INTRA-ABDOMINAL HERNIAS DUE TO DEVELOPMENTAL AND ROTATIONAL ANOMALIES*

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THE TERM *internal hernia* has been applied loosely to a large variety of intra-abdominal incarcerations and strangulations with little regard for the pathogenesis of specific types. Apparently one reason for this situation is that the symptomatology of all types of internal strangulation obstructions is approximately the same. Therefore, one can lose sight of the important variations in the development of certain types. We believe that if a clearer understanding of internal hernias is made available, it should aid the surgeon in recognizing the pathologic change present at operation.

It is common, but not necessarily proper usage to include among the internal hernias almost any incarceration within the peritoneal cavity. Such a concept can include not only all obstructions due to adhesions, surgical artefacts and gutters, but if carried one step further, can embrace intussusception, volvulus and all other incarcerating lesions and strangulating obstructions. Although such an all-inclusive concept is not necessarily illogical, it serves no useful purpose and may be highly confusing. There is another group of hernias, not confined to the normal limits of an intact endoabdominal fascia, which has been erroneously labeled internal hernia. Such hernias include obturator, sciatic, diaphragmatic and Morgagni hernias. They have been called "internal" since they are not usually discernible by external examination. We do not recognize these as internal since they fit within the definition of external hernias, occurring as they do through defects in the encompassing abdominal wall.

Limiting ourselves to the confines of the peritoneal cavity, we are confronted with a further problem of separating acquired artefacts from developmental anomalies. An acquired artefact can include surgical, traumatic, and even congenital defects in mesenteries, omenta and other structures, as well as postoperative adhesions and gutters. It is obvious that in most of these defects the incarceration of bowel follows the creation of the defect. In such cases bowel finds its way into the defect by virtue of peristaltic movement and a natural property of the bowel to occupy the entire peritoneal cavity. In the case of adhesions, however, incarceration of bowel may either follow the formation of an artificial peritoneal subdivision, or the bowel may become incarcerated *in situ* by virtue of an adhesive band spanning any sector of the peritoneal cavity which contains small bowel. Yet, this type of incarceration is not truly developmental.

There remains the group of intra-abdominal hernias which comprise the subject of this report. They are the result of incomplete development and rotation of the gastro-intestinal tract. It has been suggested that these hernias are the only true type of internal hernia. For the sake of clarity we prefer to call them intra-abdom-

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inal hernias due to developmental and rotational anomalies.

These hernias are rare, as evidenced by the fact that only 12 unequivocal cases were found in a 10-year survey of the records of Cook County Hospital, a charity institution of 3500 beds. These 12 cases are summarized in Table I.

The original concept of the genesis of these hernias was that the incarceration occurred by the entrance of a loop of bowel into one of the numerous peritoneal fossae which perhaps was somewhat larger than usual. This concept is parallel to the usual course of events in the case of an external hernial opening. It was assumed that by a process of gradual stretching of the pocket, more and more of the bowel entered until a greater length, if not the entire small intestine, was so swallowed.

The late Edmund Andrews, however, suggested that this concept was erroneous and that most, if not all, fossal internal hernias are actually anomalies of rotation and development.[1] Andrews reasoned that at least the duodenal types of hernias and probably also the cecal, are actually anomalies of intestinal rotation, in which the small bowel is trapped behind the transverse mesocolon, as the cecum rotates from the right to the left side of the abdomen and eventually becomes fixed together with the ascending colon to the posterior peritoneum. This explanation was hinted at earlier by Hertzler[4] in his monograph (1919) on the peritoneum. He wrote ". . . it seems possible that right duodenal hernias are caused by the covering over of the intestinal walls by the meson of the ascending colon, which afterwards becomes fixed, thus forming a permanent housing of the small intestine."

Recent authors, such as Callander,[2] Longacre[6] and Gardner[3] have since elaborated on this concept of genesis of internal hernias. While most thought has been given to the duodenal hernias, the same mechanism may be responsible for the cecal group and perhaps for the remaining types as well.

