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MONROE A. McIVER

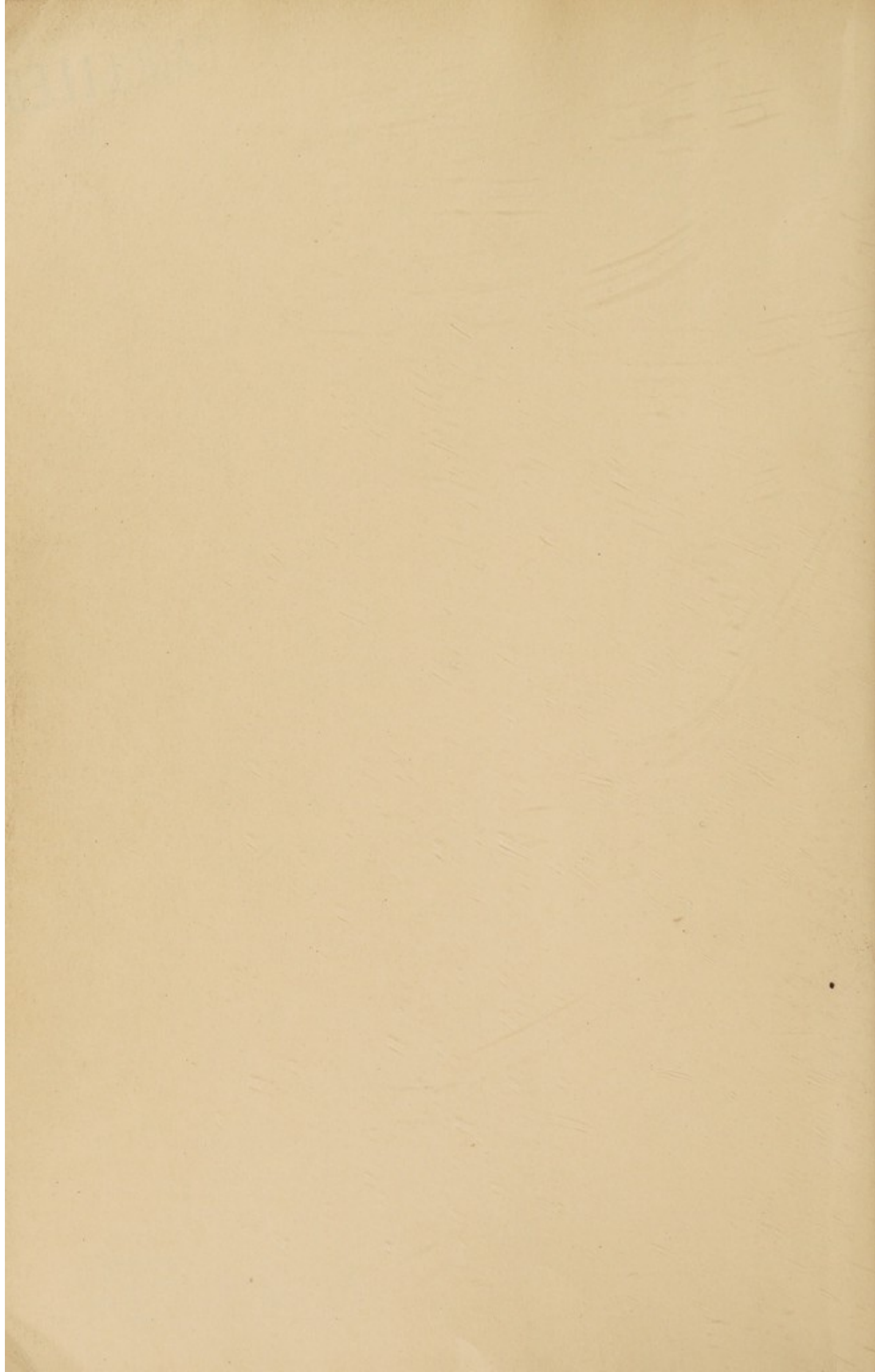
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ACUTE INTESTINAL OBSTRUCTION

BY

MONROE A. McIVER, M.D.

Surgeon-in-Chief, Mary Imogene Bassett Hospital, Cooperstown, N. Y.

62 ILLUSTRATIONS



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TO THE CLINICIANS AND INVESTIGATORS
WHOSE WORK IS PRESENTED
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PREFACE

ACUTE intestinal obstruction is a serious disease. The mortality is high, and has not been materially lowered during the past thirty years. Moreover, with the increasing number of laparotomies, the disease is of increasing importance as a postoperative complication, not only during convalescence but also as a late result of operative procedures. Such a situation obviously demands thoughtful consideration, and is a challenge to the profession.

It is not alone the severity of the disease, however, that renders it of such peculiar interest, but several other aspects as well. It is associated with almost every phase of abdominal surgery; the underlying pathological lesions are diverse and ally it to a wide range of gastrointestinal diseases; and the physiologic aspects, particularly as regards changes in body fluids and the mechanism of functional obstructions, present many interesting and complex problems.

The literature on intestinal obstruction is so extensive that no attempt has been made in this monograph to present an exhaustive bibliography; the author has tried, however, to furnish references to key articles on different phases of the subject. The following scheme is used in the presentation of the material:

There are three major divisions: PART I, which gives a general picture of the disease; PART II, which deals with points in diagnosis and methods of treatment; and PART III, which discusses the experimental work that has been carried out to determine the cause of death from intestinal obstruction. The first chapter of Part I defines the subject and presents a survey of the development of our knowledge of the disease and of methods of treatment. In the second chapter the methods of classifying cases are outlined and the important factors affecting all types are discussed. The next eight chapters take up in turn the various types of mechanical obstruction, discussing the

etiology, pathology and clinical aspects peculiar to each. Three chapters then describe the outstanding features of the pathological picture and some of the alterations in physiology, notably changes in body fluids, dehydration, and pain, vomiting and distention. The last three chapters of Part I are devoted to functional obstructions: the etiology and mechanism, with a résumé of pertinent points in intestinal physiology; the experimental work on this subject; the clinical picture presented by obstructions of both paralytic and spastic types; and prevention and treatment.

The first two chapters of Part II take up the various points in diagnosis: the general clinical picture; the method of studying the patient; the value of laboratory and x-ray studies; special points bearing on the diagnosis of certain varieties of obstruction; and differential diagnosis. The seven chapters that follow are devoted to methods of treatment: preoperative care; operative procedures; special points in the treatment of certain types of obstruction; postoperative care; and complications, operative and postoperative. Chapter XXVII presents statistics on the mortality.

The whole of Part III is given up to a presentation and evaluation of the experimental work carried out on the cause of death from acute intestinal obstruction. In the first chapter experimental methods are outlined and their relation to clinical obstructions is pointed out; since many of the conditions encountered in clinical surgery cannot be duplicated in the laboratory, however ingenious the experiments, the specific problems must be checked and evaluated against a background of clinical experience. The various theories as to the cause of death, and the experimental work related to them, are discussed in the five following chapters. The final chapter gives the author's conclusions as to the cause of death in the different types of obstruction.

The past decade has seen renewed activity in the investigation of many of the problems of intestinal obstruction; and it therefore seems a particularly fitting time to attempt to

bring together the existing wealth of clinical and pathological material and the considerable body of experimental work that is contributing to a clearer understanding of the fundamentals of the disease and its treatment.

Since the appearance of this monograph in serial form in *The American Journal of Surgery*, new material and references have been incorporated, which will, it is hoped, bring the work up to date. The author wishes to express his thanks to friends who have read the material and have made valuable suggestions; to his wife for her great assistance with the manuscript; and to Mr. Paul B. Hoeber and his editorial staff for the helpful way in which they have handled the technical matters connected with publishing the book.

M. A. McI.

COOPERSTOWN, N. Y.

January 9, 1934

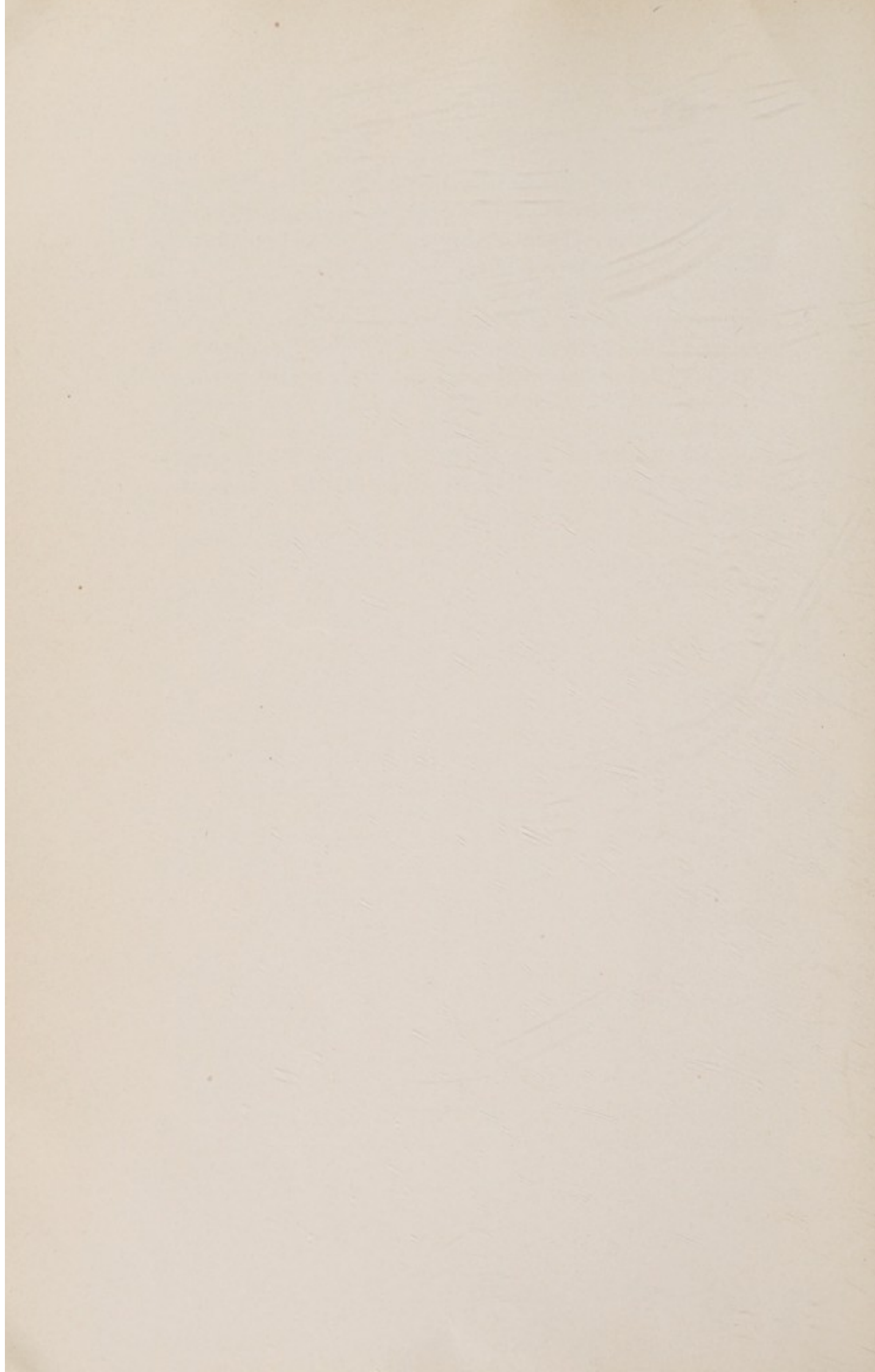




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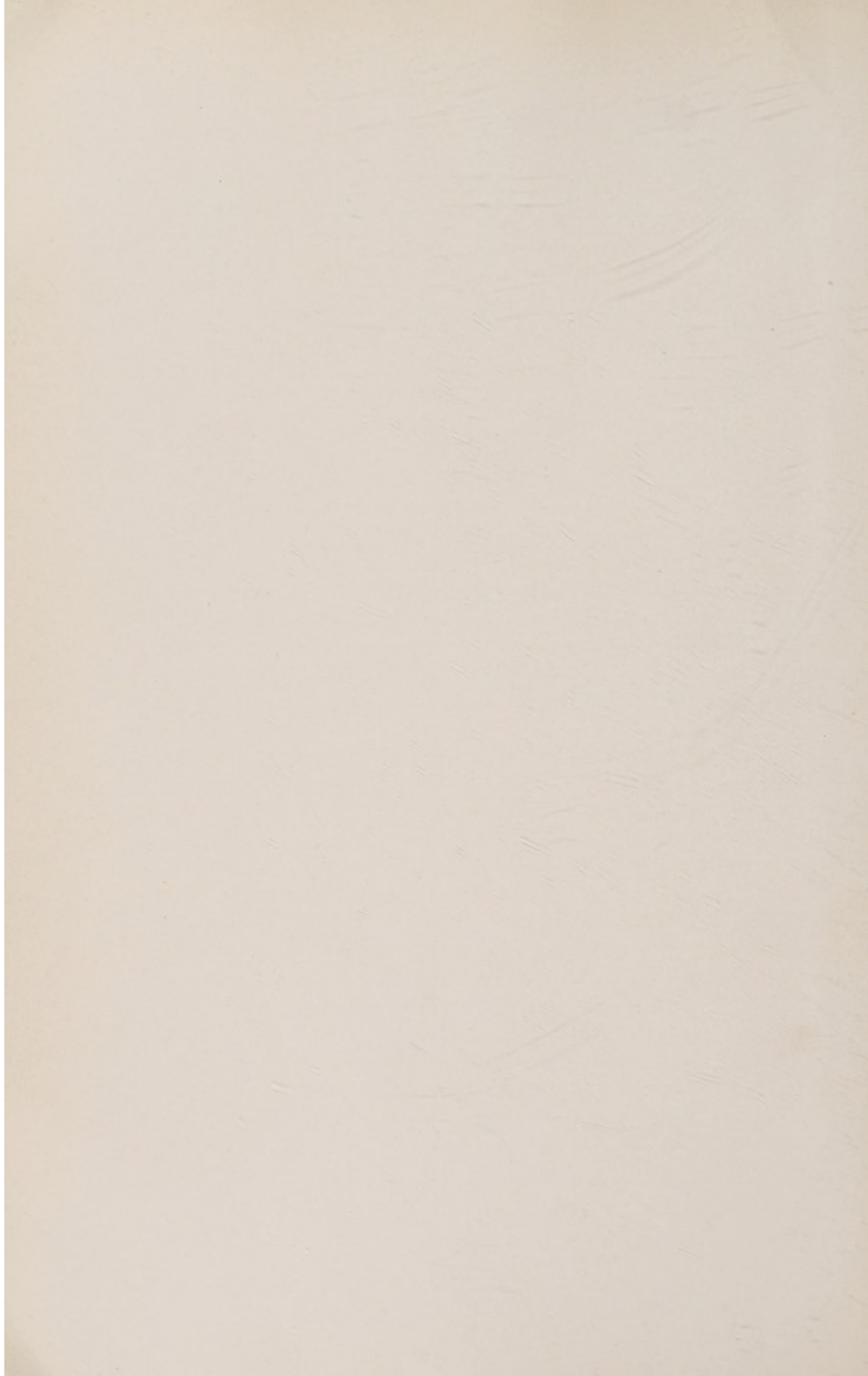


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PART I
THE DISEASE





ACUTE INTESTINAL OBSTRUCTION

CHAPTER I

INTRODUCTION

TO DEFINE concisely "acute intestinal obstruction" is impossible; for under that term, by universal consent, a number of diverse diseases or conditions are grouped. Among the characteristics which these conditions have in common, the one most consistently found is an abnormal stoppage of intestinal evacuations; and it is from this fact that the name is derived. This stoppage is not always brought about by an organic occlusion of the lumen, and, indeed, when mechanical blockage is present it is by no means always the most important element in the situation, for damage to the circulation of the bowel may be a more serious factor. Cases of intestinal obstruction, in most instances, have also in common a well-recognized set of signs and symptoms, of which pain, vomiting and distention are the most frequently encountered; but it is only in general terms that this symptomatology can be applied to the whole group, for certain of the classical symptoms are often absent. The mode of onset of the disease, the course, intensity and total duration of the illness, show striking differences in the several types of obstruction, varying from an onset of dramatic suddenness and a course of marked intensity and brief duration, such as is found in volvulus, where the patient may be desperately ill almost from the first moment and may die within two days or less, to a most insidious beginning and relatively slow course, such as is often seen in simple blockage of the colon. Turning from the clinical picture to a consideration of the etiology and pathology, it is apparent when the major types are contrasted that the causes

are various and unrelated, and the pathological pictures completely different, as, for example, the obstructions that arise in connection with peritonitis, and those caused by blockage of the lumen.

This lack of uniformity in clinical picture, etiology and pathology creates a situation which is complicated and lends itself to misunderstanding and confusion, more especially since in spite of its very broad meaning the term intestinal obstruction is often used as though it represented a single clear-cut entity. In order to gain a comprehensive picture of the whole disease, which is essential for any intelligent application of diagnosis or treatment or for interpreting the results of experimental investigation, groups must be established of cases that may logically be considered together, and an analysis must be made of their likenesses and differences. Before attempting such a study, it will be profitable to gain some perspective by reviewing briefly the principal steps in the development of our knowledge of the disease and the methods of treatment.

Three stages leading to our present conceptions of acute intestinal obstruction may be distinguished since the beginning of the 19th century. These stages of course overlap; but in a general way they show the principal steps in development. In the first period, roughly covering the whole of the 19th century, the development centers about the study of the anatomy and pathology of the disease and the relation of these to the clinical picture. To this period belongs the recognition of the different types and causes of obstruction and the mechanism by which they come about. The foundations had been laid by the anatomists of the 17th and 18th centuries, who had become familiar with the anatomical peculiarities of most of the major types of organic occlusion but had made little or no progress in relating their morphological findings to the clinical picture. It was not until the great renaissance of medicine in the early part of the 19th century that fairly complete and sound ideas concerning the pathology and symptomatology began to take

form, and cases that today would be grouped as intestinal obstructions began to be so classified and to be withdrawn from the heterogeneous collection which included under the title of "ileus" all types of peritonitis, typhoid fever, simple colic, ascites and most cases showing persistent vomiting, abdominal distention or abdominal pain.

The culmination of this stage of development came in the last twenty-five years of the 19th century and the first decade of the 20th, when the development of abdominal surgery made possible a much better knowledge of the pathological changes occurring early in the disease and of the finer points of classification and symptomatology. The achievements of this period will be found in the works of Brinton, Leichtenstern, Bryant, Ashhurst, Fitz, Nothnagel, Gibson, Treves, and Wilms.¹⁻⁹

The second period, starting in the latter part of the 19th century and culminating in the first quarter of the present century, is characterized by a study of the toxic factors. With the increasing number of abdominal operations and autopsies, the pathological picture presented by acute intestinal obstruction became clearer, and it was soon apparent that frequently these findings did not fully explain the acute symptoms and the prostration manifested by the patient during the illness, nor adequately account for the death that so often ensued. The search for less obvious factors which might explain the observed phenomena has centered around the belief that the course of events following intestinal obstruction could best be explained by the action of some powerful toxin. The rise of bacteriology gave stimulus and direction to the investigations. The bacterial invasion of the peritoneal cavity and blood stream was first investigated, but it was found that while this occurred in certain instances it would not account for the death of the patient in the far larger number of instances where the findings were negative. Attention was next turned to the contents of the obstructed bowel, which were obviously foul and grossly contaminated with bacteria; and it was easily proved by injecting this material into other animals that it was usually highly toxic.

The possibility that some factor other than bacteria may be responsible for the acute illness has also been studied at length: notably, that a toxic secretion is formed by the mucosa of the obstructed bowel or that certain of the digestive ferments become toxic under conditions of obstruction. A great deal of work has been done in an attempt to isolate and identify a single, chemically pure toxin from the obstructed bowel; but while a number of substances have been isolated that proved to be highly toxic, there has been no general agreement as to their actual role in the disease.

The workers in this second period are numerous. Particularly associated with it are the names of Von Albeck, Clairmont, Roger, Murphy and Vincent, Hartwell and Hoguet, Whipple, Draper, Brooks, Sweet, and Dragstedt.¹⁰⁻¹⁹

In the third and present period, the chief interest or point of advance has been a study of the less obvious bodily changes that come about as secondary results of obstruction, especially as shown by the chemical and physical changes occurring in the blood and body fluids, notably the marked dehydration and a lowering of the blood chlorides. That these secondary effects are of great practical importance and that their correction can prove in certain instances to be a life-saving measure has been thoroughly demonstrated by Hartwell and Hoguet,¹⁴ Haden and Orr,²⁰ and others. Since, however, these changes do not occur in all types of intestinal obstruction, when treatment calculated to correct them has been applied indiscriminately, the results have naturally been disappointing.

A knowledge of at least the basic facts established during these three periods, and their interrelation, is essential to anyone who is to have the clinical responsibility for patients with intestinal obstruction. The contributions of the first, or pathological, period are obviously fundamental, for as has already been pointed out, without a clear conception of the various major types of obstruction and their peculiar pathology the picture is one of utmost confusion. During the second period, the studies of the toxic factors in the disease have thrown much

light upon the bacteriology, chemical nature, mode of absorption and biological effects of the contents of the obstructed bowel, although the correct evaluation and interpretation of many of these experiments is still in doubt. The investigations made during the third period concerning the changes in the blood and body fluids that occur in certain types of obstruction and the measures that may be taken to correct them, are of first importance and can be understood only if one has a clear conception of the fundamentals of the altered physiology and the pathology in different types of obstruction.

Therapeutic measures have, of course, been influenced by all these findings; but surgical treatment has rarely followed systematically on the advance of the theoretical knowledge of a disease, and in its early beginnings was empirical and designed to relieve certain symptoms.

Attempts to treat by surgical measures cases of intestinal obstruction have from very early times to the present proceeded along two main lines: first, by indirect methods whose original object was to relieve certain symptoms, particularly distention, by draining the intestine above the point of obstruction; and second, by operations intended to find and remove the cause of the obstruction.

The most primitive of the indirect methods,—tapping the distended coils of intestine by blind puncture through the abdominal wall with a hollow needle,—goes back to very early times, when this operation (paracentesis) was used to relieve distention of the abdomen both from ascites and from accumulations in the intestine. The making of an external opening in the colon for relief of distention of that organ due to obstruction has been practiced since the beginning of the 18th century at least, for Littré²¹ described this operation in 1710. A later modification by Amussat²² consisted in opening the colon in the left lumbar region; his name was frequently applied to this form of colostomy. It was apparently, however, not until over a hundred years later that Dupuytren²³ established drainage from the small intestine above the point of obstruction (enter-

otomy*); and it remained for Nélaton,²⁵ whose name was usually given to the operation, in about the middle of the last century, to establish this as an accepted surgical procedure carefully thought out and executed.

The value of enterostomy was soon realized and it is in use today, modified in technique and choice of location but not in principle. Obviously, however, this procedure could usually relieve only cases of simple blockage of the bowel and did little or nothing to relieve the more frequent cases where in addition to the occlusion of the lumen there was interference with the mesenteric circulation to the involved segment.

Almost simultaneously with the employment of these indirect operations, direct attempts were made to relieve the condition by opening the abdomen and removing the obstruction. Before the days of asepsis and anesthesia, these procedures were indeed desperate adventures and of course few in number. The first reasonably authentic report was made in 1679 by Bonetus,²⁶ and describes an intussusception successfully reduced by a young surgeon who "had long followed the camps." About two hundred years later (1874), Ashhurst²⁷ was able to collect only 74 cases, in 14 of which the operation was carried out for obstruction from intussusception and in the remaining 56 for causes "other than intussusception."† Fourteen years later, however, the number of recorded cases had risen to 328;²⁸ and after that time, with the growth of abdominal surgery, the number of direct operations became legion.

About thirty years ago, the last edition of Treves' classical contribution, "Intestinal Obstruction," appeared. In addition to giving Treves' own contribution to the subject, this volume summarizes the work of Brinton, Leichtenstern, and others, and thus may be taken as the foundation for any further treatment of the subject; the descriptions of the anatomical

* Travers,²⁴ in 1812, stressed the value of the establishment of an intestinal fistula in cases of strangulated external hernia.

† Ashhurst's²⁷ articles carried a comprehensive and interesting account of the early history of operations for intestinal obstruction, furnishing a total list of the early cases, and a number of abstracts of the histories.

and pathological aspects of the disease furnished by these earlier workers are not likely to be surpassed at any time. During the last thirty years, however, important and radically new concepts of the disease and its treatment have been made. The uses and limitations of various types of surgical procedure have been revised; and a better understanding has been reached of the toxic factors in the various types of obstruction. The secondary changes that occur in the blood and body fluids as a result of the obstruction, and the importance of the prompt use of adequate amounts of physiological salt solution—the specific treatment that can correct these otherwise fatal changes—have been recognized. Supplementing this work has come a better comprehension of the importance of the inorganic chemical constituents of the digestive juices, especially of the sodium and chloride ions, and the relation that these substances bear to the subject of dehydration. The introduction of the roentgen ray has not only added to the knowledge of the motor functions of the intestinal canal, but has revolutionized the diagnosis of intestinal diseases, especially in relation to obstructions of the colon, where the use of the barium enema in connection with the x-ray can not only demonstrate the site of an obstruction but also lead to the early diagnosis of lesions, notably carcinoma, which if neglected would inevitably produce an acute obstruction. The use of the duodenal tube has been a great contribution to the preoperative and postoperative care of patients, especially as regards the prevention and treatment of functional obstructions.

In the following chapters an attempt is made to present the new concepts of the disease and to interpret the results of the important recent investigations in relation to the clinical field.

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CHAPTER II

GENERAL CONSIDERATIONS

Cases of acute intestinal obstruction must be considered first in two main groups which differ completely from each other in etiology and show considerable variations in symptomatology and pathology: first, obstructions due to mechanical factors; second, those of functional origin.

The old word "ileus," as a term for intestinal obstruction, is not used in this monograph. In the early days it was used to denote a heterogeneous list of abdominal diseases; there still is not complete agreement as to its use, and there is a tendency on the part of certain authors to discard the term.¹ When it is used it is generally subdivided as follows²:

Mechanical obstructions	Mechanical ileus
	{ Paralytic obstructions; absence of peristalsis = Adynamic ileus
Functional obstructions	{ Spastic tonic condition of intestinal musculature = Dynamic ileus

Some writers,¹ however, refer to mechanical obstruction as "adynamic ileus"; while other authors use the term "ileus" simply to refer to the paralytic obstructions. The terminology that will be used in the remainder of this volume will be "mechanical obstructions" and "functional obstructions," the latter being subdivided into paralytic and spastic obstructions.

The group of mechanical obstructions, with which this book chiefly deals, is a more clear-cut entity than the group of functional obstructions, and is, generally speaking, a more serious disease. Into the group of obstructions due to functional disturbances of intestinal motility is gathered a somewhat ill-defined collection of cases, the etiology and pathology of which are not very thoroughly understood. These cases are

by no means to be disregarded, however, for they often present grave difficulties in diagnosis and treatment.

CAUSES OF MECHANICAL OBSTRUCTIONS

Mechanical obstructions of the intestinal canal come about in a number of quite dissimilar ways. The bowel may be occluded by pressure from without, as, for example, by a constricting band. Or, the occluding element may lie within the lumen as it does in obstructions caused by neoplasms or foreign bodies. A quite different mode of occlusion is found where the intestine is twisted or kinked in such a manner that the lumen is obliterated, the so-called volvulus. And still another method is represented by the intussusceptions, where the intestine is occluded by a process of invagination within itself.

While the exact pathology found in different types of mechanical obstruction varies widely, the conditions fundamentally responsible for most of the acute obstructions are: developmental defects, inflammatory reactions within the peritoneal cavity, and neoplasms.

Under the heading of developmental defects come, first, the obstructions of early infancy due to imperfect development of the digestive tract, as, for example, atresias and other anomalies of this class^{3,4}; and, second, the obstructions coming on later in life as a result of developmental structures or anomalies within the abdominal cavity, such as Meckel's diverticulum, remnants of the urachus, the appendix, congenital bands, ileocecal⁵ and mesenteric cysts,⁶ ectopic spleen,⁷ and so forth. Congenital anomalies of the mesentery and abnormalities of intestinal rotation may at times be responsible for obstructions.⁸⁻¹⁰ The large group of obstructions caused by hernias must also fundamentally be considered as resulting from developmental defects.

That inflammatory reactions in the peritoneal cavity are responsible either directly or indirectly for a large group of obstructions is obvious. The bands, adhesions, inflammatory lymph glands, etc., that arise from this cause are the result not

only of bacterial inflammation of the peritoneum but also of mechanical injury, usually incidental to abdominal operations. During the last forty years that have marked the rise of

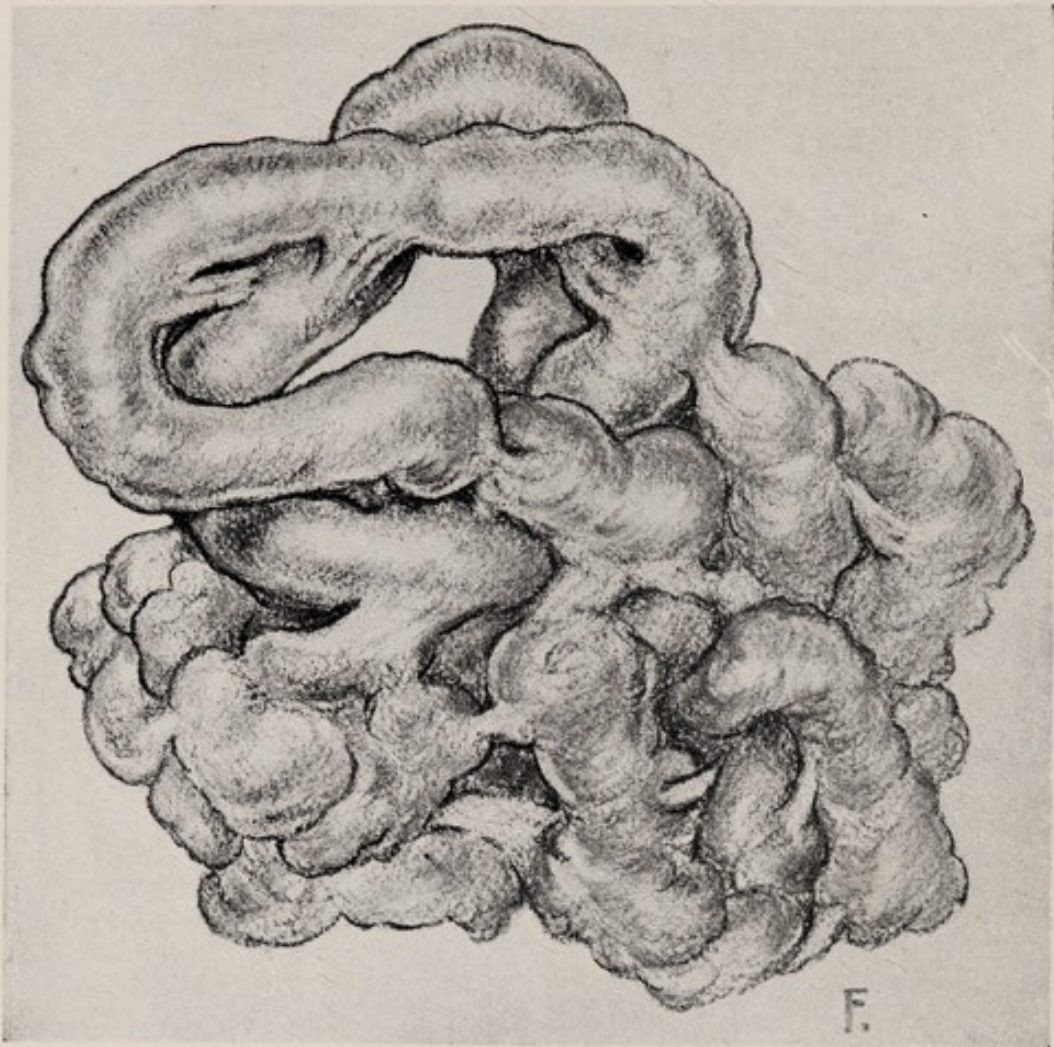


FIG. 1. Numerous bands and adhesions between loops of small intestine.

This patient had a duodenal ulcer and also sub-acute obstruction. Bands and adhesions were due to a healed peritonitis, etiology of which was uncertain.

abdominal surgery, obstructions following laparotomies have formed an increasingly large proportion of the total cases¹¹; (see Table xx, p. 326). Chronic ulcerative processes, usually tuberculous,¹² rarely syphilitic,¹³ involving the bowel wall itself may lead to a stricture of the intestines and subsequent obstruction. Obstructions of this type are usually

subacute or chronic; but occasionally the blockage may be complete, or a foreign body may become impacted in the narrowed lumen and convert a chronic into an acute obstruction. Benign strictures may also result from non-specific inflammatory changes.¹⁴ An important cause is the inflammatory change which may follow serious interference to the mesenteric blood supply: at times, although the intestinal loop survive, an extensive inflammatory reaction may be set up and the gut wall be converted into scar tissue, with marked narrowing of the lumen.¹⁵

The third fundamental cause is neoplasms, which in the great majority of cases actually rise from the bowel itself and usually cause the obstruction by producing stenosis of the lumen. Since cancer of the colon makes up the great majority of all tumors of the intestine, obstructions from neoplasms are usually found in the large intestine.

CLASSIFICATION OF CASES

In order to consider the various types of obstruction in detail, some grouping of cases is necessary. Different clinics have adopted different methods, no one of which alone is completely satisfactory. The following classification, based partly on etiology and partly on clinical features, has been in use at the Massachusetts General Hospital for thirty years or more,¹⁶⁻¹⁸ and is adopted here as affording a working basis:

Obstruction by bands and adhesions, occurring:

 Early after operation

 Late after operation

 Without previous operation

Obstruction by volvulus

Obstruction by intussusception

Obstruction by rarer causes:

 Congenital anomalies

 Gallstones and other foreign bodies

 Internal hernias

 Meckel's diverticulum

Obstruction by mesenteric thrombosis and other vascular lesions

Obstruction by neoplasms

Obstruction by strangulated external hernias.

INCIDENCE OF THE VARIOUS TYPES OF OBSTRUCTION

The incidence of the various types of obstruction already outlined is shown, for a series of 335 cases recently studied,¹⁸ in Figure 2.*

It will be observed that strangulated external hernia is the etiological factor in almost one-half the total number of cases; bands and adhesions are the cause of another large group; neoplasms are responsible for a third; intussusceptions and volvuli come next; while the remaining cases are in the rarer groups.

The figures from different clinics are of interest, but some difficulty is encountered in trying to make a composite picture of the incidence of the different types. The term "intestinal obstruction" is such a broad one that different investigators may limit their studies at different points (one, for example, may exclude obstructions from carcinomas; another may omit cases complicated by peritonitis), or, again, the same types of obstruction may be included but they may be grouped under different headings. The points of difference in selection and grouping of cases are illustrated by Table 1, which gives the classifications employed in various large clinics. It will be noted that Braun and Wortmann¹⁹ omit strangulated external hernias from their series. There is also a difference in the consideration of the important group of obstructions complicated by peritonitis: many of these cases present particular difficulties in classification because there is often both a functional and a mechanical element and it is hard to say which predominates. Braun and Wortmann group obstructions with peritonitis separately. In the figures from the

* These data represent a ten-year period at the Massachusetts General Hospital. During the same period the total number of surgical admissions was 39,936 or about 4000 a year.

Massachusetts General Hospital, cases of peritonitis with the paralytic functional type of obstruction are excluded; a certain number of cases, however, where peritonitis was present

INCIDENCE OF TYPES OF OBSTRUCTION IN A TEN-YEAR SERIES OF CASES AT MASSACHUSETTS GENERAL HOSPITAL

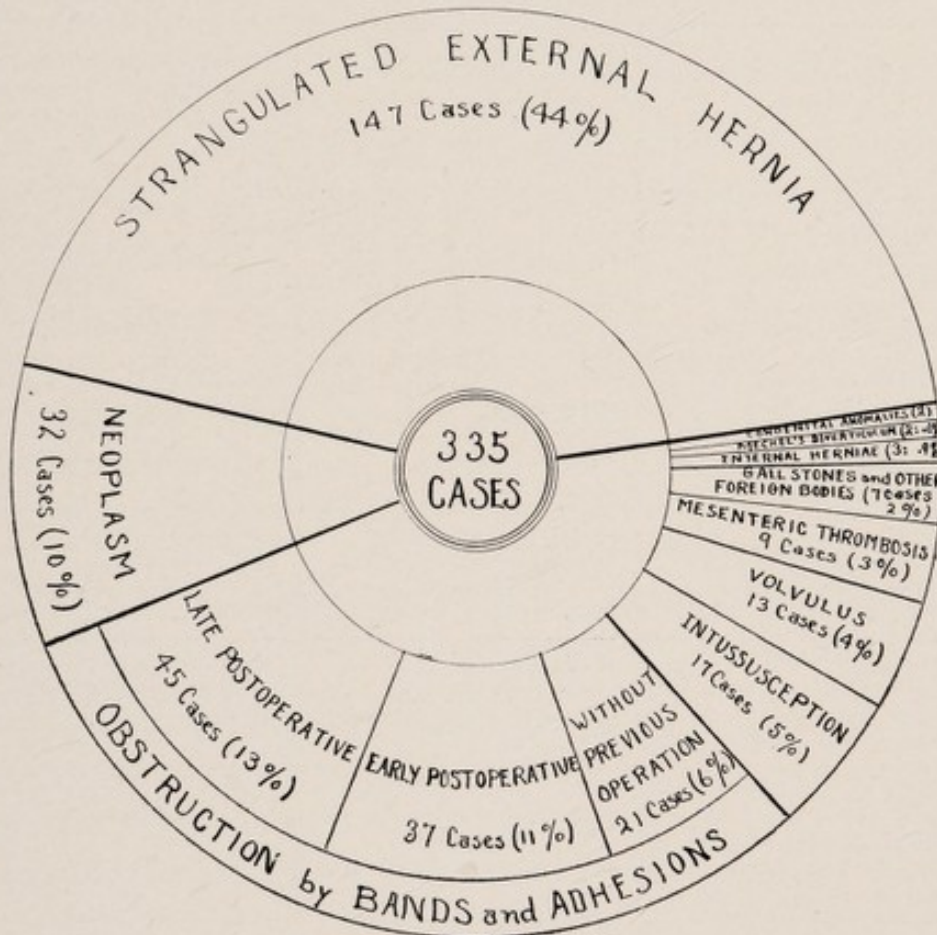


FIG. 2.

It will be observed that strangulated external hernia was the etiological factor in almost one-half the total number of cases; bands and adhesions were the cause of another large group; neoplasms were responsible for a third; intussusceptions and volvuli came next; while the remaining cases were in the rarer groups. (McIver¹⁸)

as a complication but where the mechanical factor of inflammatory adhesions was the important element, have been included under "early postoperative obstructions." Souttar makes no mention of peritonitis in his table.²⁰

There are four groups that are usually given the same titles: volvulus, intussusception, tumors (or carcinomas) and

gallstones. It will be noted in Table 1 that the percentage incidence of the two latter types at the different hospitals is fairly uniform. From the literature one has the impression

TABLE 1
INCIDENCE OF VARIETIES OF OBSTRUCTION AT DIFFERENT CLINICS

Massachusetts General Hospital 1918-1927		Statistics by Braun and Wortmann, Berlin, 1903-1922		Combined Statistics from Seven London Hospitals, Reported by Souttar in 1925	
Type of Case	Number of Cases	Type of Case	Number* of Cases	Type of Case	Number of Cases
Early Postoperative (bands and adhesions).....	37 (11%)	Obstruction with Peritonitis.....	44		
Late Postoperative (bands and adhesions).....	45 (20%)	Bendings, Kinks, etc. Intestinal knots.....	94 4	Adhesions.....	342 (18%)
Bands and Adhesions without previous op.	21	Strangulation†.....	64	Internal strangulation.....	223
Internal Hernias.....	3 (1%)	Hernias, internal and diaphragmatic.....	8	(Internal hernias incl. under above)	
Volvulus.....	13 (4%)	Volvulus.....	63	Volvulus.....	74 (2%)
Intussusception.....	17 (5%)	Invagination.....	43	Intussusception‡....	630 (21%)
Gallstones and Other Foreign Bodies.....	7 (2%)	Gallstones.....	7	Gallstones.....	28 (1%)
Mesenteric Thrombosis	9 (3%)				
Congenital Anomalies..	2 (1%)				
Meckel's diverticulum..	2 (1%)				
Neoplasms.....	32 (9%)	Carcinoma of the large intestine.....	48	Carcinoma.....	358 (11%)
Strangulated External Hernias.....	147 (44%)		...	Hernias.....	1409 (46%)
		Inflammatory stricture of small intestine.....	4		
Totals.....	335		379		3064

* Strangulated external hernias are not included in the series of Braun and Wortmann; since, therefore, the percentages would not be comparable with those of the other two series, they are not given.

† Strangulations are divided by Braun and Wortmann into two groups: "Strangabklemmungen" (54) and "Strangabklemmungen"(10).

‡ Souttar divides his intussusceptions into those with tumors (17) and those without (613).

that volvulus is more common in the European clinics than in this country; and this is borne out by the figures of this table. The high percentage of intussusceptions reported by Souttar is explained by the fact that intussusception is predominantly a disease of infancy and early childhood, and the number of cases reported in any series will be influenced by the ratio of adults to children admitted to the clinic furnishing the figures.

DISTRIBUTION OF CASES BY AGE

Figure 3 illustrates the incidence of the different varieties of obstruction at different ages, and brings out certain interesting points.

It will be noted that obstructions from congenital anomalies and from intussusceptions occur for the most part in infancy and childhood. Obstructions arising from bands and adhesions in patients not previously operated upon showed, in this series, two definite peaks: one in the second and third decades and another in old age, middle life being relatively free; this is a rather striking finding, but may be simply a coincidence in this small group. Bands and adhesions occurring either early or late after operation are found for the most part in young adults or those in middle age, corresponding to the period of greatest frequency of abdominal operations. Mesenteric thromboses and other circulatory lesions reach their peak in old age, although cases do occur as early as the third decade. Obstructions from neoplasms as might be expected reach their peak in old age. In the group of strangulated external hernias, while all ages are represented, a very striking peak occurs in the fifth and sixth decades; and while there is no actual peak in early childhood, it is interesting to note that except for congenital anomalies and intussusceptions strangulated external hernia is the only cause, in this series, of obstruction in the first year of life.

DISTRIBUTION BY SEX AND RACE

Intestinal obstruction occurs far more frequently in males than in females. In the series of cases recently studied at the

DISTRIBUTION OF VARIOUS TYPES OF OBSTRUCTION ACCORDING TO AGE OF PATIENT

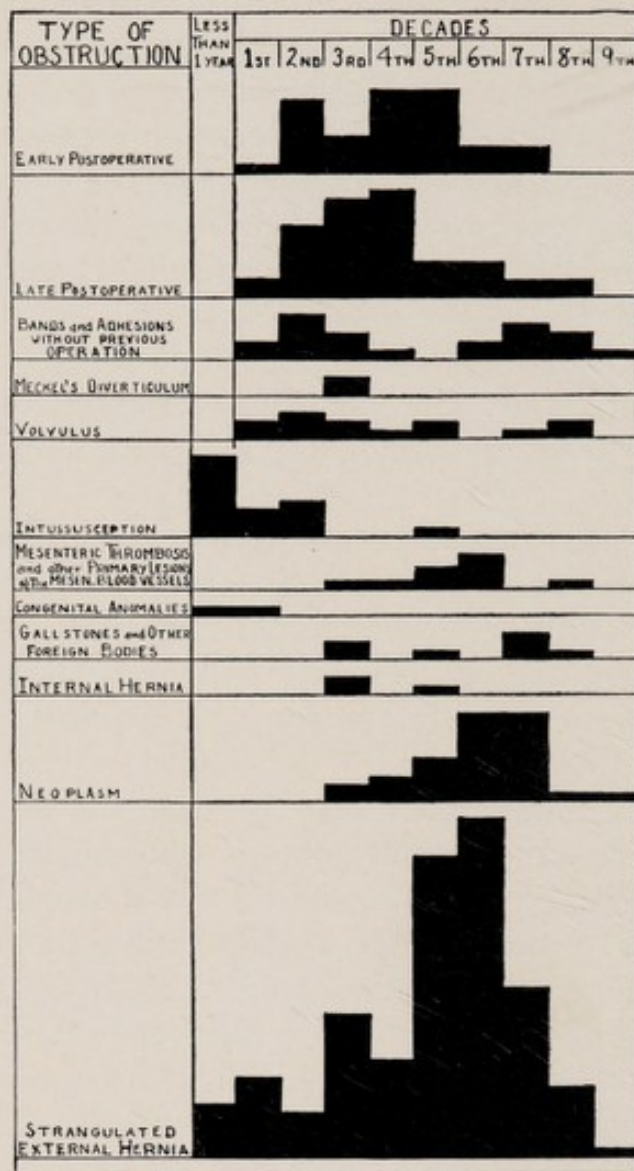


FIG. 3.

It will be noted that obstructions from bands and adhesions occur for the most part in young adults and those of middle age. Mesenteric thromboses and other circulatory lesions reach their peak in old age. Obstructions from neoplasms, as might be expected, also occur for the most part after the fifth decade. In the group of strangulated external hernias, while all ages are represented, a very striking peak occurs in the fifth and sixth decades; and while there is no actual peak in early childhood, it is interesting to note that except for congenital anomalies and intussusceptions, strangulated hernia was the only cause, in this series, of obstruction in the first year of life. Intussusceptions occur almost entirely in infancy and early childhood. (McIver¹⁸)

Massachusetts General Hospital there were 216 males and 119 females. Among all the varieties there was only one group in which the obstruction occurred appreciably more often in females than in males, namely the group of "gallstones and other foreign bodies," where there were only 2 males and 5 females.

Acute intestinal obstruction is somewhat more common in the negro race than in the white.²¹⁻²³ The reason for this is not known; possibly it is related to the fact that keloid formations are more common in negroes.

IMPORTANT FACTORS AFFECTING ALL TYPES OF MECHANICAL OBSTRUCTION

Before going on to a detailed analysis of the different types of mechanical obstruction, two factors which largely determine the nature of the disease in each particular case may be considered. These are, first, the condition of the circulation of the bowel; and, second, the level of the obstruction.

CIRCULATION TO THE BOWEL. It must be emphasized that the most important factor governing the pathology and symptomatology, and largely determining the severity of the illness, is, paradoxically enough, not the blockage of the intestinal stream but the condition of the circulation to the involved segment of bowel. Interference with the circulation of the bowel may come about in two ways. First there may be gross interference with the mesenteric circulation (strangulation); second, the capillary circulation in the wall of the bowel itself may be damaged by the increased intrainestinal pressure of extreme distention, or local changes may be produced by the pressure of a band or foreign body at the point of obstruction.

Mesenteric Circulation. It is necessary to remember that the blood supply to a great part of the bowel is vulnerable: the free motility of large portions of the intestinal tract is due in part to the fact that the intestine is attached to the posterior abdominal wall by the delicate web-like mesentery which

carries but affords little protection to the arteries and veins supplying the intestinal circulation. From the point of view of function, the mesentery is as much a part of the intestinal

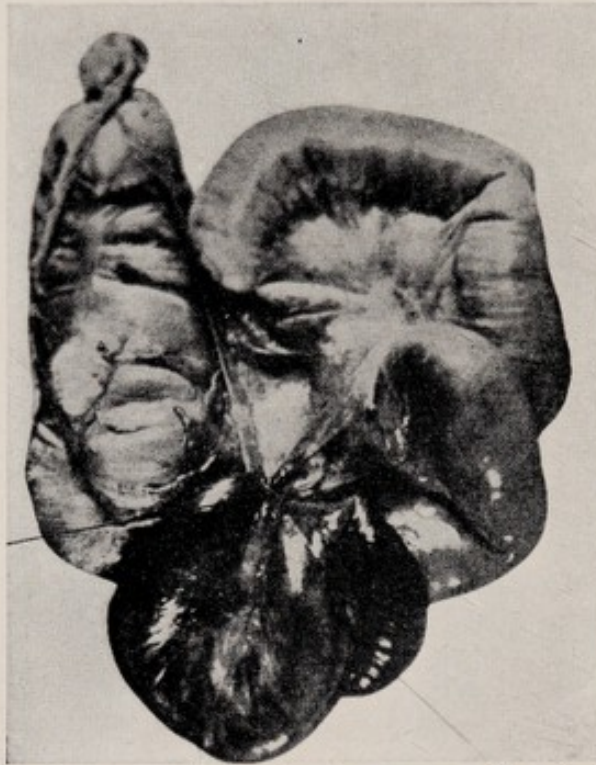


FIG. 4. Strangulation by a constricting band; gangrene of involved loop. (Braun and Wortmann¹⁹)

tract as the air-line is of a diver's suit, and the compression of either is equally disastrous. This grouping of the mesentery and the intestine together is of great practical importance, for most of the twists, knots, invaginations and strangulations that obstruct the intestinal lumen also in varying degrees snarl or compress the mesentery.

In cases of obstruction where the mesenteric circulation is impaired, the severity of the illness is, broadly speaking, proportional to the degree of interference. Where the interference is extensive, the course of the disease is fulminating in the extreme and the pathology and the alterations of the physiology are quite different from what they are in cases where there is

merely blockage of the intestinal lumen; there is little time for secondary effects, such, for example, as would result from the loss of digestive secretions by vomiting. If the obstruction is not relieved, the patient dies of a toxemia or a general peritonitis, and at autopsy gross changes in the bowel wall are observed (Fig. 4). The more serious nature of this type of obstruction, usually termed "strangulation," as contrasted with simple obstruction has been an established fact in the surgical literature for a long time, having been accurately summarized in 1885 by Bryant.*²⁴ See also p. 346.

Table II, A and B, shows the incidence and effect on mortality of interference with the circulation in cases of acute intestinal obstruction from causes other than neoplasm and strangulated external hernia. It will be noted that 66 of the 156 cases listed in Table II, A, showed some degree of interference with the mesenteric circulation, although in many instances this was not extreme, and that the mortality among these patients was 53 per cent as contrasted with a mortality of 37 per cent in patients without this complication. In Table II, B it will be seen that in a thirty-year period about half the patients had interference with the mesenteric circulation, and that the mortality was 62 per cent as contrasted with a mortality of 41 per cent among patients where the mesenteric blood supply was not disturbed. It is interesting to note in this table that the proportion of patients having this serious complication was about the same in the three ten-year periods.

*Bryant writes: "Hitherto it has been the custom to place cases of strangulation of the bowel amongst those of obstruction, indeed, to consider them as only one of its forms. There is, I am convinced, in this arrangement a grievous error; since, in strangulation of the intestine, obstruction is only one of its symptoms, but not the cause of danger or of death; whereas, in cases of intestinal obstruction, the obstruction is the prominent and dangerous feature, and from it, or it chiefly, the consecutive changes are brought about."

And again: "In fact, in this instance, as, in all other cases of acute or a like nature [strangulation], the symptoms are directly due to the arrest of the circulation of the venous blood through the strangulated bowel, and not to the obstruction to the passage of the intestinal contents. The action of the bowels may, as a clinical symptom, be one of value to prove the patency of the intestinal tract, and to suggest, consequently, the completed removal of the cause of the strangulation; but it is well to remember that the symptoms excited by strangulation of the bowel are not due to obstruction alone."

Among the obstructions by neoplasm (Table III) only one case showed interference with the mesenteric circulation. This is due, of course, to the fact that in the great majority of these cases the obstruction comes about through blockage of the lumen by a growth originating within the bowel wall.

All the cases due to strangulated external hernia (Table III), by definition, showed some degree of interference with the mesenteric circulation. These cases, however, from the point of view of early diagnosis, ease of management, and absorption from the involved loop, present features distinctive from internal strangulations and are discussed in detail later.

Circulation in the Bowel Wall. The small blood vessels coursing in the relatively thin bowel walls are subject to considerable distortion when distention occurs, and the circulation is correspondingly impaired. The view that increased pressure impairs the circulation and causes subsequent damage to the

TABLE II
INTERFERENCE WITH MESENTERIC CIRCULATION
A

Classification	Number of Cases	Number Showing Interference with Circulation	Mortality in Cases with Interference (Per Cent)	Mortality Cases without Interference (Per Cent)
Postoperative, early.....	37	2 (5%)	100	43
Postoperative, late.....	45	17 (38%)	29	28
Bands and Adhesions Without Previous Operation.....	21	8 (38%)	50	38
Meckel's Diverticulum.....	2	2 (100%)	0	0
Volvulus.....	13	9 (69%)	55	25
Intussusception.....	17	17 (100%)	53	0
Mesenteric Thrombosis.....	9	9 (100%)	100	0
Congenital Anomaly.....	2	0	0	100
Gallstones and other Foreign Bodies.....	7	0	0	29
Strangulated Internal Hernias.....	3	2 (67%)	50	0
Total.....	156	66 (42%)	53	37

TABLE II. (Continued)

B

THIRTY-YEAR PERIOD, SAME TYPES OF CASES AS THOSE SHOWN IN TABLE II-A

<i>Period</i>	<i>Number of Cases</i>	<i>Number Showing Interference with Mesenteric Circulation</i>	<i>Mortality in Cases with Interference (Per Cent)</i>	<i>Mortality in Cases without Interference (Per Cent)</i>
1898-1907	121	59 (49%)	78	57
1908-1917	118	54 (45%)	55	30
1918-1927	156	66 (42%)	53	37
Total.....	395	179 (45%)	62	41

Table II, A and B, shows the number of cases in the various types of obstruction (exclusive of those caused by neoplasm or strangulated external hernia) in which there was interference with the mesenteric circulation, and its effect upon mortality. It will be noted that in the obstructions occurring early after operation this complication is relatively infrequent. (Scudder,¹⁶ Richardson,¹⁷ and McIver.¹⁸)

TABLE III

<i>Classification</i>	<i>Number of Cases</i>	<i>Number Showing Interference with Circulation</i>	<i>Mortality in Cases with Interference (Per Cent)</i>	<i>Mortality Cases without Interference (Per Cent)</i>
Neoplasms.....	32	1* (3%)	100	28
Strangulated External Hernias.....	147	147 (100%)	18	0

* There were also 2 cases where neoplasm caused mesenteric thrombosis; these are listed under that heading.

In Table III it will be noted that interference with the mesenteric circulation in obstruction by neoplasm is rare.

It will be observed that the mortality in strangulated external hernias is low in spite of the fact that there is interference with the mesenteric circulation in every case. In this type of obstruction there are certain other important factors, to be discussed later, which account for the lower mortality. (McIver¹⁸)

bowel wall is old, having been strongly advocated in 1898 by Kocher.²⁵ The dusky and at times markedly cyanotic appearance of the distended intestine constitutes a striking finding at operation. When the capillary circulation is damaged degenerative changes occur in the gut and have been reported both following experimental obstructions and at autopsies on humans.^{26,27} Van Beuren²⁷ points out that any increase in the diameter of the bowel is tripled in the circumferential measurement. It is obvious, therefore, that severe degrees of distention

result in great thinning of the intestinal wall, with stretching and flattening of its blood vessels,—the maximum interference with the circulation occurring on the antimesenteric border, where areas of infarction and perforation may at times result.

Great distention often accompanies obstruction of the colon by neoplasm. This is particularly true in obstructions of the sigmoid when partial obstruction has existed for some time before becoming complete. In these cases the cecum is particularly likely to suffer from the effects of distention, areas of necrosis and perforation occurring on the anterior surface where the circulation is most vulnerable. In the 32 obstructions from neoplasms listed in Table III, there were 4 cases in which there was obvious damage from distention, perforation or the beginning of necrosis of the cecum being found at operation. The interference with the circulation in the bowel wall is undoubtedly of importance also in those cases where it has not progressed to the extent of producing areas of infarction or gangrene, and may be an important factor in producing damage to the mucosa, which may in turn permit absorption of some toxic substance from the obstructed intestine. It is often difficult to estimate grossly the extent of this interference. Among 156 cases shown in Table II, A, for example, while 66 cases showed interference with the mesenteric circulation, there were only 9 instances in which it was evident from the description of the bowel at operation that extensive damage to the capillary circulation in the wall had resulted from distention; in 4 of these cases, bloody peritoneal fluid was found at operation. Interference with the capillary circulation of the bowel wall, however, is probably an important factor in a larger proportion of cases than is indicated by these figures. (See also pp. 140, 175 and 380.)

The local changes produced by pressure of a band or foreign body at the point of obstruction are discussed on page 141.

LEVEL OF THE OBSTRUCTION. In the group of cases where there is simple obstruction of the lumen of the intestine and

the mesenteric circulation is intact, the marked variations in the course of the disease are largely determined by the anatomical level of the obstruction in the intestine, particularly with reference to its location in the large or the small intestine.

When one considers the great differences in the more obvious functions of the several parts of the gastrointestinal tract, it seems reasonable that occlusion of one part should result in a train of events quite different from that following occlusion in another portion. For example, into the upper part of the small intestine an enormous amount of digestive secretions containing sodium and chloride ions of vital importance is poured during the course of twenty-four hours,—an amount, as will be shown later, probably equal in volume to that of the total circulating blood. If, therefore, occlusion occurs high in the intestinal tract, these fluids and electrolytes can of course not be absorbed in the lower portion; and frequently they are lost from the body in the excessive vomiting which is likely to take place. The loss of these fluids and salts is serious. In obstructions of the colon, on the other hand, in the early stages at least, absorption from the small intestine can go on to some extent and vomiting is not likely to take place in large amounts; in the colon, furthermore, all degrees of stagnation of fecal material occur more or less normally, so that adaptation to conditions of obstruction can take place more adequately and be carried on for a longer period of time. It is, therefore, natural that the illness produced by a simple obstruction of the colon is likely to run a longer, less severe course than an obstruction located in the small intestine.* Broadly speaking, but not invariably, it is also true that obstructions located in the lower ileum do not cause as fulminating an illness as those located in the jejunum or duodenum;† but while one can usually differentiate clinically between an obstruction in the colon and one in

* The clinical difference between obstructions located in the large and in the small intestine has been recognized for a long time. Brinton²⁸ wrote in 1867: "On the whole, it may be safely affirmed that obstruction in the small intestine runs a more rapid course, is marked by symptoms of greater severity, and is attended with greater danger, than when located in the large intestine. It is easy to understand this contrast, though diffi-

the small intestine, it is often difficult, except in a most general way, to attempt on the basis of signs and symptoms to determine the level of an obstruction of the small intestine.

Table IV shows the distribution of the obstructions at different levels of the intestine in a series of 335 cases. The number of cases occurring in the small intestine was 249; in the large and small intestine together, 27; in the colon, 52. Of those

TABLE IV
LEVEL OF THE OBSTRUCTION

<i>Part of Bowel Obstructed</i>	<i>Classification of Cases</i>			
	<i>Group I*</i>	<i>Group II</i>	<i>Group III</i>	<i>Total Cases</i>
Jejunum.....	7	1	0	8 (2%)
Ileum.....	59	2	0	61 (18%)
Small Intestine..... (not further specified)	53	0	127	180 (54%)
Ileocolic Intussusception.....	14	0	0	14 (4%)
Large & Small Intestines.....	0	0	13	13 (4%)
Large Intestine.....	16	29	7	52 (16%)
Not Determined.....	7	0	0	7 (2%)
Totals.....	156	32	147	335

* Group I : All cases except those due to neoplasms or strangulated external hernias.

Group II : Obstructions due to neoplasms.

Group III: Obstructions due to strangulated external hernias.

Table IV shows the portion of the bowel obstructed. It will be noted that in 74 per cent of the cases the point of obstruction was located in the small intestine; these obstructions were for the most part in the lower small intestine; there were only 8 cases in which the obstruction was located in the jejunum. Both the large and the small intestine were involved in 8 per cent of the cases; the large intestine alone in 16 per cent. (McIver¹⁸)

cult adequately to resolve it into all its elements. Not only has the large intestine functions which are less complex, less delicate, and less indispensable to life than those of the jejunum and ileum, but even its greater size and mechanical distensibility, perhaps also its special powers of absorbing liquids, render its mere obstruction of less moment;—we may almost say, less divergent from its normal condition.”

† In the experimental work with dogs there is often an extraordinary difference between obstruction in the upper and in the lower portion of the small intestine; high obstruction produces death in a few days, the animals with low obstruction living much longer. This has colored the statements in regard to human cases where the differences are not so well defined.

in the colon, 29 were caused by neoplasms. There is one group of high obstructions occurring in connection with a posterior gastroenterostomy, in which either the distal or the proximal loop of the upper jejunum that was used for the anastomosis becomes obstructed. These cases are by rather universal consent not grouped under the ordinary headings of acute intestinal obstructions but are described as "malfunctioning posterior gastroenterostomies" or "vicious circle after posterior gastroenterostomy."* If these cases were included in the series in Table IV, the total percentage of high obstructions would be in the neighborhood of 6 per cent instead of 2 per cent; but even so, the frequency of high obstructions is relatively low; for the relation of this fact to treatment of dehydration, see p. 331.

SUMMARY. There are two main groups of obstructions: mechanical and functional. (Functional obstructions are discussed later in the monograph.) Mechanically the bowel may be obstructed by pressure from without, by internal occlusion of the lumen, by bends and twists, and by invaginations; the important fundamental causes are developmental defects, inflammatory reactions and neoplasms. For the purpose of analysis in subsequent chapters, the cases are grouped on the basis of etiology and clinical manifestations.

Two factors largely determine the course of the disease. The first of these is the condition of the circulation in both the mesentery and bowel wall. The mesentery is a vital part of the intestine, and if it is twisted or compressed (strangulated) the result is disastrous; damage to the circulation in the bowel wall by distention or by local pressure may also have serious consequences. The second important factor is the level of the obstruction. Simple obstructions of the colon are not so rapidly fatal as those of the small intestine; and, generally speaking, the higher the obstruction in the small intestine the more fulminating the course of the disease. High obstructions of the small intestine are relatively rare.

*These are probably the most comparable to experimental simple, high obstructions.

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CHAPTER III

VARIETIES OF OBSTRUCTION

OBSTRUCTION BY BANDS AND ADHESIONS

ETIOLOGY AND PATHOLOGY. When the etiology of the bands and adhesions within the peritoneal cavity is considered, it may be said in general that these structures either are congenital

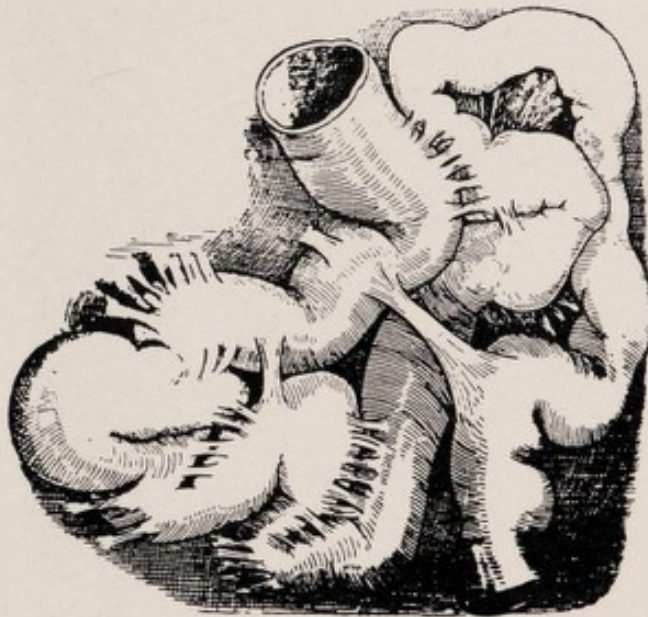


FIG. 5. Numerous dense bands binding loops of intestine to one another. (Von Froriep¹)

in origin or come about as a result of injury to the peritoneum; or that both these causes play a role: for example, a Meckel's diverticulum may become adherent as a result of inflammation.

Bands and adhesions arising as a result of injury to the peritoneum may be caused by bacterial inflammation or by mechanical trauma. The character of the adhesions differs according to their age. The fibrous exudate thrown out immediately after a peritoneal injury glues the intestinal coils together lightly; but even in this stage may cause obstructions. These have such distinctive features that they are considered in a separate chapter under the heading "Early Postoperative

Obstructions." In many instances these soft, fibrinous adhesions are in great part absorbed without undergoing the process of organization into fibrous tissue; but often they are converted



FIG. 6. Dense, investing, fibrous membrane, which has shortened, convoluted and angulated four-fifths or more of small intestine. (Dowd²)

into connective tissue, varying from a few light membranous attachments to very dense scar tissue and hard cicatricial bands (Fig. 5), present everywhere throughout the peritoneal cavity and binding the abdominal viscera to each other and to the abdominal wall so tightly that practically no free peritoneal cavity can be demonstrated on abdominal section. Dowd² points out (Fig. 6) that occasionally a chronic peritonitis occurs which affects the visceral peritoneum and resembles in certain respects the pathology found in chronic multiple serositis. The exact factors that determine how much scar tissue shall remain after injury to the peritoneum are for the

most part unknown. It is recognized that the response of the peritoneum to injury differs greatly in different individuals:³ in some patients very slight trauma is apparently responsible for very numerous and dense adhesions; while in others, although a severe peritonitis has occurred, later abdominal section may reveal very little evidence of the previous disease.

In the main, the old, well-organized bands and adhesions have two principal forms: membranous folds or sheets; and isolated cord-like structures that are known as bands or false ligaments. According to Treves,⁴ the bands may be brought about by molding and stretching of an unformed mass of adhesive tissue into a cord by movements of the abdominal viscera, the intestines rolling over and under and about an adherent membrane until it is molded almost as a piece of clay may be molded when rubbed between the palms. This formation is most likely to come about when the situation of the adhesions is such as to keep the membrane on the stretch. These structures may be only small filaments, or may be dense strong cords; they may be short pedicles binding coils of intestines to adjacent structures; or they may be long, extending from one side of the abdomen to the other. They may arise from localized areas of peritonitis, such, for example, as are caused by the breaking down of tuberculous lymph nodes; or an intestinal ulcer may cause a localized peritoneal inflammation which is the starting point for the band. Tuberculosis of the peritoneum may bring about widespread adhesions, which at times may result in intestinal obstruction. Frequently, however, in this disease the obstruction produced is subacute.⁵

The omentum, becoming adherent to some of the abdominal viscera, may not infrequently be converted into a band or cord by some such process as described for the conversion of adhesions into cords.

Another structure capable of acting as a band is Meckel's diverticulum. Although at times the omphalomesenteric duct may have been so completely obliterated that there is no true diverticulum, nevertheless the obliterated duct or the remains

of its vessels may exist in the form of a cord stretching from the lower ileum to the umbilicus; according to Leichtenstern,⁶ remains of this embryonic structure may also be represented by



FIG. 7. Obstruction by appendix, acting as solitary band. (Warren and Gould⁷)

a band running from the ileum to the root of the mesentery. The most common condition that converts a Meckel's diverticulum into a band is a local inflammatory process which anchors the free end of the diverticulum to the mesentery or to some other abdominal structure. In rare instances, when the diverticulum is long, it may form knots and thus snare loops of intestine.

The remains of other congenital or developmental structures found within the abdominal cavity may at times act as bands. The appendix (Fig. 7), fallopian tubes, appendices epiploicae or pedunculated tumors may also act as bands by becoming attached through inflammatory processes.

MECHANISM OF SIMPLE OBSTRUCTIONS AND STRANGULATIONS. Bands and adhesions bring about the actual obstruction in a number of different ways, and the pathological picture may be exceedingly complex and interesting. The subject is thoroughly covered by detailed descriptions and numerous illustrations of the pathological anatomy in the works of Brinton,⁸ Leichtenstern,⁹ Treves,⁴ Wimls,¹⁰ and other writers

of the 19th century; the various compressions, twists, kinks and strangulations that may be found at operation or autopsy were of particular interest to the pathologists, anatomists and surgeons during this period.

In addition to obstruction of the lumen caused by bands and adhesions, there is frequently a constriction or compression of the mesentery, which may vary from a mere slowing of the venous return to complete occlusion of the circulation. The changes in the bowel wall of course depend upon the extent of the damage to the vascular supply, and may vary from simple congestion, edema and cyanosis to complete infarction and gangrene. (Fig. 4, p. 22). The importance of this factor has already been discussed in Chapter II. Obstructions are usually spoken of as "simple" or "strangulated," depending on whether or not the mesenteric circulation is constricted.

Simple Obstructions. In general it may be said that adhesions or bands bring about simple obstructions by compressing or flattening the intestine against some solid structure (Fig. 8)—such, for example, as the posterior abdominal wall or the scar of some previous abdominal operation—or by attaching loops to one another (see Fig. 5), to their mesentery, or to some other abdominal structure, thus distorting or pinching the intestine so that the lumen is occluded.

Strangulations. The fundamental methods by which bands and adhesions produce strangulation are relatively few. In general this comes about in one of two ways: bands form bridges or arches, under which intestinal loops become engaged and strangulated; or long cords are formed into nooses or knots which capture a loop.

Strangulation under bands is the more frequent of the two types. All that is essential for the production of this type of strangulation is a firm band attached at two points to form an arch, and a firm surface to make the floor of the arch. The points of attachment may be diverse: it is common for at least one end of the band to be attached to the mesentery; other common sites of attachment are the posterior abdominal wall,

the brim of the pelvis or the pelvic organs. The bands are often multiple. They may be γ -shaped and the triangle between the arms of the γ may form the opening through which

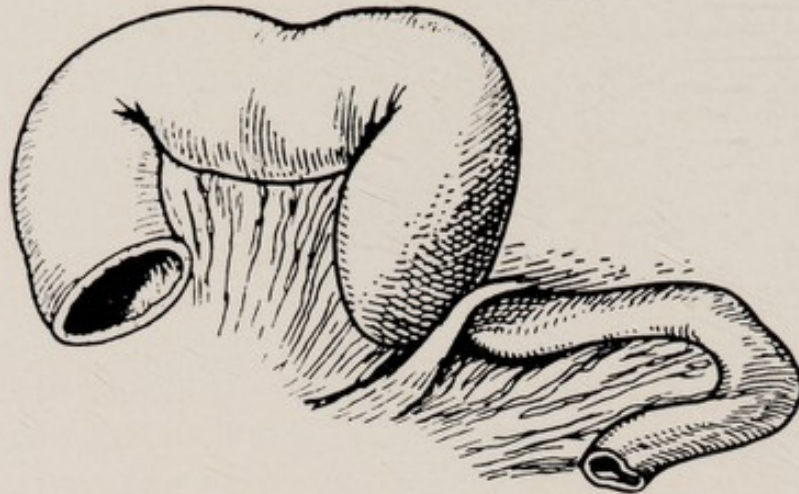


FIG. 8. Compression by isolated band. (Braun and Wortmann¹¹)

the loop of bowel passes. It is obvious that arches formed by bands have considerable similarity to the opening of a hernial ring, and the actual passage of the bowel through such an opening and its subsequent strangulation is essentially similar to the strangulation of a bowel in a hernial sac, whether internal or external.

Long cords or bands lying within the abdominal cavity are capable of forming nooses and simple or complicated knots which may capture and strangulate a loop of bowel. Figure 9, A, B, C illustrates various ways in which this can come about. It will readily be seen that the pathological picture presented at operation or autopsy, in this type of obstruction, may be exceedingly complex and difficult to unravel; and it is obvious to what extent the mesentery must suffer with the bowel.

There are rare instances in which there is knotting or intertwining of intestinal loops themselves. According to Leichtenstern,¹⁵ this most commonly involves the sigmoid flexure and a loop of ileum; although at times loops of small intestine only

may be involved. Probably peritoneal adhesions or abnormalities of the mesentery are responsible for most of these cases.

Closely related to the above forms of strangulation are



FIG. 9A.

Snaring of a loop of intestine by a long lax band attached at both ends. Band lies as a ring; loop becomes accidentally engaged and is then strangulated. (Leichtenstern¹⁴)

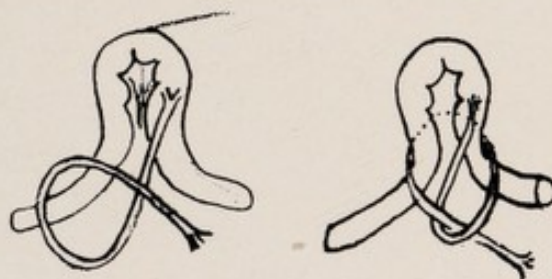


FIG. 9B.

Strangulation by a simple knot. Long loose band is fastened at one end to intestine and hangs as a simple coil (A); top of loop passes through coil, forming a knot (B). (Leichtenstern¹⁴)



FIG. 9C.

A more complicated form of knot. (Leichtenstern¹⁴)

those in which a loop of bowel is trapped through openings in the mesentery, in the omentum or in certain ligamentary attachments normally present in the abdomen, as, for example, the broad ligaments of the uterus or the suspensory ligament of the liver. These are discussed under the heading of "Internal Hernias," Chapter VII, page 92.

The exact mechanism by which strangulation comes about has been the subject of much study. There are doubtless many ways in which the initial compression of the mesenteric vessel,



FIG. 10. Strangulation by solitary band attached at either end to mesentery. (Treves¹²)

occurs. The band responsible for the strangulation is frequently attached to the mesentery, and relatively slight twists or torsions of the involved loop may be sufficient to hinder the mesenteric blood flow. Where a loop has become incarcerated under a band or through a hernial opening, distention may in certain cases be an important factor in causing the initial pressure on the mesenteric vessels, and at times draws more intestine through the hernial opening, thus increasing the amount of bowel involved. The course of events in such cases is as follows: a loop of bowel becomes trapped; gas is forced into this incarcerated loop, and as it becomes distended a valve-like action takes place so that the gas does not escape;*

* The earliest experiments on this point were carried out by Bern,¹⁶ who drew a loop of bowel through a small opening and found that if he blew air slowly through a

as distention of the loop increases, more intestine may be drawn into the trap (Anger,¹⁹ Gatch et al.,²⁰ and others) (Figure 11). As the process continues, pressure may be exerted on the

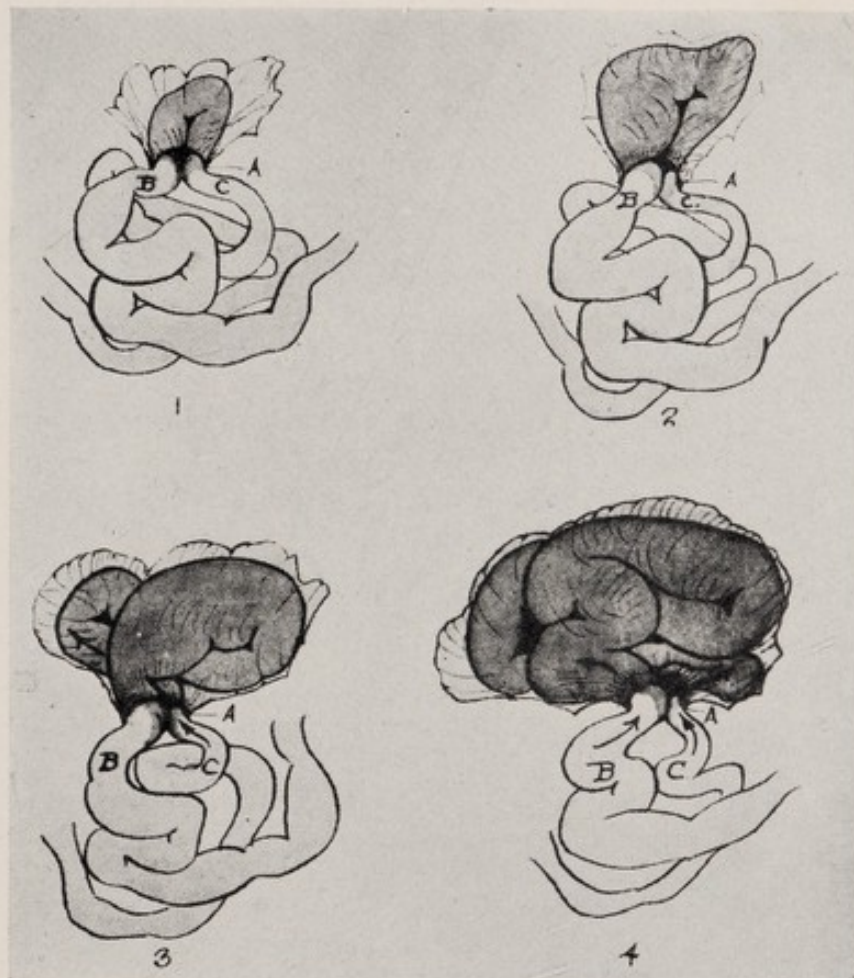


FIG. 11.

Gas trap mechanism. A, ring or band which constitutes opening into a pocket of peritoneum or mesentery. B, afferent intestinal loop and C, efferent portion. In Diagram 1 process is just beginning; in diagram 2 bowel in pocket is overdistended by gas forced in from (B) and trapped by valve-like action of the ring; 3 and 4 show successive steps in inclusion and incarceration of bowel, as traction from pocketed and distended loop pulls more intestine into trap, as shown by arrows. (Gatch et al.²⁰)

mesenteric blood vessels; and it is clear that once this has occurred the action will be progressive. First the thin-walled

catheter into one end of the bowel, it escaped easily through the other end; whereas if he suddenly blew in a large amount, the bowel became distended and none of the air escaped. (See also Rost¹⁷ and Treves.¹⁸)

veins will be affected; engorgement of these vessels and subsequent edema of the mesentery and bowel wall will increase the pressure; and this in turn will further constrict the vessels.

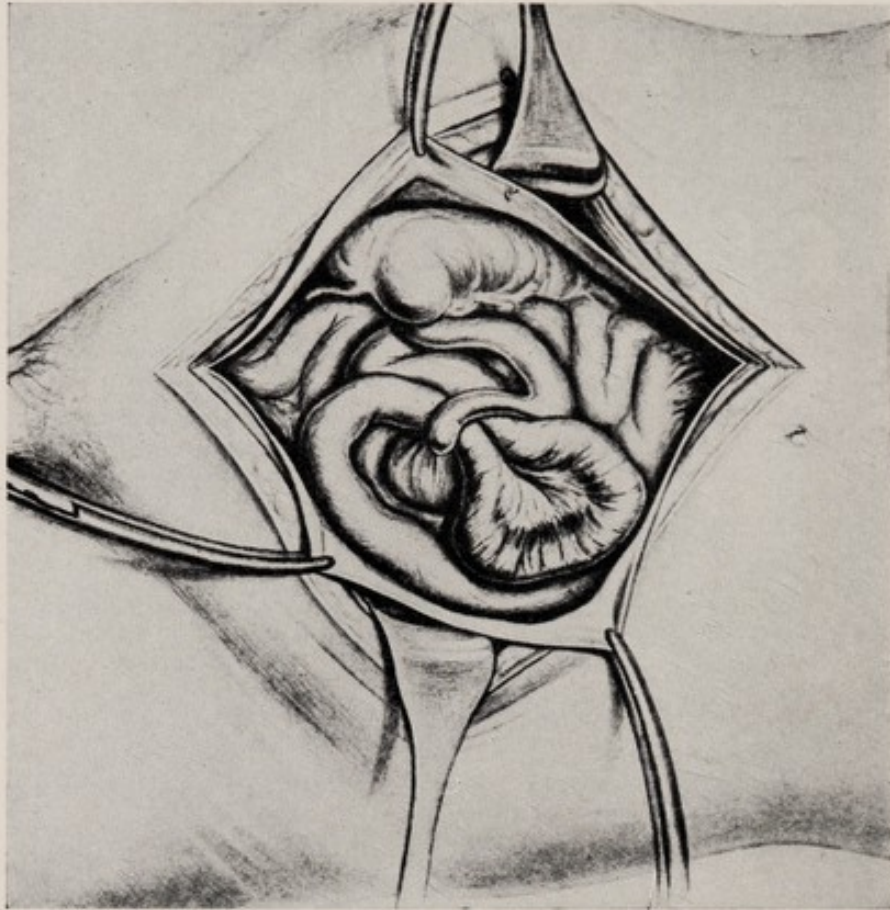


FIG. 12. Loop of small intestine strangulated beneath a Meckel's diverticulum. (Coley and Fortune¹³)

Under these conditions, an exudate quickly forms within the obstructed loop, which increases the distention and adds to the seriousness of the situation. The anoxemia of the strangulated loop will tend to set up violent peristaltic movements in the involved segment, which further complicates the picture and perhaps accounts for the twists and torsions not infrequently found in connection with these internal strangulations.

