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ABDOMINAL PAIN AS EXEMPLIFIED IN ACUTE APPENDICITIS:

A CLINICAL AND BIOLOGICAL CONSIDERATION.*

BY

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THE EVOLUTION OF OUR KNOWLEDGE OF ABDOMINAL PAIN.†

ALMOST the first considerable contribution to our knowledge of the mechanism of abdominal pain was made by J. Ross of Manchester¹ in 1887. Ross held that there were two kinds of pain in visceral disease—true splanchnic pain, felt in the affected organ; and associated somatic pain, referred to the cerebro-spinal nerves of the body wall. Lennander² established the insensibility of the exposed gastro-intestinal tract to the ordinary painful stimuli, and attributed all abdominal pain to stimulation of nerves in the sensitive parietal peritoneum. Mackenzie, whose views form the orthodox teaching of the current textbooks, accepted Ross's views on somatic pain, but rejected splanchnic pain as non-existent. He believed that painful stimuli passed from the viscera through the afferent splanchnic nerves to the spinal cord, but were only appreciated by the brain as arising from the sensory nerves of the parietes. Mackenzie's theories of a viscerosensory and visceromotor reflex have been widely accepted, and will be discussed later. Hurst³ in 1911 published observations which have restored our belief in true splanchnic pain; he proved that this type of pain is produced by a single adequate stimulus—namely, increased tension in the muscular wall of the viscus concerned. Yet Hurst does not reject Mackenzie's views on the viscerosensory and visceromotor reflex mechanism.

My purpose in this paper is to discuss the light which a study of acute appendicitis throws on the mechanism of abdominal pain; and since Mackenzie's theories were based very largely on his observations in cases of appendicitis, this disease may reasonably be used to test those theories.

THE TWO PAINS IN APPENDICITIS.

The conventional teaching on the pain in acute appendicitis is that the attack is ushered in by pain referred to the region of the umbilicus or a little above it, and that after a few hours the pain becomes localized in the right iliac fossa or moves down into that region.

The Initial or Central Pain.

The first point that I wish to emphasize is that the initial pain is entirely different in character and in its mode of origin from the pain which appears in the right iliac fossa a few hours later. The initial pain is felt in the centre of the abdomen, and though the patient may refer it to the mid-line at or above the umbilicus, he often describes it as "all across," sweeping his hand evenly across the central region of the abdomen. It is, in short,

very imperfectly localized. The pain is often described by the patient as "like an ordinary bellyache, but more severe." It is frequently gripping in character and varies in intensity; the more severe spasms usually occur at more or less regular intervals, and last for a few seconds, during which the patient moans and writhes restlessly in bed. This early pain is entirely unassociated with any tenderness on palpation, and the patient may rub or press on his abdomen in a vain attempt to secure relief—a thing that he never does when the second pain has appeared.

This initial pain in appendicitis is, I believe, a true splanchnic pain, and, like all true splanchnic pain, is due to increased tension on the muscular wall of the viscus concerned. Some degree of obstruction to the lumen of the appendix, causing retention of inflammatory exudate distal to the obstruction, is an essential factor in its production. Sometimes—in fact, it is almost the rule in the most fulminating cases—we find a hard, laminated faecal concretion engaged in the relatively narrow base of the appendix, while distal to it a collection of foul pus under great tension rams the concretion home into the base. In this condition, so well described by Wilkie⁴ as acute appendicular obstruction, the central pain in the umbilical region continues until general gangrene or localized perforation of the appendix relieves the tension within it and the pain disappears. In other cases a stricture at the base, the result of some former ulceration, gives rise to the same sequence of events. In others a kinking at the base combines with inflammatory swelling of the mucosa to occlude the outlet into the caecum. In yet rarer instances a foreign body is the occluding agent. A critical examination of the specimens removed in a large number of cases of early appendicitis has convinced me that wherever this splanchnic pain is present some degree of obstruction to the lumen, with retention of inflammatory exudate under tension beyond it, will be found. It is possible that the inflamed condition of the appendix lowers the threshold of painful stimulation and allows a relatively slight distension to cause pain; but without increased tension visceral pain is impossible.