It is not sufficient to classify a developmental hernia as congenital, since it is obvious that an internal incarceration due to congenital adhesions or defects may occur at or near birth without a developmental or rotational anomaly.

The general anatomical groups of peritoneal fossae in which incarcerations commonly occur are:

1. Paraduodenal
   a. Left paraduodenal
   b. Right paraduodenal
   c. Transmesocolic (transmesenteric)
   d. Foramen of Winslow
2. Pericecal (paracecal, retrocecal)
3. Intersigmoid
4. Internal supravesical

PARADUODENAL HERNIAS

The region of the duodeno-jejunal junction is by far the most frequent location of developmental internal hernia. The latest comprehensive survey of the literature by Hansmann and Morton (1939) indicates that paraduodenal hernias comprise 53 per cent of all reported internal hernias. Four of our 12 cases were of the paraduodenal type. No less than ten peritoneal fossae have been described about the point of emergence of the jejunum from the transverse mesocolon and into at least five of these, various types of paraduodenal hernias are thought to occur. In addition, hernias through the transverse mesocolon and those of the foramen of Winslow are geographically and also probably etiologically related to those of the duodenal fossae. We believe the reason for the relative frequency of paraduodenal hernias is made clear in light of the genesis of these conditions.

An understanding of the origin and nature of paraduodenal hernias, in terms of the developmental concept, requires a review of the embryological stages of intes-

