ACUTE INTESTINAL OBSTRUCTION

THE TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

By

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WITH 13 ILLUSTRATIONS

LONDON

J. & A. CHURCHILL LTD.

104 GLOUCESTER PLACE

PORTMAN SQUARE

1945

PREFACE

ACUTE intestinal obstruction is one of the common problems facing the general surgeon and it is undoubtedly one of the most difficult emergencies which he is called upon to treat.

The pathology induced by the lesion is as variable as the site, the extent, and the degree to which the bowel is involved. Not only so, but the release of an obstruction can initiate further pathological processes which, if not treated, may lead to a fatal termination.

The need for early diagnosis of acute intestinal obstruction cannot be over-emphasised, as only then can the surgeon escape having to deal with advanced and rapidly advancing pathological processes for which at present he has but a partial answer.

The treatment of the condition is best considered under four main headings :—

1. The Pathology induced by Obstruction and the Release of Obstruction.
2. The Early Diagnosis.
3. Methods of Treatment.
4. Their Application to Special Types of Obstruction.

The order outlined above will be followed in this book, which is an attempt to state the treatment of acute intestinal obstruction based on extensive reading of the subject, experimental work, and the treatment of more than five hundred cases suffering from this condition.

The author also has the advantage of first-hand study in both British and American hospitals of the methods of treating acute intestinal obstruction and this account is published in the hope that it will aid those who have not had such opportunities. The aim is to give a concise answer based on pathology and surgical experience to the questions of " What to do " and " How to do it " to those faced with its varied problems.

It is a pleasure to acknowledge the help received from surgeons on both sides of the Atlantic, but especially from those on the " other side " who have pioneered so much of the progress herein recorded, and also to thank Dr. J. Rennie, Dr. J. Clark and my colleagues, as well as the hospital management, who all have accorded me far more freedom and opportunity than are generally the lot of a younger surgeon in this country. The experimental

work mentioned in the text was carried out through the courtesy and encouragement of Professor H. N. Green in the Department of Pathology, Sheffield University, and the expenses were defrayed by the Medical Research Council. My thanks to the publishers for their unfailing help are gratefully recorded.

Finally, no surgeon can fail to be conscious of his indebtedness to his patients, common folk whose sufferings and recovery or death are an eternal challenge to his Work, his Faith, his Hope and his Charity.

JUDSON T. CHESTERMAN.

CONTENTS

ACUTE INTESTINAL OBSTRUCTION

PART I

THE PATHOLOGY INDUCED BY ACUTE INTESTINAL OBSTRUCTION

ANY obstruction of the alimentary tract tends to lead to distension of the area proximal to it. The obstructing mechanism may directly affect the vascular supply of the bowel involved or the blood supply may be indirectly impaired later by the distension produced. Hence the two main pathological problems are the effects of distension and the effects of strangulation. Distension is more marked in simple obstructions, as primary strangulation leads either to gangrene and perforation or to a fatal loss of blood volume before severe distension can occur, though distension itself may precipitate strangulation in otherwise simple obstructions. Strangulation without complete obstruction may occur, but is a rare occurrence.

The Effects of Distension

The Character and Source of Distension. An obstructed loop of bowel is found to contain fluid and gas in varying proportions. Most of the fluid is derived from the secretions which are poured mainly into the upper reaches of the alimentary tract.

Rowntree has estimated the daily output as follows :—

Saliva	1,500 c.c.
Gastric juice	2,000–3,000 c.c.
Bile	300–500 c.c.
Pancreatic juice	500–800 c.c.
Succus entericus	3,000 c.c.

which averages some 7,000 c.c. per diem, but Gamble has placed the average daily output even higher at 8,200 c.c., a figure which incidentally corresponds to more than twice the normal plasma volume of the blood.

Distension or section of the extrinsic nerve supply of the bowel tends to increase the secretion, but it is doubtful if this increase occurs when acute obstruction is also present (Montgomery and Swindt, 1934).

Small quantities of the fluid present are due to ingestion and exudate into the lumen.

The possible sources of gaseous distension are swallowed atmospheric air, diffusion into the bowel lumen from the blood stream and the effect of the digestive processes and bacterial action on the intestinal contents.

Hibbard (1936) in a careful review of this subject concludes that these three possibilities account for 68, 22½ and 9½ per cent. respectively of the gas found in the bowel in acute intestinal obstruction.

