Remarks

ON

THE DIAGNOSIS OF ACUTE ABDOMINAL CRISES.*

BY

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IN these two lectures I propose to discuss the diagnosis of acute abdominal crises in a general sense, and to consider first of all, individually, the various symptoms and signs met with in such crises, attributing to each its relative value as a factor in diagnosis, and as an indication for treatment; and secondly, these same symptoms arranged in groups, such as are observed clinically in the different types of abdominal crises.

First, then, let us consider the diagnostic value of such symptoms as may be present in an acute abdominal crisis.

GENERAL SYMPTOMS.

The general symptoms include:

1. Various degrees of shock or collapse.
2. Alterations in the frequency and character of the pulse.
3. Changes in the temperature.
4. Alterations in the type of respiration—"thoracic" type.
5. Other symptoms, such as a dry and furred condition of the tongue, hiccough, strangury, and tenesmus.

Speaking generally, one may say that, although these general symptoms are useful in directing attention to the possibility of a crisis having occurred, yet they give no clue to its nature, nor are they alone of much value as indications for treatment. They may occur alike in serious and comparatively trivial abdominal lesions, as evidenced, on the one hand, by the profound degrees of shock which may accompany a simple contusion of the abdominal wall, or be present during an attack of colic; while, on the other hand, cases of perforation of a viscus are sometimes seen with practically no rise of temperature or pulse, and with very little, if any, shock. In fact, so unlikely is it that any material assistance will be obtained from a consideration of the temperature or pulse that I feel strongly that fewer cases would pass unrecognized in their early stages were it to become the universal rule never to take the temperature nor count the pulse in a suspected abdominal crisis; they are only too likely to mislead. The persistence of shock and of the other general symptoms is of far greater diagnostic importance than is their initial degree.

LOCAL SYMPTOMS.

It is, then, mainly upon the local symptoms and signs—those referable to the abdomen itself—that we have to rely in deciding questions of diagnosis and treatment in any acute abdominal crisis. These include:

1. Abdominal pain.
2. Cutaneous hyperalgesia ("viscero-sensory" reflex).
3. Tenderness, local and general.
4. Nausea, vomiting.
5. Muscular rigidity ("viscero-motor" reflex).
6. Abdominal distension.
7. Presence of free gas in the peritoneal cavity.
8. Presence of free fluid in the peritoneal cavity.
9. Local abdominal or pelvic swelling.
10. Certain changes ascertainable upon rectal examination.

1. Abdominal Pain.

It is now well recognized that the viscera are themselves quite insensitive to touch or pain, and it has been shown by Mackenzie and many others that pain resulting from a lesion of a viscus is felt not in that viscus itself, but is referred to the peripheral distribution of the spinal nerves arising from those segments of the spinal cord with which that viscus is connected through its sympathetic nerve supply. Since the abdominal wall is supplied by the lower six dorsal nerves and the abdominal viscera are connected via the sympathetic with these same nerves, it follows that pain arising from a morbid state of an abdominal viscus is referred to the peripheral distribution of the lower six dorsal nerves—that is, to the mid-line

of the anterior abdominal wall. It must, however, be remembered that these nerves have both posterior and lateral branches, so that in some cases pain is referred to the back or around the side of the chest and abdomen as well as, or even instead of, to the middle line anteriorly.

So far as the alimentary canal is concerned, reflex pain from the stomach is referred to the peripheral distribution of the fifth to eighth dorsal nerves—that is, between the xiphoid cartilage and the umbilicus; that from the duodenum close above the umbilicus; that from the small intestine and appendix (eighth to eleventh dorsal) to the "umbilical" area (the umbilicus corresponding to the tenth dorsal); and that from the large intestine to the mid-hypogastric line. From the liver, gall bladder, and bile ducts (seventh to ninth dorsal) pain is referred to the lower part of the epigastrium and also sometimes to the lower angle of the scapula (eighth dorsal). From the close association between the liver and diaphragm, supplied by the phrenic nerve, we find reflex pain transmitted along the superficial cervical nerves arising from the spinal cord at the same level as the phrenic (third and fourth cervical), resulting in pain felt over the outer and anterior aspects of the shoulder—the characteristic "shoulder tip" pain in gall stones, much less frequently observed clinically than the stress laid upon it in textbooks would lead one to anticipate.

