A CLINICAL LECTURE ON ACUTE INFLAMMATION OF THE CÆCUM AND ITS APPENDIX.

Delivered in the Richmond Hospital, Dublin.

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So much has been written about typhlitis in connection with modern developments of abdominal surgery, and so much said as to the exact diagnosis and the treatment of the various inflammations which attack the cæcum, its appendix, and the tissues about them, that I desire to clear the ground for myself and others who may sympathise with my difficulty in accepting all that has been put forward in this context. The question of exact diagnosis is that which puzzles me most, and I must confess to inability to follow or apply in general the nice symptomatic distinctions which some writers draw between inflammation of the cæcum, of its appendix, of the areolar tissue behind it, or of the peritoneum which covers it. If localisations so exact exist—and I doubt their frequency—their discovery is more likely to be made on the operating table or in the *post-mortem* room than in the bed, and they are therefore of little help to the surgeon in guiding him as to the treatment he should adopt.

The cases to which I propose to refer are those of acute inflammation. What I have to say does not apply to relapsing inflammations, which present acute recurrences with chronic intervals. With them we have a more educated acquaintance; we recognise their clinical features; during their quiescent intervals we can often determine their exact nature by physical examination, and we have now attained definite views as to their proper treatment. I only mention them for the purpose of excluding them; they almost invariably have their origin in a diseased appendix, and usually demand removal of that organ during a chronic interval. It is because they always first bring themselves into prominence by an acute attack, and because operative treatment during that attack is so unfavourable when compared with laparotomy during a quiescent interval, that I desire to formulate my experience concerning the nature and treatment of acute attacks of typhlitis, be they primary or secondary. Of these relapsing inflammations we may, for the present purpose, dispose with the remarks:—

a. That they generally demand laparotomy, usually for the purpose of removing a diseased appendix.

b. That the operation, if done during an interval of freedom from acute disease, is seldom dangerous or unsuccessful.

The knowledge of these facts makes us more anxious to be able to overcome initial attacks of typhlitis by minor treatment, on account of the grave danger of operation at such a period, and of the frequency with which a first attack proves the only attack. The same reluctance to operate should obtain during subsequent acute relapses, in view of the confidence with which we may open the peritoneum when they have subsided.

If I use the term "typhlitis," I do so for want of a better, and not because it is in any way exact. I mean it to comprehend inflammation in or about the right iliac fossa of the varieties variously named cæcitis or typhlitis, perityphlitis, paratyphlitis, and appendicitis. That an acute attack of one of these may occur without the other is evident, but the persistence of one of them without the other is a rare incident, and one not usually open to exact diagnosis.

I have seen a very large number of cases of acute primary typhlitis, and in nearly all of them have found it impossible to make an exact diagnosis as to the seat of the lesion, and in spite of the much writing which should have made us exact, have found myself lamentably behind the numerous

arbitrary writers on this subject. To be sure, it is generally safe to accuse the appendix in these cases, because it presents the most characteristic symptoms and signs, because it undoubtedly is usually the seat of the initial inflammation (for reasons to be estimated hereafter), and—less worthy reason—because by the time operation or the progress of disease has rendered matters more evident, the enterprising and exact opinion will be justified by finding it in a state of inflammation which, if not primary, cannot be proved to be secondary. In other words, when it is, exceptionally, not the seat of the initial disorder it is almost sure to be attacked subsequently.

In a most instructive paper by Mr. Treves,¹ he deals with the influence of the bacterium coli commune in producing typhlitis, and the circumstances under which it assumes the abnormal virulence which enables an otherwise harmless inhabitant of our intestines to become so destructive. With what he says on that branch of the subject it is not possible to disagree; it is logical and practical; but when seeking for the circumstances under which the appendix becomes the seat of recurrences he hardly goes far enough. It is not necessary to assume deformity or abnormality to explain why with, or without, the assistance of this bacillus the appendix becomes so frequently the seat of inflammation. If it be conceded that a foul or constipated large intestine predisposes to the virulent development of this micro-organism, it must be allowed, *a fortiori*, that the narrowest, most obscure, and most unused by-way of the bowel is the likeliest breeding place for mischief; also, the frequency with which appendicitis appears only once and does not relapse is a strong argument in favour of the view that the normal appendix is generally that affected. There is one passage in Mr. Treves's paper which appeals most strongly to experience; it is that in which he remarks that the number of cases in which there has been only one attack is much greater than that in which there have been recurrences. This is a fact that will speak loudly to those who have seen a large number of cases of typhlitis, and it is at once an argument against the ready adoption of operations in first attacks, and against the probability of a deformity of the appendix being present. Recurrences are rare in children, and of greater frequency in vigorous adult life.

