HARVEIAN LECTURES

ON

THE MODE OF DEATH FROM ACUTE
INTESTINAL STRANGULATION AND
CHRONIC INTESTINAL
OBSTRUCTION.*Delivered before the Harveian Society of London.*

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LECTURE III.—ON THE DIFFERENTIAL DIAGNOSIS OF ACUTE
INTESTINAL STRANGULATION AND TYPHLITIS.

In the two lectures it has been my privilege to deliver, attention has been directed to the subjects of acute intestinal strangulation, acute and chronic intussusception, and chronic intestinal obstruction; and I hope I have not been too dogmatic in my observations. My desire has been to separate, as clearly as I could, these three very different pathological as well as clinical conditions; and to demonstrate to you, by pathological facts and clinical data, how patients die from these conditions, in order that I might point out how a fatal result may be warded off or prevented.

I have said little about the difficulties of diagnosis, nor how to avoid them; and yet, of all points, these are the most important; since, I take it, it is on account of the uncertainty of diagnosis that our treatment of these cases has been uncertain, tentative, expectant, unsatisfactory, and (I must add) unsuccessful. In the present lecture, I propose to consider this subject of diagnosis, and to see if any way of deliverance from the difficulty is to be found.

It is unnecessary to dwell upon the symptoms of an acute intestinal strangulation, since a sudden attack of abdominal pain, accompanied with vomiting, attacking a patient previously in apparent good health—the two symptoms being more or less paroxysmal, and associated with obstruction—mark its nature; and they do so, whether or not they are complicated with an external hernial swelling. When the latter condition obtains, a strangulated hernia is readily diagnosed; when no such swelling is present to make the special diagnosis sure, the symptoms are still enough to indicate the existence of some internal strangulation, although its special form may be obscure.

It is true, that a sudden perforation of the intestine from ulceration—an acute attack of peritonitis following some caecal or pericaecal trouble—or an acute attack of gall-stones, may simulate to a degree a case of acute intestinal strangulation; and yet, I think, on investigation, the diagnosis of these two former conditions ought not to be difficult. My observations will consequently chiefly point to these; the third, or gall-stone difficulty, I leave for my medical friends to unravel.

CASE XLIX.—A lady, aged 67, who had previously been perfectly well, was suddenly seized with intense abdominal pain and vomiting; and these symptoms rapidly passed on to collapse and death within twelve hours. After death, a perforating ulcer of the duodenum was found.

CASE L. *Perforating Ulcer of the Duodenum: Peritonitis: Abdomen not distended.*—Charles R., aged 28, was admitted on July 2nd, 1877. Three weeks previously, after some nausea, he had been attacked with vomiting in the morning, and had not been well since. He had often suffered from constipation. On June 30th, after having taken a warm bath, he was seized with severe pain in the abdomen across the epigastric region, and with vomiting. After twenty minutes the pain ceased, but returned in half an hour, and continued until the following afternoon, when it ceased entirely. The bowels had acted on the day before he had been taken ill. The vomiting, of greenish material, had since been persistent. On July 2nd, two days after the commencement of symptoms, when he was admitted, he lay on the left side, with his legs drawn up. The eyes were sunken and surrounded by a dark areola. The hands were cold. The pulse was not to be counted at the wrist. The abdomen was scarcely, if at all, distended. Temperature 100.4° F. Nutrient enemata were given, and a grain of opium every four hours. The bowels acted repeatedly. Next morning, the man declared himself better; but the collapse increased, and he died at 2 p.m. on that day, July 3rd. At the necropsy (239), acute

peritonitis existed. Gas bubbled up in separating the intestines from the liver. It came from a perforating ulcer in the pylorus and first part of the duodenum. The ulcer looked as if it had existed for a long time.