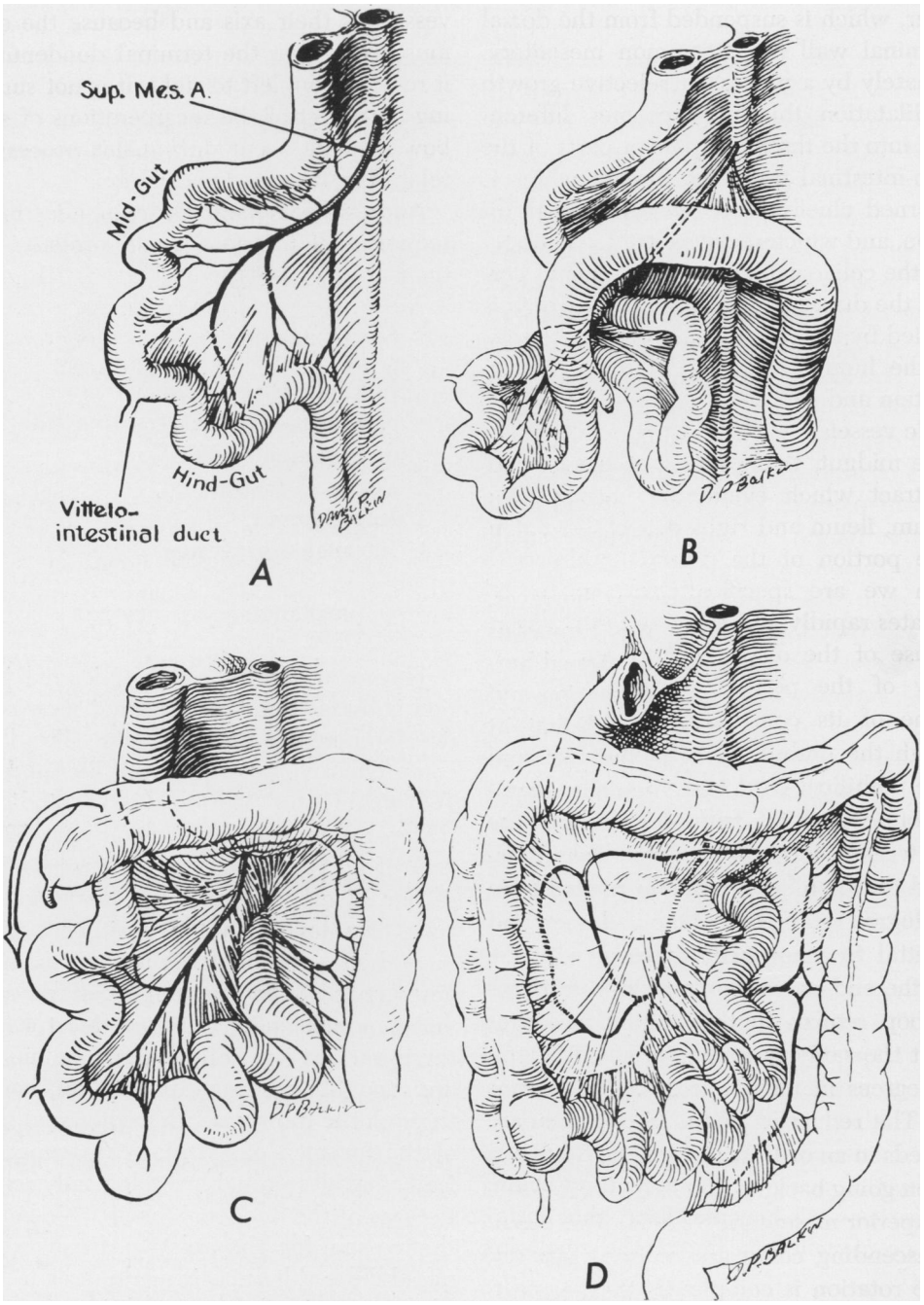

FIG. 1. Normal Intestinal Rotation. A. Loop formed by midgut. B. Rotation of midgut and extracelomic position. C. Orderly return of intestinal loops into peritoneal cavity below the transverse mesocolon and further rotation of 180 degrees in counterclockwise direction. D. Descent of cecum and fixation of ascending colon to posterior parietal peritoneum.

tinal rotation (Fig. 1). The primitive alimentary canal is a straight tube of uniform caliber, which is suspended from the dorsal abdominal wall by a common mesentery. Ultimately by a process of selective growth and dilatation, this tube becomes differentiated into the three component parts of the gastro-intestinal tract; the foregut which is concerned chiefly with the function of digestion, and which receives its blood supply from the celiac axis; the midgut which performs the duties of adsorption, and which is supplied by the superior mesenteric vessels; and the hindgut, essentially the organ of excretion and supplied by the inferior mesenteric vessels.

The midgut, that portion of the alimentary tract which eventually becomes the jejunum, ileum and right half of the colon, is the portion of the digestive tube with which we are specifically concerned. It elongates rapidly and is thrown into a loop. Because of the disparity between the capacity of the peritoneal cavity and the volume of its contents, this loop bulges through the umbilical orifice into the base of the umbilical cord and occupies this extracelomic position from the fifth to the tenth week of intra-uterine life. During this period the umbilical bowel loop undergoes a 90 degree rotation, counterclockwise from a sagittal to a horizontal plane.

At the end of the tenth week the umbilical loop returns to the abdominal cavity and at the same time rotates an additional 180 degrees in the counterclockwise direction. The reduction of the bowel normally proceeds in an orderly fashion, the proximal portion going back first and passing beneath the superior mesenteric vessels. The cecum and ascending colon are reduced last and as the rotation is completed, they come to lie in the right upper quadrant. Subsequently the cecum descends to the right lower quadrant and the colon becomes fixed to the posterior parietal peritoneum.

In view of the fact that the rotations of the midgut are on the superior mesenteric vessels as their axis and because the colon must cross over the terminal duodenum as it rotates from left to right, it is not surprising that most of the incarcerations of small bowel which occur during this process are related to the duodenal region.

Andrews reasoned that anomalies in this sequence of rotations could produce both the right and left types of duodenal hernia. A lesser degree of deviation from the normal would produce the right duodenal hernia in which the sac lies to the right of the duodenum with its aperture looking toward the left (Fig. 2). In this form, the superior mesenteric artery or its continuation, the ileocolic artery, lies in the free edge of the sac. The formation of this anomaly is due to delayed and incomplete rotation of the umbilical loop. The cecum then does not lie superior to the small intestine, and as it migrates from the left to the right, the small bowel is caught in the mesentery of the ascending colon. When the right colon fuses with the posterior parietal peritoneum, the small bowel loops become imprisoned behind this mesentery, thus forming the usual picture of the right paraduodenal hernia. The fossa has been labeled in the older literature as the fossa of Jonnesco.