Distension is further increased by lessened absorption. (*Vidi infra.*)

Effects of Distension on the Bowel Wall. The onset of acute simple mechanical intestinal obstruction produces an increased activity of the bowel, especially on the proximal side, but also on the side distal to the obstruction. This activity increases with moderate degrees of distension and leads to a shortening of the proximal loops of bowel by contraction of their longitudinal muscle coat. (Lengthening may occur in chronic obstruction, occasionally after the release of an acute obstruction, or in the later stages of "shock.") The bowel is not only shortened, but the increased blood in the dilated blood vessels and the exudate which forms in its wall increases its weight up to some 33 per cent., even when allowance is made for the reduction in length.

The resistance of the bowel wall to bursting is, to all intents and purposes, proportional to the diameter of the lumen, for the variations in the thickness of the gut wall are too small to be a factor of much consequence. Thus the pelvic colon and the ileum withstand increase of intraluminal pressure better than the larger and more proximal segments of the colon and small intestine respectively. This law of hydraulic stress is equally true in acute intestinal obstruction, though here the power to withstand increments of intraluminal pressure is markedly reduced. The actual intraluminal pressures found which are capable of producing pathology vary not only from one animal to another, but also at different times in the same animal, but in both man and animals striation of the peritoneal coat of the intestine means that rupture of whole wall is imminent. The sustained pressures as estimated in man vary from 4 to 19 cm. of water for the ileum and from 12 to 52 cm. of water for the colon, with considerable increase during periods of strong peristalsis.

The degree of distension present varies with the site of obstruction. The upper reaches of the small intestine are usually decom-

pressed by vomiting, a factor which brings but little relief in lower ileal obstructions where great distension often results from partial volvulus of the obstructed loop. The degree of colon distension depends on the efficiency of the ileo-cæcal sphincter, which may allow regurgitation from the cæcum or by non-relaxation produce a " closed loop " obstruction.

The term ileo-cæcal sphincter should be used in preference to ileo-cæcal valve, as both in man and the commonly used experimental animals it appears as a sphincter and acts as a sphincter, and a valve-like appearance only occurs during marked relaxation of the region or some hours after death, when a valve-like appearance with a less well-formed inferior lip becomes evident. The sphincter's function is much the same as that of the pylorus, namely, to regulate the flow of intestinal contents in either direction. It is well known that regurgitation takes place through the ileo-cæcal sphincter when a barium enema is given, but here the pressure used is 90 cm. of barium emulsion (the sustained pressure of acute colonic obstruction is rarely more than 25 cm. water), the quantity is large and it is also given after purgation and fasting, two conditions which are known to relax the sphincter. The author has studied the action of the ileo-cæcal sphincter in acute colonic obstruction in cats.

Three methods were used. In the first two a " Perspex " window was inserted into the abdominal wall and the colon was inflated by a rectal tube or through a colostomy tube brought out through a small stab incision while the anus was occluded. The third method was to use a colostomy and rectal clamp on exteriorised bowel which was kept moist with warm saline. Known quantities of air or fluid could be injected at known pressures in any of these techniques and the effects on the ileo-cæcal sphincter noted.

It was found that on the same cat at different times the injection might pass into the ileum, while at other times even if the pressure was raised to 200 cm. of water the sphincter did not relax. It was further noticed that regurgitation took place only in the resting bowel and that when peristalsis and segmentation were active before the injection started the sphincter remained contracted, even though amounts up to 200 c.c. of fluid were injected at a pressure of 200 cm. of water. In these latter cases the sustained intraluminal pressure on all occasions and in every animal rapidly became balanced between 15 and 20 c.c. of water,

whether fluid was run in or out of the bowel, owing to the postural tone of the actively contracting colon. Also in these cases, as the obstruction continued increased peristalsis was seen in the ileum and reversed peristalsis occurred in the colon, but both tended to fade out near the cæcum. The sphincter only relaxed to allow of the passage of ileal contents (as shown by colouring the ileal contents) into the cæcum, which continued in spite of great distension to receive the chyme. Rectal dilatation sometimes increased and at other times diminished the activity of the obstructed bowel. These observations fit in with the clinical findings that sometimes the small gut is distended in acute colonic obstructions, though whether from ileal regurgitation or ileo-cæcal achalasia is uncertain. Radiographs of my own cases differ markedly from the results given by Wangensteen, for 60 per cent. show ideal distension, a condition he considers rare and attributes to valvular incompetence.

It is easy by using a " Perspex " window to observe that there is increased peristalsis as distension occurs, that the bowel distal to an obstruction functions normally, that reverse peristalsis occurs in the colon, though this is far more difficult to be certain about in the small intestine. As the distension increases the " stretch " reflex of unstriated muscle comes into play and the strength of contraction, though not the rate, is increased. This increase in work leads to fatigue and the typical clinical picture of recurring attacks of severe intestinal colic.

