A Lecture

ON

ILEUS: ACTIVE AND PARALYTIC

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BY

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"ILEUS" was thus described by Barnard¹: "an ancient term incapable of precise definition, and therefore should not be used." In our everyday practice, however, this title outlines to all of us the picture of a motionless distension of the intestine in the absence of any obstructive lesion.

There are two main causes of intestinal distension:

1. *Obstruction* to the passage of contents is associated with overaction and hypertrophy.² Clinically peristalsis is seen and felt.
2. *Inertia* of the intestinal muscle from (a) active inhibition, and (b) interference with the circulation; either ultimately leading to distension.

Intrinsically the distinction is so sharp, both clinically and pathologically, that I submit the following definition of "ileus": "Distension from intestinal inertia."

Four factors are concerned: (a) an organ of independent functioning power (even when removed from the body),³ co-ordinated and controlled for the needs of the body by (b) *sympathetic nerves*—inhibitory to bowel movements and vaso-constrictor to the arteries; (c) *parasympathetic nerves*, whose stimulation excites intestinal motility; and (d) the blood supply of the intestine. The co-ordination of these constitutes a physiological balance.

It is generally recognized that ordinary simultaneous stimulation (whether experimental or pathological) of these antagonistic nerves always leads to a domination of the sympathetic or inhibitory group,⁴ and a motionless intestine results.

Now in the etiology of ileus (or "inertia distension") at least four primary types demand consideration: (a) the effect of bacterial toxins on the bowel wall; (b) mechanical obstruction; (c) "active ileus"; (d) "paralytic ileus."

The Effect of Bacterial Toxins on the Bowel Wall (Nothnagel).⁵

The observation of active intestinal movements after death from "ileus" (Nothnagel) would appear to exclude this as a cause.

Mechanical Obstruction.

As an example, in carcinoma of the colon, the stage of overaction and hypertrophy signifies an effort to drive the

FIG. 1.—a-b, a'-b' represent the distension of the intestinal lumen; A-A' and A'-A' represent the increase of transverse (longitudinal) mesenteric tension; B, fixed point of mesentery; B', point to which mesentery would be dragged if it were not fixed; B-A and B'-A', being equal, B-A' and B-B' show vertical mesenteric tension.

"inert area" of the intestine down the intestinal tract. The ultimate compression of the congested and swollen mucosa into the narrowed lumen completes the picture of

final obstruction. The gradual transition from the "active" stage to "intestinal inertia," with the typical cachexia, is attributable to two factors: (1) compression of capillaries and veins in the intestinal wall. This leads to increasing failure of intestinal muscle. (2) Tension on the mesentery in two directions (Fig. 1). Mesenteric tension was recognized by Nothnagel⁷ and Barnard,⁸ and I have demonstrated it experimentally.

Here I must remind you that increase of pressure in a bent tube tends to straighten it; and that, owing to the increased length of the intestine over that of its mesenteric attachment, the same law holds good for the gastro-intestinal tract.

Increased mesenteric tension has two results:

(a) Mechanical stimulation of the contained sympathetic fibres, and the resulting inhibition of intestinal movement.⁹

(b) Tension on, and obliteration of, the most collapsible structures—that is, mesenteric veins.¹⁰ This leads to venous stasis and paralysis of intestinal muscle.

These last two factors can each, separately or conjointly, institute intestinal inertia or "ileus" by a different mechanism. Accordingly there are two varieties to be recognized.

"Active Ileus."

This is intestinal inertia excited by sympathetic inhibitory impulses, and it is conveniently illustrated by the example of acute inflammation of the appendix adjacent to the ileal mesentery ("five o'clock appendix").

FIG. 2.—Diagram of the area of active inhibitory nerve impulses (afferent and efferent) in acute appendicitis. A, appendix; C, caecum; I, ileum; AC, ascending colon; S, sympathetic nerve supply to the inflamed area. Dotted line=extent of direct intestinal inhibition.

In sharp contradistinction to the retrocaecal or pelvic appendix, it is in this situation (Fig. 2) that acute appendicitis is associated with the classical symptoms from the onset, by reason of the mesenteric reflexes it evokes. Such a lesion stimulates in the immediate neighbourhood (Fig. 2):

(a) The efferent sympathetic. Intestinal movement is inhibited in the dotted area.