Under certain conditions recurrent attacks of appendicitis may occur in which this splanchnic central pain is the only evidence of the disease, and great difficulty in diagnosis results. A striking instance of this difficulty was afforded by a man, aged 21, who gave a history of four severe attacks of epigastric pain, each lasting for a few hours, during the course of four months. There had been neither tenderness nor muscular rigidity during the attacks. Exploration a week after the last attack showed that the only viscus affected was the appendix, which was swollen and congested in its distal portion. There was a narrow stricture at the base, and, distal to this, a collection of thick yellow pus. In the ten months that have passed since the operation there have been no more attacks.

It is evident that this patient was suffering from recurrent attacks of acute obstructive appendicitis of too mild a type to cause perforation, and that his pain was of the pure splanchnic appendicular type. The reason for the entire absence of local pain and rigidity I shall discuss later, when dealing with the second pain. The brief intermittent colicky attacks of central abdominal pain so common in children, and described by physicians as "umbilical colic," are very commonly due to spasmodic efforts to expel a concretion or a nest of threadworms from the appendix, and form another example of pure splanchnic pain.

Although the splanchnic pain in acute appendicitis is felt in the umbilical or lower epigastric region, and not in the region of the appendix, it should not be described as a referred or reflected pain, as no radiation of pain or reflex process is involved. The appendix is developmentally a part of the mid-gut loop. The brain can only appreciate painful stimuli arising from any portion of the mid-gut as vaguely situated in the centre of the abdomen, and stimuli of appendicular origin are no exception to the rule. It is characteristic of this splanchnic pain that it is dull and aching in character—as patients express it, "like an ordinary bellyache"—and vaguely localized; in other words, it is a form of Head's protopathic pain.

*A post-graduate lecture delivered at the Manchester Royal Infirmary.
†For reasons of space this introductory section has been abridged.

The Second or Localized Pain.

A few hours after the onset of the acute attack the second pain makes its appearance in the right iliac fossa. The initial splanchnic pain is usually still present, but it tends, either gradually or suddenly, to sink into insignificance. The new pain is entirely different in character from its forerunner. It is localized, severely at first, to the right side. It has a sharp or stabbing character. Any movement of the abdominal muscles, as in deep breathing, vomiting, or coughing, accentuates the pain, and the right iliac fossa becomes exquisitely tender on palpation. The patient no longer writhes in pain, but lies still in bed, resenting any movement and even complaining of the pressure of the bedclothes. The respiratory excursions of the lower abdominal wall are now increasingly limited.

Since the cutaneous and muscular phenomena interpreted by Mackenzie as the visco-sensory and visco-motor reflex have received such general attention, we must now consider them in detail.

CUTANEOUS HYPERALGESIA IN ACUTE APPENDICITIS.

Mackenzie's theory of a visco-sensory reflex was based on his observation that in certain abdominal diseases areas could be mapped out in which the skin of the abdominal

FIG. 1.—To illustrate Mackenzie's theory of a visco-sensory and visco-motor reflex. S=Skin. M=Abdominal muscles. P=Parietal peritoneum. I=Intestinal tract.

wall could not be lightly pinched up and drawn away from the underlying muscles without evoking pain. Impressed by the similarity between these areas and the skin areas mapped out by Head in cases of herpes zoster Mackenzie made the assumption that afferent stimulation through the splanchnic nerves from the inflamed viscus caused an irritable focus in its own centre of grey matter in the cord, as a result of which the sensory nerves from the skin supplied by the same spinal segment became excessively irritable. (Fig. 1.) Stimuli, such as a light pinch of the skin, which normally cause no pain, in the presence of visceral inflammation, so Mackenzie taught, become painful. Then, regarding his visco-sensory hypothesis as proved, he next argued that from these hyperalgesic areas we could, by comparison with Head's diagrams, deduce the segmental innervation of the affected viscera. He also laid great stress on the value of these areas of hyperalgesia in the diagnosis of acute abdominal catastrophes.

Sherren⁵ attempted to systematize the matter by investigating the hyperalgesia present in a series of 124 cases of acute appendicitis. He found cutaneous hyperalgesia in 40 of these, and described a cutaneous triangle of hyperalgesia over the right iliac fossa in a majority of the positive cases. Sherren's views as to the mechanism of this reflex clearly conform closely with those of Mackenzie, for he believes that hyperalgesia is never present where the appendix is gangrenous or perforated, and goes so far as to teach that disappearance of the hyperalgesia may constitute an important indication for immediate operation. He also states that cutaneous tenderness does not vary with the position of the appendix.