CLINICAL GROUPING OF OBSTRUCTIONS BY BANDS AND ADHESIONS. Obstructions caused by bands and adhesions form

a large class (Figure 2, p. 17). Because of certain clinical and pathological differences, these cases are considered in three groups: obstructions occurring in patients who have had no previous operation; obstructions occurring late after operation; and those arising early after operation.

Obstructions in Patients Who Have Had No Previous Operation. The bands and adhesions in these cases are usually of congenital origin or the result of bacterial inflammation. Cases are reported in the literature where trauma to the abdominal wall was of sufficient severity to injure the abdominal viscera and so produce localized peritonitis with adhesions which were later responsible for obstructions; but these cases are rare. In most instances it is impossible to determine, at the time of the operation for obstruction, the exact etiological factor responsible for the bands or adhesions, which usually represent anatomical landmarks of old, healed peritonitis. Tuberculosis is not infrequently a factor (see Case 1). Among 21 cases recently reported,²¹ mesenteric lymph glands, enlarged or partly broken down, furnished a chronic inflammatory focus in 5 instances; tuberculous peritonitis was present in one case; an acute, inflamed appendix acted as a band constricting the terminal ileum in one case.

In the afore-mentioned series, the obstructions were described as being due to adhesions in 13 instances; to bands in 8.* In 3 of these latter cases the omentum acted as a band. The mesenteric circulation was impaired in 8 instances, but in only 3 was it of sufficient severity to demand a resection.

In this group of cases the obstruction was located in the small intestine in 16 instances; in the large intestine in 5.

The following is a representative case in this group of obstructions:

CASE 1. Obstruction by bands and adhesions in a patient who had had no previous operation:

* It is an interesting finding that when the obstruction was described as being caused by a band, the mortality was less than half as great as when it was described as due to adhesions. This applies also to obstructions occurring late after operation. The term "adhesions" was probably used to denote more widespread pathology; also a definite band is more easily dealt with at operation.

No. 264,358, M. G. H. Male, aged seventeen. Patient complained of severe abdominal pain of eleven hours' duration, referred to the umbilical region; cramp-like in character, but present almost continuously; somewhat more severe on the left side of the abdomen than on the right. Had vomited shortly after onset of pain and several times since; vomitus was clear, bile-stained, odorless fluid. No bowel movements since the onset of the attack. Temperature 97.4°F.; pulse 50.

On physical examination there was some tenderness all over the abdomen, more marked on the left side; no distention; no visible peristalsis; no mass could be made out. Provisional diagnosis of intestinal obstruction with strangulation was made.

At operation there was a considerable amount of dark, bloody peritoneal fluid. A strangulated loop of ileum, dark and cyanotic in color, was found in the left side of the abdomen. The strangulation had been caused by a firm band attached to the mesentery and to a partially broken-down lymph gland ("presumably tuberculous") lying near the root of the mesentery. There was also a partial rotation of the involved coil.

The band was divided and the intestine liberated. Under observation the color improved; the loop was considered viable and returned to the abdomen. After a somewhat stormy convalescence the patient made a complete recovery.

Comment: The adhesions about the partially broken-down tuberculous lymph gland that were found in this case represent a not uncommon cause of obstruction. On x-ray examination the calcified gland may be mistaken for a renal stone. For further discussion of points in the diagnosis of calcified abdominal lymph nodes, see Golden and Reeves.²²

Although this patient was operated upon within twenty-four hours of the attack, the viability of the bowel was open to some question; this illustrates the fact that a rapid course is run by cases of obstruction caused by acute strangulation; if operation had been delayed a few more hours a resection would have been required.

Late Postoperative Obstruction. Intestinal obstruction by bands and adhesions may follow any abdominal operation; it is most common after appendectomies for acute appendicitis. In the recent Massachusetts General Hospital series of 45 cases, the original operation was as follows:

Appendectomy.....	27
Pelvic operations on uterus or adnexa.....	8
Repair of hernia (strangulated in 3 cases).....	4
Miscellaneous.....	6

Since many of the appendectomies took place in other hospitals, it is impossible to estimate the number of cases that required drainage; the figures available, however, indicate that the number was probably high. In 7 instances the patient had undergone more than one operation before the obstruction occurred.

“Late postoperative” is here taken, somewhat arbitrarily, to mean four or more weeks after the original operation. These obstructions are more common during the first year after operation, but may occur at any time. In the 45 cases of this series, 16 occurred within the first year; the average interval, exclusive of the first year, was six years; the longest was twenty-five years.

Late postoperative obstructions are almost always in the small bowel. In the foregoing series the location was as follows:

Jejunum.....	2	
Ileum.....	19	
Small intestine (not further described).....	22	43
	—	
Large intestine.....	2	

Strangulations are not infrequent in connection with the old, thoroughly organized adhesions of this group. In the foregoing series of 45 cases, strangulation was present in 20 instances; among these a gangrenous gut was found and resected in 5 instances. A gangrenous gut requiring resection was also found in 2 cases of volvulus (listed under that heading) occurring late after operation.*

The following case is representative of the late postoperative group:

CASE II. Obstruction by bands and adhesions arising late after operation:

No. 251,400, M. G. H. Female, aged twenty-three. Patient gave a history of appendectomy two years before admission; the wound had not been drained.

Two days before admission patient was seized with sudden, severe, cramp-like pain, described as being “all over the abdomen” but more severe over the upper half. On admission the pain was constant and severe.

* Two other cases of volvulus occurred late postoperatively, but are listed under “volvulus.”

There had been vomiting shortly after onset of pain and a number of times since; the vomitus was not fecal in character. No bowel movements since the onset of pain. Temperature 98.6°F.; pulse 100; white blood count 26,000. Physical examination showed some tenderness across the lower abdomen; no visible or audible peristalsis. In view of these symptoms and of the fact that the patient had the scar of a previous abdominal operation, a diagnosis of acute intestinal obstruction was made. Immediate operation was advised, but was refused.

The morning after admission (twelve hours later) the patient consented to operation. On opening the peritoneum, dark-colored, bloody fluid escaped. The coils of the small intestine were greatly distended. The terminal ileum was obstructed by a band which crossed the bowel just above its entrance into the cecum; the mesenteric circulation was somewhat impaired at this point, but the bowel was considered viable. The obstructing band was cut and an enterostomy was performed a short distance above the point of obstruction. The patient made a satisfactory recovery from the anesthetic; but distention and vomiting persisted. The patient appeared to be very toxic and died the third day after operation.

Comments. The inciting cause of this obstruction was an adhesion resulting from the appendectomy of two years before. The patient came to operation late, in part due to refusal of operation on admission. The high white count of 26,000 was suggestive of strangulation.

The exact cause of death in this case is not clear from the data at hand. Apparently there was no return of peristalsis after the relief of the obstruction: possibly the bowel was paralyzed from the effects of obstruction and distention; it is also possible that a mistake was made as to the viability of the bowel.

SUMMARY. One group of obstructions is brought about by bands and adhesions, which may be either congenital or the result of injury to the peritoneum. The obstructions may be simple or strangulated; in the latter type, distention of the involved loop may play an important rôle.

Clinically these obstructions are considered in three groups. First, obstructions occurring in patients who have had no previous abdominal operation; the etiology of the adhesions in these cases may be hard to determine. Second, obstructions occurring late after operation; strangulations are frequent. Third, obstructions arising early after operation (discussed in the next chapter).

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CHAPTER IV

VARIETIES OF OBSTRUCTION (Continued)

BANDS AND ADHESIONS (Continued)

EARLY POSTOPERATIVE OBSTRUCTIONS. Obstructions caused by bands and adhesions arising early after operation form an important and interesting group. The obstruction is usually due to recent inflammatory adhesions and is often complicated by local paralysis of the bowel from peritonitis; indeed, certain of these cases really form a borderline group between mechanical and functional obstructions, the functional element being superimposed upon a partial mechanical obstruction. (The purely functional obstructions from general peritonitis, the so-called "paralytic ileus," are not included in this group; see "Functional Obstructions.")

Early postoperative obstructions in the various Massachusetts General Hospital series have been, somewhat arbitrarily, defined as those occurring within the first four weeks after operation. As a matter of fact, the great majority of them occur in less than two weeks following operation, about half of those in the 1918-1927 series having taken place before the fourth day. The average time between the original operation and the operation for obstruction was about eleven days. Of the 335 cases of acute obstruction forming this series, early postoperative obstructions, as shown in Figure 2, were responsible for 37 cases, or about 11 per cent of the total number.¹

Etiology and Pathology. As indicated by Table v early postoperative intestinal obstructions may complicate the convalescence from abdominal operations of any character, but are particularly likely to occur when peritonitis is present.

The adhesions responsible for these obstructions may be due to the mechanical trauma of operative procedures or to

TABLE V
EARLY POSTOPERATIVE OBSTRUCTIONS
(Second and Third M. G. H. Series)

<i>Original Operation</i>	<i>Number of Cases</i>
Appendectomy..... (with drainage, 29)	35
Pelvic Operations in Women.....	10
Operations for Carcinoma of the Rectum.....	7
Operations for Carcinoma of the Sigmoid.....	2
Resection of Jejunum and Closure of Gastroenterostomy Stoma.....	1
Closure of Perforated Gastric Ulcer.....	1
Repair of Ventral Hernia.....	1
Miscellaneous.....	9
Total.....	66

bacterial peritonitis. Any surface that has been denuded of its peritoneal covering is potentially a source of obstructive adhesions. A drain placed among the coils of the small intestine is a particularly common source of mischief.

The adhesions may be localized or widespread. Occasionally they may become surprisingly strong and abundant in a short period of time; but usually, due to their short duration, they are not firmly organized. They may bind a loop of bowel to the operative scar or to some inflamed structure in the pelvis, or to the posterior abdominal wall. They may act as a fixed point around which the bowel is twisted. When the obstructed loop lies in proximity to a localized septic process, as, for example, a tubo-ovarian or appendix abscess, the adhesions may be merely depositions of fibrin, glueing the intestinal coils together and causing localized kinks (Fig. 13) or valve-like closures. These might amount to little were it not for the distended, atonic condition of the bowel; but, as Cannon^{2,3} has shown, in order to carry the intestinal contents beyond such an obstacle as a kink, it is necessary for peristalsis to be vigorous. Wilkie⁴ also is convinced on the basis both of clinical and of experimental evidence that recent fibrinous adhesions and kinks may be an important factor in intestinal obstruction. Guiou⁵ makes the point that the distention is also important, for as the diameter of the gut increases, acute

angulations and obstructive kinkings are more likely to occur. The frequency of acute angulations and adhesions of the terminal ileum following suppurative appendicitis has been

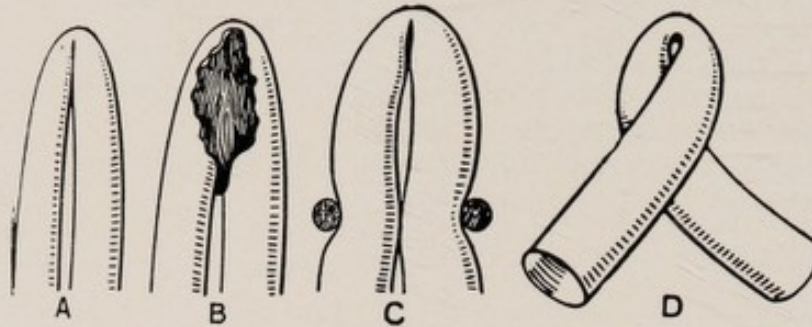


FIG. 13. Various forms of kinks: (A), free kink; (B), fixed kink; (C), held kink; (D), traction kink (garden hose kink, of doubtful occurrence in the intestine.) (Guiou.⁵)

stressed by Cheever,⁶ Richardson,⁷ and Roeder.⁸ (See also pp. 175 and 200.)

Interference with the Circulation. Strangulations are not common among the early postoperative obstructions caused by recent inflammatory adhesions. Among 69 cases of obstruction occurring early after operation at the Massachusetts General Hospital during a twenty-year period, there were only 2 instances of strangulation by bands and 3 by volvulus. This is in striking contrast to the frequency of strangulation among late postoperative obstructions, where dense bands of scar tissue are so frequently responsible for the obstruction. Since strangulation is so seldom present in early postoperative cases, it is rare that bloody fluid is found in the peritoneal cavity; when this is present, strangulation must of course be suspected.

Level of the Obstruction. Most of the early postoperative obstructions occur in the lower portion of the small intestine. In about half of the cases in the foregoing series it was definitely stated that the obstruction was in the lower ileum. In 2 of the remaining cases it was said to be high in the small intestine (mid-jejunum); in 9 it was merely assigned to "the small intestine" without further specification; in 7 instances there

was no statement in the record by which the level could be estimated.

Age and Sex. Early postoperative obstruction occurs far more frequently in males than in females: in the Massachusetts General Hospital series there were 30 males and 7 females, a rather striking sex distribution.

Most of the cases occur between the ages of ten and fifty (see Fig. 3). Appendicitis, the most common cause of these obstructions, is, of course, primarily a disease of early adult life.

Course of the Disease. The symptoms of obstruction shown by the early postoperative group are in general those of acute obstruction from any cause: vomiting, pain, distention and obstipation; but owing to the fact that these obstructions come on during a convalescence, the picture is often masked. If the histories of a number of these cases be studied, it will be found that the obstruction usually does not occur out of a clear sky: the onset is often insidious, the picture changing almost imperceptibly from that of a stormy convalescence with a considerable amount of vomiting and pain (which, however, might be expected to quiet down under conservative treatment), to one where frank obstruction, demanding operative intervention, is recognized.

The picture is frequently complicated by varying degrees of peritonitis; in 12 of the 37 cases in the foregoing series this played a definite rôle. If peritonitis exists, it may be difficult to decide whether mechanical obstruction is present or whether there is simply a functional, paralytic condition of the bowel ("paralytic ileus"). Cramp-like pains and evidence either on inspection or auscultation of increased peristaltic activity are valuable signs pointing to the existence of a mechanical obstruction.

In another group of cases the early disturbance of the operation may have passed, the peritoneal infection have subsided and the convalescence be proceeding in a satisfactory manner, when the first symptoms of obstruction manifest

themselves, usually in from ten days to two weeks after the operation. The diagnosis is likely to be easier in this group.

See also Diagnosis, p. 239, and Treatment, p. 296.

Case III illustrates a number of points in relation to mechanical obstruction occurring in connection with peritonitis:

CASE III. Appendectomy with drainage followed by early postoperative obstruction.

No. 279,896, M.G.H. Female, aged thirty-two. Eight days before the operation for obstruction the patient entered the hospital complaining of pain in the abdomen of five days' duration. The pain had been almost constant and quite severe. During the two days before admission the patient had vomited a number of times. There had been no bowel movements for several days, although cathartics and enemas had been used. For the past twenty-four hours the patient had noticed distention of the abdomen.

On physical examination the patient appeared to be acutely ill, with marked distention of the abdomen. There was tympany over most of the abdomen, but dullness in the flanks. The temperature was 100°F., pulse 122, white blood count 4000. Lavage of the stomach yielded about 180 c.c. (6 oz.) of foul-smelling fluid. It was not at all clear whether the patient had organic obstruction or whether the symptoms were secondary to peritonitis.

First Operation. When the peritoneal cavity was opened a large amount of turbid fluid escaped. The intestines were everywhere distended and injected and showed depositions of fibrin. The appendix was found to be gangrenous and perforated. There was little or no walling off. The appendix was removed and a drain placed in the right iliac fossa and another in the pelvis. When the drain was inserted in the pelvis there was an escape of a considerable amount of foul-smelling pus. After operation the patient was placed in Fowler's position and was given morphine liberally. No fluids were given by mouth, but large quantities under the skin and by rectum.

On the first day after operation the patient was in poor condition, pulse barely palpable. On the second day her general condition showed some improvement: the abdominal distention continued, but there was no vomiting. For the next two days the condition remained about the same; but there was considerable vomiting. The stomach was washed out several times with a Levine tube, and the tube left in the stomach for twenty-four hours; there was profuse drainage.

During the course of the next four days the patient's condition showed improvement; and with this improvement there was a rise in white blood

count and temperature. The abdominal signs continued to be about the same, and frequent use of the stomach tube was required to prevent dilatation and vomiting.

On the eighth day postoperative, on passage of the stomach tube a large amount of foul-smelling fluid was obtained; the abdominal distention was a little more marked, and visible peristalsis was noted in the left upper quadrant. The patient complained of cramp-like abdominal pain.

Operation for Obstruction. Under local anesthesia a jejunostomy was performed by the Witzel method. At this operation no exploration was carried out. The coils of the intestine in the upper abdomen were distended, but no active peritonitis was present. The obstruction was presumably in the lower ileum in the region of the drains.

Following the jejunostomy, the patient showed marked improvement. There was profuse drainage from the catheter in the jejunum amounting to almost 4000 c.c. in twenty-four hours. This continued for a number of days, the lost fluid being adequately replaced by subcutaneous injections of normal saline solution. The patient's general condition showed steady improvement. The tube was removed from the jejunum eight days after the operation.

The patient was discharged in good condition thirty days after the second operation.

Comment. This case illustrates a number of points in the type of obstruction occurring early after operations undertaken for peritoneal infection. At the time of the first operation the patient was overwhelmed with the systemic effects of a general peritonitis, as shown by the high pulse rate, low temperature and subnormal white blood count. The intestinal symptoms during the early stages were undoubtedly those of functional disturbance of motility secondary to the peritonitis. During this stage the treatment was directed primarily toward localizing this infection (Fowler's position, morphine, fluid subcutaneously). The functional atony of the intestinal tract was marked; drainage back into the stomach was profuse, but use of the small stomach tube was considered adequate to cope with the situation.

About eight days following the appendectomy, the generalized bacterial peritonitis being for the most part overcome, there was an onset of mechanical obstruction, as indicated by visible peristalsis. This sign, taken in connection with the character and the increase in amount of the fluid obtained from the stomach, was deemed adequate indication for operative drainage of the intestinal tract. The symptoms of obstruction were completely relieved by a jejunostomy without exploration or attempt to break up adhesions; and, as so frequently happens with this type of obstruction, no further operative procedures were required: the light adhesions were

either completely absorbed, or the involved loop of bowel regained its tone so completely that those remaining were no longer capable of arresting the intestinal stream.

This case also illustrates the great loss of fluids that may take place. It is extremely important that careful study of intake and output of fluids and measurements of blood chlorides should be carried out on these cases in order that the water and the essential sodium and chloride ions, may be adequately replaced. See Table xvi, page 306.

The history of Case iv illustrates instances where the obstructive adhesions are caused primarily by operative procedures rather than by peritonitis.

CASE IV. Obstruction following exploratory laparotomy and appendectomy.

No. 288,275, M.G.H. Male, aged thirteen. The original operation was a laparotomy and exploration for repeated hemorrhages from the bowel. No neoplasm or other cause for the bleeding was found; the appendix, which was normal, was removed.

The patient made an uneventful convalescence until ten days after the operation, when he began to have sharp epigastric pain, and vomited. The signs of obstruction (pain, distention, vomiting, and failure to obtain results with enemas) persisted, and an exploration was decided upon.

At operation, the upper small intestine was found greatly dilated; the lower, collapsed. The omentum and two loops of small intestine were firmly adherent to the previous operative incision. There was also a rotation of the adherent loops around the point fixed by adhesions. The twist was straightened out and a jejunostomy was carried out by the Witzel method. A large amount of thin, fecal fluid was immediately evacuated through the catheter.

The first few days' convalescence was stormy; and large amounts of intravenous and subcutaneous fluid were required to combat dehydration. The symptoms of obstruction subsided and the patient was discharged symptom-free about three weeks later.

Comment. The obstruction in this case was due to adhesions resulting from operative trauma to the peritoneum. There was no acute peritonitis with the functional element that it so often carries. Note that the obstruction came on relatively late after the operation with a sudden onset and the classical signs of acute obstruction. The diagnosis is often much easier in this type of obstruction occurring after operation than in cases such as that quoted above where active peritonitis confuses the picture.

Obstructions early after operation may also arise as a result of operative procedures which alter the normal relations

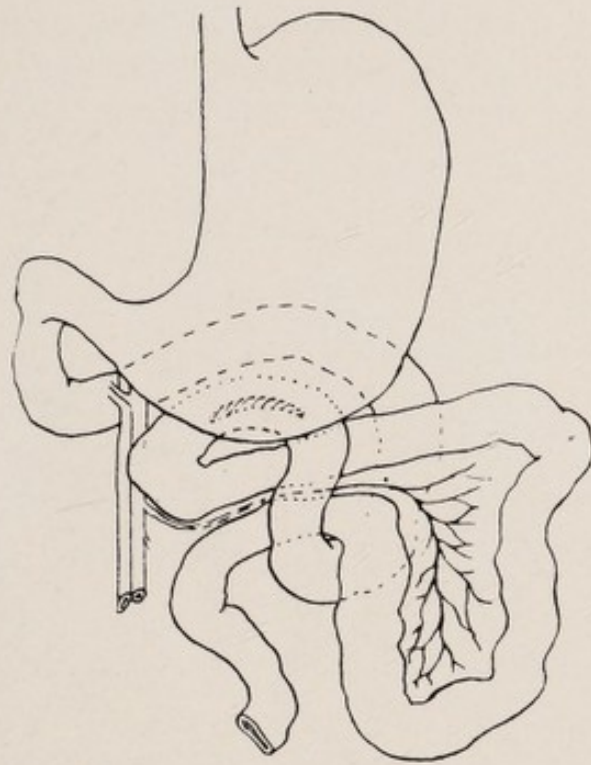


FIG. 14. Strangulation occurring after a posterior gastroenterostomy. (Keene.⁹)

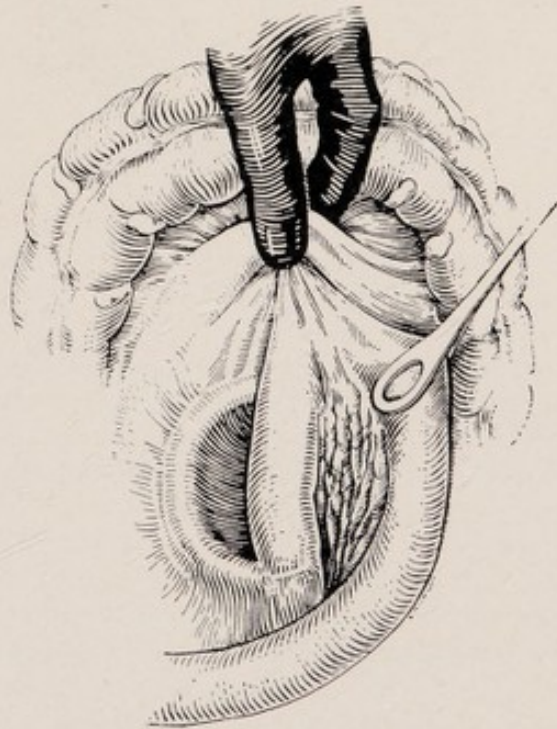


FIG. 15. Boundaries of opening through which herniation may occur following a posterior gastroenterostomy. (Armitage.¹⁰)

of the intestinal tract: anastomoses, enterostomies, colostomies and the like may result in abnormal apertures or arches through which the bowel may herniate and become obstructed (Figs.

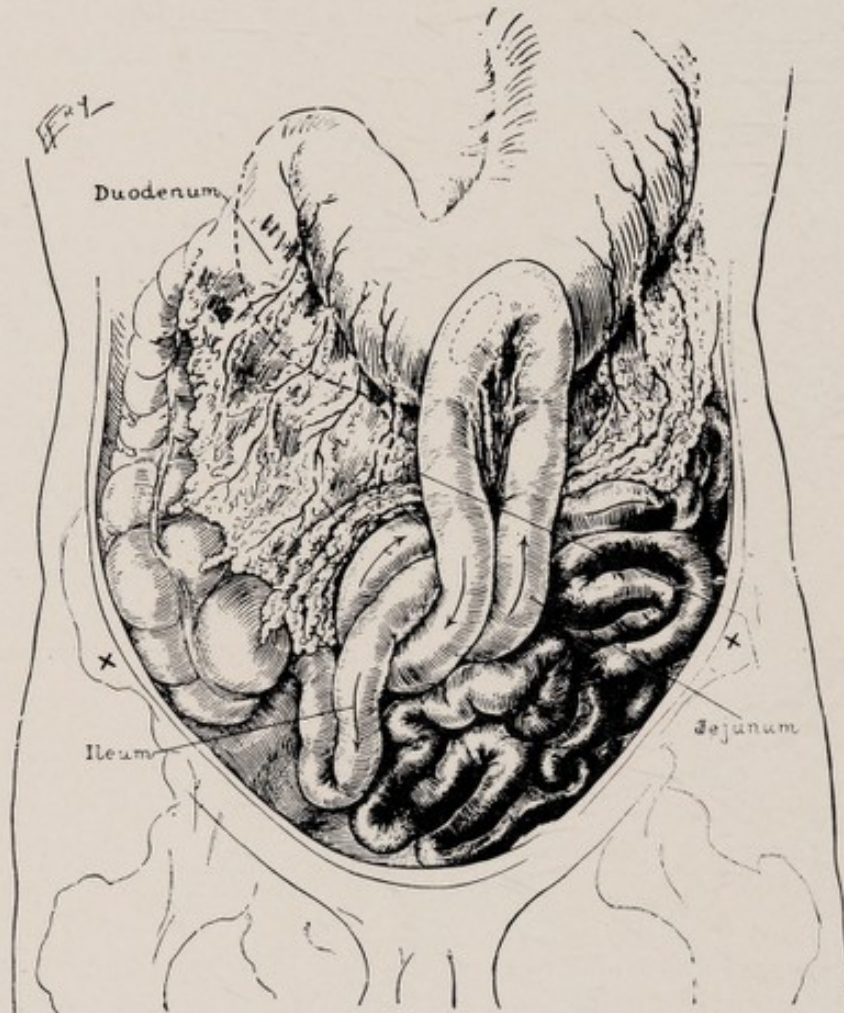


FIG. 16. Hernia of small intestine behind short anastomotic loop of jejunum following posterior gastrojejunostomy. (Mayo and Magoun.¹¹)

14-18; also Chap. VII). Case v illustrates obstruction following a colostomy.

CASE V. *Obstruction following operation for cancer of rectum.*

No. 245,924, M.G.H. Male, aged forty-three. Amputation of the rectum for carcinoma was performed, combined abdominal and perineal, one stage.

On the second day postoperative the abdomen was somewhat distended and the patient was hiccoughing. One day later, the colostomy was opened,

with some immediate relief. The distention, however, increased and no result was obtained from an enema. "Fecal" vomiting occurred. The stomach was washed out and a moderate amount of foul-smelling fluid



FIG. 17. Hernia of small intestine around colostomy. To prevent the type of hernia shown in this illustration Mayo suggests placing the colostomy far to the left of the mid-line. After the loop of sigmoid has been brought out of the abdomen the mesocolon is sutured to the left parietal peritoneum by a continuous puckering suture. Two interrupted sutures are then placed at the juncture of the sigmoid and its mesentery just below the outer cut edge of peritoneum as shown in Figure 18. (Mayo and Magoun.¹¹)

obtained. During the next two days some gas was expelled through the colostomy, but distention persisted and the patient's general condition was not satisfactory; vomiting recurred.

Six days after the original operation an exploratory procedure was carried out. It was found that an obstruction was present, caused by coils

of small intestine passing between the attachment of the colostomy and the left abdominal wall. As the distention had increased, the obstruction had become complete. The intestines were restored to their normal position,

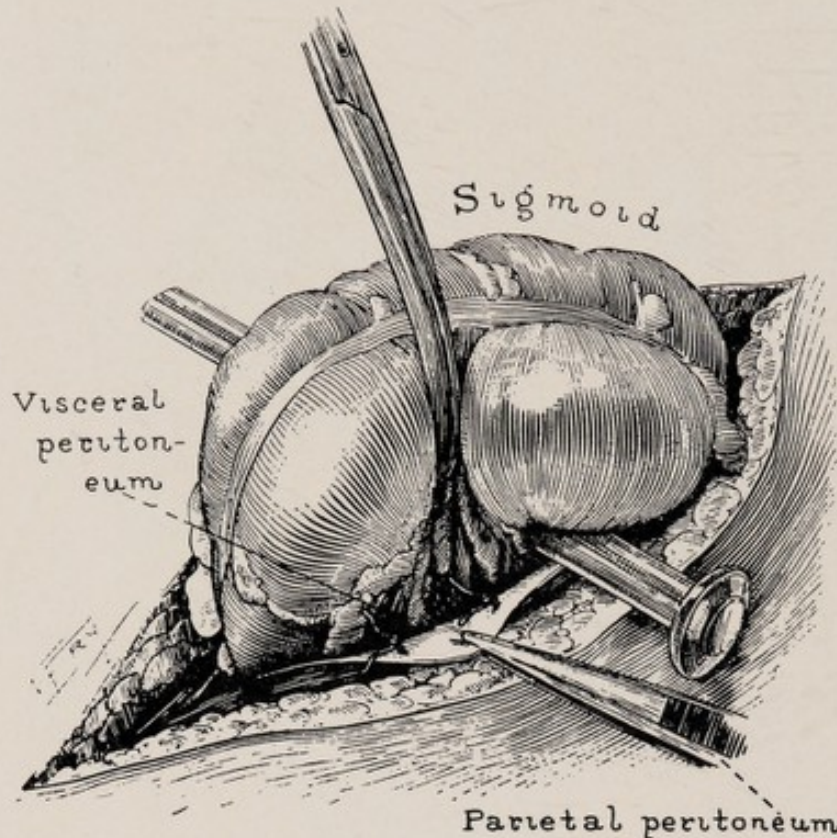


FIG. 18. Two interrupted sutures through cut edge of parietal peritoneum and upper portion of mesosigmoid to prevent retraction of the colostomy loop. (Mayo and Magoun.¹¹)

and an ileostomy was performed.

The patient was in poor condition after the operation and died fifteen hours later.

Comment. The obstruction did not result from injury to the peritoneum either by bacterial peritonitis or by operative trauma, but from what was essentially an internal hernia. It is advisable to fix the mesosigmoid to the left abdominal wall so that it is not possible for a loop to herniate through this opening.

Case VI illustrates a rather rare type of obstruction coming on early after operation:

CASE VI. *Volvulus occurring early after operation.*

No. 259,416, M.G.H. Female, aged twenty-six. The first operation was for subacute appendicitis. The appendix was removed and the abdomen closed without drainage.

On the second day postoperative the note states that the abdomen was somewhat distended. The fourth day after operation there was further distention, a sudden onset of violent epigastric pain, and vomiting. The stomach was washed out and an enema given with some relief. Some time after the onset of the epigastric pain an indefinite, soft mass was made out in the right lower quadrant of the abdomen.

On the fifth day after operation the symptoms of obstruction were so pronounced that the patient was again operated upon. A volvulus of the cecum was found, the terminal 6 inches of the ileum and the ascending colon being involved in the twist. The volvulus was reduced and a cecostomy performed. The patient made an uneventful convalescence.

Keene,⁹ Armitage,¹⁰ and Mayo¹¹ have reported cases* where following a posterior gastroenterostomy the small bowel herniated through the opening formed by the transverse mesocolon, the posterior abdominal wall and the loop of intestine lying between the ligament of Treitz and the gastroenterostomy (see Fig. 14). Molesworth¹² has described a somewhat similar condition occurring after an ileocolostomy †: here the bowel herniated through a ring bounded in front by the anastomosis, above by the transverse mesocolon, behind by the posterior abdominal wall, and below by the mesentery of the small intestine. Mayo and Magoun's article¹¹ on the internal hernias that occur as a result of operative procedures is very interesting and comprehensive.

SUMMARY. Early postoperative obstructions often form a borderline group between mechanical and functional obstructions; at times adhesions and atony from peritonitis both play a rôle. The adhesions may be localized or widespread; they often are not firmly organized. They may furnish a fixed point around which a volvulus occurs. The obstructions are

* These cases occurred late after operation; but I have observed a case where the same thing occurred during convalescence.

† This case also actually occurred late after operation, but might well have occurred early after operation.

usually located in the lower levels of the small intestine. While strangulations are not common, herniation of the bowel through apertures created by operative procedures does at times occur.

Obstruction may come on insidiously in the course of a stormy convalescence; or the convalescence may be proceeding satisfactorily when the obstruction occurs, ten days to two weeks after operation.

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CHAPTER V

VARIETIES OF OBSTRUCTION (Continued)

OBSTRUCTION BY VOLVULUS

Intestinal obstructions produced by the rotation or twist of an intestinal loop form an interesting though relatively rare group.* The underlying cause of the volvulus may be some congenital anomaly, such as abnormalities in the fetal rotation of the gut, or lack of mesenteric or cecal fixation.^{1,2} A giant sigmoid or a megacolon may be the predisposing factor: Weeks³ has collected 63 such cases from the literature. In another group of cases, bands and adhesions, tumors of the mesentery, etc., may be the etiological factors. A volvulus may occur in association with a hernia⁴ (see p. 122).

Either the large or the small intestine may be obstructed by this mechanism. In the large intestine the most common sites are the sigmoid flexure and the cecum. Due to their close anatomical relation, a volvulus of the cecum may involve the ascending colon and terminal ileum. A mobile cecum or sigmoid may become twined around a coil of ileum, or may serve as an axis around which a section of small intestine may become coiled; these cases are extremely rare, but have been reported by Treves,⁵ Leichtenstern,⁶ Wilms,⁷ and others.

When a section of gut undergoes rotation around its mesenteric axis, or in some instances around its own long axis, an isolated loop is formed (see Fig. 19) with obstruction of the lumen at both ends. There is also more or less compression of the mesenteric vessels, particularly of the thin-walled veins. The amount of the interference with the mesenteric circulation may vary from slight congestion to complete venous blockage, depending upon the degree of rotation and the tightness of the

* Minor degrees of torsion of intestinal loops are often found when strangulation by a band occurs. The term "volvulus" is usually reserved for the more extreme twists and rotations.

twist. It is frequently severe. The obstructed loop may also become enormously distended, which further embarrasses its blood supply. Due to the twofold interference with the circu-

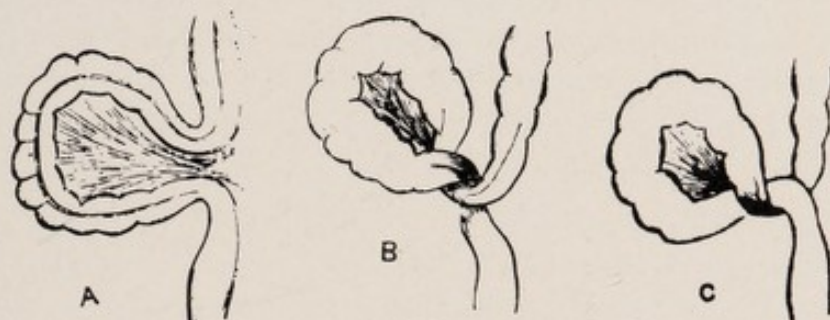


FIG. 19. Mechanism of volvulus of sigmoid. (Treves.¹⁰)

lation, through pressure on the mesenteric vessels and from gaseous distention within, changes in the bowel wall often occur early and may proceed to complete infarction and gangrene, with resulting peritonitis. In the 1918-1927 Massachusetts General Hospital series, 9 of the 15 cases of volvulus showed serious interference with circulation; in 5 instances, changes in the bowel wall were so extensive as to require resection.

Cases of volvulus are usually grouped under three heads: volvulus of the sigmoid; volvulus of the cecum; and volvulus of the small intestine.*

VOLVULUS OF THE SIGMOID. According to the figures of Braun and Wortmann,⁹ volvulus of the sigmoid flexure occurs almost three times as frequently as that involving the cecum and ascending colon; although in the cases of volvulus in the recent Massachusetts General Hospital series the numbers found in these two locations were equal.

Volvulus of the sigmoid takes place by rotation of the loop around its mesenteric axis (Fig. 19); for its occurrence it is of course necessary to have a mobile loop of sigmoid. There are many theories as to the inciting cause; undoubtedly it differs in different cases. At times adhesions which bind parts of the redundant loop together in a distorted position or fasten por-

* Volvulus of the stomach can also occur, but is rare.⁸

tions of the loop to the pelvis or descending colon undoubtedly play a part. Bloodgood¹¹ believes that gaseous distention of such an adherent loop is an important factor in producing the twist; also as the distention increases, the expanding bowel exerts traction on the mesentery and the twist is drawn tighter. A congenital enlargement of the sigmoid is sometimes found in association with volvulus,^{3,12} and may be responsible for its occurrence. Treves¹³ considers that obstinate constipation in elderly patients is an important etiological factor; on the other hand, there have been cases that have followed shortly after a diarrhea. It has been stated that overeating after a long fast predisposes to volvulus of the sigmoid; and that this type of obstruction was of not infrequent occurrence in Old Russia where a period of feasting often followed a long period of fasting.¹⁴

Clinical and Pathological Features. The disease may occur at any age; but it is more common after middle life. The onset may be accompanied by severe pain and vomiting. The pain is likely to be located in the left lower quadrant with radiation across the lower abdomen; it may be of sufficient severity to produce collapse. There is usually nausea and vomiting. Obstipation is complete; and enemas are without result. The severity of the symptoms are generally in proportion to the degree of interference with the circulation. There is often an elevation in the white blood count, with an increased proportion of polymorphonuclear leucocytes.

There may be a history of habitual constipation, and the patient may have experienced in the past similar attacks of pain that were relieved by enemas.¹¹ Volvulus of the sigmoid may complicate the convalescence from an abdominal operation.^{11,15} On physical examination, marked distention of the abdomen may be a striking feature. Frequently there is localized tenderness and spasm in the lower left abdomen, and occasionally a mass may be made out in this region.

The history of Case VII illustrates some of the features of the disease:

CASE VII. *Obstruction by volvulus.*

No. 275,846, M.G.H. Female, aged thirty-eight. Patient had been troubled by obstinate constipation for two weeks. About twenty-four hours before admission she was taken with severe colicky pain, located in the left lower quadrant of the abdomen. The pain had been increasing in severity. Enemas had been given without results. Abdominal distention had increased rapidly.

On physical examination the abdomen was found distended and tympanic; it was tender in the left lower quadrant, where a well-defined, soft tumor 4 or 5 inches in diameter could be made out. During a paroxysm of pain this tumor became hard and more prominent.

At operation a volvulus of a redundant sigmoid was found. The volvulus was untwisted with some difficulty. The bowel was evidently viable.

The convalescence was uneventful. Three years later the patient entered the hospital and a hernia in the operative wound was repaired. There had been no recurrence of the symptoms.

At times the degree of distention of the sigmoid is enormous, the loop occupying practically the whole abdominal cavity and causing embarrassing pressure on the diaphragm.

VOLVULUS OF THE CECUM AND ASCENDING COLON. While volvulus of the sigmoid always takes place around the mesenteric axis, volvulus of the cecum may occur in a number of different ways. It may occur around a line which is at right angles to the long axis of the cecum: in other words, the cecum may be bent back upon itself so that the lower part of its posterior surface becomes the anterior and lies with the appendix along the ascending colon. Or it may be rotated or twisted clockwise on its own long axis. There are, however, cases in which the cecum and ascending colon are provided with a long mesocolon which allows the coil to be twisted on its mesenteric axis just as in the case of the sigmoid; and at times, a redundant loop of cecum and ascending colon may become intertwined around some coil of small intestine or may serve as an axis around which small intestine is coiled.⁵

Most cases of volvulus of the cecum depend fundamentally upon some congenital anomaly associated with developmental defects in rotation of the colon.^{2,5} Homans¹⁶ has reported cases

illustrating different types of developmental anomalies of the cecum that were responsible for volvulus.

The cecum is capable of great distention and in cases of

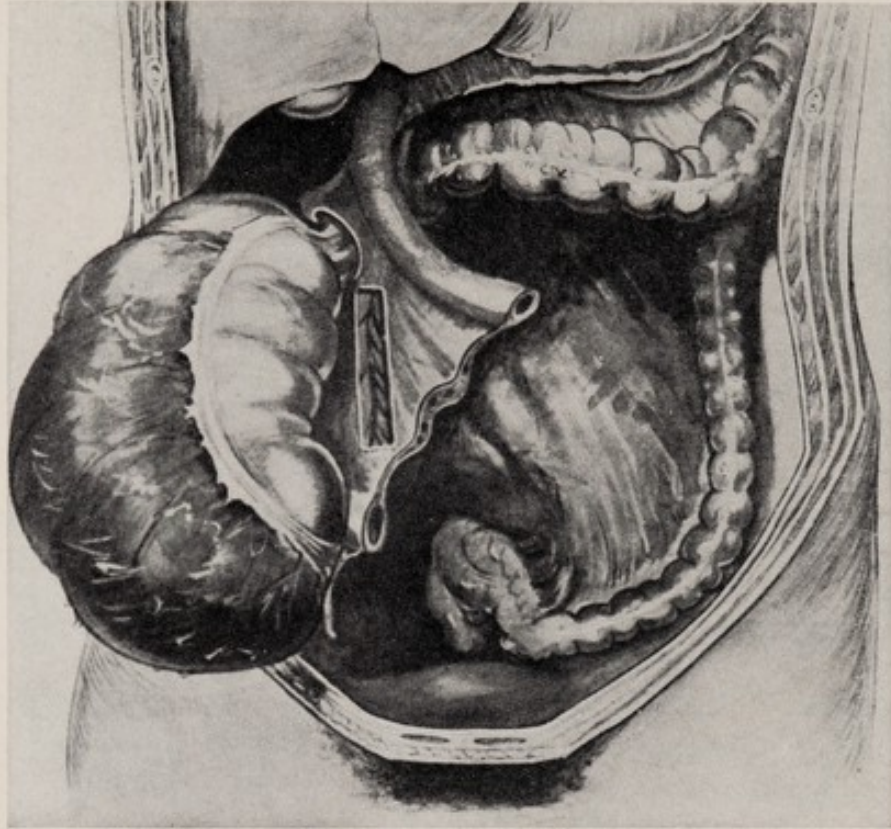


FIG. 20. Volvulus of cecum in association with malrotation. Note that peritoneal coat of cecum has ruptured from distention. (Dott.²)

volvulus may reach an enormous size. In one of Kirby's cases¹⁷ it measured 45 cm. long by 30 cm. in diameter.

Rarely, volvulus of the cecum may occur during the convalescence from an abdominal operation.¹⁸

CASE VIII. *Obstruction by volvulus of the cecum.*

No. 245,586, M.G.H. Female, aged nineteen. The patient entered the hospital thirty hours after the onset of the attack. The day before admission she had "attended a fair at school and had eaten heavily of a number of things." After a few hours' abdominal discomfort, the patient had been taken with severe pain, which was described as dull in character. There was vomiting at the onset of the pain, and this had been repeated a number

of times; the vomitus was watery and greenish. Enemas had been given without result. The abdominal distention had been increasing.

On examination the patient appeared acutely ill. There was enormous

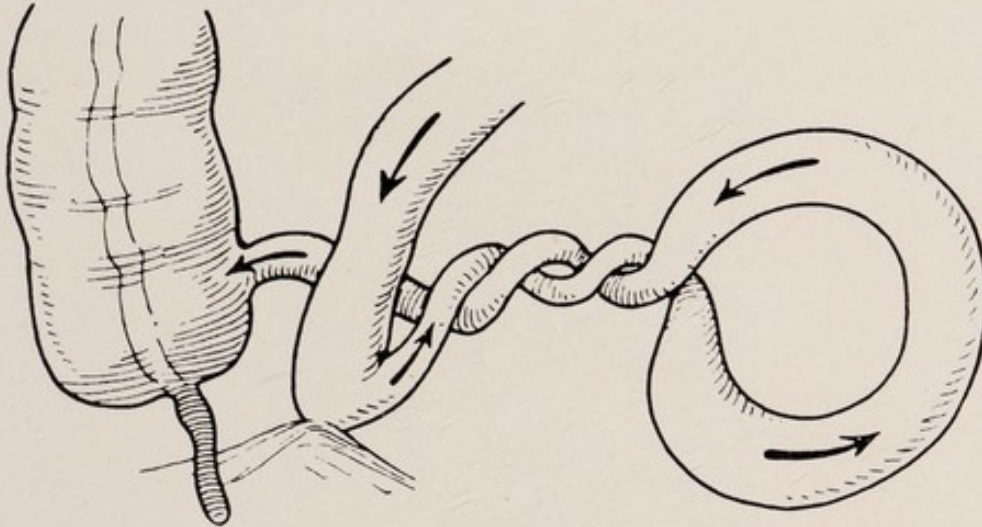


FIG. 21. Volvulus of small intestine resulting from fixation by adhesions. (After Göbell; see Braun and Wortmann.²⁰)

distention of the abdomen, marked tenderness and muscle spasm in the right lower quadrant. There was a questionable mass occupying the right lower side of the abdomen. Temperature 99.4°F.; pulse 132.

An exploratory operation was carried out, and bloody fluid with a foul odor was found in the peritoneal cavity. A volvulus of the cecum and ascending colon was found. There was a long mesocolon, and the cecum and ascending colon had made one complete twist on the mesentery. The cecum was greatly distended, and was black and gangrenous in appearance. The gangrenous mass was delivered and a catheter was sewn into the terminal ileum. The patient was in poor condition at the termination of the operation, and died eight hours later.

VOLVULUS OF THE SMALL INTESTINE. Volvulus of the small intestine may occur anywhere from the jejunum to the terminal ileum, the more common location being in the lower portion of the ileum. One or more loops may be involved. Rarely the torsion may involve the whole mesentery of the small intestine; Weible¹⁹ was able to collect 65 such cases from the literature.

Fixation or distortion of the mesentery by bands and adhesions is probably the most frequent cause of volvulus of the small intestine (Fig. 21). At times mesenteric lymph

glands or mesenteric cysts may be etiological factors. The occurrence of volvulus may complicate the convalescence from an abdominal operation. This occurred in 3 instances in the

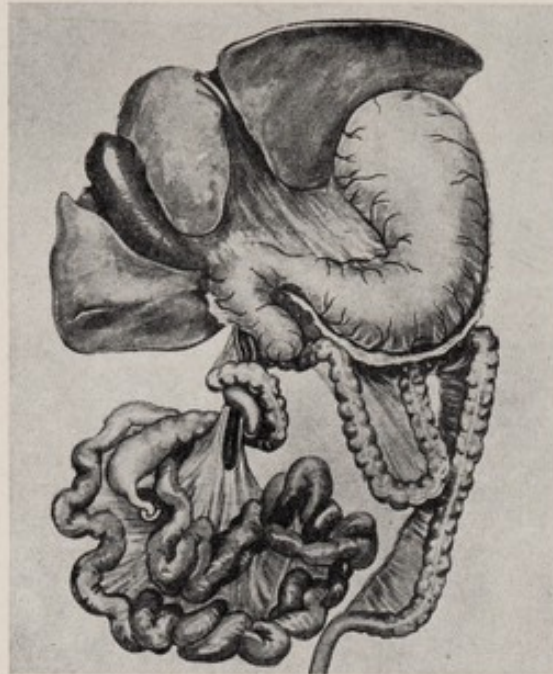


FIG. 22. Volvulus neonatorum, showing dilatation of duodenum and stomach above point of constriction. (Dott.²¹)

Massachusetts General Hospital series; in two of these cases the small intestine was involved, in one the cecum. There were 2 cases of volvulus in this series in which no previous operation had been carried out. The operative notes upon one of these cases showed that the mesentery was very long and that the intestine was adherent in the pelvis. In the other case a complete twist of the mesentery of the small intestine had taken place in a girl aged sixteen years; after the volvulus was untwisted, the bowel had seemed to be viable, but thrombosis of the mesenteric veins set in and the patient died about forty-eight hours later, the autopsy showing thrombosis of the mesenteric vessels with complete infarction of the small intestine.

The clinical course of obstruction of the small intestine by volvulus does not, in general, differ from that in other cases where strangulation of the small intestine is present.

VOLVULUS NEONATORUM. Dott² has drawn attention to a variety of volvulus occurring in the newborn, which he calls "volvulus neonatorum." The child often appears normal for a few days and meconium is passed. With the onset of the volvulus, vomiting sets in; the vomitus is deeply bile-stained. The upper half of the abdomen only is distended: since there is obstruction only at the upper extremity of the twisted gut, and free exit through the colon below, the lower bowel does not become distended (Fig. 22). Melena may or may not be evident. More recently, Dott reported a case²¹ where the lesion was diagnosed and successfully operated upon. Rixford's case¹ where this type of lesion was found in a five-year-old child is of interest. Donald²² has reported a somewhat similar case in an adult.

See also Diagnosis, p. 240, and Treatment, p. 300.

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CHAPTER VI

VARIETIES OF OBSTRUCTION (Continued)

OBSTRUCTION BY INTUSSUSCEPTION

DEFINITION. By the term intussusception is meant the invagination of one segment of the intestines into another. It is an important and serious cause of acute obstruction of the intestine. Its relative frequency in various clinics is shown in Figure 2 and Table 1. Since this disease is commonly one of infancy and early childhood, the frequency of its occurrence in relation to other types of acute obstruction in any particular clinic depends upon the ratio of children to adults admitted. Thus, in the figures of Ladd and Cutler¹ from the Children's Hospital, Boston, out of 102 cases, including all varieties of acute obstruction, intussusception was responsible for the obstruction in 88 cases (86 per cent). Peterson's² figures, also based on the occurrence of acute obstruction in infancy and childhood, show about the same relative frequency of obstruction from intussusception to obstruction from other causes.

The anatomy of an intussusception forms an interesting picture, the details of which are shown in Figure 23. The common terms used in describing an intussusception are also shown in this diagram: the outer layer is known as the "sheath" or "receiving layer" or "intussusciens"; the invaginated portion is known as the "intussusceptum," and is composed of two layers, the entering and the returning; between these layers is found the mesentery and at times mesenteric glands or the appendix. The most advanced portion of the intussusceptum is the "apex" of the intussusception, the point of entrance of the intussusceptum is the "neck." The entering and returning layers have their serous surfaces in apposition; the returning layer and the sheath have their mucosae apposed. Although the facts are well known, a great many diagrams that illustrate the

anatomy frequently do not stress the most characteristic and important feature, namely, the fact that not only is one segment of the bowel telescoped into another, but the entering or "swal-

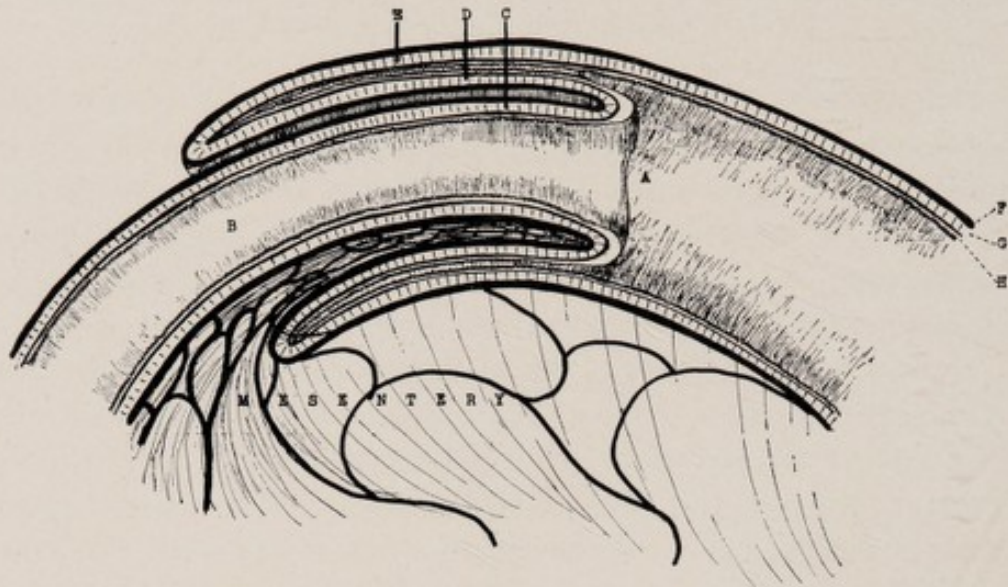


FIG. 23. Diagram showing different parts of an intussusception. Sheath or receiving layer is known as intussusciens; swallowed portion is intussusceptum. Note compression of mesentery between layers. (A), Apex of intussusceptum. (B), Neck of intussusceptum. (C), Entering layer of intussusceptum. (D), Returning layer of intussusceptum. (E), Intussusciens. (F), Peritoneum. (G), Muscularis. (H), Mucosa.

lowed" portion consists not only of bowel wall but also of mesentery. This vital structure is caught between the entering and returning layers (see Fig. 23) and forms a somewhat wedge-shaped mass with its apex attached to the most distal point of the intussusceptum and its base lying at the neck of the intussusception. It is obvious that as the intussusception increases, the mesentery will be put more and more on the stretch, and will be more and more compressed at the neck. The pull of the mesentery gives the characteristic curved shape to the intussusception.

There are occasional cases in which the invagination is not single, but compound; it may be double or triple; the latter condition is very rare. In these cases there is first a primary

single invagination, such as is shown in the diagram, and then secondary invaginations which occur around the first. Cases have also been reported where two or more invaginations occurred simultaneously in different portions of the intestinal tract.³

Another rare form is the so-called "retrograde" intussusception. In the common forms of intussusception the invagination occurs in a descending direction: in other words, the apex points downward in the intestinal canal. In retrograde intussusceptions the apex points upward; the intussusception having occurred in an ascending direction. According to Treves,⁴ a primary descending intussusception may be associated with a secondary ascending or retrograde intussusception. Cases of retrograde gastrojejunal⁵ intussusceptions have been reported following posterior gastroenterostomy.

Intussusceptions are frequently found at autopsy that have evidently occurred only a short time before death, and for this reason are called "agonic" intussusceptions. They are small, and show no evidence of congestion or inflammation. They are probably associated with the violent spasmodic contractions and violent peristalsis that are commonly encountered if the blood supply is cut off from the intestines.⁶ According to many writers they are most commonly met with in children who have died of some disease of the central nervous system. They are often multiple and frequently of the ascending or retrograde type. They are, of course, of no clinical significance; and the only point is to make a correct diagnosis of the condition at autopsy.

VARIETIES OF INTUSSUSCEPTION. Intussusceptions are of three major varieties, the classification being made on an anatomical basis. The terminology which was generally accepted until 1873, and which is still the simplest, describes as "enteric" those cases in which the small intestine alone is involved; as "colic" those in which invagination of the colon alone occurs; and as "ileocecal" all cases of intussusception in which the terminal ileum and cecum are involved.

Enteric Intussusceptions. Enteric intussusceptions, those in which the invagination involves the small intestine alone, form a relatively small group (about 10 to 15 per cent^{7,8}); but since this group occurs chiefly in older children and adults, the exact percentage would depend on the ratio of infants to older individuals admitted to any given clinic.⁸ An obvious organic lesion is often responsible for the intussusception; among the inciting causes may be mentioned Meckel's diverticulum, a polyp, carcinoma, hemangio-endothelioma, tuberculous ulceration⁹ and possibly an inflamed Peyer's patch. Watts stressed the importance of adenomata.¹⁰

Colic Intussusceptions. Colic intussusceptions, those involving the colon only, constitute about 6 per cent of all cases of intussusception. In those occurring in early life, Perrin and Lindsay⁷ stress the importance of mucosal folds and lymphoid follicles as etiological factors. In the colic intussusceptions of adult life, just as in the enteric intussusceptions, some type of tumor is ordinarily responsible. Chronic intussusceptions having carcinoma as a basis occur not infrequently.

Intussusceptions Involving the Terminal Ileum, Cecum and Colon. There is hopeless confusion in the terminology at present used for the large and important group of intussusceptions involving the terminal ileum, cecum and colon. This is due in part to the fact that a vast number of terms have been created to describe differences in the starting-point or method of growth of these intussusceptions, and writers have used dissimilar terms to describe similar varieties, and similar terms to describe dissimilar varieties. Another factor that has led to confusion is that, even granting a uniform use of terms, it is often difficult, or impossible, at operation to identify accurately the anatomical points that serve as a basis for classification; and the distinguishing features are often lost in the process of reduction. It is thus hard to be sure in which group a given intussusception involving the terminal ileum and cecum should be classified. It is suggestive in this connection

that in studying 363 cases of intussusception Close¹¹ found a statement as to the type in only 185 instances.

In examining the literature we find that most writers divide

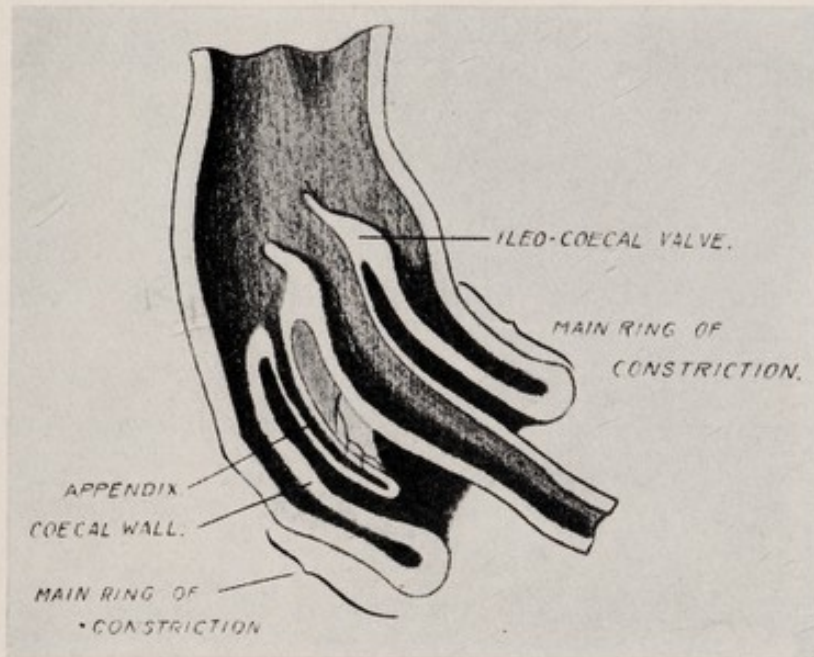


FIG. 24. Ileocecal intussusception. Ileocecal valve heads intussusception. Cecal wall lying next to appendix will, after reduction, often present a characteristic "dimpled" appearance due to edema and congestion. Note "main ring of constriction." (Perrin and Lindsay.⁷)

the old form "ileocecal" into two main groups: ileocecal and ileocolic. There is general agreement that the first of these groups, the ileocecal, should include those intussusceptions where the ileocecal valve forms the apex of the intussusception* (see Fig. 24). There is good reason for also including as ileocecal those cases formerly classified as "caput-ceci," "cecocolic" and "ileocecal-caput-ceci," where the base of the cecum was supposed to form the apex of the intussusception.†

* Corner¹² considers this variety very rare, while Perrin and Lindsay feel that it makes up about 46 per cent of all intussusceptions.

† It has been noted for a long time that after an intussusception involving the terminal ileum and cecum had been reduced a dimpling of the cecum was often present. This was considered by certain authors to indicate that the head of the cecum had formed the apex of the intussusception and they therefore gave to cases in which this occurred the special names "cecocolic," "caput-ceci" and "ileocecal-caput-ceci." Fitzwilliams

The term used for the second subdivision of the old form "ileocecal intussusception," namely, "ileocolic," has given rise to considerable confusion. Leichtenstern,¹³ who originated the term, defined this type of intussusception as "a prolapse of the ileum through the ileocecal valve"; Fitzwilliams,³ and Perrin and Lindsay,⁷ doubt whether a prolapse ever takes place, believing that what really occurs is the passage through the ileocecal valve of an intussusception that has started in the ileum close to the valve. (See Fig. 25. *) Treves¹⁴ and Leichtenstern recognized that this latter method of invagination occurred at times and designated it as "iliaca-ileo-colica" intussusception.

There remains to be described one other not uncommon form of intussusception occurring in the region of the terminal ileum and involving the cecum and colon. In this type the intussusception forms in the terminal ileum, usually within 12 inches of the ileocecal valve, passes down to the ileocecal valve and enters the colon by pushing the valve before it, at times becoming impacted in the valve. During the reduction of this type of intussusception, when the cecum is completely emptied, an enteric intussusception is found. Clubbe¹⁵ places this form in his "enterocolic" group†; Perrin and Lindsay prefer to classify it as a "compound" intussusception and consider that it is much more frequent than is generally

and Perrin and Lindsay consider that the dimpling is not to be interpreted in this way but that it is a secondary feature, caused by pressure and edema, the ileocecal valve having really formed the head of the intussusception. They therefore include these varieties among their ileocecal intussusceptions and urge that the terms cecocolic, caputceci and ileocecal-caput-ceci be abandoned. Even granting that in certain instances the end of the cecum may form the apex of the intussusception, this probably occurs very seldom; and considering the close proximity of the ileocecal valve to the end of the cecum, the present writer feels that for the sake of simplicity these intussusceptions had best be grouped as ileocecal.

Cases have been reported in which there first occurred an invagination of the appendix into the cecum, followed by an invagination of the base of the cecum.¹²

* It will be noted in the illustration that if the intussusception were seen after the enteric intussusception had unfolded itself into the cecum, it would be impossible to tell whether or not it had started as a prolapse. Fitzwilliams contends that it does not.

† Clubbe has no group called ileocolic; but includes the ileocolic of Perrin and Lindsay as a subdivision of his "enterocolic."

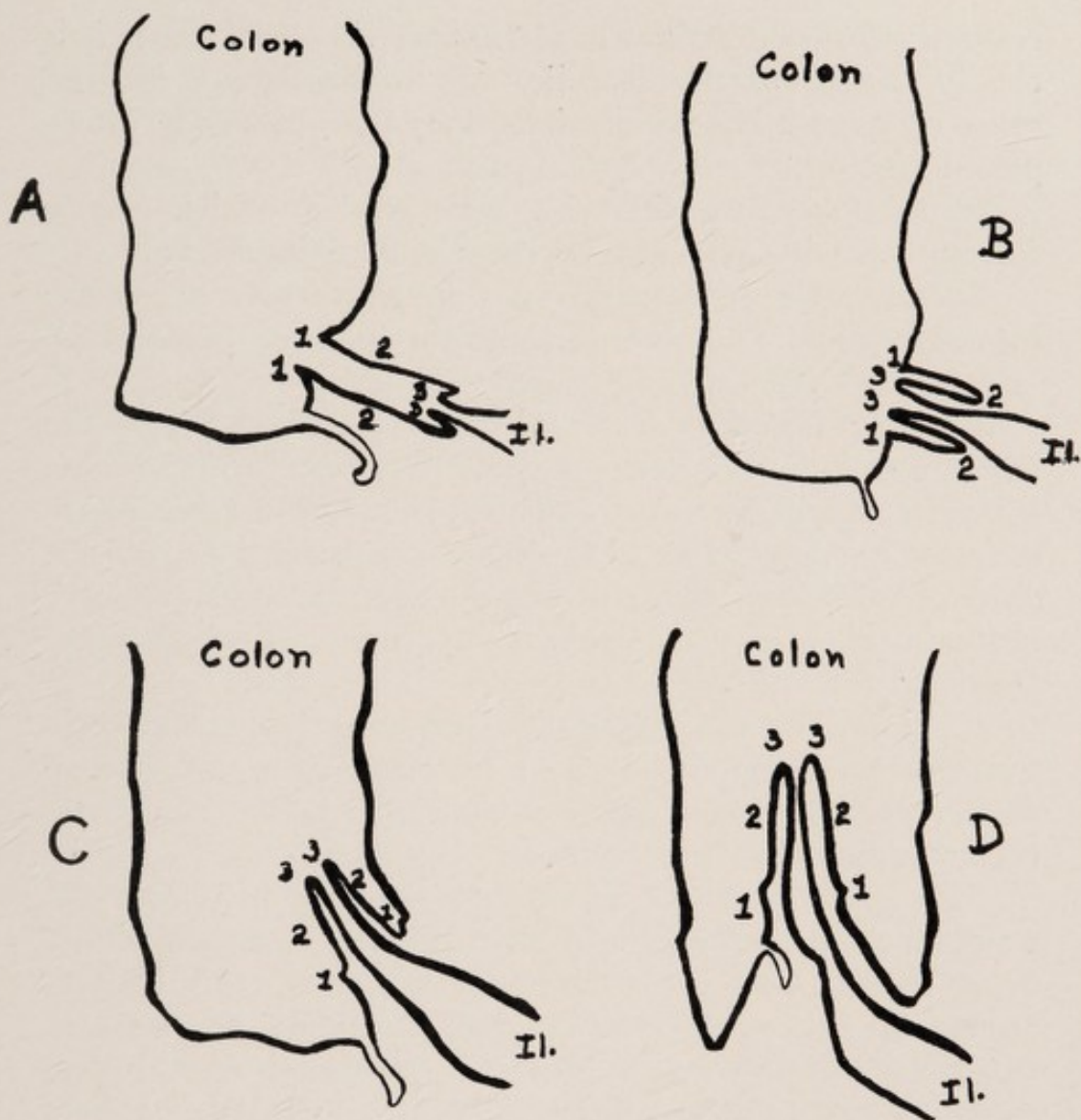


FIG. 25. Mechanism of formation of an ileocolic intussusception according to Fitzwilliams. Numbers denote fixed points on ileum. (A), an enteric invagination starting at 3. (B), 3 remaining the apex passes through valve, while 2 rolls round at neck from *outer to middle* layer. (C), all ileum of sheath has now passed round neck into middle layer. (D), growth proceeding, valve, cecum, and colon roll round into middle layer. As in all other forms of intussusception, apex has remained constant. (Fitzwilliams.³)

realized; Corner describes it as "enteric ileocecal." Since it is closely related to the ileocolic, and to distinguish it from other compound intussusceptions, I propose calling it "compound ileocolic."*

In summary, the following classification, which closely corresponds with Perrin and Lindsay's, is recommended:

Ileocecal: Forms in which the ileocecal valve heads the intussusception. This term includes the so-called "cecocolic," "caput-ceci" and "ileocecal-caput-ceci."

Ileocolic: Forms in which the ileum is found within the cecum but the valve does not form the apex. This condition may come about as a result of an intussusception forming in the terminal ileum close to the valve and passing through the valve (see Fig. 25). It is a disputed point whether an ileocolic intussusception may also come about, rarely, as a prolapse of the ileum through the valve.

Compound Ileocolic: Cases in which an enteric intussusception starting in the ileum about 12 inches from the ileocecal valve enters the colon by pushing the valve before it, at times becoming impacted in the valve.

The great majority of intussusceptions involving the terminal ileum and cecum occur in infancy and early childhood. Usually no organic pathology can be demonstrated as responsible for these intussusceptions, unless the view of Perrin and Lindsay, and others, be accepted, that hypertrophy of the lymphoid tissue is commonly present.

MECHANISM OF THE INVAGINATION. The questions of how and why invaginations of the intestines take place have interested investigators for a long time. A great many theories or speculations have been advanced to account for the phenomena, and all points are not yet clear. There are, however, certain facts that must be taken into consideration.

* There are other types of compound intussusception that may occur in the region of the ileocecal valve. For example, Perrin and Lindsay quote a case in which the intussusception started as an ileocolic intussusception and when 9 inches of the ileum had passed through the ileocecal valve the colon was invaginated into itself.

In the first place, one must assume a perverted action of the normal peristalsis. It is in attempting to explain the underlying cause of these abnormal intestinal movements and the exact form that they take that conflicting theories arise.

The modern view inclines toward the belief that there is an organic basis for the abnormal intestinal movements in the great majority of cases;⁷ it is a well-known fact, for example, that intussusceptions frequently occur in connection with a tumor which may be a polyp, carcinoma, Meckel's diverticulum, submucous angioma and so forth, attached to the internal wall of the bowel. It has been suggested⁷ that some intussusceptions which have heretofore been considered purely functional may have an organic basis in the presence of hypertrophied lymphoid tissue, particularly around the ileocecal valve and terminal ileum.

There are, however, intussusceptions for which no organic basis can be found. Surgeons have observed that minor degrees of intussusception take place and reduce themselves in the course of operations. Also minor degrees of invagination are easy to produce in animals and often occur spontaneously (Treves¹⁶ and Alvarez¹⁷).

Meckel's diverticulum, which so frequently furnishes the organic basis for an enteric intussusception (occurring in almost half the total cases analyzed by McIver⁸ in which organic pathology was responsible for the intussusception; see also Hertzler and Gibson¹⁸ and Lower¹⁹), may be used as a representative of the type of intussusception brought on by a tumor or polyp attached to the bowel wall. Figure 26 shows the relation, in an illustrative case, of the diverticulum to the intussusception. It will be noted in the first place that the diverticulum is inverted into the lumen of the intestine. In regard to this point, cases of intussusception associated with a diverticulum have been reported (Porter²⁰) in which there was no inversion of the diverticulum; but in the great majority of cases (Harkins,²¹ Hertzler and Gibson¹⁸) inversion of the Meckel's diverticulum into the lumen of the gut is present.

Hertzler and Gibson consider that the inversion occurs as a secondary process; but this seems most unlikely. To most authors the inversion has seemed to be a very important



FIG. 26. Intussusception caused by Meckel's diverticulum. A specimen found at operation, shown in diagrammatic form. The Meckel's diverticulum was inverted in the lumen of the gut. It will be noted that the diverticulum did not form the apex of the intussusceptum, but was attached near the proximal end. (McIver.⁸)

factor in the later production of the intussusception, and numerous speculations have been indulged in as to the method of its occurrence. Two theories stand out as being most plausible. The first is that an active peristalsis of the walls of the diverticulum in an effort to expel its contents brings about the inversion. The second, which has received considerable attention, is that an inflammatory process occurs around the opening of the diverticulum into the lumen of the intestine, producing a thickening of the walls of the diverticulum, and that this process extends until the mucous membrane protrudes into the lumen of the intestine, where it is caught up and pushed along by the intestinal peristalsis.

In regard to the second point, namely the manner in which the intussusception is produced, it may be said that the mechanism is probably the same whether the foreign body attached to the intestinal wall be an inverted Meckel's diverticulum, an adenoma or a single polypus. An explanation commonly offered is that the diverticulum or other tumor is dragged along by the peristaltic movements of the intestine, the point of attachment being thereby invaginated and forming the apex of the intussusception. Wardill²² discusses this point (with reference to the intussusceptions caused by polypoid growths in general) and points out the fact that this theory cannot be upheld since in a number of cases the polypus does not form the apex. He believes the true explanation to be that

there is a spasmodic contraction of the gut around the polypus and an inhibition of the part immediately distal to this; his article carries illustrations of this point. In some of the cases reported in the literature (Porter²⁰), however, the inverted diverticulum does occur at the apex of the intussusception.

The most extensive experiments on the mechanism of the invagination have been carried out by Nothnagel.²³ This author showed that if electric stimulation was applied to a segment of intestine a tetanic constriction followed, the portion of the bowel below the contracted area was pulled over the contracted portion like a sheath, and the growth of the intussusception took place by the pulling of more and more of the lower segment of bowel over the contracted portion. The experiments of D'Arcy Power²⁴ on the artificial production of intussusceptions essentially confirms the views of Nothnagel.²³ Nothnagel considered that the longitudinal muscles of the intestinal wall were chiefly active in pulling the normal intestine up over the spastic constricted portion. Fitzwilliams³ and Treves¹⁶ in general agree with this view; the latter describing the action of the longitudinal fibers as follows: "The action of the longitudinal fibres must extend beyond the line where the contracted and non-contracted parts of the intestine meet. If they be considered to act from the contracted segment as from a fixed point it is evident that they will tend to draw the wide non-contracted segment over the narrow and contracted piece. In this way, by the drawing of one part of the intestinal tube over another part, the intussusception is formed." Treves,²⁵ however, does not consider that this method of growth applies to the variety of intussusception where a portion of the ileum prolapses through the ileocecal valve. Here, according to this author, the invagination may increase for some time solely by the prolapse of more and more ileum, the sheath remaining unchanged. Later, when no more ileum can enter, the cecum and ascending colon may be drawn into the sheath. Fitzwilliams³ devotes considerable space in his article to the mechanism of the invagination.

PATHOLOGICAL ANATOMY. Little that is fundamentally new in the pathology of intussusception has been added since Leichtenstern's classical monograph in 1873.¹³ It is easily understood that the pathological picture presented by an intussusception depends largely on the extent of involvement of the mesenteric circulation. A simple invagination of the bowel that does not constrict the mesenteric vessels may result in very slight changes in the bowel wall, and the condition may be tolerated by the patient for weeks, or even months (one variety of the so-called "chronic intussusception"). In acute intussusception, however, constriction of the mesenteric vessels, particularly the veins, is the important factor. As already pointed out, the mesentery forms a wedge-shaped structure imprisoned between the entering and the returning layers (see Fig. 23); as the intussusception grows, more and more of the mesentery is constricted at the narrow neck. To this mechanical action is added the swelling and edema of the intestinal wall following the venous stasis and congestion, which further compresses the mesenteric vessels, particularly at the neck of the intussusception; the mesentery itself then becomes swollen and edematous and adds to the congestion. The changes that take place in the bowel wall under these circumstances are similar to those that are later described under strangulation (p. 143): they may vary from simple congestion and edema to complete infarction and gangrene. The intussusceptum, or swallowed portion, suffers first due to damage to its blood supply; and gangrene may be limited to the intussusceptum. In this respect, infarction of the bowel in intussusception differs from the infarction that follows strangulation by bands; for the gangrenous portion of the bowel may be entirely walled off from the general peritoneal cavity by the enveloping sheath, which at times furnishes complete protection against peritonitis or the absorption of toxins.* In certain cases the intussusceptum, after

* There are two types of obstruction in which infarction of the bowel may occur and absorption be at a minimum: one, in certain cases of intussusception; and the other in certain cases of strangulated external hernia.

undergoing degenerative processes, may be sloughed off at the neck of the invagination and be passed by rectum, constituting a spontaneous cure.* On the other hand, it may cause further trouble: cases have been reported where the necrotic portion of the bowel after being separated constituted a foreign-body obstruction lower in the intestinal canal; and Nothnagel says that the separated intussusceptum may set up a peristalsis so violent as to cause a rupture of the bowel at the point of recent attachment, with a resulting peritonitis.

The histological picture of the changes taking place in an intussusception is described in detail in the work of D'Arcy Power.²⁶ The layer of bowel forming the outer sheath (the intussusciens) may remain essentially normal; it may, on the other hand, due to the pressure and inflammation of the necrotic intussusceptum, be involved in the gangrenous process; or an intermediate condition may be found in which the wall is congested, thickened, and edematous. The mucosa may show ulcerations which may perforate, allowing the intussusceptum to protrude through the slough; the lumen of the bowel may be clogged or blocked by the swelling of the tissues or by clots of blood and débris. Angling or knuckling of the intussusceptum produced by the tension on the mesentery may also obliterate the lumen.

The different layers making up the intussusception may become so firmly adherent one to another as a result of inflammatory changes that reduction is impossible. Reduction may also be impossible simply from inflammation, congestion and edema, even in cases that come to the surgeon reasonably early.

Many interesting and curious details of the pathological changes found in association with intussusceptions are given in the works of Treves²⁵ and Nothnagel.²³ Corner¹² points out that where a Meckel's diverticulum is patent an intussusception may protrude at the umbilicus.

* The ulcer left at the neck of the invagination after sloughing has taken place may, in the process of healing, lead to a stricture of the bowel. (Nothnagel).

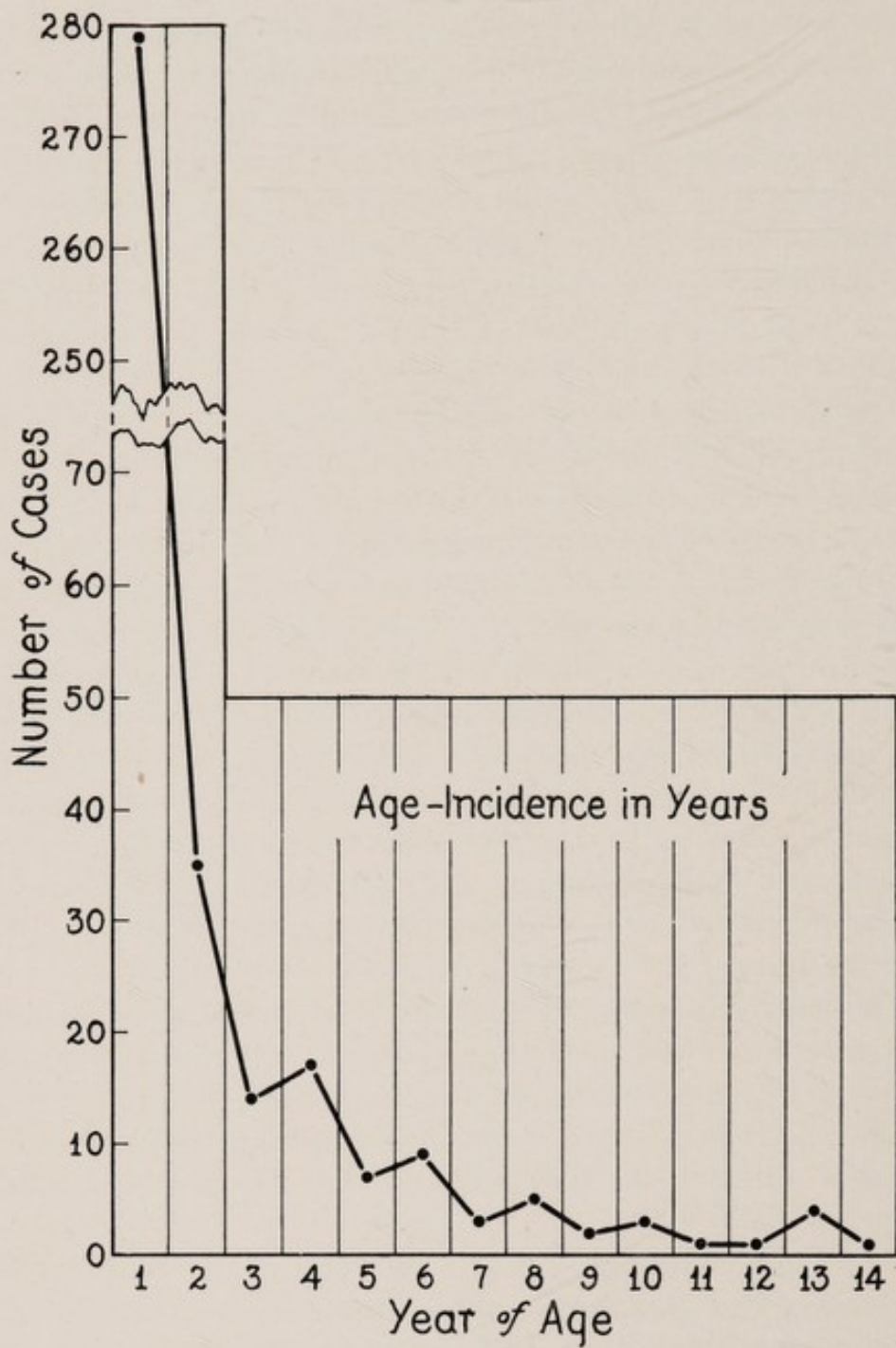


FIG. 27. Curve illustrating age incidence of acute intussusceptions, in years: 382 cases are included in the curve, which ceases at age of fourteen years, for convenience of insertion; 18 other cases occurred between ages of fourteen and fifty-eight, making the total 400. (Perrin and Lindsay.⁷)

CLINICAL FEATURES. Intussusceptions are divided clinically into acute and chronic types. The latter show no symptoms of acute intestinal obstruction and are not considered in

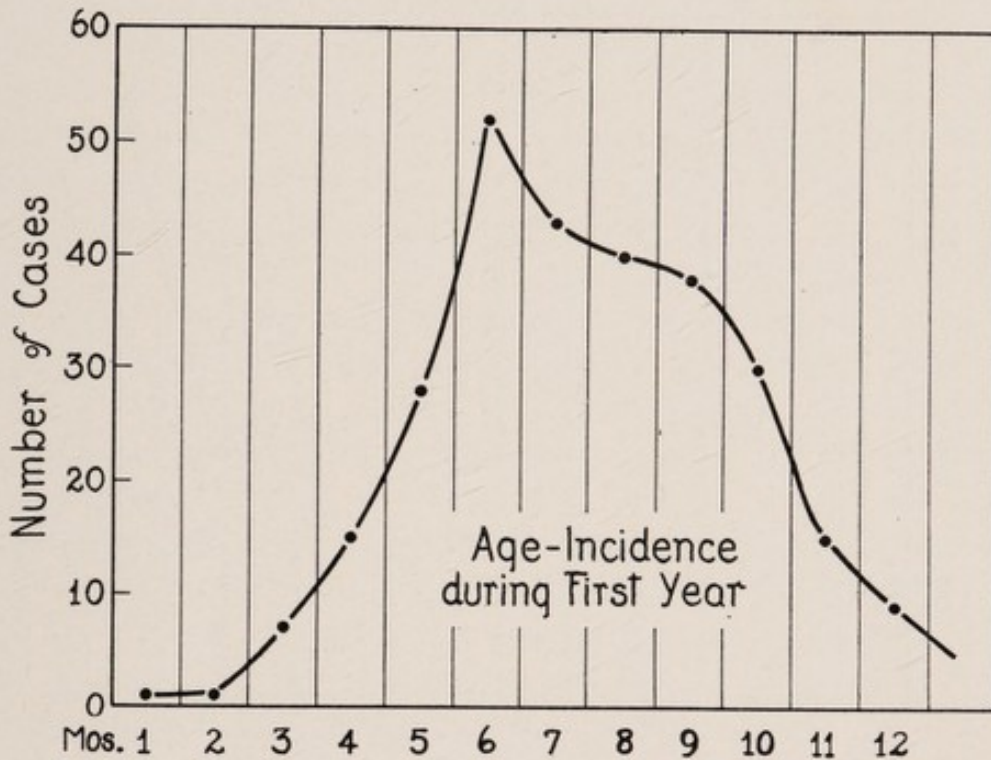


FIG. 28. Graph of age incidence of acute intussusception, in months during first year. (Perrin and Lindsay.⁷)

this monograph; the reader is referred to articles by Jones²⁷ and Marsh²⁸ for a comprehensive description of the chronic type.