Increased activity also leads to dilatation of the blood vessels, which as distension progresses makes the circulation more sluggish and may lead, both in man and in experimental animals, to thrombosis and intramural strangulation. (See under Strangulation.)

Nervous impulses derived from distended loops of intestine are said to alter the blood flow in that loop and also in some unexplained way to hasten somewhat the death of the animal, which can be delayed by peripheral denervation, but not by removal of the cœliac ganglia.

Microscopic examination of the distended but viable bowel wall shows œdema, shortening, and clubbing of the villi, with dilatation of the blood vessels, but without intramural hæmorrhages.

Effects on Absorption. Normal absorption from the bowel takes place either through the blood vessels or through the lymphatics of the mesentery, but in acute intestinal obstruction

there is evidence that transperitoneal absorption may also occur.

Numerous experiments have shown that the venous absorption of water and the normal products of digestion is retarded by obstruction and that the reduction, which may amount to 80 per cent., is roughly proportional to the degree of distension present, provided the viability of the bowel wall is not impaired. It has also been shown under similar conditions that histamine is not absorbed from the lumen of the bowel in an active form (as if injected direct into the portal vein its usual effects are obtained) and that there is also a delay in the absorption of strychnine. Transfusion of portal blood from dogs with advanced acute intestinal obstruction into the systemic circulation of other dogs has failed to show any toxic or depressor substances present, which suggests that such substances if absorbed must pass into the circulation by another route.

Distension, by raising the venous pressure, has been shown to increase the lymphatic absorption of certain dyes injected into the lumen of the bowel and also of bacteria.

Claims have been put forward that a toxic substance may be found in the thoracic duct in cases of acute intestinal obstruction, but ligature of the thoracic duct or of the lymphatic pedicles in the root of the mesentery does not increase the survival period of experimental animals.

Toxic substances do not make their appearance in the peritoneal cavity until the viability of the gut has been affected, so transperitoneal absorption can only become a pathological factor in strangulation. (See section on Strangulation.)

The Systemic Effects of Distension. Gross abdominal distension obviously interferes with normal intrathoracic physiology by embarrassing both cardio-vascular and respiratory function, but lesser degrees of distension may produce results which are only just beginning to be appreciated.

The rise of intraperitoneal pressure, though it lags behind that of the intra-enteric pressures, is sufficient to retard the venous return and so prolong the circulation time from the legs by compressing the veins in the iliac fossæ. The inferior vena cava is not affected owing to its thick fibrous sheath.

The portal circulation is also affected, but in the reverse direction, as there is a lowering of the venous pressure by 50 per cent. or even more from the normal 12 cm. of saline pressure. This is due to the lessened venous return and consequently is more

marked in strangulating obstruction. This reduction of portal venous pressure is said to lead to hyperglycæmia and so to fatty infiltration from glycogen lack, with presumably reduction in all normal liver functions.

The stasis and resulting anoxia will, in both the systemic and portal systems, lead to damage of the capillary walls, fluid loss, and reduction of the circulating blood plasma.

The Effects of Strangulation

It has already been pointed out that distension may lead to intramural strangulation, a condition most often seen in the cæcum in advanced acute obstruction of the pelvic colon in those cases where the ileo-cæcal sphincter prevents regurgitation and so allows gross cæcal distension to occur. Sudden hyperacute distension of the bowel may produce the same effect, for the author has seen intramural cæcal strangulation in blast injury when there was no macroscopic evidence of any lesion in the body wall, the other viscera, or the mesenteric vessels.

However, the more usual causes of strangulating obstructions are those lesions which affect both the intestinal continuity and its blood supply from the onset.

Types of Strangulation. The most common mechanism is one which obstructs the lumen of the bowel at two places and produces venous obstruction with a varying degree of arterial occlusion, as in the majority of strangulated herniæ. This mechanism, if mild, will produce only venous obstruction, while, if severe, complete arterial occlusion will also take place. Arterial occlusion alone probably only occurs in cardio-vascular disease.

Effects on the Bowel Wall. These effects have been studied by direct observation after the insertion of a " Perspex " window and confirm Scott's (1938) observations.

Strangulation by tying a tape round a loop of bowel soon produces a deep purple discolouration. The degree of distension of the loop depends on the type and the degree of the vascular obstruction, the distension being greatest when complete venous obstruction is associated with free arterial supply and least with complete occlusion of both the arteries and the veins. The formation of exudate before gross gangrene occurs runs parallel to the distension and is not marked in arterial occlusion, though the bowel in arterial occlusion is rapidly covered with a fibrinous exudate which causes it to adhere closely to the parietal peri-

toneum. The degree of hæmorrhage and exudate into the bowel wall is directly proportional to the degree of venous obstruction, but in arterial occlusion the wall may be thinner than normal and may show anæmic infarction when vascular obstruction is complete.