The left paraduodenal hernia is a manifestation of a further degree of the same malformation (Fig. 3). In this form the sac lies to the left of the duodenum, and the inferior mesenteric vessels course in the fold which constitutes the free margin of the sac. The mechanism of this imprisonment begins with a reversed rotation of the umbilical loop so that when the small bowel is reduced into the abdomen, it is caught in the mesentery of the descending colon and is eventually incarcerated there by the fixation of the left colon to the posterior wall. The name of Landzert has been attached to this type of hernia.

TABLE I.

Case	Age and Sex	Type	Contents	Symptoms	Clinical Diagnosis	Treatment	Result
1	30—male	Left paraduodenal	3 ft. ileum and ascending colon, gangrenous with multiple perforations.	Abdominal pain, distension, vomiting—5 days.	Perforated viscus with walling off.	None	Patient died 4 hours after admission. Autopsy.
2	38—female	Left paraduodenal	Unstated length of gangrenous bowel with peritonitis.	Abdominal pain, distension, vomiting—2 days.	Acute abdomen.	Supportive.	Death day of admission. Autopsy.
3	1 week—male	Left paraduodenal	Loop of small bowel incarcerated.	Distension, vomiting since birth.	Congenital intestinal obstruction.	Surgical. Unroofing of fossa.	Recovery.
4	44—male	Right paraduodenal	Incarcerated loop of small intestine.	Abdominal pain, vomiting 2 days; chronic constipation.	Intestinal obstruction.	Surgical reduction and closure of fossa.	Recovery.
5	53—male	Paracecal	Terminal ileum and cecum —apparently viable.	Abdominal pain, distension, vomiting 2 weeks.	Intestinal obstruction.	Surgical. Reduction of bowel; closure of fossa.	Death 6 days post-op. following enema. No autopsy.
6	50—female	Paracecal	Gangrenous jejunum, unstated length.	Right abdominal pain, vomiting "several" days.	Ruptured appendix with peritonitis.	Surgical. Reduction.	Gangrenous bowel found at autopsy.
7	73—female	Paracecal	Infarction of bowel, incarcerated.	Symptoms of circulatory collapse.	Myocardial infarction.	Medical. Picture dominated by circulatory collapse.	Death. Autopsy.
8	36—male	Retrocecal	Retrocecal appendix and terminal ileum. Ileum adherent to post. peritoneum. Bowel viable. Appendix removed.	Right abdominal pain 3½ hours.	Acute appendicitis.	Surgical. Appendectomy and reduction.	Recovery.
9	42—male	Transmesenteric	3 ft. gangrenous ileum.	Fecal vomiting and cramps—3 weeks.	Intestinal obstruction with perforation.	Surgical Reduction and anastomosis.	Death 5 days post-op. No autopsy.
10	13 days—male	Transmesenteric	Incarcerated obstruction without infarction.	Vomiting and distension since birth	Congenital intestinal obstruction.	Surgical. Reduction.	Recovery.
11	63—male	Intersigmoid	Ileum (length unlisted), gangrenous; in fossa formed by leaves of sigmoid mesentery.	Abdominal pain, distension, obstipation, vomiting 5 days.	Bilateral pneumonia.	Supportive.	Death 6 days after admission. Autopsy.
12	47—male	Supravesical	8″ loop ileum incarcerated.	Low abdominal pain and distension 3 days.	Intestinal obstruction.	Surgical reduction and closure of fossa.	Recovery.

Numerous variations in this anomalous rotation are seen. The two other recognized types of internal herniation in the duodenal region, the transmesocolic and those of the foramen of Winslow are probably further manifestations of anomalous rotation, whose exact mechanism has as yet not been worked out. The same arguments for a prenatal origin of these conditions prevail as do for the more thoroughly studied varieties. Our series contains two cases of transmesenteric hernia.