The acute cases fall into two major groups: those occurring in infants and young children; and those occurring in adults. According to Bolling,²⁹ about 75 per cent of all acute cases occur within the first year of life; Perrin and Lindsay⁷ reported 78.5 per cent as occurring within the first two years (Fig. 27), the first year showing a very marked peak between the fifth and sixth months (Fig. 28).

Males are affected approximately twice as frequently as females, a rather striking fact that has never been adequately explained. The disease shows a definite seasonal variation

(Fig. 29), the greatest number of cases occurring in spring with a secondary peak in winter. The theories advanced to explain this seasonal variation are not convincing. It might

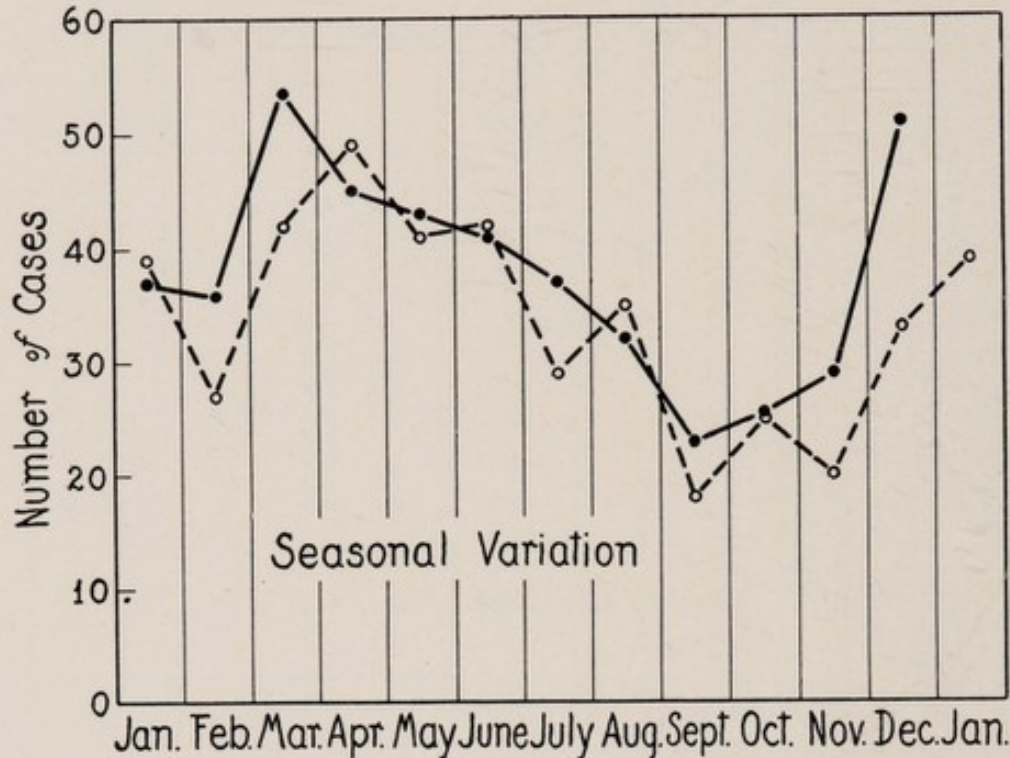


FIG. 29. Curves illustrating seasonal variation of acute intussusception, compiled from cases reported by Perrin and Lindsay⁷ and by Fitzwilliams.³ (Solid line, Fitzwilliams; broken line, Perrin and Lindsay.)

be expected that the disease would reach its height during the summer season when the various types of infectious diarrhea are more prevalent; but it is evident that such is not the case, although Bolling²⁹ reports a small group where the intussusception occurred during an attack of "ileocolitis."

Indiscretions of diet are frequently mentioned as causing intussusception; but Close's¹¹ and Peterson and Carter's³⁰ studies do not bear out this belief. Intussusception may occur as a complication of Henock's purpura. (See Diagnosis, p. 246.)

Intussusceptions may recur after operation. In Close's series of 363 cases, this occurred in 14 instances, usually within a month or two after operation, but in several instances after an interval as long as a year.

The clinical course in infants is usually typical. A previously healthy, well-nourished infant cries out while nursing or passing a stool; with the paroxysm of pain the knees are drawn up, and there may be a noticeable pallor. Vomiting is likely to take place soon after the onset of the attack. One normal stool may be passed; after that, only blood and mucus; occasionally some flatus may be passed. The paroxysm of pain may recur at frequent intervals. Even after the lapse of twenty-four hours, the infant may appear surprisingly normal and without signs of shock, so that from the general appearance it may be difficult to realize that the situation is so serious. In the later stages the infant may look extremely ill and show signs of collapse; in the most serious cases where death follows operation there is likely to be a marked postoperative elevation of temperature.¹

On physical examination of the abdomen the characteristic "sausage-shaped" mass can usually be palpated. Distention is not likely to be marked in the early stages. On rectal examination the apex of the intussusception may be felt, or, rarely, it may be seen protruding from the anus.

In contrast to the uniformity of the picture in infants and children, the picture in the adult is usually not characteristic and the diagnosis is often made only at operation. The clinical signs and symptoms may point strongly to an acute obstruction; but the typical features of intussusception (passage of bloody mucus by rectum, and the characteristic abdominal mass) are often absent.⁸ One reason for their absence is that these intussusceptions are frequently of the enteric variety. A more detailed discussion of intussusception in the adult will be found in articles by Eliot and Corscaden,⁹ Watts,¹⁰ and McIver.⁸

See also Diagnosis, p. 244, and Treatment, p. 297.

Cases IX and X are typical of intussusception in infants:

CASES IX and X. *Obstruction by intussusception.*

No. 7705, MIBH. Female, aged three months. The infant was full term, normally delivered, and had been well up to the onset of the present

illness. There had been no digestive disturbances except slight constipation.

On the morning of admission the infant waked at 5 A.M., cried and drew up her legs as though in pain. A mild cathartic was given by the parents, and almost immediately a small amount of bloody mucus was passed by rectum. The infant was seen about six hours later. There were periods in which she cried as though in pain. There was some pallor of the mucous membranes; but the general condition appeared good. The abdomen was soft and not distended. In the region of the left upper quadrant and extending across toward the mid-line a rounded mass could be felt very definitely at one time, while examination a few minutes later showed that it had disappeared; after a short interval the child began to cry again, and again the mass could be felt. This probably coincided with a wave of violent peristalsis. Rectal examination was negative. The white blood count was 11,000; temperature 98°F. A barium enema showed the main column of barium stopping rather sharply in the region of the splenic flexure. (See Fig. 53, p. 236.) The roentgenologist considered the picture very suggestive of an intussusception.

A diagnosis of intussusception was made, and an abdominal operation was carried out under ether anesthesia. At operation an intussusception was found, the apex lying in the transverse colon to the left of the midline. The intussusception was easily reduced by taxis. After the small intestine had been "milked out" of the colon, it was noticed that a second intussusception was present in the ileum about 12 cm. from the ileocecal valve. This intussusception was about 4 or 5 cm. in length and was reduced with slightly more difficulty because of the edema and swelling. The portion of the bowel involved in the intussusception was edematous and cyanotic in appearance, but appeared definitely viable; it was obvious that no resection was required. In the mesentery of the small intestine at the point of the intussusception there were several enlarged lymph glands about 1.5 cm. in diameter which may have been an etiological factor in the production of the intussusception. The infant had an uneventful convalescence.

Comment: This intussusception was of the compound ileocolic variety. The diagnosis was fairly obvious: there was the typical picture of a previously healthy infant suddenly crying out and drawing up the legs as though in pain, and shortly afterward passing bloody mucus by rectum. The fact that the mass could be felt at times and at other times could not be somewhat atypical. A few hours' delay in this case might have been very serious, since the circulation to the involved section of bowel was obviously seriously jeopardized. It was, of course, a mistake on the part of the parents to give the infant a cathartic: unfortunately this is a common first procedure in cases with abdominal pain; in this case it undoubtedly aggravated the symptoms.

No. 5415, MIBH. Male, aged six months. The infant had been well previous to the time of the present admission. The mother states that on the morning of admission the baby appeared to have severe abdominal colic, crying out and drawing up his legs. Castor-oil was administered; this was promptly followed by vomiting and the baby began to pass bloody mucus from the rectum. An enema produced more of this bloody discharge; there was no bowel movement. A few hours later the mother noticed a mass in the left lower quadrant of the abdomen and brought the infant to the hospital.

Examination showed an extremely sick child: the mucous membranes were pale; the pulse rapid; the child was very apathetic and lay quietly on the examining-table, although at intervals he cried out as though in pain, drawing up his legs. A definite mass, somewhat "sausage-shaped" in outline, could be felt in the lower left quadrant and on rectal examination this could be palpated in the pelvis. The abdomen was not distended. There was no muscle spasm and no marked tenderness. The white blood count was 18,000, differential showed polymorphonuclears 82 per cent, small lymphocytes 17 per cent, large lymphocytes 1 per cent. Temperature was 99°F.

Diagnosis of intussusception was made, and immediate operation advised. The abdomen was opened under ether anesthesia. The apex of the intussusception was located in the sigmoid. It was reduced by taxis. The first part of the reduction was simple; but as the apex neared the cecum there was difficulty in effecting complete reduction; this, however was accomplished. Although edematous and somewhat discolored, the lower portion of the ileum was evidently viable. There was a mass of enlarged mesenteric glands in the ileocecal region, the largest being about 2 cm. in size, and a dense, fibrous band which passed across the ileocecal valve and appendix and terminated in the group of glands. This band was divided; the abdomen was closed and the child returned to the ward in fair condition. The infant showed a rather marked elevation of temperature for the first two days; after that time there was an uneventful convalescence.

Comment: The history and physical findings in this case (sudden onset of abdominal pain in a previously healthy infant, followed by the passage of bloody mucus, and after a few hours a palpable mass in the abdomen) were typical of intussusception. A strong cathartic was administered at the onset of the illness, which was unwise and only served to aggravate the symptoms.

CASE XI. *Obstruction from enteric intussusception due to Meckel's diverticulum.*

No. 284725, M.G.H.; Male, aged twelve. History meager, and rather unreliable, since the boy, was somewhat irrational and mother spoke no English. Patient is said to have had attacks of mild abdominal pain,

lasting fifteen or twenty minutes, during past eight months; occasionally vomited during these attacks.

Forty hours before admission the patient was taken with severe, generalized, abdominal pain, which was attributed to green apples eaten the day before. Vomited a number of times during the first day; pain persisted. Was given castor oil, which was vomited. During the day of admission the pain was said to have been more severe in the right lower quadrant; vomited five or six times, the vomitus being greenish in color and without odor. Bowels had not moved for two days previous; no symptoms referable to cardiorespiratory or genitourinary system.

Physical examination showed a slightly built boy of twelve, apparently in considerable pain, excited and disturbed mentally. Abdomen was scaphoid in appearance, moving only slightly with respiration. Marked muscle spasm over the whole abdomen, most marked on the right side; also marked generalized tenderness, perhaps somewhat more pronounced on the right side; no masses could be felt. Rectal examination was unsatisfactory because of lack of cooperation; definite tenderness, however, could be elicited. Temperature 104°F.; white blood count 27,000.

It appeared obvious from the physical examination that we were dealing with some acute pathological condition in the abdomen requiring surgical treatment. It was thought most likely that this was a fulminating appendicitis, although intestinal obstruction was considered.

Operation was undertaken under ether anesthesia, the abdomen being opened through a right rectus muscle-splitting incision. When the peritoneum was opened, free fluid, slightly dark in color, escaped. On exploring with the hand, a mass was felt in the pelvis which on delivery proved to be an intussusception about 20 cm. from the ileocecal valve. A few centimeters of the intussusception were reduced and the bowel found to be gangrenous. It was impossible to reduce the intussusception further, so the whole affected portion was resected. No anastomosis was attempted at this time, since the patient was in very poor condition: he took the ether badly; his pulse soon rose to 150; he became very cyanotic, and respiration stopped. Under artificial respiration breathing again started and no more ether was given. Clamps were left on the distal and proximal ends of the bowel and these were brought out through the wound, which was rapidly closed around it. At the end of the operation the patient was given glucose intravenously and was returned to the ward in fair condition.

The pulse and temperature remained elevated for several days after operation, gradually returning to normal. Clamps were removed from the bowel the second day postoperative, and drainage was free. There was some breaking down of the wound and digestion by the intestinal secretions; but the boy's general condition was good after he had recovered from the acute phase.

About three weeks after the first operation, the wound was opened, a few inches of the ends of the intestine were resected and an end-to-end anastomosis was carried out. The appendix, which was adherent in the region of the wound, was removed.

The boy was discharged twenty-one days after the second operation in good condition. He was seen again three weeks later in good health and symptom-free.

SUMMARY. Intussusception is predominantly a disease of infancy and early childhood. One of the most important features of the pathological anatomy is the compression of the mesentery between the entering and the returning layers.

Cases in which the small intestine alone is involved in the invagination are usually described as "enteric"; those in which the colon alone is involved, as "colic." There is dispute in regard to the classification of the large groups of intussusceptions in which the terminal ileum and cecum are both involved: it is suggested that these be classified as "ileocecal," "ileo-colic" and "compound ileocolic," according to the method of their formation.

There are various views in regard to the mechanism of invagination. There is often an organic basis for the intussusception: in the enteric and colic types an adenoma, carcinoma, polyp or Meckel's diverticulum may be the etiological agent; hypertrophied lymphoid tissue around the ileocecal valve may be a factor in the production of intussusceptions occurring in this region.

The pathological changes that take place in the invaginated bowel depend upon the degree of interference with the circulation, and vary from congestion to gangrene. The intussusceptum suffers first.

Clinically the cases fall into two major groups: a large group occurring in infants and young children, and a small group occurring in adults. On analyzing the first, it is found that there is a seasonal variation and that males are affected twice as often as females. The clinical picture in infancy is usually typical and easily recognized; adult intussusceptions (usually of the enteric variety) may be difficult of diagnosis before operation.

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CHAPTER VII

VARIETIES OF OBSTRUCTION (Continued)

RARER TYPES

INTERNAL HERNIAS. Intestinal obstruction may result from herniation through abnormal openings or slits, congenital or acquired.

Among the most interesting of these herniations are those into the abdominal fossae (see Fig. 30). Many of the anatomical and clinical features of this group are described in contributions by Moynihan,¹ Short,^{2,3} Copenhaver,⁴ and Masson and McIndoe.⁵ The common sites of these fossae are in the region of the ligament of Treitz—the so-called fossa duodenojejunalis—and in the region of the junction of the ileum with the cecum, from which arise the ileocecal fossa, the subcecal fossa, etc. There is also one which is sometimes found in relation to the sigmoid—the intersigmoid fossa, the opening in the mesocolon occurring on the left side of the sigmoid over the bifurcation of the iliac vessels. Extremely rarely the bowel may herniate into the lesser peritoneal cavity through the foramen of Winslow and become obstructed.^{4,6}

A group of hernias that are usually classed with those just described are hernias of the intestines through the diaphragm into the thoracic cavity. These openings of the diaphragm may result from congenital defects or from injury.⁷ Where obstruction occurs in such hernias the mortality is usually high.⁸⁻¹²

Herniation and subsequent obstruction may also occur through openings in the mesentery, omentum, and broad ligaments of the uterus.¹³ According to Treves¹⁴ the openings are most frequently found in the mesentery of the lower ileum and are generally circumscribed by an anastomosis between the ileocolic branch of the superior mesenteric artery and the last of the intestinal arteries. The openings may vary

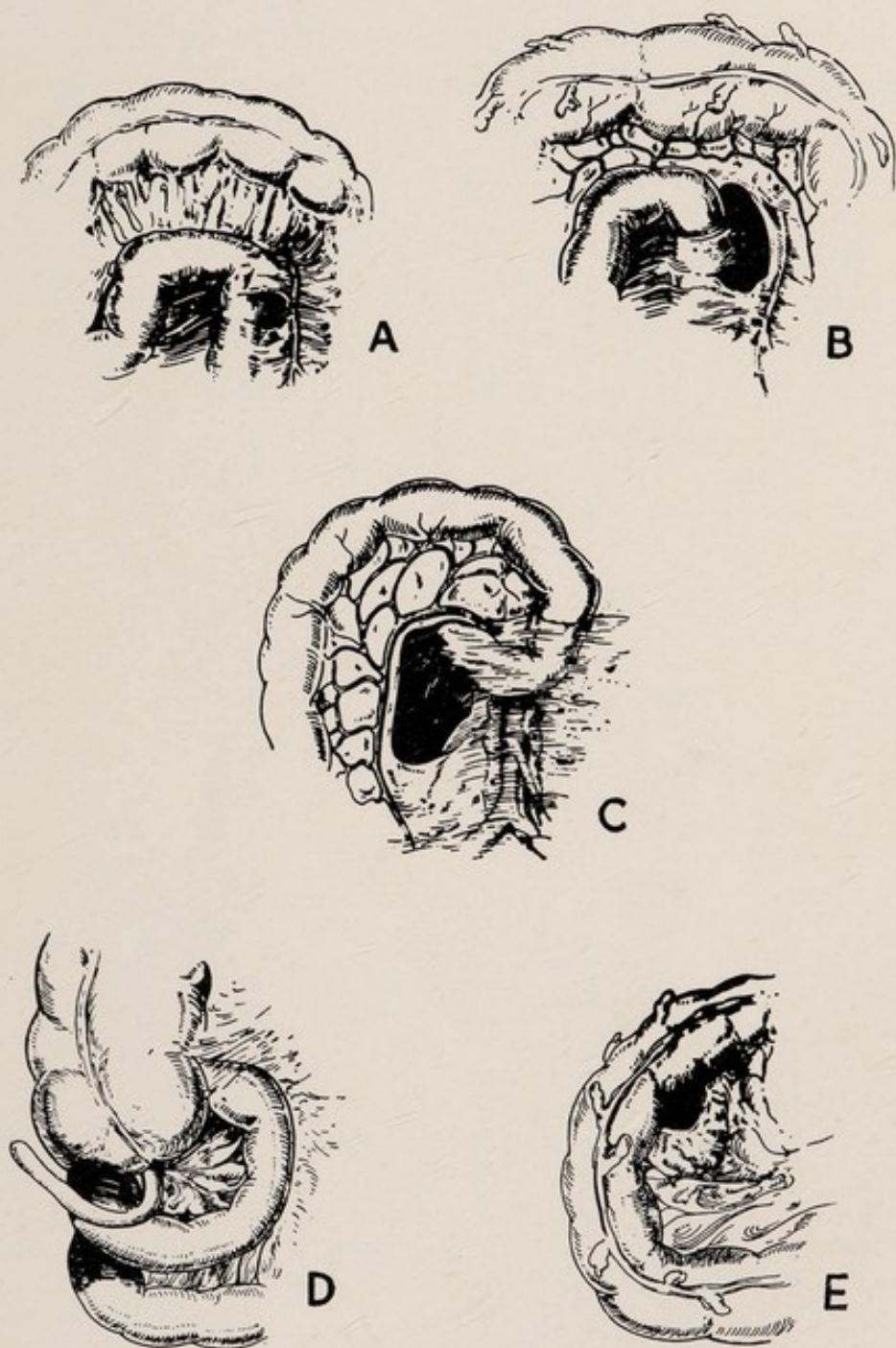


FIG. 30. More common types of retroperitoneal fossae. (Short³)

considerably in size and not infrequently permit the entrance of a considerable portion of bowel. According to the aforementioned author, these holes in the mesentery may at times

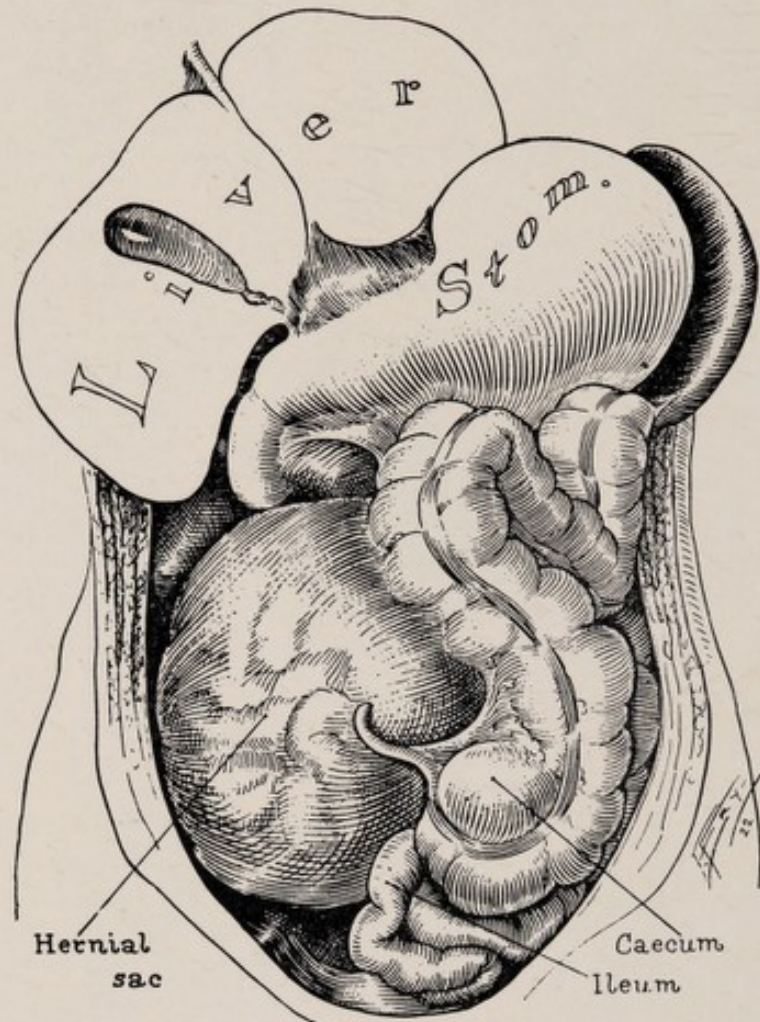
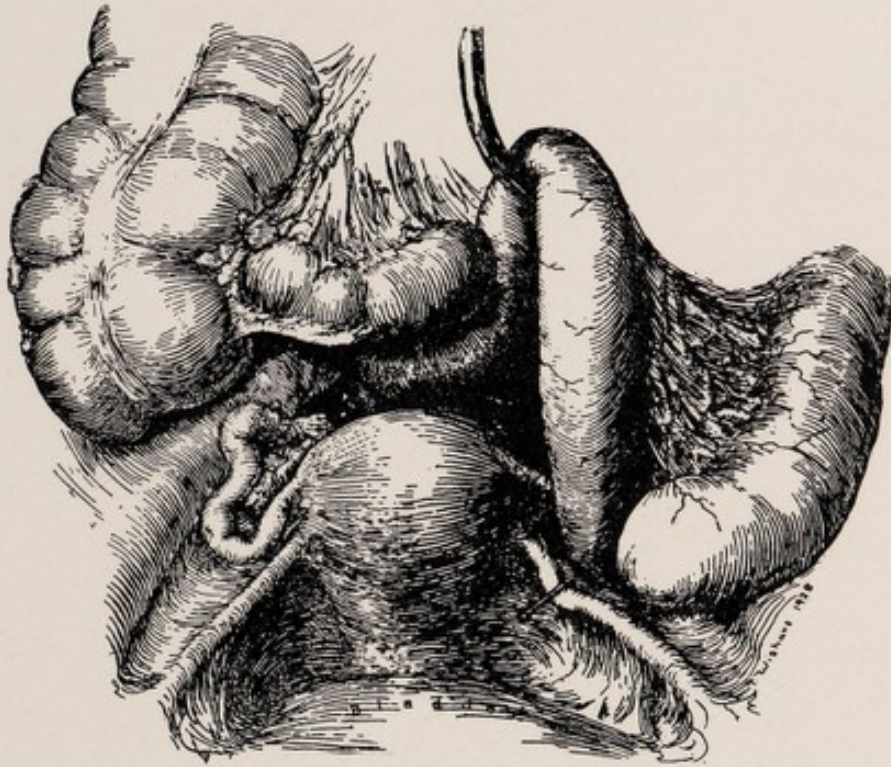


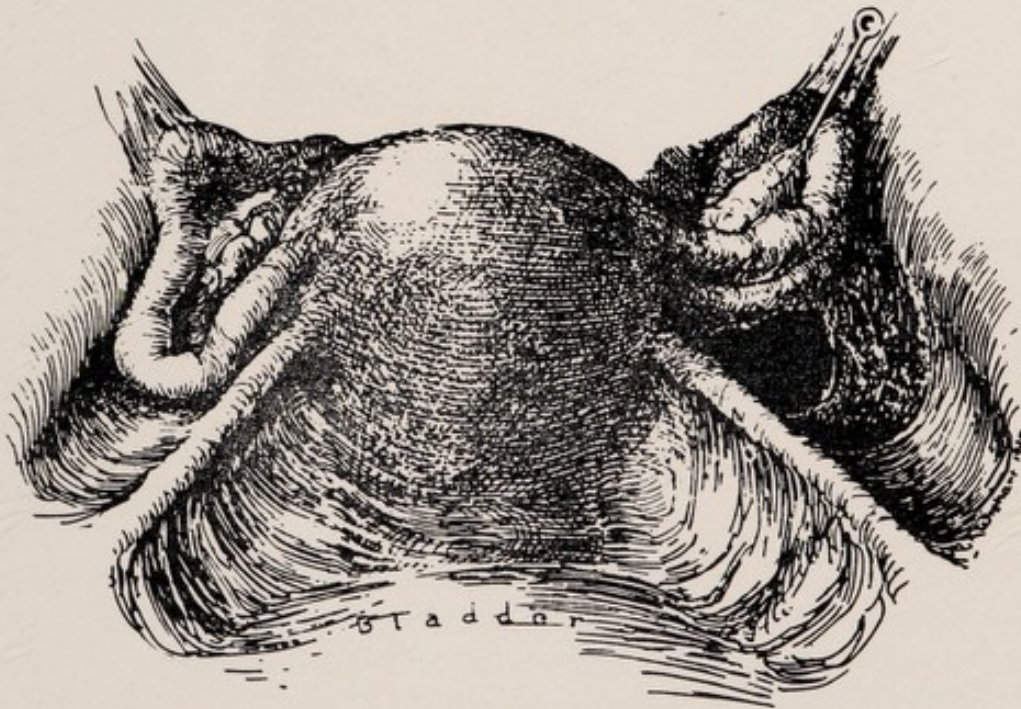
FIG. 31. Pericecal hernia with transposition of cecum. Sac contained practically all of small intestine which had entered through an opening beneath ileocecal valve into root of mesentery of caecum, extending upward and to right. (Copenhaver⁴)

be traced to abdominal injury. Openings in the omentum may be due to injury or congenital causes.

Herniation through abnormal openings resulting from operation are discussed under "Postoperative Obstructions," pp. 52-57.



A



B

FIG. 32. Strangulation of loop of small intestine in opening of left broad ligament. (Janes¹³)

A. Condition found at operation.

B. Opening in broad ligament after reduction of bowel.

In reference to the whole group of internal hernias, it may be said that herniation may occur without any symptoms of obstruction. Obstruction and strangulation usually follow,

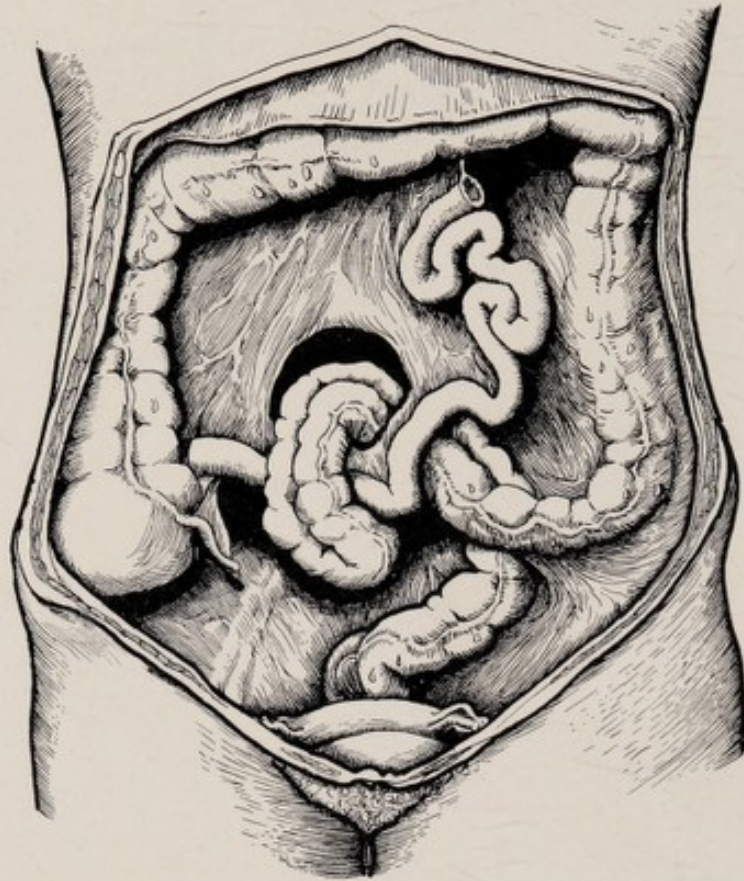


FIG. 33. Complicated type of internal hernia through mesentery, in which both sigmoid and ileum were involved. (Smith¹⁵)

A. Sigmoid after it has passed once through defect in mesentery near ileocecal junction. Sigmoid was twisted through 180°.

however, once herniation has occurred.

CASE XII. *Strangulated internal hernia.*

No. 282,569, M. G. H. Male, aged thirty. Past history not remarkable. Three days before admission the patient was taken with severe lower abdominal cramp-like pain and vomiting. There had been no bowel movement for the past three days and enemas had been given without result.

On physical examination the patient appeared critically ill; pulse 120. There was board-like rigidity of the abdomen. White blood count, 25,000.

Immediate operation was carried out. When the peritoneal cavity was opened, turbid fluid with a foul odor escaped. It was found that the small

intestine had herniated through an opening in the lower portion of the mesentery. Approximately 2 feet of the intestine were gangrenous; this loop was resected and the ends brought out through the abdominal wound.

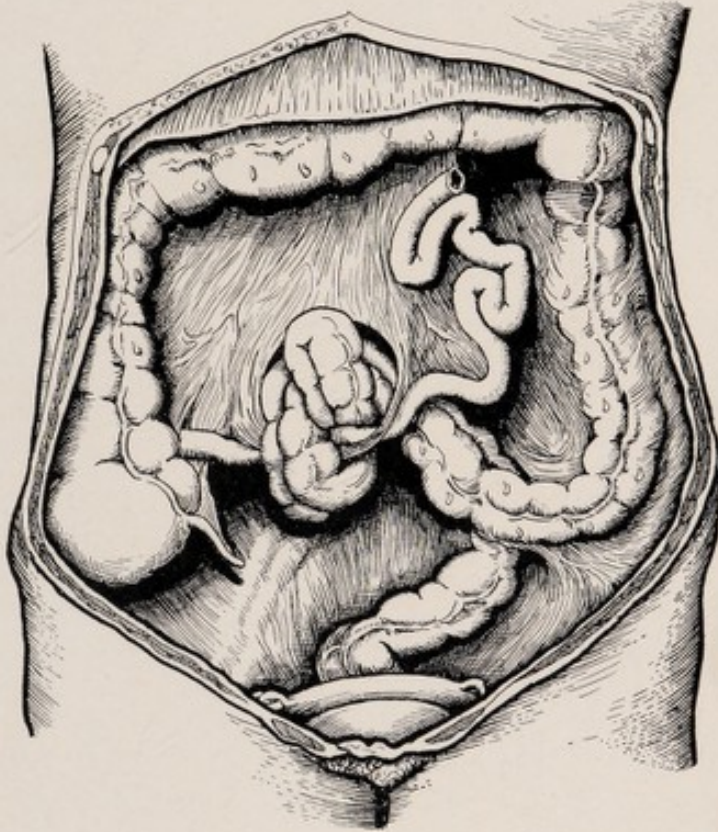


FIG. 33.

b. Sigmoid after it has passed through mesentery a second time, carrying with it adjacent ileum.

After a stormy convalescence the patient recovered. An end-to-end anastomosis was carried out at a later operation.

CONGENITAL ANOMALIES

As pointed out in Chapter II, congenital malformations may be fundamentally responsible for a large and varied group of obstructions. In this section are considered only those occurring in infancy where a gross malformation is directly responsible for the obstruction. The group is relatively small and the mortality high.

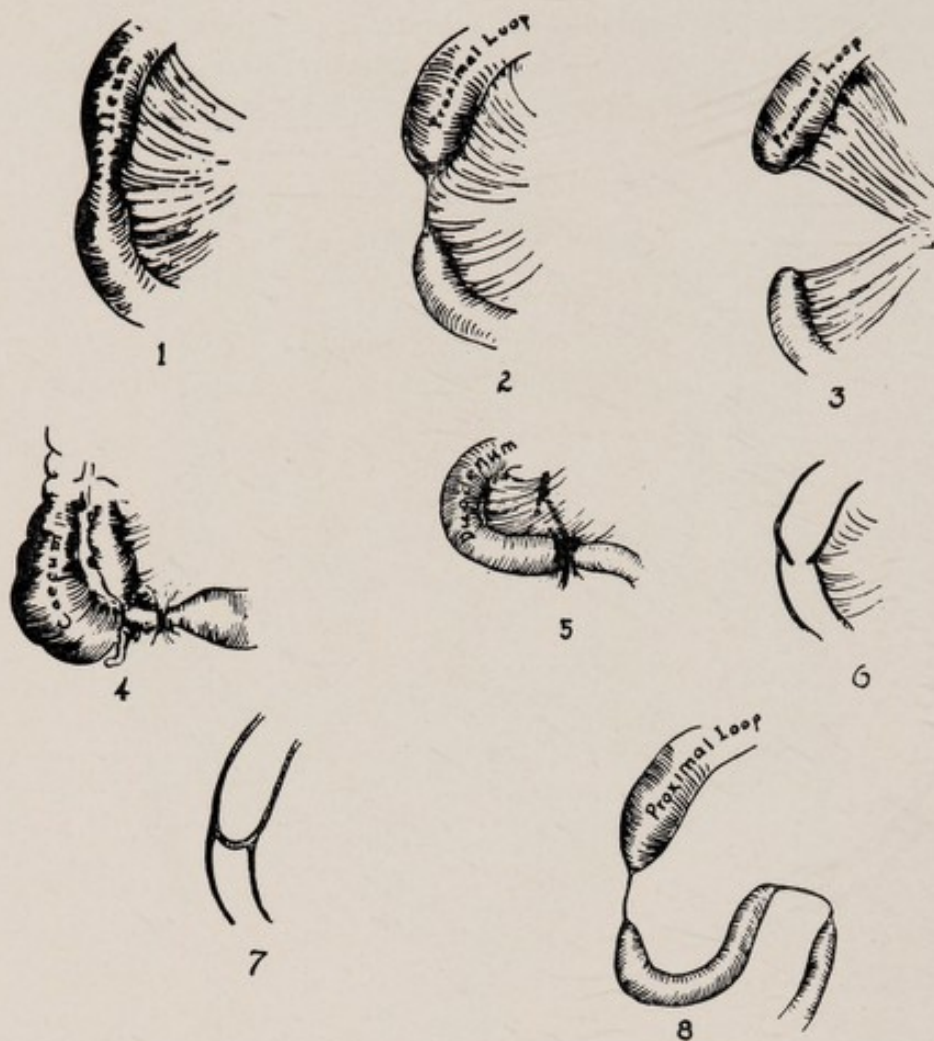


FIG. 34. Various types of congenital malformations described by Cole.¹⁶ (1) Stenosis with partial obstruction. (2) Occlusion with blind loop. (3) Blind loop with gap in mesentery. (4) Obstruction by fibrous band (found usually in third part of duodenum or terminal ileum). (5) Obstruction at ligament of Treitz; occasionally an anomalous superior mesenteric artery is chief factor in producing obstruction. (6) Cross-section of intestine showing perforated diaphragm; this occurs usually in duodenum or terminal ileum. (7) Cross-section of imperforate diaphragm. (8) Multiple occlusion of ileum or jejunum; this constitutes about 15 per cent of atresias of intestine. Clinically, dilatation of proximal loop with collapse of distal loop is usually even more pronounced than shown in this illustration.

Congenital anomalies causing obstruction are of two types: the atresias;¹⁶⁻²¹ and the congenital defects of rotation that are responsible for volvulus^{22,23}. According to Davis and Poynter,¹⁷ an atresia may occur at any point in the intestinal tract; they are encountered with especial frequency in the duodenum. (See Table VI.) In about 15 per cent of the cases there were multiple atresias. The developmental defects responsible for volvulus were discussed in Chapter v.

TABLE VI
CONGENITAL OCCLUSIONS: CASES OF ATRESIA

<i>Position of Atresia</i>	<i>Number of Cases</i>
Duodenum, above papilla.....	59
Duodenum, below papilla.....	75
Jejunum.....	60
Ileum and cecum.....	101
Colon.....	39
Multiple atresia.....	67

It will be noted that congenital occlusions are most frequent in the duodenum and in the region of the ileum and cecum. The atresias in this series were multiple in about 15 per cent of the cases. (Davis and Poynter.¹⁷)

MECKEL'S DIVERTICULUM

Meckel's diverticulum as an etiological factor in intussusceptions has already been discussed (Chap. VI). The diverticulum may also cause obstruction in other ways. Chief among these is the strangulation of a loop of gut, the diverticulum acting as a band^{24,25} (see Chap. III). This occurred in 101 instances in the series collected by Porter.²⁴ Rarely a long diverticulum having its distal end free may act as a noose and snare a loop of bowel. Occasionally a Meckel's diverticulum is responsible for a volvulus. In some cases the diverticulum may itself become twisted and in the twist involve the portion of bowel from which it originates.²⁶

The clinical picture presented by these cases of obstruction from a Meckel's diverticulum is that of strangulation from any cause. These cases are, however, especially serious because the diverticulum, due to its poor blood supply, is likely to undergo early necrosis and perforation, with resulting peritonitis.

GALLSTONES AND OTHER FOREIGN BODIES

GALLSTONES. Among the rarer types, obstruction from gallstones occupies a rather important place. Of the 335 cases of intestinal obstruction at the Massachusetts General Hospital in 1918-1927, a gallstone was responsible for the obstruction in 5 instances. Occasionally stones are found in the intestinal tract, which from their composition might be considered to be gallstones, but which are apparently formed in the intestinal tract, from the constituents of bile, rather than in the gall bladder. Hellström²⁷ has collected 7 cases, including 2 of his own, in which stones consisting almost entirely of colic acid were found in the gut. These stones were evidently not formed in the gall bladder, but in the intestine, for the gall bladder was normal, and in some of the stones there was a nucleus of vegetable matter that could have come only from the alimentary canal. Phillips²⁸ suggests that all "gallstones" removed from the intestinal canal should be analyzed with this possibility in mind.

The large gallstones (not infrequently measuring 4 or 5 cm., or more, in diameter) that are capable of producing obstruction, gain entrance to the intestinal tract by ulceration between the gall bladder and the intestine. After a stone has entered the gut it may be increased in size by the deposition of phosphates or carbonates from the intestinal tract; or, mineral matter (such as magnesia) that has been taken as a medicine, may be deposited around a gallstone and increase its size.²⁹

The gallstone may become impacted in the intestinal canal simply because of disproportion between the size of the stone and the diameter of the lumen. In certain cases the presence of the foreign body may incite a spasm in the intestinal musculature and this may be largely responsible for the onset of the acute obstruction.

The stone producing the obstruction may be found in the duodenum or upper jejunum; or, as more frequently happens, it

may pass to the last 2 or 3 inches of the ileum or to the ileocecal valve before becoming impacted. In 32 cases in which the position of the stone was given, reported by Leichtenstern,³⁰ the location was as follows:

Duodenum and jejunum.....	10 cases
Middle ileum.....	5 cases
Lower part of the ileum.....	17 cases
	—
	32

Rare cases have been reported in which the stone was located in the large intestine or cecum.

Changes in the bowel wall at the point where a stone becomes impacted may be slight; or, in certain cases, there may be ulceration or necrosis. The stone may at times produce a diverticulum.

Clinical Course. Obstruction by gallstones occurs much more frequently in the female than in the male: the 5 cases reported in the 1918-1927 Massachusetts General Hospital series all were in females; in Leichtenstern's³¹ series of 41 cases there were 32 females and 9 males. The disease is usually one of old age, although it occasionally occurs in middle life. It is usually possible to elicit a history of attacks of cholelithiasis or cholecystitis in the past. See also Diagnosis, p. 241.

The disease may run an acute course from the beginning; but often it is subacute for a number of days or even weeks before becoming acute. The count of the white blood cells in the 2 cases of the Massachusetts General Hospital series where it was recorded, was elevated (17,000, 24,000); the temperature is usually normal or only slightly elevated. The following are rather typical histories:

CASE XIII. *Obstruction by gallstone.*

No. 285,537, M. G. H. Female, aged sixty-seven. Patient gave a history of six days' upper abdominal pain and continuous severe vomiting; there had been only one bowel movement during this period.

Physical examination showed marked distention of the abdomen; there was some generalized tenderness; no muscle spasm.

The patient was operated upon the day after admission. At operation a gallstone about 4 cm. in diameter impacted in the lower ileum was found to be responsible for the obstruction. The stone was removed and an ileostomy done just above this point. The distention was not relieved after the operation and there was no drainage from the tube. The patient died one day postoperative.

CASE XIV. *Obstruction by gallstone.*

No. 248,412 M. G. H. Female, aged sixty-eight. Patient had had no serious illness and there had been no previous attacks of abdominal pain. On admission complained of generalized abdominal pain and vomiting of three days' duration; the pain had been more severe in the region of the epigastrium, and had been cramp-like in character. During the three day period there had been no bowel movements, and enemas gave negative results.

Physical examination was essentially negative except for a rather markedly distended abdomen. On auscultation, peristalsis was active.

The patient was operated upon at once. At operation there was a large quantity of free fluid in the abdomen, somewhat turbid in appearance. On exploration a large gallstone was found in the lower ileum. This was removed. There was a large mass of dense adhesions in the region of the gall bladder and duodenum; the omentum was adherent to this area. These adhesions were not disturbed.

After a somewhat stormy convalescence the patient recovered.

Comment. It is interesting that although there was "a mass of adhesions" in the region of the gall bladder and evidently the stone had entered the small intestine by perforations between the gall bladder and an adjacent coil of intestine, no history was obtained suggesting previous attacks of gallstone colic.

CASE XV. *Obstruction by gallstone.*

No. 223,351, M. G. H. Female, aged forty-six. Patient gave a past history of two attacks of abdominal pain, ten years and one year ago respectively; she had never been jaundiced. The present illness began two weeks before admission; the patient had had constant severe pain, cramp-like in character, in the upper half of the abdomen during this period. Patient had vomited ten to twelve times daily during the two weeks, the vomitus being "fecal" the morning of admission. Enemas daily for the two weeks had produced some results.

Physical examination showed the right half of the abdomen to be very tender; there was no tenderness in the left half. There was a moderate degree of muscle spasm over the whole of the right upper quadrant, and slight spasm in the right lower quadrant. Temperature 97°F.; pulse 104; white blood count 17,000.

At operation numerous adhesions were encountered; certain of these were freed, but no definite point of obstruction was located. The patient's condition became critical and the operation was terminated. The patient died fourteen hours later.

The autopsy showed acute intestinal obstruction from an occluding gallstone in the ileum. There was necrosis of the gall-bladder wall and the wall of the duodenum, the ulceration connecting these two structures.

Comment. Apparently in this case the early symptoms were due to acute cholecystitis. This attack terminated with the discharge of a gallstone into the intestinal tract where it caused obstruction.

VARIOUS OTHER FOREIGN BODIES. Other contents of the intestinal tract may at times cause obstruction. Such obstructions are rather infrequent and have been caused by a miscellaneous lot of material. Accumulations of food³²⁻³⁶ that have a high cellulose content may at times produce an obstruction. In 2 cases of the recent Massachusetts General Hospital series, indigestible vegetable matter had become impacted in the terminal ileum; in one instance this material was the husks of corn, in the second instance it was bran. Davis³⁵ has reported a similar case. Impacted vegetable matter occasionally produced obstruction in diabetic patients before the days of insulin, when enormous quantities of green vegetables formed the chief part of the diet. Leichtenstern³⁶ has described "sponge-like stones" that are formed chiefly of masses of indigestible vegetable fragments.

Hair-balls,^{37,38} pieces of wood and other bizarre collections of material have been reported as causing obstruction. These cases are usually met with in the insane or feeble-minded.

Obstruction from enteroliths or "intestinal stones" occurs in rare instances. The stones may be composed of various inorganic salts³⁹ as, for example, calcium or magnesium phosphate, calcium carbonate, etc. The mineral matter composing the enteroliths may have been taken as a medicine over a long period of time⁴⁰; and, as has already been pointed out, bile salts or pigments may enter into the formation of such stones.

A number of instances⁴¹⁻⁴³ have been reported where masses of *Ascaris lumbricoides* have produced acute intestinal

obstruction. In the obstructions produced by these parasites there may be two elements: the mechanical blockage of the lumen, and a spastic contraction of the gut due to a toxin



FIG. 35. Tuberculous stricture of ileum. (Treves⁴⁷)

given off by the worms. Herrick and Emery⁴⁴ were able to demonstrate that extracts made from *Ascaris* produced an increased tone and rate of contraction of intestinal musculature. An article by Morton and Archer⁴³ deals with the general surgical aspects of *Ascaris lumbricoides*, and describes a technique for demonstrating their presence by x-ray.

Inspissated "sebaceous material" causing obstruction in the new born has been reported.⁴⁵ Bullova and Brennan⁴⁶ report one case of obstruction by inspissated meconium.

When various mechanical devices, such as buttons, have been used for making intestinal anastomoses, these devices themselves have occasionally produced obstruction.

Occasionally a relatively small foreign body may become impacted in a benign or malignant stricture such as is shown in Figure 35.

Bismuth and barium taken by mouth for the purpose of x-ray examination are capable of converting a subacute into an acute obstruction. The danger of administering such materials for diagnostic purposes where there is a possibility of obstruction is recognized; but occasionally the seriousness of the situation is not appreciated and their use has been followed by an acute obstruction. This is a very grave type of obstruction: it may not be realized early that an acute obstruction has been produced; and it may be difficult, or impossible, to get rid of the barium even after the intestine has been opened: if peristalsis has stopped by the time the operation is undertaken the barium lies in the intestine as an inert mass and it may be impossible to get rid of it even though an enterostomy is carried out. Case XVI is of this sort:

CASE XVI. *Obstruction due to bismuth administered for x-ray examination.*

No. 241,088, M. G. H. Female, aged thirty-nine. Fifteen years before the patient's present admission she was operated upon for acute appendicitis. About six months before entrance she had an attack of what was evidently subacute obstruction. Since that time she had been greatly troubled with obstinate constipation, for which she frequently used cathartics. At times she had attacks of abdominal pain and occasionally vomited. There had been considerable loss of weight.

For the past two months the vomiting and pain had greatly increased in amount. Constipation had been very severe. She had vomited at least once a day for the past month.

In the course of her study on the medical wards she was given barium by mouth for a gastrointestinal examination. The day following this the vomiting increased in amount; the pain was about what she had experienced in the past. On the following day both vomiting and pain were worse and she had considerable distention. The vomitus was very profuse, brownish in color, but was said not to be foul. No result was obtained from enemas.

She was operated upon under gas-oxygen anesthesia with the addition of a small amount of ether. The small intestine was greatly dilated and contained a large amount of bismuth. An enterostomy was carried out in the lower ileum, but very little bismuth drained out through the catheter. The patient's condition did not warrant extensive exploration. Numerous adhesions were found and some were separated; but no definite point of obstruction was located. The patient died about five hours postoperative.

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CHAPTER VIII

VARIETIES OF OBSTRUCTION (Continued)

OBSTRUCTION BY NEOPLASMS

Obstructions from neoplasms form an important group, constituting about 17 per cent of all cases of obstruction, exclusive of those caused by external strangulated hernias (Fig. 2). The group assumes even more importance if the age incidence is considered, for it is the most common cause of obstruction (again exclusive of hernias) in patients past middle life. (Fig. 3.)

Tumors causing obstruction may be divided into two main groups: the first and much the more important comprises the primary tumors originating in the bowel; the second, the metastatic implantations.

Of the 32 cases of neoplasm reported in the 1918-1927 Massachusetts General Hospital series, 19 were due to primary carcinoma of the bowel, and 6 to metastatic carcinoma. There was one case of primary tumor of the mesentery, the exact type of which was not determined. As would be expected, in so small a series there were very few carcinomas of the small intestine: only 2 were of this type.

PRIMARY NEOPLASMS. Primary carcinoma arising from the large intestine is the most frequent type of primary neoplasm responsible for acute obstruction. Sarcomas¹ and carcinomas²⁻⁵ arising from the small intestine are rare tumors; they do not often produce complete obstruction.

While a growth situated anywhere from the cecum to the sigmoid may produce an acute obstruction, those arising from the sigmoid account for about half of these obstructions of the large intestine. Table VII gives Miller's⁶ analysis of 129 cases of carcinoma of the colon admitted to the Johns Hopkins Hospital from 1889 to 1919, showing the location of the tumor and the presence or absence of obstructive symptoms. In this series a carcinoma of the sigmoid was accountable

for an acute obstruction in 12 instances, the remaining 13 acute obstructions being about equally distributed among the other anatomical divisions of the large intestine. It will be noted that carcinoma of the cecum is a relatively common tumor (see also Erdmann and Clark⁷ and Rankin and Scholl⁸) but complete obstruction is rare. Miller's figures (Table VII) do not include carcinoma of the rectum. Carcinomas of the rectum occur about as frequently as carcinomas of all the rest of the large intestine taken together; but only rarely do they produce complete obstructions.

TABLE VII
SITE OF GROWTH IN THE THREE CLINICAL GROUPS

Site of Growth	Number of Cases	Acute Obstruction	Chronic Obstruction	Non-obstructive
Cecum.....	50 (38%)	2	24	24
Ascending Colon.....	5 (4%)	1	1	3
Hepatic Flexure.....	12 (9%)	2	6	4
Transverse Colon.....	7 (5%)	4	2	1
Splenic Flexure.....	8 (6%)	2	3	3
Descending Colon.....	6 (5%)	2	1	3
Sigmoid.....	40 (31%)	12	16	12
Hepatic Flexure combined with 2nd Growth in Ascending Colon.....	1 (0.7%)			
Total.....	129	25	53	50

Miller divides his cases of neoplasm of the large intestine into those showing acute obstruction, those showing chronic obstruction and those with no symptoms of obstruction. It will be noted that by far the largest number of obstructions occur in the sigmoid. Neoplasms involving the cecum, while often causing symptoms of chronic obstruction, rather rarely cause acute obstruction. (Miller⁶)

Etiology and Mechanism. Three important types of carcinoma arising in the large intestine may be distinguished.

The first type is adenoma destruens, a typical malignant tumor having a tendency to ulcerate early and extend relatively rapidly. It may, according to Ewing,⁹ be a bulky tumor in the cecum and is prone to produce wide superficial extensions in the bowel wall. It is often found in the sigmoid, and may produce acute obstruction relatively early.

The second type is the stenosing fibrocarcinoma. Here the lesion may be sharply circumscribed, and has a tendency to encircle the lumen, producing a tight, annular stricture due to the cicatricial contraction. These annular neoplasms may be present in any portion of the colon; and they may at times be multiple. Ewing suggests that this type often starts as an adenoma destruens and that relatively resistant tissue leads to early fibrosis and cicatrization. As would be expected from the stenosing tendency of this type, acute obstructions frequently result.

The third type of carcinoma in the large intestine is the gelatinous adenocarcinoma, which has a tendency to ulcerate extensively; degeneration and sloughing of the tumor in the lumen of the bowel keeps pace with the external growth of the neoplasm, so that, although a large bulky tumor may be present, complete obstruction does not usually follow. Codman¹⁰ points out that intestinal tumors of large size, usually sarcomas, rarely cause complete obstructions, for they are likely to slough out centrally as they grow peripherally.

An acute obstruction from neoplasm is usually produced, therefore, either by an adenoma destruens, or, more commonly, by a stenosing fibrocarcinoma. Blockage of the lumen generally comes about by a combination of the growth of the tumor mass and the process of cicatrization. Inflammatory swelling and edema usually play an important rôle, as is shown by the fact that if an obstruction is relieved by drainage of the bowel above, and the inflammatory process is allowed to quiet down, there will frequently be a resumption of the passage of fecal matter by rectum. Occasionally a mass of undigested food may, by becoming impacted in the stenosed lumen, convert a partial obstruction into a complete one. Not infrequently a carcinoma of the colon will lead to a chronic intussusception, the tumor mass in the lumen acting as a foreign body; the intussusception usually involves a rather small section of the bowel.

METASTATIC TUMORS. Neoplasms that are not primary in the bowel wall may produce acute intestinal obstruction. These tumors are for the most part carcinomas. They frequently arise from the pelvic organs in the female: the uterus and adnexa. Occasionally the primary growth may be situated in the other abdominal structures, such as the stomach, gall bladder, etc. Rarely, a sarcoma or other type of metastasizing neoplasm may be responsible for an obstruction.

Metastatic neoplasms may produce obstruction by involving the bowel in local extensions of the growth. It is fairly common to have the sigmoid involved in such a manner; thus, for example, a carcinoma arising from the ovary may extend around the lower portion of the sigmoid and completely occlude the lumen. On the other hand, a metastasis may occur anywhere in the peritoneal cavity, and by involvement of some neighboring loop of bowel produce obstruction; and at times a carcinoma that has become implanted in a mesenteric lymph node may spread upwards in the lymphatics of the mesentery and encircle the bowel. Occasionally a lymphoblastoma involving the mesenteric lymph glands may, likewise, produce an acute obstruction. Among these rarer types of tumors must be mentioned endometrial implants,^{11, 12} which may infiltrate the intestinal wall and encroach upon the lumen.

PATHOLOGY OF THE BOWEL ABOVE THE OBSTRUCTION. The bowel above the neoplasm is dilated. If a chronic obstruction has existed for some time before the acute attack, the bowel wall may be thickened, hypertrophied and edematous. Some of the greatest degrees of abdominal distention seen in cases of acute obstruction are encountered in obstructions of the left side of the colon; and distention is apt to be a particularly striking feature in cases where an acute obstruction supervenes on a partial obstruction which has been present for some time.

Interference with the Circulation. The point has been made a number of times that interference with the circulation of the

bowel is of two types: one in which the mesenteric vessels are occluded; and another in which the blood supply of the smaller arterials, venials and capillaries of the wall itself is impaired.

Capillary Interference. Interference with the capillary circulation comes about as a result of distention of the bowel wall and is frequently encountered in obstructions of the large intestine by neoplasm. Gaseous fermentation is more active in the colon than in the small intestine, and interference with the circulation of the bowel from the resulting distention is of relatively frequent occurrence. Since the cecum is the most distensible portion of the large intestine, damage to the capillary circulation is most frequently found in this portion of the canal. As would be expected, it is the anterior surface of the cecum (that is, the part farthest away from the entrance of the blood supply) that suffers first. The changes may vary from dusky cyanosis to actual necrosis and gangrene, perforation of the cecum and general peritonitis being the not infrequent termination of an unrelieved obstruction of the colon.

Perforation of the bowel with general peritonitis is sometimes the result not of the effects of distention but of an ulcerative, gangrenous process in the obstructing neoplasm itself; this, however, is not of very frequent occurrence.

Mesenteric Interference. Although the large intestine is particularly likely to suffer from distention, which causes interference with the circulation in its wall, it is relatively immune to interference with its mesenteric blood supply. In the first place, most of the obstructions by neoplasms of the large intestine are the result of a stenosing process or of occlusion of the lumen; so that the mesentery would naturally not be involved in the process. In the second place, the only portion of the colon that ordinarily has a definite mobile mesentery is the sigmoid; the vessels to the remainder of the large intestine are better protected than are the vessels in the mobile mesentery of the small intestine. For these two reasons, obstructions of the large intestine, in general, even those caused

by bands and adhesions, rarely interfere with the mesenteric blood supply. Almost the only exceptions to this are in the case of volvulus of the sigmoid or cecum, and those in which a portion of the large intestine is strangulated in a hernial sac.

Rarely neoplasms arising in the mesentery, usually lymphosarcomas, may involve the blood vessels of that structure and produce infarction of the adjacent segment of bowel; there was one example of this condition among the tumors in the Massachusetts General Hospital series.

Occasionally, also, a carcinoma spreading along the mesenteric lymphatics may surround and occlude a large vessel and thus likewise produce an infarction of a segment of large intestine. Case XVII illustrates this condition:

CASE XVII. Obstruction from carcinoma of the sigmoid complicated by metastatic occlusion of the inferior mesenteric artery.

No. 251,331, M. G. H. Female, aged fifty-five. The patient had been losing strength and weight for several months and had had mild intestinal disturbances and increasing constipation for six months. For four days before admission there had been no bowel movements, and the patient had been vomiting; there had also been increasing abdominal distention. It was said that one week before the attack she had passed a tarry stool.

The patient was operated on under local anesthesia. The colon was found greatly distended, and a colostomy was performed. The patient died eighteen hours after operation.

Autopsy showed a carcinoma of the sigmoid, producing obstruction at that point. There was also an extension of the neoplasm around the inferior mesenteric artery, completely occluding it. This had resulted in hemorrhagic infarction of the left side of the colon, with necrosis and perforation.

CLINICAL COURSE OF THE DISEASE. Subacute Stage. Since obstruction from neoplasm depends upon the growth of a tumor and the gradual stenosis of the lumen of the bowel, the process is ordinarily slow and progressive, and there is usually a partial or chronic obstruction for some time before the occlusion of the lumen becomes complete. The symptoms shown during this stage of the disease depend in large part upon the completeness of the obstruction: they may vary

from the mildest symptoms, only to be elicited by the most careful questioning, to symptoms that obviously suggest obstruction.

The symptoms of chronic obstruction are usually those of an obstinate and often progressive constipation; but on the other hand the presenting feature may be an obstinate diarrhea. The condition described by the patient as "diarrhea" often consists of the frequent passing of a small amount of thin watery movement, not seldom mixed with mucus or blood; there may be tenesmus.^{13,14} Not infrequently the patient is conscious of increased peristaltic activity, with borborygmus; if the obstruction is located in the colon this is usually associated with pain across the lower abdomen. There may be attacks which the patient describes as "indigestion" or "bilious attacks," consisting of a sense of heaviness in the abdomen, often in the epigastrium, with an aversion to food and occasional attacks of nausea and vomiting. Not infrequently the patient complains of a fullness of the abdomen, often described as "bloating."

The symptoms of chronic obstruction may at any time be converted into those of acute obstruction. One may be able to recognize a definite time at which there was a marked exacerbation of the symptoms; or the condition may pass into that of an acute obstruction in a very insidious manner. About two-thirds of the acute obstructions from neoplasms, whether from a primary or a metastatic tumor, have a history of definite symptoms of subacute or incomplete obstruction antedating the complete obstruction by days or weeks or in some cases by several months. In the recent Massachusetts General Hospital series the period of incomplete obstruction was from ten days to several months, the average duration being just short of nine weeks.

Acute Stage. At the time of the patient's entrance to the hospital the obstruction may be complete and acute; but in the greater number of cases it is not, and it is often difficult to say whether the case should be classified as complete or

incomplete obstruction. In the foregoing series only those cases were called "complete" in which the symptoms and physical signs were so marked that immediate surgical intervention was indicated. In Miller's review⁶ (Table VII) he divides the patients with carcinoma of the large intestine into three groups with reference to obstruction: those showing acute obstruction, those with chronic obstruction, and those with non-obstructive lesions; and states that in every 10 patients with carcinoma of the large bowel presenting themselves for treatment, 4 will show no features of obstruction, 4 will give a definite history of chronic obstruction, and 2 will either be actually obstructed or will give a history of an acute attack from which they have recovered.

Signs and Symptoms. The symptoms of acute obstruction from neoplasm are in general those of acute obstruction from any cause where strangulation is not present: constipation, pain, distention and vomiting. Since most of these obstructions are located in the large intestine the symptoms have a tendency to be less fulminating than in types of obstruction where the small intestine is involved,¹⁵ and considerable time is likely to elapse between their onset and the time of operation. In the recent Massachusetts General Hospital series the average time between the onset of acute symptoms and the operation was five days. In some cases the obstructive symptoms were very severe over a period of ten days to two weeks; the obstruction was probably not complete over that period, but it was impossible to say at what time the blockage became absolute.

Since strangulation is rarely present in obstructions from neoplasm, the pain may not be particularly severe; it is, however, almost constantly present to some degree. In the early stages the pain is likely to be described as cramp-like or colicky; in the later stages it may lose this characteristic and become constant, although it remains colicky longer in obstructions of the large intestine than it does in obstructions of the small intestine. It is usually localized across the lower abdomen, although occasionally at the onset it is referred to the region

of the epigastrium. In some instances acute discomfort from distention is the most outstanding complaint.

Vomiting comes on early with the onset of the acute obstruction, but is not likely, at least in the early stages, to be so profuse or frequent as in obstructions of the small intestine; in the later stages, after the obstruction is well established, and as the small intestine becomes more and more involved in the obstructive process, the vomiting may be profuse and of the typical "fecal" character.

Distention is likely to be a very marked feature in obstructions of the large intestine. In the early stages, visible peristalsis of the large intestine above the point of obstruction may be seen at times. In attempting to say whether an obstruction is complete or not, the question of whether there is any passage of flatus either voluntarily or on the administration of an enema is an important criterion. There is usually little or no systemic reaction, the temperature and white blood cell count being generally normal.

See also Diagnosis, p. 242, and Treatment, p. 301.

Age and Sex. Since carcinoma of the colon occurs almost twice as often in the male as in the female, we naturally find a similar ratio in intestinal obstruction by carcinoma.

The ages of the patients in a recent series are shown in diagrammatic form in Figure 3. Following the incidence of carcinoma in general, it will be seen that obstruction from neoplasm is a disease of late middle life.

Two typical histories follow:

CASE XVIII. *Obstruction by carcinoma of the sigmoid.*

No. 228,768, M. G. H. Male, aged fifty-seven. Patient had two weeks of gradually increasing constipation, requiring large doses of cathartic to obtain a movement. For the past seven days there have been no bowel movements. During this time the patient has vomited practically everything taken by mouth and enemas have produced no results, either feces or gas. During this time there has been indefinite, generalized abdominal pain, cramp-like in character.

On physical examination, the temperature was normal and the white count 8000. The examination was essentially negative except for the

abdomen, which was distended, with visible peristalsis seen to the right of the umbilicus. The left upper quadrant appeared more distended than the remainder of the abdomen. There was a question of an indefinite mass in the left lower quadrant.

The patient was operated upon soon after admission and a carcinoma of the sigmoid was located. A colostomy was done above the growth. The patient recovered uneventfully from this operation. Two weeks later the tumor was resected. The patient was discharged relieved.

Comment: The patient's age, together with the rather mild course of the disease, would suggest the diagnosis of obstruction by carcinoma of the large intestine.

CASE XIX. *Obstruction by carcinoma of the sigmoid.*

No. 248,046, M. G. H. Male, aged sixty. For the past few months the patient has had obstinate constipation relieved by cathartics. He has suffered from general malaise, loss of appetite, weakness and gradual loss of weight. Eleven days before admission the abdominal discomfort and distention increased. Enemas gave unsatisfactory results. Four days before admission he began to vomit foul-smelling material at frequent intervals. The distention and abdominal discomfort were extreme.

Physical examination showed an emaciated man with evidence of considerable dehydration. The abdomen was distended, tense and tympanic. No mass was felt; there was no visible peristalsis. Temperature and white blood count were normal.

The abdomen was explored at once, under novocaine and ether anesthesia. The whole colon was greatly distended. A mass could be felt in the region of the sigmoid. A section of the sigmoid above the growth was brought out as a colostomy. The patient did not rally from the operation and died the next day. Autopsy showed general peritonitis and hemorrhagic edema of the lungs, in addition to the carcinoma of the sigmoid.

Comment: This case represents an end stage of obstruction by carcinoma of the sigmoid. The patient had had complete obstruction for several days before operation.

The operation of choice for obstructions of the left side of the colon by neoplasm is a cecostomy.

PARTIAL OR SUBACUTE OBSTRUCTIONS. During the years from 1918 to 1927 inclusive, there were at the Massachusetts General Hospital 24 cases in which the patients were operated upon for subacute obstructions due to tumor. There were 12 other patients in whom the obstructive symptoms were a rather marked feature of the disease but not severe enough

to warrant their consideration even in the group of subacute obstructions. Of the 24 cases with subacute obstruction, the blockage in 19 instances was due to a primary carcinoma of the large intestine; in 5 instances to metastatic involvement of the small intestine. The average duration of symptoms in these patients before their entrance to the hospital was six weeks.

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CHAPTER IX

VARIETIES OF OBSTRUCTION (Continued)

OBSTRUCTION BY STRANGULATED EXTERNAL HERNIA

Strangulation of a loop of bowel in an external hernia is the most frequent single cause of acute intestinal obstruction, accounting as it does for almost half the total number of cases.

DEFINITION. Strangulated hernias must not be confused with incarcerated hernias. The latter term describes the condition existing where a loop of bowel has found its way into a hernial sac, and, due to adhesions or other cause, cannot be returned to the abdominal cavity; such a condition may be tolerated for years without any serious symptoms. The essential feature of the condition known as "strangulated hernia" is interference with the circulation to a loop of bowel contained in a hernial sac; in the great majority of cases there is complete obstruction of the lumen of the bowel at the points where the ends enter and leave the sac. When a strangulation comes about, acute symptoms appear at once and are usually progressive in character. In going over any large series of so-called strangulated hernias, there are certain borderline cases in which it is difficult to decide whether the case should be classified as a strangulation or as an incarceration; the patients not infrequently have presented themselves following the onset of the incarceration of a loop in a hernial sac, and it is hard to say whether or not the element of strangulation is present. Clinically, the criterion for differentiation is the severity of the symptoms (pain, tenderness over the sac, vomiting, etc). At operation the major point for differentiation would be the presence or absence of changes indicating interference with the circulation to the involved loop.

MECHANISM OF STRANGULATION IN EXTERNAL HERNIAS. In an external hernia the point of constriction is usually at the hernial ring, although occasionally it may be at the tight

neck of the sac. This is important; for occasionally the hernial sac with its contents may be returned to the abdominal cavity by taxis (without operation), the so-called "reduction en bloc," without recognition of the fact that there is bowel strangulated within the sac. A loop may occasionally be strangulated by bands and adhesions within a hernial sac; or there may be rotation of the coil within the sac. A hernia of considerable size may exist for a number of years with no symptoms of incarceration or strangulation. This depends in large part upon the size of the hernial ring; the smaller rings, as is well known, having a greater tendency to produce strangulation than the hernias with large openings. A loop of bowel may be forced through a tight ring and immediately become strangulated; or after having been incarcerated in a hernial sac for a number of years may suddenly, without apparent cause, become strangulated. Strangulation may follow immediately upon some action that increases intra-abdominal pressure, such as straining, lifting weights, coughing, etc.

Warren and Gould¹ summarize the various older theories of the mechanism of strangulation in hernias as follows: the strangulation occurs because of the elastic compression of the ring; compression of the lower end of the strangulated loop by the distended upper portion; angulation of the distal end of the loop; valvular folding of the mucous membranes; torsion of the loop; interposition of the mesentery; or fecal impaction. None of these alone appears to accord a satisfactory explanation of the strangulation in all cases: an adequate theory must explain both the obstruction of the bowel and the interference with the circulation.

Warren and Gould¹ consider that the inflammation of an incarcerated loop is a fundamental factor and that venous congestion is the first step in the inflammatory process. This is doubtless true; but the fundamental causes responsible for the onset of the inflammation are not always clear. It appears likely (see mechanism of strangulation, p. 38) that distention of

the incarcerated loop may be an important factor in bringing about the strangulation.

RETROGRADE STRANGULATION. Rost² mentions a rare form, the so-called "retrograde" strangulation, in which two loops of bowel enter the hernial sac, while a third loop, which connects the two incarcerated loops but has not itself entered the hernial sac, becomes gangrenous. This is attributed to kinking of the mesentery of the third loop.

Closely allied to retrograde strangulations are those cases in which a volvulus complicates the hernia. Miller³ divides these cases into six groups: (1) volvulus of a portion or all of the herniated bowel; (2) volvulus of the small bowel, one loop of which becomes herniated; (3) volvulus of a distal oral loop above an actual strangulated hernia; (4) volvulus, generally of the large bowel, distal to a simulated strangulated hernia; (5) volvulus of the herniated bowel immediately after its reduction; and (6) volvulus of the herniated bowel long after its reduction. The diagnosis of this complication before operation is difficult; occasionally an abdominal mass may be palpated.

RICHTER'S HERNIA. There is an interesting type of hernia known as Richter's hernia (Fig. 36),⁴ in which a portion of the bowel wall only is constricted in the hernial opening without obstruction of the lumen. This portion may actually become gangrenous. Richter's hernia occurred in 2.2 per cent of the 1487 cases of strangulated hernia reported by Frankau.⁵

LITTRÉ'S HERNIA. In this rare type of hernia a Meckel's diverticulum is present in the sac. Sweet⁶ has recently collected all the cases reported in the literature in which the diverticulum was incarcerated or strangulated in the hernial sac.

An acute intussusception has also been reported⁷ as forming part of the contents of a hernial sac.

INCIDENCE OF STRANGULATION AMONG DIFFERENT TYPES OF HERNIA. The incidence of the different types of hernia and the relative frequency of strangulation in these types are shown in Table VIII. It will be noted that inguinal hernia is by far the

most common form; strangulation, however, is relatively more frequent in the small groups of femoral and umbilical hernias. The distribution of 147 cases of strangulated hernias (Massachusetts General Hospital 1918-1927 series)¹⁰ among the different types is shown in Table IX. The large proportion of strangulated inguinal hernias is due to the fact that this type of hernia is so common.

There is one rare and usually fatal form of hernia not represented in Table IX, namely, the obdurator hernia. Gibson¹¹ reported 7 cases (collected from the literature) of strangulated hernias of this type, among which there were 6 deaths.

TABLE VIII
INCIDENCE OF DIFFERENT TYPES OF HERNIA AND RELATIVE FREQUENCY OF STRANGULATION
IN THESE TYPES

	Total Cases	Inguinal		Femoral		Umbilical		Incisional Postoperative Ventral	
		Num- ber of Cases	Stran- gulated	Num- ber of Cases	Stran- gulated	Num- ber of Cases	Stran- gulated	Num- ber of Cases	Stran- gulated
Mt. Sinai Hosp. New York.	4,139	3,208 (77%)	4%	294 (7%)	32%	167 (4%)	15%	377 (9%)	3%
Beller & Colp ⁸ 1925									
N. Y. Hospital	2,587	2,300 (89%)	1.8%	97 (3.7%)	25.7%	29 (1.1%)	17.2%	145 (5.6%)	5.5%
Erdman ⁹ 1926									

It will be noted that inguinal hernia is by far the most common form; strangulation, however, is relatively more frequent in the small groups of femoral and umbilical hernias. Beller and Colp⁸ and Erdman.⁹

The portion of bowel strangulated is illustrated by Table X. It will be noted that the small intestine is the part involved in the great majority of cases.

PATHOLOGY. The changes taking place in the bowel wall following strangulation are described later (p. 143). They may vary from simple congestion to complete infarction and gangrene; thrombosis of the veins to the involved segment

may occur. The greatest pressure comes at the opening of the hernial sac, and the bowel may show evidences of gangrene at this part while the remainder of the loop in the sac shows only slight changes. Beller and Colp⁸ divided the pathological process into the stages of congestion, inflammation and gangrene.

TABLE IX
STRANGULATED EXTERNAL HERNIAS

<i>Type of Hernia</i>	<i>Number of Cases</i>
Inguinal.....	84 (56%)
Femoral.....	34 (21%)
Umbilical.....	18 (18%)
Ventral.....	9 (6%)
Epigastric.....	2 (2%)
Total.....	147

It will be noted that about half the total number of strangulations occurred in inguinal hernias. While, as shown in Table VIII, strangulation is a relatively infrequent complication among these hernias as compared with the femoral and umbilical hernias, the total number of inguinal hernias is so large that the actual number of strangulations is greater in this group.

TABLE X
PORTION OF BOWEL OBSTRUCTED

<i>Portion of Bowel Strangulated</i>	<i>Number of Cases</i>
Small intestine.....	127 (86%)
Small and large intestine.....	13 (9%)
Large intestine.....	7 (5%)
Total.....	147

It will be noted that the small intestine is the part involved in the great majority of cases.

The fluid present in the hernial sac will, of course, vary according to the condition of the bowel: in the early stages of congestion it may be a clear, yellow fluid; where the bowel shows more serious involvement, it may be blood-stained; and if the bowel has already become permeable to organisms, it may be foul-smelling and show flakes of fibrin.

The frequency with which the bowel changes have gone on to actual necrosis in the different types of hernias is illustrated by Table XI. It will be noted that out of the 147 hernias,

necrosis is recorded in 19 instances, or about 13 per cent. This is an end stage of the process of strangulation and, of course, represents delay in bringing the patients to operation.

TABLE XI
CASES OF EXTERNAL STRANGULATED HERNIA SHOWING NECROSIS OF BOWEL

Type of Hernia	Portion of Bowel Obstructed	Duration of Symptoms	Type of Operation	Anesthetic	Result
Femoral 8	Small intestine	5 days	Resection; ends brought out.....	Spinal procaine	R*
		7 days	Resection; ends brought out.....	Ether	R
		3-4 days	Resection; ends brought out.....	Novocaine	D*
		30 hours	Resection; end to end anastomosis....	Ether	D
		7 days	End-to-end anastomosis.....	Ether	D
		2 days	End-to-end anastomosis.....	Apothesine spinal	R
		4 days	Inversion of necrotic area and enterostomy 8 inches above repair.....	Novocaine	D
	8 days	Small necrotic area perforated; catheter placed in opening.....	Novocaine	R	
Inguinal 5	Small int.	16 hours	End-to-end anastomosis.....	Spinal novol	R
	Small int.	? ?	Resection; lateral anastomosis.....	Spinal by apothesine, sup. by ether	D
		3 days	Inversion.....	Novocaine	R
	1 day	Gangrenous loop brought outside of peritoneum but not outside of muscle	Novocaine	R	
Trans. colon or sigmoid	4 days	Loop brought out; Mixer tube.....	Novocaine	D	
Umbilical 5	Small int.	4 hours	Resection; ends brought out.....	Ether	D
	Small int. and trans. colon	24 hours	Resection; lateral anastomosis.....	Sp. novocaine	D
		5 days	Loop of bowel brought out; Mixer tube.....	Ethylene	D
		4 days	Resection of small intestine; anastomosis.....	Ether	D
	Trans. colon	15 hours	Ends brought out.....	Ether	D
Epigastric 1	Small int.	2 days	Resection; lateral anastomosis.....	Novocaine	R

* R: recovered. D: died.

Table XI gives certain data in relation to cases of external strangulated hernia with necrosis of the bowel; duration of symptoms, type of operation performed, result, etc. (McIver¹⁰)

The strangulated loop, if not relieved, may perforate and set up an abscess cavity; or it may go on to complete gangrene, with separation of the slough at the neck of the sac. The whole hernial mass may then rupture externally and a spontaneous cure result, with the formation of a fecal fistula; the older

textbooks of surgery record many examples of this end process; but usually before this occurs the patient dies from a peritonitis or from the obstruction of the lumen of the bowel. (See also, Travers.¹²)

TOXEMIA IN STRANGULATED HERNIA. There are really two elements responsible for the toxemia in strangulated external hernia. The first is the toxic absorption which takes place from the strangulated loop. At times, due to the constriction at the hernial opening or at the neck of the sac, the strangulated loop is completely isolated from the general cavity, with the result that absorption of toxic products from the gangrenous loop is minimized; occasionally a spontaneous cure is effected, as just mentioned.

The second element is the simple obstruction of the portion of bowel proximal (oral) to the strangulated loop, a factor of considerable importance in the pathological and clinical picture.

The fact that strangulated external hernia presents two distinct elements was essentially recognized as far back as 1812; and Travers'*¹² interesting experiments and monograph on the cause of death in strangulated hernia is a valuable contribution. Briefly, he contended that the cause of death was not the strangulation of the bowel, because the bowel was outside the peritoneal cavity; but that death was due to the obstruction of the intestine above the strangulation.

CLINICAL PICTURE AND COURSE OF THE DISEASE. *Sex.* The male is subject to strangulated hernia about twice as frequently as the female. The umbilical and femoral types of

* Travers writes: "I have repeatedly formed herniae in animals, imitating the secondary species of strangulation by drawing a loop of intestine through a small muscular orifice, and the primary by making a tight ligature upon the protruded gut. The former produces inflammation of the strictured bowel, and if the obstruction is complete, the animal dies of general peritoneal inflammation. The latter rapidly induces sphacelus (gangrene), in which state the gut bursts, and speedily gives issue to the contents of the upper bowels. The animal is immediately relieved by the discharge; an artificial anus is formed; and though the experiment is ultimately fatal, the peritoneal surface invariably remains healthy." And again: "The local injury would scarcely affect the system if it were not for the derangement of the intestinal function. It will be found that the symptoms are in all cases proportioned in severity to the degree of obstruction." (Travers, 1812.) See also page 383.

hernia, however, become strangulated in females about three times as often as in males.

Age. Figure 3 illustrates the ages at which strangulated external hernias occur. It will be noted that while all ages are represented, the period from forty to sixty years is particularly prone to this disease.

Signs and Symptoms. The clinical picture presented by a strangulated hernia is for the most part very characteristic and uniform. The severity of the symptoms usually depends upon the completeness of the strangulation. Pain is practically always present and is likely to come on suddenly with the onset of the strangulation; it is usually severe and at times agonizing. At the onset the pain may be localized in the epigastrium or across the lower abdomen, and is usually described as cramp-like in character. As the obstruction continues, the pain and tenderness, due to increasing inflammation and infection, are more completely localized over the hernial sac.