In these two cases are embodied the ordinary symptoms of an acute and a subacute case of duodenal perforation. In both there were the sudden central abdominal pain, accompanied with vomiting. In the acute case with these symptoms there was collapse, from which the patient hardly rallied. In the subacute case, collapse did not come on till the second day, when it was marked and persistent. In the one case obstruction was hardly a symptom, since there was no time to show it. In the second it did not exist, since the bowels acted freely and repeatedly. In the subacute case it was to be noticed also that there was no abdominal distention.

In both cases, therefore, the first symptoms were those of acute strangulation; but in the acutest, the collapse was more rapid and persistent than is usually met with in strangulated bowel, and in the second case it was equally decided.

In strangulated intestine, however acute it may be, I should like to remind you that collapse is not an early or marked symptom in the generality of cases, although it may be such in very exceptional examples. I have known it occur in an acute strangulated hernia of the congenital kind in a young man. In a general way it may, however, be said that collapse is not an early symptom of strangulation; it comes on slowly, with the pathological changes of the strangulated gut, and, when present, denotes a condition of strangulated bowel, which means its death.

The collapse of perforation is sudden, and most like that of cholera; the sunken hollow eye, the deep far-away voice, feeble pulse, clammy congested skin, all denoting death. The collapse of strangulation is slower in its onset, and slower in its progress, and rarely so marked and death-like, till death of the patient, with death of the strangulated bowel, is at hand. If I may, therefore, roughly express it, the symptoms of perforation are more acute than the acutest case of strangulation. Collapse is an early symptom, and more persistent; intestinal obstruction need not be present. "A perforating ulcer," wrote my lamented friend, Hilton Fagge, "of any part of the direct channel of the alimentary canal is commonly fatal in a few hours, or in a day or two at the latest. Hence, I believe, that, when peritonitis runs a more protracted course than this, there is a very strong presumption that it started from the caecal appendix." (*Guy's Hospital Reports*, vol. xx, third series, 1876, page 221.) These points, I believe, are likely to be of value for diagnostic purposes.

I propose now to pass on to a much larger class of cases—to a class which, it may be said, simulates cases of acute strangulation more than any others; I mean cases of perforating ulcer of the caecum or its appendix, for I have reason to believe that both of these occurrences are met with in practice.

"Perityphlitis, or, more properly, typhlitis or caecitis," wrote Hilton Fagge (*Guy's Hospital Reports*, vol. xx, 3rd series, 1876), "is a process of ulceration, which almost always begins in the mucous, and spreads to the serous surface. Sometimes, this is accompanied with sloughing of the little tube, or of its extremity. The ulceration is generally set up by the presence of a concretion, which may vary in size from a pea to a plum-stone. This sometimes consists of a substance resembling wax, but much more frequently it is composed of hard dry faecal matter, mixed with mucus and with earthy salts. Such concretions may look very like cherry or other fruit-stones, and have often been mistaken for them. Indeed, this mistake has been so frequently committed, that many pathologists are disposed to doubt whether ulceration of the caecal appendix is ever set up by foreign bodies of the kind just mentioned. It is, however, certain that seeds, pills, bristles, pieces of bone, shot, etc., have been found in the appendix. On the other hand, ulcers of this part of the bowel, penetrating to its serous coat, have sometimes been tuberculous."

Chronic caecitis, or typhlitis, is not likely to be mistaken for intestinal strangulation, unless it be complicated with an attack of acute peritonitis, the result of the bursting of an abscess or perforation; and, under these circumstances, the previous history of the case will throw light upon existing symptoms. I have before me the notes of nine fatal cases of caecitis, and propose to give you abstracts of them, and, later on, an analysis of the symptoms.