It appears to me necessary to consider two points in connexion with hyperalgesia in acute appendicitis—namely, its value in diagnosis, and the mechanism of its production.

The Value of Hyperalgesia in Diagnosis.

There is a marked discrepancy between the statements of different observers as to the frequency of hyperalgesia in acute appendicitis. Sherren, in 124 cases, found hyperalgesia in 40, or 32 per cent. Robinson,⁶ in 123 cases, found hyperalgesia in 26, or 21 per cent. Zachary Cope⁷ found hyperaesthesia (not amounting to pain) in 110 of 185 cases, or 59 per cent. Further, both Robinson and Cope differ from Sherren in finding that hyperalgesia and hyperaesthesia occur quite frequently with a gangrenous or ruptured appendix, and with this opinion my own observations are in full agreement.

A phenomenon that can be detected by different observers with a frequency that varies from 21 to 59 per cent. would appear to be of but little help in diagnosis. Unless, given certain pathological conditions in the appendix, hyperalgesia can be detected as a constant phenomenon, I hold that it is merely of academic interest.

A further consideration which invalidates hyperalgesia as an aid to diagnosis is the extreme frequency with which it can be elicited over the appendix in the neurotic abdomen, or those cases simulating chronic appendicitis, where on exploration no evidence of organic disease in the appendix or any adjacent organ can be found.

The Mechanism of Production of Hyperalgesia.

All observers are agreed that when hyperalgesia is present in acute appendicitis it is found, in the vast majority of cases, in some part of the right iliac fossa only. If we accept Mackenzie's hypothesis that it is due to stimuli arising in the appendix and travelling by way of the splanchnics to the spinal cord, we should expect it, however, to be bilaterally symmetrical. The appendix is a portion of the embryonic mid-gut loop. It becomes fixed in the right iliac fossa relatively late in the development of the embryo, and long after it has received its splanchnic innervation. As a developmentally median organ its nervous connexions are presumably with both sides of the spinal cord, and consequently Mackenzie's hypothesis as to the origin of the hyperalgesia would appear to be at variance with anatomical evidence. Zachary Cope, in the article quoted previously, suggests that irritation of the sensitive parietal peritoneum may in many cases determine the presence of right-sided hyperaesthesia, yet he goes on to argue that some evidence is furnished by his cases to show that the appendix is supplied mainly by the tenth dorsal segment; thus he tacitly accepts Mackenzie's visco-sensory reflex.

My own conclusions are that the hyperalgesia is a phenomenon in which the splanchnic afferent nerves take no part. I believe that the nerves of the exquisitely sensitive parietal peritoneum affect their cutaneous branches by radiation, and that the process is precisely analogous

FIG. 2.—To illustrate the mechanism of peritoneo-cutaneous radiation, and the peritoneo-muscular reflex. S=Skin. M=Abdominal muscles. P=Parietal peritoneum.

with the cutaneous hyperalgesia along branches of the fifth cranial nerve so often present in toothache. (Fig. 2.) The communication or synapsis rendering this radiation possible probably takes place in the posterior root ganglia of the nerves concerned. On account of its inconstancy, however, the practical value of hyperalgesia in clinical medicine has been greatly overestimated.

MUSCULAR RIGIDITY IN ACUTE APPENDICITIS.

We have, however, in connexion with the localized pain of acute appendicitis an objective phenomenon of prime importance because of its constancy under certain well-defined conditions. This is the deep tenderness on gentle palpation with the flat of the hand over the appendix, associated with reflex and involuntary muscular rigidity. In order to appreciate the extent and degree of this muscular rigidity it is necessary to have the patient lying at ease on his back, with the head well supported on a pillow. Gentle pressure with a warm hand on the unaffected left iliac fossa through a full respiratory cycle gives a standard of comparison. The hand is then transferred to the area of pain in the right iliac fossa, and again exerts gentle pressure through one or more respiratory cycles. The hard, unyielding contraction of the affected area of the flat muscles, the corresponding immobility of the muscle in full expiration, and the marked tenderness on pressure over the contracted muscle can then be readily appreciated and the extent of the rigidity determined. The area of muscular rigidity and tenderness corresponds accurately with the area to which the patient points when asked to indicate the position and extent of his pain.

The Value of Muscular Rigidity and Tenderness in Diagnosis.