Vomiting may begin with the onset of the pain and continue with increasing frequency throughout the course of the disease. Just as in other types of obstruction, the vomitus is at first merely the contents of the stomach or upper small intestine, and as the disease continues may become "fecal," consisting of the foul fluid that collects above the point of obstruction.

There may be complete cessation of bowel movements from the time of strangulation, or the lower segment of bowel below the strangulation may be emptied. In types of hernia where a section of the wall of the bowel only is strangulated, without obstruction of the lumen (Richter's hernia), bowel movements may continue throughout the course of the disease; under these conditions diarrhea is not infrequent.

On physical examination there is usually an obvious hernial sac which is tender and irreducible. If the hernia has persisted for some time there may be signs of inflammation, such as redness and edema. The onset of the strangulation is likely to have been sudden; it not infrequently follows strain-

ing of some sort that increases the intra-abdominal pressure; but at times no such history is obtained. In cases where the strangulation is complete, the pain may be severe in character from the onset.

Four case histories illustrating different types of strangulated external hernia follow:

CASE XX. Obstruction by strangulated right inguinal hernia.

No. 247849, M. G. H. Male, aged twenty-six. The patient gave a history of having had a right inguinal hernia, always easily reducible, for ten years. For the past four years he had worn a truss. Four hours before admission he was taken with a sharp pain in the right inguinal region; the pain has been constant and there has been no movement of the bowel since its onset. Patient noticed that the hernia was down in the sac, and he has not been able to reduce it.

Examination showed an irreducible, tender mass in the right inguinal region, having the typical characteristics of a strangulated hernia.

The patient was operated on under novocaine and ether anesthesia. When the sac was opened, the gut was found to be blue-black in color, but the circulation improved under observation. The intestine was considered definitely viable and was returned to the abdomen; the hernia was repaired. Convalescence was uneventful.

Comment: This is a rather typical case of strangulated hernia. The diagnosis is easy and if operation is promptly performed the result is usually satisfactory. Most of these cases can be handled under novocaine anesthesia.

While the diagnosis of a strangulated hernia is usually obvious both to the patient and to the physician, there are times when a small hernial mass may be overlooked. This is particularly true in regard to a small knuckle of bowel in a femoral hernia. The two following histories illustrate this type of case:

CASE XXI. No. 266491 M. G. H. Female, aged 73. The patient had had pain for four or five days before admission. No coherent history could be obtained. On a previous admission to the medical wards a diagnosis of arteriosclerosis and cholelithiasis had been made but no operation advised. On the present admission a diagnosis of intestinal obstruction was made; no hernia was found on physical examination.

Laparotomy showed a dilated intestine which when followed down led to a strangulated loop in the left femoral canal. The bowel was reduced and found to be viable. The patient also had a gallstone impacted in the

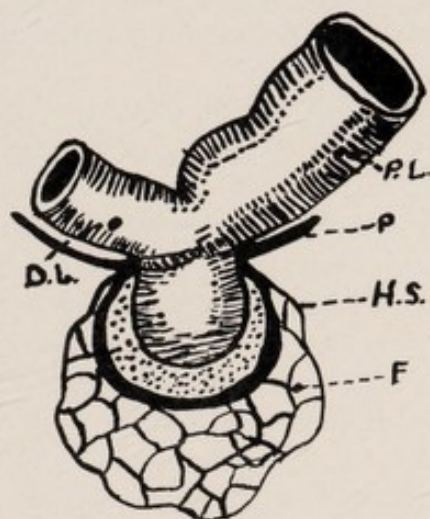


FIG. 36. A Richter's hernia shown in diagrammatic form. It will be noted that only a portion of the bowel wall is contained in the hernial sac and that the lumen of the intestine is not completely obstructed. (Walton.⁴) P.L., Proximal dilated loop of gut. P., Peritoneum. H.S., Hernial sac. F., Wall of sac loaded with fat. D.L., Distal contracted loop of gut.

cystic duct. After a stormy convalescence the patient was discharged, relieved.

Comment: The cholelithiasis undoubtedly obscured the picture of obstruction, and the small femoral hernia was overlooked before operation.

CASE XXII. *Obstruction by Richter's hernia.*

No. 290977, M. G. H. Male, aged fifty-nine. The patient was taken with severe pain in the right lower quadrant of the abdomen eight days before admission; there was vomiting at that time. The pain and vomiting ceased within forty-eight hours of their onset; but constipation persisted, little or no results being obtained by enemas. The past history was negative, except for the fact that the patient had been jaundiced since childhood. Twenty years before admission he had received treatment for syphilis.

Physical examination of the abdomen showed a large, firm mass occupying most of the left side of the abdomen; the mass descended on inspiration. This mass had the characteristics of an enlarged spleen. During observation in the hospital the signs of obstruction increased and vomiting was profuse.

An exploratory laparotomy was carried out under novocaine anesthesia. In addition to the enlarged spleen, dilated and collapsed bowel was found. On following down one of the collapsed coils it was found that it entered the

right femoral ring. The hernial sac was then exposed in the right groin and it was found that a portion of the circumference of the bowel was constricted at the hernial ring, Richter's type of hernia (see Fig. 36). The strangulated portion of the bowel was necrotic, and when the sac was opened there was an escape of fecal material. A catheter was inserted into the bowel and held in place by purse-string sutures.

The patient recovered after a somewhat stormy convalescence. At the time of discharge from the hospital it was understood that he was to return later for a splenectomy.

Comment: The foregoing case represented a difficult diagnostic problem. The patient had chronic jaundice with splenomegaly, and the fact that he also had a strangulated Richter's hernia was entirely overlooked before operation. This case illustrates the importance of careful examination of all the usual hernial openings in any case showing symptoms of obstruction.

The accidental escape of contents from a necrotic bowel, such as occurred in this case, is extremely serious and very frequently leads to a fatal outcome from a virulent peritonitis. In another case of Richter's hernia, where this accident occurred, the patient died in a few days, the autopsy showing widespread gas bacillus infection, with emphysematous necrosis of the liver and spleen and extensive subcutaneous emphysema.

Occasionally a loop of bowel that has been damaged by interference with the circulation may be sufficiently viable so that no gangrene or perforation occurs, and yet so much scarring takes place that a stricture later results.¹³ The following case illustrates this point:

CASE XXIII. Strangulated inguinal hernia with subsequent stricture of the involved segment of bowel.

No. 222859, M. G. H. Female, fifty seven years old. Three months before the present admission the patient had been operated upon for strangulated inguinal hernia: three inches of intestine that were strangulated in the sac had been very dark in color but had improved somewhat under observation and had been considered viable and returned to the abdominal cavity; the hernia had been repaired.

The patient returned with a history of increasing abdominal pain, nausea and vomiting. A diagnosis of intestinal obstruction was made and the abdomen explored. A stricture of the small intestine was located. This was resected and an anastomosis carried out. The patient made an uneventful recovery.

The pathological report on the section of bowel removed was as follows: "The specimen consists of a piece of small intestine 5 cm. long, whose

surface is rough and red. The lumen is stenosed and admits the point of the scissors with some difficulty. The wall is thickened and fibrous. Microscopical sections from the central portion show the wall of the gut entirely destroyed by scar tissue. No mucosa remains, and the inner surface is formed by a connective tissue which contains focal collections of wandering phagocytes. Small collections of round cells are also present in the fibrous tissue. The peritoneal surface is highly vascular. It would seem probable that this scar tissue had been formed by the organization of blood clot which arose in the course of an infarction."

Comment: While the intestine was not injured so severely by strangulation in the inguinal hernia that necrosis followed, the wall of the gut was gradually converted into scar tissue which on further contraction produced the obstruction. This is an unusual late complication of strangulated hernia.

See also Diagnosis, p. 244, and Treatment, p. 302.

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CHAPTER X

VARIETIES OF OBSTRUCTION (Continued)

OBSTRUCTION BY OCCLUSION OF THE MESENTERIC VESSELS

Occlusion of the larger mesenteric blood vessels is usually followed by acute intestinal obstruction, due to changes produced in the intestinal wall. It is open to question whether such obstructions should be classed with those of mechanical origin, since there is no blockage of the intestinal lumen, or with the functional obstructions. Since, however, there is an organic basis for the disease and since these obstructions are in certain instances subject to direct operative procedure, they are in this monograph placed among the mechanical obstructions (see p. 203).

The two lesions of most frequent occurrence are thrombosis (arterial or venous) and arterial embolism. In some cases both lesions are present, thrombosis following primary arterial embolism (Jackson, Porter and Quinby¹). Among the cases of mesenteric occlusion in the recent Massachusetts General Hospital series,² there were six of mesenteric thrombosis and three in which the circulation was occluded in other ways. In one of these latter instances a carcinoma arising in the sigmoid had extended around the inferior mesenteric artery, completely occluding the artery and resulting in hemorrhagic infarction of the area of colon supplied by this vessel. In another instance, a group of enlarged glands of metastatic origin had pressed on the superior mesenteric artery and produced intestinal infarction. In the third instance, arteriosclerotic changes in branches of the superior mesenteric artery had been so extensive as to result in the hemorrhagic infarction of the segment of ileum which they supplied.

ETIOLOGY. The diseases which lead to the formation of thrombi or emboli are of etiological significance in occlusions of the mesenteric vessels.^{1,3,4} Among these may be

mentioned endocarditis, atheroma of the aorta, and arteriosclerosis, particularly of the mesenteric arteries. The embolus frequently arises from vegetation on the mitral or aortic valves; or dislodgment of a thrombus from the left auricle may occur. Occasionally an atheromatous patch in the aorta may be the source of an embolus. In the venous type of thrombosis, conditions causing stenosis in the portal system are of importance. The thrombus may be primarily in the mesenteric veins and ascend into the larger radicals; or the reverse process may take place. Infections, particularly suppurative conditions in the peritoneal cavity arising from the appendix, may be responsible for the thrombosis. Thrombosis of the mesenteric vessels may result from the trauma of volvulus, strangulated hernia, or intussusception. Cirrhosis, syphilis, pylophlebitis, etc., should be mentioned as causes of secondary or descending venous thrombi.

PATHOLOGY. In the cases analyzed by Jackson, Porter and Quinby,¹ the occlusion was arterial in 61 per cent, venous in 39 per cent. The changes produced in the bowel by the occlusion of an important mesenteric vessel are various: they are influenced by a number of factors, of which the more outstanding are the size of the vessel occluded, the rapidity with which the interruption of the circulation takes place and the adequacy of the collateral circulation. If the blood supply to a considerable segment of intestine is suddenly interrupted by the occlusion of an artery, a hemorrhagic infarct usually results. Welch and Rolleston⁵ have established the fact that this is due not to the regurgitation of blood from the veins, as had previously been thought, but to the entrance of blood through the capillaries from collateral arterial circulation. Rarely, an anemic infarct follows obstruction of a mesenteric artery (2 per cent in Trotter's series⁶). For a comprehensive discussion of the mechanism of infarction, the work of Welch⁵ should be consulted. The mesentery itself becomes edematous and congested; and bloody fluid is usually present in the peritoneal cavity. Degenerative changes in the affected segment of bowel

may vary from congestion, edema and other inflammatory reactions, to complete infarction, necrosis and perforation. Klein's³ article gives a comprehensive and interesting discussion of the varied pathological pictures that may be produced by vascular lesions of the mesentery; as this author points out, occlusion even of the superior mesenteric artery may at times take place without fatal consequences. (See also case reports by Ross⁷ and Reed,⁸ where spontaneous recovery took place.)

AGE AND SEX. Sixty-four per cent of the cases of Jackson, Porter and Quinby¹ occurred in men, 36 per cent in women. The ages of the patients are shown as follows:

Age (years)	Per Cent
10 to 19.....	4
20 to 29.....	12
30 to 39.....	16
40 to 49.....	22
50 to 59.....	18
60 to 69.....	15
70 to 79.....	8
80 to 89.....	3
90 to 99.....	2

It will be noted that mesenteric occlusion is largely a disease of middle or old age, although cases are occasionally met with in the young.

COURSE OF THE DISEASE. As would be expected from the varied pathological pictures presented by this disease, the clinical manifestations are also varied; the course of the illness depends upon the size of the vessel affected and the ability to establish adequate collateral circulation. Jackson, Porter and Quinby divide their cases into two groups: those which run an acute course, and those which run a chronic course. The first group is much the larger. In these cases there may be a sudden onset of the colicky pain characteristic of obstruction, often at a time when the patient is in good health. Unless the condition is relieved, death usually follows in a few days or even hours, although occasionally recovery may take place without operation even when the symptoms have been acute and severe.^{3,7,8} The pain is usually generalized over the abdo-

men; it may be constant and extremely severe, although not infrequently there are exacerbations with comparative comfort between attacks. Vomiting is a very common symptom and if the infarcted area is high in the intestinal tract the vomitus may contain blood. If there is any passage of stools they are very likely to contain blood; Jackson, Porter and Quinby found blood in the stools in 41 per cent of their cases. There may be an absence of bowel movements from the onset of the attack; or there may be a number of bloody movements, followed by constipation. Distention comes on early and may be severe. The abdomen is usually tender. The temperature is likely to be nearly normal, although in the fulminating cases it may be subnormal. A marked increase in the white blood cells is a rather characteristic finding in the disease.⁴

The group of cases which run a chronic course is smaller. The onset of these cases is insidious. They may go on to a spontaneous cure.¹

See also Diagnosis, p. 242, and Treatment, p. 300.

The following is an illustrative case of mesenteric thrombosis:

CASE XXIV. *Obstruction by mesenteric thrombosis.*

No. 269,845, M. G. H. Male, aged fifty-nine. He had been operated upon for cholelithiasis ten years before, and since that time had had intermittent attacks of abdominal pain. On admission the patient stated that he had suffered during the past month from attacks of generalized abdominal pain. He had vomited three days before entrance, but this had not been repeated. There had been no bowel movement for the past six days. On physical examination the abdomen was found to be distended and somewhat tender. No masses were palpated and no peristalsis could be seen or heard. The temperature was 100.2°F., pulse 126, white blood count 22,000.

The patient was in poor condition before operation.

The operation was carried out under ether anesthesia. The peritoneal cavity contained bloody fluid. An infarction of 1½ feet of the upper jejunum was found; the mesentery was thickened and edematous. The gangrenous segment of small intestine was resected. The patient died on the table.

The following is an abstract of a case reported by Ross⁷:

CASE XXV. *Obstruction by mesenteric thrombosis; verification at operation; recovery without resection*

Case No. v. D. H. G. Male, fifty-one years of age. The day before admission the patient was suddenly taken with severe epigastric pain, which in the course of an hour or two became generalized. The pain was paroxysmal in character, leaving the patient with a dull ache between paroxysms. He vomited several times; the bowels did not move after the beginning of the illness. Past history negative.

Examination of the abdomen showed little or no distention. Exaggerated peristalsis could be heard on auscultation. White blood count 13,000; 76 per cent polymorphonuclears. Enema without result. Lavage showed gastric contents with fecal odor.

Patient was operated on under the diagnosis of intestinal obstruction. At operation a thrombosis was found involving a branch of the superior mesenteric artery which supplied a segment of ileum. There was considerable hemorrhage into the mesentery and a small amount of free blood in the abdominal cavity. The segment of bowel affected was in fair condition, apparently being taken care of by the collateral circulation. It was decided that resection was not necessary, and the abdomen was closed.

The patient was discharged in good condition on the fourteenth day after operation.

Comment. This case illustrates the point made in the text: that occasionally a mesenteric thrombosis may occur and yet the collateral circulation be sufficient to maintain the viability of the bowel.

The next case is quoted from an article by Jackson, Porter and Quinby¹:

CASE XXVI. *Obstruction by mesenteric thrombosis.*

No. 14. Male, aged sixty-four. Three days before admission the patient was seized with sudden, severe pain in the abdomen, somewhat to the right of the umbilicus; diarrhea but no vomiting at the onset. Since yesterday there had been constant abdominal pain with increasing distention. There has been persistent nausea with vomiting, and no bowel movements for two days.

Physical examination showed a fairly well-developed and nourished man; temperature 100°F., pulse 80, respirations 23, white blood count 24,000. The abdomen was distended and there was some tenderness, especially in the region of the umbilicus.

The patient was operated upon. A large quantity of free, clear fluid was found (culture sterile). A loop of obstructed bowel was located which was dark red in color with here and there bright yellowish-green mottlings

about $\frac{3}{4}$ inch in diameter, covered with thick fibrin. The mesentery attached to this loop of bowel was of fair color, thickened, but bled scarcely at all when incised. The gangrenous loop, which showed a sharp line of demarcation, was resected and an end-to-end anastomosis was carried out. Examination of the resected loop showed that the mesenteric artery and vein were filled with a more or less adherent thrombosis, especially so in the artery, the inner coat of which was proliferated.

The general condition of the patient appeared improved following the operation; but there was soon a recurrence of the previous symptoms, marked distention of the abdomen, and no movement of the bowels or passage of gas could be obtained. The patient died three days after operation. Autopsy showed that further thrombosis, and gangrene in the bowel proximal to the anastomosis, had taken place.

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CHAPTER XI

THE CLINICAL AND PATHOLOGICAL PICTURE

PATHOLOGICAL ANATOMY

Turning now to the changes produced by obstructions, we will consider first the local changes that take place under conditions of simple obstruction, and later the special changes found when the obstruction has been complicated by interference with the blood supply.

SIMPLE OBSTRUCTIONS. The pathological changes in the intestine following simple obstructions vary with the duration of the illness and the lesion causing the obstruction. In general, the longer the obstruction has persisted the more marked are the changes. Many of the more serious degenerative changes are the result of distention, which interferes with the circulation to the bowel wall.* This does not appear at once, since an appreciable length of time must usually elapse after the onset of obstruction before the distention becomes of sufficient importance to impair the blood supply.

The coils of intestine above the obstruction are dilated; those below collapsed. In the later stages the blood vessels over the surface show evidence of hyperemia and congestion; there is frequently a definite bluish, cyanotic tinge, indicating capillary and venous stagnation. The intestinal walls may show evidence of congestion and edema; at times they may be distended to almost paper thinness. The mucosa usually shows evidence of intense congestion and edema and is swollen and purplish-red in color; small hemorrhagic areas may be found. The wall is often extremely friable and must be handled with the greatest circumspection to avoid a tear or perforation. Hartwell, Hoguet and Beekman¹ have studied very carefully the changes in the intestinal wall following simple obstruction in dogs. In general, the lesions they described were congestion,

*See p. 25.

hemorrhagic areas and areas of superficial necrosis. (See also Braun and Wortmann.²)

These lesions are often all that can be demonstrated in the intestinal tract in uncomplicated, fatal cases of obstruction; and at times even these cannot be found. Elman and Hartmann³ in over half of their experimental obstructions were not able at autopsy to demonstrate lesions in the bowel wall. Carlson and Wangenstein⁴ also reported only slight changes: congestion and edema. At times, however, extensive lesions may be found. The distended bowel may show ulcers due to interference with the circulation of the bowel resulting from distention (Bryant,⁵ Kocher,⁶ Morison,⁷ Van Beuren⁸); Van Beuren stresses the importance of the time factor in the damage caused by distention. The ulcers may progress to actual perforation. These changes are most often seen in their extreme form in distention of the cecum and may progress to extensive areas of gangrene and perforation. Extensive local changes in the bowel wall take place at the actual point of obstruction: for instance, a band passing over the bowel wall may produce pressure ulceration at the point of contact. In the area where a gallstone has become impacted there may also be areas of pressure necrosis. Experimental studies¹ have shown that even very localized damage to the bowel, as, for example by a constricting ligature, may be an important factor in the clinical course of the obstruction. (Further discussion will be found on p. 344.)

In cases where an acute obstruction is superimposed upon a chronic stricture, extensive inflammatory changes may be found above the obstruction, causing catarrhal or phlegmonous ulceration. This type of ulceration, according to Kaufmann,⁹ is due in some cases to distention, in other instances to toxic necrosis from the chemical effect of stagnating feces and the secondary bacterial action. Kaufmann mentions cases in which obstructing, stony-hard scybala have produced ulcers which were actual imprints of fecal masses. These ulcers are due to anemia brought on by local pressure; and the disintegration may go

on to perforation. The bowel wall above a stricture that has persisted for some time may show marked hypertrophy, dilatation and inflammatory reactions. A detailed account of these changes is given by Treves.¹⁰

Intestinal Contents. The contents of the bowel, if the obstruction is in the small intestine, is usually the thin, brownish, foul-smelling fluid commonly described as "fecal" fluid (see p. 228). The amount of gas present varies in different cases; it is usually abundant in obstructions of the large intestine.

Normally, few bacteria are found in the stomach, duodenum and upper jejunum (Cushing and Livingood,¹¹ Herter¹²). The ileum contains a diverse flora of potentially pathogenic bacteria, embracing a large number of different types of anaerobic as well as of aerobic organisms (Herter), the numbers becoming more numerous as the ileocecal valve is approached. The colon, of course, always contains enormous numbers of organisms. Among the anaerobic organisms that may normally be present in the intestine, Herter mentions *B. putrificus*, *B. aerogenes capsulatus*, *B. sporogens*, *B. tetani*, and *B. histoliticus* have also been mentioned (Williams¹³).

On the basis of the fact that few bacteria have been reported as occurring normally in the stomach, duodenum and upper jejunum, it has been assumed by some writers that this holds under conditions of obstruction. This is by no means true: the fluid above the obstruction contains enormous numbers of organisms regardless of whether the obstruction is located high or low in the intestinal tract. It has been shown (Cannon, Dragstedt and Dragstedt¹⁴) that the intestinal flora following obstruction becomes dominated by the proteolytic types of bacteria. Williams has stressed the great increase in the numbers of *B. welchii* that will ordinarily be found in the contents of obstructed loops, and this has been confirmed by McIver et al.¹⁵ and others.* (See also Chapter xxxi.)

* This organism is not always found, however. Recently, anaerobic cultures of the loop contents from a patient acutely ill with high obstruction showed no gas-producing bacilli.

STRANGULATIONS. The mechanism of strangulation has already been discussed (p. 35). The pathological changes that are found in the bowel wall depend upon a number of factors, of which the more important are the completeness of the interference with the circulation and the time that has elapsed since the onset of the obstruction. Due to the thin walls of the veins and their low internal pressure, interference with the venous return occurs quickly. The bowel wall soon shows evidence of the venous stasis: the color changes to a dusky, mahogany red, which turns to black as the venous occlusion becomes more complete. As the blood supply fails, a violent and disordered type of peristalsis has at times been noted.¹⁶ The lumen of the intestine becomes distended with a bloody fluid exudate containing a large amount of particulate matter, largely blood cells and bacteria. The fluid that rapidly accumulates in the peritoneal cavity is also blood-stained, a finding which furnishes valuable evidence of strangulation in exploratory operations for intestinal obstruction.

If the strangulation is not relieved, the changes rapidly progress to actual gangrene of the intestinal wall. The mucosa is the first structure to show the disintegrative effects, and in complete venous occlusion these can occur very early (Murphy and Vincent¹⁷). With the necrosis of the mucosa, an important barrier to bacteria has been destroyed, and a rapid infiltration of the walls by microorganisms occurs. The appearance of the gangrenous loop is characteristic: the peritoneal surface loses its normal sheen, becoming dull and lusterless; it may show a thin layer of grayish fibrin over the surface, often binding the affected loop lightly to the surrounding loops or to the parietal peritoneum; thrombosis of the involved mesenteric vessels may often be demonstrated. All tone in the muscular structure is lost and the wall feels pasty and doughy. Perforation may occur, with a resulting diffuse peritonitis; or peritonitis may appear without a macroscopic perforation.¹⁸

The pathological changes occurring in the bowel in intussusception and mesenteric thrombosis have already been discussed (pp. 80 and 134).

PERITONEAL CAVITY. In the early stages of simple obstructions there is usually an increase in the amount of free peritoneal fluid. Where strangulation is present this fluid is likely to be blood-stained. After degenerative changes have occurred in the bowel wall, peritonitis is a relatively frequent complication. This may be due to a gross perforation in the obstructed bowel; or, where gangrene is present, the intestinal wall may be so permeable to bacteria that peritonitis occurs without a gross perforation. Some degree of peritonitis is a common finding at autopsy in patients dying of obstruction. This is particularly true of those dying after operations which involve openings into the bowel: it is not easy to avoid soiling the peritoneal cavity with the highly septic contents of the bowel; and there are a certain number of cases where in attempting to free an adherent loop the friable bowel wall gives way and some of the contents is spilled: a most serious accident. Among 123 autopsies of obstruction cases at the Massachusetts General Hospital, general peritonitis was listed as the principal or contributing cause of death in 66 instances.

PULMONARY LESIONS. A pneumonic process in the lungs may be found at autopsy either as a terminal manifestation of the disease or resulting from the aspiration of septic vomitus at operation. In the latter case, if the patient survives long enough, the process may go on to lung abscess or gangrene.

The following quotation from Leichtenstern¹⁹ is of interest:

We have yet to mention briefly a lesion which is found in the lungs of those who have succumbed to ileus. I refer to pneumonia caused by the presence of foreign bodies in the lungs, particles of vomited matter (fecal matter, when the vomiting is stercoraceous) carried in during inspiration. This can occur the more easily as the patients are often rendered comatose by large doses of opium and the reflex irritability of the mucous membranes of the respiratory passages is diminished. Like most pneumonias of this kind, they occupy mainly the lower or middle lobe of the right lung, where infiltrations (sometimes distinct lobular ones, sometimes united in forming lobar ones) are found with a dirty-gray or dark green surface on section, central sphaclated softening, and formation of gangrenous cavities filled with the offensive matter. The surrounding lung tissue is more rarely in a

condition of hepatizing inflammation than in that of soft, moist infiltration. Microscopical examination of the gangrenous spot clearly shows, as I have occasionally demonstrated, that aspiration of the contents of the intestines has taken place, for we find there pieces of muscle stained with bile, bits of vegetables, grains of starch, etc. (Leichtenstern, 1876.)

Among the 123 cases of acute intestinal obstruction autopsied at the Massachusetts General Hospital, serious pulmonary complications existed in 29 instances, as follows:

Bronchopneumonia.....	16
Lobar pneumonia.....	2
Lung abscess.....	1
Lung abscess with empyema.....	1
Gangrene and abscesses of the lung.....	1
Gangrene of lungs.....	2
Edema of lungs.....	4
Pulmonary embolus.....	2

NERVE LESIONS IN THE CELIAC GANGLIA. Changes have been reported by Myerson²⁰ as occurring in the nerve cells of the solar plexus in a case of volvulus; the chief changes found were a disappearance of the Nissl bodies and an abnormal location of the nucleus.

SUMMARY. Simple Obstructions. Pathological changes in the intestine following simple obstruction depend upon the nature of the obstruction and its duration. A band passing over and obstructing the bowel may produce pressure necrosis at the point of contact; gallstones and other foreign bodies may produce ulceration from pressure at the point of impaction. Distention may interfere with the circulation and so produce degenerative lesions. If an acute obstruction be superimposed upon a chronic obstruction, hypertrophy and extensive inflammatory reactions may be found above the stricture. The bowel content consists of thin, brownish, foul-smelling fluid containing large numbers of aerobic and anaerobic organisms.

Strangulations. The changes following strangulation depend upon the completeness of the strangulation and its duration. The venous circulation is first affected. The bowel wall becomes congested, and if the strangulation is not relieved, gangrene

with its characteristic features follows. A thick bloody exudate containing a large number of bacteria accumulates in the lumen of the strangulated bowel.

The peritoneal cavity usually shows an increase of free fluid. If strangulation is present the fluid is likely to be bloody. In the late stages a general peritonitis is a frequent complication. Pulmonary complications are relatively common.

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CHAPTER XII

THE CLINICAL AND PATHOLOGICAL PICTURE (Continued)

GENERAL FINDINGS

We will now consider the more general bodily changes produced by intestinal obstruction.

TEMPERATURE. In general, it may be said that intestinal obstruction does not produce marked changes in body temperature unless some complication is present. In the early stages of simple obstruction the temperature is usually normal or only slightly elevated; it may be subnormal. Later in the disease there may be a slight fever, probably due to infection around the point of obstruction. In obstructions by strangulation, a high temperature will occasionally be found late in the course of the disease; in such cases there is usually considerable peritonitis in the region of the involved loop. Following operations for intussusception in children and infants a marked rise in temperature may occur, even though no peritonitis be present¹; the cause for this reaction is not clear. In rare instances where there is infarction of extensive portions of the intestine there may be a subnormal temperature.

RESPIRATION. In the early part of the disease some quickening of respiration is caused by pain sensations. In the later stages the respirations are usually rapid and shallow: Braun and Boruttau² considered that this was a manifestation of anemia of the respiratory center due to low blood pressure, and regarded the damage to this vital center as an important factor in a fatal outcome.

CIRCULATORY SYSTEM. *Pulse.* At the onset of all types of the disease there is likely to be some increase in heart action, shown by the accelerated pulse. This would be expected to accompany the acute pain, and probably represents increased

stimulation of the heart by way of the sympathetic accelerator fibers.* After the initial stages, the pulse may return to normal, particularly if the pain has been controlled by morphine; although in cases of strangulation at least a slight elevation of pulse rate is likely to continue. It may be said in general that if the disease progresses in an unfavorable manner there is an accompanying increase in the pulse rate; but, as pointed out by Eisberg,³ one should not be deceived as to the seriousness of the situation by the fact that the pulse is of normal rate and good quality, for its character may not be affected until late in the disease and then may rather suddenly become rapid and thready. Cases of mesenteric thrombosis have been reported⁴ where the pulse rate was strikingly slow early in the disease.

Blood Pressure. It is generally agreed that in the early stages of obstruction there are usually no marked changes in the arterial blood pressure. Frequent reference will be found in the literature⁵ to the collapse that accompanies the onset of acute obstruction; but I know of no report of low blood pressure shown by such patients; Tileston and Comfort⁶ are among the few authors who have recorded blood pressure readings on patients, and their data are meager. It seems likely that when early collapse does occur it is in the nature of prostration from acute pain and is not accompanied by the low blood pressure associated with true shock; exception to this statement may be found in instances of extensive infarction or strangulations which involve considerable portions of the intestinal tract.

In the terminal stages of obstruction there is a progressive fall in the systemic blood pressure. Most writers regard this merely as one manifestation of a toxemia that is bringing about a suspension of all the vital processes. Braun and Boruttau,² however, believe that the fall in blood pressure is of primary importance; they attribute to this the other signs of collapse, and consider that most of the other serious effects of the

* Sweating is another evidence of sympathetic activity often noted at this time.

disease may be considered as secondary to the resulting anemia. They believe that the outpouring of fluids and vascular stasis in the splanchnic area decrease the circulating blood volume and thus initiate a fall in blood pressure (see p. 391). It is obvious that once the blood pressure has fallen to a low level, very serious secondary effects may promptly follow, the patient passing into a depressed state resembling in many respects that seen in traumatic shock. (See p. 311.)

Accompanying the fall in systemic blood pressure there is evidence of capillary stasis, as shown by the grayish or dusky pallor exhibited by patients with obstruction as the end approaches.

Blood Cytology. Usually little change in the red blood cells is produced by intestinal obstruction. If the concentration of the blood is marked, there may be a relative increase in red blood cells. Considerable variation, on the other hand, may be shown by the white blood cells. If the obstruction is a simple one, the white count is usually normal; occasionally there may be some elevation, due probably to infection in the region of the obstruction. If strangulation is present the count is usually elevated and is likely to go higher as the disease progresses. The ratio of polymorphonuclear leucocytes is usually increased. Cases of mesenteric thrombosis are likely to show a striking elevation of the white blood cells.

Blood, Bacteriology. It is generally agreed that bacterial invasion of the blood stream does not occur except as a terminal phenomenon.

Blood Chemistry. The changes which may take place in the chemical composition of the blood plasma during obstruction are so important that they are given a separate chapter.

KIDNEYS. That intestinal obstructions are accompanied by a diminution in the secretion of urine often amounting to anuria was known to the early observers (Leichtenstern,⁷ Brinton,⁸ and Nothnagel⁹). This suppression of excretion was attributed by these early workers in part to dehydration and

in part to a reflex inhibition of kidney secretion; they applied this latter explanation particularly to those cases of strangulation where diminished secretion of urine accompanied the onset of the attack.

It is only comparatively recently that careful investigations of the disturbance of kidney function caused by intestinal obstruction have been carried out. In 1914, Tileston and Comfort,⁶ while investigating the non-protein nitrogen and urea of the blood in a number of diseases, studied 3 cases of acute intestinal obstruction and found a great increase in both of these constituents; the volume of the urine was small and showed albumin and casts. McQuarrie and Whipple¹⁰ studied this subject in more detail in relation to intestinal obstruction, and showed that there was a great increase in urinary nitrogen and a marked increase in non-protein nitrogen of the blood; they also reported a decrease in kidney excretory function, as shown by the inability of the kidney to excrete the normal amounts of urea, sodium chloride and phenol-sulphonphthalein. Along with this they reported a decrease in the output of fluids.

That a serious disturbance of kidney function, or at least a decreased secretion of urine and a heaping-up in the blood of nitrogenous products, accompanies intestinal obstruction has been abundantly confirmed by all later workers (Haden and Orr,^{11,12} Gatch et al.,¹³ Gamble and McIver,¹⁴ and others); there has been, however, a difference of opinion as to the mechanism of this impairment of the kidney function. Hartwell, Hogue and Beekman,¹⁵ and later Brown, Eusterman et al.,¹⁶ have reported degenerative lesions of the kidneys in association with upper intestinal obstruction. Most of the workers, however, have not been able to demonstrate organic lesions of the kidney. McQuarrie and Whipple¹⁰ believe that although they could find no anatomical lesion, the impaired function is due to the direct action of a toxic substance upon the renal epithelium. Marriott and others,^{14,17} on the other hand, believe that no injury to the kidney exists but that under

conditions of dehydration this organ is not able to function properly; in other words, it cannot separate a normal urine from concentrated blood. This explanation of decreased kidney function would seem to be the most reasonable, at least in those cases showing dehydration, and would account for the prompt return to normal under appropriate treatment.

LIVER. The function of the liver under conditions of intestinal obstruction has not been extensively investigated. That there may be serious disturbance was suggested by Werelius,¹⁸ chiefly on the basis of the fact that he observed in his experimental animals a marked decrease in the flow of bile following the production of upper intestinal obstruction. Colp and Louria,¹⁹ however, not only were unable to confirm this observation but found that the liver function as determined by the phenoltetrachlorophthalein method was normal; they were, moreover, unable to demonstrate any structural changes in the liver. Haden and Orr's studies¹² on the fibrin content of blood following obstruction do not support the idea that liver insufficiency plays a rôle in intestinal obstruction. Werelius's belief that there is serious disturbance of liver function thus requires further proof.

BASAL METABOLISM. Disturbances of the basal metabolic rate have not been recorded in intestinal obstructions. Presumably when the terminal shock-like state is reached the metabolism is depressed, as it is in traumatic shock (Aub²⁰).

SUMMARY. Unless complications are present there is usually no marked deviation from the normal in temperature, pulse and respirations. The blood pressure also is usually normal early in the disease. The blood count is likely to be normal unless strangulation is present; when it is present, the leucocyte count is likely to be elevated and is often strikingly high in cases of mesenteric thrombosis. There is a decrease in kidney function, as shown by the diminished secretion of urine and by various laboratory tests; there is also frequently an increase in non-protein nitrogen of the blood. There is difference of

opinion as to whether there is actually kidney damage or whether all the changes can be explained on the basis of dehydration; there is considerable evidence in favor of the latter view. There is apparently no serious disturbance of liver function. Changes in the chemistry of blood and body fluids are discussed in the next chapter.

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CHAPTER XIII

THE CLINICAL AND PATHOLOGICAL PICTURE (Continued)

CHANGES IN BODY FLUIDS

Important changes frequently occur in the composition of the blood plasma following intestinal obstruction, the more important being a *decrease in the chlorides* and an *increase in the alkali reserve* and a *rise in the non-protein nitrogen*. Along with the chemical changes, a marked degree of *dehydration* may occur.

The fact that patients with intestinal obstruction frequently give evidence of extreme loss of water was familiar to the early writers, Barlow,¹ Brinton² and Leichtenstern.³ The last named author mentions dryness of the mucous membranes and skin, intense thirst, choleraic face and voice, sunken eyes, cramps in the calves of the legs, and anuria. All of these symptoms, he states, point to a rapid withdrawal of water from the blood; and this he considers due to vomiting and profuse sweating. These changes are most typical and pronounced in simple high obstruction; but they may also occur in low obstruction if the vomiting is profuse and continues over a period of several days. They are rarely found in the type of obstruction characterized by strangulation, because in such cases the whole course of the disease is likely to be too short for extensive loss of fluid by vomiting.

Closely associated with the loss of blood chlorides and, as a rule, proportionately increasing as the chlorides are lowered, there is, as stated above, an increase in bicarbonate or alkali reserve. This represents an important adjustment in the blood, tending to maintain the normal reaction in the face of the large loss of the acid radical, chloride. The literature bearing on the lowering of the blood chlorides in intestinal

obstruction may here be briefly reviewed. In 1912, Hartwell and Hoguet⁴ showed that dogs with high intestinal obstruction (simple blockage of the bowel) could be kept alive for several weeks if given, by subcutaneous injection, large quantities of salt solution. Unless so treated, the animals died within four to six days. They believed that the efficacy of this measure consisted in sustaining the water content of the body, and advanced the theory of dehydration as the cause of the symptoms in this type of intestinal obstruction. In 1920, MacCallum and his co-workers⁵ showed that in experimental pyloric obstruction the blood chlorides are lowered and the alkali reserve is increased. In 1923, Haden and Orr⁶ also found a lowering of blood chlorides in high intestinal obstruction, and confirmed Hartwell's experiments demonstrating the efficacy of salt solution in prolonging the life of animals. Since their first contribution to this subject, Haden and Orr, in numerous investigations and clinical studies, have stressed the importance of the lowering of the blood chlorides; and it is largely due to their efforts that the medical profession has been convinced of the great importance of this finding and the value of instituting appropriate treatment. In their earlier papers, these authors advanced the theory that the reason the chlorides were lowered in the blood was that they were withdrawn from the plasma to neutralize some toxin absorbed from the obstructed intestine. In their later publications, however, they modified their opinion on this point, coming to agree with Gamble and McIver,⁷ White and Bridge,⁸ Atchley and Benedict⁹ and others, who contend that the decrease of chlorides is due to their loss in the digestive secretions by vomiting.

MECHANISM OF DEHYDRATION. In papers appearing in 1925, Gamble and Ross,¹⁰ and Gamble and McIver⁷ regarded the loss of sodium and chloride ions as largely responsible for the fatal effects of high intestinal obstruction; for, while agreeing with Hartwell and Hoguet⁴ that death in this type of obstruction was due to dehydration, they considered the

dehydration the result of the loss of these electrolytes, using here the general physiologic concept that the volume of a body fluid is sustained by its total ionic content. The substances

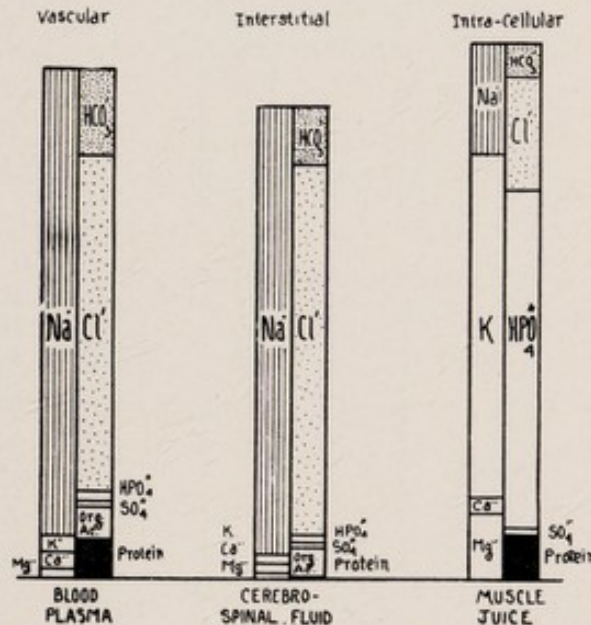


FIG. 37. Acid-base composition of body fluids. This diagram is constructed from average values for individual factors expressed in terms of acid-base equivalence: i.e., as cubic centimeters of tenth normal solutions per 100 c.c. of fluid. Base factors are superimposed in left hand and acid factors in right hand of each column. They represent, as is actually the case, a structure composed not of salt but of individually sustained concentrations of ions. Exact acid-base equivalence indicated by equal height of two parts of each column is obtained by adjustability of bicarbonate ion concentration (HCO_3) to any change elsewhere in structure.^{11,13}

which are of chief importance in supporting the volume of the blood plasma and of the interstitial body fluids are sodium and chloride ion. The reason for this is, simply, that these two substances are quantitatively the chief factors in the total ionic content of these fluids, as may be seen by reference to the diagram describing the acid-base composition of the body fluids given in Figure 37.¹¹ It may be noted that, according to the third column in this diagram, sodium and chloride ion are present in relatively small amounts in intracellular water. The significance of this fact will be referred to presently. That these two substances are also the chief inorganic factors in the composition of the digestive secretions is shown by

Figure 38. This diagram is constructed from data obtained from specimens of digestive secretions collected from animals by the methods of Pawlow and others.¹² The total fixed base in

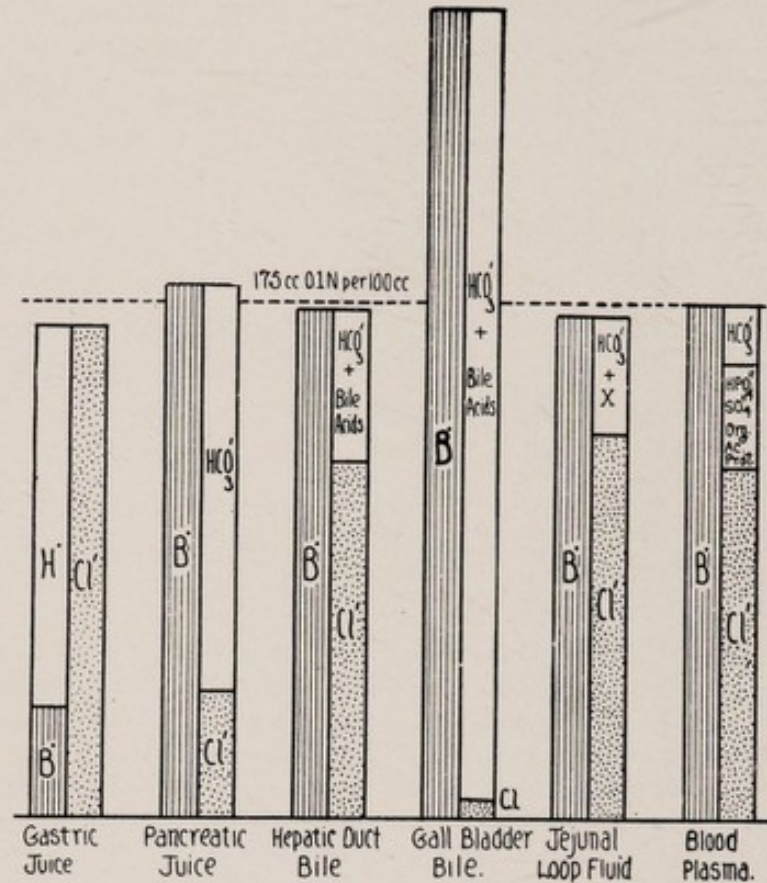


FIG. 38. Acid-base composition of digestive secretions. As in Figure 37, this is constructed from measurements of component factors expressed as cubic centimeters of tenth normal per 100 c.c. of fluid, base factors being superimposed on left hand and acid factors on right hand of columns. Pancreatic juice data from dog; other data from cats. (McIver and Gamble¹³)

these secretions (B in Figure 38) is, as in the case of the blood plasma, composed almost entirely of sodium. A diagram of the composition of the blood plasma (of the cat) is also included in order to make clear the fact that the digestive secretions contain fixed base and chloride ion in amounts which are appreciable in terms of the plasma content of these substances. The inorganic substances in the digestive secretions are, of course, derived from the plasma. Under ordinary

circumstances they are in great part returned to the plasma following the periods of digestive activity, and there is also abundant replacement from the food intake. Evidently, if there is interference with their reabsorption, as is the case in the presence of pyloric or high intestinal blockage, a continuous loss from the blood plasma will occur. According to the conception which is here being used, the consequent depletion of the total ionic content of the plasma and of the interstitial body fluids will be accompanied by an approximately parallel loss of water with the result that a normal total ionic concentration tends to be sustained at the expense of reduction of volume. That is, dehydration is regarded as the result of the loss of electrolytes; and an important corollary of this view is that dehydration cannot be repaired by the administration of water alone: an attempt to restore and sustain the original volume of the blood plasma and of the interstitial body fluids must include replacement of the lost electrolytes. An additional theoretical point has been brought out by Gamble and Ross;¹⁰ viz., that loss of sodium rather than loss of chloride ion is the significant factor in determining the extent of dehydration, since the loss of chloride ion is compensated for within the body fluids by a corresponding increase of bicarbonate ion.

EXTENT OF LOSS OF WATER AND OF ELECTROLYTES IN DIGESTIVE SECRETIONS. The enormous volume of the digestive secretions which in the course of twenty-four hours normally enters the upper part of the gastrointestinal tract and is reabsorbed from the lower bowel is usually not appreciated. From the data which Rowntree¹⁴ has collected from various sources, it may be estimated that for an adult the secretion of digestive fluids, taken together, amounts to between 5 and 7 liters daily. This is two or three times the volume of the blood plasma which is the immediate source of these secretions.

It has been shown that circumstances causing severe dehydration may reduce the volume of the blood plasma by one third.¹⁵ This finding by no means indicates the actual extent

to which water and electrolytes have been withdrawn. It has been found that, following experimental pyloric and duodenal obstruction in rabbits, several times the total initial plasma content of water, of fixed base and of chloride ion enters the stomach during the survival period, as shown in Table XII.

TABLE XII
ESTIMATIONS OF LOSS OF WATER, FIXED BASE AND CHLORIDE ION FOLLOWING PYLORIC AND DUODENAL OBSTRUCTION, IN TERMS OF ORIGINAL BLOOD PLASMA CONTENT; DATA FROM RABBITS

	Pylorus Obstructed C.c.	Duodenum Obstructed C.c.
Water lost.....	203	235
Initial plasma H ₂ O.....	83	77
H ₂ O lost ÷ initial H ₂ O.....	2.4	3.1
	C.c. Tenth Normal	C.c. Tenth Normal
Fixed base lost.....	270	539
Initial plasma B.....	140	130
B lost ÷ initial B.....	1.9	4.1
Chloride lost.....	309	265
Initial plasma Cl.....	85	79
Cl lost ÷ initial C.....	3.6	3.4

It will be noted that several times the total initial plasma content of water, of fixed base and of chloride ion is lost during the survival period. These substances are largely replaced from the interstitial body fluids, with resulting dryness of the skin, shrinkage of the subcutaneous tissues and other clinical signs of dehydration. (Gamble and McIver⁷)

A rapid and extensive replacement of water and of electrolytes is thus indicated. The immediate source of these materials is the interstitial body fluids. Reduction of the volume of these fluids provides the familiar clinical evidences of dehydration, i.e.: dryness of the skin, shrinkage of the subcutaneous tissues and, in infants, deep recession of the fontanel. It is probable that intracellular body fluids are only to a slight extent drawn upon, for the reason that these contain relatively small amounts of the electrolytes, sodium and chloride ion, particularly required for plasma repair (Figure 37, column 3, showing composition of muscle juice).

Obviously, this arrangement defends cell volume; and direct measurements of the water content of parenchymatous tissues from severely dehydrated animals have shown an extremely small loss. The data in Table XII show that a considerable part of the body's content of water and of sodium and chloride ion may be rapidly lost. When similar circumstances are encountered clinically, replacement measures should be correspondingly extensive and sustained.

CHANGES IN THE BLOOD PLASMA CAUSED BY LOSS OF DIGESTIVE SECRETIONS. *Concentration Changes Due to Loss of Water.* As a result of the reduction of the volume of the blood plasma there occurs, as might be expected, an extensive increase in the concentration of plasma protein, and a rise in the red count and hematocrit reading. That loss of water from the plasma causes physical changes in the blood, increase of viscosity, for example,¹⁶ which must seriously interfere with its respiratory and other functions, is shown by the fact that in severe dehydration a tenfold reduction of volume flow may occur.¹⁷ Haden and Orr¹⁸ have recently shown a huge reduction of the oxygen content of venous blood in the presence of high obstructions.

Changes of Acid-Base Structure Due to Loss of Sodium and Chloride Ion. The important change to be here considered is a change in the reaction of the plasma due to alteration of the bicarbonate concentration, and this in turn is referable to the relative amounts of sodium and of chloride ion withdrawn from the plasma. The latter relationship may be briefly explained with the help of Figures 37 and 38. In Figure 37 it may be seen that the sum of the acid factors in the plasma is represented as exactly equivalent to the total of fixed base. This equivalence is maintained in the presence of changes in the individual factors, by the adjustability of the bicarbonate ion concentration. In Figure 38, column 1, the composition of gastric juice, chloride ion is seen to be in large excess of fixed base.¹² Withdrawal of these substances from the plasma in the proportions indicated in this diagram may be expected

greatly to decrease the extent to which fixed base in the plasma is covered by chloride ion; and to this extent the bicarbonate ion concentration will be increased. Actually it has been well established that continued loss of stomach secretions following experimental obstruction of the pylorus does produce an alkalosis, often of a degree sufficient to cause severe tetany. The columns representing the inorganic factors in pancreatic juice and in gall-bladder bile¹⁹ show that, in complete contrast to gastric juice, these secretions contain fixed base in much greater amounts than chloride ion. The inference is thus provided that loss of these secretions will tend to reduce the plasma bicarbonate. This expectation has been verified by finding a marked degree of acidosis, as measured by bicarbonate reduction, in the blood plasma of a dog following construction of a pancreatic fistula which completely drained away the external secretion.²⁰

In the presence of upper intestinal obstruction there is obviously opportunity for loss of all these digestive secretions. The accompanying change in plasma bicarbonate has been found to be widely variable: usually there is an extension, which is often large; occasionally there is no appreciable change from the normal value, and infrequently there is a considerable reduction. According to the foregoing discussion, this variability of plasma bicarbonate is understandable as the result of differences in the relative amounts of stomach and of duodenal digestive secretions which have been lost, loss of the former tending to produce alkalosis and of the latter, acidosis.

Besides the loss of gastric juice, of pancreatic juice and of bile, it is quite likely that another factor is present: viz., loss of fluid derived from the intestinal mucosa. The probable composition of this fluid is indicated by the analysis of a specimen drained from an isolated upper jejunal loop, which is represented in Figure 38. There is some doubt as to whether this fluid should be regarded as an irritatively stimulated digestive secretion or as an exudate. In any case, however,

it is seen to contain approximately the same amount of fixed base and of chloride ion as does the blood plasma. Loss of this fluid will cause reduction of the volume of the blood plasma and of the interstitial body fluids, but should not alter the plasma chloride (or bicarbonate) concentration.

To turn our attention to the concentration of plasma chloride: from the considerations given it is evident that not infrequently in the presence of upper intestinal obstruction the circumstances are such that the loss of chloride ion is not in excess or is only slightly in excess of the loss of fixed base, in terms of the plasma concentrations of these substances, with the result that plasma chloride remains at or only slightly below its usual value, although the absolute loss of chloride ion from the plasma and interstitial fluids may have been extremely large. To use a very simple illustration of the situation: if a large part of the contents of a flask of salt solution is poured out, the chloride concentration of the solution remaining in the flask is not altered. Even when the plasma chloride is found greatly reduced, the extent of reduction of concentration is by no means a measure of chloride loss, since several times the total normal plasma content may have been withdrawn, as shown in the table. A point of great practical importance to be derived from these statements is that it is entirely incorrect to regard the plasma chloride concentration as an index of the degree of dehydration and of the extent to which replacement by salt solution is required.

From the foregoing discussion it is clear that the question of dehydration and the disturbance of the acid-base balance in the blood is intimately related to the loss of the digestive secretions by vomiting; and that the only way to correct these changes is by the administration of adequate volumes of fluid containing sodium chloride. A further discussion of this will be found under "Treatment" p. 254.

CHANGES IN THE NON-PROTEIN NITROGEN OF THE BLOOD. A marked increase in the non-protein nitrogen of the blood is frequently found following acute intestinal obstruction. It

occurs even more consistently than does the lowering of the blood chlorides, for while the latter is not usually found in the fulminating cases of obstruction where strangulation is present and the disease runs too short a course for extensive vomiting to occur, an increase in the non-protein nitrogen of the blood is generally present.

Several factors may be responsible for the increase in non-protein nitrogen: in the first place, there is an increase in tissue destruction due to the effects of dehydration and perhaps in certain cases due to the action of some circulating toxin²¹; in the second place, dehydration brings about a decrease in kidney function (see p. 150), so that a heaping up of the non-protein nitrogen constituents of the blood takes place.

OTHER FINDINGS IN THE BLOOD CHEMISTRY. Other changes in the blood under conditions of obstruction have been recorded. Guest et al.²² have reported marked changes in the distribution of phosphorus in the blood, which they correlate with a progressive loss of chlorides from the blood cells. Haden and Orr^{16,18,23,24} have noted large reductions in the oxygen content of venous blood; increased viscosity; increase in blood fibrin; and increase in sedimentation rate of erythrocytes. Ribeyrolles²⁵ has reported the occurrence of hemolysis following certain types of obstruction.

SUMMARY. Dehydration and other alterations in body fluids are most marked in simple high obstruction. Outstanding findings in the composition of the blood plasma are a decrease in chlorides, an increase in the alkali reserve and a rise in non-protein nitrogen; there is also a reduction in its total volume. For the dehydration and the alterations in the acid base balance, the loss of sodium and chloride ions in the digestive secretions is largely responsible. The immediate source of these essential electrolytes is the plasma, and this in turn is replenished largely from the interstitial fluids, for the intracellular fluids contain relatively small amounts of sodium

and chloride ions (see Fig. 37). The plasma bicarbonate varies with the relative amounts of stomach and upper intestinal secretion lost, loss of the former tending to produce alkalosis and of the latter acidosis. Under conditions of high intestinal obstruction the loss of gastric secretion usually predominates and a condition of alkalosis ensues.

As a result of the reduction of volume of blood plasma there occurs an increase in concentration of plasma proteins, a rise in red count and haematocrit readings and an increased viscosity. These changes seriously interfere with the function of the blood. The rise in non-protein nitrogen may be caused by an increased tissue destruction and a decreased kidney function resulting from the dehydration. See also Chapter XXXIII, p. 387.

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CHAPTER XIV

THE CLINICAL AND PATHOLOGICAL PICTURE (Continued)

MECHANISM OF PAIN, VOMITING AND DISTENTION

PAIN. Three important manifestations in acute intestinal obstruction of mechanical origin are pain, vomiting and distention. Of these, abdominal pain is one of the most important; it is nearly always present and is usually severe. The character and localization of the pain in different types of obstruction are taken up in some detail under Diagnosis. In the present chapter will be considered some of the more fundamental questions dealing with the origin of pain and the pathways over which it is transmitted.

Pathways of Transmission. The exact way in which sensations of pain from the intestinal tract are registered in consciousness has puzzled both physiologists and clinicians. Pain sensations are rarely (if ever) actually localized by the patient in the intestines but are referred to some portion of the anterior abdominal wall; and at times the skin in the region to which the pain is referred may become hypersensitive (cutaneous hyperalgesia) and the underlying muscle tonically contracted (muscle spasm). The areas to which the pain is referred are fairly constant for certain portions of the intestinal tract; pain from the jejunum, for example, is referred to the midline above the umbilicus.¹ Presumably the particular locality to which the pain is referred depends upon the level at which the afferent impulses from the diseased viscus enter the spinal cord, the pain sensations being projected along the corresponding nerve roots to that portion of the abdominal wall which they supply.

Not only are pain sensations from a diseased intestine not localized in the intestine, but, somewhat paradoxically, despite the severe pain that is felt as a result of pathological processes in the intestines, these viscera may be handled,

pinched, cauterized or subjected to any of the other common types of stimulation without any resultant pain. It is well known that the parietal peritoneum, including the mesenteric attachment of the intestines, is extremely sensitive to the usual forms of stimulation; as regards the former, this is readily explained, because sensory spinal nerves run in the loose connective tissue just beneath the layer of endothelial cells. Whether the mesentery also contains spinal sensory nerve endings is uncertain; but it has been suggested² that the pacinian corpuscles found in this region constitute such organs. It is generally agreed that the intestines themselves are supplied only by the vagus and splanchnic nerves. The disputed point, therefore, is whether or not there are true pain sensations from the intestines, transmitted by afferent visceral fibers. Lennander³ held that there were not, and attributed all sensations of pain either to a pull or to pressure exerted on the mesentery or parietal peritoneum by a violently contracting loop, or to an involvement of the mesentery or parietal peritoneum in an inflammatory reaction. Mackenzie⁴ also did not believe that sensations of pain could come directly from abdominal viscera; he maintained that a portion of the spinal cord, upon receiving afferent impulses from a diseased viscus, became abnormally sensitive ("irritable focus"); and that normal sensory impulses from the muscles and skin entering this hypersensitive area were interpreted as pain which was localized, not in the viscus, but in peripheral structures (see Fig. 39). According to this view, pain from a duodenal ulcer, for example, is not felt in the duodenum but is referred to that part of the anterior abdominal wall corresponding to the distribution of the sensory nerves entering the segment of the spinal cord rendered irritable by afferent impulses from the diseased duodenum. Mackenzie's theory seems to have received some confirmation from the work of Weiss and Davis,⁵ who were able to abolish certain types of visceral pain by anesthetizing with a local anesthetic the area of skin to which the pain was referred.

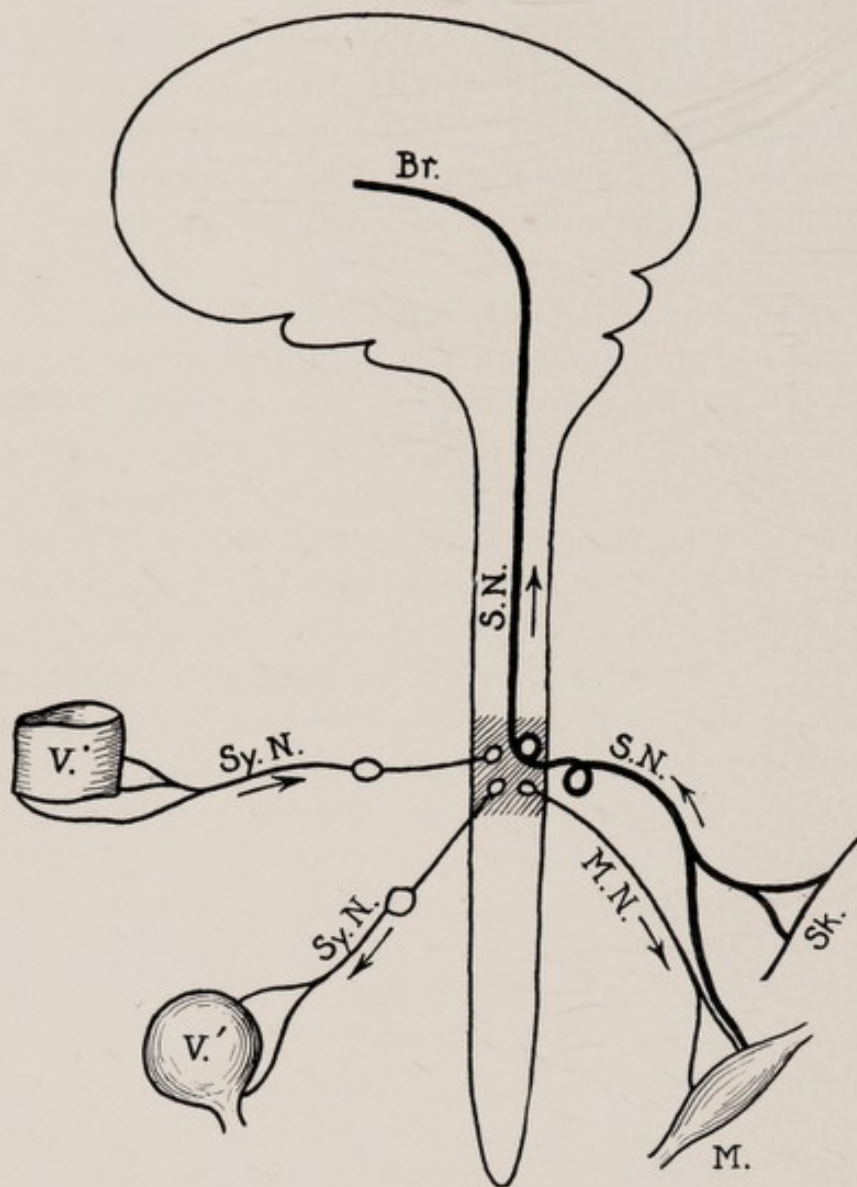


FIG. 39. Mechanism of visceral pain, cutaneous muscular hyperalgesia (viscero-sensory reflex), the visceromotor reflex and organic reflex. An adequate stimulus proceeding from organ, v, by sympathetic nerve, SyN, to center in spinal cord extends to cells of nerves in its neighborhood, and stimulates them to activity, when function peculiar to each nerve is exhibited. Thus stimulus affecting cells of a pain nerve, SN, results in perception of pain which is referred by brain to peripheral distribution of nerve in external body wall (Sk, m); affecting cell of a motor nerve, MN, causes a contraction of muscle, m, supplied by motor nerve; and affecting cells supplying other viscera (as v') stimulates them to their peculiar function (as contraction of a hollow muscular viscus, or increased secretion of a secretory organ). If stimulus is of sufficient strength it may leave an irritable focus in spinal cord (shaded area) as shown by a persistent hyperalgesia of skin and muscle (Sk, m) and by a persistent contraction of muscle (m). (Mackenzie⁴)

On the other hand, Ross,⁶ Nothnagel,⁷ Hurst,⁸ Ryle⁹ and Morley¹⁰ have contended that true pain sensations can arise from the viscera themselves, with appropriate stimulation. As regards pain from the intestines, the important factor, according to Hurst and Ryle, is abnormal tension in muscle fibers due to spasm or to failure to relax in the presence of increased intrainestinal pressure. Morley¹⁰ regards intestinal pain as the result of increased tension on the sympathetic nerve endings in the intestinal musculature. That true sensations of pain can arise from the intestines seems most likely, in view of the recent work of Mixer and White¹¹ and others, on sensations from the thoracic viscera conveyed by sympathetic fibers (see also Kuntz¹²). Ross, Hurst and Morley believe that there are two types of pain from the abdominal viscera; one direct from the viscera; the other referred. Head¹³ agrees with this, and considers that the pain arising from the involved organ is "dull, boring and wearing," thus differing from the sharp aching and stabbing of referred pain. It seems probable that the view of two types of pain is correct; although there is no one theory that satisfactorily explains all the observed phenomena.

Alvarez's¹⁴ article on abdominal pain reviews the literature on the pathways of pain and concludes that most of the sensory fibers leave by way of the splanchnic nerves, but points out that the situation is complex and that further study is required on many points.

It has been demonstrated both clinically and experimentally (Cannon and Murphy¹⁵) that acute intestinal obstruction is accompanied by a violent type of peristalsis, and it seems clear that part of the pain experienced is associated with this excessive intestinal activity; clinically it will occasionally be observed on inspection of the abdomen that the pain complained of is synchronous with a vigorous wave of peristalsis. The patient frequently recognizes this pain as similar in character to former attacks of intestinal colic, such, for example, as result from the use of strong cathartics,

or from eating the proverbial green apple, a type of pain undoubtedly associated with hyperperistalsis and spasm. Hurst⁸ believes that peristalsis itself is unable to cause pain: that only when the onward passage of intestinal contents is prevented by organic or spasmodic obstruction is there a rise of intestinal pressure sufficient to produce pain. It seems likely, however, that any violent, disordered type of peristalsis or a localized tonic contraction of a segment of intestine might give rise to painful sensations and that this type of pain comes directly from the gut. The reference of these colicky pains is usually as follows: upper jejunal pain is apt to be referred to the mid-line between the umbilicus and the ensiform cartilage; sensations from the rest of the small intestine tend to be referred to the region of the umbilicus or across the abdomen above that point. Pain from the large intestine is usually referred across the abdomen below the umbilicus. (Fig. 40.)

There is probably another element present in the pain of intestinal obstruction, particularly in strangulations; although in such cases the very violent tonic contractions accompanying interference with the blood supply (Foster and Hausler,¹⁷ McIver et al.¹⁸) might, in part, account for the pain, there is probably also direct injury to the nerve endings in the mesentery. Not only does direct compression of the mesentery stimulate the nerve endings in this location, but edema and later inflammatory changes are also a source of irritation.*

Peritonitis is a third element that may play a role in the pain, particularly in strangulations and in the later stages of simple obstruction. Patients with this complication frequently show extreme localized tenderness and muscle spasm in the region overlying the strangulated loop, due to the involvement of the parietal peritoneum in the inflammatory process.

* Brinton¹⁹ considered that there was pain from congestion and dilatation of blood vessels in the region of the obstructing band; basing this chiefly on the fact that there were such a large number of nerve fibers accompanying the blood vessels to the abdominal viscera.

VOMITING. Vomiting is probably the next most constant symptom associated with acute obstruction of the intestines. Since it occurs almost simultaneously with the onset of

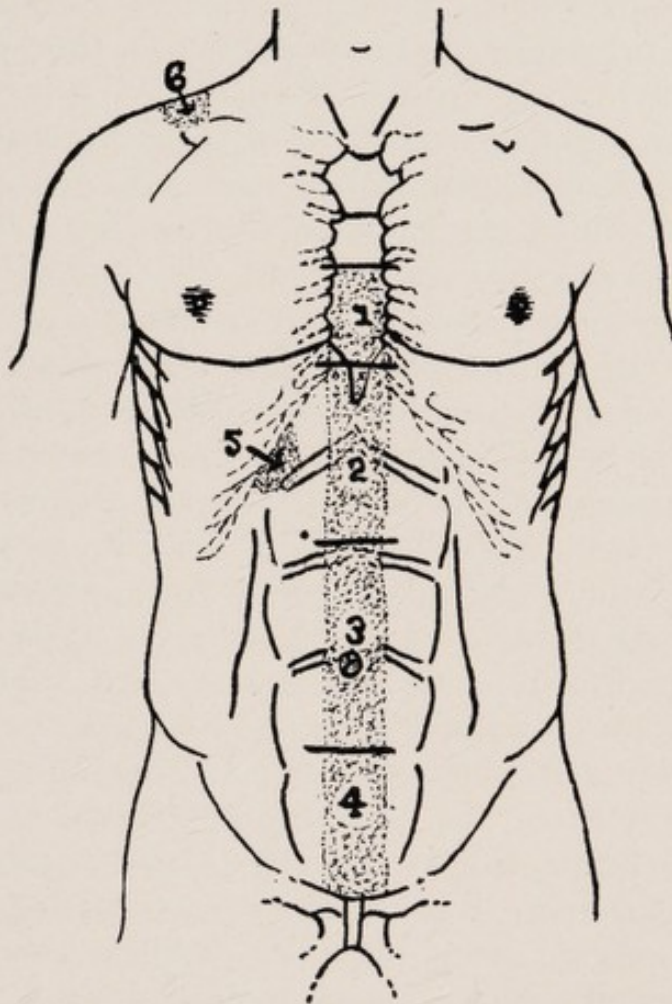


FIG. 40. Areas to which pain is likely to be referred from various parts of digestive tract: (1) from esophagus; (2) from stomach; (3) from small intestine; (4) from colon; (5) and (6), regions to which pain may be referred in diaphragmatic pleurisy; (5) from border of diaphragm, and (6) from its dome (according to Capps and Coleman). (Modified from Mackenzie,⁴ by Homans¹⁶)

the attack, it must, at least in the early stages, be reflex in nature, as it is at the onset of most of the acute pathological conditions in the abdominal cavity. The stimulus reaches the vomiting-center, which according to Hatcher and Weiss²⁰ is located in the medulla within the sensory nuclei of the vagi, by way of either the vagus or the splanchnic nerves. (See also

Walton et al.²¹) As the disease progresses the vomiting may be a manifestation of the general toxic state of the patient rather than a reflex phenomenon. In the late stages regurgitation from the stomach, rather than actual vomiting, often takes place; the dilated and atonic stomach never completely empties itself.

The mechanism by which the fluid that accumulates in the small intestine reaches the stomach has been a matter of some dispute. All the early writers regarded it as due unquestionably to reverse peristalsis; from the time of Galen²² to the seventeenth century, reverse peristalsis was regarded as the essential feature in intestinal obstruction, indeed as the cause of "ileus" (Sydenham^{23*}). Later Brinton²⁴ vigorously contested the idea that reverse peristalsis occurred. He believed that a normal wave of peristalsis passing along the intestine toward the occluded part tended to produce two distinct currents in the tube. One of these currents near the intestinal wall passed downward, carrying the contents of the bowel in the direction of the peristaltic wave; this increased the pressure in the bowel, and since a continued passage downward was prevented by the obstruction, an upward movement in the central part of the column of fluid occurred, and this upward current carried the liquid back into the stomach as succeeding waves of peristalsis passed downward toward the obstruction. Brinton compared this action to that of a hollow piston moving down a tube which is closed at one end; as the piston moves downward, fluid is

* The following quotation is from Sydenham: "This terrible disorder (ileac passion), which has hitherto generally been esteem'd fatal, is owing to the inversion of the peristaltic motion of the guts, when their contents are forced upwards, and thrown out by vomiting, so that the strongest glysters become emetic, as do likewise cathartics, immediately after being taken. And I judge the exquisite and intolerable pain attending this disorder, proceeds only from the inverted peristaltic motion of the bowels, whose natural formation is such as by their many folds to promote the descent of the faeces in the properest manner; and therefore whenever they are forced to yield to a motion opposite to that of their fibres, a pungent pain is occasioned."

Sydenham considered that in organic obstruction reverse peristalsis occurred above the obstruction only. He therefore called it "spurious ileac passion."

forced up through the hole in the piston. More recent studies have shown that both Brinton and the very early writers are correct. Cannon and Murphy¹⁵ were able by means of the fluoroscope to see the intestinal contents "squirted" backward towards the stomach when a powerful wave of peristalsis passed downward toward the obstruction. These authors, however, further demonstrated that at times a true reverse peristalsis also occurs. (See also Alvarez^{25,26}.)

Another belief was held by Nothnagel,²⁷ who regarded the act of vomiting as a force capable in itself of carrying the intestinal contents upward to the stomach. The occluded bowel, he considered, became dilated with fluid above the obstruction, the column of fluid extending backward to the stomach; when the act of vomiting occurred, the diaphragm and abdominal muscles contracted, the intra-abdominal space was reduced, pressure was exerted on the intestine with its contained fluid, and since the pressure could not be relieved by forcing the fluid downward, it was of necessity forced upward, into the stomach. Some such mechanism may occur in the late stages, for at that time probably all types of peristalsis are absent and yet profuse vomiting, or at least regurgitation, may continue to the end.

While the loss of fluids with the contained sodium and chloride ions is, of course, detrimental to the body as a whole, the drainage back into the stomach and vomiting must be a beneficial compensatory phenomenon relieving intractable pressure and so conserving the circulation in the bowel wall. It is possible that in humans the ligament of Treitz and the sharp angulations of the duodenum tend to prevent a perfectly free flow backward into the stomach and perhaps this is one reason why the insertion of a catheter into the jejunum at times gives such beneficial results.

DISTENTION. Distention is such an important element in obstruction, both as an etiological agent in certain types and as the cause of many of the secondary lesions and symptoms, that it is almost impossible to discuss any phase of the subject

without touching on this topic. This is emphasized in most of the recent work on obstruction, and was in part recognized by the early writers, who noted that good results were occasionally attained by blind tapping of distended intestinal loops. Before taking up the mechanism of distention, a brief summary is given of some of its important effects, with references to the pages where these are discussed in more detail.

Distention is a source of great discomfort to the patient, and adversely influences his general condition. Mechanically, it may embarrass the heart action and respiration. Perhaps its most serious action is in damaging the capillary circulation of the bowel: the resulting degenerative changes may progress to ulceration or to actual necrosis of the bowel wall; and even when the changes are not as extreme, some writers believe that the mucosa may be so injured as to permit toxic materials to be absorbed.* Distention has also been considered actually to force toxic products from the gut into the absorbing channels (p. 380). By promoting stasis in the large capillary bed of the splanchnic area it may be a factor in the production of shock (p. 311).

In mechanical obstructions, if distention has persisted for some time it may produce an atonic condition of the intestinal musculature, so that even after the mechanical blockage has been removed, a functional obstruction remains, superimposed upon the mechanical (p. 202). In functional obstructions distention may further injure an already handicapped intestine, thus forming a link in a vicious circle. It may also stimulate the secretory function of the intestine, so that once distention has occurred it may itself cause further secretion which in turn increases the distention (p. 177). Following operations where anastomoses have been carried out it may seriously endanger the integrity of a suture line, both mechanically and by interference with its blood supply (p. 288).

As an etiological factor, distention plays a rôle in the production of internal (p. 38) and external strangulations (p. 121) and of volvulus. It may be a factor in the production of serious

* See also pages 24, 111, 140, 141, 284, 329, 350, 367, 379, 380.

kinks and twists of the intestine, particularly when peritonitis is present, and may convert a subacute into an acute obstruction (pp. 47 and 211). From the point of view of diagnosis, distention is one of the cardinal signs, and may be demonstrated at a very early stage, particularly by means of x-ray plates taken without opaque media (pp. 229 and 233).

We will turn now to a discussion of the mechanism of distention.

Source and Character of Distending Fluids. Distention is brought about by an increased pressure of either fluid or gas or of both together in the intestinal lumen. We will first consider the intestinal fluids, their source and character.

The fluid that accumulates in such large amounts above an obstruction has certain typical characteristics. It is the same foul-smelling, yellowish fluid with white flecks of particulate matter that is described under "fecal vomiting," page 228. It is derived from several sources. In the first place, there is the fluid that reaches the intestines from the stomach, consisting of ingested fluids, saliva and gastric secretions. To this is added, in the upper duodenum, the secretions from the liver and the pancreas; and finally there is abundant outpouring from the intestines themselves. Most of the increased secretion is probably best regarded as the result of an irritative process. That injury or irritation of the nerve endings in the intestinal walls may be an important factor is suggested by the fact that cutting the nerves to an intestinal loop is capable of producing copious secretion, the so-called "paralytic secretion" (Mendel,²⁸ Falloise,²⁹ Molnar³⁰). Lim et al.³¹ and Herrin and Meek³² have shown that mechanical distention of the stomach or an intestinal loop by means of a balloon will result in a copious secretion; so perhaps once distention has occurred the distention itself acts as a stimulation to continued secretion. Perhaps some rôle may be played by an increase in the osmotic pressure of the loop contents. Boese and Heyrovsky³³ considered on the basis of their animal experiments that in obstructions of the colon a hypersecretion of the mucous glands

and a transudate due to injury of the mucosa and blood vessels occurred. Dragstedt³⁴ has suggested that certain products of decomposition formed in the obstructed loop may, when absorbed, act as secretagogues and further augment the secretion from the digestive glands and intestines. Where there is blockage of the venous mesenteric circulation, such as is found in strangulation, the obstructed loop becomes distended by a bloody fluid exudate brought on by an increase in the capillary pressure and by damage caused by a poor supply of oxygen³⁵; the particulate matter, which consists chiefly of blood cells and bacteria, may constitute 40 per cent of the exudate in these cases (McIver, White and Lawson¹⁸). The early experiments of Kader,³⁶ dealing with the characteristics of distention following various types of strangulation and interference with the mesenteric blood supply, are of interest.

Not only is there a great outpouring of fluid into the intestine under conditions of obstruction, but there is a retardation of absorption which further favors the onset of distention.

Source and Character of Distending Gases. An increased volume of gas in the intestinal lumen may at times be a very important factor in the production of distention.

The gases of the intestinal tract were first systematically studied by Planer³⁷ in 1860 and the work later amplified by Ruge,³⁸ Tappeiner,³⁹ Tacke⁴⁰ and others. These writers agree that the gases commonly present in intestinal meteorism are carbon dioxide, oxygen, nitrogen, hydrogen, methane, and sometimes hydrogen sulphide, the proportions being subject to considerable variation. In considering the relation of these gases to distention, their origin must be taken into account.

One well-recognized and important source for certain of the gases is found in the decomposition of intestinal contents; this is probably one of the most important causes of gaseous distention under conditions of mechanical obstruction; corroboration is found in the fact that obstructions of the large intestine, where fermentation is active, are usually characterized by marked gaseous distention. The amount of gas

in the obstructed small intestine probably depends to some degree on whether the intestine was empty at the time the obstruction occurred; or, if not, to what stage digestion had

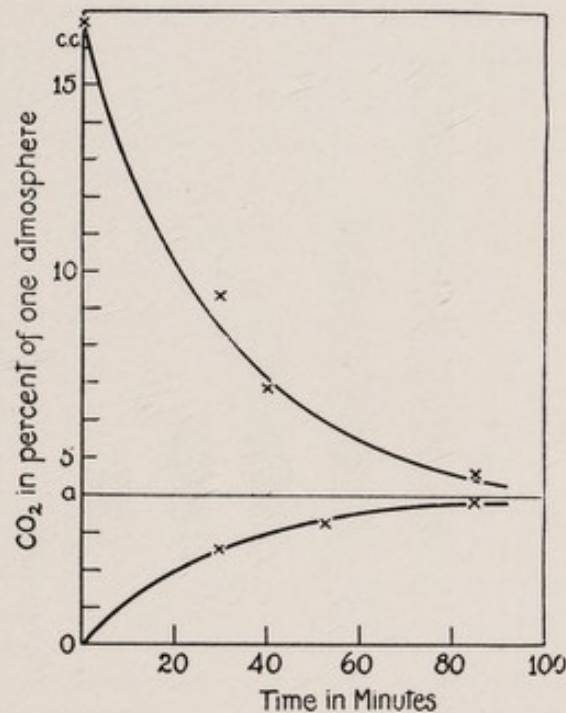


FIG. 41. When air is injected into stomach, CO₂ enters until concentration equals that of blood. Crosses related to lower curve show rate at which this proceeds. When air containing a high percentage of CO₂ is injected into stomach, CO₂ diffuses out of stomach into blood. Crosses along upper curve show rate at which this process goes on. Crosses represent actual measurements; curves are calculated on assumption that CO₂ is passing by diffusion and not by secretion. Close agreement between crosses and curves suggests that CO₂ enters and leaves stomach by process of diffusion. Line *a* represents concentration of CO₂ in blood. (McIver, Redfield and Benedict³⁵)

proceeded, since the presence of partially digested food of course greatly increases the likelihood of active fermentation and gas formation. The work of early German writers, already mentioned, and more recent studies by Boycott and Damant,⁴¹ furnish data on this subject and emphasize the influence of different types of diet on the kind of gas produced. Fine and Levinson⁴² consider liquid carbohydrates a particularly important source of distending gases.