(b) The afferent sympathetic. The efferent response evokes: (i) proportionate immobility of the whole intestinal tract; and (ii) proportionate closure of the pylorus against the passage of food.

(c) The afferent vagus. The resulting contraction of the proximal stomach against a closed pylorus empties this viscus of food.

The whole picture of the empty stomach and motionless intestine is that of a protective mechanism. It is to be regarded as a physiological response to injury or infection; as a mechanism designed to immobilize and rest the inflamed part; to prevent the ingestion of food; and to localize the inflammatory process.

One further protective mechanism is evident. The presence of Paccinian (or touch) corpuscles in the mesentery—long recognized—was emphasized years ago by Keith.¹¹ He described them as "end organs for receiving pressure." Their stimulation excites the reflex immobility of the abdominal wall—again a protective mechanism. The whole protective picture is proportionate to the extent of the exciting cause—clearly the resulting distension may reach a sufficient degree to interfere with the venous return, and so to initiate the onset of "paralytic ileus."

"Paralytic Ileus"—Intestinal Inertia of Circulatory Origin.

A classical example is that of "acute intestinal strangulation," in which the importance of the circulatory factor was recognized by Barnard.¹² This is a clinical analogy to the experimental ligation of the intestinal veins with the arteries intact. The result is:

(i) Severe stimulation of the Paccinian corpuscles, leading to shock,¹³ vomiting, and initial immobility of the abdominal wall.

(ii) As soon as the pressure in the obstructed veins exceeds that in the intestinal lumen, gases pass from the blood into the viscus by osmotic pressure, and progressive inertia and distension begin.

(iii) When in due course the pressure in the bowel exceeds that in the veins, gases will pass by osmosis back into the blood stream; and these, exhaled by the lungs, will give a faecal odour to the breath—a most significant diagnostic symptom. The progressive extension of mesenteric tension so induced leads to a proportionate spread of venous engorgement, and thus:

(iv) Fluid from the veins passes, not only into the peritoneum, but into the intestinal lumen. In due course absorption of fluids and gases from the bowel accentuate the initial shock with a profound toxæmia. Such a process, if unrelieved, is essentially progressive; so that, when the gastric musculature is involved and failure of the pyloric sphincter occurs, the intestinal contents obtain access to the stomach, and the result is stercoral vomiting. The condition is now analogous to that seen in "acute dilatation of the stomach."

(v) *The ileo-colic sphincter.* Normally the large bowel is adapted to tolerate a degree of intermittent obstruction, created by the voluntary control of the anus. From this the small intestine (which is not so adapted) is protected by the ileo-colic sphincter. This Elliott¹⁴ showed to contract as the result of sympathetic stimulation, anaemia, and adrenaline. (That this function of the sphincter is of importance is shown by the changes in the ileum following colectomy—changes which are hardly noticeable after a year, when the same operation is performed by implanting the ileo-colic sphincter into the sigmoid and so preserving its function.) Whereas, then, the ileo-colic sphincter is closed in "active ileus" (from sympathetic stimulation), it will, in common with the rest of the intestine, be paralysed by venous engorgement. The free access of the infective contents of the colon to the small bowel may be one of the reasons for the highly increased "infectivity" of the contents when intestinal paralysis follows venous obstruction.

Clearly severe toxæmia and faecal odour of the breath are not features of "active or protective ileus," but are evidence of venous stagnation and "paralytic ileus." In minor degrees such ileo-colic incompetence may play a part in chronic disease. Further, if this picture is correct, the absence of toxæmia in obstruction (when the venous return is unimpaired) is in sharp contrast to the rapid toxæmia of strangulation, due to the obstructed venous return.

The clinical lesson stresses the significance of the breath as an indication of intestinal derangement associated with a defective venous return.