By noting the exact position and extent of the muscular rigidity and tenderness in an early case of appendicitis, at a stage when the initial splanchnic pain has given place to the sharper pain in the right iliac fossa, but before perforation and spreading peritonitis have confused the picture, it is possible to predict with the greatest accuracy the position in which the inflamed appendix will be found. When the appendix is curled up on the outer side of the caecum the maximum rigidity is found close to the anterior superior iliac spine; when the appendix is pointing upwards towards the spleen the maximum rigidity is nearer to the umbilicus than the conventional McBurney's point. In the rare undescended caecum the maximum rigidity is higher than usual, and corresponds with a point just below the lower margin of the right lobe of the liver. Where the appendix hangs over the brim of the pelvis the rigidity is in the lower part of the rectus muscle, well below and internal to a line joining the anterior superior spine and the umbilicus. With a prolapsed caecum which has dropped low into the pelvis there may be no rigidity or tenderness on palpating the abdominal wall, but in such a case rectal examination will usually reveal marked tenderness on pressure by the finger from within towards the right side of the rectum. In this early stage the accuracy with which the inflamed appendix can be located is remarkable. One striking instance will suffice.

A woman, aged 50, became ill with pain in the umbilical region and vomiting. When seen fifteen hours after the onset there was no abdominal rigidity or tenderness, but on rectal examination pressure towards the right wall of the pelvis caused intense pain. I diagnosed an inflamed pelvic appendix. For domestic reasons some delay ensued, and I saw her again twelve hours later. She had now typical tenderness and rigidity in the right iliac fossa, while the tenderness per rectum had almost disappeared. I remarked before operating that she must have a very mobile caecum, which had moved up out of the pelvis between my two visits, dragging the appendix with it. Operation disclosed precisely that condition. The caecum was so loosely attached that it could be lifted through the wound six inches above the abdominal wall, and the appendix, which was long and free from effective adhesions, was acutely inflamed and covered with flakes of plastic lymph. The appendix and caecum were found lying in the iliac fossa well above the brim of the true pelvis. From the condition found I have no doubt that the change in position that I had predicted had actually taken place.

THE MECHANISM OF REFLEX MUSCULAR RIGIDITY AND ASSOCIATED DEEP TENDERNES.

The arguments that I have advanced against Mackenzie's hypothesis of a viscerosensory reflex apply with an even greater cogency to the theory of a visceromotor reflex by which he attempted to explain the phenomenon of protective rigidity in appendicitis. The almost invariably unilateral position of the rigidity makes the agency of the splanchnic nerves improbable, because, as I have argued above, the appendix is developmentally not a right-sided but a median organ; and the far greater constancy of muscular rigidity than of cutaneous hyperalgesia in acute

appendicitis lends greater strength to this argument. Mackenzie himself appreciated the difficulty, as he admits in his work on *Symptoms and their Interpretation*, where he says (p. 175, fourth edition): "I cannot satisfactorily account for the predominant symptoms from the appendix being so distinctly one-sided, seeing that it is developmentally a portion of the digestive tube." But even if we could accept the tacit assumption underlying Mackenzie's theory that there is a specially intimate connexion through the splanchnic nerves between the appendix and the right side of the spinal cord, how are we to reconcile it with the fact—which I have so many times verified—that the area of muscular rigidity and deep tenderness corresponds exactly with the position of the appendix? If the visceromotor reflex were a fact, the muscular rigidity produced by it would be constant in position, since it would be determined by the segmental innervation of the appendix, and would be entirely unaffected by the position in which the appendix happened to be lying in relation to the abdominal wall.

I find, then, that the clinical investigation of the distribution of muscular rigidity in appendicitis reveals facts that are entirely inconsistent with Mackenzie's visceromotor reflex theory. Nor am I aware of any physiological experiments which make it probable that such a reflex exists, nor of any anatomical evidence of a communication in the cord between splanchnic afferent and somatic efferent nerve fibres. The whole theory appears to me to be an unsound extension of Ross's theory of somatic referred pain, based upon a mistaken interpretation of clinical and operative findings. Mackenzie did not appear to appreciate the exquisite sensitiveness of the parietal peritoneum to even slight degrees of inflammation. Had he done so I believe that so acute a mind as his would have perceived that in a peritoneo-muscular reflex lies the explanation of this protective muscular rigidity, and that the splanchnic afferent nerves have nothing to do with the reflex rigidity in appendicitis.