All pain, then, felt in the early stages of an acute abdominal crisis is of this reflex character, and is not, therefore, localized to the situation of the viscus primarily at fault. Later pain, however, that due to irritation of the parietal peritoneum, is always felt at the site of irritation, and has, consequently, considerable localizing value. It is well known that there is a rich nerve-plexus in the connective tissue underlying the parietal peritoneum, and as this is in connexion with the cerebro-spinal system, unlike the visceral peritoneum which is supplied entirely by the sympathetic, accurate localization is possible. Let us consider the subjective pain felt during an attack of acute appendicitis. Its most characteristic feature is that it commences not over the region of the appendix at all, but is referred to the area of the umbilicus—in fact any acute abdominal attack in which the initial pain is felt in the right iliac fossa is extremely unlikely to be appendicitis. The later transference of pain to the right iliac fossa indicates extension of the irritation to the parietal peritoneum. The closer the appendix is situated to the parietal peritoneum of the right iliac fossa the earlier will the latter be irritated, and the sooner will pain be there localized. It must, however, be remembered that the appendix is sometimes placed deeply among the coils of small intestine or among the pelvic viscera, so that there may be no pain localized to the right iliac fossa at any stage, the initial "umbilical" area pain becoming steadily generalized. Absence of local pain may be, therefore, either a favourable sign, indicating that the infection is so slight as never to irritate the parietal peritoneum (and the appendicular origin of many of these cases is overlooked), or an unfavourable sign, indicating an appendix deeply placed among the viscera and therefore likely to cause diffuse peritonitis. When well-marked local pain appears in the right iliac fossa soon after the initial "umbilical" area pain one may confidently expect to find the appendix not far removed from the parietal peritoneum. These statements refer to first attacks only, for if as the result of previous attacks adhesions attach the appendix to the parietal peritoneum, the latter may become irritated almost from the very first and the pain be correspondingly early localized.

2. Cutaneous Hyperalgesia.

In the normal state impulses are constantly passing both in an afferent direction from the viscera to the spinal cord and efferently from the cord to the skin and muscles of the abdominal wall without our being aware of their existence. If a viscus becomes diseased the afferent impulses will naturally be of a more irritant nature, and the segment or segments of the cord receiving those impulses will be in a state of hyper-excitability. If a cutaneous nerve connected with one of these hyper-excitability segments receive a stimulus—such as a light pinch, which normally would have been quite insufficient to evoke any sense of pain, and which applied elsewhere

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to the body would fail to do so—it is very probable that pain will be felt; that is, the area of skin pinched is said to be "hyperalgesic." Theoretically the whole cutaneous distribution of the hyperexcitable segment might be expected to be hyperalgesic, but in practice it is found that certain areas are especially susceptible. These have been well worked out by Mr. David Ligat, and are described in detail in his Hunterian Lecture;¹ by his kind permission I reproduce his diagram showing the "functional" areas where hyperalgesia is elicited in disease of the various viscera. His method of exciting the reflex response is to grasp the skin and subcutaneous tissue firmly between finger and thumb and draw them away from the deeper layers of the abdominal wall. "If an abnormal reflex is produced, it is at once recognized—the patient winces; he

Diagram showing the areas where hyperalgesia is elicited when present. The width of the shaded area does not denote lateral spread, but merely represents degree of intensity of reflex in typical cases. 1. Gastric and duodenal area: maximum point midway between ensiform cartilage and umbilicus. 2. Small gut area: maximum point at junction of upper and adjacent fourths of a line drawn from umbilicus to symphysis pubis. 3. Great gut area: maximum point at junction of lowest and adjacent fourths of a line drawn from symphysis pubis to umbilicus. 4. Gall bladder area: the maximum point is situated where a horizontal line drawn from the tip of the tenth rib crosses a vertical line drawn midway between the nipple line and the middle line of the abdomen. 5. Appendix area: maximum point at junction of inner and middle thirds of a line drawn from the anterior superior spine to the umbilicus. 6. Fallopian tube area: maximum point at junction of lowest and adjacent fourths of a line drawn from the middle of Poupart's ligament to the umbilicus. (Reproduced by kind permission of Mr. David Ligat from his Hunterian Lecture, published in the *Lancet*, May 3rd, 1919.)

diagnosis of such disease seem probable upon other grounds, the absence of any hyperalgesia must be ignored. The sudden disappearance of hyperalgesia, after having been once definitely elicited—as, for instance, in acute appendicitis—is always a grave sign, indicating interruption of the reflex arc, as by thrombosis of the mesentery of the appendix or gangrene.

3. Tenderness.

Localized deep tenderness, which must be carefully differentiated from superficial cutaneous hyperalgesia, is always a valuable sign. The viscera themselves are quite insensitive to pressure or pain, so that when, in disease of a viscus, pain is elicited by pressure over any area of the abdominal wall such pain may be due to either (1) cutaneous hyperalgesia (a true visco-sensory reflex), or (2) local parietal peritoneal irritation from stimulation of the subserous nerve plexus connected with the cerebro-spinal system (and therefore capable of accurate localization), and here there is no question of any reflex being concerned; it is not due to direct pressure upon the diseased viscus itself. Thus the pain elicited by deep

pressure over "McBurney's" point in acute appendicitis, and localized by the patient to the point pressed upon, arises, not from direct pressure upon an inflamed appendix, but from pressure stimulation of the subserous nerve plexus rendered unduly irritable by extension to it of irritation from such appendix, no reflex being involved. Were it otherwise—that is, were the pain due to pressure stimulation of the inflamed appendix itself—then the stimuli, on reaching the spinal cord via the sympathetic, would be referred to the "umbilical" area, as already mentioned, and not to McBurney's point. I believe this is what actually does happen occasionally in cases met with in the very earliest stages of acute appendicitis, before the irritation has had time to extend to the subserous plexus, where, although the patient complains of pain when pressed over McBurney's point, yet, if carefully questioned, he will locate that pain to the region of the umbilicus.