(vi) *The abdominal wall.* Increase of intra-abdominal pressure, by further compression of the larger veins, accentuates venous stasis. Accordingly "paralytic ileus" tends to occur sooner in subjects who are stout, or have firm abdominal walls, than in those with flabby muscles. For instance, it is remarkable to see the degree of distension which sometimes follows Caesarean section without marked distress or constitutional symptoms.

PRACTICAL ISSUES.

The clear-cut distinction between these two forms of ileus—active and paralytic—should form the foundation of our clinical practice. Accordingly we recognize two main groups of primary exciting causes of intestinal inertia and subsequent distension: (a) extra-abdominal; (b) intra-abdominal.

A.—Extra-abdominal.

A brief reference only to some of the more important conditions which may confuse or aggravate the clinical picture is possible.

1. *The Psychic Factor.*—Excessive or prolonged sympathetic stimulation of psychic origin inhibits both secretory and motor functions of the gastro-intestinal tract. In acute forms cessation of intestinal movement was recognized by Cannon,¹⁵ Leonard Hill,¹⁶ Langdon Brown,¹⁷ and

many others. In milder form such derangements may result from excessive mental activity; anxiety and apprehension; fear; unhappiness, etc. If long-continued, the gastro-intestinal sequelae may become permanent, and so accurate diagnosis of the primary disease may be exceedingly difficult.

2. *The Pulmonary Factor.*—Abdominal distension associated with pneumonia and empyema is a common experience, especially as a terminal feature. Mannaberg¹⁸ believed it to be due to peritonitis (that is, infective agencies), but it is probably circulatory in origin.

3. *Cardiac.*—Heart failure has long been recognized as a cause of venous stasis in the viscera (Nothnagel, loc. cit.) and consequent distension.

In both the latter groups the absence of that characteristic odour of the breath constitutes an important clinical distinction.

4. *The Renal Factor.*—This factor (in renal colic, for example) induces an "active" or inhibitory ileus by sympathetic stimulation. Again the odour of the breath in addition to the history, etc., is a useful guide unless the circulatory type—"paralytic ileus"—supervenes.

A striking example came under my notice last summer. I operated on a man of 51 years for pain and repeated vomiting, absolute constipation associated with an enormous degree of abdominal distension, and visible coils of gut. No gross lesion of the alimentary tract could be found, but he had a haemorrhage into a left hypernephroma, which I subsequently removed, when the distension subsided as the result of a temporary caecostomy.

B.—Intra-abdominal.

Post-operative Ileus ("Paralytic Ileus").—I have long believed (and invariably acted on the belief) that if we would aid (and, if necessary, supplement) Nature's methods of immobilization and rest of the gastro-intestinal tract after laparotomy, this dreaded sequel would be excessively rare. The mechanism is the same for trauma as for infection, both of which, as we have already seen, primarily evoke local and general immobility by direct and indirect sympathetic stimulation. This, as already stated, results from injury to the mesentery and also to the parietal peritoneum. The stimulation of the contained Paccinian corpuscles further immobilizes reflexly the abdominal wall.

This picture of a protective mechanism is designed to ensure rest, and should be the basis of our treatment, characterized largely by masterly inactivity. "Active" (physiological) ileus is to be promoted by various measures.

Starvation.—Common sense indicates a middle course between absolute starvation of fluids and plethora. The former induces restlessness and misery from thirst, and violates the principles of rest, both bodily and psychic. The latter may induce vomiting and flatulence. A medicine glass of fruit juice stood at the bedside and sipped at will, or chewing fruit (for example, pineapple, grapes, etc.), will suffice to check thirst, clean the mouth, and supply carbohydrates.

Rectal salines (less irritating and more easily retained than water) with 5 per cent. glucose, at approximately four-hourly intervals, are less disturbing than continuous proctolysis.

The Fowler position is adopted for two reasons: (a) relaxation of the abdominal muscles adds to comfort; (b) gravitation of peritoneal fluid to known regions (Box¹⁹) and the more innocuous sites.

Sedatives.—Local and general rest are promoted by sleep. Blood pressure tracings during the exhibition of emotions (anxiety, apprehension, fear, etc.) so closely approximate those seen in the initial stages of shock that, for these reasons, for personal comfort, and for humanitarian purposes, the liberal administration of morphine (or its allies) is an essential routine.