The cumulative effect of correlating pre-operative symptoms with operative findings in acute appendicitis has convinced me that the second or localized pain in the disease is entirely dependent on stimulation of the sensitive parietal peritoneum. (Fig. 2.) In cases such as the first one quoted above, where recurrent attacks of obstructive appendicitis gave rise to the epigastric splanchnic pain alone, the parietal peritoneum was shielded from the appendix by a fairly thick omentum; and as the degree of inflammation was too mild to penetrate through the thick walls of the appendix and the omentum the parietal peritoneum was not affected. This explains the complete absence of localizing pain or rigidity, and the difficulty in diagnosis. An appendix wrapped round by omentum and coils of small intestine as a result of some former attack will, in fat patients, often give rise to no characteristic right-sided pain or rigidity until it has perforated. This absence of rigidity is particularly liable to occur if the caecum and appendix are lying low in the true pelvis. But a degree of inflammation so slight as to cause but a little congestion of the outer serous coat of the appendix and a trifling fibrinous deposit will, provided it lies within reach of the parietal peritoneum, cause the most accurately localized tenderness and rigidity. Should perforation occur the rigidity and tenderness extend *pari passu* with the inflammation of the parietal peritoneum, while from the moment of perforation all the initial splanchnic pain disappears.

THE PERITONEO-MUSCULAR REFLEX AND PERITONEO-CUTANEOUS RADIATION.

Since the parietal peritoneum is supplied by the same cerebro-spinal nerves as the overlying muscles and skin, the accuracy with which an inflammatory stimulus from within the abdomen is localized can be easily understood. It is necessary, however, to insist that a true nervous reflex mechanism through the governing centre in the spinal cord is involved. The tonic contraction of the affected area of muscles, recognized on palpation as a hardness that never relaxes during the whole respiratory cycle, makes it impossible for us to believe that we are pressing on the inflamed parietal peritoneum itself. The pressure required to appreciate the muscular rigidity is too light to have any

influence of that kind, and even on inspection alone the tonic muscular contraction may readily be appreciated, if we observe the limitation of respiratory movements in the lower rectus muscle.

The peritoneo-cutaneous radiation is best appreciated by a consideration of the shoulder-tip pain resulting from stimulation of the parietal peritoneum lining the under-surface of the diaphragm. In some observations on this shoulder-tip pain¹ I have drawn attention to the fact that whenever the sensory terminations of the phrenic nerve under the diaphragm are stimulated, either mechanically or by inflammatory exudate, a pain is felt over the corresponding shoulder in the area supplied by the third and fourth cervical nerves. This shoulder-tip pain is of special interest as proving a peritoneo-cutaneous reflection or radiation of the painful stimulus. By the descent of the diaphragm in the embryo this area of parietal peritoneum becomes widely separated from its corresponding skin area, and thus an embryological event, dependent on the development of the lungs, enables us to observe the working of the pain-producing mechanism. While I have proved the invariability of shoulder-tip pain where the diaphragm is stimulated, I have not found the same constancy of cutaneous hyperalgesia as tested by light pinching over the acromion process. In some cases the patients describe the pain as akin to rheumatism in the shoulder, and deny any tenderness on light pinching or deep pressure. In some instances tenderness is felt, but only on deep pressure on to the acromion process, while in others definite hyperalgesia is felt on light pinching.

It cannot be doubted that a similar radiation or spreading of painful stimuli from the parietal peritoneum to the skin overlying it via the spinal cord occurs in the anterior abdominal wall, though the proximity of skin to peritoneum has prevented its recognition. When the under-surface of the diaphragm in a patient under spinal anaesthesia is stimulated by the insertion of a gauze swab between the liver and the diaphragm, the patient complains of pain in the shoulder-tip area, but feels no pain locally. So, when the parietal peritoneum is stimulated I believe that the pain is felt in the skin and subcutaneous tissue alone. The exact position in the nervous system of the synapses necessary to produce this radiation is uncertain. The teaching of Mackenzie and Ross would lead us to expect to find it in the grey matter of the posterior horn of the cord, but there would appear to be a greater probability that it lies in the posterior root ganglia. (Fig. 2.)