A second source for the distending gases is found in the diffusion of the blood gases into the intestinal lumen. The walls

of the intestine are permeable to gases and an active interchange takes place, tending to keep the gases on the two sides of the intestinal mucosa in equilibrium. The mechanism of this

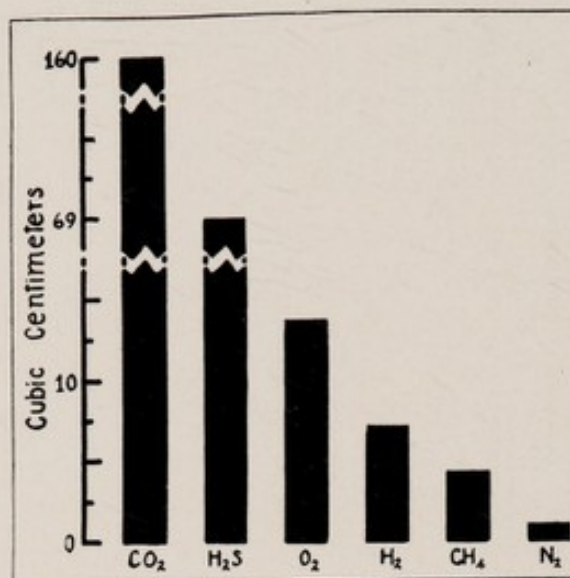


FIG. 42. Relative rates of absorption, expressed in cubic centimeters, of carbon dioxide, hydrogen sulphide, oxygen, hydrogen, methane and nitrogen, from a 25 cm. loop of small intestine of cat. Height of columns represents number of cubic centimeters of gas absorbed over a period of one hour. In case of carbon dioxide, because of rapidity of its disappearance, and of hydrogen sulphide, because of its toxicity, gas was withdrawn after lapse of a few minutes, and rate of absorption for one hour was calculated. Rate of absorption of atmospheric air was also tested in these experiments, although results are not shown in this diagram, and was found to be low, only 4 c.c. being absorbed from a 45 cm. loop in five hours. This is to be expected, because of close similarity (except for content of CO₂) between composition of atmospheric air and that of blood gases. (McIver, Benedict and Cline⁴³)

exchange is discussed in detail in a report by McIver, Redfield and Benedict.³⁵ (See Fig. 41.)

A third source for the intestinal gases found in distention must be considered. The gas normally found in the stomach consists primarily of atmospheric air admitted by swallowing. Under certain conditions the amount of air is considerably increased⁴³⁻⁴⁶; and the experiments of McIver et al.⁴³ showed that accumulations of air in the stomach are readily carried into the intestines and that in certain obstructions, particularly those following peritonitis or operative trauma, this may be an important factor in the production of distention.

Considerable quantities of air were found by these authors in the stomach of patients following operation; and the removal of this air at frequent intervals during the first twenty-four to forty-eight hours following operation markedly decreased the incidence of postoperative distention. Experiments also showed that air injected into the stomach of animals was often passed downward into the intestine; and that animals with peritonitis in which the pylorus was ligated so that air could not be passed into the intestines, showed a dilatation of the stomach but no distention of the intestines, while animals in which the pylorus was not ligated showed dilatation of both stomach and intestines, proving that the intestinal distention was due to air passed downward from the stomach.

The Body's Methods of Eliminating Intestinal Gas. The expulsion of gas by motor activity of the intestine is an important method of eliminating abnormal accumulations. Since, however, the gases cannot be driven by peristalsis beyond the point of intestinal obstruction, a second method must be relied on, namely, absorption into the blood stream.

The diffusion of blood gases into the intestinal lumen was mentioned in a preceding paragraph: the reverse process, namely absorption of gases from the intestine into the blood and elimination through the lungs, is, under normal conditions, equally active. The importance of this mechanism of elimination of gases is illustrated by Figures 42 and 43; particular attention is called to the first column of Figure 42, which shows the normal rapid absorption rate of CO_2 .

If, under conditions of obstruction, gas accumulates in the intestines faster than it can be eliminated by absorption into the blood stream, distention ensues. As the distention increases, the circulation to the bowel wall is interrupted, so that the absorption of gas is still more diminished; this, in turn, causes further distention, and a vicious circle is instituted. Impaired absorption is particularly serious in the case of CO_2 for this gas is given off in large volumes during ferment-

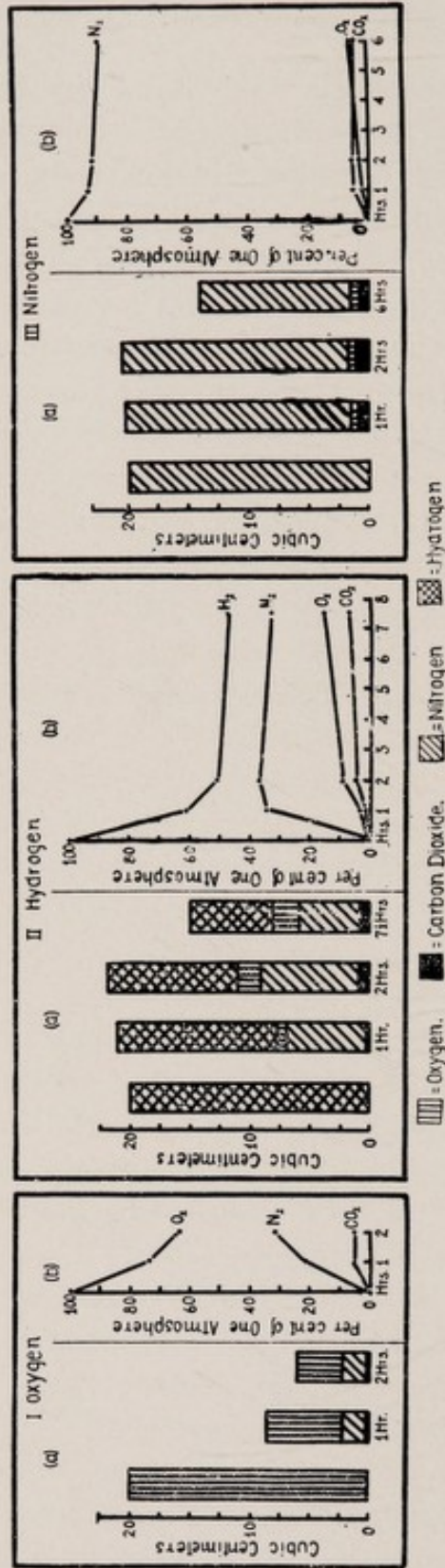


FIG. 43. Course of events following introduction of (i) O₂, (ii) H₂ and (iii) N₂ into an isolated loop of small intestine. Gases were injected severally, allowed to remain for period of time indicated, drawn off and analyzed. First column in diagram (a) represents in cubic centimeters volume of gas injected; succeeding columns represent total volume of gas and its composition when withdrawn from intestinal loop after stated intervals of time. Percentage results of analyses are plotted in diagram (b). Line descending from upper left indicates, in percentage, absorption of injected gas. Lines ascending from lower left indicate entrance of blood gases. (McIver, Redfield and Benedict³⁵)

tation, and under normal conditions would be rapidly absorbed by the blood stream.

When sudden gross interference with the mesenteric circulation occurs, experiments have shown³⁵ that if the loop is empty of gas at the time of the blockage of the mesenteric circulation, the thick, bloody exudate previously described occurs promptly, but there is usually very little accumulation of gas; if, on the other hand, the intestinal loop contains some gas at the time of the venous obstruction, the volume of gas increases, due chiefly to an accumulation of CO₂.

SUMMARY. Pain. The parietal peritoneum and mesentery are sensitive to the usual forms of painful stimuli, which can be explained on the basis of their sensory spinal nerve supply. The intestines are not sensitive to such stimuli; sensations of pain do arise from pathological processes in the bowel, but are usually felt not in the bowel itself but in definite regions of the abdominal wall which are fairly constant for different levels of the intestinal tract. It is a moot point how the sensations from the intestines are transmitted. It has been suggested that they arise in the parietal peritoneum or mesentery and not in the intestines themselves; or that afferent impulses from the diseased gut set up an "irritable focus" in the spinal cord and that normal sensory impulses entering this hypersensitive area are interpreted in consciousness as pain and localized in peripheral structures; on the other hand, many writers believe that true pain sensations arise from the viscera themselves and are transmitted over visceral fibers. It is possible that there are two distinct types of pain.

The pain of obstruction is often associated with violent peristalsis and spasm. In strangulations it seems probable that injury to the nerve endings in the mesentery may play a rôle. In the late stages of obstruction inflammatory reactions and peritonitis may be responsible for the pain.

Vomiting. The reflex impulse that produces vomiting reaches the vomiting center by way of either the splanchnic or the vagus nerves. The fluid from the obstructed intestines is, in the

early stages, either carried backward to the stomach by reverse peristalsis or forced backward as a descending wave of peristalsis encounters the obstruction. In the later stages, at least, the increase in intra-abdominal pressure created by contractions of the abdominal muscles and diaphragm may force the fluid upward into the stomach.

The passage of fluid from a distended intestine into the stomach relieves intraintestinal pressure and is therefore to some degree beneficial.

Distention. Distention is an important and serious manifestation of obstruction, adversely influencing the general condition of the patient and locally interfering with the blood supply of the bowel wall. It is also a factor in the etiology of strangulation and volvulus.

Distention results from collections of fluid and gas. The fluids are largely made up of the digestive secretions together with an abnormal secretion from the stomach and intestines. Where strangulation is present, a bloody exudate containing many bacteria accumulates in the involved loop. There are three sources of intestinal gases: fermentation, diffusion from the blood, and the passage downward of air from the stomach. The gases are normally expelled by peristaltic activity or are absorbed by the blood and eliminated through the lungs. The absorption rate of carbon dioxide is rapid.

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CHAPTER XV

FUNCTIONAL OBSTRUCTIONS

MECHANISM AND ETIOLOGY

In the preceding chapters we have been considering the intestinal obstructions of mechanical origin. We shall now take up the mechanism, etiology and clinical types of obstructions caused by functional disturbances of intestinal motility.

Functional disturbances of intestinal motility may be the result of local or intra-abdominal pathology, or may be a reflex response to general or distant bodily pathology. For example, we may have an atonic paralysis of the intestines as a result of acute peritonitis; or, on the other hand, the passage of a renal stone or certain other lesions of the kidney may cause reflex vomiting and a cessation of peristaltic movements. (Fig. 44.) Intestinal motility is also influenced by the general bodily state (see p. 205).

The mechanism governing the highly coördinated movements of peristalsis is complicated. It depends upon the muscle structure of the gut wall, with its local nerve supply, and is also influenced by an extrinsic nerve supply through the splanchnic and vagus nerves. Thus injury to the muscle or nerve elements in the gut wall might abolish peristalsis; or reflex impulses transmitted over the extrinsic nerves might bring about the same result. The situation is further complicated by the fact that the bowel may respond to one and the same injury at times by an atonic paralysis or inhibition of motility, and at other times by a spasmodic, tonic contraction of some segment of the intestine which effectually interferes with the passage of intestinal contents and so blocks the intestinal stream. Because of this fact it has long been the custom to describe the functional obstructions as "paralytic" and "spastic." Inhibition of intestinal movements (paralytic type) is much the more frequent process underlying functional obstructions, and it is largely this type that has been studied experimentally.

In order to understand more clearly the effects of injuries to various parts of the system governing intestinal movements, it is desirable to review briefly the major facts of normal

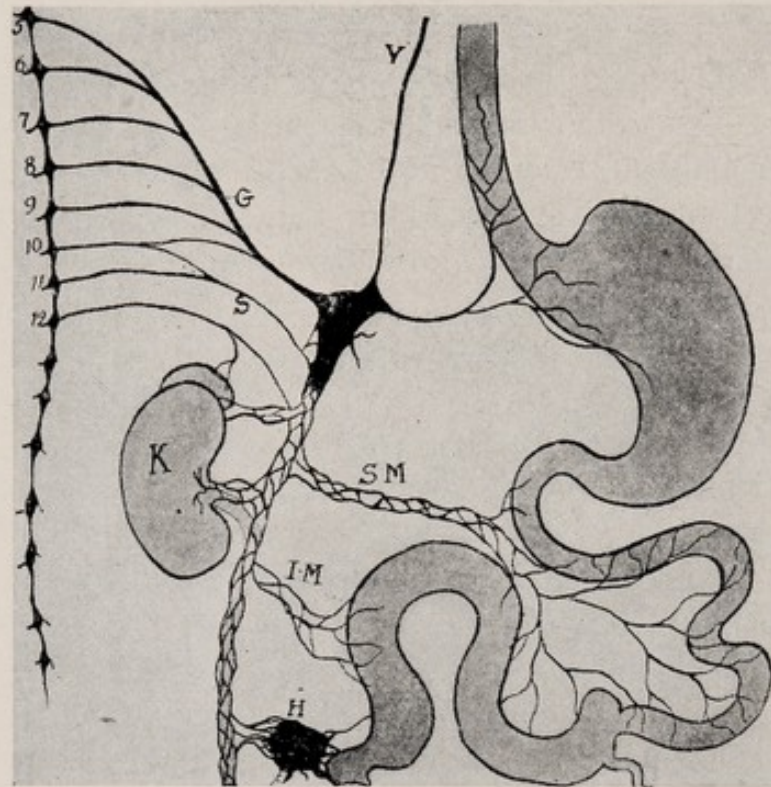


FIG. 44. Intimate connection between nerve supply of kidney and of intestines. K, kidney; V, vagus nerve to celiac ganglion; G, greater splanchnic nerve; S, smaller splanchnic nerve; SM, superior mesenteric plexus; IM, inferior mesenteric plexus; H, hypogastric plexus. Note how nerves of kidney and ureter communicate in celiac ganglion with those of stomach and intestine. (Eisendrath¹)

peristalsis and the experimental work on functional obstruction.

POINTS IN INTESTINAL PHYSIOLOGY BEARING ON FUNCTIONAL OBSTRUCTIONS. *Peristalsis.* The intestinal contents are normally propelled through the great length and numerous convolutions of the intestinal tract by a series of coördinated muscular contractions known as "the peristaltic wave." Cannon² has described the wave as occurring in two forms: one, a slowly advancing contraction that goes only a short distance; the other, a swift movement that sweeps for longer distances along the canal. This latter in its most marked form may spread

over the whole length of the small intestines in about one minute; it is called by Meltzer and Auer³ the "peristaltic rush."

In addition to the peristaltic waves, there are rhythmic

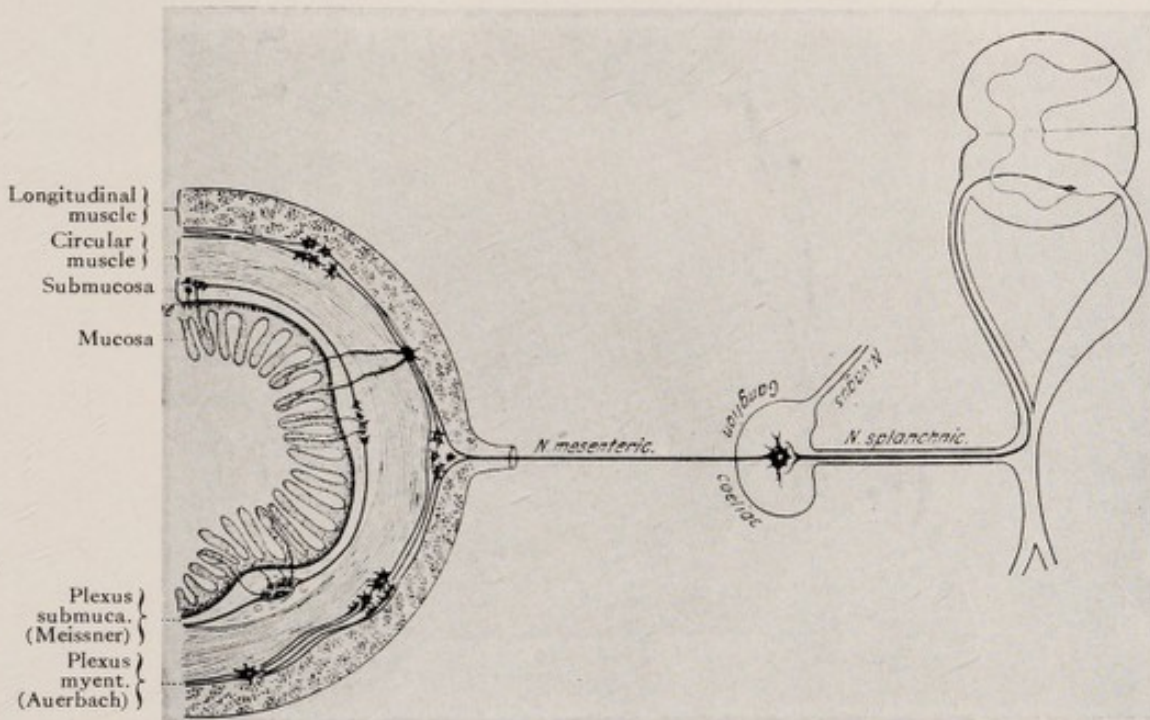


FIG. 45. Schematic representation of intrinsic nerve supply to gastrointestinal tract, showing location of submucous plexuses, etc. (Müller; see Braun and Wortmann⁵)

contractions of the intestinal musculature which segment without advancing the contents; and associated with this are gently swaying movements of the loop, the so-called "pendulum movement."⁴

Intestinal Innervation. The intestines have an intrinsic and an extrinsic nerve supply (see Figs. 45 and 46).

Intrinsic Innervation. The intrinsic innervation consists of two nerve plexuses in the intestinal wall: the submucous plexus (Meissner's plexus); and the more important myenteric plexus (Auerbach's plexus), which lies between the circular and the longitudinal muscle layers and serves to conduct stimuli and to coordinate movements. Upon this plexus depends the local reflex, the "myenteric reflex" (Cannon⁶), which, according to Bayliss and Starling,⁷ governs the orderly progress of the

intestinal contents, a wave of contraction being preceded by a wave of relaxation. Alvarez⁸ questions the importance of the myenteric reflex in the propagation of the peristaltic wave.

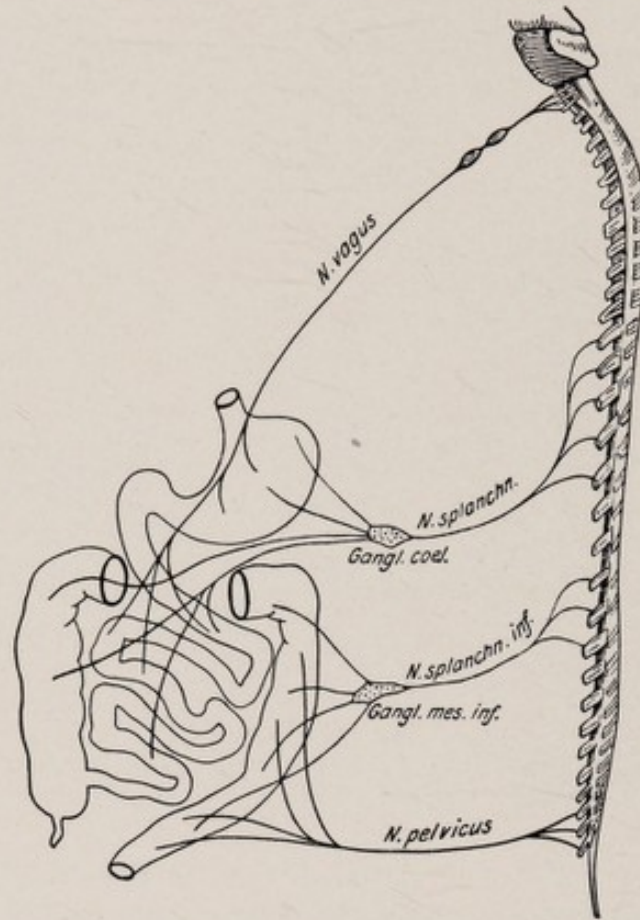


FIG. 46. Schematic representation of extrinsic nerve supply to gastrointestinal tract. (Müller; see Braun and Wortmann⁵)

According to his view, the downward passage of a wave is due to gradients of irritability, tone and metabolism between the upper and the lower portions of the intestinal tract. Cannon⁹ considers the myenteric reflex important in the propagation of peristalsis; but clearly states that it does not govern the rhythmic contractions of the small intestine, or the rhythmic peristalsis and antiperistalsis of the colon, and probably not the rhythmic waves of the stomach.

Extrinsic Innervation. Although the intrinsic innervation is complete in itself, and capable of carrying out in an orderly

manner all types of peristalsis without connection with the central nervous system,¹⁰ under normal conditions it is influenced by impulses from the central nervous system, transmitted over two opposed systems which together constitute the extrinsic innervation: the motor system, represented by the cranial (vagus) and sacral autonomic nerves, whose impulses tend to stimulate intestinal movements and increase tone; and the inhibitory system, consisting of the sympathetic fibers (splanchnics), whose impulses tend to abolish intestinal movement. The sympathetic system also carries motor fibers to the pyloric and ileocecal sphincters.

The cranial autonomic system, through the vagus, supplies fibers to the intestinal canal from the esophagus to the terminal ileum, diminishing in influence as it descends; the sacral system starts at the anal end of the canal and reaches upward along the colon with diminishing influence as it ascends. Stimulation of these nerves produces powerful contractions of the intestinal musculature. It is not known where the ganglion cells of these motor neurons to the stomach and intestines are situated; and therefore we do not know with certainty that the vagus carries preganglionic fibers to these organs. Gaskell,¹¹ however, thinks it probable that the motor cells are situated in Auerbach's plexus.

The sympathetic fibers arise in the thoracico-lumbar region and reach the abdomen by way of the major and minor splanchnic nerves, the preganglionic fibers ending in ganglion cells in the celiac plexus. It has not been finally settled whether there also exist ganglion cells belonging to the sympathetic system in the intestinal wall; but the evidence as presented by Gaskell¹² seems against it. Stimulation of the splanchnic nerves inhibits peristalsis and constricts the ileocecal and pyloric sphincters. The blood vessels are also constricted by stimulation of these fibers.

Neurogenic and Myogenic Theories of Intestinal Movements. Intestinal peristalsis is carried out by a neuromuscular mechanism consisting of the smooth muscle of the intestinal

musculature, and the intrinsic nerve supply already described. The respective parts played by nerve and muscle in the initiation and propagation of intestinal movements is a moot point in intestinal physiology. The views on this question are embodied in two theories: the neurogenic and the myogenic. While there is no conclusive evidence on which to decide between the two theories,* it may be pointed out that the chief types of intestinal movements, that is, the rhythmic contractions and the peristaltic waves, may depend upon different mechanisms, the former simpler and more primitive movements being simply a function depending upon the well-known inherent ability of smooth muscle to contract in a rhythmic manner, while the more complicated and highly developed peristaltic wave may depend upon the nervous elements for its initiation and propagation. This is a point of some practical importance in considering the functional disturbances of motility; for diverse types of injury may affect different portions of the neuromuscular mechanism.

EXPERIMENTAL WORK ON FUNCTIONAL OBSTRUCTIONS. As already stated, the injuries causing the inhibition of intestinal motility which in its severe forms constitutes complete intestinal paralysis, may be grouped as general or distant bodily injuries, and local or intra-abdominal injuries, the former bringing about reflex disturbances of motility. The occurrence of obstructions due to these major types of injury has been recognized from the time of Henrot¹⁵; but the exact way in which they come about is not generally agreed upon.

Experimental Work on the Effect of General or Distant Bodily Injuries. Cannon and Murphy¹⁶ showed experimentally that distant, pathogenic processes were capable of inhibiting intestinal movements. They studied this phenomenon under

* It has been difficult, for a number of reasons, to establish either of these theories conclusively, particularly because of the intimate anatomical relations between muscle and nerve: the muscle bundles are surrounded by nerve filaments, and even the smallest collection of muscle fibers shows ganglion cells. Methods tending to destroy one without the other may be inconclusive because of the surprising resistance to anemia of this type of ganglion (Cannon and Burket,¹³ Gunn and Underhill¹⁴). An introduction to the literature will be found in the references of Cannon, Gaskell and Alvarez.

several conditions. They noted, in the first place, that asthenic states occurring in the course of general infections tended to abolish or inhibit intestinal peristalsis. Next, they studied the effects of powerful sensory stimuli which they produced by injury to the testicles of the anesthetized cat, and found that such sensory stimulation also caused an inhibition of intestinal movements. These authors further showed that the stoppage of intestinal movements under the foregoing conditions was due to inhibitory impulses from the spinal cord, transmitted over the splanchnic nerve; for when they sectioned this nerve the inhibition was abolished. As regards the inhibition of peristalsis during asthenic states* it was a striking observation that following section of the splanchnic nerves intestinal peristalsis was observed even in moribund animals. This reflex mechanism, whereby distant lesions are able to bring about an inhibition of intestinal movements, is now generally accepted. The experimental observations of Cannon and Murphy have been recently confirmed by King.¹⁷

Experimental Work on the Effect of Local or Intra-abdominal Injury. There are numerous kinds of intra-abdominal injury which are capable of affecting intestinal motility, and there is no reason *a priori* to suppose that the mechanism is the same in all cases. Only two types of obstruction produced by local injury have been studied experimentally in any detail. These are, first, those following mechanical trauma to the gut by rough handling; and, second, those following bacterial or chemical peritonitis.

Injury by Mechanical Trauma to the Gut. Cannon and Murphy,¹⁶ in the work referred to in a preceding paragraph, also investigated experimentally the inhibition of intestinal peristalsis that follows trauma to the gut. They found that they were able to produce strong inhibition of intestinal movements by rough handling of the intestines

* It is well known clinically that patients with uremia may have marked inhibition of intestinal movements, great distention and vomiting. Instances have occurred where they have been operated upon under a mistaken diagnosis of intestinal obstruction. The exact mechanism of this type of functional inhibition of peristalsis is not known.

(of the anesthetized cat) and considered that the inhibition of movements following this type of injury was probably due to direct injury to the neuromuscular mechanism of the intestinal wall. They were of the opinion that inhibitory impulses from the spinal cord did not play any rôle in this instance, for the inhibition was not removed by section of the splanchnic nerves.

This work has recently been confirmed by Olivecrona.¹⁸ This author, however, considered that another possibility, aside from injury to the neuromuscular mechanism of the bowel or inhibitory impulses from the spinal cord, might explain the inhibition: namely, that there might be a local reflex by way of the celiac plexus. Having first confirmed the observation of Cannon and Murphy that section of the splanchnic nerves did not abolish the inhibition of intestinal movements produced by trauma, he removed the celiac plexus as thoroughly as possible; and, after allowing time for the degeneration of the nerve fibers, traumatized the intestines and found that the usual post-traumatic inhibition was almost completely absent. These experiments were few in number, and not altogether conclusive; but Olivecrona considered that they proved that trauma to the gut may produce inhibition by local reflexes through the celiac plexus, rather than as a result of injury to the neuromuscular structure of the gut wall itself.

Injury from Peritonitis. Aside from gross mechanical trauma to the intestines, peritonitis of bacterial origin may, as is well known, seriously interfere with intestinal motility. The various views as to the mechanism by which peritonitis brings about functional inhibition of intestinal movements are vague. Most writers (Wilms¹⁹) assume that both muscle and nerve plexuses are affected by the edema and inflammation resulting from the action of bacteria and their toxins. This was also essentially the view of Cannon and Murphy,¹⁶ who assumed that the mechanism was the same as in the case of mechanical trauma (namely, direct injury to the intestinal musculature and its intrinsic nerve supply) and that section of the splanchnic, therefore, should not abolish the inhibition. Arai,²⁰ however,

found that when he produced peritonitis by intraperitoneal injection of sublethal doses of bacteria, inhibition of movement occurred; if, however, the splanchnic nerves were first



FIG. 47. Case No. 8426, M.I.B.H. General peritonitis; showing coils of dilated intestines glued together by light fibrinous adhesions. It is easy to see how obstruction results from these adhesions and kinks combined with atony and distention of the bowel. (See also Fig. 48, showing a histological section of an inflamed bowel wall from a case of peritonitis.)

cut, no inhibition followed the peritonitis. This was also true when he produced a chemical peritonitis by injection of an irritating solution (5 c.c. per kg. body weight of Lugol's solution). He concluded that in peritonitis inhibition of intestinal movements was produced by inhibitory influences from the spinal cord. Olivecrona¹⁸ was not able to confirm Arai's observation that section of the splanchnic nerve abolished the inhibition of intestinal movements caused by

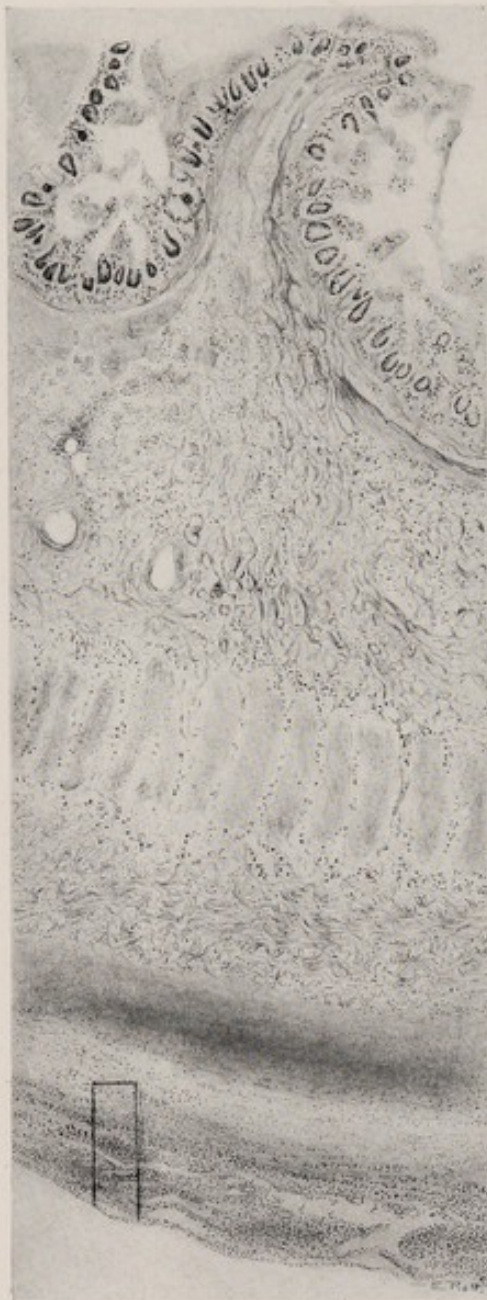


FIG. 48A.

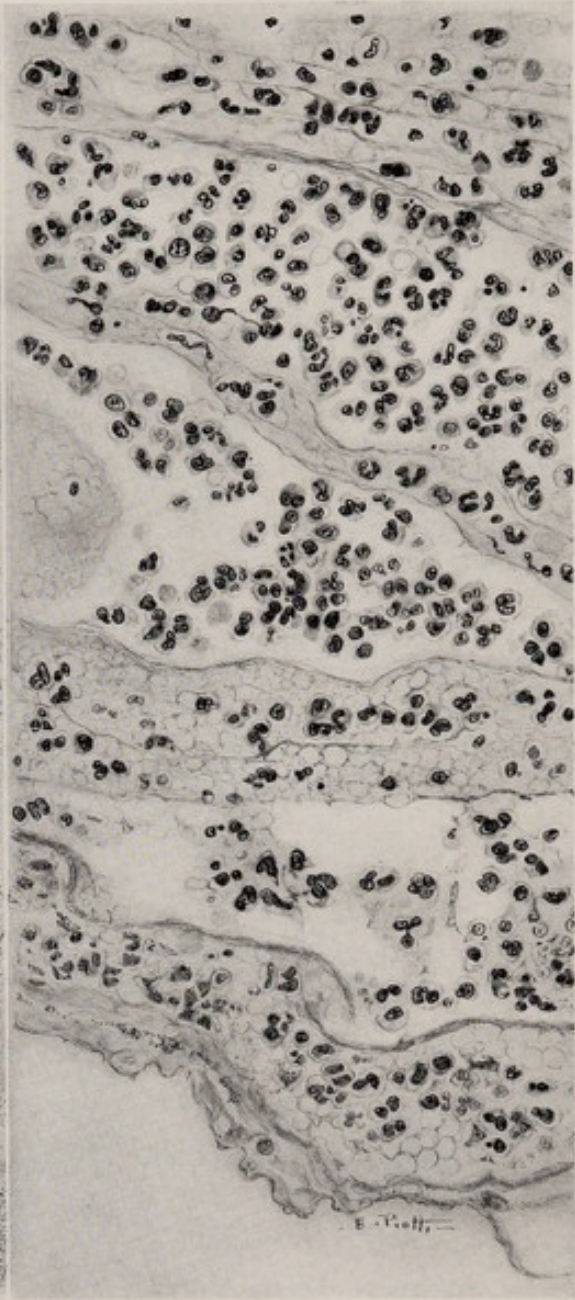


FIG. 48B.

FIG. 48. Case No. 8122, M.I.B.H. General peritonitis:

A. Section through intestine. Low power. Note exudate on serosa, and intense inflammatory reaction in bowel wall, marked degenerative reaction in muscle layers, edema, and infiltration with leucocytes. (See also Fig. 47.)

B. High power of indicated portion, Figure 48A.

peritonitis of bacterial origin. He explained the difference between his own findings and Arai's by the fact that he had produced a rapidly advancing, fatal peritonitis; and drew the conclusion that in the milder grades, paresis of intestinal movements was brought on by inhibitory influences from the spinal cord, while in the more severe forms the disturbances of intestinal motility were caused by injury to the neuromuscular mechanism in the wall, agreeing in this with Cannon and Murphy.¹⁶ But since he found that neither mechanical trauma nor peritonitis abolished rhythmic contractions, he inclined to the belief that the chief injury is sustained by the nerve elements rather than by the muscular structure. Lennander²¹ considered that the most important factor was damage to Auerbach's plexus. Supporting this view is the work of Askanazy,²² who considered that histologically he could demonstrate degenerative changes in this plexus, consisting chiefly of edema around the nerve cells. The importance of these observations of Askanazy is, however, open to question: Marchand²³ and Orth²⁴ found the same histological picture in typhoid, dysentery and other inflammatory conditions, usually unaccompanied by symptoms of obstruction; and Walbaum²⁵ pointed out that similar findings might be demonstrated in normal intestines as a result of post-mortem changes. Walbaum further pointed out that histological changes in the intestinal wall as a result of peritonitis could be demonstrated only late in the disease, while functional disturbances often occurred early. Hotz²⁶ considered that where obstruction and peritonitis were both present, the bowel was more injured by the obstruction and distention than by the inflammation.

SUMMARY. It may be said in summary that pathological processes outside the peritoneal cavity may cause inhibition of intestinal movements. These inhibitory impulses are transmitted over the splanchnic nerves; and following section of these nerves the inhibition is abolished. Functional inhibition of motility caused by intra-abdominal injury may be the result of damage to the neuromuscular mechanism in the gut wall;

or it is possible, at least in the less severe forms of peritonitis, that inhibitory impulses from the spinal cord, transmitted over the splanchnic nerves, may play a rôle (Arai²⁰). Olivecrona¹⁸ has contended that local reflexes through the celiac ganglion may also play a part. Since this author found that the inhibition of peristalsis produced by either mechanical trauma or bacterial peritonitis did not abolish rhythmic contractions, it would appear that the chief injury is sustained by nerve elements rather than by muscle; although this is not certain, and attempts to demonstrate the nature of the lesion by histological methods are not conclusive.

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CHAPTER XVI

FUNCTIONAL OBSTRUCTIONS (Continued)

VARIETIES OF FUNCTIONAL OBSTRUCTIONS

PARALYTIC TYPE. In considering the clinical aspects of the paralytic type of functional obstructions, the cases fall naturally into two groups: namely, disturbances of motility resulting from local, intra-abdominal pathology, and reflex disturbances resulting from distant lesions. These have already been discussed from the experimental point of view in the preceding chapter.

The more important disturbances among the first group are those that arise as a complication of peritonitis, those that follow operative procedures or other forms of mechanical trauma, and those occurring as a complication of mechanical obstruction. The most frequently encountered and most serious cases are those accompanying peritonitis.

Functional Obstruction from Peritonitis. Disturbances of intestinal motility may be caused by acute peritoneal infection in two ways: mechanically, by the production of adhesions; and functionally, by causing paralysis and atony of the intestinal canal as a result of injury to the neuromuscular structure of the gut, as discussed in the preceding chapter.* Frequently the obstruction is found to come about as a result of both these conditions, and there is present in the same case a mechanical and a functional element.

The following case is illustrative of the functional disturbances of motility associated with diffuse peritonitis; there was apparently no mechanical element in the obstruction:

CASE XXVII. Functional obstruction associated with peritonitis.

No. 256152, M. G. H. Male, aged fifty-three, entered the Hospital with a typical history and physical findings of acute appendicitis. He appeared to be acutely ill; temperature 101°F., white blood count 26,000.

* On the basis of the work described in the preceding chapter it must be admitted that even in cases where local damage to the gut is the outstanding factor there may at times be some reflex element in the inhibition of motility.

The patient was operated upon and a ruptured appendix with abscess formation was found. The abscess was partially walled off; although the notes state that there was considerable free fluid in the general peritoneal cavity.

Following the operation the patient had a stormy convalescence. Marked distention of the abdomen was present, and there was some discomfort from this source, but no colicky or cramp-like pains suggesting mechanical obstruction. Enemas, poultices and rectal tube were tried, but little gas was expelled and the distention was not relieved. The patient showed very little elevation of temperature (usually from 100° to 101°F.).

On the ninth day postoperative, the patient's general condition was said to be worse, the most outstanding symptom being the persistent distention. An enterostomy was decided upon, and an ileostomy, under novacaine anesthesia and without any exploratory operation, was carried out according to the Witzel method; a catheter was inserted into a distended loop of gut and the omentum was sutured around the point where the catheter emerged from the gut. There was no relief of distention following the operation; there was very little drainage from the catheter. The patient died the next day. At necropsy a general fibropurulent peritonitis was found.

Comment: Infection was undoubtedly the chief factor in this patient's death: the symptoms of obstruction were due to a generalized atonic condition of the intestinal tract and no improvement followed the drainage of a distended coil. It will be noticed in the postoperative notes that following the first operation there was no evidence of colicky pains or other signs of increased peristalsis such as is usually associated with mechanical obstruction.

The foregoing case seems to be an example of pure functional obstruction. As has already been stated, it is not uncommon in connection with peritonitis to have both a mechanical and a functional element present in the same case. This is illustrated by the following history:

CASE XXVIII. *Obstruction complicating peritonitis and having both a mechanical and a functional element.*

No. 287917, M. G. H. Female, aged thirty-one. The first operation consisted in drainage of a tubo-ovarian abscess. Following the operation the patient had a somewhat stormy convalescence, with a temperature range between 102° and 103°F. There was a profuse drainage from the abdominal wound. The culture of the pus from the wound showed a hemolytic streptococcus. Abdominal distention was persistent, but was somewhat reduced by enemas.

On the fifteenth day postoperative, the operative notes state that the abdominal distention was more marked and the patient was complaining of severe, cramp-like, colicky pains. The drainage from the wound was still profuse. It was decided that intestinal obstruction was present, and operation was advised.

Operation (under novacaine anesthesia) consisted in the insertion of a catheter into a dilated coil of small intestine. There was immediate relief of all symptoms of obstruction following the enterostomy, and the patient was eventually discharged in good condition.

Comment: In this case there was probably atony of the loops of small intestine in the region of the pelvic abscess, due to inflammation and infection. Kinking of the intestines by recent inflammatory adhesions doubtless added a mechanical element. The severe, cramp-like pains showed that there was no general atony, clearly indicating that vigorous peristalsis in the upper bowel was attempting to push the intestinal contents beyond a point of obstruction.

There was a twofold reason why at the time the enterostomy was performed it was deemed unwise to carry out any extensive exploration. In the first place, the exploration would have been likely to disseminate the peritonitis which now was localized in the region of the abscess. In the second place, it is known from experience that these obstructions coming on early after operation and caused by light adhesions combined with functional atony of certain coils do very well if the bowel above is drained, without any attempt to find the exact point of obstruction. There was no bloody fluid in the peritoneal cavity; and while this does not rule out the possibility of strangulation it makes it seem unlikely.

While undoubtedly the patient had some toxic absorption from the localized peritonitis, the outstanding feature of this case at the time of the second operation was the intestinal obstruction.

Functional Disturbance as a Complication of Mechanical Obstruction. The late stages of most cases of mechanical obstruction are complicated by functional disturbances of the motility of that portion of the bowel lying above the obstruction. It is not infrequently found that when patients are operated upon late in the disease the bowel does not regain its motility even when the mechanical difficulty has been relieved, the normal tone and peristalsis having been completely abolished by the long-continued distention and resultant injury to the capillary circulation of the intestinal wall. Perhaps, also, the toxic contents of the obstructed bowel plays

a rôle. Administration of ether to these late cases is likely to enhance this functional disturbance. The atony may not involve to an equal degree the whole intestinal tract lying above the obstruction, but may be largely confined to some particular loop immediately related to the mechanical obstruction.

Functional Disturbances after Operative Procedures. Minor degrees of functional disturbance are among the most common complications that follow abdominal operations,¹ even when no peritonitis is present; for, as shown experimentally (see preceding chapter), handling of the intestines is capable of producing inhibition of motility. This disturbance usually amounts to no more than slight distention; there are cases in the literature, however, where the symptoms have progressed to the point of real functional obstruction, exploratory operation or autopsy showing a general atonic dilatation of the intestinal canal but no mechanical obstruction. Clinically, this group is of minor importance, since practically all the true obstructions that come on early after operation have a mechanical basis or occur as a complication of peritonitis.

In connection with these functional disturbances due to local trauma, Richards, Fraser and Wallace² have pointed out that resections and anastomoses for gunshot wounds of the intestines are often followed by functional obstruction, the segment above the anastomosis becoming distended and paralyzed. This result may follow even though the operation take place relatively soon after the injury. Although localized peritonitis frequently plays a part in this picture, these authors consider that the functional element due to trauma to the gut is an important factor.

Functional Disturbances from Vascular Lesions. Functional disturbances resulting from vascular lesions, principally from mesenteric thromboses and embolism, are discussed in Chapter X, page 133.

Reflex Disturbances from Distant Pathological Processes. Functional disturbances also occur where there is no intra-abdominal lesion, the inhibition of intestinal movements being

caused by some pathological process in a distant part of the body, the peritoneal cavity itself being unaffected. The reflex mechanism which brings this about has been discussed in the preceding chapter. Clinically these cases are rare, and therefore from a practical point of view relatively unimportant; yet they are occasionally encountered, and represent such an interesting problem in the functional pathology of the gastroenterological tract that considerably more space in the literature is given to them than they deserve on the basis of their frequency.

Functional disturbances caused by distant lesions have been reported as occurring most frequently in connection with renal pathology (tumors, infections, operations or colic). They have also been observed following retroperitoneal hemorrhage or infection.³ Eisendrath⁴ stresses the intimate relationship between the splanchnic nerve supply to the kidneys and to the intestines. Tixier and Clavel⁵ have also discussed the reflex mechanism whereby pathological processes in the kidneys or retroperitoneal tissue produce serious gastrointestinal disturbances. (See Fig. 44.)

The following case illustrates the condition in which a serious infection of the kidney resulted in reflex inhibition of intestinal motility of such severity as to bring the patient to operation under the diagnosis of intestinal obstruction:

CASE XXIX. *Functional obstruction following infection of the kidney.*

No. 251029, M. G. H. Male, aged sixty-eight. Three days before admission the patient was taken with sudden, severe abdominal pain. There has been some localization of the pain in the right lumbar region; the pain has been of sufficient severity to require morphia. The bowels have not moved since the onset of the symptoms. There has been constant nausea and regurgitation of "watery material." There have been no urinary symptoms.

Physical examination showed the abdomen greatly distended and tympanitic. There was generalized tenderness, even to the slightest pressure.

The patient was operated upon under local anesthesia. The distention of the intestines was so great that an exploratory operation could not be

carried out. The cecum was found to be distended, and a tube was inserted into this organ.

The patient died five hours after operation. At necropsy the abdominal examination was negative except for great distention of all the intestinal tract. There was pyonephrosis of the left kidney, with stones.

Functional obstruction following fractured ribs has also been reported. Adams⁶ observed 2 such cases, Ralphs⁷ one. The following is a brief abstract of Adams' first case:

CASE XXX. *Functional obstruction following fractured ribs.*

Male, aged sixty-six. Three days before admission the patient fell and fractured several ribs on the right side. The affected side was strapped. The bowels had been regular before the accident; afterwards they did not move even with enemas. The patient became markedly distended, and vomited profusely.

An exploratory laparotomy was carried out, but no organic obstruction was found. The patient developed severe shock and died. Autopsy was essentially negative, except for great intestinal distention.

Adams' second case recovered following an ileostomy.

Intestinal motility is influenced by the general bodily state. Marked inhibition of intestinal motility will occasionally occur in the course of acute infections, notably pneumonias, the resultant abdominal distention being at times a serious complication. Symptoms suggestive of true intestinal obstruction may also develop at times in the course of uremic states and present a difficult problem in diagnosis. Not infrequently disturbances of intestinal motility of sufficient severity to be classed as functional obstructions are observed in the very aged and in young infants. The clinical syndrome may be that of obstruction and yet no organic cause be demonstrable at operation or at autopsy. During the ten-year period 1918-1927 at the Massachusetts General Hospital 9 such cases were admitted to the surgical wards: 6 of these were patients over seventy years of age, 2 were infants under one month. Probably these functional disturbances of intestinal motility were due to the poor constitutional state of the patients; they may be difficult to distinguish from mechanical obstructions. The following histories illustrate these conditions:

CASE XXXI. *Functional obstruction in an aged person.*

No. 254687, M. G. H. Male, aged seventy-three. For the past two weeks the patient had felt poorly, but had worked until thirty-three hours before admission to the hospital. At that time he was taken with abdominal pain and vomiting. The vomitus was said to be dark in color. He had vomited a number of times since the onset of the attack; there had been one bowel movement during that time. During most of this time the patient had been troubled by hiccough.

On physical examination the abdomen was found greatly distended and tympanitic throughout. It was tender on palpation.

A diagnosis of intestinal obstruction was made and the abdomen was explored under ether anesthesia. There was no free fluid in the peritoneal cavity. The intestines were generally distended and of dark color due to interference with the circulation by distention. No cause for the obstruction could be found. During the closure of the abdomen the patient vomited and aspirated a considerable quantity of vomitus, and died immediately.

Autopsy was carried out, but no organic cause for the obstruction was found.

Comment: There is not sufficient data at hand to give an adequate explanation of the functional obstruction in this aged patient. The danger of aspiration of vomitus while under a general anesthetic and the methods of avoiding this complication are discussed in a later chapter.

CASE XXXII. *Functional obstruction in an infant.*

No. 270780, M. G. H. An infant, five weeks old. The baby had been losing weight and vomiting for four days. During this time the bowels had moved only twice and in very small amount. The baby had cried out constantly and been very restless. An enema had failed to produce either gas or feces.

General physical examination was negative except for an extreme degree of emaciation and marked abdominal distention. The abdomen was tympanitic all over. The most probable diagnosis seemed to be intestinal obstruction, and an enterostomy was decided upon.

Under local anesthesia a catheter was inserted into a distended loop of small intestine. The baby died four days later. Autopsy was essentially negative, except for distention of the small intestine, and emaciation.

SPASTIC OCCLUSIONS. So far, only those functional obstructions of the bowel brought about by intestinal atony have been considered; there are also obstructions caused by localized spasms of the intestinal musculature. Peristalsis is a complicated process, depending upon a high degree of coördination of the different parts in order to bring about a purposeful

movement. If there exist areas of localized spasm that are out of time with the wave of peristalsis, the progress of the contents is blocked. While moderate degrees of intestinal spasm* are probably very common, contractions of sufficient intensity and duration to bring about the symptoms of true obstruction are rare. Authentic cases, however, have been reported, much more attention having been paid to the condition in the German literature than elsewhere.

Our knowledge as to either the frequency or the mechanism of this type of disturbance is vague. Nagel⁸ reported 2 cases from his clinic, together with 49 collected from the literature. The spasms have been reported as occurring under diverse circumstances; a number have occurred in neurasthenic or hysterical individuals, who seem particularly predisposed to these spasms of intestinal musculature. The condition may be recurrent: Jacobsen⁹ operated on the same patient twice during one hospital admission. Most of the cases have followed abdominal operations on the stomach, intestines or pelvic organs (Green et al.,¹⁰ Meyer,¹¹ Nagel,⁸ and Steindl¹²). In 1925, Reimer¹³ reported 3 cases where obstruction due to localized spasm followed intraperitoneal hemorrhage. F. Colmers¹⁴ reports 3 cases in which spastic obstruction came on in association with "grippe," the symptoms being so severe that the patients were operated upon and nothing but spasmodic contractions of the small or large intestine were found; 2 of these cases afterwards showed signs of disturbance of the central nervous system.

The views expressed by most of the authors as to the etiology are not conclusive, and the evidence on which they base their speculations is vague. Steindl¹² says that spastic occlusion can come about in several ways: as the result of injury to the peritoneal nerve plexuses; from causes directly affecting the intestinal tract; from irritation of intestinal contents; as a result of hysteria; and from causes unknown.

* The gas pains after operation probably represent, at least in part, localized spasm of the gut.

This author thinks that considerable importance should be attached to changes in the autonomic nervous system. He reports the finding at autopsy of changes in the medulla in the region of the vagus center, which he relates to the intestinal spasm. These changes, due to narcosis or absorption of toxins, consisted of perivascular infiltration and lymphocytosis. Reimer¹³ considered that changes in the central nervous system are important. Riess¹⁵ considers that spasm of the intestinal musculature is the important factor.

Local injury to the muscle or nerve plexuses is very probably an important factor in the production of the spasm around foreign bodies in the intestinal lumen, the spasm converting an incomplete blockage by the foreign body into a complete obstruction. For example, there is good reason for believing that a spasmodic contraction often occurs around a gallstone that has lodged in the intestinal tract and that might have passed through without producing an obstruction had it not been for the tonic contraction incited by its presence. Tonic contractions have been reported around collections of intestinal parasites, particularly ascarides, of sufficient persistence to produce the syndrome of obstruction. The spasm in this instance is supposedly due to a toxin secreted by the worms (see p. 104).

The pains of lead colic are probably associated with areas of local spasm and at times may be of sufficient severity to present a problem in differential diagnosis. Murphy's¹⁶ case in which an operation was performed in a case of lead colic under the diagnosis of intestinal obstruction is illuminating; an actual area of spasm of the intestinal musculature was found. His article carries an illustration of this condition.

SUMMARY. The most important of the functional obstructions are those associated with peritonitis; in these cases there is frequently present both a mechanical and a functional element.

Functional obstructions may also occur as a complication of mechanical obstruction and as the result of operative

trauma. Reflex disturbances from distant pathological processes, especially renal, may occur; the symptoms may also arise in the course of various diseases and debilitated states.

The etiology of functional obstructions due to intestinal spasm is, for the most part, not clear: the spasms have been attributed to changes in the autonomic nervous system; local injury and irritation may at times be a factor. Areas of localized spasm may be produced by certain poisons, notably lead.

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CHAPTER XVII

FUNCTIONAL OBSTRUCTIONS (Continued)

PREVENTION AND TREATMENT

Occasionally, under any regime, patients suffering from acute peritonitis or convalescing from abdominal operations, will develop symptoms of functional obstruction; inhibition of intestinal motility, abdominal distention and vomiting may assume serious proportions, particularly if the peritonitis is extensive. There are, however, certain principles of treatment that minimize the danger of this grave and distressing complication, and tend to establish a smooth convalescence. The rigor with which the measures are applied is to be governed by the seriousness of the situation.

ADMINISTRATION OF FLUIDS. In peritonitis of any extent, fibrinous adhesions glue together the coils of intestines, and the downward passage of gas and fluid from the stomach increases the intestinal distention and may thus produce obstructive kinks; the distention itself also diminishes the tone of the bowel and interferes with its circulation. Great care must therefore be taken that the stomach should not become distended with fluid and gas. In the more serious cases this means limiting the fluid intake by mouth to sips of water; or even, particularly if the patient is vomiting, prohibiting entirely the fluid intake by mouth. Thirst may be alleviated by rinsing the mouth and by keeping wet applications on the lips. It should be recalled that when liquids are swallowed, air also is carried into the stomach; and the work of McIver, et al.¹ showed that atmospheric air taken into the stomach by swallowing or retching is often passed down into the intestine and may be an important factor in the gaseous distention accompanying peritonitis. The fluid intake must, of course, be adequately provided for, and may be maintained by the administration of normal saline by rectum, subcutaneously or by intravenous injection: dehydration must be prevented if

possible, or combatted if it occurs (see Treatment, p. 254). Glucose is frequently added to the salt solution for intravenous injection, but it should be noted that hypertonic glucose may exert an inhibiting effect on intestinal activity,* which may, however, be abolished if insulin is added to the infusion.²

USE OF DUODENAL TUBE. If, in spite of the limitation of fluids by mouth, there is any suspicion that the stomach is dilated, a nasal catheter should be passed and the fluid and gas drawn off. At times it may be desirable to establish constant drainage from the stomach and intestines by leaving the tube in place for twenty-four hours or longer. The use of the duodenal tube in the management of a patient during a stormy convalescence after abdominal operation constitutes one of the major advances of the past ten years. Even in the less severe cases, the prevention of postoperative distention by this method often contributes a great deal to the patient's comfort and well-being. For further discussion of this subject and technique, see pages 180 and 259.

ADMINISTRATION OF MORPHINE. In those cases where peritonitis is found at operation, or where excessive trauma to the abdominal viscera is caused by operation, I believe that the liberal use of morphine is a most valuable prophylaxis against postoperative distention and functional obstruction. In certain cases of peritonitis, 10 mg. ($\frac{1}{6}$ gr.) of morphine every three hours may not be excessive, although the respiratory rate should be carefully watched and if it falls below 16 per minute the quantity or frequency of the dose should be reduced.

I realize that it is a paradox to recommend for atony of the intestine a drug that is commonly supposed to inhibit intestinal peristalsis. The most recent work, however, has clarified the situation. Plant and Miller³⁻⁵ in 1926 and 1928 showed, in both humans and dogs, that morphine, in the doses ordinarily

* Ochsner et al. have found that while injections of glucose of 10 per cent or stronger exert this inhibiting effect, solutions approaching isotonicity do not. (Personal communication.)

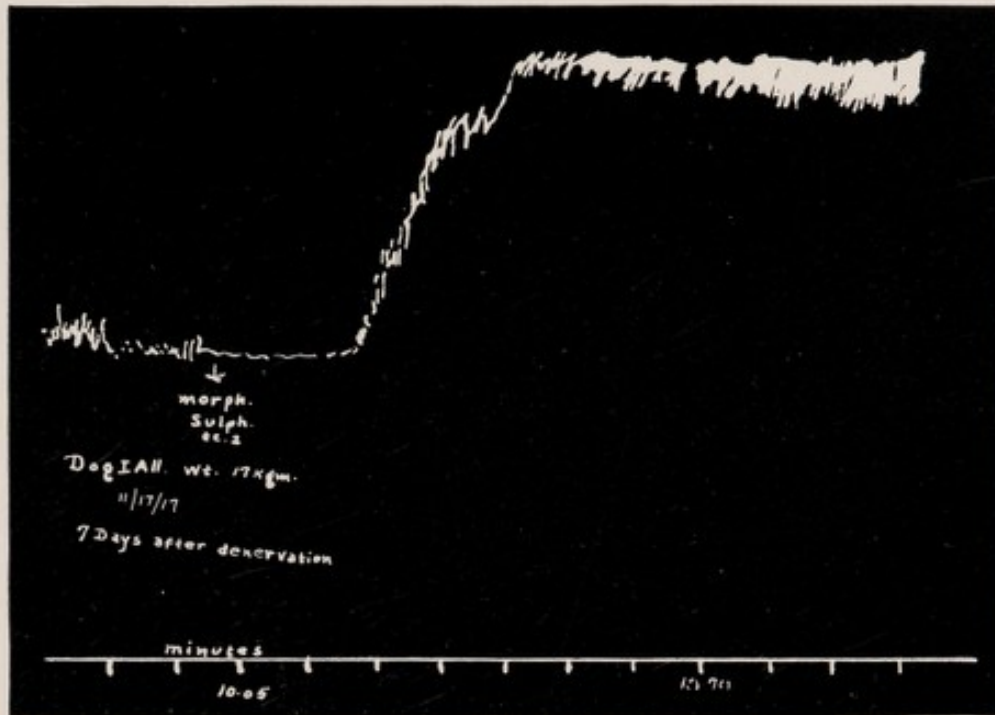


FIG. 49A.

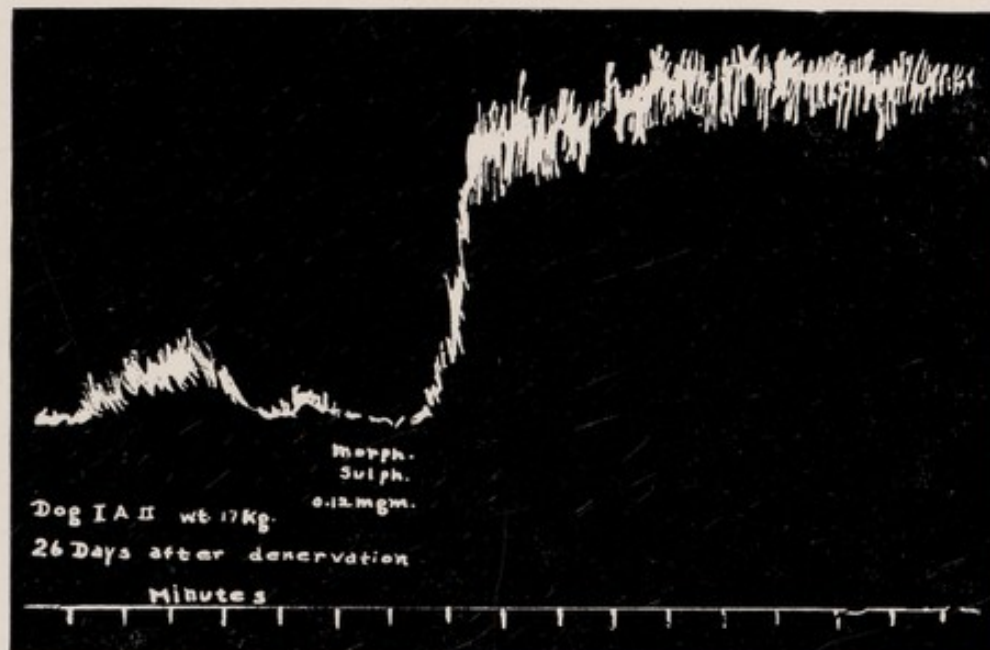


FIG. 49B.

FIG. 49. Effect of morphine on small intestine, showing marked increase in tone produced by drug. This tracing suggests that point of action of morphine is in neuromuscular mechanism in gut wall, since extrinsic nerve supply had been cut. (Denervated Thiry-Vella fistula.) Fast kymograph; time trace-minute intervals. A. Effect of 0.12 mg. morphine sulphate per kg. seven days after denervation. B. Effect of 0.12 mg. morphine sulphate per kg. twenty-six days after denervation. (Plant and Miller²)

used, increased the tone, and to some extent, the peristaltic activity of the small intestine and colon. (See Figs. 49 and 50.) In the case of the colon, the most pronounced effect was a

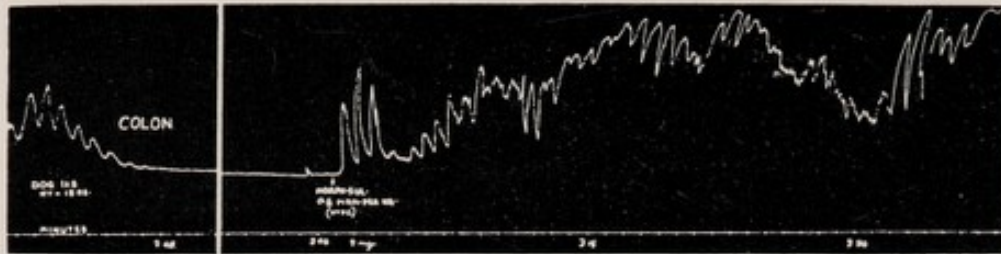


FIG. 50. Effect of morphine on colon, drug causing both an increase in muscle tone and in peristaltic activity. (Plant and Miller⁴)

marked increase in muscle tone, which might last for several hours after the administration of 10 mg. of morphine. In contrast with its effect on the intestine, morphine caused a decrease in the muscle tone of the stomach wall. These studies by Plant and Miller were reviewed in the *Journal of the American Medical Association*, but they did not appear in a surgical journal. Those who are accustomed to think of the action of morphine on the intestinal tract as purely inhibitory, would do well to look up this work; it is quite convincing, and has been confirmed by a number of observers.⁶⁻¹¹ Paine et al.¹² felt that clinically, in the group of cases they studied, morphine did not influence distention. I have repeatedly, however, seen patients with extensive peritonitis, after receiving repeated doses of morphine, pass considerable flatus within twelve to twenty-four hours after operation; and the use of the drug definitely appeared to prevent the appearance of serious distention and lessen the likelihood of obstructive symptoms.*

* Historically the use of morphine in intestinal obstructions and peritonitis is of considerable interest. Before the days of abdominal surgery the early writers were enthusiastic over the use of morphine in intestinal obstructions (Brinton,¹³ Leichtenstern,¹⁴ Thomas,¹⁵ and Nothnagel,¹⁶) and they seemed undoubtedly to have often prolonged life, and at times, perhaps, effected a cure by the use of the drug. Their cases must have represented all types of obstruction, both organic and functional. Fifty years ago Alonzo Clark¹⁷ was a strong advocate of large doses of morphine in peritonitis.

In my article on postoperative distention published in 1926,¹ I was not familiar with this work of Plant and Miller, and gave the commonly accepted explanation of the favorable effect of morphine in peritonitis, namely, that it quieted the intestinal movements, and prevented the spread of the infection, "splinting the intestines." I now believe that its action in increasing the tone of the small intestine and colon, and so tending to prevent distention, is one of the most important functions served by the drug. In regard to its use, it should be especially borne in mind that while morphine stimulates the tone of the lower intestinal tract it diminishes the tone of the stomach; so that during its use it is important to limit the intake of nourishment by mouth and to employ the nasal catheter at the slightest suspicion of dilatation of the stomach.

Is there danger of masking the symptoms of true mechanical obstruction arising after operation by the use of morphine? Where the drug is used intelligently, under the supervision of an experienced surgeon, I do not believe that the danger is great. The peristaltic movements above a point of obstruction are not abolished by the administration of reasonable doses of opiates.^{9,18} It is true that if used in too large doses it will minimize the symptom of pain; but if colicky pain is a striking symptom, of course care would be used in continuing the drug. Persistent vomiting, distention and evidence of hyperperistalsis on inspection or auscultation indicate the need for operative interference (see value of enterostomy in peritonitis, p. 292).

USE OF CATHARTICS. The use of cathartics is contraindicated in the presence of any obstructive symptoms. If there is any mechanical element in the obstruction, forcing the bowel contents downward is likely to convert a partial into a complete obstruction. Other dangers in the use of purgatives in the presence of obstructive symptoms were forcibly stated by Barlow¹⁹ about 100 years ago.*

* Barlow writes¹⁹: "But I believe that it is not so generally considered that active purgatives may produce serious mischief, even when they do *not* invert the action of the tube, and when they *do* reach the seat of the obstruction; whereas it is evident that

STIMULATION OF PERISTALSIS BY DRUGS. Drugs such as pituitrin and eserin and their derivatives, that have a tendency to set up violent types of peristalsis and to cause localized spasms of the intestine, should in general be avoided: they are often either totally ineffective, or if they set up temporary peristalsis the later effect may be a more complete relaxation of the intestinal musculature.^{1,16,20} As with most general statements, however, there are exceptions to the rule: in certain selected cases these drugs may occasionally be useful. Hypertonic salt solutions for stimulation of peristalsis might also, as suggested by Hughson and Scarff²¹ and Ochsner et al.,²² be considered in selected cases.

ENEMAS. An occasional low enema is useful in getting rid of accumulations of gas in the colon. A free movement of the bowels is not so important, provided the patient is passing flatus and not becoming distended¹; and a too frequent use of enemas is to be avoided, particularly if they are not being freely expelled. An important function of the colon is to dehydrate the feces, and if the colon is being constantly filled with watery solutions that are not freely expelled, it seems probable that fermentation is increased. Small enemas containing glycerine and magnesium sulphate frequently give satisfactory results.*

APPLICATIONS OF HEAT TO THE ABDOMEN. Flaxseed poultices on the abdomen, combined with a rectal tube and a dose of morphine, are often an exceedingly effective and harmless method of getting rid of flatus and reducing distention.

POSITION OF THE PATIENT. If peritonitis is present, Fowler's position seems useful in localizing the infection in the lower abdomen and diminishing toxic absorption.

SUMMARY. In summary, it may be said that symptoms of functional obstruction are best prevented by measures tending

in the case of George J— any forcible effort to propel the contents of the bowel beyond the seat of obstruction must have been productive of much irritation immediately above it, and have increased the risk of laceration."

* A formula that has proved useful is as follows: Saturated solution of magnesium sulphate 60 c.c. (2 oz.), glycerine 120 c.c. (4 oz.), and water to 240 c.c. (8 oz.).

to avoid or relieve distention: limitation of the fluid intake by mouth; liberal administration of morphine, which increases the tone of the small intestine and colon; the use of flaxseed poultices, rectal tube, and occasional enemas. All these measures should be supplemented, as indicated, by the use of the duodenal tube. Cathartics should be avoided; and, in general, the use of pituitrin and eserine likewise.

In connection with the management of cases showing symptoms of functional obstruction, the sections on the treatment of early postoperative obstruction and on the value of enterostomy in peritonitis should be consulted.

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PART II
DIAGNOSIS AND TREATMENT



CHAPTER XVIII

DIAGNOSIS

GENERAL CONSIDERATIONS

In approaching the question of the diagnosis of acute intestinal obstruction, it should be clearly borne in mind that if the life of the patient is to be saved, diagnosis must be made early in the disease. The terms "early" and "late" in the disease have a different significance according to their application to the fulminating or milder varieties of obstruction. Where strangulation is present, the lapse of twelve hours or less may be sufficient for irreparable damage to the involved intestine and for the development of serious systemic effects; while in simple obstruction of the colon several days may go by without the development of serious local or general effects. There are many variations between these two extremes. As pointed out by Eisberg,¹ even in strangulations there are considerable differences in the clinical course of the disease, depending on the length of loop involved and on whether the blood supply is suddenly or gradually occluded.

Diagnosis in the early stages of the disease is often difficult. This is demonstrated by the fact that although the symptom complex and major features of the disease have been taught to five or six generations of medical students and have been stressed at medical and surgical meetings for the past two or three decades, the patients continue to be brought to operation late and the mortality continues high. Modern laboratory diagnostic methods in general are not very helpful in arriving at a diagnosis of acute intestinal obstruction. Thus the diagnosis must often rest on the old clinical basis of signs and symptoms; and yet the physical signs in the early stages of the disease may not be very constant. Muscle spasm and tenderness may be absent; visible peristalsis can be seen only occasionally; abdominal distention may be absent or slight; the temperature and white blood count may be normal;

and while the cardinal symptoms of abdominal pain, vomiting and obstipation are usually all present, they vary in intensity and prominence and are, of course, also associated with other diseases which do not demand operative interference. As a practical matter the real difficulty may be, not in diagnosing the presence or absence of obstruction, but in distinguishing between organic and functional obstruction. The two types may have many basic signs and symptoms in common, yet the former (organic) demands operative interference, the latter only occasionally does so. The distinction is of particular importance in obstruction occurring during the convalescence from an abdominal operation, especially when peritonitis complicates the picture.

STUDY OF THE PATIENT

An intestinal obstruction may come on suddenly, with the patient in perfect health; or there may be a history of chronic obstruction before the acute attack. The symptoms are frequently severe from the beginning, although sometimes the onset is insidious. Where the pain is very intense, the patient may show some signs of prostration early in the disease; but usually this is not the case; the presence of this symptom early in the disease points to strangulation.^{2,3} If the pain is extreme, the pulse-rate may be elevated; it usually, however, remains fairly normal until the late stages of the disease and then becomes rapid and thready.⁴ The temperature is usually normal, or only slightly elevated. The patient frequently has a rather characteristic facial expression that might be described as one of anxiety. If there has been much loss of fluid by vomiting, the usual signs of dehydration (sunken eyes, dry tongue, depressed fontanelles in infants, and loose, inelastic skin) are present. The amount of urine passed is likely to be small, its volume furnishing some indication as to the extent of the dehydration.

In elderly patients, abdominal distention, the peristaltic type of colic, and vomiting, suggest the possible diagnosis

of obstruction from neoplasm of the colon. In these patients a past history can not infrequently be obtained of progressive constipation, bloody mucus in the stools, or frequent small movements, which tends to confirm the first impression. In infancy and early childhood, severe abdominal colic associated with a lack of bowel movements should suggest the possibility of intussusception; if bloody mucus is passed, the suspicion is greatly strengthened. Intussusception is outstandingly the chief cause of obstruction in infancy and early childhood, as is shown by the figures from children's clinics: Ladd and Cutler⁵ reported 105 cases of obstruction in infants and children, of which 88 were caused by intussusception, and 17 by other conditions; Peterson⁶ reported 55 cases of intestinal obstruction in the same age group, intussusception being responsible in 46 instances.

The presence of an abdominal scar in a patient with symptoms consistent with an acute obstruction should at least put the physician on his guard. Since abdominal laparotomies have become more common, postoperative intestinal obstruction is more frequently encountered.⁷⁻⁹ This increase is shown in the following figures from the Massachusetts General Hospital: in the ten-year period 1898-1907 there were 37 cases of obstruction following early or late after abdominal operations; in the period 1908-1917, there were 57; in 1918-1927, there were 82. In many of the late postoperative obstructions the element of strangulation is present; see page 43.

In studying the patient suspected of having acute intestinal obstruction, particularly detailed information is needed on certain points, which are summarized below. Since, however, the suspected "intestinal obstruction" may prove actually to be lead colic, an abdominal crisis of tabes, or some other condition equally removed, a careful history that is not limited to the presenting symptoms, and a thorough and complete physical examination are required.

PRESENT ILLNESS

Abdominal pain: Location; duration; intensity; character (has it been cramp-like, "doubling-up"?). Periods of partial or complete remission? Diminishing or increasing in severity? Radiation?

Vomiting: Time of onset; frequency; character (accompanied by nausea? projectile?). Amount; appearance of vomitus (color and odor).

Bowel movements: Number since onset of illness; time of last movement; its character; did it contain blood or mucus?

Has gas been passed by rectum? How frequently? Have cathartics been given? What result? Have enemata been given? How many? What results?

Urine: Amount passed; has it been highly colored?

Distention: When first noticed? Has it been more severe than at present?

Medication: What kind? Morphia? Cathartics?

Diet: Solid food? Fluids? Retained?

PAST HISTORY

Previous abdominal operations: Date on which they occurred; character; was wound drained?

Question as to *existence of any hernia.*

Has patient had any *symptoms in the past* suggesting partial or complete obstruction, such as abdominal pain, vomiting, distention, excessive or unusual gurgling peristaltic sounds, constipation or diarrhea?

PHYSICAL EXAMINATION

General condition: Does patient appear acutely ill? Signs of collapse? Does he appear dehydrated (drawn features, sunken eyes, dryness of tongue)?

Abdomen: Flat? Distended? Uniformly so? Visible peristalsis? Tenderness, spasm or masses on palpation? Tympanitic; areas of dullness or flatness, or fluid wave on percussion? Peristaltic activity on auscultation normal? Increased? Absent?

Rectal examination: Masses? Tenderness?

LABORATORY STUDIES

Blood: Count of white blood cells, including differential count; non-protein nitrogen; blood chlorides.*

Urine: Usual examination.

X-ray: Examination (no barium by mouth).

There are few diseases in which the classical signs and symptoms are all present in any individual case; and acute obstruction forms no exception to this rule. Pain, vomiting and obstipation are, however, so commonly encountered that they must receive special consideration.

* Not of much value in differential diagnosis; see page 232.

CLINICAL ANALYSIS OF SYMPTOMS

PAIN. Acute obstruction is usually ushered in by abdominal pain. This cardinal symptom is almost always present, although it varies considerably in character, severity and location. With simple obstructions of the small intestine it is colicky or cramp-like early in the disease and has a tendency to be intermittent, working up to a climax and then subsiding. After twelve to twenty-four hours the patient insists that the pain is constant, not coinciding with the waves of peristalsis which in some cases may be seen or heard at intervals. In obstructions of the large intestine the colicky character of the pain lasts longer than it does in obstructions of the small gut. Where strangulation exists, the infiltrated, distended loop of intestine causes pain that is steady and agonizing from the beginning, rather than colicky in character, and which may be of sufficient severity to bring on a state of collapse. In cases where a chronic obstruction has been gradually converted into an acute obstruction, however, the pain may not be severe; and in certain types of obstruction (notably post-operative obstructions following serious operative procedures, or where peritonitis complicates the picture) pain may be absent, or if present may not be the most outstanding feature.

The location of the pain in obstructions of the small intestine is likely to be the region of the umbilicus or epigastrium. If the obstruction is situated in the colon, the pain is likely at first to extend across the lower abdomen, although in the early stages it may at times be referred to the epigastrium; when the obstruction has existed for some time, the pain is often localized over the site of the obstruction, probably because of edema and infection. If there is a strangulated loop of gut the pain may also be referred to the region overlying it. At times the patient is unable to localize the pain, passing his hands all over the abdomen when asked to show where it is.

VOMITING. Vomiting is present with the rarest exception and usually occurs very promptly after the onset of the obstruction. The amount of vomiting is likely to vary with the level of the obstruction and the stage of the disease. In general the vomiting is more frequent and profuse in high obstructions, particularly in the early stages of the disease; in the later stages it may take place frequently and in large amounts regardless of the level of the obstruction. In the later stages it may be observed that the patient goes for a number of hours without vomiting, and then, suddenly and with little vomiting effort, regurgitates a large amount of foul-smelling fluid from a dilated stomach. In these cases the stomach-tube is most useful in determining the true state of affairs.

The vomitus from obstruction presents no characteristic features in the early stages of the disease, consisting either of food recently eaten or of clear or bile-stained gastric juice. Later in the disease the vomitus does have characteristic and distinct qualities: it is a thin, yellowish fluid containing small whitish particles that settle out on standing; and the characteristic odor is foul in the extreme. This fluid represents the contents of the small intestine, although, of course, not the normal contents: it is a mixture of the secretions of the digestive glands and of the intestinal tract, which are poured out in great quantities under conditions of obstruction, and it is heavily infected with bacteria. It is this material which is usually described as "fecal" or "stercoraceous," receiving its name from the fecal-like odor imparted by the action of the colon bacillus and other putrefactive bacteria; it does not contain any actual feces, since true vomiting of feces is found only where there is a fistulous communication between the stomach and the colon.

Between the onset of the obstruction and the appearance of the characteristic vomitus, a time interval elapses, which in certain types of obstruction may be as long as several days; so that while inspection of the vomitus may confirm the diagnosis,

it may also indicate that the most favorable time for operation has passed.

OBSTIPATION. Since complete intestinal obstruction necessarily stops the intestinal stream, it might be supposed that cessation of bowel movements and failure to obtain them after appropriate stimulation would be pathognomonic of the disease. The situation, however, is not quite so simple. If the patient is seen early after the onset of the attack, the fact that the bowels have not moved even with the use of enemas may not be conclusive evidence of obstruction: obstinate constipation and reflex inhibition of intestinal motility are findings not at all uncommonly associated with other conditions in which abdominal pain occurs, as, for example, renal colic. On the other hand, even though the bowels have moved after the onset of the pain, this is not conclusive evidence that obstruction does not exist. Failure to obtain even gas on the administration of subsequent enemas, however, is a finding that cannot be disregarded; and in conjunction with abdominal pain should suggest to the attending physician the necessity of at least placing the patient under surgical observation.¹⁰

In attempting to decide whether a partial obstruction has become complete, the question as to whether or not flatus has been passed is particularly important, since the patient with complete obstruction fails to expel even gas after the administration of an enema. If, however, vomiting and distention persist, and particularly if the character of the vomitus suggests intestinal contents, one should not be led into delaying operation too long because a little gas is being obtained on administration of an enema.

PHYSICAL SIGNS

The physical signs of intestinal obstruction may be pronounced in some cases, in others absent, or slight.

DISTENTION. The most consistent physical finding is abdominal distention. Since it takes an appreciable length of time for fluid and gas to accumulate in sufficient quantities

to produce a demonstrable distention of the abdomen, this is not an early symptom unless there has been some chronic obstruction before the onset of the acute attack.

Distention does not always occur; it may be absent, or slight, when the obstruction is situated at a high level in the small intestine; and in certain cases of internal strangulation the whole course of the disease is so short and severe that there is not time for noticeable distention to occur. If, however, a case is seen late in the course of the disease, or if it has been improperly treated, the distention may be very great. It is seen in its most extreme degree where the obstruction is in the left side of the colon,⁷ particularly where an acute obstruction is superimposed upon a chronic obstruction; and is also likely to be found in its severer forms where in addition to an organic obstruction there is a functional element introduced by peritonitis.

MUSCLE SPASM AND TENDERNESS. The presence or absence of tenderness depends largely upon the type and duration of the obstruction. In strangulations, tenderness over the involved loop of bowel is frequently found early in the disease, particularly if the loop lies in contact with the abdominal wall. In cases of simple obstruction, tenderness and muscle spasm (abdominal muscles) are often absent early in the illness; usually, however, the abdomen is held rather tightly and some generalized tenderness is often present.* Codman¹² considers that the feeling of increased intra-abdominal tension obtained on palpation is quite characteristic. Later in the obstruction tenderness is present over the edematous, infiltrated loop of obstructed gut. In obstruction of the colon there is likely to be tenderness especially over the cecum.

In a recent series of 188 cases of intestinal obstruction¹³ (obstructions by strangulated external hernia excluded) abdominal tenderness on palpation was recorded in 72 instances, muscle spasm in 41.

* According to Dr. D. F. Jones¹¹ the degree of tenderness also depends much upon whether or not the bowel is in spasm; when it is contracted the tenderness over it is marked.

TUMORS. Occasionally it may be possible by palpation or percussion to detect a distended loop of gut (Wahl's sign); this finding, however, is infrequent.

In cases of intussusception it is fairly common in infants to be able to palpate the characteristic "sausage-shaped" tumor in the region of the cecum or ascending colon, or on the left side in the region of the descending colon or sigmoid. In the recent Massachusetts General Hospital series, out of 11 cases in infants, a mass could be palpated in 8 instances. This is in contrast with the group of older patients with intussusception, where a mass could be palpated in only one out of 6 cases. On rectal examination it may be possible to palpate the apex of an intussusception. As has been stated before, the apex of the intussusception may protrude at the anus.

VISIBLE PERISTALSIS. Visible peristalsis can occasionally be made out above the point of obstruction. It was recorded in 27 of the 188 cases (obstructions from all causes except external strangulated hernia) recently reported by McIver.¹³ It occurred with the greatest frequency in the early and late postoperative obstructions. Some of the most extreme examples are encountered not in complete but in partial obstructions. When strangulation is present, peristalsis is not often visible.

Auscultation of the abdomen may yield evidence of increased peristaltic activity even when none is visible on inspection. In attempting to determine the existence of mechanical obstruction when peritonitis is present, the absence of any sounds of peristaltic activity is strongly suggestive that the symptoms of obstruction may be due to a paralytic distention of the intestines.

CLINICAL INDICATIONS AS TO THE LEVEL OF THE OBSTRUCTION

One is able not infrequently to draw inferences as to the level of the obstruction on the basis of the clinical picture presented. If there is no element of strangulation, the low obstructions run a milder course than the high obstructions.

In high blockage, the profuse, characteristic vomitus of obstruction comes on quickly and is an outstanding feature, so that these patients very rapidly show evidence of considerable dehydration; distention takes longer to develop and, indeed, may be slight even when the disease is advanced; the course of the disease is rapid and unless relief is obtained death is a matter of a few days.

Simple obstructions of the colon, on the other hand, may require a number of days before the disease reaches its maximum intensity. Vomiting is not so profuse in the early stages, and evidences of dehydration appear more slowly. Distention usually assumes marked proportions.

The clinical picture presented by patients with obstruction of the ileum usually lies somewhere between that presented by the high jejunal obstruction on the one hand, and obstructions of the colon on the other. In general it may be said that in simple obstructions the symptoms become less fulminating as one approaches the ileocecal sphincter.