I have in mind a medical man (intent on analysing his symptoms) who developed, after operation for obstruction, an alarming degree of distension of from four to five days' duration, relieved only by liberal administration of morphine and radiant heat to the abdomen.

Purgation and Feeding.—Diverse practice and teaching on this point are evidence that current views are based on empirical rather than scientific grounds. There is no

general rule. The subsidence of an inflammatory or traumatic lesion is evidenced by a return of intestinal movement. Clinically this is ascertained by the patient; the use of the stethoscope; and the spontaneous passage of flatus. These constitute a demand (whenever they occur) either for temporary assistance by pituitrin injection and turpentine washouts, or for initiating a normal routine with a purgative followed by a gradual return to normal diet—each case being considered on its merits. The former is a temporary measure which can always be employed to prevent or relieve a distension which may threaten to become, in itself, a source of distress. The momentary effect is a negligible violation of the principle of rest. Purgation, if adopted before there is evidence of a return of intestinal activity, only causes hyperaemia, outpouring of fluids and gas into the intestines, and distension. Finally it may actually precipitate the onset of "paralytic ileus." Accordingly adherence to principle will forbid the administration of a purgative until there is evidence of the return of intestinal activity.

"Paralytic Ileus."

It is evident that distension arising from physiological ileus may, in any case (often as a result of mismanagement), reach a degree sufficient to compress the veins, and initiate paralytic ileus—in any instance this may be accentuated by cardiac failure, pulmonary complications, etc., as already stated. The odour of the breath, the state of the tongue, and the constitutional changes are all evidence of the onset of venous obstruction and the interchange of gases referred to. Measures for prevention are, in part, measures for cure. Accordingly we should add the following to the routine outlined.

Radiant heat to the abdomen—recommended to me many years ago by Sir George Makins—has formed so integral a part of my routine that my own nursing sister applies it to every case until it can evidently be dispensed with. To its routine employment I attribute a relative immunity from serious "paralytic ileus." Its action is to reduce deep congestion by causing superficial hyperaemia.

Cardiac insufficiency and pulmonary complications—both of which have been discussed as potentially initiating or accentuating venous obstruction—must be watched and controlled. The injection of strychnine 1/30 grain, atropine 1/100 grain, adrenaline (1 in 1,000) 10 minims (sometimes called the A.S.A. (or Trinity) hypodermic of Box), given six-hourly for four doses (and repeated in half doses if necessary), is an excellent precautionary measure. With its employment the pulse rate and thirst must be watched and controlled.

Progressive "Paralytic Ileus."

Nothnagel's²⁰ observations would appear to contradict the theory of Reichel,²¹ that ileus is due to bacterial invasion of the bowel wall. Accordingly clinical methods, directed towards the restoration of intestinal motility, should be concentrated upon the relief of distension and intra-abdominal pressure. Interval injections of pituitrin followed by turpentine or human bile washouts promote a diminished intra-abdominal pressure, and so a restoration of intestinal circulation.

Gastric lavage further reduces abdominal distension and favours the return to a normal circulation. This measure (which appears never to be harmful) is urgently indicated when vomiting has commenced.

Avoidance of purgation.—It has been pointed out that intestinal inertia is only accentuated by purgation—morphine (or an allied sedative) is indicated. I have in mind an instance of an over-anxious doctor administering 1 grain doses of calomel hourly to a lady during the stage of post-operative protective ileus. The resulting picture of acute obstruction was relieved by morphine and radiant heat to the abdomen.

I must emphasize the rule that the only sure indication for purgation and feeding is evidence of the return of normal intestinal activity.

In the rare instances when this routine is ineffective a light general anaesthetic should precede a spinal anaes-

thetic. This may result in a copious evacuation, from the removal of inhibitory reflexes. If this occurs after the abdomen is open (and mechanical obstruction is excluded) a catheter passed per rectum as high as possible is sufficient. Otherwise a temporary ileostomy or jejunostomy (by Witzel's method) is indicated. The initial discharge of faeces from this procedure is usually followed by an absence of drainage for twenty-four to thirty-six hours—a feature which often causes unnecessary anxiety to those in charge.