PERICECAL HERNIAS

There are at least four peritoneal fossae in the ileocecal region into which hernias are said to occur. In general these hernias are less frequent than are those of the duodeno-jejunal region. They, too, have been accepted as herniations into preformed pouches, although Andrews believed that they were also embryonic entrapments of small bowel behind the mesentery of the cecum and terminal ileum. Their genesis is easily explained on the basis of a minimal error in rotation with incarceration behind the cecum during the final phases of descent and fixation of the right colon. A related and much more common anomaly is the retrocecal or retroperitoneal appendix, which is also caught behind the mesentery of the ileocecal region during the same phase of rotation. The so-called mesentericoparietal hernia of Waldeyer is an example of paracecal hernia.

Three cases of paracecal hernias and one case of retrocecal hernia occurred in our series. The findings are presented in Table I.

INTERSIGMOID HERNIAS

A very rare form of internal herniation has been described in which a portion of small bowel is incarcerated in a fossa between the two loops of the sigmoid colon and its mesentery. There have been too few such cases and too little opportunity for careful study to permit conclusions as to their step-by-step development.

There was one intersigmoid hernia in our series (Table I).

SUPRAVESICAL HERNIAS

Hernias into the supravesical fossa are infrequently reported, but probably occur much more often and fail to be recognized. They fall into two quite distinct categories, the external variety which bulges out of the abdominal cavity in the hypogastric, inguinal or femoral regions, and the internal variety which dissects into the supravesical fossa and remains within the confines of the intact endoabdominal fascia. The latter type, therefore, constitutes one of the varieties of intra-abdominal hernia.

The supravesical fossa is a triangular depression of the peritoneum bounded by the bladder below and the two obliterated hypogastric arteries on either side. It is divided into a right and a left compartment by a median fold representing the obliterated urachus. Of the multiplicity of names applies to internal hernias originating in this space, the term *internal supravesical* is most appropriate. Four subdivisions of internal supravesical hernias are described on the basis of the relation of the fundus of the sac to the bladder; *i.e.*, prevesical into the space of Retzius, supravesical, intravesical and paravesical.

The factors held responsible for the development of supravesical hernias are (1) increased depth of the fossae, due to congenitally incomplete regression of the hypogastric arteries and urachus; (2) loss of prevesical fat; and (3) inflammatory fibrosis of subperitoneal tissues with retraction. They are more common in males, only four of the reported cases having been seen in females. All reported cases have been in adults, and chiefly within the middle-age group.

The clinical manifestations are those of small bowel strangulation plus urinary symptoms. Localized swelling and tenderness in the suprapubic region may be elic-