LABORATORY STUDIES

As already stated, laboratory examinations do not usually furnish a great deal of aid in establishing the diagnosis of acute intestinal obstruction. The blood chemistry findings early in the disease may be completely normal. So much has been written within the past decade on the low blood chlorides found in obstruction, that the impression may exist that this is a helpful diagnostic point. As a matter of fact, such is rarely the case. In the first place, since the low blood chlorides are the result of extensive vomiting, this finding is not present at the onset of the illness in any type of intestinal obstruction; and in the most common and fulminating types, in which strangulation is present, lowered blood chlorides may not be found at any stage of the disease. Furthermore, profuse vomiting occurs in connection with a number of diseases other than intestinal obstruction, and if it has been of sufficient amount will, of course, result in a lowering of the blood

chlorides*; so that however helpful the laboratory finding of lowered blood chlorides may be from the point of view of indicating the presence of dehydration and the need for adequate administration of salt solution, it is only rarely that this finding is helpful in establishing the diagnosis. The same thing applies, with even more force, to an elevation of the non-protein nitrogen of the blood: it is true that after intestinal obstruction has become established the non-protein nitrogen is usually elevated; but, obviously, this finding occurs in a number of other diseases also.

The white blood count is frequently normal, especially in the early stages of simple obstruction. Where a strangulation is present the white count is likely to be high,¹⁴ emphasizing the need for prompt operation.

X-RAY EXAMINATION

X-ray examination has a definite field in the diagnosis of acute intestinal obstruction; it is important, however, that the findings be interpreted in the light of the clinical picture, for otherwise grave errors can arise.

The administration of opaque substances by mouth for the purpose of determining the existence of an intestinal obstruction is highly dangerous, for it may convert a partial into a complete obstruction (see p. 105). Roentgenograms of the abdomen taken without the administration of any opaque medium, however, may be quickly and easily made without undue disturbance of the patient and often furnish valuable evidence as to the presence or absence of obstruction and as to its level, particularly with reference to its location in the large or small intestine. The detection of multiple fluid levels, which can at times be visualized in the obstructed intestine, is a particularly important point in the diagnosis by x-ray.¹⁵⁻¹⁸ To bring these out the roentgenogram must be taken either with the patient in the upright position (sitting or standing),

* The digestive secretions may also be lost as a result of a severe diarrhea, and, in children particularly, a lowering of the blood chlorides may result from this condition.

the picture being taken antero-posteriorly; or with the patient lying on his back, the picture being taken laterally (see Figs. 51 and 52). In obstructions of the small intestine the accumulations of gas may give the outline described by Case¹⁵ as the "herring-bone" appearance; or at times a "ladder" arrangement of the coils may be observed. The diameter of the dilated coils should be carefully noted; also the presence or absence of gas in the colon. In determining the level of an obstruction in the small intestine, Swenson and Hibbard¹⁹ consider that the relatively abrupt disappearance of the striae in the shadow of the distended jejunum as it approaches its junction with the ileum, is an important point. On the basis of experimental studies these authors believe that accumulations of gas can be demonstrated in the intestine three or four hours after the onset of obstruction, whether mechanical or functional in origin, and that the fluid levels can be demonstrated three or four hours later.

An *x*-ray picture of the abdomen, taken without opaque medium, has been found by Ginzburg²⁰ particularly helpful in making a differential diagnosis where obstructive symptoms have appeared in a patient who has had a previous abdominal operation and where the diagnosis might well, therefore, be obstruction by bands and adhesions. If in these patients the *x*-ray shows dilated loops of small intestine and absence of gas in the colon, a diagnosis of mechanical obstruction connected with the previous operation is emphatically suggested; if, on the contrary, gas can be demonstrated in the colon, this diagnosis must be strongly questioned. In Ginzburg's series, ten patients who had previously undergone abdominal operation and who entered the clinic with symptoms suggesting obstruction, were later shown to have such diseases as neurosyphilis, uremia, coronary arterial disease, renal colic, and so forth; the *x*-ray was useful in ruling out the question of obstruction.

On the basis of experimental work, Hibbard, Swenson and Levine²¹ believe that in the diagnosis of occlusion of the

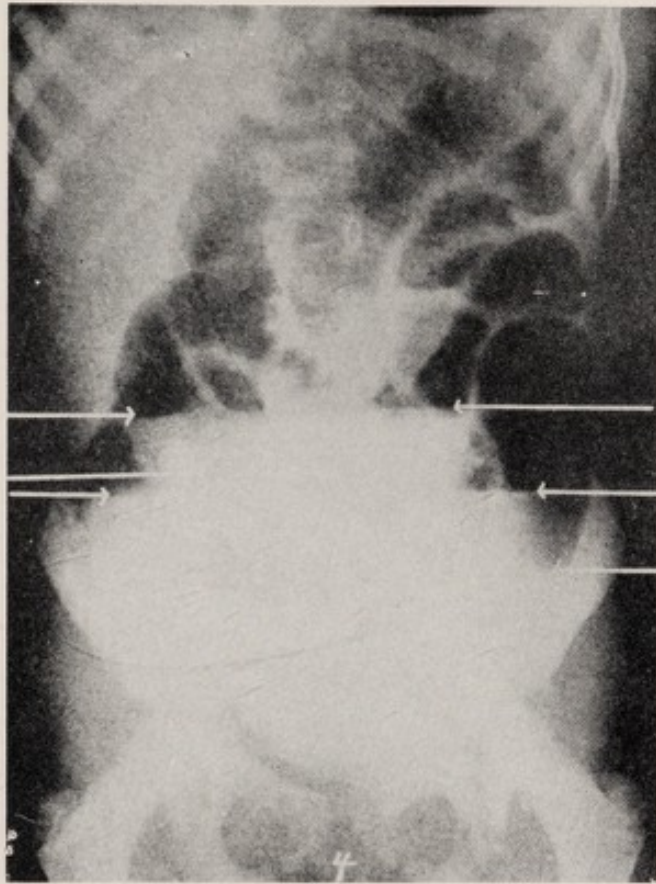


FIG. 51. Multiple fluid levels in acute obstruction; patient in upright position. (Ochsner and Granger.¹⁷)

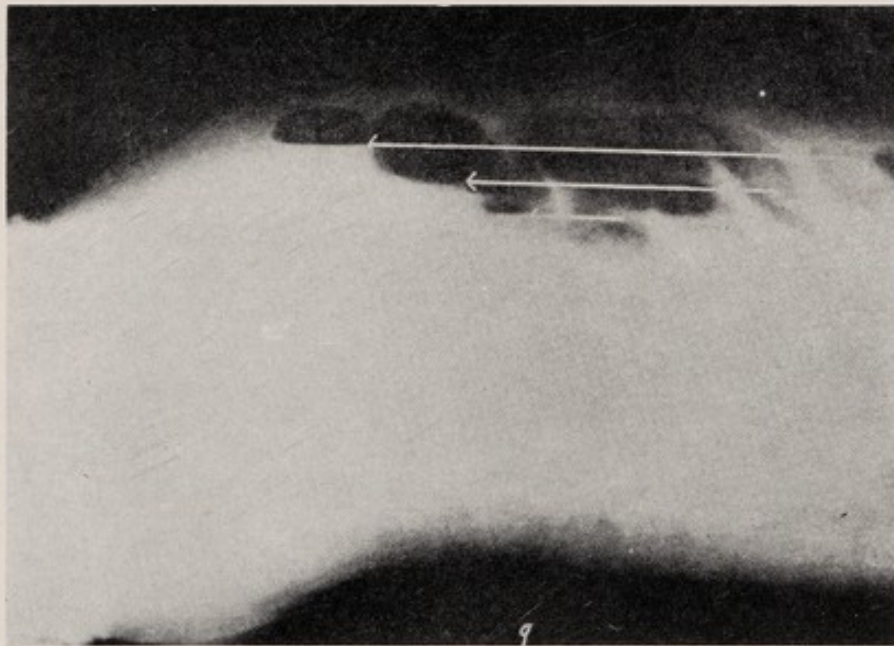


FIG. 52. Multiple fluid levels in acute obstruction; patient in horizontal position. (Ochsner and Granger.¹⁷)

mesenteric vessels a flat plate of the abdomen is of some value, although the picture cannot be distinguished from that produced by other types of obstruction.

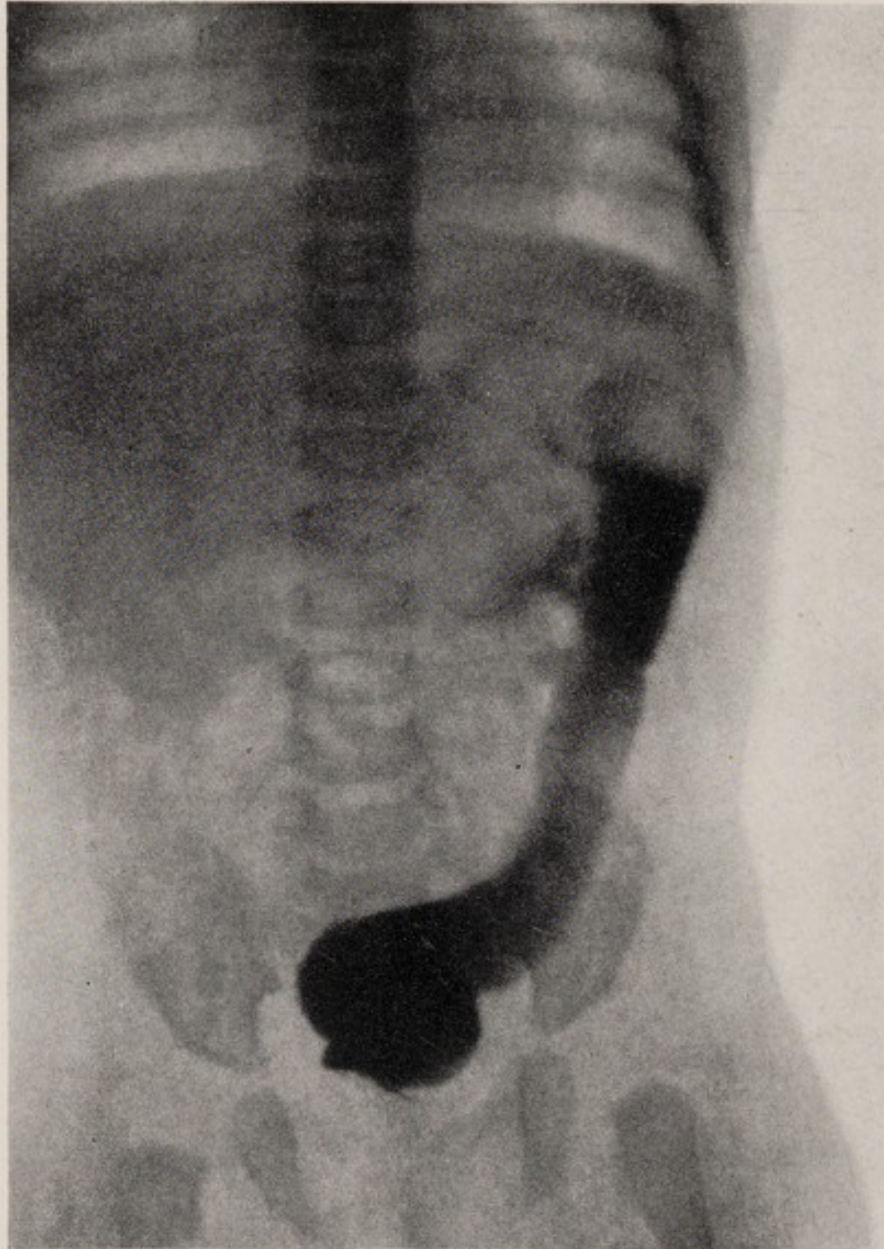


FIG. 53. Intussusception in a child of three months. Barium enema, showing column of barium stopping in region of splenic flexure. Appearance of barium at region of point of stoppage suggests intussusception. (See Case IX, p. 85.)

A barium enema is often of great assistance in the x-ray diagnosis of various types of obstructing lesions of the colon;

for while obstructions of the large intestine can often be distinctly determined on a flat plate, by noting the outlines of the distended colon and the presence of fluid levels, at times difficulties may arise which can be clarified by the enema. In addition to its use in the diagnosis of acute and subacute obstructions by neoplasms, it may be helpful in the diagnosis of volvulus of the sigmoid (Ginzburg was able to make this diagnosis without opaque media in four out of five cases), and in doubtful cases of intussusception.^{22,23} (See Fig. 53.)

The *x*-ray is also helpful at times in differentiating between functional and mechanical obstructions. If gas can be demonstrated in both the small and the large intestine, the obstruction is probably functional. The reverse, however, is not always true: signs of obstruction of the small bowel without gas in the colon do not necessarily indicate mechanical obstruction but may be demonstrated in certain cases of functional disturbance from peritonitis and from other causes. The *x*-ray may be especially misleading in determining whether an obstruction arising early after operation is functional or mechanical in origin, for the picture sometimes suggests mechanical obstruction where none exists: it should be remembered that gas in the small intestine may be demonstrated shortly after operation in most cases,* although the distention does not usually reach proportions where it is of serious clinical significance. Swenson and Hibbard suggest that since in functional obstruction from peritonitis the walls of the dilated loops sometimes appear to be thick, due to exudate over the surface of the gut, the presence of this sign may sometimes be helpful in distinguishing functional from mechanical obstruction.

* McIver et al.²⁴ have brought out the fact that most patients suffer from some degree of intestinal distention after abdominal operations, due to the entrance of air from the stomach. See also p. 180.

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CHAPTER XIX

DIAGNOSIS (Continued)

In the previous chapter we have considered the cardinal signs and symptoms of acute intestinal obstruction. The clinical picture may be briefly summarized as follows:

The attack is likely to be ushered in by abdominal pain, of a colicky and cramp-like character at the onset, often becoming constant after the first twelve to twenty-four hours; and by vomiting, which becomes more profuse and persistent as the disease progresses, taking on the characteristics of "fecal" vomiting in the late stages. While the lower bowel may be emptied once after the onset of the attack, either voluntarily or by means of enemas, subsequent enemas usually fail to produce either fecal material or gas. Abdominal distention usually develops, although it may be negligible in amount if the obstruction be situated high in the small intestine. Visible peristalsis may occasionally be seen and in the presence of the symptoms already referred to is characteristic of the disease; but it is not present as commonly as one might suppose from the textbooks, and is rarely seen where strangulation exists. The temperature and pulse rate are usually normal, although not infrequently a subnormal temperature is encountered early in the disease.

While the foregoing picture is in general characteristic of acute intestinal obstruction, certain varieties of the disease present special clinical features, which will now be briefly discussed; certain points in differential diagnosis will also be considered.

SPECIAL POINTS IN THE DIAGNOSIS OF CERTAIN VARIETIES OF OBSTRUCTION

EARLY POSTOPERATIVE OBSTRUCTION. The signs and symptoms of obstruction shown by the early postoperative group are in general those of acute obstruction from any cause:

vomiting, pain, distention and obstipation; but owing to the fact that these obstructions come on during a convalescence from abdominal operation, the picture is often masked. When the classical symptoms come on suddenly during a convalescence that is proceeding normally, the diagnosis is usually easy: the difficulties arise when the obstructive symptoms come on somewhat gradually during a stormy convalescence. The question often to be decided is whether the symptoms are due simply to a disturbance of the motor function of the intestine by operative trauma or peritonitis; or whether an organic obstruction is present.* The presence of cramp-like or colicky pains or visible peristalsis, or the detection of hyperperistalsis on auscultation, indicates that there is an attempt to push intestinal contents beyond some obstructed point, and suggests that some mechanical element is present. The character of the vomitus should be carefully noted and any change to "fecal" vomiting should be detected at the earliest possible moment. The amount of distention in these cases shows considerable variation: if the obstruction is high, it may be slight. Occasionally, localized distentions pointing to the presence of distended coils of bowel can be made out. A rising white count in the presence of obstructive symptoms is suggestive of strangulation.

The clinical variations shown by the obstructions that occur early after operation have been comprehensively discussed by Richardson.⁴ (See also p. 46.)

VOLVULUS. Volvulus of the small intestine cannot usually be distinguished from any of the other forms of internal strangulation of the small intestine.

* The use of spinal anesthesia has been suggested for the differential diagnosis between functional and mechanical obstruction. Following the administration of spinal anesthesia in functional obstructions a movement of the bowels and passage of flatus may occur. The advocates of this procedure advise prompt operation if this favorable result does not take place.¹ The possibility that violent peristaltic movements set up by the spinal anesthesia may produce rupture of a gut damaged by obstruction, should be borne in mind.

In connection with the use of spinal anesthesia, the work of Ochsner et al. on splanchnic anesthesia in the treatment of experimental ileus is of interest; and numerous references to the literature on this subject will be found in their articles.^{2,3}

Volvulus of the sigmoid at times has more distinctive features. In general it occurs in middle age or later. Not infrequently there is a history of obstinate constipation preceding the attack; it is also not uncommon to have a history of attacks in the past that suggested obstruction but were milder in character and subsided after the administration of enemas.^{5,6} The pain and tenderness are likely to be localized in the left lower portion of the abdomen; the pain may be intense and associated with considerable prostration.⁷ At times a mass can be made out, its outline corresponding with the dilated loop of sigmoid. A barium enema may show the point of obstruction.

In volvulus of the cecum also there may be a history of earlier attacks⁸; and at times a mass, representing the twisted cecum, can be made out in the right side of the abdomen.

GALLSTONES. As would be expected from the etiology, this type of obstruction is a disease chiefly of later life.⁹ It is one of the few types of obstruction that is more common in females than in males. The obstruction may be complete from the beginning; or there may be a subacute period before the intestine is completely blocked. The severity of the symptoms varies, depending in part upon whether the gallstone has become impacted in the upper intestine, or, as more frequently happens, lodges near the ileocecal valve. In the early stages the pain is usually colicky in character and referred to the umbilicus. Not infrequently a history that suggests gallbladder pathology can be obtained.^{10,11} Occasionally upper abdominal pain, nausea, vomiting and tenderness, corresponding with the discharge of a gallstone into the upper intestinal tract, may have preceded the obstructive symptoms. At times an x-ray of the abdomen may show the presence of a gallstone; and, rarely, the passage of gallstones by rectum may be reported.

Essentially, the diagnosis of obstruction by gallstones should be suggested by obstructive symptoms developing in an elderly individual whose past history indicates gall-

bladder disease. The common cause of obstruction in this age group is carcinoma of the colon; but in this latter disease there is usually a history of increasing constipation, or of frequent small movements, at times containing blood and mucus, preceding the acute attack. The distention is likely to be more marked than in obstruction by gallstones.

MESENTERIC THROMBOSIS. The diagnosis of occlusion of the mesenteric circulation by thrombosis or embolism is difficult, and is often made only at operation or autopsy: in Trotter's¹² series of 360 cases, the diagnosis was made before operation or autopsy in only 13 instances. There are, however, certain points which may suggest the diagnosis.

While the disease may occur at any age, even in childhood, it is more commonly a disease of later life. It is usually associated with some disease of the circulatory system, such as endocarditis, atheroma of the aorta or arteriosclerosis; and the patient may give a history of embolic phenomena in other parts of the body before the onset of the abdominal symptoms or simultaneously with it. There are also other diseases with which it is sometimes associated: Meyer¹³ reports that it occurred in 3 patients with polycythemia.

The classical signs and symptoms are severe abdominal pain,¹⁴ vomiting, melena, distention of the abdomen with tympanites and at times a shifting dullness in the flanks. Blood was present in the stools in 41 per cent of the cases reported by Jackson et al.¹⁵ When the process of infarction involves the upper intestine, the vomiting of blood has been reported. The patient may show signs of collapse,¹⁶ with a fall in body temperature. A low pulse rate is sometimes found at the onset of the attack.¹⁷ The leucocyte count is likely to be quite high: in the series of 92 cases collected by Meyer¹³ the count in all but 3 instances was above 18,000, the highest being 45,000.

OBSTRUCTION OF THE COLON BY NEOPLASM. The symptoms of obstruction of the colon from neoplasm have a tendency to be less fulminating than if the obstruction were located in the

small bowel. Symptoms of subacute obstruction (usually increasing constipation, or in certain cases frequent small watery movements occasionally mixed with blood or mucus, cramp-like pains radiating across the lower abdomen, increasing gurglings of gas and fluid and a sensation of inability to empty the bowels,^{18,19}) are likely to antedate the acute attack. This is predominantly an obstruction of later life.

Since strangulation is very rarely present in these obstructions, the pain may not be particularly severe. It is, however, almost constantly present. In the early stages it is usually localized across the lower abdomen; when the obstruction has existed for some time, the pain and tenderness are often localized over the site of the obstruction, probably because of edema and infection. In some instances acute discomfort from distention is the most outstanding complaint. Vomiting comes on early with the onset of the acute attack, but is not likely in the early stages to be so profuse or frequent as in obstructions of the small intestine; in the later stages, after the obstruction is well established and as the small intestine becomes more and more involved in the obstructive process, the vomiting may be profuse and of the typical "fecal" character. Distention is likely to be a very marked feature. In the early stages, visible peristalsis of the large intestine above the point of obstruction is not infrequently seen. Rectal examination is important and may disclose a mass, particularly when the growth is situated near the rectosigmoid juncture.²⁰

In an attempt to say whether or not an obstruction is complete, the passage of flatus either voluntarily or on the administration of an enema is an important criterion. The vomitus should be carefully observed; if it takes on the character of small intestinal contents, prompt surgical intervention should be undertaken even though some flatus is being passed by rectum.

There is usually little or no systemic reaction, the temperature and white blood cell count being generally normal.

X-ray examination with or without the administration of a barium enema is often helpful.

STRANGULATED EXTERNAL HERNIAS. The diagnosis in cases of strangulated external hernia is usually obvious. In typical cases, a hernia which has previously been reducible becomes irreducible. This condition is accompanied by severe, often intense, pain, which may not be confined to the hernial sac but may take the form of generalized abdominal cramps. Vomiting is usually present, starting soon after the initial pain. Obstipation is usually present; although early in the illness the bowel below the point of obstruction may be emptied voluntarily or by means of an enema, and in types of hernia where a section of bowel wall alone is strangulated, without obstruction of the lumen (Richter's hernia), bowel movements may continue throughout the course of the disease. Under these conditions, diarrhea is not infrequent.

The late manifestations are those usually found in acute intestinal obstruction from any cause: distention, fecal vomiting and prostration.

Although the diagnosis is usually so easy, there are occasional cases where it is not obvious. The patient may not be aware that he has a hernia, and a small, tense sac may be overlooked, particularly if it occurs in the femoral canal. In the recent Massachusetts General Hospital series of 147 cases of obstruction by strangulated external hernia, there were 3 instances in which the diagnosis was not made until laparotomy had been performed and a knuckle of gut found strangulated in the femoral canal. In any patient with symptoms suggesting intestinal obstruction, the usual sites of hernia should be carefully examined to exclude the possibility of a small, strangulated hernia.

INTUSSUSCEPTION. Most of the cases of intussusception occur in infants or young children, frequently at an age when the patient is unable to describe his symptoms. With the onset of the attack the infant is likely to show unmistakable signs of abdominal pain, at times drawing up the legs and crying

as the cramp-like pain takes place. Vomiting frequently takes place at the onset of the obstruction and is likely to be repeated; it may not be very profuse. Often the pain is considered to be one of the commoner varieties of intestinal colic and there is delay in calling the physician; or at times the physician prescribes for the infant over the telephone without a physical examination.

After the onset of the attack there may be one normal bowel movement; after that, the passage of bloody mucus occurs and is a very constant symptom in infants.²¹⁻²³ The infant may lie in a rather apathetic state, showing evidence of marked toxemia; the signs of prostration may occur relatively early in the disease. On the other hand, even after a number of hours have elapsed he may not show a great deal of systemic reaction. On physical examination there is usually not a great deal of distention, and in the classical case a "sausage-shaped" tumor may be felt in the right or left side of the abdomen; at times, however, the intussusception is under the lower border of the liver and no mass can be felt. On rectal examination it is often possible definitely to palpate the apex of the intussusception.

The syndrome shown by infants with intussusception is usually so typical that it seems almost incredible that cases should so often come to the surgeon late. Among the 9 deaths from intussusception in the series recently reported from the Massachusetts General Hospital there were only 2 patients who were received at the Hospital in less than forty-eight hours after the onset of symptoms. This group of obstructions seems to be outstandingly one in which the general practitioner can contribute to a lowering of the mortality by sending the patients to the surgeon earlier.

While intussusceptions occurring in infants and young children are usually of the ileocecal or ileocolic variety (involving both small and large intestines), the rare cases of intussusception in adults are likely to be of the enteric variety (involving the small intestine alone) or the colic (involving the large intes-

tine only), and are usually caused by a benign or a malignant tumor²⁴; this latter is usually encountered in elderly individuals. In adult patients the clinical syndrome is not so typical as in children. There are frequently attacks of abdominal pain preceding the final attack. With the acute attack, vomiting and pain are present; distention is usually not marked; bleeding from the rectum is infrequent; and an abdominal tumor is only occasionally felt. From the general symptoms, however, in spite of features which are not typical of intussusception in infants, it should be easy to realize at least that an acute obstruction is present and that operative interference is indicated.

There is a relatively rare disease, namely, purpura abdominalis or Henoch's purpura, which may be confused with intussusception because in this disease also there are frequently abdominal cramps associated with the passage of blood by rectum. Other signs of purpura, such as the rash over the abdomen and the petechial hemorrhages that may follow after the application of a tourniquet (the "tourniquet test"), may clarify the diagnosis. Even if it proves to be purpura, however, the question of intussusception may still be involved, for, as pointed out by Bailey,²⁵ the hemorrhagic infiltration of the wall of the small intestine that often takes place in purpura may result in intussusception. Bailey reports 9 cases where this occurred, with 7 recoveries following operation. In cases of doubt an exploratory laparotomy should be carried out.

DIFFERENTIAL DIAGNOSIS

Diseases that may be confused with acute intestinal obstruction are in general those that give abdominal pain and at the same time cause functional interference with intestinal motility. The list of diseases having a symptom complex which may, at least in part, resemble that of acute obstruction, is rather long and heterogeneous, and at times the differential diagnosis is much more difficult than most writers would

have one believe. A few outstanding examples of different types may be mentioned and briefly commented upon.

Among the more general constitutional diseases that may have abdominal symptoms of sufficient prominence to be confused with intestinal obstruction, should be mentioned lead colic, the gastric crises of tabes, and uremia. It should not be forgotten that pneumonia and cardiac thrombosis are capable of causing severe upper abdominal pain which may be accompanied by nausea and vomiting. Among the rarer medical conditions that have been mistaken for obstruction, Treves²⁶ mentions cholera and poisoning by arsenic. Angio-neurotic edema may at times cause intense abdominal pain, nausea and vomiting; Osler's article²⁷ and Withington's²⁸ on this disease are very interesting.

The differential diagnosis often depends upon finding in the course of the history and physical examination, clear evidence that some disease other than intestinal obstruction is responsible for the symptoms: for example, the fixed pupils and absent knee-jerks of tabes, the lead line on the teeth in lead poisoning, and so forth.

The pain associated with the passage of a calculus, either biliary or renal, may be mistaken for the colic of intestinal obstruction. Usually the characteristic location and radiation of the pain in these conditions,* or associated findings such as blood in the urine or at times slight jaundice, will serve to establish the correct diagnosis.

X-ray examination without opaque medium, or with the barium enema, may be helpful in arriving at a differential diagnosis; see page 233.

The following case illustrates the symptoms of obstruction associated with the passage of a renal calculus:

CASE XXXIII. No. 5317, M. I. B. H. Female, aged fifty-eight. The patient was sent to the hospital with a diagnosis of acute intestinal obstruction. She gave a history of four days of abdominal pain, profuse vomiting, and

* The characteristic pain of gallstones is, of course, likely to be located in the right upper quadrant of the abdomen and frequently radiates through to the back, especially

cessation of bowel movements. The pain was located in the left lower side of the abdomen and flank, and radiated to the left inguinal region; it had been very intense at times and paroxysmal in character. The vomitus had not been foul smelling at any time. The patient had been well and strong during her past life, "had never had a doctor." A number of years before, she had had an attack of left-sided abdominal pain resembling somewhat the present attack but shorter in duration and less severe in character.

Physical examination showed an obese woman with some abdominal distention. She complained of pain in the left side of the abdomen, but stated that it was not as severe as it had been previous to entrance. There was no muscle spasm; definite tenderness over the left side of the abdomen and in the left costovertebral angle. It was noted on examination of the extremities that the patient held the fingers somewhat tense; the thumb was in the abducted position. On applying the tourniquet to the arm (Trousseau's test) the typical carpal spasm of tetany occurred. Temperature 101.6°F., pulse 96, respirations 22; white blood count 6000, polymorphonuclear leucocytes 81 per cent, small lymphocytes 16 per cent, large lymphocytes 2 per cent, mononuclear leucocytes 1 per cent. Examination of the urine showed a faint trace of albumin, a few red cells and a few white blood cells. The blood chlorides were 495 mgs. per 100 c.c.

The unilateral nature of the pain, with the radiation to the left inguinal region, suggested the diagnosis of renal colic. When the urine was found to contain red blood cells, this impression was strengthened. A roentgenogram of the abdomen showed a shadow in the course of the left ureter that was characteristic of a calculus. This was later confirmed by a cystoscopic examination. Within the course of a few days the patient passed the calculus.

Comment: The fact that the patient's pain and tenderness were largely confined to the left side of the abdomen and flank, together with the characteristic radiation of the pain, was very suggestive of some lesion of the kidney and ureter. As has already been pointed out, functional disturbances of the gastrointestinal tract are not uncommonly associated with such pathology. In this case they were marked, particularly the vomiting. The gastric secretions had been lost to such an extent that gastric tetany occurred: this is not infrequent in organic obstruction at the pylorus; but

to the "shoulder blade." The pain of renal colic may be localized largely in the region of the costovertebral angle, in which case it is not likely to be mistaken for intra-abdominal pathology; where the pain is felt in the abdomen it is likely to be localized in one side of the abdomen or flank and radiate downward to the scrotum or penis in the male or the vagina in the female; the pain may be referred down the thigh on the affected side. Contrast the pain in these conditions with the pain in intestinal obstruction (see p. 227).

it is rare that the vomiting in cases with no organic obstruction is sufficient in amount to produce this result.

The distention yielded to the simple measures of flaxseed poultices and rectal tube. Large volumes of normal salt solution were administered to the patient subcutaneously. The vomiting subsided, as did likewise the signs of gastric tetany. No operative procedures (other than cystoscopic examination) were required.

Various acute conditions arising in the abdominal cavity cause a sudden onset of intense pain, nausea and vomiting, and may resemble the picture of intestinal obstruction with strangulation. Among these may be mentioned the twist of an ovarian cyst or of a pedunculated fibroid, the perforation of an ulcer, acute pancreatitis, etc. Rarely, the intensive colic accompanying the onset of a fulminating appendicitis might be confused with the pain from an obstruction, but usually the localized tenderness and other findings will clear up the diagnosis. Frequently in this group it is not possible to make an accurate differential diagnosis; but it is usually clear that prompt surgical intervention is indicated, whatever the diagnosis, and operation should not be delayed. In the following case certain symptoms of obstruction were associated with acute appendicitis and general peritonitis:

CASE XXXIV. No. 8124, M.I.B.H. Male, aged forty-nine. The patient was sent to the Hospital with a diagnosis of intestinal obstruction. About two weeks before this he had had an attack of abdominal pain, extending across the lower abdomen, which persisted for about twenty-four hours. The pain was somewhat colicky in character, but was dismissed by the patient as "indigestion." Four days before admission the pain recurred; the patient took several doses of cathartic without relief of the pain and with little fecal result. Two days before admission he woke from sleep with a very severe pain across the lower abdomen; a physician was called and morphine was administered hyperdermically; the patient vomited several times. Since that time the patient has had little pain, but marked distention of the abdomen; there have been no bowel movements and enemas have produced only a small amount of gas. The patient was known to have had diabetes and nephritis for a number of years.

Physical examination showed a moderately obese man with distention of the abdomen; he was having no pain. On palpation there was slight

tenderness on deep pressure over the right lower quadrant; over this region there was definite muscle spasm. Rectal examination was not remarkable. The patient was sweating profusely. Temperature 101.2°F., pulse 116, respirations 24, white blood count 14,000, with 90 per cent polymorphonuclear leucocytes. The urine showed heavy traces of albumin and sugar, with many hyaline and granular casts, few white blood cells, a rare red blood cell; diacetic and acetone were present in large amounts; the blood sugar was 235 mgs. per 100 c.c.

Although this patient did have certain signs of intestinal obstruction (cessation of bowel movements and essentially negative results from enemas, together with abdominal pain and vomiting), it seemed reasonably certain that these signs were not primary, but were secondary to an inflammatory process within the peritoneal cavity. The history, together with the elevation of the temperature and the leucocytosis, abdominal tenderness and spasm, all pointed to this diagnosis.

The patient was intensively treated for a few hours by insulin and intravenous administration of solutions of saline and glucose, by the medical department. Under this regime the blood sugar was lowered and the acetone and diacetic disappeared from the urine. The abdomen was then explored under novocaine and gas anesthesia. A ruptured appendix and general peritonitis were found. The appendix was removed and a drain inserted. The patient's general condition became progressively worse, and he died on the second postoperative day, with marked elevation of pulse, temperature and respiration. There was no vomiting following the operation and the abdominal distention diminished somewhat.

Comment: This case represents a functional disturbance of intestinal motility from peritoneal inflammation. The diagnosis was reasonably clear. The functional obstruction really played no important role in the picture, the patient dying of a progressive peritonitis.

The distention and obstinate constipation shown by patients with Hirschsprung's disease may at times suggest obstruction; in case of doubt, a barium enema will show the true state of affairs.

Fecal impaction in the rectum in the aged may at times reach such proportions as to constitute a temporary obstruction. The obstruction, however, is rarely complete, and a rectal examination and enema will usually settle the question.

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CHAPTER XX

TREATMENT

The general principles governing the treatment of a case of acute mechanical obstruction of the intestine are simple: they consist of relief of the obstruction, and correction as far as possible of the systemic effects of the obstruction. The need for prompt operation has long been stressed; but realization of the importance of the treatment of the secondary effects of obstruction is a relatively recent advance.¹⁻² It is now generally recognized that many of the signs and symptoms shown by the patient are due not solely to the absorption of a toxin, as formerly thought, but also to loss of water and sodium and chloride ions from the body by vomiting. The restoration of these substances is an extremely important factor in the treatment,³ although the amount of replacement needed is, of course, in proportion to the amount of fluid lost, which varies greatly in different types of obstruction.⁴⁻⁵ *

The management and treatment may be chronologically considered under three heads: treatment before operation; operation; and after care.

PREOPERATIVE TREATMENT

Since promptness in operating is of paramount importance, no preparatory procedure that materially delays surgical relief should be considered. Usually, however, after the decision to operate has been reached there is an interval, while the necessary arrangements for operating are being made, which can be utilized to good advantage in preparing the patient. Proper treatment at this time will often put the patient into much better condition for operation. This applies particularly to the late case of simple obstruction; here, instead of rushing the patient to the operating room, some time may be advantageously spent in improving his general condition, especially

* See p. 387.

as regards treatment of dehydration and emptying the dilated stomach and upper intestine.

RELIEF OF PAIN. If the pain is severe, morphine should be administered. Since the diagnosis has been made, morphine is no longer contraindicated for fear of masking symptoms, and even partial relief from the agonizing types of pain will often materially improve the patient's general condition and relieve the mental anxiety that is often an important feature.

MAINTENANCE OF BODY TEMPERATURE. It is important that any patient prior to operation be adequately protected against exposure. It is especially important in the type of case which shows signs of collapse or is in danger of developing such a condition after operation that the normal body temperature be maintained as a prophylaxis against shock.⁶ The physician and nurse should be careful to avoid undue exposure during examinations, or while the skin preparation for operation is being carried out, during the administration of enemas, etc. If indicated, the patient may be wrapped in warm blankets and heat applied to the extremities.

TREATMENT OF DEHYDRATION. One of the most striking clinical features of many cases of obstruction is a marked dehydration. This is proportionate to the amount of vomiting that has taken place; for in the vomitus is lost not only water but also the sodium and chloride ions, which are essential in maintaining the normal volume of body fluids. These substances are best returned to the body by the administration of normal salt solution;* as has already been shown in the chapter on body fluids,† no other type of solution will take the place of one containing sodium chloride.¹¹ As demonstrated

* In their early work, Haden and Orr advocated the administration of hypertonic saline⁷ on the theory that the blood chlorides were low, due to the fact that they were called upon to neutralize a toxin.⁸ These authors have abandoned this theory,⁹ and it seems physiologically more correct to use an isotonic solution, which also permits the administration of large volumes of fluid. The work of Hughson and Scarff¹⁰ on the markedly stimulating effect of hypertonic salt solution on intestinal peristalsis is of interest; it would seem unwise, however, to increase peristaltic activity before relief of the obstruction.

† See p. 155.

experimentally by Haden and Orr⁷ and White and Bridge,¹² water alone, or glucose alone, is without benefit. Not infrequently, however, it may be desirable to add glucose to the salt solution, for the patient may have been starving for some time and the glycogen reserve of the liver be exhausted. If the slow intravenous infusion of a solution containing glucose be continued over several days, as described later, considerable amounts of glucose can be administered in the course of twenty-four hours and the caloric requirements proportionately met. Glucose is usually added in the concentration of 5 or 10 per cent; it is better to use the former concentration, which is isotonic, if the solution is to be given subcutaneously, for it should be borne in mind that the fluid is not taken up so rapidly from the tissues when it contains glucose, especially if the solution be hypertonic.

Method of Administering the Fluids. Administration of fluids by mouth is contraindicated: they bring on vomiting, which increases the loss of chlorides, and produce distention of the intestinal tract above the point of obstruction (Foster¹³ and others). There are a number of methods by which fluids can be given satisfactorily, and these are discussed in the order of ease of administration: (1) by rectum; (2) by hypodermoclysis; (3) intravenously. Where the dehydration is severe, all three methods of administering fluids may be used.

The large amount of fluid that will be absorbed from the rectum when the body is depleted is well known. There is usually no contraindication to administration by rectum, either in the form of a continuous Murphy drip, or by injections of 120 to 180 c.c. (4 to 6 oz.) repeated every three or four hours. Normal salt solution should be used.

The subcutaneous administration of salt solution, either as normal sodium chloride or as modified Ringer's solution,¹⁴ is one of the simplest and most effective methods for supplying large volumes of fluid to a patient with intestinal obstruction. It may be used alone or in conjunction with the rectal and intravenous methods. It is customary to inject fluids under the

breast or into the subcutaneous tissues of the thigh. Kolodny¹⁵ feels that hypodermoclysis under the pectoral muscles interferes with thoracic respiration and favors postoperative pulmonary complications; he advises, therefore, that after laparotomy the infusion be given in the thighs. It is usually possible in the adult to give 1000 or 2000 c.c. in the course of a single injection without producing too great distention of the tissues; the flow of fluid must be clamped off from time to time when the rate of entrance markedly exceeds the rate of absorption.

Injection of normal salt solution (with or without the addition of 5 to 10 per cent glucose) directly into the venous circulation is the most rapid and often the most satisfactory method of administering fluids. The usual and well-known precautions for intravenous injection of any fluid should be observed¹⁶⁻¹⁹; the solution should, of course, be sterile, chemically pure, and free from any particulate matter,* and it is desirable that it be made up only a short time before its injection. The fluid should be injected at body temperature, and care should be taken that it be not given too rapidly. The importance of regulating the speed of injection is stressed in the work of Hirshfeld et al.²⁰ These authors showed that the rapid injection of a relatively harmless substance might produce a serious disturbance which they call "speed shock," whereas even a substance ordinarily recognized as toxic might be tolerated if given slowly. The rate must be governed somewhat by the urgency of the situation: if the patient is seriously ill and only a short time is available before operation, it may be desirable to give 500 to 1000 c.c. to an adult during the course of half an hour or an hour; this may be safely done and will at times bring about marked improvement. Ten per cent

* It is especially important that new rubber tubing be carefully washed and boiled to remove any substances from the inner surface, since these are at times responsible for reactions; the whole apparatus should be autoclaved before use. The ampoules of 50 per cent glucose that may be obtained on the market are useful and may be added to the solution immediately before injection; since employing these, over a period of several years, the author has not seen a reaction following intravenous use of glucose.

glucose in normal saline is often a useful solution under these circumstances.¹⁸ If a longer period of time is available, a larger volume can be given and is often desirable, the amount depending upon the extent of the dehydration, and upon the age, size and general condition of the patient, particularly as regards his cardiovascular and renal systems and his toleration of the treatment.

A slow, continuous intravenous infusion which may be kept up for hours or days is useful in selected cases. Matas,²¹ Penfield and Teplitzky,²² and Hendon²³ were among the first in this country to call attention to the usefulness of this method, although the subject had previously been extensively dealt with in the European literature.^{24,25} Various other articles²⁶⁻³¹ may also be consulted for details of technique. No complicated apparatus is required: a simple and satisfactory set consists of a container of 1 to 2 litres' capacity, connected by rubber tubing to a needle or cannula to be inserted into the vein, with a screw pinchcock between the reservoir and the cannula to regulate the flow, and a "drip bulb" which permits the drops to be counted. A convenient vein of the arm may be utilized, and the arm immobilized on a splint or pillow if desired. Lahey³⁰ has called attention to the advantages of employing an ankle vein if the patient is restless or delirious. The temperature of the solution may be kept up by hot water bottles about the container and tubing; or a heating unit as suggested by Matas²¹ may be employed. Titus³² has developed a more complicated apparatus. The rate of flow should be about 150 to 200 c.c. per hour. Lahey³⁰ feels that concentrations of glucose stronger than 5 per cent produce a thrombosis of the vein receiving the solution.

The slow "intravenous drip" does not supplant but supplements the older methods. It is undoubtedly useful in serious cases, and can be continued over a period of days to maintain the body fluids and, to a certain extent, to meet the nutritional requirements. It should not be forgotten, however, that the introduction of substances into the body through the venous

circulation is a most abnormal procedure, and unlooked-for effects on the circulation or distant organs can occur. A number of at least potential dangers from the intravenous injections of glucose solutions were pointed out in an editorial appearing in the *Journal of the American Medical Association*³³; Titus et al.³⁴ have suggested that prolonged injection of glucose overstimulates insulin production by the pancreas; a depressing action of glucose on intestinal activity has been reported by Gage et al.³⁵ (see p. 212). Jones and Eaton³⁶ found a low level of serum protein in patients that developed edema after surgical procedures; they consider that the reduction of protein intake is an important element in the situation, and also that the administration of an excessive amount of fluid and salt after operation, especially by the intravenous route, is an additional factor. Matas²¹ points out the danger of excessive amounts of salt solution in patients with renal damage or with certain circulatory and pulmonary diseases. When the intravenous infusion is in progress, one should watch for the development of any edema of the dependent parts or about the eyes; the output of urine should of course be carefully recorded. When glucose is being given, the urine should be tested for sugar: if more than a trace is found the glucose intake should be cut down. See also postoperative management, page 305.

GASTRIC LAVAGE. Patients with obstruction who are having the regurgitant type of vomiting, or where there are other reasons for suspecting a dilated stomach, should have a tube passed and the stomach emptied. Often this not only temporarily improves the general condition of the patient, but renders vomiting during the operation less likely. This latter point is of particular importance if a general anesthetic is to be used, for the patient may regurgitate and aspirate the septic material into the lungs, thus setting up a diffuse, fulminating pneumonia; or he may even aspirate so much material that he dies on the table from asphyxia. The danger from this serious complication is not completely removed by emptying the

stomach before operation, since the stomach may rapidly fill again from a dilated intestine. In the more serious cases a small tube may be passed into the stomach through the nose and be left there during the operation.

“DECOMPRESSION” OF THE STOMACH AND INTESTINES BY THE DUODENAL TUBE. Decompression of the stomach and obstructed intestine by means of a duodenal tube introduced through the nares has been advocated both as a preoperative measure and, in certain types of obstruction, as a form of treatment replacing operation. The value of this procedure in the prevention and treatment of postoperative distention, nausea and vomiting, dilatation of the stomach, subacute obstruction, and so forth, has long been recognized; for reference on this point see articles by Ward,^{37,38} McIver et al.,³⁹ Paine et al.,⁴⁰ and others, also the section on distention in this monograph, page 175. The application of mild suction to the tube in order to insure continuous gastric drainage was suggested by Ward³⁷ in 1925 for use in the management of patients with general peritonitis and intestinal obstruction. More recently Wangenstein and Paine,^{41,42} who have used the “decompression” technique as a method of improving the patient’s condition before operation and also as treatment for subacute obstructions and for certain of the simpler types of acute obstruction of the small intestine (“adhesive obstruction”), have reported that in selected cases the symptoms have subsided under this treatment and no operation has been required.

How far the use of this method can be carried as regards intestinal obstruction remains to be seen: its advantages and limitations cannot be fully defined until a greater body of experience is at hand. It is certain that the cases for this method of treatment should be carefully selected, and this requires good judgment supported by wide clinical experience. If any one fact in regard to acute intestinal obstruction is established, it is that early operation results in a low mortality rate; therefore where the diagnosis is suspected, grave responsi-

bility rests upon the surgeon who delays operation. With this in mind it may be said that the "decompression" procedure is valuable in certain borderline cases where obstructive

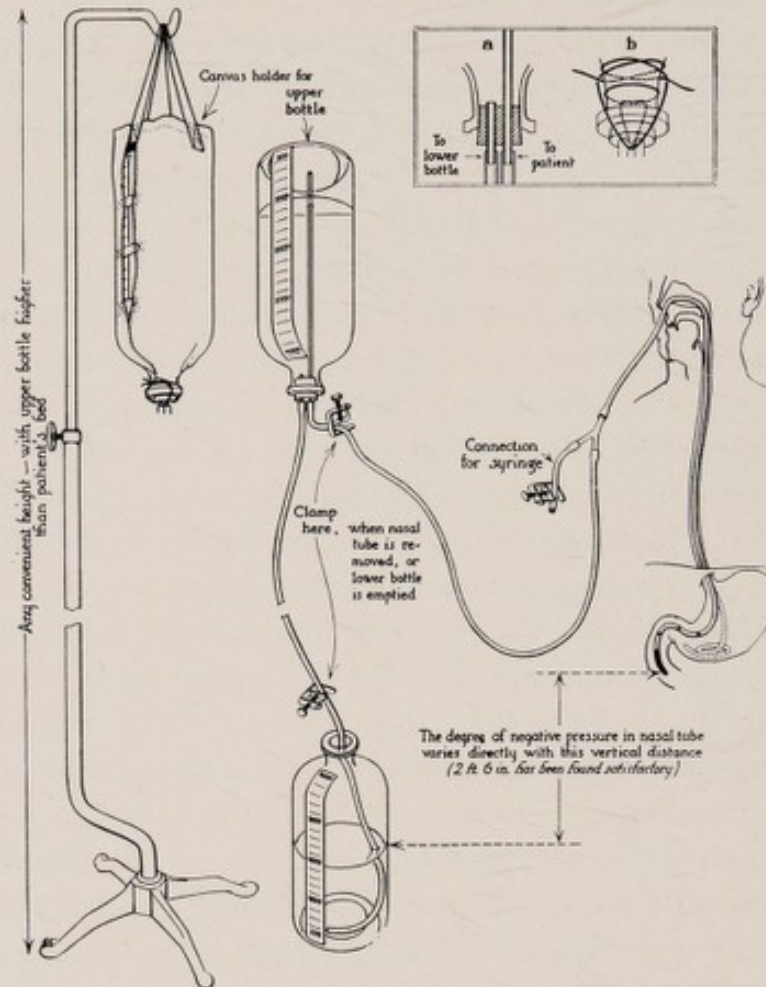


FIG. 54. Diagram of suction apparatus. Upper bottle is hung by a canvas sling from an irrigation standard. Lower bottle rests on floor. A "Y" connection is attached to duodenal tube so that a syringe may be conveniently used to clear tube if it becomes plugged. (Wangensteen and Paine;⁴² see also Ward.³⁷)

symptoms exist but where the indication for operation is not clear; it may be possible to tide over some of these subacute cases without operation. In a group of the obstructions occurring early after abdominal operations, where the obstruction is often caused by fibrinous adhesions rather than by dense bands and where in the past an enterostomy has often brought about a cure without further operative procedures, the use of a

duodenal tube and suction may at times obviate the necessity for the enterostomy. High simple obstructions that occur as a result of a malfunctioning posterior gastroenterostomy may respond satisfactorily to this procedure. As has been pointed out in Chapter xvii, it is also often valuable in the prevention and treatment of functional obstruction. On the other hand, this method of treatment, to the point of delaying early operation, is clearly contraindicated in the more fulminating types of obstruction. Where strangulation is suspected, any temporizing is obviously hazardous and may lead to a fatal outcome. There are also other cases where delay in operative intervention may be fatal: cases, for example, in which isolated fibrous bands cross a loop of intestine and while not strangulating the blood supply often produce necrosis at the points of contact with the bowel wall; or cases of obstruction caused by Meckel's diverticulum, where ulceration and perforation may occur early due to the poor blood supply of the diverticulum. The use of this method of treatment to the exclusion of operation is also, as pointed out by Wangenstein and Paine,^{*42} contraindicated in obstructions of the descending colon, because of the danger of gangrene and perforation of the cecum due to unrelieved distention.

In summary it may be said that in the improvement of a patient's general condition before operation and in the management of his convalescence, the duodenal tube undoubtedly has a great field of usefulness. As a procedure supplanting operation, it would seem to have a distinctly limited field.

ANESTHETIC. The anesthetic to be used must be fitted to the individual case. In Table xiii are shown the types of anesthesia used in cases of intestinal obstruction (exclusive of strangulated external hernia and neoplasms) at the Massachusetts General Hospital during the twenty-year period

* These authors feel that frequent x-ray plates of the abdomen, without opaque media, are useful in checking the results of treatment and the progress of the case where the duodenal tube is being used.

1908-1927 inclusive, and the attendant mortality. The low mortality shown by the cases receiving ether is in agreement with the figures recently reported by Miller.⁴³ As regards the Massachusetts General Hospital figures, the high mortality attending local anesthesia as contrasted with general, is probably explained on the basis of the fact that the cases received early for operation were generally done under ether; whereas the more desperate types of cases, usually those received late, were usually operated upon under novocaine anesthesia.

Ether. There are many early cases where ether is a safe anesthetic, and the complete muscular relaxation that it produces is desirable from the point of view of a thorough exploration. In the later stages of the obstruction, when the patient is beginning to show systemic manifestations of the disease, ether has a number of drawbacks.

In the first place, shock and collapse are much more frequent after the use of ether, even in patients who seemed to be in reasonably good condition at the time the operation was started. Its action in these cases resembles its effect on patients with severe traumatic injuries, where its use often tends to induce shock if this is not already present, or to depress further the patient who already shows signs of shock. An interesting study of the effects of ether in shock was made by Cattell.⁴⁴ This investigator found that a condition of sensitiveness to ether was brought about by any circumstances which tended to depress the general condition of the animals. Upon etherizing the experimental animals there occurred a preliminary drop in blood pressure, probably due to the depressing action of ether on the heart. In normal animals, this was quickly compensated for by the vasoconstrictor action of ether on the peripheral vessels; but when the animals were in shock, no such compensation occurred and the blood pressure continued to fall, reaching zero before the eye reflexes disappeared.

Secondly, the local effects of ether on the bowel may be injurious. Ether normally inhibits the peristaltic activity and

decreases the tone of the intestine; and the bowel which is distended and fatigued from obstruction may not resume its activity after operation, even though the obstruction has been relieved. In the less extreme cases, the convalescence may be rendered more serious by the depressing effect of ether on intestinal activity and tone.

Thirdly, the postoperative pulmonary complications after ether anesthesia should be remembered. Although statistically they are not more common following ether than after other forms of anesthesia, it should be recalled that the more serious cases, which might be expected to have a higher incidence of pulmonary complications, are usually done under local anesthesia. For a recent analysis of postoperative pulmonary complications, see King's article.⁴⁵

Novocaine. Local anesthesia is often the anesthetic of choice in obstruction, particularly in the more serious cases where no extensive exploration is contemplated. There is no depressing effect on any of the bodily functions; and since the reflexes about the throat are intact, the danger of aspirating vomitus is avoided. It has the disadvantage that complete muscular relaxation is not obtained, so that exploration may be difficult; also intestinal manipulation requiring traction upon the mesentery may be painful. It is a form of anesthetic, however, that has a wide field of usefulness in obstruction. Most of the operative procedures required can be carried out satisfactorily without employing supplementary anesthetics. It is particularly indicated in the very sick individual requiring only an enterostomy; and practically all cases of strangulated external hernia can be operated upon satisfactorily without resort to other anesthetics. Its use is by no means limited, however, to those two types of case.

Nitrous Oxide. Nitrous oxide with oxygen is occasionally a satisfactory anesthetic. The gas has little or no depressing action, and actually raises the blood pressure. Unless skilfully given, relaxation of the abdominal muscles is poor, and the straining is likely to push distended coils of intestine out of the

wound, and render exploration difficult and dangerous; at times, however, it may be useful as an adjunct to local anesthesia. Because of the danger of aspirating vomitus, the mask should not be strapped on.

Ethylene. Ethylene has proved a satisfactory anesthetic in many cases of obstruction. In some clinics, however, it is not

TABLE XIII
TYPES OF ANESTHESIA*

Type of Anesthesia	1908-1917			1918-1927		
	Number of Cases	Number of Deaths	Mortality (Per Cent)	Number of Cases	Number of Deaths	Mortality (Per Cent)
Ether.....	99	41	41	99	35	35
Spinal anesthesia.....	7	1	14	1	0	0
Local (novocaine).....	4	2	..	29	20	69
Spinal and general.....	2	1	..	0	0	0
Novocaine and general.....	4	4	..	10	6	60
Gas-oxygen.....	2	0	..	8	6	75
Ethylene.....	0	0†	..	9†	1	11
Total.....	118	156		

* Richardson;⁴⁶ McIver.⁴⁷

† The use of ethylene was abandoned because of the explosion hazard.

used because of the explosion hazard. Its advantage lies in the fact that it gives better relaxation than nitrous oxide, without having the depressing effect of ether. This anesthetic also may be used advantageously to supplement local anesthesia.

Spinal Anesthesia. Spinal anesthesia is increasing in popularity in many types of gastrointestinal surgery.⁴⁸ It is at times a satisfactory and desirable anesthesia in obstruction:

it gives good muscular relaxation and has the advantage that under its influence intestinal peristalsis and tone are stimulated. It has, however, certain disadvantages. It interrupts the vasoconstrictor impulses passing by way of the splanchnic nerves to the blood vessels of the abdominal viscera, often producing considerable fall in blood pressure. In the deeply toxic patient this drop in blood pressure may be profound and serious, for considerable strain on the splanchnic circulation may already exist and anything that tends to abolish the compensatory mechanism may be disastrous. If, therefore, the patient shows any sign of shock or is in such a precarious condition that the onset of shock seems likely, spinal anesthesia should be given only after careful consideration of its dangers.

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CHAPTER XXI

TREATMENT (Continued)

THE OPERATION

GENERAL CONSIDERATIONS. The location and character of the obstruction having first been determined as exactly as possible (particularly as to whether the large or small intestine is involved and whether or not strangulation is present), it is important to form an opinion in regard to the patient's general condition. It should be realized that a patient with intestinal obstruction is often a much poorer risk than might be supposed from the objective findings: the compensation that these patients have been able to establish is easily broken by anesthesia and operative procedure, and once this is broken they quickly pass into a state of collapse and shock from which recovery may be difficult or impossible. With this in mind, the type and extent of operation must be fitted to the individual patient. The chance of recovery for the extremely ill patient is better if the minimum amount of operative procedure be carried out; and it is easy to be led into doing too much. Excessive manipulation of the abdominal viscera, especially traction on the mesentery, or evisceration, is to be avoided. Speed is desirable; but precision and gentleness are essential.

In performing the operation the greatest care should be used in attempting to free adhesions or to deliver an adherent loop: a friable, inflamed, distended loop of bowel may be almost as easily torn or punctured as wet paper; and if this accident occurs the result is usually fatal. Even if no gross perforation occurs, many surgeons believe that the permeability of an obstructed bowel is at times so increased that a peritonitis may result from undue handling.

Ordinarily, as soon as the peritoneum is opened dilated coils of intestine present themselves in the wound. Great care is necessary that they do not escape, especially if the patient

makes a sudden straining movement, for not only is such an accident conducive to shock, but the dilated coils may be extremely difficult to return to the abdomen. Care should be taken to cover immediately with warm, moist towels or large gauze sponges any coils of intestine that are exposed.

CHOICE OF PROCEDURE. The first important question to decide is whether the abdomen is to be explored, or whether some simple procedure such as an enterostomy shall be carried out without exploration. The answer to this question may be clear from the preoperative study of the patient; or it may depend upon the operative findings, as for example evidence, upon opening the peritoneal cavity, of strangulation which had not been suspected beforehand.

In the seriously sick patient, if there is no evidence of strangulation and the obstruction is in the small intestine, an enterostomy alone is usually sufficient without any exploration; the intestine should not be allowed to come out of the abdomen. If it has been determined before operation that the obstruction is in the large intestine, a cecostomy should be done with a large tube (unless the obstruction is in the ascending colon or at the ileocecal valve, which can easily be determined); no exploration should be done, and the intestine should not be allowed to come out of the abdomen. In doing either an enterostomy or a cecostomy, if the intestine is much distended an aspirating needle attached to a suction apparatus should be inserted to empty the loop that is to be opened.

Aside from the foregoing group of cases, there is the larger group where an exploration is indicated. The hand should be inserted into the abdominal cavity and the experienced surgeon should be able to feel any band, growth or volvulus, in fact any local distention or thickening of the intestine. In certain instances a collapsed intestinal coil may be followed up to the point of blockage, or a coil of dilated intestine followed down to the obstruction, and the nature of the pathology determined. The situation may be very simple, as in the case of a single band compressing a loop of gut, or of an annular

carcinoma of the colon. On the other hand, it may be extremely puzzling, as for example in cases where the intestine is snarled and rotated around some point fixed by a mass of adhesions. Tracing a section of collapsed intestine may lead one to an opening of an abdominal fossa where an internal strangulation has occurred, or to some hole in the mesentery or omentum through which herniation and strangulation has occurred; palpation in the region of the terminal ileum may reveal an impacted gallstone or an inflammatory reaction around a Meckel's diverticulum which may be responsible for the obstruction; or the characteristic mass of an intussusception may be felt. The intestine should not be allowed to come out of the abdominal cavity during the exploration.

If by this conservative method of exploration it is impossible to locate the obstruction, and if it is imperative that it be found, it may be necessary to resort to partial or complete evisceration. Cheever¹ suggests that the intestines, as they are brought out of the peritoneal cavity, be placed in a large sheet of rubber dam and covered with pads wet with warm saline solution. D. F. Jones² says that only in cases of absolute necessity should the intestines ever be brought out of the abdominal cavity, for he believes that even with the utmost precautions this procedure is dangerous to the life of the patient.

LOCATION OF THE INCISION. As regards the incision in a case of intestinal obstruction, in general it may be said that when no definite indication exists as to where this should be made, it had best be located over the lower abdomen on or near the midline.³ A right para-umbilical incision which can be extended upward or downward is often useful and furnishes access to a large area of the abdomen.⁴ The great majority of obstructions involve the lower bowel and are to be found in the lower abdomen or pelvis, and the incision should furnish ready approach to this region. The incision should be of sufficient length to permit exploration to be carried on with ease and rapidity.

CHARACTER OF THE PERITONEAL FLUID. As soon as the peritoneum is opened, most valuable information may be obtained from the character of the peritoneal fluid. In cases of obstruction there is an increased amount of clear, straw-colored fluid; if the fluid is of a dark, bloody color, a strangulation must be suspected and sought for. In the recent series of 335 cases at the Massachusetts General Hospital there were 21 instances in which blood-stained fluid was present and a gross interference with the mesenteric blood supply found; in a number of instances the fluid was described as foul-smelling, indicating extreme damage to the bowel. There were 4 cases in which blood-stained fluid was present but no definite interference with the mesenteric circulation found; the fluid in these cases was probably the result of the great distention and congestion of the obstructed intestine. There were also a few cases in which, although there was interference with the mesenteric circulation, it was definitely stated that there was no bloody fluid. Its presence must depend upon the degree and duration of the strangulation. It represents an important operative finding; but its absence, particularly in cases brought early to operation, does not preclude the possibility of strangulation.

At times the character of the peritoneal contents may suggest that a mistake in diagnosis has been made: the purulent fluid may suggest some inflammatory disease, or the escape of gas and fluid indicate the perforation of an ulcer.

ORIENTATION. The immediate problem is to obtain some idea as to the level of the obstruction. This may be easy or may be difficult. If a collapsed small intestine can be located, it is obvious that the obstruction is in the small intestine and that it must be sought for at a level higher than the collapsed portion. On the other hand, if, in addition to dilated coils of small intestine, distended colon (easily identified by the characteristic bands and appendices epiploicae or the attachment of the omentum) is found, the obstruction is presumably situated in the colon distal to the distended portion. It is sometimes time-

saving to examine the cecum and terminal ileum first. These structures serve to orient one for further exploration, while their distention or collapse at once furnishes evidence as to

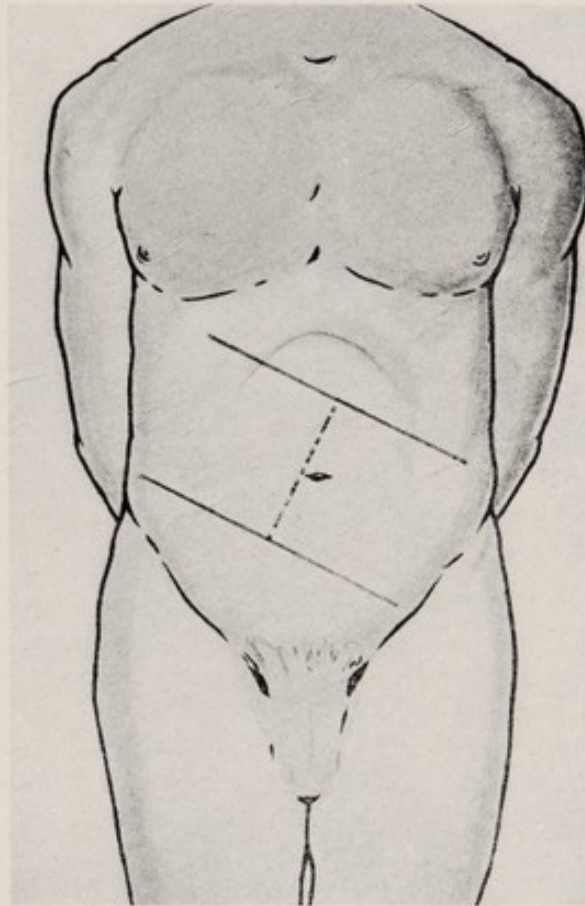
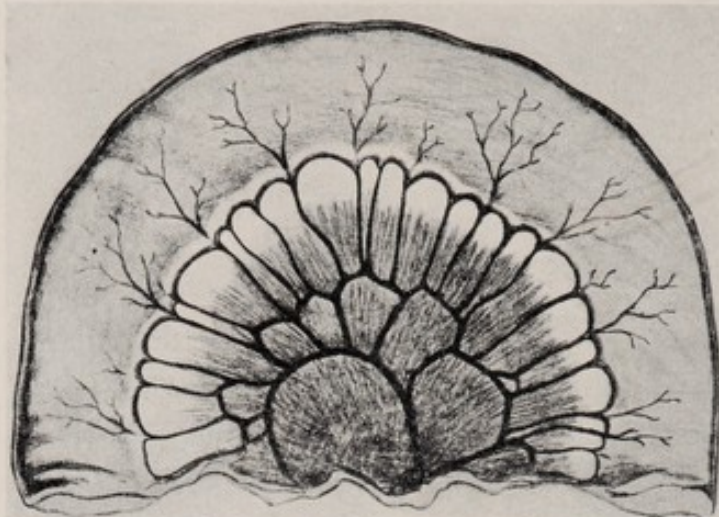


FIG. 55. Broken line is approximately line of mesenteric root as located on abdominal wall. Oblique solid lines divide abdomen into three compartments containing upper, middle and lower thirds of small intestines. (Monks.⁵)

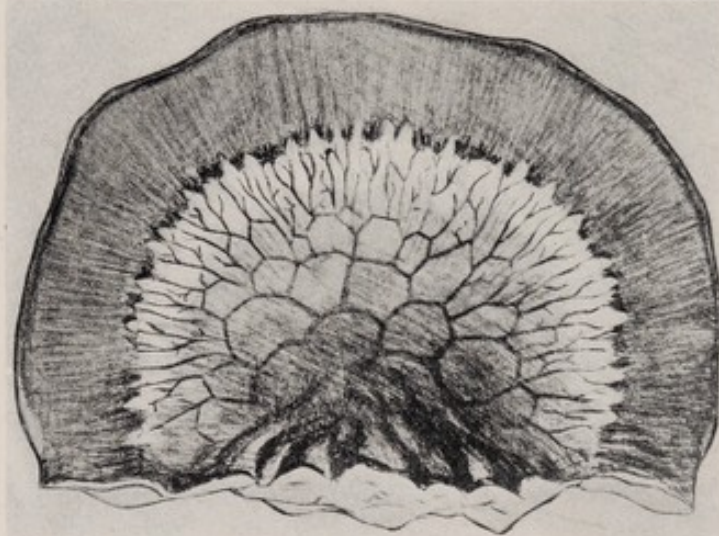
whether the obstruction is located in the large or small intestine.

Monks, in 1903⁵ on the basis of the attachment of the mesentery and certain distinctive anatomical features of the intestine and its blood supply, called attention to a number of points that are useful in determining approximately to which portion of small intestine any given loop belongs. Diagrams from his article are reproduced here. In Figure 55 the broken line is approximately the line of the mesenteric root as located on the abdominal wall. The oblique solid lines drawn at right

56A



56B



56C



FIG. 56 A, B and C. [For legend see facing page.]

angles to this line divide the abdomen into three compartments containing the upper, middle and lower thirds of the small intestine. The correspondence between the different parts of the small intestine and the oblique attachment of the mesentery is obvious. The upper 6 feet or so of the tube are generally confined to the left hypochondriac region. The upper small intestine is in general of larger diameter than the lower portion of the intestine; the walls are thicker, and due to greater vascularity the color is pinker. These distinctive features, however, may be largely obliterated or obscured by the effects of distention. The size and arrangement of the mesenteric blood vessels offer characteristic points and may furnish a clue as to the identity of an intestinal loop; Figure 56 A, B, C illustrates these points. The mesenteric vessels opposite the upper part of the bowel are larger than at any other point, and diminish in size until the lower third of the gut is reached. In general, the mesentery of the upper intestine is thinner and more translucent than that of the lower intestine.

The foregoing points in regard to the approximate identification of any given portion of the small intestine may or may not be helpful in a particular case. They may, however, furnish valuable hints, and the student would do well to note at the autopsy table, in the dissecting room, or during the course of general abdominal work, the small anatomical variations that distinguish different levels; for at times the information may be valuable and prevent unnecessary manipulation in an emergency.

EVACUATION OF DISTENDED INTESTINE. Distention of the coils is a factor which often renders exploration difficult in

FIG. 56. A. Loop of intestine at 6 feet, showing vasa recta. Secondary loops are a prominent feature. (Monks.⁵) B. Loop of intestine at 17 feet. Mesentery is opaque, and small tabs of fat begin to appear along mesenteric border of gut. Vessels are represented by a somewhat complicated network and are seen with difficulty in thick fat of mesentery. C. Loop of intestine at 20 feet. Gut appears to be thick and large. Mesentery is quite fat and opaque, and large and numerous fat tabs are present. Vessels, which are complicated, are seen with difficulty and are represented by mere grooves in fat.

cases of obstruction. In some of the more extreme cases one will have to decide whether or not to abandon the idea of exploration and simply do an enterostomy; if there is reason to suspect strangulation the exploration must, naturally, be continued. In the course of exploration in very distended cases it may be desirable to empty certain of the more dilated coils. This may be necessary in order to reach the desired portion of the abdomen, to make it possible to bring out a portion of the bowel for purposes of an enterostomy or cecostomy, or to return to the abdominal cavity coils that have been drawn out onto the surface of the abdomen. Owing to the highly septic nature of the intestinal contents in obstruction, however, this is always a dangerous procedure and must not be undertaken lightly. The procedure is best carried out with a needle or small trocar attached by rubber tubing to a suction apparatus; if the aspirating needle is put into the intestine and allowed to drain without suction, leakage around the needle is likely to occur. The puncture wound may be closed with a purse-string suture; gauze packing should be carefully placed to absorb any unavoidable leakage.

Codman,⁶ Cheever¹ and others⁷ advise in selected cases (in general, late cases of small intestinal obstruction) the insertion into the intestine above the point of obstruction of a "Monks tube" or a stiff, woven catheter, over which the bowel may be "threaded" and the distended intestine emptied of its contents. This procedure is carried out, not only with the idea of reducing the distention, but also in order to draw off the toxic contents of the obstructed intestine as a therapeutic measure. The method, however, is not used in most clinics, and should certainly not be attempted by anyone who has not had wide experience in abdominal surgery. (See also Elman.⁸)

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CHAPTER XXII

TREATMENT (Continued)

THE OPERATION (CONTINUED)

PROCEDURES FOR RELIEVING THE OBSTRUCTION. Depending upon the pathology found at exploration, there is great latitude as to the choice of operative procedures. In certain cases, the cause of the obstruction may be directly removed: the division of a constricting band, the untwisting of a volvulus, the reduction of a strangulated hernia or intussusception, may be all that is required and may be very easily carried out. When a direct attack is inadvisable or impossible, the blockage may be indirectly relieved by establishing drainage above the obstruction through an enterostomy or, occasionally, by anastomosis of the intestinal loop above the obstruction to one below, thus side-tracking the intestinal stream around a benign or malignant stricture. Where the intestine has suffered considerably from the effects of obstruction and is dilated and atonic above the blockage, an enterostomy may be indicated in addition to direct removal of the cause of the obstruction, in order temporarily to divert the intestinal stream and prevent overloading and further distention of a portion of bowel that is functionally incapacitated. In cases where extensive interference with the circulation of the bowel exists, resection of the gangrenous bowel and reestablishment of the intestinal continuity may be required in addition to relief of the obstruction; with rare exceptions the anastomosis should be reserved for a secondary operation.

The operations in the recent Massachusetts General Hospital series were grouped somewhat arbitrarily under the following heads: relief of the obstruction only; relief of the obstruction and drainage; drainage only; resection with immediate or delayed anastomosis; miscellaneous procedures which could not be grouped under the foregoing headings. The results are shown in Table XIV.

It will be noted that those cases in which relief of the obstruction alone was carried out had by far the lowest mortality. These cases were, in general, those that came to operation early in the disease.

Resection. Question as to the viability of a segment of intestine comes up frequently in operations for acute obstruction. The cyanotic, engorged appearance of a strangulated loop of gut is characteristic. It may be easy or difficult to decide whether it is viable and should be returned to the abdominal cavity or whether it should be resected. A wrong decision usually means a fatality. (See p. 313.) There are a number of points that help one to decide whether the intestine has been damaged beyond the point where it may safely be conserved.

The peritoneal coat should be observed: if this has lost its normal sheen and glisten, the outlook is not good. Palpation is helpful, for the viable intestine has a certain tone which can be felt and which is replaced in the later stages by a relaxed and sodden feeling. Peristalsis should be looked for: if it occurs, spontaneously or after pinching or applying hot salt solution, it is a favorable sign and indicates that the neuromuscular apparatus has not undergone serious degeneration. Note whether the whole of the involved loop contracts: a small area where pressure has been especially severe may break down even though the remainder of the damaged loop be viable.* The mesentery should be palpated for pulsations in the mesenteric arteries, including the vasa recta at the mesenteric border.¹ The veins should be examined for the presence of clot. Jacques et al.² consider that the consistency of the strangulated intestine and the return of color after relief of the obstruction are the most valuable signs in determining viability. If the circulation is obviously inadequate,† the bowel should

* Eisberg¹ reports a case in which although peristalsis was noted at operation, the gut later became gangrenous. I have seen a similar case, where peristalsis could be seen over the greater part of the loop. When in doubt, resect; or leave the loop outside the abdominal cavity.

† Considerable experimental work has been carried out on animals to determine how much interference with the mesenteric circulation could be produced without resultant gangrene of the bowel. There have also been numerous experiments to determine the

be resected; or, in the desperate cases, the gangrenous intestine may be drawn out onto the abdominal wall and a catheter inserted in the proximal (oral) portion as advocated by Eisberg.¹ In doubtful cases the loop should be wrapped in a sponge with warm saline, and a little time allowed for the circulation to be re-established. Improvement in color and other convincing signs of returning circulation may appear promptly; if they do not appear after a reasonable time, resection should be carried out. There may be an occasional borderline case where one is loath to return to the abdominal cavity a loop of questionable viability, but equally reluctant

point at which ligations of the mesenteric vessels could best be carried out without fatally interrupting the circulation of the bowel.

The early anatomical and pathological studies of Mall,² Welch,⁴ and Dwight⁵ laid the foundation for subsequent work on this subject, particularly as regards the working out of the anastomotic connections in the mesentery and bowel. Most of the more recent experiments have consisted of stripping the mesentery from varying lengths of intestine, or of ligating various mesenteric vessels, and observing the survival period of the animal and the pathological reactions of the involved segment of bowel. Eisberg¹ points out that the ligation of the vessels of the second mesenteric arcades (see Monks' illustration, p. 274), has proved safest in experimental work. This author also feels that trauma to the vessels and prolonged spastic muscular contracture of the gut wall is important in preventing the return of adequate collateral circulation. Another factor pointed out as important is the speed with which vascular occlusion takes place, the gut suffering severely when it occurs rapidly, even though a short segment is involved, while interference with the blood supply to considerable areas is much better tolerated if the vascular occlusion takes place slowly.

Bost⁶ and Rothschild,⁷ repeating older experiments by various investigators,³ separated the mesentery from varying lengths of intestine and found that the animal survived (in one instance, where 8 inches of the intestine had been separated from its mesentery;⁶ the mesentery was sutured back in place and the omentum wrapped about its attachment to the bowel.) It is an old clinical practice to wrap the omentum around intestines whose viability is questionable. The experiments of Scudder⁹ and Wilkie¹⁰ dealt with this point. Wilkie says: "From these experiments I concluded that the wrapping of a portion of the intestine, the viability of which is doubtful, in the great omentum is of some value in preventing gangrene and perforation; but it is a practice whose range of usefulness is distinctly limited." Scudder came to essentially the same conclusion. Apparently in some cases vascular connections are made between the omentum and the damaged gut; but in most cases probably the chief value of the omentum is in preventing the spread of peritonitis.

In regard to the clinical application of this experimental work it should be remembered that most of the ligations of blood vessels and separations of the mesentery were carried out on healthy intestines, while in acute strangulation in humans not only is the mesenteric circulation impaired, but often, also, the capillary circulation in the bowel wall is handicapped by congestion and distention due to the intestinal obstruction.

to carry out the radical procedure of resection of gut that might survive. In such a case, the loop in question may be left outside the abdominal cavity and the peritoneum carefully closed around it. Under these conditions the loop could be promptly removed if it did not survive, and the amount of toxic absorption in the meantime would be small;* if on the other hand it proved viable, it could at a suitable time be returned to the abdominal cavity.

Where a small, localized area of necrotic bowel is found, such, for example, as might result from the direct pressure of a band upon the bowel wall, the damaged area may be infolded without resorting to resection.^{13,14} Where it is not possible to infold the necrotic area, at times a local excision may be carried out without cutting across the mesenteric border of the intestine; the wound is closed as in an end-to-end anastomosis.

In cases requiring resection, great precaution should be taken (by walling off the operative field with gauze and by careful application of clamps) to avoid spilling any of the intestinal contents and so setting up a rapidly fatal peritonitis. A suction apparatus to remove any fluid may be used to advantage.

After a gangrenous segment has been removed and all bleeding controlled, the question as to the re-establishment of the continuity of the intestine arises. In selected cases it may possibly be desirable to proceed at once with an appropriate anastomosis (either end-to-end or side-to-side) by means of suture, or, where time is especially important, by using a Murphy or a Jaboulay button. High fistulae are often poorly tolerated, especially by young children; and at times an immediate anastomosis, although a risky procedure, is better than a delay and secondary operation. In the usual case, however, particularly in dealing with a very sick individual or when technical difficulties to anastomosis exist (as for example when there is great disproportion between the dilated gut above

* The experiments by Travers¹¹ and White and McIver,¹² show that toxic absorption under these conditions is small even when gangrene is present. (See also Eisberg's case.¹)

and the contracted portion below), it is wiser not to attempt to carry out an immediate anastomosis but to bring the severed ends outside the peritoneal cavity, closing the peritoneum

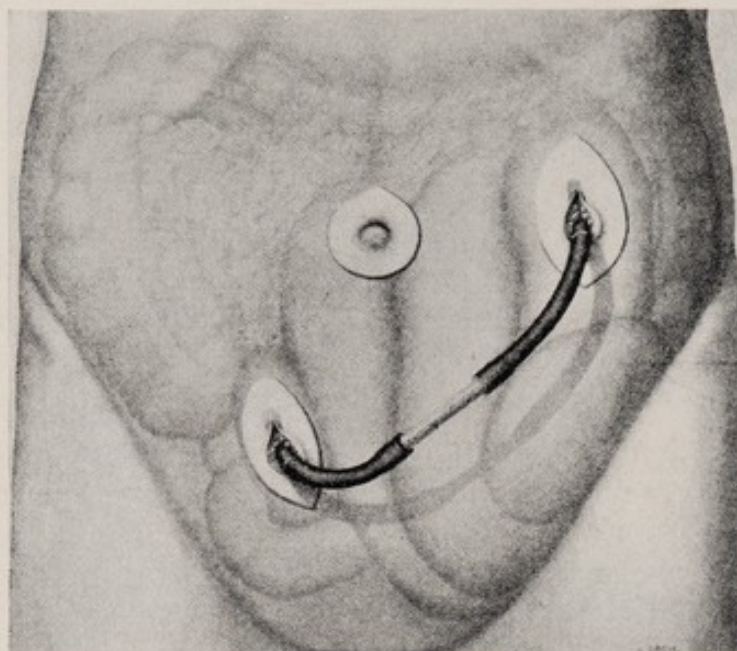


FIG. 57. Device joining enterostomy tubes, permitting secretions from intestine above an obstruction to pass into collapsed gut below. (Wilkie.¹⁵)

around them, and wait until the patient has recovered sufficiently before restoring the intestinal continuity. It may be useful to tie into the ends flanged glass tubes ("Mixer tubes"; "Paul tubes") to lead off the drainage. In certain cases where drainage is profuse, it is possible later to connect the ends of the tubes so that the intestinal stream from above passes into the lower bowel for absorption, and dehydration and malnutrition are diminished (see Fig. 57).¹⁵ Or, one may collect the drainage and inject it into the lower segment. These procedures are particularly applicable to a high fistula; if the fistula is low, so that there is a large absorbing surface above, they are usually not necessary. The secondary operation may be postponed for days or weeks, depending upon the condition of the patient or upon how satisfactorily the fistula can be managed. Shedden¹⁶ has recently called attention to the fact that an extraperitoneal Mikulicz type of operation, where the

dividing walls between the loops of gut are gradually cut through with an appropriate clamp, may be utilized in establishing the continuity of the small as well as of the large intestine. (See also Shelley¹⁷ and Lahey.¹⁸)

In the group of resections in Table XIV, the mortality was high. Once the bowel has become gangrenous there is only a short time interval in which surgery can save the patient; if many hours elapse before operation, so much toxic absorption takes place that removal of the damaged gut is without avail. There were 22 resections in the recent Massachusetts General Hospital series, with 16 deaths, a mortality of 73 per cent. In 9 of these patients the anastomosis re-establishing the continuity of the intestinal tract was carried out immediately; there were 7 deaths. In 13 instances the ends of the intestine were brought out and anastomosis reserved for future operation; there were 9 deaths. Enterostomy was combined with resection in 4 instances.

“Sidetracking” Operations. Indirect operations for relieving the obstruction have already been mentioned. Before taking up the principal one of these, enterostomy, the question of “sidetracking” operations will be considered.