CASE LI.—Richard O'R., aged 18, a labourer in chemical works, was admitted on March 31st, 1880, under my care. A month previously, he had been suddenly seized with colic, the pains being above his right groin, and intermittent. After the free use of castor-oil, he was relieved, but a pain down the front and inner side of his thigh was left. In two weeks, his thigh began to flex upon his pelvis, and the knee to be everted. He could not, moreover, straighten the limb,

and any attempt to do so excited severe pain. When admitted, the boy looked ill. His right thigh was flexed up to a right angle upon the pelvis, and everted. It could be more flexed without pain, but it could not be extended without giving rise to agony. There was a great hard swelling in the right iliac fossa, and swelling beneath Poupart's ligament, and down the inner side of the thigh. The patient's bowels were regular, and motions natural. It should be added that the case had been sent into Guy's Hospital as one of hip-disease. In a few days, the swelling in the thigh became larger and resonant. It was, therefore, freely opened, when pus, gas, and feces escaped. Two incisions were made, one in the thigh, and the other within and above the anterior superior spinous process of the ilium backwards, parallel with its crest. The above cavity was well washed out with iodine-water, and drained. The patient was relieved by this measure, but died a month later from hæmorrhage, due to ulceration of the deep circumflex iliac artery. At the necropsy (156), a large sloughing cavity was entered on passing the finger through the wound beneath Poupart's ligament. The cæcum was felt with an opening into it. On injecting the vessels, it was found that the deep circumflex iliac artery, which lay in the posterior wall of the abscess, was completely divided by ulceration. A clot existed at its free end. Above the ileo-cæcal valve in the ileum were a few ulcers, not unlike healing typhoid ulcers. A large ulcer had perforated the valve, leaving an opening large enough to admit the finger. In the cæcum were other ulcers, one of which had perforated, and led into the abscess-cavity. The iliac abscess had burrowed in the course of the psoas, and had travelled between the muscles down the thigh. The hip-joint was bathed with pus, both internally and externally; the cartilages were deeply stained.

REMARKS.—This case is full of interest; first, in the attack of so-called colic, a month before the boy's fatal illness, when the pain was above his right groin; secondly, in the fact that, when the pain of colic was relieved, a pain passing down the thigh, in the course of the anterior crural nerve was left; thirdly, in the gradual flexion of the thigh upon the pelvis, and its rotation outwards with inability to extend the limb; fourthly, in the formation of an abscess extending beneath Poupart's ligament down the inner side of the thigh, and the presence of flatus in the abscess; fifthly, in the rapid relief which followed free incisions; and lastly, in the purely accidental ulceration of the artery which led to sudden death. Had this mischance not ensued, in all probability a good result would have had to be recorded.

Some ten years ago I saw, with Dr. Smith, of Highbury, a precisely similar case to this, although it was in an older man. In it, the abscess, which was resonant (as in the former case), not only extended downwards, but also upwards into the right loin. I treated it with free incisions, irrigation, and drainage, and the man quite recovered.

CASE LII. Ulceration of the Cæcal Appendix: Concretion: Suppurative Peritonitis.—Edward W., aged 37, was admitted on January 2nd, and died on the 5th, 1874. Having been quite well five weeks beforehand, he had strained himself when at work, and could not afterwards flex the right thigh. He also felt a lump above Poupart's ligament. His present illness began fourteen days before admission, with pain in the hypogastric region, not aggravated by pressure. On December 29th he went to bed as usual, and woke with a distinct sensation of movement in the right iliac region, and with pain. He vomited also, and was purged until January 2nd, when the vomit became stercoraceous; and in this condition he was admitted, with the abdomen distended. A tumour could be felt in the right iliac fossa. There was no hernia. The rectum and the bladder were normal. The urine was albuminous, and full of lithates, of specific gravity 1036. The man died on January 5th.

At the *post mortem* inspection (3) the intestines were found matted together by peritonitis. The appendix cæci was of port-wine colour, and ulcerated at the apex, with a cavity about it which contained concretions.

REMARKS.—This case well follows the last; since in it, as in the other, the pain was first felt in the right groin above Poupart's ligament, and with it there was some pain in flexing the right thigh. It is to be noticed also that with the acute symptoms there was purging, not obstruction. In fact, in the light of present knowledge, all the evidence pointed to cæcal trouble.

(To be continued.)