Local tenderness is especially met with in the inflammatory group of abdominal crises—appendicitis, cholecystitis, salpingitis, and pancreatitis. The site of maximum tenderness is not alone sufficient to differentiate between these various conditions, since as already explained it merely indicates the site where the subserous nerve plexus is most acutely irritated by extension from the inflamed viscus. Thus local tenderness in the right iliac fossa may be due to the appendix, the right Fallopian tube, a low-lying gall bladder, or escaped contents from a perforation of the duodenum. In the differentiation between these valuable help may be derived from the detection of any of the hyperalgesic areas.

General abdominal tenderness, unless associated with muscular rigidity of the involuntary type, shortly to be described, is of no special importance. When so associated it usually indicates peritonitis.

At the risk of repetition I would emphasize the importance of a clear appreciation of the difference between the three signs already considered—pain (subjective), cutaneous hyperalgesia, and local deep tenderness, and would point out that in appendicitis they are manifested each in a different area—the subjective pain has reference to the "umbilical" area, the cutaneous hyperalgesia to the "appendix" area (see Ligat's diagram), and the local tenderness usually to McBurney's point.

4. Vomiting.

Like the pain, the vomiting which sets in at or soon after the onset of an acute abdominal crisis is a purely reflex phenomenon, and not, therefore, of any diagnostic value. Such reflex vomiting, however, usually subsides within twenty-four hours and does not tend to recur. A recurrence of vomiting after having definitely ceased for some time is always a grave sign, and usually indicates the onset of peritonitis. Persistence of vomiting from the onset is also of serious import, and should raise a strong suspicion of intestinal obstruction.

5. Muscular Rigidity.

Stimuli received from a diseased viscus by the spinal cord may irritate the motor cells of the same segments, and so cause a reflex contraction of the abdominal muscles supplied by those segments—the "viscero-motor" reflex of Mackenzie—rendering the abdominal wall in that area rigid. This is Nature's effort at protection, and is in every way analogous to the muscular spasm around a joint in a state of acute synovitis, fulfilling the same purpose—in fact, the peritoneum is the great visceral joint. The exact site and extent of the rigidity will depend upon the nerve connexions of the viscus primarily at fault with the spinal cord segments; but as it is always much more difficult to accurately map out areas of rigidity than areas of hyperalgesia, the visco-sensory reflexes will be found to be of greater service than the visco-motor in determining the primary focus of disease. Though the visco-motor reflex may exist quite apart from peritoneal irritation, yet in the vast majority of acute abdominal crises peritoneal irritation is the exciting factor. Normally the peritoneum is moistened by a small amount of serous fluid to the presence of which it has become accustomed; any material alteration of the chemical constitution of this fluid will serve as an irritant and start a visco-motor reflex. Mere increase in the amount of the fluid is not sufficient to do so—for example, the absence of

abdominal rigidity in simple ascites. Naturally, we may expect that the greater the chemical alteration the greater the consequent muscular contraction, and this we find to be the case. Thus the contraction is best marked where acid fluids are suddenly shot into the peritoneal cavity, as in rupture of gastric or duodenal ulcer. On one occasion I had the opportunity of examining the abdomen within two minutes of a perforation of a duodenal ulcer, and found the muscles intensely rigid even in that short time. Next in order come the contents of the intestine, then bile; urine, even though slightly acid, seems to be still less of an irritant. There is, however, one fluid that may be suddenly poured out even in large quantity into the peritoneum and yet prove so little of an irritant that it fails to start a visceromotor reflex, and that fluid is blood. Almost identical in chemical composition with the normal peritoneal fluid, it does not act as an irritant. Hence we may find an entire absence of muscular rigidity overlying an extravasation of blood into the abdominal cavity, at any rate in its earlier stages. Later, as the blood clots, slight irritation may be set up, and some slight rigidity be detected. This absence of a visceromotor reflex over an underlying intraperitoneal haemorrhage is an extremely important fact to bear in mind, and unless regard be paid to it it is easy for a large effusion of blood into the peritoneal cavity to be entirely overlooked.

Differentiation of Involuntary from Voluntary Muscular Rigidity.—A patient expecting pain from an examination of the abdomen, or an intentional malingerer, will often voluntarily contract the abdominal muscles and so simulate a visceromotor reflex. Since voluntary muscular contraction is of no value in diagnosis, and depends upon the temperament of the individual patient, it is of the highest importance to distinguish it from the true involuntary rigidity of a visceromotor reflex. The chief points of difference are: (1) gentle palpation for several minutes with the flat of a warm hand, with regular steady pressure, will cause voluntary rigidity to disappear or diminish; (2) distraction of the patient's attention by questioning him as to his symptoms, or directing his notice to some other part of his body, will cause voluntary rigidity to relax; (3) in voluntary contraction there is always a momentary relaxation of the muscles between the end of one expiration and the commencement of the succeeding inspiration, sometimes extending a little way into inspiration. *True involuntary muscular rigidity never relaxes, however gentle the examination, however one distracts the patient's attention, nor is any relaxation met with at any phase whatsoever of the respiratory movements.*

Defective Mobility.—Muscular rigidity may also be observed clinically as defective mobility during respiration, and it is always useful to commence an examination of the abdomen by asking the patient to take a few full breaths, carefully noting any defective movement of any part of the abdominal wall. It will usually be found that any area which does not move on respiration will, on palpation, present the features of involuntary muscular rigidity; the converse is not, however, always true, and I have seen cases where respiratory movement seemed perfect nevertheless show definite evidence of involuntary rigidity on palpation. Moreover, by simple inspection one cannot distinguish with the same facility as by palpation between voluntary and involuntary rigidity.