Short-circuiting the intestinal stream around a point of obstruction is particularly indicated where the obstruction is the result of some inoperable neoplasm. Certain cases where the obstruction is caused by such a mass of adhesions and inflammatory reactions that it is unwise to attempt to deal with it directly,^{19,20} and others where the obstruction comes about from some benign stricture, may also be advantageously treated by this method.²¹

At times the sidetracking operation is useful as a first step in a two-stage operation, the attack upon the primary cause of obstruction being reserved for a secondary operation after all obstructive symptoms have subsided.

Turner²² draws attention to the dangers of certain types of exclusion operations, where by lateral anastomosis the ileum is joined to the transverse or left side of the colon because of

a malignant growth in the proximal portion of the colon. The author makes the point that a cecostomy should be carried out in this case; for occasionally the cecum, lacking a vent (the ileocecal valve at times preventing any discharge back into the ileum), will be so greatly distended that it becomes gangrenous or bursts. (See also Morison's article.²³) Stretton²⁴ reports a case where, following the transplantation of ileum into the sigmoid, a blind pouch composed of cecum and a portion of colon became so distended that resection was required. Codman's case²⁵ where a permanent cecostomy was left is of interest. Complications²⁶ may follow a sidetracking operation (ileo-ileostomy or ileocolostomy) for benign obstruction of the terminal ileum. Holm²⁷ has reported serious pathological changes occurring in the blind loop of ileum, and advises resection of the "sidetracked" ileum if possible, either as a part of the primary operation or as a second stage.

TABLE XIV
OPERATIVE PROCEDURES
(156 CASES, MASSACHUSETTS GENERAL HOSPITAL TEN-YEAR SERIES)

<i>Type of Operation</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality (Per Cent)</i>
Relief of obstruction, only.....	68	13	19
Relief of obstruction and drainage of the bowel....	27	15	55
Drainage of the bowel, only.....	33	19	58
Resection } Immediate anastomosis.....	9	7	} 73
} Delayed anastomosis.....	13	9	
Miscellaneous†.....	6	5	83
Total.....	156	68	44

It will be noted that those cases in which relief of the obstruction alone was carried out had by far the lowest mortality: these cases were for the most part those that came to operation early in the disease. The higher mortality in the other groups indicates in general the seriousness of the condition found at operation and is not due to poor choice of operative procedure.²⁸

† There were 2 cases in which because of the condition of the patient the operation was abandoned; and one case in which due to a diagnostic error the obstruction was not relieved.

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CHAPTER XXIII

TREATMENT (Continued)

THE OPERATION (Continued)

ENTEROSTOMY. Opening a distended segment of intestine above the point of obstruction and so establishing drainage is one of the oldest and most primitive procedures for the relief of intestinal obstruction. The fact that in selected cases the operation still has an accepted place shows upon what a sound physiological basis it rests: namely, the relief of increased intra-intestinal pressure with its resultant damage to the capillary circulation and the bowel wall.

Indications for the Use of Enterostomy. Enterostomy has a wide field of usefulness¹⁻⁴ and is employed under a number of different circumstances that may conveniently be considered in the following groups:

1. Not infrequently patients with obstruction are brought to the surgeon late in the disease, when they are desperately ill and obviously in such a condition that no extensive operation could be considered. Most of these patients will die whatever is done; but an enterostomy without exploration will occasionally effect a recovery (provided, of course, that no strangulation exists), or bring about sufficient improvement so that more extensive procedures can be carried out later.

2. Enterostomy may be used as a supplementary procedure under various conditions, as follows:

In cases where, after the obstruction has been freed, the bowel is found to be atonic, dilated, and functionally handicapped by the effects of the obstruction, it is often wise to do an enterostomy to relieve the tension and distention and allow the bowel to recover its tone.*

* In 1812 Travers, speaking of strangulated hernia, wrote:

"First, that there is a state of the intestine wanting the criteria of gangrene, in which the operation does not arrest the symptoms caused by obstruction. Secondly, that such intestine when examined after death remaining unaltered, no signs of disorganization being present, there is much reason to suppose that it has been palsied by the

Where an anastomosis has been carried out, an enterostomy above is often a factor of safety in protecting the suture line against damage from distention.

In cases where a resection has been carried out, an immediate anastomosis may for various reasons be unwise. Here, quite a different type of enterostomy is indicated: namely, the bringing of both ends of the intestine outside of the abdomen, the anastomosis being delayed for a more favorable occasion.

3. The use of enterostomy in obstructions that occur early after abdominal operations will be discussed in Chapter XXIV. In brief, it may be said that an extensive exploration or attempted lysis of newly formed inflammatory adhesions is often dangerous and unnecessary; enterostomy is frequently the only procedure indicated.^{5,6}

4. The value of enterostomy in peritonitis will presently be discussed in some detail. It may be said in passing that the operation has its greatest usefulness in cases where there has been some localization of the infection, the obstructive symptoms being due to recent inflammatory adhesions and atony of those intestinal coils in immediate contact with the infected area.

Location of the Enterostomy. The location of the enterostomy must depend upon the circumstances and findings in the individual case, and upon the level of the obstruction. When the obstruction is located in the colon, a cecostomy is usually the operation of choice; when the obstruction is high in the small intestine, the enterostomy must necessarily be high; when the obstruction is low in the small intestine one can, under varying

duration and severity of the stricture. Thirdly, that the return of a bowel unequal to the resumption of its muscular or peristaltic action, counteracts the intention and defeats the success of the operation, for though the strangulation is removed, the only dangerous consequence of it, the obstruction, continues." (See page 126.)

Travers, quoting Bichat, says: "If we consider the paralysing effect of acute inflammation attacking a muscle; the inability of the oesophagus, the urinary bladder and other parts to obey their natural stimuli when so affected; we shall have no difficulty in accounting for the torpor of a bowel recently released from strangulation. Not only is the tissue of the organ gorged with a preternatural quantity of blood, but of blood unfitted by its stagnation for the maintenance of the vital functions." (Travers, 1812.)

circumstances, elect to make the enterostomy low, near the seat of the obstruction, or high, in the jejunum. At times the best that can be done is to utilize the first distended loop of gut presenting itself in the wound. When enterostomy is being carried out to protect an anastomosis, it had best not be placed at too great a distance above the suture line.

The older idea in regard to the best location for the enterostomy was that it should always be made in the segment of intestine just above the point of obstruction; in other words, since most of the obstructions are situated in the lower portions of the intestinal tract, it should be a low enterostomy. This is a logical view, for since drainage from a distended intestine depends upon peristaltic action, it would seem that if a vent were placed near the point where the contents are arrested, the peristalsis would continually push material out through the artificial opening.

For a number of years, however, the older dictum as to the location of the enterostomy has been questioned. In 1910 and 1916, Bonney^{7,8} advocated placing the enterostomy, not low in the intestine near the point of distention, but high in the jejunum. His contention was that the segment near the obstruction usually contained gas only, while the greatest accumulation of fluid occurred in the jejunum; and that the jejunum, therefore, was the logical place to establish drainage. In 1913, McKenna^{9,10} also advocated jejunostomy, under the belief that duodenal secretions were responsible for the toxemia and that a jejunostomy was the best means of draining the duodenum. Both Bonney and McKenna reported cases successfully treated by jejunostomy.

Just why a jejunostomy may be more successful than an opening nearer the point of obstruction is not altogether clear. The idea that the symptoms of obstruction are caused by a toxic secretion from the duodenal mucosa has been generally abandoned; and Bonney's idea that the coils of intestine near the obstruction contain only gas is often not borne out by observation at operation. It is true, however, that the greatest

outpouring of fluids occurs high in the intestinal tract, and that drawing this off by a jejunostomy prevents over-distention of the lower intestinal segments. Whatever the reason back of

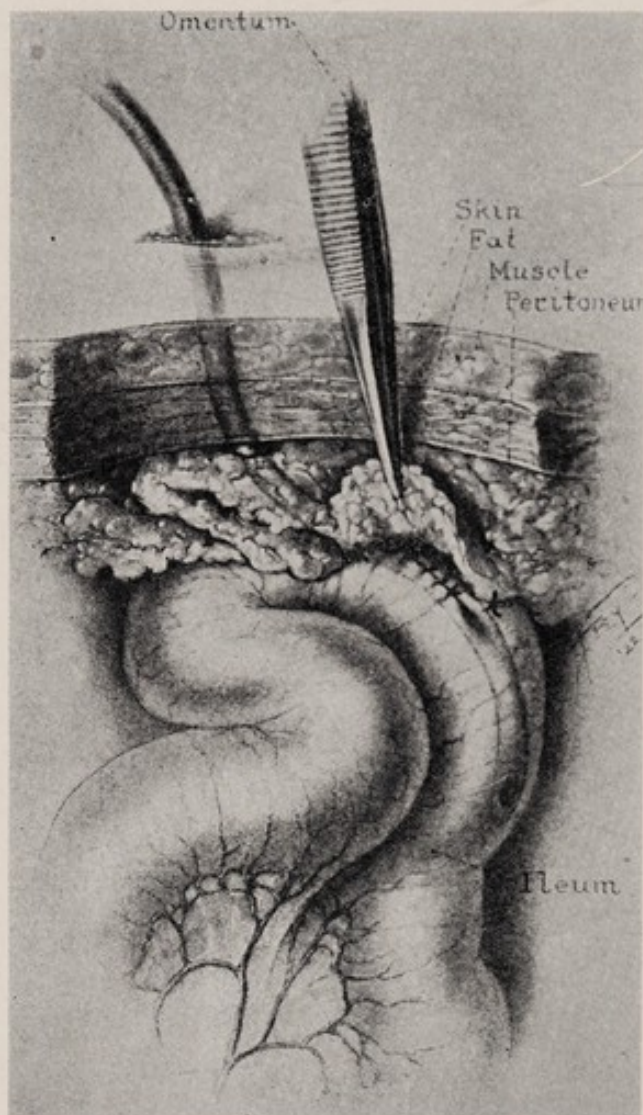


FIG. 58. Mayo's technique for catheter enterostomy. (Mayo.¹⁴)

it, certain cases do brilliantly with a jejunostomy;¹¹ it is especially useful in those cases where peritonitis plays a rôle. The use of a valvular type of enterostomy lessens one serious objection to the operation, namely, an uncontrollable loss of fluids, and also prevents the digestion of the abdominal wall

by active ferments that so often occurred with the older type of enterostomy when it was placed high in the intestinal tract.

Technique. When the opening into the small intestine is not intended to be permanent, the valvular type of enterostomy, where a catheter is employed, has largely replaced the older methods, where the opening into the intestine was sutured to the edge of the abdominal wound or a flanged glass tube was tied into the intestinal opening. The valvular enterostomy is usually performed according to the method described by Long¹² and Mayo,¹³ which is based on the Witzel technique for performing a gastrostomy (Fig. 58).¹⁴ If the loop is distended it may be desirable to empty it by inserting a needle attached to a suction apparatus. If possible, intestinal clamps should be applied before the opening for the insertion of the catheter is made, one clamp being applied first, the contents milked out of the section of bowel that is to be opened, and then a second clamp applied.⁶ Placing a purse-string suture which may be drawn tightly around the catheter as soon as it is inserted is at times a useful manoeuvre in preventing leakage while the bowel is being infolded around the catheter. On completion of the intestinal suture, the free end of the catheter should be passed through a small opening in the omentum; the omentum is then tucked down over the suture line and around the point where the catheter emerges from the gut wall. This is important in preventing contamination of the peritoneal cavity and in ensuring prompt closure of the enterostomy when the catheter is withdrawn. The intestine may or may not be anchored to the peritoneal edges of the abdominal wound.

When the valvular type of enterostomy has been used, prompt closure usually follows withdrawal of the catheter. When this does not take place, there are various procedures which may be employed (see p. 316 under "Complications").

When a tube is being used in performing a cecostomy, care should be taken that it is of large size, for lumps of solid material in the colonic contents readily plug a small opening.

Care of the Enterostomy. If the enterostomy is to accomplish its purpose, it is essential that satisfactory drainage take place. Fundamentally this depends upon the peristaltic action of the bowel; but constant care is required to make sure that when a tube is used no kinking or blockage of its lumen takes place. The injection and aspiration every few hours of a small quantity of salt solution will at times be found effective in maintaining drainage.

When the more acute symptoms of obstruction subside and the foul drainage ceases, normal saline or other types of nutritive solutions may advantageously be injected into the bowel through the enterostomy opening and so assist in maintaining the body fluids and general nutrition.¹⁵

Value of Enterostomy in Acute Peritonitis. In approaching the subject of the value of enterostomy in acute peritonitis, one should realize that the situation is complex. The peritonitis may be widespread or sharply limited. At times an abscess exists, surrounded by coils of small intestine. Acute peritonitis may affect intestinal motility in two ways: it may cause a functional disturbance through atony of the bowel; or it may produce light adhesions which interrupt the intestinal movements by kinking or twisting the bowel. Both these factors are often present. See Figs. 47 and 48.

Most cases of diffuse, acute peritonitis show some degree of functional disturbance of intestinal motility,* although at times the disease runs such a fulminating course that this aspect does not have time to assume any importance in the clinical picture; the death is then obviously one from infection and is comparable to death due to infection of any of the large serous cavities such as the pleura or the pericardium. A general atonic dilatation of the whole intestinal tract, however, often occurs (the so-called "paralytic ileus") which may result in symptoms of obstruction: abdominal distention,

* In considering a patient with peritonitis it should not be forgotten that a quiet intestine and a moderate degree of distention may possibly be advantageous in walling off or limiting the infectious process.

vomiting and obstipation. The outcome in these cases depends chiefly on the ability of the patient to overcome the infection; if that takes place, the bowel will usually resume its normal activity. (See "Functional Obstruction," p. 200.)

If, however, the course of the disease in these patients with paralytic distention be carefully observed over several days, a few cases will be noted in which, although the patient is successfully convalescent and is localizing the infection, distention and profuse vomiting continue. On auscultation, gurglings can be heard and the patient may complain of gas pains; enemas give unsatisfactory results. These symptoms point toward the development of a mechanical element in the obstruction, due to light fibrinous adhesions;* and an enterostomy is often a lifesaving procedure.⁵ There are also comparable cases in which from the outset the peritonitis is to a large extent limited to the lower abdomen or pelvis and involves only those coils lying in the immediate vicinity of the inflammatory process (Handley¹⁷† and Wilkie¹⁸). In this type of case the symptoms of obstruction often arise during the convalescence from operations for acute appendicitis; further discussion of these cases will be found under "Early Post-operative Obstruction," p. 296, where an enterostomy is recommended.

In the foregoing cases, where the peritonitis is at least partially localized and where there is a mechanical element in addition to the atony, an enterostomy undoubtedly gives excellent results. The debatable point comes in regard to the group where a diffuse peritonitis alone causes varying degrees of intestinal atony, distention and vomiting. There has been a tendency on the part of some authors^{20,21} to advise an enterostomy in practically all cases of peritonitis in the belief that a high percentage of patients die not from peritonitis but

* Wilkie has shown experimentally that this type of adhesion is quite capable of producing obstruction.¹⁶

† Handley¹⁷ and Anspach¹⁹ have advised sidetracking the intestinal stream around inflamed, atonic coils by an anastomosis of the small intestine above the point of involvement to the colon below.

from a secondary, functional obstruction. The soundness of this view is open to question. Certainly an enterostomy performed on a patient dying of toxic absorption from an infected peritoneum is a futile gesture which can only hasten the end. Also, if the whole bowel is atonic, it is unreasonable to expect that satisfactory drainage from the enterostomy tube will take place in the absence of the propulsive force of peristalsis.

As a practical matter of fact it must be admitted that it is often difficult, clinically, to pick out the case with peritonitis that will benefit by enterostomy. If the distention is pronounced and does not yield to simple methods such as poultices and rectal tube, and if vomiting is profuse, an enterostomy done under local anesthesia is at times justified and should not be delayed until the patient is moribund. In the presence, however, of an advancing, fulminating peritonitis, and in the absence of any sign of peristaltic action (cramp-like pains, audible or visible peristalsis), the results will often be disappointing;²² and in certain of these cases perhaps as much can be accomplished by means of a small tube inserted into the stomach through the nares, as by an enterostomy. See pages 212 and 259.

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CHAPTER XXIV

TREATMENT (Continued)

POINTS IN THE TREATMENT OF CERTAIN TYPES OF OBSTRUCTION

EARLY POSTOPERATIVE OBSTRUCTION. The type of operative procedure to be selected for the relief of early postoperative obstruction depends, obviously, upon the general condition of the patient and the findings within the abdominal cavity at the time of operation. Certain broad principles, however, may be applied. Since these obstructions for the most part depend upon recently formed adhesions, usually accompanied by a certain amount of functional atony, if the bowel can be adequately drained, the peristalsis above the point of obstruction will be quieted and the propulsion of more material into the obstructed segment prevented. The secondary changes in the bowel wall resulting from distention will also be prevented. In certain cases the use of the duodenal tube with suction (see pp. 212 and 259) will serve as both prophylaxis and treatment. In the more serious cases, or where the patient does not respond to this method, an enterostomy above the point of obstruction is frequently all that is required; and this can, of course, be carried out under local anesthesia without a thorough exploration, which is of great advantage in a very sick patient. Symptoms of obstruction may subside after such a drainage operation, the light adhesions being absorbed and the bowel regaining its tone without further procedures. How much exploration is required is a matter for the operator to decide: it depends upon a number of factors, particularly upon the condition of the patient and the presence or absence of acute peritonitis.

Interference with the mesenteric circulation is rare in these cases; thus if no bloody peritoneal fluid is found it is presumptive evidence that strangulation is not present; when strangu-

lation does occur, a rising white blood count may indicate its presence.

As already stated, it is dangerous to attempt to separate a mass of inflammatory adhesions: the bowel is exceedingly friable and is likely to tear; and since its contents are highly infectious, if such an accident does occur it is likely to be followed by a fatal peritonitis. There is also the danger that in an attempt to free an involved segment of bowel, a localized area of infection may be opened with a resulting spread of the peritonitis.

Occasionally a volvulus may be present or herniation through some internal opening may have occurred.¹ While these types of obstruction are rare in the early postoperative group, the operator should bear in mind the fact that they are at times encountered. Volvulus occurred postoperatively in 3 instances in the 1918-1927 Massachusetts General Hospital series. In one case a jejunostomy was performed without exploration; the symptoms were not relieved, and a later exploratory operation revealed the twist; the patient died. The two other postoperative volvuli were discovered and relieved at exploration; these patients recovered.

The operations carried out in the remaining 37 cases of early postoperative obstruction at the Massachusetts General Hospital were as follows:

	CASES	DEATHS
Enterostomy alone.....	17	9
Enterostomy with lysis of adhesions.....	10	5
Entero-enterostomy with lysis of adhesions.....	1	0
Lysis of adhesions alone.....	9	3
	—	—
Total.....	37	17

OBSTRUCTION FROM INTUSSUSCEPTIONS. The operation for intussusception, in cases received early, is one of the easiest in surgery, namely, reduction of the intussusception by taxis. The invaginated bowel should be "milked" out, not pulled out; occasionally slight traction is necessary to remove the last portion of ileum from the cecum. After reduction of the

intussusception, the bowel should be carefully palpated to see if a polyp or diverticulum or tumor can be felt; these structures are not infrequently responsible for enteric intussusceptions, and may cause recurrences.²

The difficulties are encountered in the later cases, where reduction is impossible, due to edema and swelling, or where the various layers are glued together by inflammatory adhesions. In Bolling's³ experience most of the difficulties in reduction encountered in his operative series were due to swelling and edema rather than to adhesions. These irreducible intussusceptions, frequently gangrenous, may be dealt with in a number of ways; but all ways show a high mortality. The simplest and most direct method is a resection of the intussusception, followed by immediate or delayed anastomosis. This is particularly applicable to adults with enteric intussusception, a rather rare variety. In the ileocecal and ileocolic varieties (types of intussusception usually found in children) this procedure is too formidable. It must be emphasized that any extensive procedure in a young child is almost certain to be fatal. The method advocated by Coffey,⁴ namely, resection of the intussusception after delivering the intussusceptum through an incision in the outer layer, is a radical procedure and involves contamination with the septic bowel contents. This latter objection applies also to Brown's⁵ suggestion that an incision be made into the cecum, freeing the constricted neck of the intussusception.*

A method recently suggested by Montgomery and Mussil⁶ would seem to have possibilities: these authors advise taking advantage of Nature's method of occasionally effecting a spontaneous cure,† and propose merely fixing the intussusception in place by interrupted sutures at the neck, then making an anastomosis between the ileum and the colon, distal to the intussusception. The intussusceptum presumably sloughs off into the intestine; in their cases it caused no further symptoms. They report 2 cases in which operation was successfully carried

* Brown and Coffey reported only one case each.

† See p. 80.

out by this method. Another operation was suggested by Codman⁷ in 1908 for use in irreducible cases in which the small intestine was invaginated into the large intestine. This con-



FIG. 59. First stage of Mikulicz operation for volvulus of a sigmoid megacolon. (Weeks.⁸)

sists essentially in ligating and dividing the impacted mesentery close to the neck of the intussusception, dividing the bowel between clamps and bringing the ends out of the abdominal wound; after a few days, when the intussusceptum has become gangrenous, it can be drawn out of the intussuscipiens by gentle traction. The procedure was advocated particularly for desperate cases, to replace resection and hopeless efforts at reduction. Shelley⁹ has reported the successful use of a modified Mikulicz resection in a late irreducible case with perforation.

Any one of the foregoing methods seems better, in irreducible cases that are not suitable for the ordinary type of

resection, than merely doing an enterostomy. Enterostomies are usually of little value in any case, and are of none when the intussusception is not reduced.¹⁰ Occasionally a life may be saved by drawing the intussusception out onto the abdominal wall and doing an enterostomy in the distal end of the gut.

OBSTRUCTION BY VOLVULUS. The extent of the operation for volvulus of the small intestine depends upon the pathological condition found at operation; untwisting of the loop and division of any bands and adhesions may be all that is required. If the circulation has been so damaged that the bowel is not viable, resection must be carried out or the bowel must be extraperitonealized.

In volvulus of the sigmoid, where the bowel is viable the question of recurrence of the twist must receive consideration. Deaver and Magoun¹¹ have advised a lateral anastomosis between the proximal and the distal limbs of the loop, with later resection of the loop if the volvulus recurs. Where the twisted loop of sigmoid is not viable, it had best be drawn out of the wound and a Mikulicz type of resection carried out. (See illustration of Weeks'⁸ case showing resection by the Mikulicz method in volvulus of a sigmoid megacolon; Fig. 59). It may be well to perform a cecostomy as part of the first operation. Bloodgood¹² stresses the importance of the careful division of adhesions and of covering the raw surfaces with peritoneum as a prophylaxis against recurrence. It is often a useful procedure to pass a tube upward through the rectum and draw off the contents of the loop after the sigmoid has been untwisted.

In volvulus of the cecum, a cecostomy should usually form part of the operation: it not only drains the obstructed loop, but anchors the cecum so that recurrences are not likely.¹³

OBSTRUCTION FROM MESENTERIC THROMBOSIS OR EMBOLISM. The patient with obstruction from mesenteric thrombosis or embolism is often desperately ill; and local anesthesia is desirable. The operative procedure usually consists of resec-

tion of gangrenous intestine, with or without immediate anastomosis. In the more desperate cases, the gangrenous intestine may be drawn outside the peritoneal cavity and the wound closed around its mesentery. Extremely rarely, recovery takes place without resection,^{14,15} for occlusion of a mesenteric vessel is not always followed by infarction: the collateral circulation may be sufficient to maintain the blood supply to the bowel. In a case of this type reported by Ross,¹⁵ an exploratory operation was performed; after inspection of the involved loop, it was decided that the circulation was sufficient to prevent necrosis; no further procedures were carried out, and the patient recovered.* (See p. 137.)

OBSTRUCTION BY NEOPLASM. It is now well established that operative procedures upon cases of acute obstruction of the large intestine by neoplasms should be directed toward draining the bowel above the point of obstruction, usually by a colostomy or a cecostomy, without any attempt at a direct attack upon the tumor. This procedure can usually be carried out satisfactorily under local anesthesia. It is wise to wait for from ten days to two weeks after this first operation, so that all symptoms of obstruction may subside and the bowel regain its normal tone, before attempting a radical operation upon the neoplasm. Occasionally it will be possible in obstructions of the cecum or ascending colon to carry out, as a first stage operation, a lateral anastomosis between the ileum and the transverse colon, diverting the intestinal stream around the point of obstruction. A supplementary ileostomy may be indicated.

In obstructions from metastatic carcinomas, only palliative procedures, of course, are to be considered. These might consist either of simple drainage above the point of obstruction, or of some type of anastomosis sidetracking the intestinal stream around the point of obstruction.

* The author has recently operated upon a similar case where the involved loop of bowel seemed viable; an enterostomy was carried out to relieve the distention; the patient recovered.

In the rare cases in which a primary tumor obstructs the small intestine, it is usually possible to excise the growth at the first operation. The continuity of the intestinal tract may be re-established at the same time or later, at the discretion of the operator.

OBSTRUCTION BY STRANGULATED EXTERNAL HERNIAS. For strangulated external hernias, local or spinal anesthesia is usually the anesthetic of choice; although if the case is seen early a general anesthetic is frequently safe.

In the uncomplicated cases, all that is required in the way of operative procedures is exposure and opening of the sac, cutting of the constricting ring, followed by reduction of the hernia and repair of the defect. In the cases where the intestine

TABLE XV
OPERATIVE PROCEDURES AND MORTALITY IN 19 CASES OF STRANGULATED EXTERNAL HERNIA WHERE NECROSIS OF THE BOWEL WAS FOUND AT OPERATION (MCIVER¹⁶)

<i>Type of Operation</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality</i>
Resection, ends brought out.....	7	5	71 per cent
Loop of questionable viability brought outside peritoneum.....	1	0	0
Resection, immediate anastomosis.....	8	5	62 per cent
Inversion of necrotic area and enterostomy 8 inches above repair.....	1	1	100 per cent
Small necrotic area perforated; catheter placed in opening.....	1	0	0
Inversion of small necrotic area.....	1	0	0
Total.....	19	11	58 per cent

is found to be gangrenous, it must be resected. Following this, immediate anastomosis may be carried out, or the ends of the intestine may be brought outside the wound and anastomosis reserved for a secondary operation: the decision as to which procedure is preferable in the individual case, and as to what extent the operation may be justifiably prolonged, must rest on the judgment of the surgeon. An enterostomy is frequently indicated in the late case or where an anastomosis has been

carried out. In the Richter type of hernia, where a part of the bowel wall alone may be necrotic, the area of necrosis may be infolded without resort to resection, or the necrotic portion removed by excision of a v-shaped section of the bowel wall, leaving intact the mesenteric border.

Table xv shows the operative procedures and mortality in 19 cases of strangulated external hernia where necrosis of the bowel was found at operation.

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CHAPTER XXV

TREATMENT (Continued)

CARE OF THE PATIENT AFTER OPERATION

When ether has been used as the anesthetic for the operation, the patient should be carefully watched during the recovery stage to guard against the aspiration of vomitus.

As already stated, maintenance of the body temperature, both in the operating room and after returning to the ward, is extremely important. The frequent pulse and blood-pressure records that have presumably been taken during the operation should be continued on the ward until the danger of "operative shock" is past.

ADMINISTRATION OF FLUIDS. Everything that was said in the chapter on preoperative care of the patient, in regard to combating dehydration by the administration of salt solution, applies with equal force to the treatment after operation. In the more serious cases, the normal saline solution may be administered subcutaneously, intravenously and by rectum. When the body has been seriously depleted of water and the essential sodium and chloride ions, it is important that this loss be corrected as soon as possible. As to the amount of salt solution to be administered, no didactic rules can be laid down: it depends upon the extent of the dehydration when treatment is started, and upon the daily output of fluids. One should take into account the clinical evidence of dehydration and correlate this with the laboratory findings; in the severe cases, 4 to 6 liters may not be too much in the course of twenty-four hours; the milder cases will naturally require much less.

A careful chart should be kept of the intake (by all routes) and output of fluid, the latter including a record of both vomitus and urine. The keeping of this chart in twenty-four-hour periods is a simple matter and the record furnishes valuable data for use in determining the degree of dehydration and

TABLE XVI

Day post op.	Output				Intake		Remarks
	Vomit (c.c.)	NaCl (gm.)	Urine (c.c.)	NaCl (gm.)	Fluid (c.c.)	NaCl (gm.)	
3rd	3500	25.2	No record	No record	5000	27	Plasma chlorides 503 NPN 57
4th	3600	25.9	No record	No record	5000	27	Plasma chlorides 440
5th	700	4.5	No record	0	5000	45	Patient being treated by small stomach tube
6th	No record	No record	No record	No record	5000	45	
7th	600	3.7	No record	0	4000	36	Plasma chlorides 500
8th	4166	26.9	310	.6	2500	18	Plasma chlorides 560
Jejunos- tomy	Intestinal Drainage (c.c.)						
9th	2240	15.96	520	3.25	4420	39.7	Marked clinical im- provement. No vomiting
10th	2700	19.2	550	1.48	3160	24.3	Plasma chlorides 595
11th	4500	28.0	250	4.5	5315	34.3	Taking a small amount of fluid by mouth
12th	3400	18.5	400	2.3	2600	19	Plasma chlorides 630
13th	5800	13.3	590	2.0	4000	10	
15th	1100	7.5	No record	No record	3515	10?	Taking semi-solid food
17th Enteros- tomy tube re- moved							Bowels moving nor- mally

Case No. 279896. M. G. H. Intestinal obstruction following appendectomy with drainage for acute appendicitis. The appendix was ruptured. Second operation (8th day after appendectomy): jejunostomy. Enterostomy tube removed on ninth day after

in planning further treatment. A low volume of urine is usually conclusive evidence that the patient is not receiving sufficient fluid. When a high enterostomy has been carried out, the amount of drainage in the course of twenty-four hours may be surprisingly large (an amount greater than 5 liters has been reported; see Table xvi) and of course demands adequate replacement. In adding up the total fluid output for a twenty-four-hour period one should not forget that considerable quantities are lost by evaporation from the skin and lungs; the amount is particularly large (sometimes as much as 2 or 3 liters) in patients who are sweating, and should receive consideration in an estimate of the quantity of fluid to be replaced. The importance of loss of fluid in this way has been stressed in recent articles by Coller and Maddock.^{1,2}

The administration of fluids by mouth should in all but the simplest cases be carried out with caution, in order to be certain that the intestine has regained its tone. For a number of hours after the operation it may be wise to withhold fluid by mouth entirely, except for wetting of the mouth and throat for relief of thirst, and then to give only a few sips of water at a time until it is certain that it will be tolerated. When it is evident that the patient is able to assimilate water, other liquids may be added. Milk should be withheld for a number of days: it produces a bulky residue and tends to cause distention.

Distention. Convalescence after operation for obstruction may be stormy, particularly in late cases or where peritonitis complicates the picture: vomiting and abdominal distention are frequently encountered. It is important that any dilatation of the stomach be recognized and early treatment instituted, not only because if unrelieved it may lead to more generalized intestinal distention, but also because it may itself give rise to serious symptoms.³ Chapter xvii, dealing with the prevention

jejunostomy; bowels moving normally. This case illustrates the large amount of fluid and sodium chloride that may be lost in the vomitus and intestinal drainage. It emphasizes the fact that large quantities of fluid containing sodium chloride may be needed for adequate replacement. (Studies in this case carried out by Dr. J. C. White.)

and treatment of functional obstruction, and the section on the use of the duodenal tube (p. 259) should be consulted; see also pages 175 and 314.

Drugs. There are no drugs which are especially indicated during the convalescence. During the first twenty-four to forty-eight hours after operation there is no contraindication to the use of morphine for relief of the pain incident to the operation, and it may be of benefit in increasing intestinal tone and in combatting distention.*

Laboratory Studies. Blood samples should be taken for estimation of the chlorides. It is helpful also to have carbon dioxide determinations to indicate the presence of alkalosis or acidosis; and hematocrit readings may be of use in showing the extent of water loss from the plasma. The non-protein nitrogen determinations should also be made. The blood chloride and non-protein nitrogen measurements are usually of the most practical importance, furnishing as they do some indication as to the extent of the loss of chlorides and the degree of the dehydration; but as has already been pointed out, since the total volume of body fluid is reduced in dehydration, the measurements of the plasma chlorides do not indicate accurately the extent of their loss from the body.†

* See p. 212.

† See p. 163.

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CHAPTER XXVI

TREATMENT (Continued)

COMPLICATIONS

Any of the complications associated with laparotomies may occur in connection with operations for obstruction. There are, however, certain ones that are peculiar to intestinal obstruction or have special importance in relation to it, which will now be taken up. A number of these have already been discussed, but will be mentioned again for the sake of completeness.

The complications may conveniently be divided into those that occur at operation and those that manifest themselves postoperatively.

COMPLICATIONS OF THE OPERATION. *Anesthesia.* The complications from anesthesia have already been discussed.*

Shock. Shock is most frequently encountered following operative procedures and anesthesia, or as a terminal manifestation in the untreated disease. The symptom complex is characterized by grayish pallor, cold extremities, sweating, and a rapid pulse rate accompanied by a low systemic blood pressure. The type of anesthesia, length of operation and amount of manipulation of the intestines, particularly evisceration or traction on the mesentery, are factors of importance in its production. The escape (as the result of operative accident) of any of the highly septic intestinal contents into the abdomen introduces the element of fulminating peritonitis; and, as is known from experience gained during the World War, infection may at times be the predominating factor in the onset of shock.¹

Aside from the complication of infection, why are patients with intestinal obstruction so prone to develop shock following operation? The answer must be that when the disease is well established, the adjustments that have occurred in order to

* See p. 261.

maintain the systemic blood pressure are taxed to their limit and the margin of safety is small. Once the compensation is broken, the collapse is likely to be rapid and irreversible. Patients with intestinal obstruction resemble in many respects those who come to operation following trauma, with a loss of blood that is significant but not sufficient to lower the systemic blood pressure: it is well known both clinically and experimentally that the further loss of even a relatively small amount of blood, or at times even brief operative procedures or anesthesia, may throw the patient into a severe state of shock. (P. 262.)

The close analogy between patients with intestinal obstruction who develop shock following operation and patients who develop shock following hemorrhage and trauma is probably due to similarity in the underlying conditions. It is well established that a low blood volume is one of the most constant and fundamental findings in traumatic shock and hemorrhage.² In intestinal obstruction, the following factors tend to lower the blood volume. First, there is the great outpouring into the intestines of fluid and inorganic constituents of the digestive juices,* which are to a large extent lost in the vomitus. Second, in addition to this direct loss of fluid, the puddling of blood in the splanchnic area is often considerable: the blood vessels of the intestine above the obstruction are usually found, at operation, to be engorged, while cyanosis often furnishes direct evidence of capillary stasis; Elman³ believes that the sudden decompression of a distended intestine greatly favors the stagnation of blood in injured capillaries. Third, in infarctions from venous occlusions, in addition to the stagnation of blood in the vessels, extravasation into the bowel wall and intestinal lumen takes place; there is also usually considerable accumulation of bloody fluid in the peritoneal cavity.

In an estimation of the significance of these vascular changes in the splanchnic area, the experiments of Erlanger and

* It has already been pointed out that this may equal two or three times the initial plasma volume of the animal. The replacement takes place from the interstitial tissue fluids and probably taxes to the limit this source of fluid.

co-workers⁴ and of Beard and Blalock⁵ are helpful. Severe degrees of shock were produced in their experimental animals by various types of procedure causing direct injury to the abdominal viscera (usually by exposure and manipulation of the intestines). Erlanger and his co-workers⁴ also studied shock following occlusions of the inferior vena cava and the aorta. In shock produced by manipulation of the intestines, these authors consistently found great engorgement of the splanchnic area, the capillaries and venules of intestinal villi being distended and packed with red corpuscles; a considerable loss of fluid from the bowel as a result of transudation was also noted. They considered these findings of sufficient magnitude to account for the reduction of the effective blood volume to a point where failure of the circulation occurred. On this subject see also the work of Mann,⁶ Wiggers,⁷ and Scott and Wangensteen.⁸

It seems reasonable to suppose that the mechanism of shock under the foregoing experimental conditions has many points in common with the shock developing in connection with intestinal obstruction.

Another element in the production of shock in cases of intestinal obstruction must be considered, direct proof of which is lacking, but in favor of which many plausible arguments may be advanced. This theory assumes that some toxic substance is absorbed from the obstructed bowel (see Part III), which causes, among other systemic effects, dilatation and increased permeability of the capillaries, with a resultant lowering of the blood volume.

Whatever the exact mechanism of shock (and there seems good evidence for believing that a number of different factors enter into the situation), it is probable that a reduction of blood volume is fundamental. This conception has direct practical importance in relation to treatment: it implies that efforts should be directed toward restoring the normal volume of circulating fluid as rapidly and efficiently as possible.

See also p. 391.

Errors of Technique or Judgment. One of the most serious errors of technique that can occur is contamination of the peritoneal cavity with intestinal contents. Normally the contents of the upper small intestine are relatively sterile; but an obstructed intestine at any level contains a virulent, mixed bacterial flora which is highly septic; and a rapidly fatal peritonitis usually follows any soiling of the peritoneal cavity with this material. This accident, as shown by autopsy records, is responsible for many deaths in patients operated upon for obstruction. One of the common ways in which contamination occurs is by perforation or tearing of the gut during manipulation. The attempt to separate adhesions attached to friable, inflamed bowel is a dangerous procedure. Contamination while an enterostomy is being carried out or when the gut is punctured for any cause, is to be carefully guarded against: the intestinal loop should always be carefully walled off by gauze sponges before being punctured, so that the extent of the contamination is limited if any untoward accident occurs. At times it is wise to empty the loop that is to be opened by means of an aspirating needle attached to a suction apparatus.

The difficulties in arriving at a decision concerning the viability of the gut in certain borderline cases where the mesenteric circulation is damaged have already been discussed.* The following case, quoted from Eisberg,⁹ illustrates the results of a wrong decision:

CASE XXXV. *Illustrating mistake as to viability of bowel (Eisberg).*

S. N. 9. Male, aged sixty-four. Type of obstruction: strangulated right femoral hernia. Pathology found at operation and operative treatment: hernial sac contained several cubic centimeters of serosanguineous fluid and 20 cm. of black gut with strangulated omentum. Omentum resected. Luster and color returned to intestine after application of hot pads sufficiently to warrant its replacement. Postoperative course: patient died four days after operation with signs of paralytic ileus. Wound inspection: gangrenous segment of gut found.

Remarks: viability of the gut questionable, resection considered. Circulation apparently returned but subsequently became impaired, resulting in gangrene of the segment.

* See p. 279.

COMPLICATIONS OF CONVALESCENCE. *Pulmonary Complications.* Pulmonary complications are not uncommon after operations for obstruction. The patient is frequently in a weak and debilitated condition, and pneumonia, usually of the bronchial type, not infrequently arises during the early post-operative period. Occasionally it is caused by the aspiration of vomitus. Pulmonary embolism is one of the rarer complications; it more commonly occurs after strangulations than in other types of obstruction.

Tetanus. Rarely, cases of tetanus have been reported as complicating the convalescence from operations for acute intestinal obstruction and from other operations on the intestinal tract. This is a very unusual complication, which is somewhat surprising considering how frequently the *B. tetani* are found in the intestinal canal under normal conditions.^{10,11}

*Persistence or Recurrence of Symptoms of Obstruction.** Gastrointestinal symptoms should clear up rapidly after a successful operation; if they do not, there may be several explanations. Of course, a certain amount of distention and pain is associated with most laparotomies; the importance of the symptoms depends upon their severity and the underlying cause, which must be determined if possible. Recurrence or persistence of serious distention, vomiting and pain may mean that the true cause of the obstruction was not found at operation or was not adequately dealt with: for example, many adhesions may have been present and a wrong decision made as to which ones were responsible for the obstruction; or some twist of the intestine may have been overlooked. It is also true at times that even though the mechanical obstruction has been relieved, the intestine has been so injured by fatigue and distention that there is no return of peristalsis. In other words, a functional obstruction has been superimposed on a

* Repeated attacks of subacute or acute obstruction may also occur late after operations for obstruction, particularly when many adhesions (see pp. 14, 31-33) are present. These patients present a most difficult and distressing problem, for the adhesions often recur and even increase in extent after separation. Various types of solutions and procedures (see Ref. 3, Chap. III) have been used to prevent this unfortunate occurrence, but often without success. The patients should be tided over the subacute attacks by conservative treatment if possible.

mechanical obstruction. If an enterostomy was not included as part of the first operation in these cases, it may be performed now, although it is not always successful in relieving the symptoms. The recurrence of symptoms may, on the other hand, indicate the onset of peritonitis, uremia or some other complication.

Expectant treatment consists in keeping the stomach empty and, if necessary, maintaining constant drainage by use of a small stomach tube. A rectal tube and hot applications on the abdomen may be used in combatting distention. If hiccoughing occurs, the administration of carbon dioxide may be found helpful in relieving this distressing condition. See also p. 211.

Occasionally the obstruction recurs after operation: the return of vomiting and pain after a period of relief is suggestive of this diagnosis. Visible peristalsis may occasionally be seen. If the symptoms do not subside promptly under appropriate treatment, the patient should be operated upon again.

Intestinal Fistulas. A fistula may arise from some localized damage to the intestine, as a result of faulty anastomosis, or following various types of enterostomy. It may be deliberately planned, as, for example, when two ends of gut are brought out through the abdominal wound after resection of a damaged segment of intestine. The introduction of the valvular type of enterostomy with the use of a catheter has greatly lessened the number of persistent or troublesome fistulas in cases of obstruction. Articles by Cameron¹² and Eliot¹³ furnish interesting data on the general pathology and incidence of intestinal fistulas.

The seriousness of a fistula depends largely upon its location: the higher it is in the intestinal tract, the more serious a menace to life it constitutes. In general, a fistula situated low in the small intestine is very well tolerated, for adequate absorption can take place above. Of course an opening in the colon is usually quite compatible with an active life.

Children tolerate intestinal fistulas poorly, and when it is necessary to establish one in a child it should be closed early.

When a fistula is unusually slow in closing, the possibility of obstruction below that point should be kept in mind; this

general surgical principle applies to the intestinal tube as well as to the ureters or bile ducts.

Local Manifestations and Treatment of Fistulas. An intestinal fistula may produce both local and general manifestations. The local changes produced by the escape of intestinal contents may vary from a slight irritation of the skin, in the case of a low fistula, to extremely rapid and serious digestion of the abdominal wall, in the case of a fistula located in the duodenum or upper jejunum. This action is of course brought about by the digestive juices.

Methods of prevention and treatment are somewhat varied. In the first place, one should avoid if possible the establishment of a high fistula.* If one is inevitable, an early operative closure is sometimes indicated; while at other times simpler methods of dealing with the situation may be tried first.

Various methods have been advocated to bring about partial or temporary closure of the opening in the intestine. A T-shaped rubber tube¹³ may be inserted in the gut through the fistula, serving to conduct the intestinal contents into the lower gut. An ingenious button that can be held against the internal opening has been described by Dowd.¹⁴ When the ends of the gut have been brought outside of the abdominal wound, glass tubes, connected by a rubber tube, may be tied into the ends of the gut and the intestinal contents conducted from the upper to the lower segment of bowel¹⁵ (see Fig. 53). Where none of these methods is practical, it is often desirable to use a constant suction apparatus^{12,16} so that the irritating secretions may be removed before they can attack the tissues. In addition to suction, Potter¹⁷ has found effective the prone position with the patient supported on a Bradford frame. Various ointments have been applied to coat over and protect the tissues from the digestive action. Among the older ones is zinc oxide, which is still useful at times; and more recently, a paste of kaolin and glycerine has been advocated,¹⁸ its efficacy depending on the fact that kaolin is capable of absorbing enzymes in considerable amounts. Potter^{19,20} has advocated applications of beef juice with hydrochloric acid; Warshaw and

* The danger is not great with a valvular enterostomy.

Hoffman²¹ have modified this method, using continuous irrigations with peptone solution and acid. Other protein substances, notably dried milk,²² have been advised for use in neutralizing the action of the enzymes. Cunningham²³ has suggested protecting the skin edges with copper bronzing powder. Baker's²⁴ article on the repair of intestinal fistulas carries many interesting illustrations of various types of operative procedure that may be used in dealing with this condition.

General Effects of Intestinal Fistulas. There are a number of ways in which an intestinal fistula may affect the general health of the patient. In certain cases, severe malnutrition may develop, due to loss through the fistula of the products of digestion before assimilation can take place. Even more important and serious in the case of fistulas located in the duodenum and upper jejunum is the loss of the digestive secretions and the sodium and chloride ions which they contain. The loss of these substances results in rapid and extensive dehydration and changes in the normal acid-base balance of the plasma; and if the changes are not corrected by proper treatment, death will follow within a few days. The situation is closely comparable to that found in simple high obstruction of the intestine, where the loss of these secretions takes place by vomiting instead of through a fistula; the end result is the same in both cases.* This similarity between high intestinal

* Extensive experimental work has been done on different types of high intestinal fistulas. In some of these experiments the total intestinal stream has been diverted to the surface; in others the various digestive secretions have been diverted to the surface one at a time in order to study the effects of their loss. Four important digestive secretions are poured into the duodenum: gastric juice, pancreatic juice, bile, and intestinal juice. The loss of the first two is the more serious, and in untreated animals produces death. The loss of either alone produces characteristic changes in the chemistry of the blood and body fluids.

Some of the earliest work was carried out by Pawlow,²⁵ who observed that dogs with pancreatic fistulas died unless they received appropriate treatment. More recently, studies on the fatal effect of the loss of pancreatic juice have been carried out by Elman and McCaughan,²⁶ Walters and Bollman,²⁷ and Gamble and McIver.^{28,29} The last-named investigators, after collecting and analyzing the various digestive secretions, sought to correlate the results with the changes in the blood and body fluids produced by the loss of the several secretions. Walters et al. showed by an ingenious series of operations that the loss of the pancreatic secretion caused death but the loss of bile or the secretion of

obstruction and high intestinal fistulas has recently been emphasized by Morton and Pearse.³² The treatment consists in the administration of normal salt solution in quantities sufficient to counteract the loss from the fistula. It is essential that this treatment be started early, for it is much easier to maintain the patient in a normal state than to restore him to such a state after extensive dehydration has taken place.

As has already been suggested, very satisfactory results may often be obtained by collecting the secretions from the fistula and re-injecting them into the distal segment of the bowel.

In summary, it may be said that the management of an intestinal fistula includes treatment instituted to protect the tissues from irritation and destruction by the digestive enzymes; and general measures of combatting dehydration and other changes in the body fluids that result from the loss of the digestive secretions (see p. 254).

Complications Resulting from Extensive Intestinal Resection or Exclusions by Short-circuiting Operations. It is not infrequently necessary to remove a considerable portion of the small intestine because of gangrene or other conditions. Or, extensive sections of the intestinal tract may be excluded by short-circuiting operations. Due to the body's power of adaptation, if a proper dietary régime is carried out these abnormal conditions are often borne surprisingly well. In general, this régime

the duodenal mucosa did not. Walters and Bollman³⁰ also studied the effects of a gastric fistula, and found that it resulted in a chain of events similar to pyloric obstruction, where the gastric secretions are lost in the vomitus. The work may be summarized as follows:

A duodenal fistula diverting the combined secretions to the surface is rapidly fatal, the animal showing severe dehydration, low blood chlorides, usually an alkalosis, a high urea nitrogen and a lowering of fixed base. When the gastric secretion alone is lost the same picture is produced, except that the alkalosis is more marked and tetany often occurs. The loss of the pancreatic secretion alone is likewise rapidly fatal: the dehydration is extreme; the lowering of fixed base is greater than when the gastric secretion alone is lost; and instead of an alkalosis an acidosis may occur. The loss of the bile and secretion of the duodenal mucosa is not fatal (in acute experiments) and, as might be predicted from a study of their chemical composition, does not produce marked changes in the composition of the blood plasma.^{27,31}

consists of a diet rich in carbohydrates, poor in fats and containing only a moderate amount of protein.

One of the most extensive and comprehensive studies of this subject was made by Flint,³³ who studied the effects both of resections and of short-circuiting operations. This investigator showed that, in dogs, 50 per cent of the total small intestine might be removed and the animal, when kept on a favorable diet, return to normal condition; where 75 per cent or more had been resected, the animal might survive but was not likely to show a complete recovery. At first the animals showed severe diarrhea, thirst and hunger, with loss of weight; later they were able to establish a balance and return to more normal condition, the compensating processes in the intestines consisting of a hypertrophy and hyperplasia of the remaining portion of the small intestine: there was a marked increase in the transverse diameter of the gut, and the villi and crypts were increased to almost twice their original size.*

The metabolic studies showed at first an increased excretion of fat, carbohydrate and nitrogen; later, carbohydrate was absorbed to a degree considerably above normal. There were indications of extensive putrefaction in the colon. From a study of clinical material, this author concluded that, in general, human cases behaved like the animals he had studied. Palmer,³⁷ after careful metabolic studies on a patient who had undergone resection of the lower half of the small intestine for tuberculous ulceration and stricture,† found that the loss of nitrogen in the stools was four or five times that of the normal individual; while the loss of fat was five to six times the normal amount. There was also a high urinary indican.

The length of small intestine that may be removed in human cases without fatal results shows considerable variation.

* In connection with experimental resections of the intestines, total extirpation of the duodenum should be mentioned. According to an old theory, the duodenal mucosa formed a secretion that was necessary for life or for the normal functioning of the lower intestinal segments. That such was not the case was shown conclusively by Dragstedt, Dragstedt and Chase,³⁴ Grey,³⁵ and Moorhead and Landes,³⁶ who successfully removed the total duodenum without ill effects to the animal. See also p. 365.

† It is interesting that at one time after operation, the patient developed tetany.

Doerfler³⁸ reported resection of all of the small intestine except 12 cm. at the jejunal end and 20 cm. at the terminal ileum. Recently, Jerauld and Washburn³⁹ reported the removal of 579 cm. (19 feet) of small intestine, the patient recovering from this operation and surviving a second operation for acute obstruction.

While even extensive resections are often surprisingly well borne, severe symptoms of malnutrition may arise; so that a guarded prognosis should be given especially where considerable lengths of small intestine have been removed. Certain mechanical complications following exclusion operations have already been discussed (see p. 283).

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CHAPTER XXVII

MORTALITY

It is generally recognized that acute intestinal obstruction is an extremely serious disease; the mortality, even under modern conditions, is high. From a historical standpoint, the figures on mortality given by the early writers (Ashhurst,¹ Curtis,² Fitz,³ and Gibson⁴) are of interest; since, however, they were not based on consecutive cases but on cases selected from the literature, they really give no accurate information. Even in more modern reports it is difficult to arrive at an accurate estimate of the mortality because of the complexity of the picture presented by the disease and the number of factors affecting the mortality. Comparison of the figures from various clinics is somewhat confusing, for the several surveys include different groups of cases: one study, for example, may omit obstructions from carcinoma, another may exclude cases com-

TABLE XVII
MORTALITY FROM ACUTE INTESTINAL OBSTRUCTION AS REPORTED FROM VARIOUS CLINICS

<i>Author</i>	<i>Year of Report</i>	<i>Number of Cases</i>	<i>Mortality (Per Cent)</i>
Scudder ⁶	1907	121	60
McGlannan ⁷	1913	181	37
Deaver and Ross ⁸	1915	276	42
Codman ⁹	1920	41	34
Richardson ¹⁰	1920	118	41
Braun-Wortmann ¹¹	1924	379	39
Souttar ¹²	1925	3064	26
Tuttle ¹³	1925	150	50
Brill ¹⁴	1929	124	36
Miller ¹⁵	1929	343	61
Smithies ¹⁶	1930	56	41
Cornell ¹⁷	1932	235	51
McIver ⁵	1932	156	44
Vick ¹⁸	1932	6892	26
Vidgoff ¹⁹	1932	266	46

Since the different series include somewhat different types of cases (certain of the series, for example, include strangulated external hernias while others do not) the figures are not strictly comparable.

plicated by peritonitis; and such differences greatly affect the statistics.

If we bear in mind that they are not always closely comparable, the statistics from different clinics are nevertheless of importance as indicating a high mortality. Table xvii gives representative figures which have appeared in the literature. Although there is considerable range in the percentage of mortality shown by the different authors, apparently the disease has proved fatal in almost half the cases. Miller,¹⁵ in his recent study of intestinal obstruction in various hospitals of New Orleans, comes to the conclusion that if the figures commonly given are rigidly revised to include only acute, complete obstructions, they show a mortality of from 50 to 65 per cent, whereas they are usually stated as from 30 to 40 per cent.

The number of deaths from intestinal obstruction occurring in the United States during the past ten years is of interest. The data in Table xviii were kindly furnished by the Surgeon General from the publications of the Census Bureau.

TABLE XVIII
DEATHS FROM INTESTINAL OBSTRUCTION AND DEATH RATES PER 100,000 POPULATION
IN THE DEATH REGISTRATION AREA OF THE UNITED STATES

<i>Year</i>	<i>Number of Deaths</i>	<i>Deaths per 100,000 Population</i>
1932	7,367	6.2
1931	7,713	6.5
1930	7,593	6.4
1929	7,776	6.7
1928	7,755	6.8
1927	7,230	6.7
1926	7,596	7.2
1925	7,230	7.0
1924	6,807	6.9
1923	6,668	6.9
1922	6,404	7.0

In 1932, there were 1,304,109 registered deaths from all causes in the registration area; the death rate from all causes being 1090.0 per 100,000 population.

In 1922 the death registration area included 85.4 per cent of the population of the United States; in 1932 the area for which figures are given included 95.9 per cent of the population.

(Data from publications of the Bureau of the Census.)

FACTORS AFFECTING MORTALITY: BASED ON FIGURES FROM
THE MASSACHUSETTS GENERAL HOSPITAL

An analysis of all cases of acute intestinal obstruction at the Massachusetts General Hospital during the ten-year period 1918-1927⁵ has brought out the mortality figures shown in Table XIX, which will be briefly discussed.

TABLE XIX
MORTALITY IN 335 CASES OF ACUTE INTESTINAL OBSTRUCTION FROM ALL CAUSES, TEN-YEAR SERIES, MASSACHUSETTS GENERAL HOSPITAL (MCIVER⁵)

<i>Classification</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality (Per Cent)</i>
Group I..... (All cases of acute mechanical obstruction except those due to neoplasms or to strangulated external hernias)	156	68	44
Group II..... (Obstructions due to neoplasms)	32	10	31
Group III..... (Obstructions due to strangulated external hernias)	147	27	18
Total all causes.....	335	105	31

GROUP I. Group I includes all cases of acute mechanical obstruction except those due to neoplasm or strangulated hernia. Owing to previous studies by Scudder⁶ and by Richardson,¹⁰ the mortality figures in these types of obstruction at the Massachusetts General Hospital are available for the past thirty years, and are shown in Table xx.

In the analysis of these cases of intestinal obstruction, all deaths during the hospitalization, whether or not the direct result of the obstruction, were included. The cases were grouped according to the most important feature of each, and since certain cases have characteristics allying them to more than one type, the final arrangement must be considered as somewhat arbitrary.

A contrast of the different ten-year periods is interesting; while there was a definite improvement between the first and

ACUTE INTESTINAL OBSTRUCTION

TABLE XX
MORTALITY IN DIFFERENT TYPES OF CASES, THIRTY-YEAR SERIES

Type of Case	1898-1907			1908-1917			1918-1927		
	Number of Cases	Number of Deaths	Mortality (Per Cent)	Number of Cases	Number of Deaths	Mortality (Per Cent)	Number of Cases	Number of Deaths	Mortality (Per Cent)
Early postoperative (within 4 weeks after operation).....	18	13	72	29	7	24	37	17	46
Late postoperative (more than 4 weeks after operation).....	19	6	32	28	11	39	45	13	29
Bands and adhesions without previous operation.....	33	18	54	14	7	50	21	9	43
Meckel's diverticulum..	9	7	78	4	2	50	2	0	0
Volvulus.....	9	9	100	16	4	25	13	6	46
Intussusception.....	27	14	52	20	12	60	17	9	53
Mesenteric thrombosis..	1	1	100	5	4	80	9	9	100
Congenital anomaly....	2	2	100				2	2	100
Gallstone or other foreign body.....	1	1	100	1	1	100	7	2	29
Strangulated internal hernia.....	2	2	100	1	1	100	3	1	33
Total.....	121	73	60	118	49	41	156	68	44

Table xx shows the mortality in different types of acute intestinal obstruction (exclusive of those caused by strangulated external hernia or by neoplasms). In making a comparison of the three 10-year series, 1898-1927, Massachusetts General Hospital, it will be noted that there was a definite improvement between the first and second periods; but that the figures for the second and third periods are strikingly similar: while there has been improvement in some types, in other types the figures are less favorable. (Scudder,⁶ Richardson,¹⁰ McIver.⁵)

the second periods the figures for the second and third periods are strikingly similar: there has been improvement in some types, while in other types the figures are less favorable.

In considering the factors that affect the mortality of this group, certain ones stand out predominantly: (1) the length of time elapsing between the onset of the acute obstruction and its relief by operation; (2) interference with the circulation to the bowel, and (3) the age of the patient.

Time Element. The time elapsing before operation is perhaps the most important factor affecting mortality, for if sufficient time has elapsed for marked damage to the bowel to have occurred and for systemic effects to have manifested themselves, surgery is often unable to prevent a fatal termination even though the obstruction be relieved. The relation of the time element to mortality is shown for the 1918-1927 series in Table XXI.

There were 149 cases in which it was possible to estimate approximately the duration of symptoms before operation. About half the cases (73) were operated on within the first forty-eight hours, with a mortality of 26 per cent; the remainder, operated on beyond that period, showed a mortality of 60 per cent.*

TABLE XXI
RELATION OF MORTALITY TO DURATION OF SYMPTOMS BEFORE OPERATION

<i>Duration of Symptoms</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality (Per Cent)</i>
Less than 24 hours.....	36	6	17
24-48 hours.....	37	13	35
Over 48 hours.....	76	46	60

About one-half the cases (73) were operated upon within the first forty-eight hours, with a mortality of 26 per cent. The remaining half, operated on beyond that period, showed a mortality of 60 per cent. This illustrates in striking manner the importance of early diagnosis and operation in cases of acute obstruction. (McIver.⁵)

* In the three Massachusetts General Hospital series, the mortality in the group of obstructions by bands and adhesions where there had been no previous operation was higher than it was in the group of "late postoperative obstructions." The suggestion has

Interference with the Circulation to the Bowel: (a) Mesenteric Circulation. The amount of interference with the mesenteric circulation is a factor of greatest importance in determining the acuteness of the illness and the ultimate outcome; the greater the damage to the bowel the more marked the toxemia. This has been known for a long time, both from clinical observation and from experimental work (Bryant,²⁰ Murphy and Vincent²¹). Analysis of the recent Massachusetts General Hospital series brings out the validity of this point of view. Table XXII shows that 66 cases (or 42 per cent of the total number) had some degree of interference with the mesenteric circulation, although in many cases this was not extreme. The mortality in patients showing interference with the mesenteric circulation was 53 per cent, as contrasted with 37 per cent in patients not having this complication. Table XXIII shows that about half the patients during the thirty-year period had interference with the mesenteric circulation, and that the mortality among them was 62 per cent as contrasted with a mortality of 41 per cent where the mesenteric blood supply was undisturbed. It is probable that these figures would be more striking but for the fact that where there is any element of strangulation present the onset of symptoms is likely to be more fulminating and the pain often so extreme that the patient is brought early to operation; the favorable

been made¹⁰ that the presence of an abdominal scar made the diagnosis of obstruction easier and that for this reason the patients with postoperative obstruction came to operation earlier than those who had not been previously operated upon. In the latest series, however, the time element did not seem to be a factor in explaining the difference in mortality, for the patients without previous operation were, on the average, explored for obstruction earlier than the group with postoperative obstruction. The "late postoperative obstructions" operated on in less than forty-two hours after the onset of obstructive symptoms showed a mortality of 0 per cent; whereas the cases without previous operation, operated on for obstruction within the same period, showed a mortality of 37 per cent. It seems that the higher mortality must be explained on the basis of other characteristics of the two groups rather than on the time they came to operation. It should be stated in this connection that many of the obstructions by bands and adhesions without previous operation occurred in older individuals, and represent end-results of diverse pathological processes occurring at one time or another throughout life. The question of age alone probably does not explain the difference in mortality in these two groups.

time element thus to some extent offsets the more serious pathological condition.

TABLE XXII
RELATION OF MORTALITY TO INTERFERENCE WITH MESENTERIC CIRCULATION

<i>Classification</i>	<i>Number of Cases</i>	<i>Number Showing Interference with Mesenteric Circulation</i>	<i>Mortality in Cases with Interference (Per Cent)</i>	<i>Mortality Cases without Interference (Per Cent)</i>
Postoperative, early.....	37	2 (5%)	100	43
Postoperative, late.....	45	17 (38%)	29	28
Bands and adhesions without previous operation.....	21	8 (38%)	50	38
Meckel's diverticulum.....	2	2 (100%)	0	0
Volvulus.....	13	9 (69%)	55	25
Intussusception.....	17	17 (100%)	53	0
Mesenteric thrombosis.....	9	9 (100%)	100	0
Congenital anomaly.....	2	0	0	100
Gallstones and other foreign bodies.....	7	0	0	29
Strangulated internal hernias..	3	2 (67%)	50	0
Total.....	156	66 (42%)	53	37

It will be noted that in the obstructions occurring early after operation this complication is relatively infrequent; it is also rare in obstructions by neoplasms. (McIver.⁶)

(b) *Capillary Circulation.* It is difficult to estimate statistically the relation of interference with the capillary circulation to mortality. Probably all cases where the obstruction has existed for any appreciable length of time show some interference with the capillary circulation, from distention, and resulting changes in the bowel wall.

Age of the Patient. It will be noted from Table xxiv that the age of the patient has a definite relation to the death rate, the

mortality being very high in those under one year, and also, in this series, in patients over fifty years of age.

TABLE XXIII
INTERFERENCE WITH THE MESENTERIC CIRCULATION, THIRTY-YEAR PERIOD (ALL CASES OF ACUTE OBSTRUCTION EXCEPT THOSE DUE TO EXTERNAL STRANGULATED HERNIA AND NEOPLASM)

<i>Period</i>	<i>Number of Cases</i>	<i>Number Showing Interference with Mesenteric Circulation</i>	<i>Mortality in Cases with Interference (Per Cent)</i>	<i>Mortality in Cases without Interference (Per Cent)</i>
1898-1907	121	59 (49%)	78	57
1908-1917	118	54 (46%)	55	30
1918-1927	156	66 (42%)	53	37
Total.....	395	179 (45%)	62	41

This table again illustrates the higher mortality shown by cases of obstruction complicated by interference with the mesenteric circulation. (Scudder,⁶ Richardson,¹⁰ McIver.⁵)

TABLE XXIV
AGE IN RELATION TO MORTALITY

<i>Age of Patient</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality (Per Cent)</i>
Under 1 year.....	10	6	60
1-10 years.....	11	5	45
11-20.....	28	8	29
21-30.....	27	8	30
31-40.....	24	6	25
41-50.....	20	11	55
51-60.....	13	8	62
61-70.....	13	8	62
71-80.....	9	7	78
81-90.....	1	1	100
Total.....	156	68	44

It will be noted that the mortality is especially high in the first decade and after the fifth decade. These figures do not include obstructions from strangulated external hernias or from neoplasms. (McIver.⁵)

Influence on Mortality of the Modern Treatment of Dehydration. The figures from the Massachusetts General Hospital were of especial interest in relation to the effect on mortality figures of the modern treatment of dehydration. During the

last four years of the 1918-1927 period, several physicians in the clinic were especially interested in the value of normal salt solution in combatting dehydration, and every effort was made to see that all patients with intestinal obstruction received adequate treatment with salt solution. It was the general opinion that these measures were distinctly worth while and in a number of instances were life-saving procedures. When, however, the recent ten-year series as a whole was contrasted with the 1908-1917 series, no lowering of the total mortality was found; also, when the mortality of the last four years of the present study was contrasted with that of the first six years, no reduction was found. This can probably be explained by the following facts. First, as pointed out by McIver and Gamble,²² simple high obstruction in humans (the only type comparable to the experimental obstructions in animals in which salt solution is so efficacious)* is a relatively rare occurrence; this is borne out by the figures in Table xxv, which show that only 2 per cent of obstructions were situated in the upper jejunum. Second, where damage to the bowel exists, due to interference with the circulation, the factor of dehydration is relatively unimportant, for the disease runs such a rapid course that the extreme degree of dehydration and loss of chloride and sodium ions does not have time to develop. In this group, 42 per cent (if strangulated hernias and neoplasms are included, this figure rises to 63 per cent) of the cases showed some interference with the mesenteric circulation, and there were a number in which damage to the capillary circulation by distention undoubtedly played an important rôle. Thus in the great majority of cases with acute intestinal obstruction there are factors which outweigh dehydration in importance. Some degree of dehydration, however, is usually present in acute obstruction and it is often extreme. Proper attention to this factor by the administration

* As shown by Hartwell and Hogue, and numerous other experimenters, when dogs with simple high obstruction of the jejunum are treated with sufficient quantities of salt solution to replace the volume of fluid lost in the vomitus, instead of dying within a few days they may live as long as three weeks although the obstruction is unrelieved. (See p. 387.)

of adequate volumes of normal salt solution before and after operation will improve the general condition of most patients and in a certain number will be a decisive factor in the outcome.

TABLE XXV
LEVEL OF OBSTRUCTION

<i>Part of Bowel Obstructed</i>	<i>Classification of Cases</i>			
	<i>Group I*</i>	<i>Group II</i>	<i>Group III</i>	<i>Total Cases</i>
Jejunum.....	7	1	0	8 (2%)
Ileum.....	59	2	0	61 (18%)
Small intestine..... (not further specified)	53	0	127	180 (54%)
Ileocolic intussusception.....	14	0	0	14 (4%)
Large and small intestine.....	0	0	13	13 (4%)
Large intestine.....	16	29	7	52 (16%)
Not determined.....	7	0	0	7 (2%)
Totals.....	156	32	147	335

It will be noted that in 74 per cent of the cases the point of obstruction was located in the small intestine; these obstructions were for the most part in the lower small intestine; there were only 8 cases in which the obstruction was located in the jejunum. Both the large and the small intestine were involved in 8 per cent of the cases; the large intestine alone in 16 per cent. (McIver.⁵)

* Group I: All cases except those due to neoplasms or strangulated external hernias.

Group II: Obstructions due to neoplasms.

Group III: Obstructions due to strangulated external hernias.

GROUP II. Group II is composed of obstructions from neoplasms. The mortality was found to be 31 per cent. The classification of these cases on the basis of etiology and location of the obstruction is shown in Table xxvi.

Obstructions from neoplasms in general do not cause interference with the mesenteric circulation and occur for the most part in the large intestine. As would be expected, therefore, the mortality is lower than in Group I, where interference with the mesenteric circulation is common and where it is usually the small intestine that is involved. In the present series, there were 3 cases in which neoplasm directly involved the

mesenteric vessels, resulting in occlusion or thrombosis with subsequent infarction of the intestine. In the late cases of obstruction of the colon, interference with the capillary circulation of the cecum, due to pressure, of sufficient extent to cause areas of necrosis or perforation, is not infrequent.

TABLE XXVI
OBSTRUCTIONS FROM NEOPLASMS

<i>Etiology and Location</i>	<i>Number of Cases</i>	<i>Number of Deaths</i>	<i>Mortality (Per Cent)</i>
Obstruction from primary tumor.....	20	4	
Large intestine..... 17			
Small intestine..... 3			
Obstruction from metastatic tumor.....	6	1	
Large intestine..... 5			
Small intestine..... 1			
Obstruction presumably due to tumor*.....	6	5	
Large intestine..... 6			
Total.....	32	10	31

* These patients were too ill at time of entrance to the hospital to warrant any exploration. Cecostomy or colostomy only was carried out, so that the diagnosis of neoplasm was not definitely established. However, the location of the obstruction in the colon, the age of the patient and other factors led to the diagnosis of obstruction by neoplasm. (McIver.⁵)

Age of the Patient. The ages of the patients with obstruction from neoplasm, in this series ranged from twenty-eight to ninety years, the average being fifty-five years. Almost 60 per cent of the patients were over sixty years of age; and post-operative complications due to this factor often entered in.

GROUP III. The mortality of Group III, comprising strangulated external hernias, as shown in Table XXVII, was 18 per cent.

Type of Hernia. By far the largest percentage of deaths was among the cases of umbilical and ventral hernia; this was also Frankau's²³ finding in his analysis of 1487 cases. These hernias are often found in fat, middle-aged individuals who are poor operative risks. There were proportionally more deaths among patients with femoral hernias than among those with inguinal hernias.

TABLE XXVII
MORTALITY IN DIFFERENT TYPES OF HERNIA

Type of Case	Number of Cases	Number of Deaths	Mortality (Per Cent)
Inguinal.....	84 (57%)	10	12
Femoral.....	34 (23%)	6	18
Umbilical.....	18 (12%)	8	44
Ventral.....	9 (6%)	3	33
Epigastric.....	2 (2%)	0	0
Total.....	147	27	18

The largest percentage of deaths in this series was among the umbilical and ventral hernias. There were proportionately more deaths among patients with femoral hernias than among those with inguinal hernias. (McIver.⁵)

Duration of Symptoms before Operation. As in all types of intestinal obstruction, delay in operation increases the mortality rate. In this series of patients with obstruction from strangulated external hernia, one-half the number of deaths occurred in patients where symptoms of strangulation had existed for over twenty-four hours, as follows:

Duration of Symptoms	Cases	Deaths	Mortality
Less than twenty-four hours	103	13	13 per cent
More than twenty-four hours	44	13	30 per cent

Interference with the Mesenteric Circulation. All cases of strangulated hernia by definition show some interference with the mesenteric circulation. In 19 cases of the present series, as shown in Table XI, p. 125, this was so severe and so persistent as to produce necrosis of the bowel. Eleven deaths, almost half the total number, occurred among these 19 cases. When the damaged gut lies within the sac of an external hernia there is, perhaps, less rapid absorption of toxins than when it lies free within the abdominal cavity, as in other forms of intestinal obstruction; but in spite of this fact, these cases showed a mortality of over 50 per cent. The existence of gangrene of the bowel of course indicates that the patient came late to operation; 11 of these patients had had symptoms for a number of days before they entered the hospital.

The portion of the bowel obstructed, the duration of the symptoms before operation and the type of operation, are also shown in Table XI.

Anesthetic. The operation can often be satisfactorily performed under local anesthesia. Any death following an operation for strangulated hernia, where a general anesthetic has been used, should be carefully investigated to see if the outcome could have been avoided by the use of some form of local anesthetic.

FACTORS AFFECTING MORTALITY IN ALL GROUPS. *Complications.* Shock, during operation or coming on immediately afterward, is a very common complication in serious cases. Various pulmonary complications are not infrequently encountered: bronchial pneumonia, either from the general weakened condition of the patient or from aspiration of intestinal contents, is perhaps the most common; pulmonary emboli, especially in strangulated external hernias, have been recorded. (See also p. 310.)

Skill of the Surgeon and His Judgment in Selecting the Appropriate Operative Procedure. The figures from all the large public hospitals are made up from the results of a great number of different surgeons of widely varying skill and experience. This undoubtedly affects the mortality; but the factors involved are entirely too intangible to permit of any analysis.

CONCLUSION

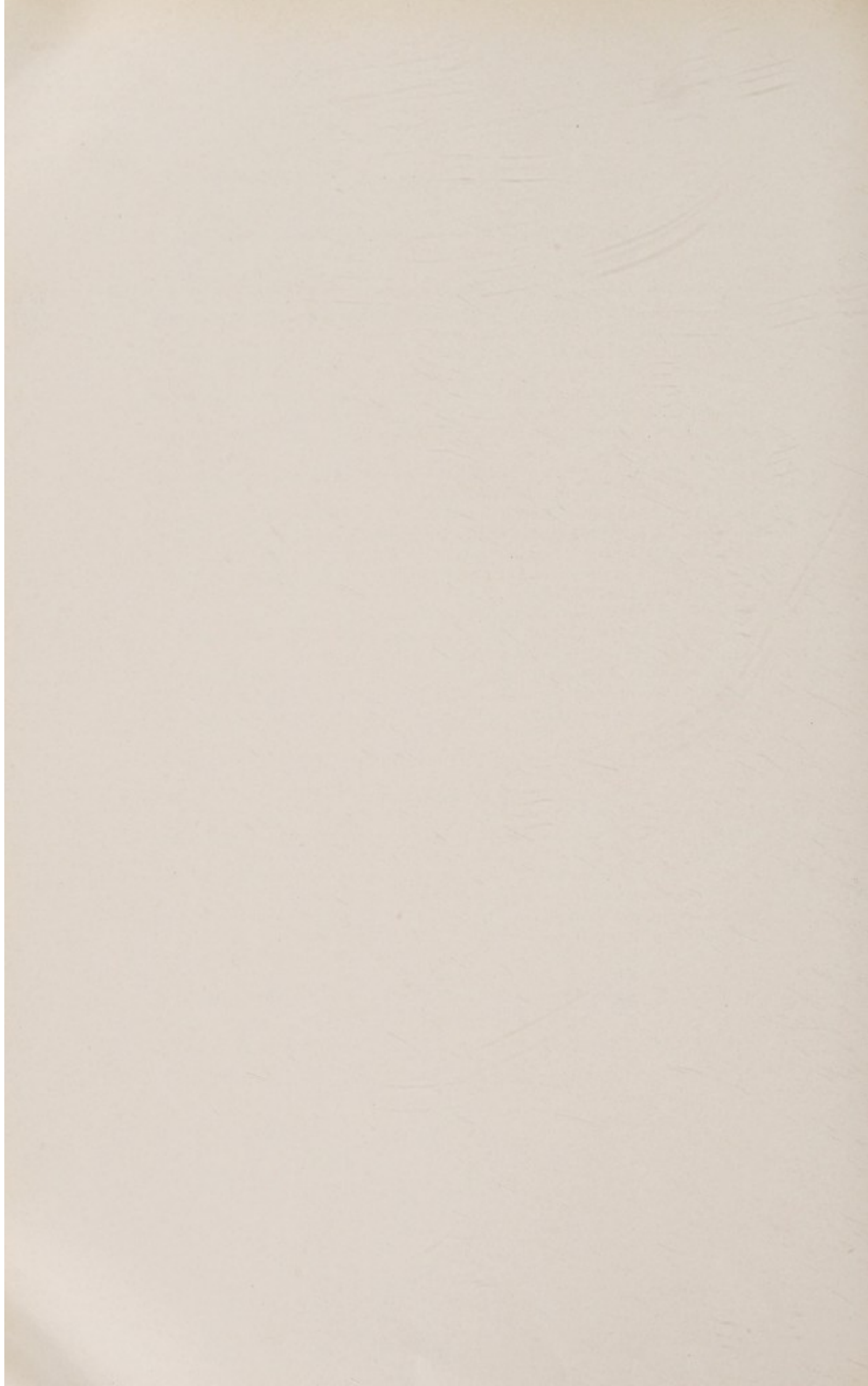
The figures given in this chapter show the seriousness of acute intestinal obstruction. Earlier diagnosis and operation, as stressed by all writers on the subject, would unquestionably contribute to a lowering of the death rate; but the pathological condition presented by many of these cases is so serious that in many types of obstruction the mortality is likely to continue high.

It would be desirable if in the preparation of statistics on intestinal obstruction, careful divisions of different types of cases were observed, so that it might be seen where improve-

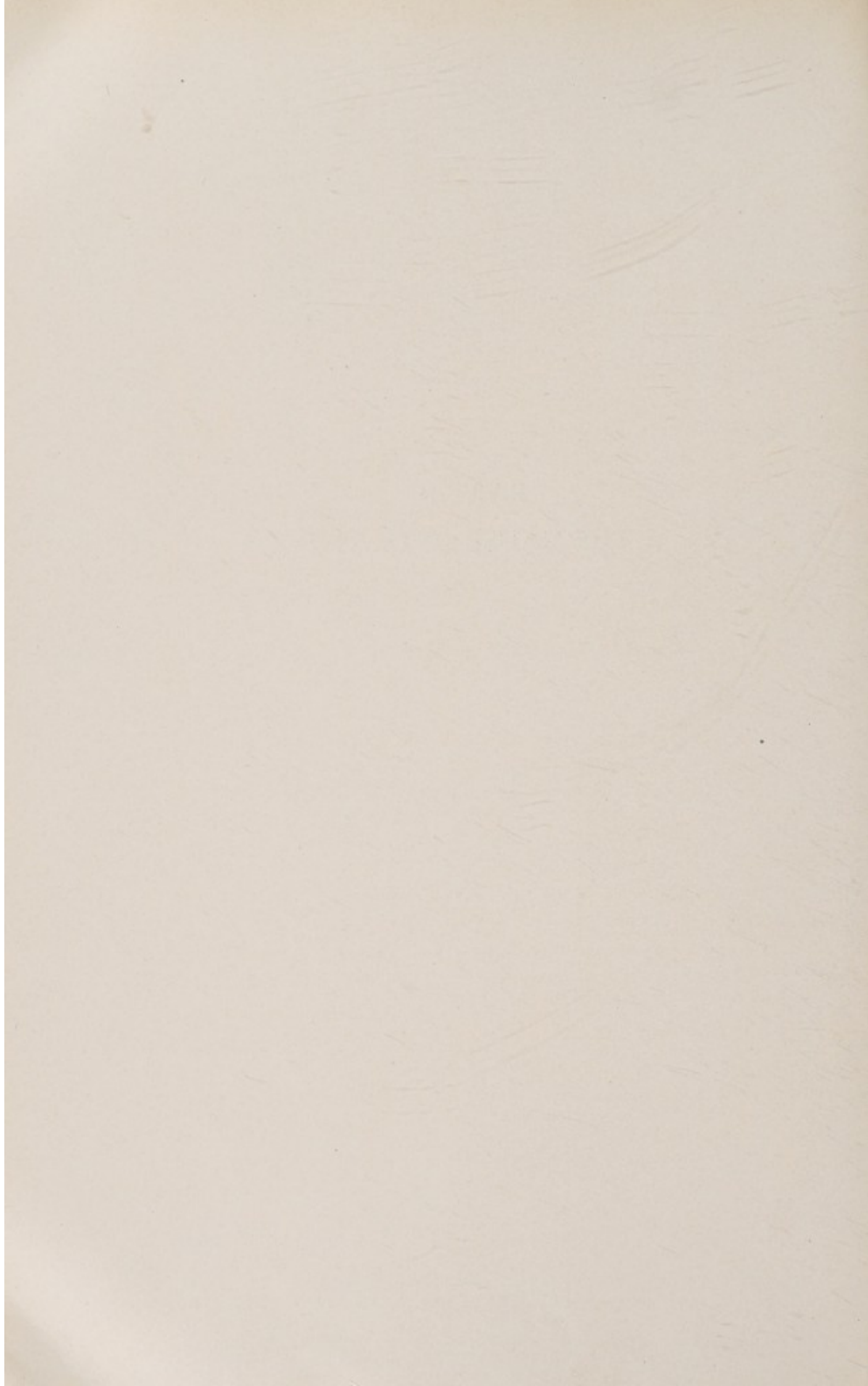
ment was most needed and possible. The figures so compiled would mean more than they do when they simply give the gross mortality of such a miscellaneous group as that embraced under the term "acute intestinal obstruction" and would show us in which types of cases progress is being made and in which types extra efforts are called for.

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PART III
THE CAUSE OF DEATH



CHAPTER XXVIII

EXPERIMENTAL METHODS USED IN THE STUDY OF INTESTINAL OBSTRUCTION

The cause of death in acute intestinal obstruction is a problem that has engaged the attention of clinicians and investigators for over a hundred years. The disease is often dramatic in the suddenness of its onset and the rapidity of its course, the patient quickly becoming extremely ill and having the general appearance and prostration associated with a profound toxemia. At autopsy the findings may not be sufficient to account for the clinical picture and death* of the patient.