Effects on the Motor Function of the Bowel. The results of strangulation on the affected loop have already been described, but its effects on the motor function of the bowel as a whole remain to be considered.

Mild degree of venous obstruction produce effects differing little from those already described under the section upon Distension, but severe venous obstruction, especially if associated with arterial occlusion, produces marked alteration in the motor function of the rest of the small intestine. In these primary strangulations there is immediate spasm of the strangulated loop and also of the gut above and below it, which frequently affects the whole of the small intestine and even the pylorus, though the effect on the stomach is variable. After a time, which varies from a few minutes to several hours, the spasm lessens and normal intestinal movements commence, first in the areas away from the lesion, but in spite of prolonged vigorous peristalsis, distenstion above the obstruction does not take place until retrograde venous thrombosis has occurred or peritonitis is present.

In acute primary strangulation of the small intestine retrograde venous thrombosis is the primary cause of distension in the bowel, either proximal or distal to an acutely strangulated loop of small intestine and if the process extends more on the distal side then dilatation of the bowel is found more marked distal to the obstruction. The bowel contains fluid and little, if any, gas and this, together with the lack of distension until a terminal phase is reached, accounts for the lack of radiographic evidence of the presence of a serious lesion on plain films in primary strangulations (Chesterman, 1944).

Strangulation of the large intestine does not appear to affect the general motor function of the small intestine to any extent until peritonitis is present.

Effects upon Absorption. Strangulation lessens the normal venous absorption in proportion to the degree of vascular obstruction present, complete arterial or venous occlusion stops venous absorption. The absorption of toxic substances such as strych-

nine and tetanous toxin are also reduced in the same way, but no conclusive evidence of the venous absorption of shock producing substances during acute strangulation has as yet been brought forward.

Lymphatic absorption is influenced by strangulation in much the same way as by distension but section of the thoracic duct or external drainage of its contents do not affect the length of survival of the experimental animal with strangulation.

Transperitoneal absorption, as has already been pointed out, does not affect the course of events in a non-strangulating obstruction and there is little evidence of any absorption of toxic products in strangulation until the viability of the bowel wall is impaired. Directly the bowel wall is no longer viable, organisms and toxic substances such as botulinum toxin readily pass into the peritoneal cavity, giving rise to peritonitis and the absorption of toxic substances and the passage of organisms into the circulation. Intestinal perforation from necrosis of the bowel wall of course rapidly increases the spread of peritonitis with all its consequences.

Systemic Effects of Strangulation. The early systemic effects of strangulation are mainly those of fluid loss. Hyperglycæmia also occurs early but its exact cause is as yet unknown.

Later the systemic effects of peritonitis are added.

Summary

The two principal lethal factors induced by acute intestinal obstruction, as far as can be at present ascertained, are related to Fluid Loss (whether by vomiting, vascular engorgement, œdema and hæmorrhages into the bowel wall, the formation of transudates and exudates into the lumen of the bowel or the peritoneal cavity, and due to the stasis induced in the systemic circulation, particularly in the legs) and the Viability of the Bowel wall.

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PATHOLOGY INDUCED BY THE RELEASE OF ACUTE INTESTINAL OBSTRUCTION

PATHOLOGICAL processes induced by the release of acute intestinal obstruction have never been adequately studied, and yet it is a well-recognised experience that fluid loss may be corrected, the anæsthesia may be satisfactory, the right operative procedure may be carried out with meticulous care and yet the patient may die in the immediate post-operative period from no very well-defined cause. This has led to a somewhat fatalistic attitude which the author is convinced is one of the reasons for the present mortality rate and it is with a better appreciation of the Vascular, Nervous and Muscular effects of the release of acute intestinal obstruction that improvement in these figures may to some extent be expected. These vascular, nervous and muscular changes appear to be inseparably linked together, but for descriptive purposes they may be taken separately, though their interdependence must be recognised throughout.

Vascular Changes. The release of strangulation produces various effects according to the degree of thrombosis present in the vessels of the affected segment.