Involuntary muscular rigidity lasts as long as the peritoneal irritation causing it lasts, or until the muscles are previously exhausted, or the reflex arc sufficiently "dulled." Even morphine, which abolishes so many of the early reflex phenomena of an acute abdominal crisis, usually, and fortunately, fails to entirely abolish this. As to its clinical significance, I believe involuntary muscular rigidity to be an absolutely diagnostic sign of a serious underlying lesion, usually with some chemical irritation of the peritoneum, and therefore as the strongest single indication for surgical treatment that we possess; nor have I ever operated in the presence of this sign without finding such definite lesion present. Unfortunately the converse is not true, and serious changes may be going on within the abdominal cavity without any overlying muscular contraction. This is especially seen in cases of intra-abdominal haemorrhage, blood serum being, as just pointed out, insufficiently abnormal to the peritoneum to start a visceromotor reflex.

6. Distension of the Abdomen—Meteorism.

That is, gas in the intestines as distinguished from free gas in the peritoneal cavity. This sign may be observed whenever the bowel is paralysed from any cause, but is especially seen in the groups of intestinal obstruction and peritonitis. A slight local meteorism is not necessarily of any serious import, but a *steadily progressive distension* of the bowels should raise a very strong suspicion of intestinal obstruction or spreading peritonitis.

7. Free Gas in the Peritoneal Cavity.

Free gas necessarily rises to the highest part of the abdominal cavity, and as the patient will usually be in the dorsal posture the gas will rise between the liver and the anterior abdominal wall and diaphragm, so leading to loss or diminution of the normal liver dullness. On the left side the splenic dullness will be similarly affected, but since the normal splenic dullness is a very inconstant quantity, its loss is thereby so discounted as to be useless for diagnosis. As a *positive* sign this loss of liver dullness is of great importance, provided there is no general distension of the bowels such as might force up the transverse colon between the liver and the diaphragm. As a *negative* sign it is valueless, and the presence of the normal liver dullness in no way excludes a perforation of the alimentary canal, since even in the presence of a perforation so insignificant an amount of gas may have escaped as not to materially affect the liver dullness. Especially valuable is the determination of the loss of liver dullness in a patient in whom, an hour or so before, its presence had been particularly noted.

8. Free Fluid in the Peritoneal Cavity.

Free fluid, shown clinically by the signs of "shifting dullness in the flanks," is not frequently observed in abdominal crises. It may, however, be seen (1) in large internal haemorrhages—for example, rupture of ectopic gestation; (2) after rupture of an organ containing much fluid—for example, distended bladder; and (3) after rupture of large cysts—for example, ovarian or hydatid. The most marked instance I have seen was in a case of perforated gastric ulcer, with large perforation of the lesser curvature; the patient, during a long journey from the country to the Manchester Royal Infirmary, had been supplied constantly with soda-water by sympathetic relatives to assuage a violent thirst.

9. The Presence of a Definite Abdominal or Pelvic Swelling.

This is of great diagnostic value, and is most frequently noted in the inflammatory group of abdominal crises—appendicitis, cholecystitis, and pelvic inflammations. Even when present its detection may be very difficult on account of overlying muscular rigidity, and a caution must be given not to use any forcible pressure in examining for it, as otherwise an appendicular abscess may readily be ruptured. A definite swelling may also be felt in some cases of intestinal obstruction, notoriously in intussusception, where its situation and shape are characteristic, and also in obstruction from malignant growths, from faecal masses, and occasionally from foreign bodies. In one case I was able to feel a large gall stone and in another a large hairball, both causing acute obstruction.

10. Rectal Examination.

This often throws light upon the diagnosis of an acute abdominal crisis, especially in the inflammatory and obstructive types. Inflamed appendices can sometimes be felt per rectum and appendicular abscesses recognized when the abdominal examination is quite negative. In obstruction from intussusception the invagination may be felt, malignant growths of the rectum causing obstruction can be detected; and, even when too highly situated in the colon for this, the peculiar "ballooning" of the bowel below them raises a strong suspicion of their presence, though this sign may occur also in peritonitis.

Having dealt with the individual symptoms and signs observed in acute abdominal crises, let us turn to a consideration of their arrangement in groups such as we meet with clinically. But, before deciding in any given case presenting symptoms suggestive of an acute abdominal crisis that the cause is referable even to the abdomen at all, there are two fallacies in particular which it is important should be borne in mind. These are: (a) The "crises"