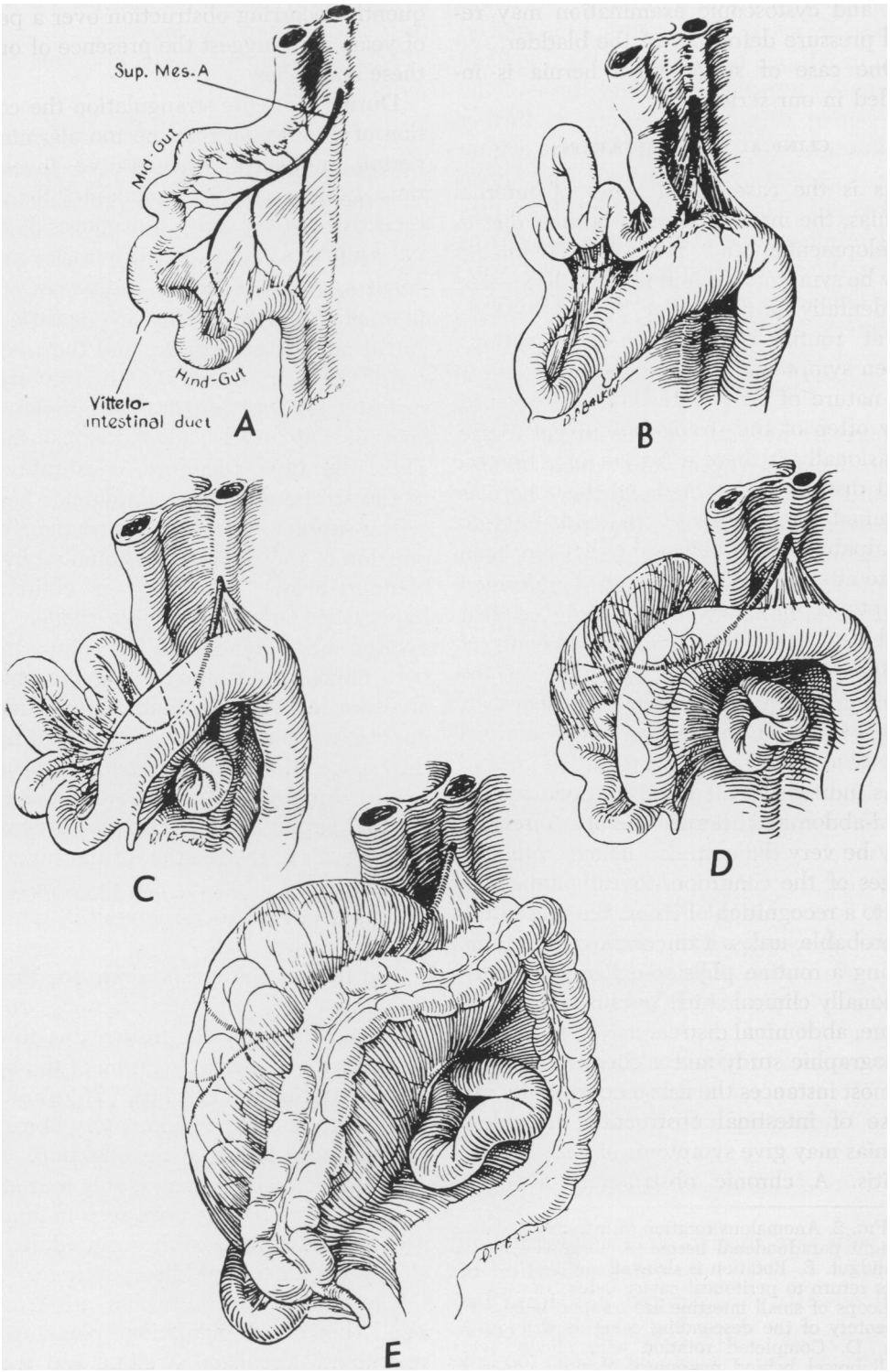

FIG. 2. Legend on opposite page.

ited and cystoscopic examination may reveal pressure deformity of the bladder.

One case of supravesical hernia is included in our series.

CLINICAL MANIFESTATIONS

As is the case in all types of internal hernias, the intra-abdominal hernias due to developmental and rotational anomalies may be symptomless and may be discovered incidentally during surgery for other cause, or at routine postmortem examinations. When symptoms are produced, they are in the nature of acute intestinal obstructions, very often of the strangulating type. Only occasionally is there a history of a chronic mild distress which leads to exploration or culminates in the acute obstructive attack. A palpable tumor, detected either during an acute attack or in the absence of abdominal symptoms, may lead to a correct diagnosis. Such tumors are localized, and may vary in tension and size with the intensity of symptoms. They may be tympanitic on percussion and impart peristaltic sounds on auscultation.

As indicated by Table I, the diagnosis of intra-abdominal hernia before operation may be very difficult. In the asymptomatic stages of the condition, investigation leading to a recognition of its existence is highly improbable, unless a tumor mass is palpated during a routine physical examination. Occasionally clinical study because of chronic, vague, abdominal distress may lead to roentgenographic study and a correct diagnosis. In most instances the first manifestations are those of intestinal obstruction. Paracecal hernias may give symptoms of acute appendicitis. A chronic obstruction or a frequently recurring obstruction over a period of years may suggest the presence of one of these anomalies.