If I have succeeded in proving my contention that the muscular rigidity in acute appendicitis is a reflex result of peritoneal stimulation alone, the diagnosis of acute appendicitis becomes a far simpler clinical problem. Mackenzie himself, obsessed by his theory of a visceromotor reflex, taught that it was impossible to separate the symptoms produced by the inflamed appendix from those produced by the inflamed parietal peritoneum. From my experience in teaching students I am sure that the widespread acceptance of Mackenzie's doctrine has introduced an unnecessary difficulty into the diagnosis of abdominal lesions. The student, picturing muscular rigidity as a reflex from the inflamed organ through the splanchnic afferent nerves, and conscious that peritonitis produces a similar effect, finds a hopeless impossibility in the task of distinguishing between the two processes, and is apt to regard abdominal diagnosis as very largely guesswork. But if he can be taught that true reflex involuntary rigidity is the result of stimulation of the parietal peritoneum, and of that alone, and that the only true pain felt in the gastro-intestinal tract is a splanchnic pain due to increased tension and exaggerated peristalsis, the investigation of a case of acute abdominal disease becomes relatively straightforward.

A BIOLOGICAL CONSIDERATION OF ABDOMINAL PAIN.

I will finally sketch in briefest outline what I conceive to be the biological or evolutionary aspect of abdominal pain. With the development of the alimentary tract and the peritoneal cavity a great responsibility for the protection of the abdominal viscera was laid upon the parietal peritoneum. To guard against bacterial invasion of the peritoneal cavity from without by penetrating wounds, or

from within by perforation of a hollow viscus, the parietal peritoneum was endowed with nerves of exquisite sensitivity to mechanical or chemical insult. Stimulation of this layer produced reflex guarding of the overlying muscles, with pain felt in the skin and on the surface of those muscles. At the same time another reflex—not dealt with in this paper, but also originating in the parietal peritoneum, the splanchnic inhibitory reflex—was provided to secure rest to the intestinal tract and so to avoid the diffusion of septic material by peristalsis.

Against the danger of intestinal obstruction another mechanism had to be elaborated. Violent peristaltic efforts of myogenic origin attempted to overcome the obstructing force, and their occurrence was registered by painful afferent stimuli through the splanchnic nerves, appreciated by the brain as spasmodic in character, and vaguely localized in the centre of the abdomen. This splanchnic pain would seem to be the more primitive in order of evolution. It is akin to protopathic pain in its dull character and in the absence of precise localization. Moreover, a considerable stimulus is needed before consciousness of pain is produced.

SUMMARY.

1. The evolution of our knowledge of the mechanism of abdominal pain is summarized.

2. The initial central pain in acute appendicitis is a true splanchnic pain, due to increased intra-appendicular tension.

3. The localized right-sided pain is due to irritation of the parietal peritoneum.

4. Cutaneous hyperalgesia is too variable in its incidence to be of value in the diagnosis of acute appendicitis.

5. The mode of production of cutaneous hyperalgesia is discussed. Mackenzie's viscerosensory reflex theory is criticized.

6. The mechanism of reflex muscular rigidity and the associated deep tenderness is discussed. Mackenzie's visceromotor reflex theory is criticized, and the role of the parietal peritoneum emphasized, protective muscular rigidity being ascribed to a peritoneo-muscular reflex.

I am indebted to Professor J. S. B. Stopford, M.D., F.R.S., for his kindness in drawing the diagrams illustrating this paper.

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AN OPERATION FOR THE CURE OF PROLAPSE OF THE RECTUM IN THE FEMALE.*

BY

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THE aim of the operation herein described is to restore the muscular retentive mechanism which prevents the bowel prolapsing after each act of defaecation.

In July, 1913, a single woman, aged 51, came to see me; she had undergone an operation for rectal prolapse four years previously, the prolapsed portion of the bowel being excised. The operation was, unfortunately, a failure. The bowel still prolapsed for nearly two inches and the anal orifice surrounded by scar tissue admitted three fingers. She had complete incontinence of faeces, and was unable to earn her living as a nurse. Her condition was deplorable, and her nervous system had suffered considerably in consequence. She was reluctant to undergo another operation, but ultimately yielded to persuasion.

The bowel was returned and the patient was placed in the elevated lithotomy position; an incision was then made around the anal orifice anteriorly and laterally for three-fourths of its circumference. The perineum was incised in the middle line from the fourchette to the anal orifice; this incision was further prolonged anteriorly into the labium of each side for about an inch and a half. The lower end of the rectum was freed by dissection anteriorly and laterally, exposing the levator ani on either side, whilst the lower portion of the posterior vaginal wall was dissected upwards for about two inches. A V-shaped piece was then cut out

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