met with in *tabes dorsalis*—"gastric," "intestinal," and "renal." It should be a cardinal rule to test the knee-jerks in every abdominal case. On the other hand, it must be realized that *tabes* does not preclude the co-existence of a true abdominal crisis, and, further, that the visceral analgesia which occurs in some tabetics and prevents the manifestation of pain, cutaneous hyperalgesia, tenderness, and rigidity, may render the recognition of such crisis a very difficult matter indeed. (b) The possibility of confusion between acute abdominal crises and acute intrathoracic diseases, especially acute basal pneumonia, acute diaphragmatic pleurisy, and acute pericarditis. This difficulty is sometimes a very real one, and surgeons of experience have not infrequently opened the abdomen only to find the peritoneal cavity normal and the lesion an intrathoracic one. Whenever acute *lobar pneumonia* follows within twenty-four or forty-eight hours of an "early appendectomy," one cannot help but suspect that the pneumonia was the real cause of the abdominal symptoms and that the operation was unnecessary; when pneumonia is due to an operation, it is of the *lobular* or bronchopneumonic type. The lower six dorsal nerves before entering the anterior abdominal wall supply the posterior and lateral aspects of the lower half of the chest wall. In disease of the chest viscera, and especially in basal pneumonia and pleurisy, pain may be referred to the peripheral distribution of these nerves—that is, to some part of the mid-line of the anterior abdominal wall—just as it is in a true abdominal crisis. Confusion is particularly liable to arise in the case of young children, since they cannot assist us with a description of their symptoms, and since in them the physical signs of a pneumonia are sometimes delayed for twenty-four or even forty-eight hours. Thus, for instance, in the early stages of an acute pneumonia in a child we may meet with any or all of the following:

1. Acute pain referred to the mid-line of the anterior abdominal wall.
2. Tenderness on palpation of the abdomen.
3. Defective mobility of the abdominal wall on respiration.
4. Rigidity of the abdominal muscles.
5. Abdominal distension (from associated constipation).
6. Nausea, vomiting, and hiccough may all be present, as well as signs of severe collapse.

If, in addition, no physical signs have yet appeared in the lungs it must be admitted that the simulation of an acute abdominal crisis is a very exact one. The points to consider in making a differential diagnosis are: (a) The frequency of respiration is out of all proportion to the abdominal symptoms or to the pulse; the respirations may be 40 or 50 with a pulse of only 100. As a working rule, a pulse-respiration ratio of less than 3 to 1 favours an intrathoracic cause, of more than 3 to 1 an intra-abdominal cause. (b) The average temperature of an intrathoracic lesion is higher than an intra-abdominal one, so that a high temperature combined with a low pulse-respiration ratio strongly suggests an intrathoracic cause, as does the occurrence of a rigor or of herpes labialis. (c) The abdominal tenderness is more superficial, and deep pressure with the flat of the hand is not only tolerated but may give relief in an intrathoracic lesion. (d) Although intrathoracic inflammation may be associated with marked rigidity of the abdominal muscles, yet this partakes of the "voluntary" rather than the true "involuntary" type. In all the cases I have seen it has been possible to detect a momentary relaxation at the end of each expiration, such as does not occur with an intraperitoneal lesion. (e) The detection of the "hyperalgesic" areas referred to previously would suggest the abdominal viscus primarily at fault; these would be absent in intrathoracic disease, but, as already explained, are by no means necessarily present in intra-abdominal disease. (f) A rectal examination may assist; marked tenderness suggests a peritoneal cause. If a careful consideration of these points still leaves one undecided, then I believe an exploratory laparotomy should be at once undertaken, as being the lesser of two evils—dangerous delay and unnecessary operation.

GROUPS OF CASES.

Having, then, excluded these possible fallacies, and having decided that we are dealing with a true abdominal crisis, we have next to consider the possible causes of this

condition. It is not my purpose now to inflict upon you a list of all the various causes of acute abdominal crises—their number is extremely large, and no one who did not possess the confidence born of youthful inexperience would attempt to devise any scheme by which they could all be recognized before either operation or *post mortem*. For my own guidance I am accustomed mentally to classify them into five groups, and although some of the rarer conditions do not fall exactly under any of such heads, yet I have found it a very convenient scheme to have in mind at the bedside. These groups are:

1. The colics.
2. The perforations.
3. The hæmorrhages.
4. The inflammations.
5. The obstructions.

This order represents roughly their average suddenness of onset. We must remember that the *early symptoms in them all may be identical*, since they depend upon the same cause—a shock to the solar plexus, evidenced clinically by pain referred to the mid-line of the abdominal wall, vomiting, and shock. These symptoms have long been described collectively by Sir Frederick Treves under the term "peritonism," and are of no value in differential diagnosis, since they may be present whatever the nature or wherever the situation of the lesion, provided only that it be of sufficient intensity to stimulate the solar plexus, directly or indirectly. Moreover, *this initial stage of shock is usually a transient one*, and is followed by a period of reaction—a dangerous calm before a mighty renewal of the storm—during which all the general symptoms may completely disappear, and it may be extremely difficult to convince oneself, let alone the practitioner or patient, that a really serious lesion is actually in existence within the abdomen. This "dangerous period" has long been recognized in connexion with acute appendicitis, but, as pointed out by that super-teacher of clinical surgery Mr. Rutherford Morison,² its occurrence in other forms of abdominal emergencies is not so widely known. Morison describes three stages of an abdominal emergency—the "triple syndrome":

1. The stage of shock.
2. The stage of reaction.
3. The stage peculiar to each lesion.

If, as too often is the case, the onset of the stage of reaction be assisted and intensified by the indiscriminate exhibition of morphine, it may prove a very pitfall of diagnosis. I well remember seeing in consultation a lady who had been seized with a violent epigastric pain and who when seen half an hour afterwards by her medical attendant was in a state of profound collapse, nevertheless, after injection of one-quarter grain of morphine, make such a complete temporary recovery that she was sitting up when I saw her four hours later, laughingly protesting that there was nothing whatever the matter with her; the only sign unmasked by the morphine was rigidity of the recti of the "involuntary" type; immediate operation revealed a perforation high up on the lesser curvature of the stomach with considerable extravasation of gastric contents.