The explanation is that the relief of pressure (and the consequent return of normal circulation) spreads consecutively from loop to loop of intestine, and is associated with a gradual spreading return of intestinal activity. This is a relatively slow process. Initially purgation only increases ileus; morphine and radiant heat are indicated to ensure rest, diminish secretion, and relieve congestion. To mention a striking example:

A woman, aged 40, had a gangrenous appendix removed and the pelvis drained. Acute obstruction supervened on the tenth day. A second operation released the volvulus round the tube sinus, and temporary ileostomy was followed by a copious evacuation. Subsequently there was little drainage for forty-eight hours. It was ascertained that pituitrin injections, enemata, and purgation had been persisted in during this period, and were only followed by increasing distension. This yielded immediately to radiant heat and the liberal administration of morphine, which gave sleep and led to natural actions of the bowels and the subsequent automatic closure of the fistula.

Intermittent and Chronic Ileus.

So far visceromotor reflexes (in their physiological or protective capacity), and circulatory (pathological) defects arising from trauma or infection, have been considered only in acute form. It is not difficult to envisage analogous symptoms arising from milder or more chronic lesions of similar character.

1. Inhibitory Impulses in Chronic Disease.

If the symptoms of acute inflammation of the "five o'clock" appendix be again referred to, and be modified to those of chronic disease arising either from inflammatory or mechanical agencies, it is not difficult to substitute: (a) flatulence and anorexia for vomiting; (b) constipation requiring laxatives for general intestinal inhibition; and (c) discomfort and referred pain from acute epigastric pain becoming localized to the inflammatory area.

In chronic or intermittent form such symptoms are only a mild but faithful mimicry of the acute crisis. As an analogy the flatulence, distension, and constipation associated with gall-bladder disease are referable to the same type of reflex; examples might be multiplied.

2. The Circulatory Factor in Chronic Disease.

It is important to realize that—the portal system being devoid of valves—the vasomotor tone, the support of the abdominal muscles, and the negative pressure of the thoracic inspiration are all factors of importance. This was realized by Leonard Hill and Barnard²² when, discussing a flabby abdominal wall, they referred to "the abdominal vessels . . . similar in nature to an easily distensible bag."

Keith²³ realized the importance of the abdominal muscles in effective expansion of the lungs when, twenty-seven years ago, he described the diaphragm as an "abdominal piston" in the "abdominal type of respiration," in contradistinction to the normal "thoracic type" with efficient abdominal muscles.

Abdominal venous stasis, associated with the resultant intestinal inefficiency, is gradually initiated by: (a) pooling in the abdominal veins—the result of imperfect tone of the abdominal wall; and (b) insufficient negative thoracic pressure—due to an inefficient expansion of the lower lobes of the lungs (an important factor in pulmonary complications following "upper abdominal" operations). In chronic disease remedial breathing instruction has been beneficially emphasized by MacMahon.²⁴

3. Inhibitory and Circulatory Factors Combined.

This is most conveniently illustrated by the congenital mobile colon, but, in preface, it is only fair to insist that the actual position of the colon means nothing whatever. What is really important is its means of support in the upright position.²⁵ The mesentery is *not* (and never was designed as) a supporting structure. As a result of this pathological imposition there are two results:

(a) Sympathetic inhibitory stimuli (with consequent general stasis, flatulence, etc.)—a mild mimicry of the mechanism and symptoms already portrayed in the picture of acute appendicitis (Fig. 2).

(b) Again the most collapsible structures suffer first. The resulting tension on the mesenteric veins leads to venous stasis (evidenced clinically by the tortuous arteries so commonly seen during operations on these subjects), and accentuates the

FIG. 3.—Showing tension on mesenteric vessels and concomitant sympathetic fibres in colopostosis.

existing intestinal inefficiency due to inhibition (Fig. 3). In subjects of visceroptosis these two factors combine to produce the symptoms which are in the experience of us all.