During an acute strangulation the condition of the patient may be too alarming to permit prolonged preoperative investigation. In a considerable number of the more recently reported cases, a diagnosis of internal hernia has been correctly made or seriously considered prior to operation. However, it is impossible to differentiate clinically the developmental internal hernias from other types of internal hernia. An acute small bowel obstruction with strangulation in the absence of external hernia and/or no history of previous surgery must suggest the possibility of an intra-abdominal hernia. If in addition there is a history of chronic mild distress or of prior obstruction, this suggestion becomes stronger. The symptoms are not specific other than of intestinal strangulation, but the presence of an abdominal tumor which emits a resonant note and gurgling peristaltic sounds greatly increases the likelihood of an intra-abdominal incarceration. In all cases of our series there occurred an acute abdominal syndrome. Several of the patients did not present themselves for admission until after several days of acute symptoms, when their condition was precarious.

The roentgenologic findings may be characteristic and a number of cases have been correctly diagnosed on the basis of the roentgen ray appearance. The scout film may show an agglomeration of small bowel loops into one portion of the abdominal cavity and an absence of such loops elsewhere. Whatever portion of the small bowel is proximal to the incarceration will show dilated, gas-filled loops. Barium enema may reveal a distortion of the colon as it encircles the loculated small bowel mass.

TREATMENT

The management depends upon the disturbances produced. Asymptomatic hernias

FIG. 2. Anomalous rotation of intestine resulting in right paraduodenal hernia. A. Original position of midgut. B. Rotation is delayed and small bowel loops return to peritoneal cavity before this occurs. C. Loops of small intestine are trapped behind the mesentery of the descending colon as rotation occurs. D. Completed rotation with almost entire small bowel behind mesentery of right colon. E. Characteristic position of small intestine in right paraduodenal hernia.