The theories that have been advanced to explain the situation are numerous: the literature dealing with the experimental evidence is confused in the extreme and is filled with contradictions. Although it has been stressed by numerous investigators,¹⁻⁴ that different types of obstructions produce quite different clinical and pathological pictures, this point is still often ignored in drawing general conclusions as to the cause of death. There has also been a failure to appreciate the fact that while certain types of experimental obstruction produced in lower animals have close clinical counterparts in human cases

* While it is true that such situations occur, the postmortem examination will usually show some obvious morbid anatomy which is the immediate cause of death, as for example, peritonitis. The peritonitis usually comes about as a result of gross perforation of the gut or increased permeability to bacteria resulting from interference with the circulation of the bowel wall. Peritonitis may also arise as a result of operative procedures, either through leakage around intestinal sutures or from soiling of the peritoneal cavity with bowel contents at operation.

In a recent review of 123 cases autopsied following death from acute obstruction, peritonitis was listed as a principal or contributing cause of death in 66 instances. It is no wonder that in the early 19th century when the condition was seen only at late operations or at autopsy, after the disease had run its full course, peritonitis (or "inflammation of the bowel") should have been considered to be always the cause of death.

The real problem is to explain the severe illness of these patients in the earlier stages of the disease, rather than to determine the immediate cause of death, which in human patients is so often some obvious complicating factor.

other types have not. In considering the literature it should be realized that experimental methods have not been sufficiently developed to afford a direct answer to many of the complex features of the problem. This has resulted in the endless repetition of certain types of experiments, with inconclusive or contradictory results. While indirect methods have in some instances furnished valuable information as to the cause of death, they have at times resulted in further complicating rather than clarifying the situation.

The first step is to understand clearly the fundamental types of experimental obstruction that have been employed, the results that they have produced, and their relation to clinical conditions encountered in humans.

TYPES OF EXPERIMENTS

The principal types of obstruction that have been used in experimental studies on acute intestinal obstruction are presented in diagrammatic form in Figures 60-62. There have been, of course, many minor modifications of technique.

Simple blockage of various portions of the intestinal lumen is shown in Figure 60. Hereafter, this type of obstruction will be referred to as "simple obstruction" with the qualification "small intestine—high" (Fig. 60 A), "small intestine—low" (Fig. 60 B), and "of the colon" (Fig. 60 C).

SIMPLE HIGH OBSTRUCTION. Experimental high obstructions (Fig. 60 A) are situated either in the duodenum or in the upper jejunum, generally within 30 to 35 cm. of the pylorus and not infrequently in the duodenum just below the entrance of the pancreatic or biliary ducts. This type of obstruction results in a very severe illness, characterized by profuse vomiting and severe dehydration. The animals soon show evidence of asthenia, apathy, muscular weakness, sometimes muscular twitchings, rapid heart rate and alterations in respiration; they not infrequently sink into coma before death intervenes.

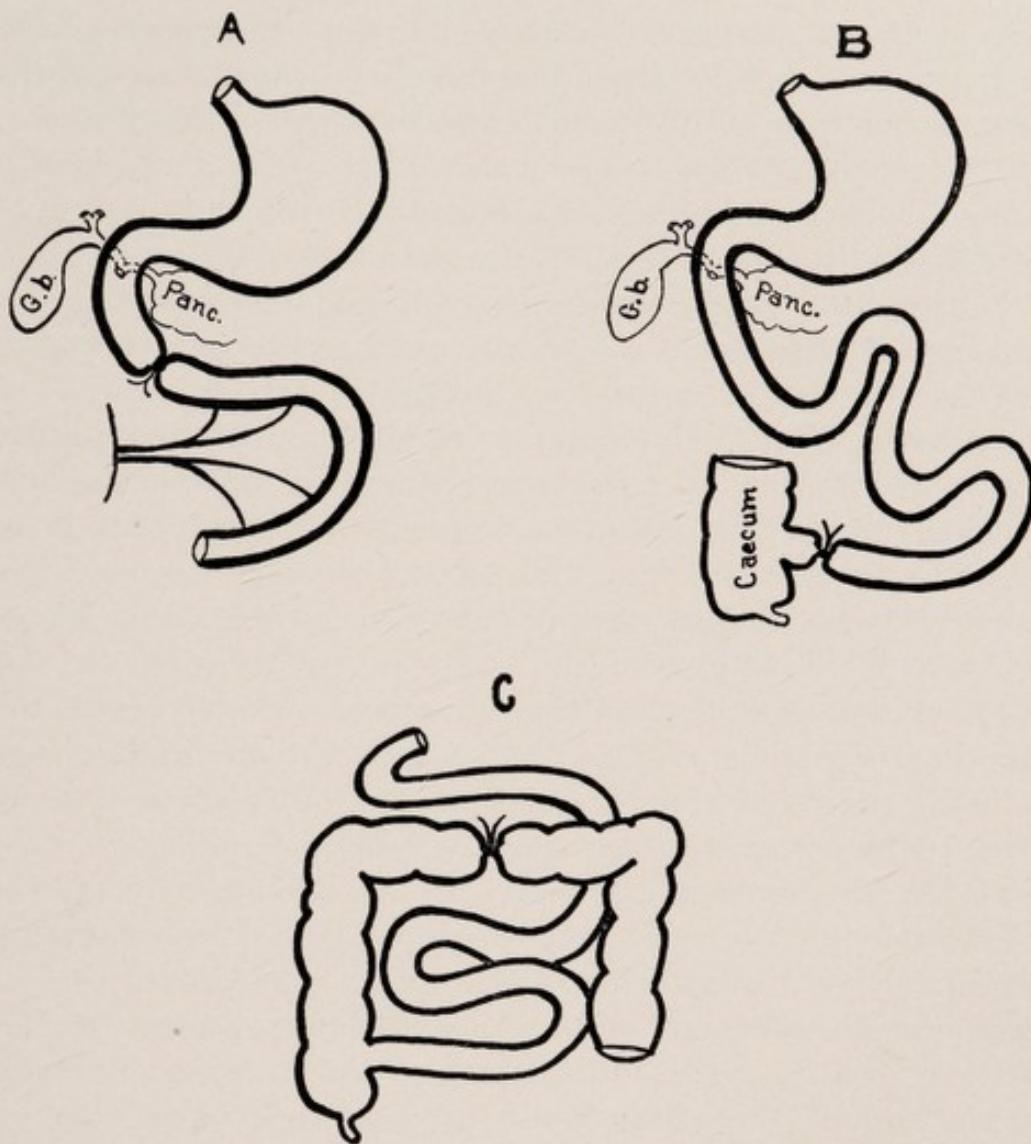


FIG. 60. Experimental types of obstruction: simple obstruction.

A. Simple blockage high in intestinal tract is a type of obstruction resulting in severe illness characterised by profuse vomiting and by severe dehydration due to loss of water and sodium and chloride ions in vomitus.

B. Simple blockage in lower ileum. Animals with this form of obstruction have a long survival period; vomiting is not likely to occur so soon or to be so profuse. This type of obstruction is less frequently used.

C. Simple obstruction of colon, a type of obstruction frequently tolerated by animals for long periods of time with few symptoms. (McIver, White and Lawson⁴)

The survival period is usually between twenty-four and seventy-two hours,⁵⁻⁷ depending largely on the distance of the obstruction from the pylorus; Draper⁷ considered that if it were in any portion of the duodenum the survival period was usually under seventy-two hours and that it occurred most rapidly if obstruction was in the second portion of the duodenum.⁸

It has been a rather usual observation in experimental work on simple obstruction that the less operative damage caused to the intestine and mucosa in producing the obstruction, the less fulminating are the symptoms of obstruction that follow. Hartwell, Hoguet and Beekman² devised a special rubber-covered clamp that could be applied so as to produce an obstruction with a minimum amount of damage to the intestinal wall. Foster and Hausler⁹ advocate section of the bowel followed by inversion of the ends. In commenting on this method they say: "Section of the intestine and inversion of the proximal and distal stumps gives a complete obstruction that is easily produced and very permanent. This technique, however, causes considerable tissue destruction and is followed by a severe constitutional reaction. The postoperative record shows that for the first five or six days, or until the inverted stumps have healed, there is a quite pronounced rise in temperature, pulse and respiration. Unless subcutaneous injections of fluid are given during this reparative period, many of the animals die. However, if the dogs survive, we have an ideal obstruction exactly comparable to that seen in human cases. The obstruction is complete, and little or no devitalized tissue is present." This statement seems to the author just the reverse of the observed clinical facts; for practically all obstructions in humans (even the simplest type) cause some local damage to the bowel at the point of obstruction, and the longer the obstruction persists, the more marked is the local inflammatory reaction. It would seem that the greater the refinements of technique in producing experimental obstructions, the less closely do they correspond with clinical cases.*

* Foster and Hausler⁹ found that when they produced high obstruction by careful inversion of the ends of the intestine, and administered salt solution during the four or

Simple high obstruction has been used extensively in experimental work and has been employed by most of the investigators who have advocated the view that death in high intestinal obstruction is due to dehydration. It must be pointed out that while this experimental form is comparable to high obstructions found in clinical practice, these are relatively infrequent (see p. 332). Cases in which a kink of the jejunum occurs in the region of a recently formed gastroenterostomy, occasional instances where a band obstructs the upper jejunum or where a gallstone lodges in this portion of the intestinal tract and brings about a similar condition, are comparable, but are not often encountered; and it is well to remember this, in view of the large amount of space devoted in the literature to this type of experimental obstruction.

SIMPLE OBSTRUCTION LOW IN THE SMALL INTESTINE. Figure 60 B shows the type of experimental obstruction which is situated in the lower portion of the ileum.

Low obstructions have not been used so extensively in experimental work, largely because the theory of toxic absorption in the more fulminating high obstructions has monopolized the attention of investigators. Also the results of experiments in low obstruction are not so consistent as in other types. The animals, commonly dogs, usually die within from two to twelve days;^{2,5,8,10,11} but sometimes they tolerate such obstructions remarkably well for surprisingly long periods. The survival periods in experiments by Hartwell et al. were five and a half days and ten days.* There is also considerable variation in the symptoms shown by the animals with this type of obstruction: the symptoms may come on promptly with profuse vomiting^{2,12}; on the other hand the animals may remain well for as long as nine days² and die of asthenia without any marked symptoms.¹

five days required for "the inverted raw ends of gut to heal," the treatment with salt solution could then be discontinued. The symptoms of obstruction (vomiting) ceased, the animal showed no symptoms except those which might be attributed to starvation; the survival period was over twenty days. See also Hartwell et al.²

* Sacrificed while still in fair condition.

Thus, low experimental obstruction, although it is anatomically comparable to a large group of cases encountered clinically, namely simple obstructions of the lower ileum without strangulation, does not produce in animals a disease corresponding as closely to human cases as the disease produced by experimental high obstruction. Complete obstruction of the lower ileum in human patients, even though there be no gross interference with the mesenteric circulation, usually brings on severe symptoms: vomiting is likely to be marked and frequent and, if the obstruction is unrelieved, the survival period is not likely to exceed a few days. It is true, however, that even in humans simple low obstructions in the ileum are not so rapidly fatal as obstructions high in the small intestine.

SIMPLE OBSTRUCTION OF THE COLON. Figure 60 c shows a simple obstruction of the colon. In general, the remarks made in regard to low obstruction of the ileum apply in even more marked degree to simple obstruction of the colon. Animals not infrequently tolerate this type of obstruction for long periods of time with few symptoms; the survival period is given^{8,10} as from seven to fourteen days; if the obstruction is at the rectum, the survival period is about twenty-eight days.^{6,8} In humans, also, simple obstructions of the colon are in general tolerated much better than obstructions in the small intestine; the clinical picture here, however, is more severe than with comparable obstructions in experimental animals, and death may be expected at an earlier date. (See also p. 27.)

CLOSED LOOP EXPERIMENTS. *With Ligature of the Mesenteric Veins (Strangulations).* Figure 61 shows obstructions complicated by interference with the venous mesenteric blood supply. These obstructions are produced by the isolation of a loop of bowel with ligatures, and occlusion of the mesenteric veins* with ties. Animals operated upon in this manner

* Murphy and Vincent¹ also performed certain experiments in which they ligated both arteries and veins. They termed this "total anaemia." It rarely if ever has a clinical counterpart.

Some of the earliest experiments on strangulation were carried out by Kader.¹³

rapidly become ill and rarely survive more than twenty-four¹ to thirty¹⁴ hours; they may die in a few hours, depending upon whether a long loop or a short loop has been used.^{3,14,15}

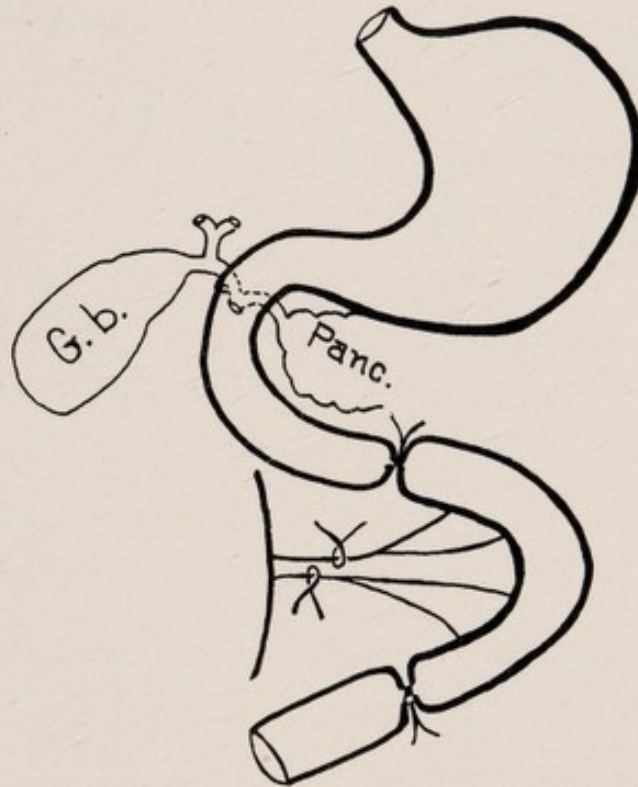


FIG. 61. Experimental types of obstruction: closed loop, with ligation of the mesenteric veins. This form of obstruction results in most serious and fulminating type of illness. Its clinical counterpart is found in cases of internal strangulation, volvulus and other types of obstruction complicated by interference with mesenteric circulation. This disease runs so rapid a course and is of such short duration that dehydration and lowering of blood chlorides do not occur to any appreciable extent. (McIver, White and Lawson ⁴)

The pathological picture shown by the bowel is similar to that already described in the pathology of strangulation, page 143.

This type of obstruction corresponds closely both in pathology and in symptomatology with a type of obstruction frequently encountered clinically, namely, cases where not only a loop of bowel has become obstructed but the attached mesentery is so involved that the venous circulation is cut off.

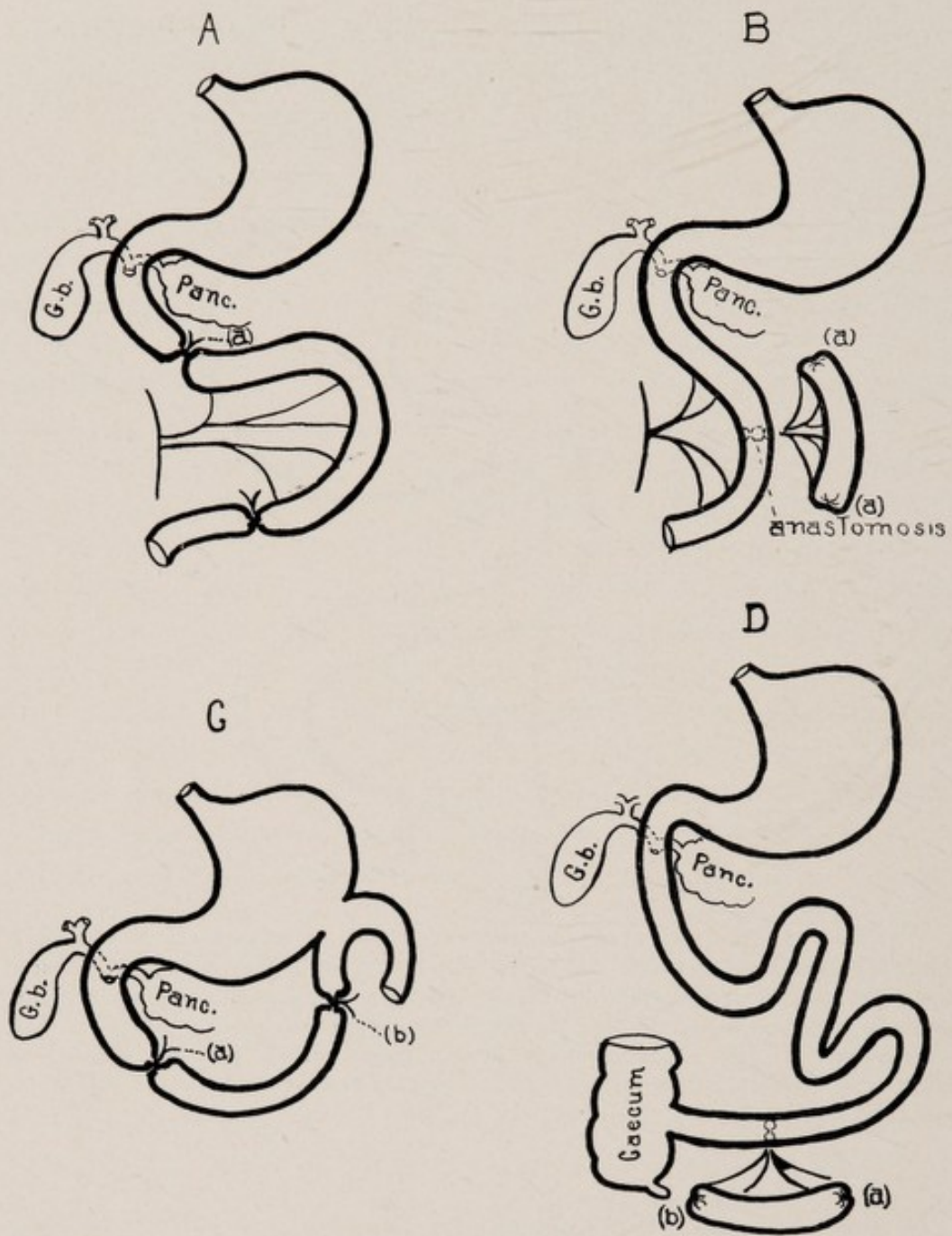


FIG. 62. Experimental types of obstruction: closed loop without ligation of the mesenteric veins.

- A. Isolated loop high in intestinal tract.
- B. Similar loop with continuity of intestine re-established by means of an end-to-end anastomosis.
- C. Similar loop with continuity re-established by means of a posterior gastro-enterostomy (Whipple's method).

[For remainder of legend see p. 349.]

The seriousness of this type of obstruction as contrasted with simple obstructions has long been recognized by clinicians;^{16,17} it includes strangulations with constriction of the venous supply produced by bands, by internal hernias and by volvuli; intussusceptions often come into this group, although here the amount of interference with the mesenteric circulation varies. (See also p. 23.)

Closed Loop Experiments without Ligature of the Mesenteric Veins. In Figure 62 A is shown a type of obstruction that has been employed very frequently in experimental work: the isolation of a segment of the intestine by ligatures, the isolated loops usually being established in the upper portion of the intestinal tract. Frequently the continuity of the intestinal tract is restored, after isolation of the loop with its attached mesentery intact, either by an end-to-end anastomosis (Fig. 62 B), or by a posterior gastroenterostomy (Fig. 62 c). This latter procedure was largely used in the work of Whipple and his co-workers.*¹⁸

* In regard to the method of re-establishing the continuity of the intestinal tract by a gastroenterostomy as advocated by Whipple (Fig. 62 c), Sweet and his co-workers¹⁹ have made the very pertinent criticism that since the gastroenterostomy may often fail to function in the presence of a ligature around the duodenum, the continuity is really not re-established. Whipple explained the fact that when he removed the lower ligature (Fig. 62 c, b) the animals still died although the loop was draining into the jejunum, by the assumption that a toxic secretion was formed in the mucosa of the duodenum below ligature (a), because this was not bathed by the normal secretions from above. As Sweet points out, however, these animals probably died because the gastroenterostomy did not function and ligature (a) constituted an upper intestinal obstruction. To confirm this he showed that if an isolated loop were formed as in Figure 62 B, with the additional procedure of anastomosis of one end of the loop into the jejunum, the animals lived indefinitely.

D. Isolated loop constructed low in intestinal tract, with re-establishment of continuity by an end-to-end anastomosis.

Type of obstruction represented by A, B and c produces a severe illness, survival period being usually twenty-four to forty-eight hours. Owing to profuse secretion that takes place in upper intestine, fluid accumulates rapidly, with a resulting distention and interference with circulation and nutrition of bowel wall. Perforation and peritonitis are frequent complications. Isolated loops in lower ileum (D) sometimes perforate from distention and lead to fatal results, but at other times are tolerated for a long period without symptoms. Difference in course of disease in obstructions of types A, B and c and those of type D is probably best explained on basis of more profuse secretion that takes place in upper intestine.

The survival period of animals having isolated loops in the upper portion of the intestinal tract is usually relatively short, the average length of life (according to Whipple and co-workers¹⁸) being 24 to 48 hours. The animals may vomit considerable amounts. Owing to the profuse secretion that takes place in the duodenum and upper jejunum, fluid accumulates rapidly in the isolated loops, with the result that there is a marked rise of pressure in the loop and consequent interference with the circulation and nutrition of the bowel wall. Perforation and resulting peritonitis are frequent complications.²⁰ When disintegration of the wall does not progress to this point, there are likely to be hemorrhagic areas and areas of necrosis in the mucosa.^{2,21} Burget et al.²² were able to keep animals with closed intestinal loops (ileum and jejunum) alive almost indefinitely by aspirating the loop and thus preventing overdistention. (See also Herrin and Meek.²³)

However much such experiments may have contributed to our knowledge of the general pathology following obstruction of isolated loops, it seems questionable whether high isolated loops without interference with the mesenteric circulation have any close counterpart in clinical intestinal obstruction.* Whipple and his co-workers⁸ considered that such loops might be comparable to cases of volvulus where there is no interference with the mesenteric circulation. This, however, is a condition rarely found at any level in the intestinal tract, and, so far as I am aware, is unknown in the duodenum or upper jejunum. Dragstedt²⁰ took the opposite view and suggested that the closed loops might find a clinical parallel in types of obstruction where there is interference with the mesenteric circulation; but he pointed out that it is problematical how closely this

* Wilkie^{24,25} has pointed out that this type of closed loop obstruction may have a clinical counterpart in certain types of acute appendicitis: the lumen of the appendix may be blocked in its proximal end and gangrene from distention result.

It is also true that in simple low obstruction of the small intestine, kinking of distended coils may prevent free drainage back into the stomach and thus a series of semi-isolated loops may be formed resembling to some extent the experimental closed intestinal loops.

comparison could be drawn. With his hesitation I quite agree; indeed, this experimental type of obstruction seems to me to have no close clinical counterpart, and it has perhaps diverted attention from types of obstruction that are more nearly comparable with clinical cases. I do believe, however, that this method of study has furnished valuable information as to the changes that occur in the bowel wall when it is subjected to increased intrainestinal pressure without vent for the distending liquid either by regurgitation back into the stomach or by passing downward in the intestinal tract. (See also page 367.)

Figure 62D shows the same type of isolated loop as is shown in Figure 62B except that it is taken from the lower ileum.

Loops isolated in the lower ileum are tolerated better than comparable loops in the duodenum or jejunum. Halsted²⁶ in 1887 reported an experiment where the animal lived for over a month after the formation of an isolated loop in the ileum.* von Baracz²⁷ also reported somewhat similar findings, which were confirmed by Whipple and co-workers.¹⁸ On the basis of these experiments an impression is prevalent in the literature that isolated loops in the lower ileum invariably produce few if any symptoms in experimental animals. Actually, this is by no means the case, as was shown by von Baracz's²⁷ early work, and also by experiments carried out by McClure¹⁰ in which all the animals died within a few days,† not infrequently with perforation of the loop; a similar report has been made by Dragstedt et al.²⁰ The general statement may be made, however, that isolated loops in the upper small intestine almost always lead rapidly to fatal results; whereas in the lower intestine the loops are sometimes tolerated for long periods with very few symptoms. (See also Hartwell and Hogue²⁸ and Murphy and Brooks.²⁹) This difference in the course of the disease in the two types is probably best explained by the fact that in the upper intestine the secretion is more

* This finding occurred during studies on various types of intestinal suture.

† The report is not definite as to the exact level of the small intestinal loops.

profuse than in the lower, and consequently damage to the circulation from distention occurs more promptly.^{20,29-31}

Clinically, isolated loops in the lower intestinal tract, without interference with the mesenteric circulation, may be encountered at times; but they are not frequent. (See pp. 283 and 318.)

TABLE XXVIII

<i>Types of Experimental Obstruction</i>	<i>Clinical Observations</i>	<i>Experimental Observations</i>	<i>Clinical and Experimental Correlations</i>
Simple high obstruction of the small intestine (Fig. 60A)	Relatively infrequent Rapidly fatal	Most commonly used type Rapidly fatal	Comparable
Simple obstruction, low in the small intestine (Fig. 60B)	Frequently encountered Not so rapidly fatal as high obstruction	Relatively seldom employed Results variable: survival period 2 to 12 days; may have profuse vomiting or may show no symptoms for considerable periods of time	Not closely comparable
Simple obstruction of the colon (Fig. 60C)	Frequently encountered Much less fulminating course than obstruction of the small intestine	Infrequently employed Dogs may live a long time and show few symptoms	Not closely comparable
"Closed loop" with ligation of mesenteric veins (strangulation) (Fig. 61)	Very common Rapidly fatal	Frequently employed Rapidly fatal	Closely comparable
"Closed loop," high, without ligation of mesenteric veins (Fig. 62A, B, C)	Not encountered clinically	Frequently employed Rapidly fatal	Not comparable
"Closed loop," low, without ligation of mesenteric veins (Fig. 62D)	Encountered rarely, as result of operation	Infrequently used Animal often survives without symptoms	Rarely comparable

SUMMARY

In Table xxviii, an attempt is made to summarize and compare the clinical and experimental findings in the major types of obstruction. It seems to be a rather universal observation in experimental work that the less damage caused to the intestine and mucosa the less fulminating are the symptoms of obstruction that follow.^{2,32} In human cases there is usually some gross damage to the mucosa and bowel wall at the point of obstruction.

The situation is complex and such a brief review can only hope to set forth approximately the true situation; it is hoped, however, that it may serve as a working basis for further study of the cause of death.

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CHAPTER XXIX

THEORIES AS TO CAUSE OF DEATH

Many theories have been advanced to explain the clinical symptoms and death that follow intestinal obstruction.

Among the older ideas, the belief that a vasomotor reflex^{1,2} was responsible for the symptom complex of obstruction received considerable attention in the latter part of the nineteenth century. The advocates of this theory were impressed by the intense pain and other signs of nervous irritation often shown by the patient, and they attributed these symptoms and the ultimate collapse to a reflex action upon the vital centers in the central nervous system resulting from injury to the nerve endings and important plexuses of the splanchnic area. The experiments of Goltz,^{3,4} in which he was able to produce shock in the frog by repeated blows on the viscera, are frequently quoted in substantiation of this theory. Severe pain sensations undoubtedly influence adversely the patient's general condition, and it is possible that injury to the rich and important nerve supply of the splanchnic area may be of more significance than is now believed.

Another of the older theories, which must be mentioned because of its importance in the past, was the belief that a bacteriemia was responsible for the illness and death of the patient with obstruction. This view has been so conclusively disproved by McClure,⁵ Hartwell and Hoguet⁶ and McKenna,⁷ that it does not seem necessary to review the evidence.

Turning to the more important theories advanced today to explain the symptom complex of obstruction, the belief that this disease is the result of the absorption of a toxin from the bowel will be discussed first. In spite of the lack of evidence on certain points and the contradictory nature of the evidence on others (for a comprehensive analysis and bibliography see Cooper's⁸ monograph), this theory is the one most widely held today; and although there is difference of opinion

as to the nature and source of the toxin, it probably furnishes the best explanation of the cause of death in those types of obstruction where damage to the bowel wall or mucosa can be demonstrated. The accumulating evidence, however, that the type of obstruction (simple high obstruction) which for a long time was considered to furnish the most striking clinical evidence of the absorption of a toxin can be satisfactorily explained on another basis,* makes one adopt a more critical attitude toward the whole toxemia theory.

TOXEMIA IN OBSTRUCTION

GENERAL OUTLINE OF THE PROBLEM. The conception that the symptoms and death of patients with acute intestinal obstruction might be due to the absorption of a toxic substance has been held for a long time.⁹ It is a plausible theory: the fluid content of the obstructed intestine is extremely foul; and it was found early in the nineteenth century that drawing off this fluid by means of an enterostomy often saved the patient's life even though the obstruction was not relieved. Furthermore, the patient with obstruction often presents the general appearance and symptoms suggestive of one suffering from poison.

The efforts of experimenters have been directed in the main, first toward proving that the disease is caused by a toxin, and second, toward determining the chemical nature of this toxin, its source and its mode of absorption. The literature on the subject is complicated by the fact that many different experimental types of obstruction have been used and the assumption frequently made that the underlying toxemia was the same in all cases. This is clearly unwarranted; the cause of symptoms must be studied separately for the various types.

The first phase of the problem, namely proving that the disease is caused by the absorption of a toxin, appears on casual inspection to be easy. It is a simple matter to draw off the intestinal content that collects above an obstruction and to

* See p. 387.

inject it intravenously (either in the state in which it comes from the intestine or after carrying out various procedures with a view to obtaining the toxin in a purer and more concentrated form) into a normal animal. The animals receiving such injections usually die promptly* with symptoms that are said to resemble those shown by animals with actual obstruction: that is to say, the animals usually vomit, go into collapse, and die in a short while.¹²⁻¹⁴ This type of experiment, with modifications, has been carried out innumerable times during the past thirty years; and the results obtained constitute the major direct evidence that the absorption of a toxin from the intestinal tract is responsible for the symptoms of obstruction. It is obvious that this method is crude in the extreme; and there are many assumptions in the line of argument for which direct evidence is lacking. There need be no serious question but that the fluid which accumulates above an obstruction usually contains toxic material.† A plethora of toxins have in fact been isolated and shown to be poisonous for animals when introduced in the appropriate manner. The toxicity of the fluid varies with a number of factors, the type of obstruction being an important point.¹⁴ The salient questions are these: Are the toxins found in the obstructed loop absorbed; and, if so, are they responsible for the symptoms and death following acute obstruction?

In attempting to show the mechanism of absorption, or indeed to show in any direct way that absorption of toxin from the obstructed bowel occurs at all, grave difficulties have been encountered.

CHEMICAL NATURE OF THE TOXIN. While most workers are agreed that the intestine, above an obstruction, contains substances which when injected intravenously into a normal animal produce toxic symptoms, there has been a wide divergence of opinion as to the nature of these toxins.

* Several observers have reported that when the Berkefeld filtrate of the contents of a strangulated loop is injected, no toxic effects are noted.^{10,11}

† At times even the normal contents of the small intestine are toxic.¹⁵

It is not surprising, considering the complex nature of the fluid that collects above an obstruction, containing as it does innumerable bacteria and a large amount of protein material in all stages of decomposition, that half a dozen or more different types of toxins should have been isolated. Nesbitt,¹⁶ among the earlier workers, reported the finding of choline and neurine; Murphy and Brooks¹⁴ thought the toxic substance resembled a ptomaine. Exhaustive studies were carried out by Whipple and his co-workers¹⁷ in order to determine the chemical nature of the toxic substance found in their closed loop experiments. They concluded that it was a primary heteroproteose, having the following characteristics: it resists autolysis and pancreatic and tryptic digestion; it is thrown out of solution by five volumes of alcohol or by half saturation with ammonium sulphate; it is readily soluble in water; it is not injured by boiling; and it is not removed by dialysis. In 1919, Dragstedt and his co-workers¹⁸ came to the conclusion that toxic amines were formed by the action of bacteria on amino acids.

Certain workers have put forward the attractive theory that histamine* is the responsible factor in the toxemia of obstruction. Its presence, in minute concentration, was first demonstrated in 1921 by Meakins and Harington²² in the cecum and colon of human patients several weeks after the removal of an intestinal obstruction²³; but after studying its absorption from the intestine and balancing all the evidence they decided that it was probably not an active agent in causing intestinal intoxication. More positive results were obtained by Gerard²⁴ in 1922. He found that histamine was present in the fluid from seven out of eight intestinal loops and that the contents of the one loop that did not show histamine did not

* Dale and his co-workers,^{19,20} in extensive studies of the pharmacological action of this drug, have noted particularly its depressor action on the systemic blood pressure, due to its effect on the capillaries. Cannon²¹ and others have considered that some histamine-like substance is the cause of traumatic shock. Since shock-like symptoms and, in the terminal stage, a low blood pressure, are met with in intestinal obstruction, the idea that histamine might be a common agent in both traumatic shock and acute obstruction aroused considerable interest.

produce toxic symptoms on injection. This author also showed the presence of a combined histamine derivative in obstructed loop contents. Sweet²⁵ believes that some histamine-like substance is absorbed from the obstructed intestine, and suggests that it exerts its injurious action chiefly by injury to the adrenals. (See also Ellis.²⁶) Andrus et al.²⁷ also believe that absorption of some histamine-like substance plays a rôle in the intoxication in certain types of obstruction. Extensive studies undertaken by Wangenstein and Loucks,²⁸ however, failed to show any absorption of histamine from a simple obstruction of the small intestine of two days' standing; they considered that absorption of histamine was slight even from strangulated but still viable segments.

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CHAPTER XXX

THEORIES AS TO CAUSE OF DEATH (Continued)

TOXEMIA IN OBSTRUCTION (Continued)

SOURCE OF TOXIN. *Toxins Derived from Food Substances.* The idea that the symptoms of the illness accompanying obstruction are due to toxins formed by the action of bacteria or digestive ferments on food is an old one and is now of merely historical interest. It is closely related to the more general idea of intoxication by intestinal products formed as a result of constipation or "intestinal stasis," the so-called "auto-intoxication"; and in the older literature considerable emphasis was laid on the increased amounts of indican in the urine, and on other evidences of intestinal putrefaction. The toxins formed in this way which have been considered responsible for the symptoms of intestinal obstruction are varied: among them may be mentioned peptone and other substances formed in the course of protein digestion; phenol, indole, and skatole; putrescine; cadaverine; a large number of organic acids; ptomaines; and, finally, neurine and choline, which are derived from lecithin. These latter substances, which are highly toxic, were isolated by Nesbitt¹ from the contents of the obstructed intestine after feeding egg-yolk.

Bokai's name² should also be mentioned in connection with early studies on toxins derived from food. This author stressed the rôle of organic acids as irritants in bringing about inflammation and increasing peristalsis.

That toxic substances of the types already mentioned may at times be extracted from intestinal contents is well recognized. Food, however, certainly does not play any important part as a source of the toxin in obstruction, for it has been shown that if the animals are starved for a considerable time before the production of the obstruction the course and duration of the disease are not essentially changed.³

Digestive Ferments as a Source of Toxin. Bile and Pancreatic Juice. The bile⁴ and pancreatic juice^{5,6} have been considered to be possibly responsible for the intoxication of obstruction, either by absorption into the blood stream under the abnormal conditions existing, or by producing through their digestive action in the obstructed intestine substances which are absorbed and which then give rise to the symptoms. The pancreatic juice, because of its powerful protein and fat-splitting ferments, has received special attention. Sweet⁵ in his earlier work thought that the pancreatic ferments were chiefly responsible for the toxemia of obstruction and that the symptoms were comparable to those shown by a patient with acute pancreatitis; he believed that both diseases were due to the same toxin produced by the same digestive ferment. He later⁷ modified this view because he thought that he could still demonstrate a toxin although the pancreatic juice was excluded. (See also experiments of McClure.⁸) Draper⁹ likewise altered his view that bile was a toxic factor in obstruction, for he was able to show that the exclusion of bile from the obstructed intestine did not alter the picture.

In connection with the rôle of bile in intestinal obstruction, the work of Brockman¹⁰ may be mentioned. This author considered that the symptoms of obstruction of the small intestine were due to absence of bile from the intestinal tract, and reported beneficial results from the administration of bile by rectum. Benedict et al.,¹¹ however, did not consider lack of bile a factor of importance in the death of the animals with acute mechanical obstruction. Jenkins'^{12,13} experiments on obstruction following short-circuiting of biliary, pancreatic and duodenal secretions into the gut below the point of obstruction, are interesting but somewhat complicated, and the results do not appear to be conclusive.

Formation of a Toxin in the Duodenal Mucosa. The belief that the absorption of a toxic substance elaborated in the duodenal mucosa is responsible for the symptoms of obstruction occupies a large place in the literature. The first advocates of

this theory, Roger and Garnier,^{14,15} and Draper,⁹ used simple high obstruction (see Fig. 60) in their experimental work; and they attributed the death of the animal to an absorption of a toxin.* In the theories of both Roger¹⁵ and Draper⁹ there was the underlying idea that the disease they produced in animals resembled a deficiency disease.† Draper was very close to the truth when he pointed out that the symptoms shown by his animals (muscular tremors, spasticity of the flexor muscles, high pulse rate, etc.) “peculiarly resembled those of parathyropriva”; evidently his animals showed symptoms of gastric tetany due to the loss of chlorides in the vomitus and consequent alkalosis. But this was before the work of MacCallum et al.¹⁸ had shown the true nature of gastric tetany. It would seem that the result of the early experiments with simple obstructions could be explained much more satisfactorily by the theory that death was due to the loss of water with the sodium and chloride ions than by the elaborate and complicated ideas centering about the existence of a toxic secretion of the duodenum. The experiments of Murphy and Brooks,¹⁹ and Dragstedt et al.²⁰ have furnished convincing evidence that the normal secretion of the duodenum is not toxic.

Turning from these experiments, in which a simple obstruction of the intestine was produced, to those in which a different type of experimental procedure was employed, the experiments of Whipple and co-workers will be considered.

In 1912, Stone, Bernheim and Whipple²¹ published the first of a series of papers that extended over a number of years, in

* In the work of these investigators there was the belief that a toxin was always present in the duodenum, but that under normal conditions it was neutralized and rendered harmless. Draper considered that the poison of the duodenum was normally neutralized by the secretions of the jejunum. (See also Ref. 16.) He was led to this belief by the fact that when he placed the obstruction a short way down the jejunum the animals lived longer than when the obstruction was located in the duodenum. In the light of modern knowledge, this could be explained more simply by assuming that there is not such a rapid loss of the digestive secretions in the lower obstruction.

† If instead of a deficiency of some complex organic substance a deficiency of water and sodium and chloride ions be assumed, simple high obstruction is a deficiency disease; but the ideas current about 1910,¹⁷ that the duodenum produced a specific secretion necessary for the life of the animal, have been thoroughly disproved. See footnote, p. 319.

which they developed clearly the idea that under conditions of obstruction the mucosa of the duodenum develops a toxic secretion. They were able to isolate a toxin from the contents of an obstructed duodenal loop, and carried out extensive studies to determine its biological properties and chemical nature. In order to understand the significance of their work we must consider these experiments in some detail, for their method represents an important and new type of experiment that has since been used extensively in attempting to solve the problem of the cause of death in intestinal obstruction.

These workers were engaged in isolating a loop of duodenum in order to obtain its secretion unmixed with pancreatic juice. They divided the duodenum just distal to the entrance of the pancreatic duct and again at the beginning of the jejunum. They re-established the continuity of the intestinal tract by an end-to-end anastomosis and delayed opening the isolated loop for twenty-four hours in order to avoid sepsis. They found that the dogs so operated upon usually died within twenty-four hours. This was the beginning of the work on closed duodenal loops. (See Fig. 62B.)

The method that these authors used in most of their subsequent work was to place a tie just distal to the pancreatic duct and a second tie at the junction of the duodenum and jejunum; they then re-established the continuity of the gastrointestinal tract by a gastroenterostomy (see Fig. 62c). They found that animals so prepared usually died in from twenty-four to sixty hours.²¹ They at first thought that drainage of the closed loop would save the animals' lives;²¹ but later²² reported that the dogs died even though the loop was drained.*

These authors found that the contents of the obstructed loop were highly toxic when injected into the blood stream of a normal animal.²¹ They further found that they could not detect the toxin if they destroyed the mucosa of the duodenum with

* Sweet's criticism, already quoted (footnote, p. 349) is that the animals still had obstruction, through failure of the gastroenterostomy to function.

sodium fluoride* before producing the obstruction;²³ and they could not demonstrate the toxin in loops from the lower ileum or colon.²⁴ They also found that when they constructed loops in the lower ileum the animals lived much longer and did not show the toxic picture presented by animals with the duodenal loops.²⁵ These facts led them to believe that the toxin was formed in the mucosa of the duodenum. They considered that it was a proteose and that an animal could be at least partially immunized against it.²⁶

The work of these authors exerted a profound influence upon the current ideas in regard to the cause of death in intestinal obstruction. On the basis merely of the experiments with simple high obstruction, as carried out by Roger, Garnier and Draper, it is questionable how wide an acceptance the theory of a toxic secretion of the duodenum would have gained; but the experiments of Whipple and his co-workers with the closed duodenal loops seemed to furnish direct evidence of the existence of such a secretion. The type of obstruction employed was supposed to simplify the situation, † for the secretions of the pancreas, stomach and liver did not enter the closed loop.

As a matter of fact, this method introduced a whole new set of conditions, for no drainage back into the stomach was possible from these blind loops, and since secretion is profuse in the upper intestine, the serious factor of damage to the bowel wall from increased pressure entered into the situation. Whipple

* The animals with the mucosa destroyed in this manner died of peritonitis and bronchopneumonia.²³

† Herrin and Meek²⁷ write in 1933: "The introduction of isolated intestinal loops by Whipple and his co-workers was the first important advance that has been made in the study of intestinal obstruction by experimental methods. Outstanding as this procedure was, it still did not allow the various factors concerned in obstruction to be dissociated from each other. We believe that our method of distending open fistulous loops accomplishes this necessary condition, and that for the first time, some of the factors can be studied independently." This last suggestion is open to question. Their interesting experiments, however, are valuable in showing that distention is capable of stimulating secretion from a loop (see p. 177), and also re-emphasize the serious effects of the continued loss of intestinal secretion; they thus have some bearing on the cause of death in high intestinal obstruction. Their experiments, however, seem more nearly comparable to high intestinal fistulae than to intestinal obstruction (see p. 317).

and his co-workers considered that their toxin was a secretion by the duodenal mucosa; another possibility, however, must be considered, namely that the toxin was formed by bacterial action, and while no direct evidence can be produced in favor of this view, considerable indirect evidence can be brought forward to show that this, and not a toxic secretion of the mucosa, is the correct explanation of the cause of death in the animals with closed duodenal loops. The evidence for this view will be presented in the next chapter.

In conclusion it may be said that the experiments with simple obstruction of the duodenum furnish no real evidence of a toxic secretion; and that the symptoms and death of the animals can be much more simply explained on the basis of the loss of digestive secretions.

The animals with closed duodenal loops show damage to the bowel wall from increased pressure within the loop; these animals probably die of a toxemia. This is very likely due to the action of bacteria, and not to a toxic secretion of the duodenal mucosa.

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CHAPTER XXXI

THEORIES AS TO CAUSE OF DEATH (Continued)

TOXEMIA IN OBSTRUCTION (CONTINUED)

SOURCE OF TOXIN (Continued). *Toxins Formed by Bacterial Action.* As stated in Chapter XXIX, the theory held by earlier workers that bacterial invasion of the peritoneal cavity and systemic circulation was responsible for the toxemia has in general been abandoned. The question that has not been settled is whether or not the action of bacteria in the lumen or wall of the intestine is responsible for the death of the animal.

All observers agree that the intestinal fluid above an obstruction contains an innumerable number and variety of bacteria. This is true for all types of obstruction; even in the upper portions of the intestinal tract, where under normal conditions the bacterial flora is sparse,¹ a rich and varied flora develops rapidly under conditions of obstruction.² It is a plausible theory that this profuse growth of organisms, incubated as it were in the obstructed intestine, elaborates a toxin which is absorbed into the body and produces the clinical picture of toxemia shown by patients with acute obstruction.

As has already been pointed out, it is usually an easy matter to prove that the contents of an obstructed intestine are toxic on injection into a normal animal. The difficulty that has been encountered by the advocates of the theory that bacterial action is responsible for the toxemia has been in proving that bacteria produce the toxic material found in the obstructed intestine and that this toxin is absorbed and causes the death of the animal.

As regards the responsibility of the bacteria for the production of the toxin in the loop contents, the early workers for the most part either assumed that this was so because the bacteria were so abundant and other factors such as food or digestive secretions had, they believed, been ruled out;² or they

carried out further experiments in order to show more directly the rôle of bacteria. Filtrates from aerobic cultures of the intestinal flora, for example, were made by Clairmont and Ranzi³ and were considered to elicit the same toxic symptoms as those produced by an injection of the toxic material from the obstructed intestine.

In 1911, Murphy and Vincent⁴ in their studies on the cause of the toxemia used a different type of obstruction: in addition to simply blocking the lumen of the bowel they occluded the mesenteric circulation in various ways. This type of obstruction has already been discussed,* so that it suffices here to say that they made it clear that interference with the circulation of the bowel wall is an important factor in the production of the symptoms of obstruction, a fact that had been recognized in the clinical field for a long time. They concluded that bacteria and endotoxins were responsible for the toxemia, because they found that when small quantities of the intestinal contents were injected intravenously into a normal animal, profound symptoms of toxemia were produced; whereas if the contents were passed through a Berkefeld filter, which removed the bacteria, the filtrate was not toxic. (See also Refs. 5 and 6.)†

In order to prove the importance of bacteria, a number of workers have attempted to produce obstructions free from bacteria: if under these conditions the animal did not become sick it seemed safe to conclude that bacteria were responsible for the symptoms shown by animals obstructed in the usual manner. The difficulty with such a type of experiment is that it is impossible to get rid of the bacteria without considerably altering the other conditions of the experiment.

Among such experiments are those of Murphy and Brooks,⁷ who isolated a loop of jejunum; one end of the loop they closed,

* See p. 346.

† The fact that no soluble toxin could be demonstrated after Berkefeld filtration of the contents of strangulated intestine was one of the factors that led Wilkie⁶ to discount the importance of toxemia in strangulation.

the other end was brought out as a fistula through the abdominal wall. The continuity of the intestinal tract was re-established by an end-to-end anastomosis. They reported that after a number of days the loop became sterile, cultures showing no growth. The draining end of the loop was then obstructed. The animal showed none of the symptoms that follow the ordinary obstruction of a jejunal loop. Four days later the contents were found sterile and were non-toxic on injection into another animal. When the loop contents became infected the usual symptoms of obstruction promptly followed.

Murphy and Brooks⁷ also employed another method to show the rôle of the bacteria in the production of the toxemia: they produced obstructions of the gall bladder and its circulation, with and without the presence of bacteria. They showed that under ordinary conditions the circulation to the gall bladder could be obstructed to the point of complete gangrene without any ill effects; whereas if the circulation to the gall bladder were blocked and at the same time organisms obtained from an obstructed intestine were injected into the gall bladder, the animals became ill. These authors considered that this illness was comparable to the toxemia of intestinal obstruction, and the conclusion was drawn that the toxemia of intestinal obstruction was due to the action of bacteria.

More recently, Dragstedt and his co-workers⁸⁻¹¹ have been strong advocates of the theory that bacteria are responsible for the symptoms of intoxication. A considerable portion of the work of these authors has been directed toward disproving the theory of a toxin secreted by the duodenal mucosa, advanced by Whipple and his co-workers.¹² Dragstedt et al. have employed a method similar in general principle to that used by Murphy and Brooks,⁷ in that they have attempted to produce obstructions free from bacteria. These authors^{8,10} first washed isolated loops in the lower jejunum with water and ether. When the loops were obstructed after this procedure the animals survived almost indefinitely; but it was found that the loops were not sterile. The survival of these animals was

attributed to the fact that ether acted as an astringent to the mucous membrane, cutting down secretion: if the secretion was not abundant no distention occurred, and consequently no damage was done to the mucosa. It was later shown that other astringents, such as tannic acid, had the same action, although they were not bactericidal.¹⁰

Dragstedt et al. used a second method¹³ of eliminating the bacteria. They found that if a loop in the upper intestinal tract were isolated with its blood supply intact, and were then washed, the continuity of the intestinal tract re-established and the loop dropped back into the peritoneal cavity, while about half the animals so operated upon died of general peritonitis, the remainder lived; and when the abdominal cavity was opened after a lapse of a number of days it was found that the loop had been sterilized by the action of the peritoneal fluids. If at this time the ends of the loop were closed the animals lived almost indefinitely and death did not follow even when obstruction to the mesenteric circulation was produced. These authors considered these experiments proof that bacteria were responsible for the disease.

Type of Bacteria Responsible for the Toxemia. Since the bacterial flora of an obstructed loop is so exceedingly abundant and so diverse in type, most workers have considered it a hopeless task to hold any one organism or group of organisms responsible for the toxemia. Cannon et al.,¹⁴ however, concluded that it was always associated with a proteolytic flora.

It is generally agreed that the normal intestinal flora includes a large number of anaerobic organisms.⁵ In 1926, Williams¹⁵ advanced the hypothesis that the Welch bacillus was the organism particularly responsible for the toxemia. In support of his theory he cited certain points of similarity between the clinical picture of intestinal obstruction and that of gas bacillus infection, and also the fact that he had found *B. welchii* in large numbers in the contents of obstructed intestines and in the vomitus from such cases. On the basis of this theory he gave *B. welchii* antitoxin to two heterogeneous

groups of patients suffering from intestinal obstruction or peritonitis, with an apparently favorable effect upon the course of the disease and with a lowering of the mortality rate.

Bower and Clark¹⁶ reported a series of 25 cases of intestinal obstruction or peritonitis, treated with *B. welchii* antitoxin. They believed that its administration had favorably influenced the course of the two diseases. Morton¹⁷ also, using a somewhat similar method, considered that the *B. welchii* was the important organism; this author carried out his experiments on dogs and used simple high obstruction.

McIver et al.⁵ were, on the other hand, not able to find sufficient evidence that the *B. welchii* played an important rôle in the production of the toxemia. The type of obstruction used in their experiments was a closed loop with ligation of its veins; the animal used was the cat. Oughterson and Powers¹⁸ and Thurston¹⁹ also were not able to find sufficient evidence to conclude that the *B. welchii* was an important factor in the toxemia.

The *B. welchii* normally found in the intestinal tract are of a rather low degree of virulence. It is obvious that this must be the case, when one considers the large number of instances in which the intestine is drained through an abdominal wound, and the rarity with which gas bacillus infection of the abdominal wound takes place, in spite of the fact that *B. welchii* may frequently be cultured from the draining wound.* It has also been shown that in intestinal loops under experimental conditions these organisms are usually relatively or entirely avirulent.⁵

It must be borne in mind that in the experimental work the chief method of judging whether or not *B. welchii* were responsible for the toxemia was to determine whether *B. welchii* antitoxin was efficacious in prolonging the life of the animals. It is obvious that this method is open to many objections. Furthermore, at best it furnishes evidence as to only

* A generalized infection with *B. welchii* does occasionally occur.

a relatively small group of anaerobes. Anaerobic organisms are without doubt usually present in large numbers in an obstructed loop, where often such good anaerobic conditions exist;²⁰ so if one is committed to the bacterial theory of the toxemia there seems no good reason against giving the anaerobic organisms a part as well as the aerobic, and the work of Meleney et al.^{21,22} on peritonitis suggests that both types of organisms may play a rôle.

While the question as to whether aerobic or anaerobic organisms are responsible for the toxemia has not been settled, the experiments of Murphy and Brooks,⁷ Dragstedt et al.¹⁰ and others furnish evidence that the toxemia in experimentally obstructed loops is due to bacterial action and not to a toxic secretion of the mucosa of the intestine. The question as to how closely these isolated loops correspond with obstructions encountered in humans has already been discussed.

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CHAPTER XXXII

THEORIES AS TO CAUSE OF DEATH (Continued)

TOXEMIA IN OBSTRUCTION (Continued)

ABSORPTION OF THE TOXIN. *The Mucosa.* It may be considered an established fact that no absorption of a toxic substance can take place through a normal mucosa.* In other words, if the most virulent toxic fluid from an obstructed loop be placed in the normal intestine of another animal, no symptoms follow.†²⁻⁴ This narrows the question to a consideration of what changes take place under conditions of intestinal obstruction which permit the poison to be absorbed. The most reasonable explanation is that the mucosa is so damaged or destroyed that it is permeable to the toxin from the lumen, or that after the barrier of epithelial cells is broken down the toxin may be actually formed in the intestinal wall itself, probably by the action of bacteria or by autolytic changes.

That degenerative changes frequently occur in the intestinal mucosa under conditions of obstruction and are the important factors in absorption has been stressed by many investigators.^{2,8-11} These changes are usually attributed to the fact that the circulation to the bowel is damaged by distention. Since increased intrainestinal pressure is important in causing damage to the circulation and has also been considered to

* It is also true that if an obstructed loop be formed and toxic intestinal fluid placed in it at the same time, the onset of symptoms and death is not hastened.¹

† Guillaume⁵ is one of the few who question this, and he brings no real evidence in favor of a contrary view. The clinical observation (stressed by Guillaume) that a patient may go into collapse after being relieved from an obstruction does not prove that this occurred because the toxic material had passed into the normal bowel below the point of obstruction and had been there absorbed. As pointed out by Brooks et al.,⁶ there are other explanations of this observation, among which may be mentioned the possibility of increased absorption from the already damaged mucosa; and in the case of strangulations there may be a puddling of blood in the dilated intestinal vessels after relief of the strangulation. Elman⁷ suggests that when even a simple obstruction is suddenly released or a distended intestine quickly decompressed, great loss of blood and fluid into the splanchnic area may take place and produce serious symptoms. (See also p. 311.)

play a direct rôle in causing absorption, it will be discussed in more detail.

Intraintestinal Pressure. As already stated, an increase in intraintestinal pressure has been supposed to influence absorption in two ways: indirectly by interfering with the circulation^{8,9} and so producing injury to the mucosa; and directly by forcing the toxin into the absorbing channels.^{1,2,12}

A number of experiments have been carried out to show the effect of increased pressure upon the circulation of the bowel. Van Zwalenburg¹³ in 1907 placed an electric light in the intestine and observed under the microscope the effect on the circulation in the bowel wall of gradually increasing the pressure in the intestine by introducing water through a canula. He found that the circulation was markedly affected by high pressure.*

Gatch et al.¹⁴ and Dragstedt et al.¹⁵ have also studied the effect of distention on the circulation of the blood and found that the flow was diminished when the pressure in the intestine was increased. Dragstedt considered that interference caused by distention was greatest in the duodenum and least in the colon. Van Beuren's studies^{10,16} of the pathological changes produced in the bowel by distention are also important: briefly these changes consisted of areas of infarction, necrosis and at times perforation, occurring for the most part on the anti-mesenteric border of the intestine. Burget et al.^{17,18} found that animals with closed intestinal loops could be kept alive almost indefinitely if overdistention of the loop were prevented by aspirating the contents; if the pressure within the loop were allowed to increase, the symptoms of obstruction would immediately return.†

In regard to the second point, namely, that an increase in intraintestinal pressure directly affects the absorption, experi-

* At 30 mm. of mercury some capillary streams were stopped. It required 130 mm. before all the circulation stopped; but as Van Zwalenburg pointed out, venous flow was probably retarded at slight increase of pressure.

† It is striking clinically that a patient with subacute obstruction may show but few symptoms so long as a minute opening for the passage of gas exists; but if the blockage becomes complete, a sudden onset of severe symptoms usually occurs.

ments were carried out in 1924 by Stone et al.¹² and in 1927 by Owings et al.¹⁹ in order to determine the relation of increased pressure to absorption. Stone believed pressure was an important factor, not because it injured the mucosa and so indirectly permitted absorption of toxic substances, but because its presence directly caused abnormal absorption, presumably by forcing the toxic material into the absorbing channels. Murphy and Brooks¹ had previously advanced the view that increased intrainestinal pressure favored absorption, and made practical use of it in one of their experiments; they believed, however, that there was an injury to the mucosa before absorption could take place. Owings et al.¹⁹ made measurements of the intrainestinal pressure both under normal conditions and following obstructions: they found it considerably elevated during obstruction.* (For further discussion of distention see p. 175.)

Dissemination of Toxins. Granted that the toxin has passed the barrier imposed by the mucosa, there are three ways in which general dissemination might take place: the toxin might be taken up by the blood stream; it might be carried by the lymphatics; or it might diffuse into the general peritoneal cavity and be absorbed from that source. One might therefore look for the toxin in the blood, the lymph or the peritoneal fluid.

Blood Stream. The assumption has often been made that absorption takes place by the blood stream. The theory is plausible, but there is no important evidence, either for or against it. Experiments^{4,20,21} showing that the blood of animals dying of intestinal obstruction is not toxic when injected into another animal prove nothing beyond that fact; the blood of animals poisoned in another manner may also be non-toxic when transferred to another animal.³ On the other hand, the work of Sugito²² and Scholefield²³ on the toxicity for mice of the blood of animals with obstruction is not convincing.

* It was, however, considerably below the high pressure created by Van Zwalenburg,¹³ Gatch,¹⁴ and others. It is possible that some other factor besides increased intrainestinal pressure affects the capillary circulation of the obstructed bowel: perhaps the permeability of the mucosa may be increased by the irritative action of the intestinal fluid so that a direct toxic action on the capillaries can take place.

Lymphatic Channel. Most of the adherents of the theory that there is an absorption of toxin from an obstructed intestine assume that absorption occurs both by way of the blood stream and by way of the lymphatics. Certain workers, however, have especially stressed the importance of the latter route. Murphy and Vincent² thought the onset of toxic symptoms was checked by the ligation of the lymphatic channels in the mesentery; their experiments were, however, not adequately controlled.

Murphy and Brooks¹ also collected material from the thoracic ducts of animals in whom obstructed loops with occluded veins had been formed, and from animals with obstructed loops in which the toxic content was under pressure. The collected fluid was found, in certain instances but not always, to be toxic on injection into a normal animal. Objections can be made to these experiments: the fluid was kept over night before injection into the normal animal; and in the control experiments slight symptoms (vomiting) were shown on the injection of thoracic duct fluid from an animal that had no obstruction. They are, however, interesting experiments and it would be most desirable to have the work confirmed.

Morton,²⁴ using colloidal silver which has a selective staining action for lymphatics, was not able to demonstrate any staining of the thoracic lymphatics unless an actual perforation of the loops occurred.

Peritoneal Cavity as a Source of Absorption. Under conditions of obstruction there is usually an increase of free fluid in the peritoneal cavity. This is particularly true where there is an interference with the venous circulation. A toxin from an obstructed loop might diffuse into this fluid and then be taken up by the abdominal lymphatics and blood vessels. This possibility has been tested chiefly with the peritoneal fluid after producing interference with the circulation of the bowel (Fig. 61). No toxic substance, however, can be detected in the peritoneal fluid^{1,4,25} by injecting it intravenously into normal animals even when large quantities are used.

Although there is no direct evidence, there is considerable indirect evidence that diffusion of a toxin into the peritoneal cavity in presence of strangulation takes place. In 1812 Travers,²⁶ in studying the cause of death in strangulated hernia, drew a coil of intestine out of the abdomen and closed the wound around it in such a way as to strangulate the blood supply. The animal lived six days; the strangulated gut had sloughed and a fecal fistula had been established. Two other animals in which an internal strangulation was produced died on the second and third days.* White and McIver† also found that where a loop of intestine with its venous supply obstructed was brought outside of the peritoneal cavity the animal lived as long as though only a simple obstruction existed. McIver, White and Lawson²⁵ showed that if a twenty-four-hour strangulated loop were suspended in salt solution in the incubator for a few hours the salt solution surrounding the loop became toxic to normal animals. Foster and Hausler²⁸ concluded that placing short strangulated loops in a rubber bag (in order to isolate them from the peritoneal cavity) prolonged the life of the animal.

STUDIES ON ABSORPTION OF OTHER SUBSTANCES, UNDER CONDITIONS OF OBSTRUCTION. Since it is so difficult to obtain direct evidence of the absorption of the toxin, indirect evidence has been sought by studying the absorption of various other substances under conditions of obstruction. The gist of those experiments is that the absorption of most substances when placed in the obstructed intestine seems to be diminished rather than increased. Braun and Boruttau²⁹ after testing the absorption of strychnine from an obstructed loop came to the conclusion that the absorption of this drug was definitely diminished. Enderlen and Hotz³⁰ later determined the absorp-

* This author recommended that in strangulated hernia where the bowel does not appear to be viable, an opening should be made in the intestine and the intestine allowed to drain. His contention is that under these conditions (i.e., strangulation outside of the peritoneal cavity) the patient dies of obstruction, not from intestinal gangrene. (See also p. 126.)

† Experiments reported in article by White and Fender.²⁷

tion rates of solutions of sodium chloride and glucose, and found that these also were decreased in the presence of obstruction. More recently Palma,³¹ using phenolsulphonphthalein, came to the same conclusion.

Various types of dyes have also been used in studies on absorption. Morton²⁴ in his studies on absorption under conditions of obstruction worked with two classes of dyes: brilliant vital red and trypan blue, representing dyes of large molecular structure; and congo red, cloth red and congo blue representing dyes with smaller molecules. These dyes were placed in obstructed loops but could not be demonstrated in the vessels or in the wall of the living intestine.

The experiments of Hettwer and Kriz^{32,33} on the absorption of protein from ligated intestinal loops are of interest and introduce a new method that may throw considerable light on the problem. These workers sensitized guinea-pigs to horse serum and found that they could produce anaphylactic reactions by introducing serum into ligated intestinal loops. This was shown to occur more readily when short rather than long loops were employed and they considered that the increased pressure in the short loops was an important factor in absorption. The experiments are of particular interest in that they furnish direct evidence of the absorption of foreign protein from an obstructed loop.

In conclusion it may be said that although these numerous experiments on the subject of absorption from the intestine under conditions of obstruction have furnished a great deal of valuable information, there is no conclusive evidence on the mechanism of absorption or the pathways by which general dissemination of the toxin occurs.

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CHAPTER XXXIII

THEORIES AS TO CAUSE OF DEATH (Continued)

THE THEORY THAT DEATH IS DUE TO DEHYDRATION

The fact that dehydration appears as a frequent and severe complication of intestinal obstruction has been known for a long time.*^{1,2} It remained, however, for Hartwell and Hoguet³ in 1912 to formulate the definite theory that death in high intestinal obstruction is due to dehydration brought about by the loss of the digestive secretions. Neither they nor more recent advocates of this theory, however, have thought that the death in all types of intestinal obstruction could be explained on this basis. The application of the theory has been clearly limited from the beginning to simple obstructions. This fact is emphasized, since at times there has been misunderstanding on this point.

It is well to examine in some detail the important evidence brought forward by Hartwell and Hoguet in favor of their views. These workers found that animals dying from simple high obstruction seldom showed any noteworthy organic change in the intestine above the obstruction.† This led them

* Leichtenstern wrote in 1876: "The change of the features to the form of the facies hippocratica, the sinking of the eyes, the diminished turgescence of the skin, the aphonia, and the dryness of the skin, are due in part to the diminished absorption of water in consequence of the violent vomiting and to the withdrawal of water from the blood and tissues by the profuse sweats which usually occur simultaneously."

Nothnagel (1904) says: "This hippocratic face is, however, due to another factor present in this disease—namely, the great loss of water from the tissues, which is directly due to the profuse vomiting and the attacks of profuse perspiration that occasionally supervene in occlusion of the bowel. The loss of water from the tissues in addition causes wasting of the skin, dryness of the tongue, torturing thirst, and aphonia, conditions which are all occasionally seen in these cases. Leichtenstern has calculated the amount of water lost from the tissues, and his investigation shows that, together with the concentration of the blood, there is relative increase in the percentage of hemoglobin. The loss of water may be very considerable and may amount to 24 per cent of the volume of the blood."

† It was quite otherwise in the case of low obstruction. Here they often found extensive changes in the mucosa (which they attributed largely to circulatory disturbances following distention), and in these instances they could not prolong life by treatment with salt solution. They therefore concluded that when these degenerative changes

to doubt that death resulted from the absorption of a toxin. In seeking some other explanation they were struck by the large amounts of fluid lost by the animals in vomiting; and by a loss of weight (2.2 kg., for example, in three and one-half days) so rapid that it could only be accounted for on the basis of loss of water. These findings led them to restore to these animals the fluid lost, using physiological saline solution. They found that whereas untreated animals died in a few days (three to ten days), the animals treated by administration of normal salt solution, in excess of that lost in the vomitus,* lived three or more weeks, and the animals sacrificed at the expiration of that period gave the impression that they would have lived almost indefinitely if the treatment had been continued. The results of their experiments were so striking that they were led to advance the view that death in simple high obstruction is usually due to dehydration.

The full significance of this really remarkable discovery was not wholly appreciated at the time, largely because attention was focussed upon the theory of toxic absorption and also because a great deal of the experimental work was being carried out with closed duodenal loops, a complicated type of obstruction which is not materially benefited by the administration of salt solution.

The views of Hartwell and Hoguet were supported in 1913 by Wilkie⁶ who stated very clearly that "simple obstruction high in the intestine differs from that lower down, chiefly in the great loss of intestinal secretions by vomiting in the former, compared with the latter, where the secretions are re-absorbed above the obstruction." This investigator obstructed the

in the mucosa occurred, whether in low or in high obstruction, death was due to the absorption of a toxin.^{3,4}

* Whether the fluid is vomited or is retained in the stomach or upper intestinal segment above the obstruction, it is lost to the animals. A most striking example is found in the rabbit: this animal is not able to vomit, and under conditions of obstruction⁵ the stomach acts as a reservoir in which large volumes of fluid collect, the animal showing the low blood chlorides and other findings of dehydration. Hartwell, Hoguet and Beekman⁴ recognized the importance of the loss to the body of fluid accumulated in the stomach and intestines.

duodenum by means of a ligature and placed a rubber tube drain above this; he found that animals so prepared lost as much as one-third of their body weight in the thirty-six hours that they lived. In 1923 Haden and Orr published the first of a series of papers⁷⁻¹⁰ in which they reported their important findings on the changes occurring in the blood chemistry following intestinal obstruction. These changes were, chiefly, a fall in the blood chlorides, an increase in the non-protein nitrogen, and a rise in the carbon dioxide combining power of the plasma. These authors also completely confirmed the observations of Hartwell and Hoguet on the efficacy of salt solution in prolonging the life of animals with high obstruction; and further showed that other solutions, although isotonic, are not efficacious in prolonging the life of the animals.* Gamble and McIver^{5,12} also stressed the importance of sodium and chloride ions, and regarded their loss as largely responsible for the fatal effects of simple high obstruction. For while agreeing with Hartwell and Hoguet that dehydration was responsible for the death of the animals, they considered that dehydration† was dependent on the loss of the sodium and chloride ions. (See "Mechanism of Dehydration," p. 156.)

White and Bridge,¹³ White and Fender,¹⁴ and Gatch et al.¹⁵ have also carried out experiments that furnish evidence that the loss of digestive secretions will adequately explain the death in high obstruction.

Opponents of the view that dehydration is the most important factor in death from high intestinal obstruction cite certain experiments bearing directly on the water content of

* These authors at first thought that the blood chlorides were low because the chloride ion combined with and neutralized some toxic substance. They later modified this view.¹¹

† Some writers, feeling that the term "dehydration" should properly refer only to the loss of water, have used the term "dehydration and dechlorination" to describe the conditions found after upper intestinal obstruction, i.e., decrease in body fluids and a low concentration of chlorides in the blood. This, however, is also incomplete; for, although it is more difficult to demonstrate, a great loss of sodium as well as of chloride ion occurs. Indeed, Gamble and Ross have offered evidence that the loss of sodium is more important than that of chloride. It would seem simpler and better to use the old terminology but to employ it with a broader meaning.

tissues. Draper¹⁶ in 1914 measured the water content of the liver, kidney, muscle and other parenchymatous tissues under normal conditions and following fasting, salivation by pilocarpine and duodenal obstruction. He found the water loss to be about 10 per cent in all three conditions. The fasting and salivated animals showed no signs of debility, while the animals with duodenal obstruction died. Whipple and co-workers¹⁷ reported somewhat similar experiments. They found that dogs, dehydrated by means of drugs, showed no evidence of intoxication and recovered their weight very promptly when given food and water. Ingvaldsen and co-workers¹⁸ measured the water content of spleen, muscle and liver following the production of high and low obstruction in dogs and found that there was only slight lowering of the water content of these tissues.

These various experiments were interpreted as showing that dehydration played no important rôle as a cause of death. Two criticisms may be made of this line of reasoning. In the first place, the loss of fluids produced by the use of pilocarpine is not comparable with the dehydration caused by loss of the digestive secretions, because sodium and chloride ions are not lost to such an extent. In the second place, the water content of the parenchymatous tissues such as muscle and liver does not furnish an accurate estimate of the degree of dehydration. As has already been pointed out, the most extensive loss of water occurs not from richly cellular tissue but from the interstitial spaces, particularly the subcutaneous tissue. This might be expected to be true on theoretical grounds (see Fig. 37), and certain experiments⁵ have indicated that such is actually the case.

The cause of death in simple high obstruction may be summarized by saying that it can be adequately explained on the basis of the loss of the digestive secretions and the secondary changes that follow.

THEORY THAT THE CAUSE OF DEATH IS ANEMIA OF THE VITAL
CENTERS RESULTING FROM CIRCULATORY DISTURBANCES
IN THE SPLANCHNIC AREA

More than twenty years ago, Braun and Boruttau¹⁹ and Wilkie⁶ came to the conclusion that their work did not support the view that death was due to the absorption of a toxin.* Braun and Boruttau were especially impressed by the fact that according to their experiments absorption instead of being increased under conditions of obstruction was actually decreased; and Wilkie was not able to produce toxic symptoms by injection of a Berkefeld filtrate of loop contents or expressed juice from the wall of a strangulated gut. Wilkie further found that animals with strangulation of long loops of intestine died within a few hours with symptoms of shock, a finding which has been more recently confirmed by Foster and Hausler²⁰ and others. Foster and Hausler considered that the time elapsing before death was too short for the development of a toxemia. Braun and Boruttau and Wilkie considered that the most outstanding finding in patients dying of obstruction was the accumulation of fluid throughout the splanchnic area, in the engorged blood vessels, in the intestines above the obstruction, and in the peritoneal cavity itself. This drainage of fluid into the splanchnic area decreases the circulating blood volume, thus lowering the efficiency of the circulation in general and of the cerebral circulation in particular. As Wilkie stated it, "splanchnic paresis with depletion of the systemic circulation is the main factor in producing the symptom complex of acute intestinal obstruction." More recently, Elman and co-workers,^{21,22} doubting that absorption of a toxin is the cause of death in simple low obstruction, have also come to believe that possibly the stagnation of blood and fluid in the splanchnic area may lead to peripheral circulatory failure and shock in this type of obstruction. Scott and

* In simple high obstruction Wilkie considered that the loss of the digestive secretions in vomiting was the important factor. (See theory of dehydration.)

Wangensteen^{23,24} considered that the early fall of blood pressure in animals with ligation of the mesenteric veins indicated that loss of blood from the general circulation was responsible for the death of the animals.

There are many points that may be mentioned in support of the theory of splanchnic paresis. The splanchnic area, when the vessels are dilated, is capable of acting as an enormous reservoir for blood: as an example of this may be cited the noticeable drop in blood pressure following section of the splanchnic nerves. The engorgement of the vessels above even a simple obstruction is often a striking feature of the picture presented by these cases at operation; and a cyanotic tinge of the obstructed intestine, indicating stagnation in the capillaries, is also often noticeable. A great outpouring of fluids into the lumen of the gut usually takes place. All these factors probably play a rôle in most types of obstruction: whether they constitute the decisive element in any type has yet to be established.

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CHAPTER XXXIV

CONCLUSIONS AS TO CAUSE OF DEATH IN DIFFERENT TYPES OF OBSTRUCTION

The widely divergent results that follow different types of experimental obstruction and the distinctive features shown by the various types of obstruction encountered in humans, have made it increasingly obvious that no one theory will explain the cause of death in all varieties of intestinal obstruction. It is also clear from the analysis of the experimental work in the preceding chapters that (as shown in Table xxviii; p. 352) while certain types of obstruction produced in the lower animals are comparable to obstructions occurring in humans, others are not.

In this chapter the author draws certain conclusions, on the basis of the evidence at hand, as to the cause of death in the different types of intestinal obstruction. It must be realized that these conclusions are only tentative and in many instances the evidence on which they are based is meager and at times self contradictory.

CAUSE OF DEATH IN SIMPLE HIGH OBSTRUCTION. In simple high obstruction the most likely explanation of the symptoms and death of the animals seems to me to be dehydration, due to the loss of the digestive secretions in vomiting. The term "dehydration" must include a loss not merely of water but also of the essential sodium and chloride ions. The severe secondary changes that occur in the body fluids and tissues as a result of the loss of these essential materials have been discussed under the section on dehydration (p. 155).

CAUSE OF DEATH IN SIMPLE LOW OBSTRUCTION OF THE SMALL INTESTINE. Simple low obstruction in humans occurs much more commonly than high obstruction and is therefore more important. Unfortunately, experimental obstructions of this type in laboratory animals do not always closely parallel the disease in humans, and therefore the data on

which to base an opinion as to the cause of death is somewhat limited. An outstanding feature at operation or autopsy is usually marked distention of the intestinal coils above the obstruction; the tension is only partially relieved by drainage back into the stomach and by vomiting, and an increase in intractant pressure occurs. This pressure undoubtedly interferes with the capillary circulation and is a factor in starting the degenerative process in the bowel wall which may eventually result in permeability to a toxin. It is possible that vascular stasis and exudation in the splanchnic area are of greater importance than is generally recognized, and warrant further study.

The part played by dehydration in this type of obstruction varies considerably. Where vomiting is profuse, the dehydration is often severe. It cannot alone, however, explain the symptoms and death; for the survival period in animals or humans cannot usually be materially lengthened by the administration of salt solution.

CAUSE OF DEATH IN SIMPLE OBSTRUCTION OF THE COLON. The lower animals with simple obstruction of the colon may live for considerable periods of time without symptoms and eventually die of inanition. In humans, also, the disease is not so quickly fatal as that resulting from obstructions of the small intestine. In neglected cases distention of the cecum may become so great that the blood supply is cut off, areas of gangrene and perforation with resultant peritonitis occur, and death results.

At times the picture presented by the patient is not unlike that found in low obstructions of the ileum; and at operation or autopsy the whole of the small intestine as well as the colon proximal to the obstruction may be distended and filled with the characteristic foul fluid. The death in these cases is presumably the same as that in low obstructions of the ileum.

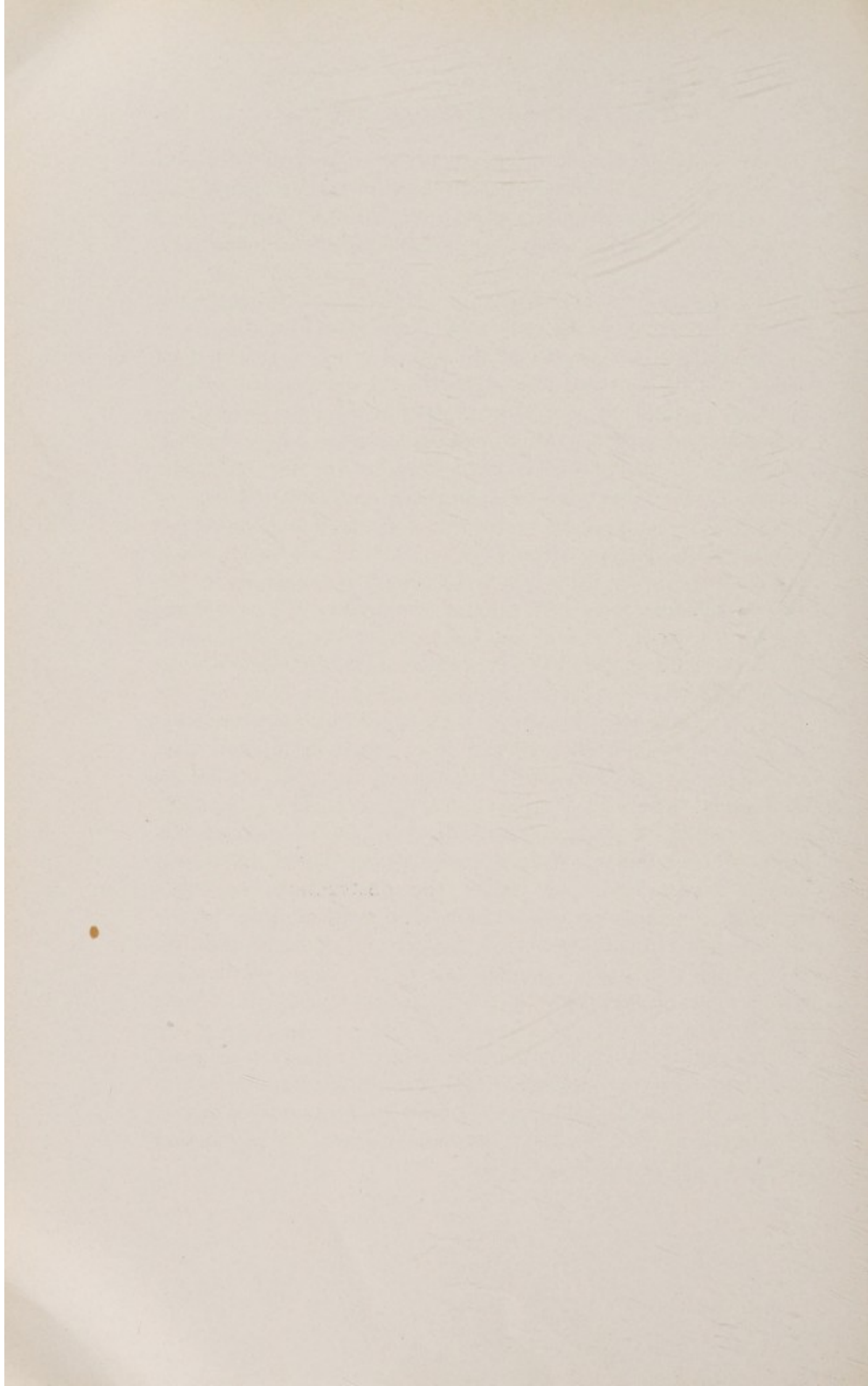
CAUSE OF DEATH IN OBSTRUCTIONS CHARACTERIZED BY INTERFERENCE WITH THE MESENTERIC BLOOD SUPPLY. Interference with the mesenteric circulation to the bowel adds

a new factor to the causes of death already discussed, and is of great clinical importance because of its frequency and seriousness. As examples may be mentioned the various types of strangulation, volvulus, intussusception or mesenteric thrombosis.

The symptoms and death in obstructions characterized by interference with the mesenteric circulation can, it appears to me, be best explained on the basis of a toxemia caused by the action of bacteria multiplying in the necrosing intestinal wall. According to this conception the toxemia would be comparable to that arising from gangrenous tissue located anywhere in the body, modified only by the fact that the intestines are suspended in a large serous cavity where the temperature is most favorable for the rapid multiplication of bacteria and for the disintegration of tissue, and from which absorption readily takes place. Degenerative changes in the bowel wall may go on to a point where a swiftly spreading peritonitis occurs, with or without macroscopic perforation.

It must be realized that the degree of interference with the circulation varies greatly in different instances. It is obvious that where the interference with the mesenteric circulation is at a minimum, the whole picture more nearly approaches that of simple obstruction, the overlapping of types producing numerous variations of the clinical picture. The symptoms are not likely to be greatly modified by the location of this type of obstruction. It is sometimes said that, just as in simple obstruction, the higher the obstruction the more fulminating the symptoms. There is no good evidence in the literature for this statement; on the face of it, it seems unlikely. The suddenness and completeness of the strangulation are probably the most important facts in influencing the course of the disease.

Reduction of blood volume by the transudate of plasma and fluids into the intestine and by stasis in the capillaries and mesenteric vessels may play a rôle in the symptoms and death. The importance of this factor probably increases in proportion to the length of bowel involved.



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