GROUP 1.—The Colics.

This group is characterized by (a) the extremely sudden onset of the pain, (b) its sudden cessation, (c) its tendency to recurrence. The pain is, moreover, referred to certain characteristic regions. In *biliary colic* it is felt in the epigastrium, radiating backwards to the angle of the right scapula, and occasionally, when the endings of the phrenic nerve are stimulated, to the tip of the right shoulder. In *renal colic* it is felt to commence in the loin and to shoot downwards towards the groin, inner aspect of the thigh, and the testicle; often definite tenderness of the testicle, which receives its nerve supply from the same spinal segments as the kidney, can be elicited, although there is no tenderness of the overlying scrotal skin, supplied from the sacral segments; exceedingly important is the association of the pain with some increased frequency of micturition, whilst the occasional coexistence of blood in the urine is diagnostic. In *intestinal colic* pain is felt all over the abdomen, especially in *lead colic*, where the pulse, during the pain, is usually retarded, vomiting and constipation are associated, and a blue line in, not on, the gums may be detected. Tenderness of the abdomen may be present, but

in many cases, especially of intestinal colic, the pain is very definitely relieved by pressure, such as never obtains in peritonitis. Muscular rigidity may occur, especially in biliary and renal colic, but I have never seen it present the unyielding character of true involuntary rigidity. Restlessness, although it gives no relief, is a marked feature of colic, but never occurs in a perforation.

GROUP 2.—*The Perforations.*

As a group this is characterized by (a) the very sudden onset of the pain, which is usually described as excruciating; (b) the severity of the initial shock, which may, however, soon pass off; (c) the marked development of true involuntary muscular rigidity from the very first; (d) there may be signs of free gas in the peritoneum. Of these (c) is the most important and demands immediate laparotomy. Some clue to the viscus perforated may be obtained from the area of most marked cutaneous hyperalgesia and from the previous history of the case—for example, of "indigestion," appendicitis, or typhoid, but time should not be wasted in this connexion, as the site of perforation can be discovered with certainty only at the operation.

GROUP 3.—*The Haemorrhages.*

Haemorrhage taking place into one of the mucous cavities of the abdominal viscera, if of such intensity as to constitute a "crisis," will probably soon show itself by escaping from one of the natural orifices and so be detected—for example, haemorrhage from the stomach, bowels, and uterus. It is haemorrhage occurring into the peritoneal cavity itself that is so likely to be overlooked and for the reason already stated—namely, that blood is not sufficiently irritating chemically to act as a stimulus to the peritoneum and thus start a visceromotor reflex, so that there is an entire absence of muscular rigidity. We have, in fact, to rely for diagnosis upon the supervention of the general signs of loss of blood, and if the blood is escaping but slowly, these may be late in appearing. Whenever, therefore, we meet with a history of sudden acute abdominal pain, with or without the other signs of "peritonism," and find a perfectly soft, supple, and undistended abdomen, we ought, although we can at once exclude the other groups of abdominal crises, to bear in mind the possibility of haemorrhage taking place into the peritoneal cavity. If in the course of a few hours we note a steadily rising pulse, with increasing pallor of the mucous membranes and of the fingernails, and restlessness, and the abdomen still remains quite soft, we may with confidence diagnose intraperitoneal haemorrhage. Apart from trauma the only frequent cause of this condition is rupture of an ectopic gestation, and this is often confused with rupture of a gastric ulcer—in fact, as my practice does not intentionally include gynaecology, all the cases I have been asked to see had been originally so diagnosed. Such an error will, however, never be made if attention be paid to the state of the abdominal muscles—in ruptured ectopic gestation involuntary muscular rigidity is entirely absent, in rupture of a gastric ulcer it is well marked. Moreover, a case of ruptured ectopic pregnancy will usually be found very restless, tossing herself about in a manner never seen in rupture of gastric ulcer, where all efforts are directed towards keeping the abdomen as still as possible. Again, nothing increases the frequency of the pulse so continuously as haemorrhage, and though in the initial shock of a gastric perforation there may be a rapid pulse yet this usually soon subsides, and rises again only as peritonitis develops.