WEST KENT MEDICO-CHIRURGICAL SOCIETY.—The third meeting of the session was held at the Royal Kent Dispensary, Greenwich Road, on Friday, December 5th, at 8 P.M. Dr. Horrocks, of Guy's Hospital, read a paper on Inflammation of the Cervix Uteri.

THE PUERPERAL FEVERS.

Read at the Opening Meeting of the Eastern District of the Metropolitan Counties Branch, October 16th.

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WHETHER there be a special puerperal fever, a fever *per se*, ranked with small-pox, scarlatina, typhoid, rubeola, or erysipelas, is a question still keenly debated. This question I propose to examine by the light of physiological study of gestation and puerperity, and the clinical observation of the disorders that characterise these two states.

We may clear the ground of one source of confusion by admitting that, if we postulate a fever to rank with scarlatina and the other zymotic fevers, then the existence of a special puerperal fever is not proved. But this is a very incomplete and unsatisfactory answer. Women die in childbed, and die in greater numbers than from any other cause, with fever of some kind. Therefore, we must enlarge the question, and ask of what nature is the fever or fevers which assail lying-in women.

Clinical observation will teach us that there is not one puerperal fever, but several. Like Meigs and Arthur Farre, we must use the plural number, and give up the attempt to describe a puerperal fever. The task before us is only to be accomplished by a process of analysis. Thus, to set the problem as it comes before us at the bedside: On the third day after labour we find our patient exhibiting the common signs of fever. She has had shivering, perhaps. The pulse and temperature are over 100; the respiration is accelerated; there is, in short, if not puerperal fever, fever in a puerpera. Thus, by a transposition, or change of terms, we are in the presence of, not a defined specific fever, as the term puerperal fever would imply, but of an indefinite fever. And now our task is to find out what is the nature of this fever. This is to be done by a process of analysis or differentiation, calling to our aid the light that history and close observation of the process of the disease may throw upon the case.

In the first place, we may state two undeniable propositions. 1. The fever implies poison, that is, toxæmia. 2. The poison may have sprung up in the patient's own system, or it may have been taken up from an external source. This great distinction is the first step in the process of differentiation or analysis. I long ago emphasised this division of fevers in the puerpera, by calling those which spring up in the patient's own system, *autogenous*; and those which were due to empoisonment from without, *heterogenous*. These terms have become classical, but their full meaning is not even yet appreciated.

Let us first examine the nature and production of autogenous fevers. These lie at the very foundation of the whole question.

But we must start with a fundamental proposition which distinguishes the diseases of the gravida from the diseases of the puerpera.

The diseases of the gravida are diseases of high nervous and high vascular tension. The diseases of the puerpera are diseases of low nervous and low vascular tension. In the gravida, the balance of osmosis is centrifugal; in the puerpera, it is centripetal. The diseases of the gravida arise out of natural conditions exaggerated in intensity. Thus, the most striking examples of high tension in excess are seen in the albuminuria and eclampsia, and the hæmorrhages of pregnancy. An active process of building is going on, and everything is made subservient to this work. But the moment this work is complete, the reverse process of demolition, and of carrying away the refuse, is begun. Absorption and excretion then are the ruling energies. Of course, an active absorption, commensurate with and subservient to the process of construction, goes on during gestation. But this is a very different thing from the absorption of the refuse stuff which, having served its purpose, has to be cast out from the body. This refuse-stuff, if not duly excreted, may be as poisonous as are the elements of urine if not duly excreted. Hence we see that, in pregnancy, thrombosis, phlegmasia dolens, and septicæmia are extremely rare, whereas, during the puerperal state, they are common.

We might add that the conditions which mark the diseases of the gravida also distinguish them from the diseases of all other persons.

Now we will briefly trace the puerperal process, which, if disturbed, may lead to autogenous fever. The following are the factors to be taken into account.

1. There is the blood of the gravida modified by the processes of construction and nutrition of the embryo and uterus. They are: excess of fibrin, diminished proportion of red corpuscles, increased pro-