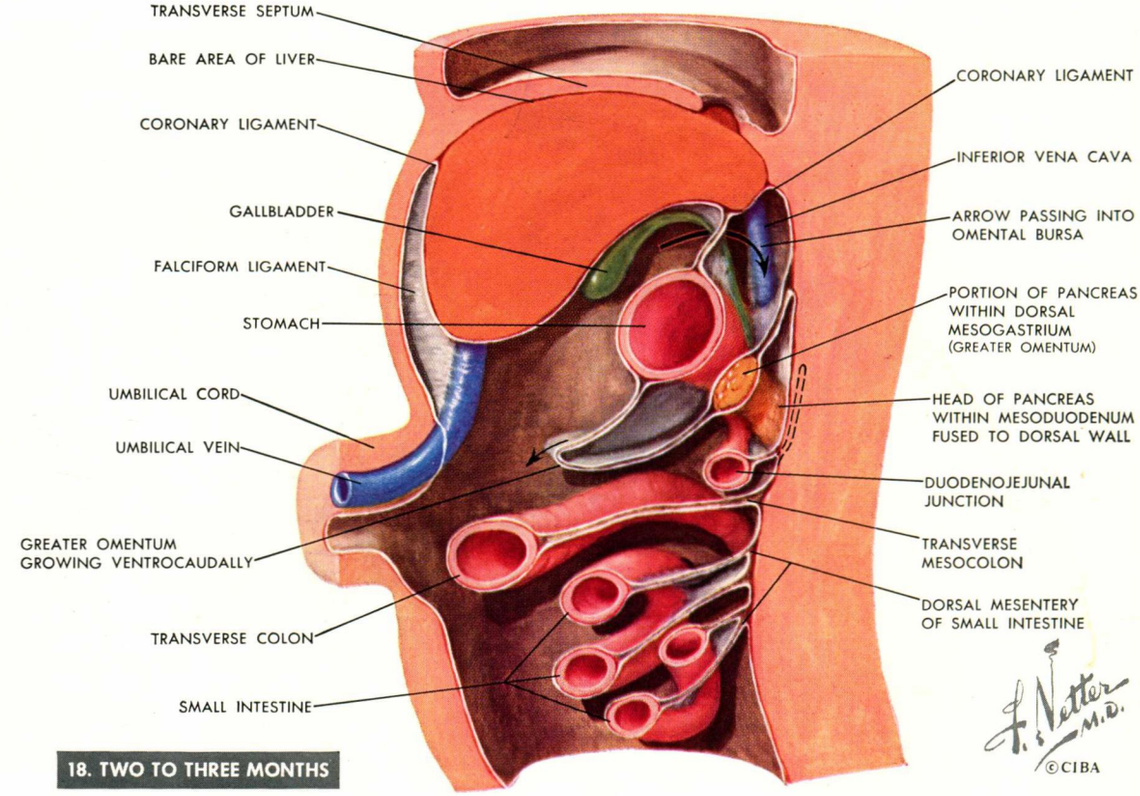
drawn last into the abdominal cavity its *cecal end continues its rotation in a counterclockwise direction* toward the right side of the abdominal cavity and then downward to the lower right quadrant of the abdomen [15]. This establishes the transverse colon in a position above the jejunum and ileum and the ascending colon close against the right side of the dorsal body wall. As the ascending colon approximates the dorsal body wall, the *original left side* of its *mesocolon fuses with the parietal peritoneum dorsal to it in a triangular fashion.* The base of this fusion triangle is the ascending colon from the ileocecal junction to the right colic flexure. The apex of the triangle is at the duodenojejunal flexure. The upper border of the triangle runs from the right colic flexure to its apex to form the *right half of the attachment of the transverse mesocolon* to the dorsal body wall. The lower border of the triangle passes from its apex to the ileocecal junction to form the attachment (*root*) *of the mesentery* of the small intestine to the dorsal body wall. In the adult the attachment or root of the mesentery of the small intestine, which is 6 or 7 in. long, runs on an angle from the level of the left side of the second lumbar vertebra to the right iliac fossa anterior to the right sacroiliac joint.

When the original left side of the serous lining of the ascending mesocolon met with the parietal peritoneum, both fused over a *triangular space,* leaving a fusion fascia, which is a double connective tissue plane remaining after the loss of the serous cells. Likewise, a layer of fusion fascia came into being between the surface of the ascending colon, which fused with the body-wall peritoneum, to constitute the bare area of the ascending colon. Since the vessels and nerves supplying the ascending colon were originally within its mesocolon, they pass to its left surface, following fusion of its mesocolon to the body wall, parallel with the plane of fusion fascia. Therefore, the ascending colon in the adult can be freed from its attachment to the body wall by first incising its visceral peritoneum as it is reflected off its lateral surface to become the parietal peritoneum all along its right border. Following the incision, blunt dissection along the plane of fusion fascia toward the midline of the body allows extensive elevation of the ascending colon, and its nerve and blood supply, away from the dorsal body wall.

When the descending colon closely approximated the dorsal body wall on the left side, as the intestine returned to the abdominal cavity from the umbilical cord, the *original left side of its mesocolon fused with the parietal peritoneum of the dorsal body wall in a quadrangular fashion* [15]. The right border of this fusion quadrangle is the original attachment of the descending mesocolon along the midsagittal plane of the dorsal body wall from the level of the duodenojejunal flexure down to the rectum. The upper border of the quadrangle passes from the *duodenojejunal flexure to the left colic flexure,* to form the attachment of the left half of the transverse mesocolon to the dorsal body wall. The left border of the quadrangle is the descending colon from its left colic flexure down to its junction with the sigmoid colon. The *lower border* of the quadrangle passes from the junction of the descending and sigmoid colon to the rectum, to form the *attachment of the sigmoid mesocolon* to the dorsal body wall.



17. TWO MONTHS



18. TWO TO THREE MONTHS

This *quadrangular fusion* of the original left side of the descending mesocolon to the dorsal wall parietal peritoneum resulted in the formation of a fusion fascia, in the same manner described for the ascending mesocolon on the right side of the abdominal cavity. A layer of fusion fascia also developed between the surface of the descending colon and the body-wall peritoneum, constituting the bare area of the descending colon. Since the vessels and nerves supplying the descending colon were originally within its mesocolon, they pass to its right surface, following fusion of its mesocolon to the body wall, parallel with the plane of fusion fascia. Therefore, the descending colon can be freed from its attachment to the body wall by first incising its visceral peritoneum as it is reflected off its lateral surface to become the parietal peritoneum all along its left border. Following the

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