GROUP 4.—*The Inflammations.*

The main characteristics of this group are: (a) The onset is less sudden than in the three groups already considered, and general symptoms of malaise may have preceded the crisis by a few days. The pain never reaches its greatest intensity in the first few moments, as in colic, perforation, and some forms of obstruction, but steadily rises to its maximum within a period which may be as long as several hours. Thus, a patient who, feeling perfectly well one minute, is in violent agony the next, is suffering from some other type of crisis than an abdominal inflammation, and a recollection of this point would have prevented many errors in the diagnosis of acute appendicitis from other crises; (b) this is the only group in which the temperature may be raised in the early stages, but the

absence of any rise of temperature should not be allowed in any way to exclude, and should certainly never be waited for to complete, the diagnosis in a doubtful case; (c) there is detected early on some definite local tenderness (compare very carefully corresponding points on the two sides of the abdomen), from which as a centre the tenderness tends to spread: the tenderness may, as in pelvic appendicitis and salpingitis, be first found per rectum; (d) muscular rigidity of the involuntary type, absent at first, appears only when the parietal peritoneum is affected, and extends concurrently with the peritoneal irritation; (e) a swelling may be definitely palpable in the region of the appendix, the gall bladder, or to one or other side of the uterus in appendicitis, cholecystitis and salpingitis respectively, the three most frequent causes of acute inflammatory crises. This swelling, especially in appendicitis, is not, as is so often thought, the swollen inflamed viscus itself, but is due to the plastic adhesion of the omentum (so aptly termed the "abdominal policeman" by Mr. Rutherford Morison), intestine or other viscera to the viscus primarily inflamed. The diagnosis between acute appendicitis and acute cholecystitis is, in the very acute cases, possible only by attention to (1) their different areas of cutaneous hyperalgesia; (2) the site of the most marked local tenderness and muscular rigidity—a guide by no means infallible, since tenderness and swelling in the right iliac fossa may be due to a gall bladder situated unusually low, while the same under the right costal margin may result from an appendix which has failed to undergo its proper descent, and has remained in the vicinity of the hepatic flexure; (3) some assistance may be obtained from the previous medical history (either of gall stones or previous attacks of appendicitis) and from the greater frequency of appendicitis in the male sex and prior to middle life.

Another frequent confusion I have noted between these two conditions has occurred at a later stage when a well-marked swelling is present, and the question is between an appendicular abscess and an empyema of the gall bladder. With a low liver (Riedel's lobe) a distended gall bladder may be at or even below McBurney's point, and, conversely, with a congenitally high appendix an appendicular abscess may be felt well above it. The diagnosis can, however, usually be made by noting: (1) a distended gall bladder is rarely so fixed by adhesions that it fails to move downwards with inspiration; an appendicular abscess is unaffected by respiratory movements; (2) a distended gall bladder has a rounded edge below, an appendicular abscess has its rounded edge above; (3) there is an area of resonance between an appendicular swelling and the liver, but not so in the case of the gall bladder.

Acute appendicitis and acute salpingitis of the right side constitute another frequent source of diagnostic confusion. The points to be considered in their differentiation are (1) the characteristic order of onset of the symptoms in acute appendicitis, so well described by the late J. B. Murphy, is much less a feature of salpingitis; (2) the initial pain is referred to below the umbilicus in salpingitis; (3) the cutaneous hyperalgesia of salpingitis is most marked at a site well below that of appendicitis; (4) the previous history of vaginal discharge, pelvic troubles, etc., may assist.

GROUP 5.—*The Obstructions.*

It is in this group particularly that improvement in the results of surgical treatment is so urgently required. In the other groups—the perforations, haemorrhages, and inflammations—the results of operative treatment have been markedly improved in recent years, and, although advancements in surgical technique and after-treatment have undoubtedly played a considerable part, yet to the earlier recognition of their existence must the chief credit be awarded, permitting as it does the application of appropriate surgical treatment at an earlier stage than hitherto. Cases of intestinal obstruction, however, unless from some obvious external hernia, are still apt either to pass unrecognized or to be temporized with until the development of the textbook symptom of *faecal vomiting* has declared itself. When, at the great Day of Judgement, we are all called upon to answer for the consequences, immediate and remote, of our actions, a truly terrible indictment awaits him who first described, in a surgical textbook, faecal vomiting as a sign of intestinal obstruction; far rather ought it to be regarded as evidence of rapidly approaching

death; nor should it ever be waited for to complete the diagnosis in a doubtful case. It usually indicates such a profound degree of toxæmia that, unless the cause of the obstruction be an external hernia or some equally readily ascertainable intra-abdominal lesion, the patient is unable to withstand the additional shock of the operation and dies "poisoned." Were as careful attention paid to the early diagnosis of intestinal obstruction as is now expended upon that of the other, perhaps more dramatic, groups of abdominal catastrophes, the present truly appalling mortality of this condition would, I am convinced, be very speedily and very materially reduced.

The chief features characterizing this group are (1) *the persistence of the vomiting*, the early reflex vomiting passing without interruption into that of obstruction; (2) *the cessation of passage of flatus*—if in a case of suspected obstruction flatus fails to pass within a period of twenty-four hours in spite of repeated enemata the diagnosis of intestinal obstruction is established with practical certainty; (3) *progressive abdominal distension*, a most important sign, and always calling for surgical intervention. To wait until distension is general is to render the operation much more difficult, to make inevitable the escape of coils of bowel during its performance—a procedure calculated more than any other to increase the intensity of the shock—besides paralysing the bowels from overstretching and increasing the toxæmia. The absence of any involuntary muscular rigidity and of early localized tenderness are negative points of some value. I have found *auscultation* of the abdomen of great assistance, and with a little practice the sounds produced by the passage of fluid contents along dilated coils and through narrowed portions of gut may be readily recognized. Sometimes there are other signs to help us in special types of obstruction. Thus in intussusception we have the passage of blood and mucus from the rectum (even before this has occurred a finger passed per anum may be covered with blood on withdrawal), and the presence of a tumour of characteristic shape felt somewhere along the course of the colon, or per rectum; this tumour is most in evidence as the peristaltic wave passes along it, and may therefore be palpable only in a spasm of pain, when the voluntary contraction of the abdominal muscles during the child's cries tends to obscure it. However, as already mentioned, there is always, in voluntary rigidity, a momentary relaxation at the end of expiration, and this will allow of the tumour being felt.