Marked arterial or venous thrombosis, separately or combined, produce gangrene and demand immediate surgical intervention. However, it should be realised that after the release of venous obstruction, the blood flowing through the damaged and dilated veins tends to lead to thrombosis or an extension of the thrombosis already present during the process of strangulation and hence resection must be made well wide of the affected area to prevent the anastomosis being involved in the spreading and often retrograde thrombosis. Venous thrombosis occurs more readily in the experimental animal if the bowel is allowed to become dry or is overheated by too warm towels. This type of thrombosis may also occur in small and patchy areas at the mesenteric insertion of the bowel or on its anti-mesenteric border following the return of apparently viable intestine to the abdomen with resulting peritonitis. The author has seen this occur twice in patients and has also observed it in cats.

A far more frequent and just as serious a problem is presented in those cases of venous obstruction without obvious thrombosis or gangrene by the still further loss of fluid into the affected area after release of the strangulating mechanism.

Experiments on cats have confirmed clinical impressions that when certain lengths of bowel are involved release of the obstruction will cause death of the animal quicker than by leaving the strangulation to take its natural course. It has long been known that venous strangulation of a long segment of intestine leads to death from fluid loss before peritonitis kills the animal, but it has not been stressed that with segments equivalent to 2 or 4 ft. in man death may occur rapidly from release of the strangulation. This form of death was noticed to occur very frequently when venous strangulation was produced on 12 to 15 in. of cat's small intestine and released before viability was impaired. The type of anæsthetic (open ether or intraperitoneal nembutal) did not affect the lethal tissue and death took place after release of the obstruction considerably faster (hours) than if the strangulation was left to take its normal course. This suggests two possibilities, excessive fluid loss into the bowel or the release into the circulation of some toxic factor produced during strangulation and liberated only when an adequate blood supply is re-established. These cats can be saved by early resection of the area and sometimes by massive intravenous infusions of normal saline provided it is given immediately after the release of the strangulation.

The possibility that death in these animals is due to the absorption of a toxic factor has also been explored. No important lethal or shock-producing factor has been shown to be absorbed during acute intestinal obstruction unless the viability of the gut has been impaired, but the possibility of the absorption of some such factor from an area of gut released from strangulation has not as yet been adequately studied. Green (1943) and Green and Bielschowsky (1943) have shown the possibility of obtaining from striated muscle a substance capable of producing all the effects which follow the release of limb ischæmia (Allen, 1939) and which they call a Muscle Shock Factor (M.S.F.) as the results of its injection resemble those of traumatic shock in man and animals. This substance (M.S.F.) probably owes all or much of its effects to the presence of adenosine triphosphate. This substance is a normal constituent of muscle and probably all tissue cells and Green (1943) postulates that it may become extracellular as the result of tissue anoxia. It is very readily broken down after death, but in the living animal quantities may be carried into the circulation with the production of " shock " and even death.

Strangulation or prolonged distension, which is accompanied by marked slowing of the venous return produces a variable degree of anoxia of the bowel wall and so gives rise to the same conditions in unstriated muscle as limb ischæmia produces in striated muscle and hence might lead to the release of M.S.F. which is carried into the circulation with serious effects on release of the obstruction, provided adequate vascular channels remain intact.

Experiments carried out on these lines were inconclusive, but they did not negative Green's idea, and the possibility of toxic absorption after the release of obstruction cannot be ruled out, but it must be recognised that fluid loss appears to be sufficient to account for the death of these animals in the conditions under discussion for the elongation of the intestine (*vidi infra*) after the release of the obstruction allows of a greater fluid loss than can occur during the strangulation.

In any case, the implications are interesting and important for it means that resection may have to be resorted to even when the gut is viable, though at present such cases should be given 1,000 c.c. of normal saline intravenously quickly and equal amount by drip transfusion. Also it negatives the practice of exteriorisation of large segments of intestine whose viability is doubtful or whose non-viability is certain.

Boyce (1941) has pointed out that certain deaths occurring after the operative relief of acute intestinal obstruction occur with hyperpyrexia, which, if not fatal within a few hours, pass on to death in uræmia. He postulates that this " Hepatorenal Syndrome " is due to an underlying liver damage or dysfunction, so that under the added strain of surgery the hepatic cells fail and release into the circulation toxic substances which produce hyperpyrexia and damage to the convoluted tubules of the kidney, which latter causes death if the hyperpyrexia is not fatal. Hyperpyrexia is a well-recognised cause of death after operation for intussusception in infants, though the author has never seen it. In fact, the only case which might be considered as belonging to this syndrome under my care was a man with acute obstruction by a carcinoma at the pelvi-rectal junction who died with hyperpyrexia three days after a colostomy had been performed. Autopsy showed no cause for the death of the patient and microscopic examination of the liver and kidneys did not present the findings described by Boyce or, for that matter, any others of significance.