As an example, we are all familiar with the patient of sturdy athletic build, with well-fixed colon, who suffers no symptoms from (or clinical evidence of) constipation, even if this extends over several days. A similar constipation in the subject of visceroptosis leads to fœtor of the breath, discomfort and distension, flatulence, anorexia, etc. Is not this comparison again a mild mimicry of the picture we have already considered when we contrasted the absence of constitutional symptoms associated with chronic obstruction with the profound changes seen in strangulation? Can it be doubted that the venous factor is equally responsible for the symptoms in chronic mesenteric tension (visceroptosis) as it is in acute strangulation?¹⁰

Again, the end-result of venous stasis and the temporary intestinal inefficiency which results, ultimately is fibrosis. Fibrosis not only involves the muscle, but, far more serious, may (and in long-standing cases undoubtedly does) involve Auerbach's plexus, thereby dislocating the whole neuromuscular mechanism of the intestine.⁴

Surely it is our duty to anticipate this eventual and incurable sequel of venous stasis by imitating Nature's normal method of mesenteric fusion, and performing the simple operation of colopexy, in suitable cases. The success of such a procedure, carried out on sound principles, depends on the selection of cases for operation; and, from what has been said, this is often no simple problem.

In conclusion, it is submitted that the term "ileus" should present to us a picture of intestinal inertia of two types: (1) active ileus—a physiological and defensive mechanism due to inhibitory nervous impulses; and (2) paralytic ileus—a pathological and progressive condition due to venous obstruction.

In chronic form the symptoms are a mild mimicry of those we are familiar with in acute abdominal crises; but the principles on which treatment should be based are identical—namely: "active (physiological) ileus" should always be imitated and supplemented; while every effort should be directed towards the prevention of increased venous pressure (from any source) and the possible onset of "paralytic ileus."

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URGENT UTERINE HAEMORRHAGE OF CONSTITUTIONAL ORIGIN.

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LOCAL or pelvic causes account for by far the majority of cases of abnormal uterine haemorrhage, which, according to Beckwith Whitehouse, may be classified into four groups: epimenorrhoea (menstrual periods too frequent), menorrhagia (periods profuse), menostaxis (too prolonged), and metrorrhagia (irregular bleeding). Minor degrees of any of these conditions are occasionally met with associated with constitutional disturbances or disorders of metabolism, such as myxoedema, exophthalmic goitre, acute infectious diseases, and the anaemias. Shaw,¹ in his very detailed work on irregular uterine haemorrhage, has noted that in 17.5 per cent. of his series of 200 cases of excessive uterine haemorrhage no explanation for its etiology could be offered. He states that it is always extremely difficult to attribute menstrual abnormalities to general metabolic and endocrinal disturbance, for in the majority of cases there is no obvious association, and it is undoubtedly true that, in the past, any association that there may be has been grossly exaggerated. He admits that it is possible that in many of the 200 cases investigated some such conditions have been present. Schmitz,² in a survey of 2,184 consecutive hospital records and 2,126 records of out-patients, found that about 36 per cent. of them complained of uterine haemorrhage. No diagnosis as to the cause of the bleeding was made in 22 cases of the in-patient series and in 23 cases of the out-patient series. Linton, Marks, and Smith³ remark that possible factors in the etiology of abnormal flowing without demonstrable pathological conditions are, among others: local or systemic endocrine dysfunction, subclinical constitutional blood disease, and systemic blood disease. According to Munro Kerr,⁴ the common blood diseases, such as anaemia and chlorosis, are associated with amenorrhoea; far more grave disorders, such as scurvy, purpura, etc., are generally accompanied by menorrhagia. Gow is quoted as having shown, in a study of mitral stenosis, that in about 20 per cent. of cases only was menorrhagia present, despite the hitherto general belief that the two conditions were associated. Munro Kerr states that haemophilia is a possible cause of uterine haemorrhage, but Osler⁵ quotes Bulloch and Fildes's claim to have established the fact of immunity in females and their denial of the authenticity of all the published cases. In Grandidier's series of 334 cases of haemophilia uterine bleeding was not instanced. Meigs⁶ has described a few interesting and unusual cases of benign uterine bleeding, excluding the usual causes, such as fibroids, inflammation, polypi, etc. They fell under the following headings: hyper-