Many cases of chronic intestinal obstruction end acutely and may be mistaken for primary acute obstruction; such can, however, usually be recognized by the presence of two features never found unless obstruction has been in existence for some considerable time: (1) The outlines of individual dilated coils may be seen beneath the abdominal wall, giving a "hill and vale" appearance, and (2) peristaltic waves may be observed passing along coils of distended bowel. In the normal state neither peristaltic waves nor the outlines of individual coils of bowel are ever visible through the abdominal walls, though rarely these phenomena may be seen beneath the thinned-out covering of large ventral herniae, in cases of wide divarication of the recti muscles, and also in extreme degrees of emaciation. Even in these instances, however, the coils observed are of normal size and not distended, and are not really exceptions to the rule that whenever we can see clearly the outlines of coils of *distended* intestine, together with a peristaltic wave passing along them, we can safely diagnose chronic obstruction, and the presence of these two features in a case whose symptoms otherwise suggest acute obstruction should lead to a recognition of its primarily chronic nature.

Time will not permit me to consider cases presenting the features of more than one of these five groups—a not uncommon combination being that of inflammation with obstruction—nor of the rarer types of acute crises which do not exactly fall into any of these groups, but perhaps I may be allowed to mention two not very uncommon emergencies of special interest—acute hæmorrhagic pancreatitis and torsion of an ovarian pedicle. *Acute pancreatitis*, though it should strictly be classified with the inflammatory group, has such a sudden and severe onset that it simulates and is usually mistaken for either a perforation of the stomach or duodenum or else an acute obstruction high up in the jejunum. The severity of the symptoms is such as to call for immediate operation, but

the actual diagnosis is not often made prior to laparotomy, when the blood-stained odourless effusion and the presence of minute areas of "fat-necrosis" in the omentum and mesentery at once direct attention to the pancreas. *Torsion of the pedicle of an ovarian tumour* is readily diagnosed if the existence of the tumour is previously known, sudden abdominal pain associated with sudden increase in size of the tumour being quite characteristic; where previously unsuspected the tumour can, as a rule, be felt either through the abdominal wall or per vaginam, at any rate in the early stages, and if of the right side is not uncommonly, in my experience, mistaken for appendicular abscess; but the size of the tumour is too large for an appendicular abscess to have attained in the given time. Later, when peritonitis of the plastic type has set in, the tumour is masked by the overlying muscular rigidity, but as this rigidity relaxes under anaesthesia it is always possible to recognize the condition prior to making the actual abdominal incision.

In conclusion, surgical technique has now reached such a high level of efficiency that further improvement therein cannot be relied upon to better the statistical results of the surgical treatment of acute abdominal crises—such can be obtained only by earlier diagnosis on the part of the general practitioner permitting the application of such treatment at an earlier stage. The time that has elapsed between the onset of the attack and the opening of the abdomen is the real deciding factor in the ultimate issue of the case—far more so than the skill of the individual surgeon. Do not expend too much time in making an exact and elaborate diagnosis: content yourself with recognizing that a grave lesion has occurred which is far more likely to recover if surgically treated. I believe I speak for all surgeons when I say that we infinitely prefer to meet in consultation the practitioner whose diagnosis, *made in the early hours* of the attack, is limited to "something gone seriously wrong in the belly," than he who, *forty-eight hours later*, can give us a cut-and-dried and possibly perfectly correct description of what an operation will reveal.

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A Clinical Lecture

ON

THE STUDY OF HEART DISEASE.*

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I TRUST that your time to-day will not be wasted in listening to the remarks that I am going to make upon such a general subject as the one I have chosen. It can hardly be called a lecture, for it is only an expression of some of the ideas that have passed through my mind during the years I have studied this subject, but I hope, nevertheless, that I may stimulate your interest.

Heart disease is to me the least special of diseases because the cardio-vascular system is essentially a mechanical apparatus for distributing blood throughout the body, and when it is damaged the circulation in important organs suffers, this injuring in turn their functions and producing symptoms which are not strictly cardiac, but yet take a part in the patient's illness, and often an important one. These symptoms may be hepatic or pulmonary, or renal, or alimentary, or nervous, or a combination of them. Thus a wide knowledge of general medicine becomes essential in order to understand, and treat with confidence, such patients. If we focus all our attention on the heart we shall overlook valuable indications of heart disease, and though alive to the changes in the heart itself, be unable to grasp the exact condition of the patient.

It is not to be supposed that the heart must not be carefully studied, but even in doing this we are liable to fall into the error of restricting this part of the investigation to one part—the valves, for example—instead of considering the organs as a whole. We can see this tendency

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