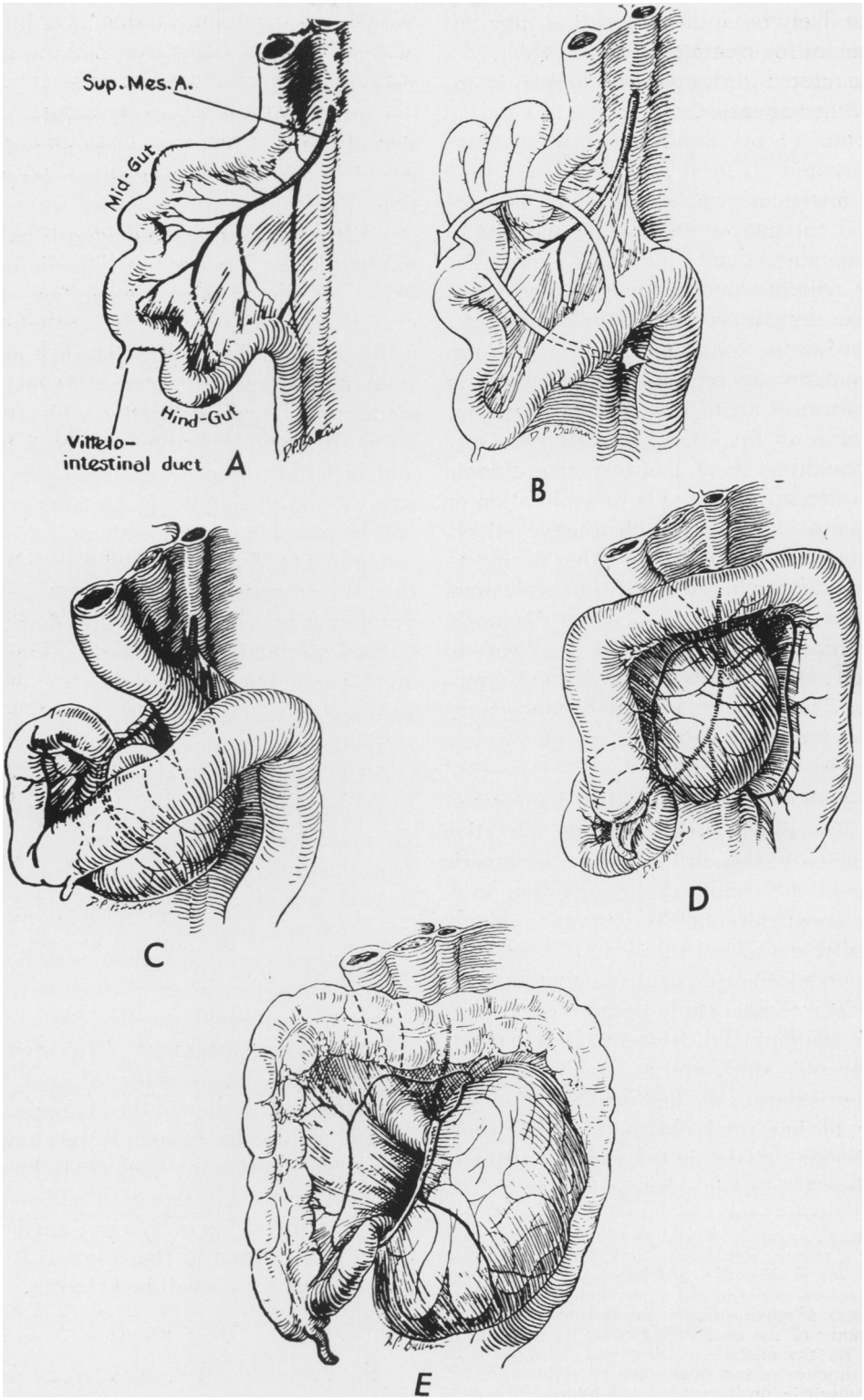

FIG. 3. Legend on opposite page.

are not likely to be diagnosed or to present themselves for treatment. However, if one is encountered during surgical operation for some other disease, the question of surgical correction of the intestinal anomaly must be evaluated. It must be remembered that many intestinal incarcerations of the type under discussion are well tolerated and produce no disturbances throughout the life of the patient. Correction of the anomaly may be difficult and not without danger. The presence of important vessels in the free margins of the sac may constitute a real hazard if their presence, identity and location is not kept in mind.

In the presence of intestinal obstruction, and particularly, if strangulation is present, early surgical release is mandatory. Procrastination can only lead to worsening of the general condition and further loss of viability of the bowel.

The findings at operation in cases of internal herniation may be bizarre and startling, and to the uninitiated may present perplexing problems. For this reason a familiarity with these conditions, including a recognition of their nature and the anatomical distortions produced, is important for anyone undertaking abdominal surgery. In some of these cases, when the peritoneal cavity is opened, no loops of small bowel are visible and the entire intestine is apparently incarcerated in a sac composed of mesentery of the ascending or descending colon. Occasionally short reaches of dilated afferent and collapsed efferent bowel may be seen entering and leaving the sac.

The primary purpose of the operation is the release of the obstruction. This can sometimes be effected by gentle traction on bowel loops entering the sac aided by guarded pressure on the sac. If this maneuver fails, it may be possible to dilate the hernial orifice by blunt stretching or by very cautious incision, being ever mindful of the possible injury of important mesenteric vessels. If reduction is otherwise impossible, an incision may be made in an avascular portion of the sac and the imprisoned intestinal loops delivered.

Further operative manipulation depends largely on the condition of the bowel. Infarcted or questionably viable bowel must be resected and continuity reestablished. If feasible, the original hernial orifice should be obliterated by suture and artificial openings made in the mesocolon for the release of strangulated intestine should also be closed.

FIG. 3. Mechanism involved in formation of left paraduodenal hernia. A. Pre-rotation status. B. Reversed rotation carrying small bowel to the right. C. Loops of jejunum and ileum reduced into mesentery of ascending colon. D. Completed rotation of colon. E. Relations found in left paraduodenal hernia.

SUMMARY

Intra-abdominal hernias due to developmental and rotational anomalies can be recognized at surgery and differentiated from internal obstructions and incarcerations due to adhesions and those due to other defects. The available evidence is presented, which implies that the hernias represent arrested stages of development and rotation. Twelve new cases are recorded, consisting of two left paraduodenal hernias, two right paraduodenal hernias, three paracecal hernias, one retrocecal hernia, two transmesenteric hernias, one intersigmoid hernia and one supravesical hernia.

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