Observation has taught me that operation in acute typhlitis is highly fatal, and should only be resorted to in very exceptional cases. Further, certain lines of minor treatment are often successful, at all events in tiding over the immediate danger, and usually in producing permanent relief. Once we allow that persons who suffer from single attacks are much more numerous than those who are subject to relapses, we have found the strongest reason, in the face of the fatality of laparotomy in the acute stage, for using every other reasonable means. I have rarely seen a case of acute typhlitis in which the large bowel was not full of old fæces, and I have still more rarely seen a case in which the colon could be unloaded in which recovery did not take place. So true is this, that of late years I have nearly always regarded the emptying of the colon as ending the immediate danger to life, provided of course that the inflammation has not proceeded to the point where an abscess has formed or perforation taken place. The question as to when laparotomy should be done in acute typhlitis is one that is often difficult to answer. To be able to formulate an accurate reply to it would be to have reduced this branch of surgery to an exact science, and to be able to set aside the advantage which experience gives. So far as I am able to state my opinion on the matter, it is that if an abscess has formed or perforation taken place, laparotomy should at once be resorted to, but that, short of these misfortunes, operation should be delayed until the last possible moment. Of course there are exceptions to such a rule, but they are only to be learned by clinical study, and cannot well be conveyed by words.

We are now at the top of an epidemic wave of operative treatment of inflammatory disease of the cæcum. It has risen too high, and like other disturbances, will presently subside to a mean level. In 1889 I ventured to express strong opinions against the propriety of usually performing laparotomy in acute intestinal obstruction due to collections

¹ BRITISH MEDICAL JOURNAL, March 9th, 1895.

within the lumen of the bowel.² This opinion I have since advocated still more forcibly,³ and experience and observation of the practice of others have led me to form an equally strong judgment against the general wisdom of operating in cases of acute typhlitis, except under the conditions just expressed, or where other means have failed. These cases are of common occurrence in all classes, and at most ages except very advanced ones. There is a pleasant poetic justice in the reward of our surgical anxieties, implied by the fact, as I have observed it, that acute typhlitis is one of those complaints commoner among patients of the better class than in those in poorer circumstances. It is more frequently met with in private than in hospital practice. This is perhaps due to the high feeding and constipation which are more usual among well-to-do people than among poor ones.

Putting aside the ultimate question of operation, it is to be considered what measures are at our disposal to empty the bowel, and so place it in as strong a position as possible to resist the poisonous effects of the colon bacillus. It is so generally the case that the inflammatory process is initiated and kept alive by fecal accumulations, and that if it has not proceeded to suppuration or perforation it will be arrested if the bowel can be emptied, that too much weight cannot be attached to the means of effecting this. There are two lines of treatment to be considered—the negative and the positive, the first embracing things that are to be avoided, the second things that are to be done. Under the former head may be grouped as both improper and in common use: (1) the local use of irritants; (2) the employment of opium; (3) the abuse of feeding; (4) reliance on the value of temperatures.

1. Local applications capable of causing any irritation are to be avoided. Anodyne or other liniments, too hot poultices, or any treatment likely to inflame the skin, ought not to be used; such measures have no good effect on the bowel, they obscure symptoms by rendering the skin tender, and should operation be unfortunately called for it is not done to the best advantage through an inflamed integument and areolar tissue.

2. Opium should be used most cautiously, if employed at all. The number of acute bowel cases in which it is pushed to such a point as to conceal symptoms is large, and most surgeons can record abundant instances in which patients have been regarded as not alarmingly ill because they have been kept narcotised until their condition was desperate. The only indication for its use is pain, and by concealing this it may keep the surgeon in a fool's paradise.

3. The abuse of feeding is a constant danger. In their anxiety "to keep up the patient's strength," solicitous friends give food to an embarrassing extent. It is difficult to make them understand that even fluids, if taken freely, can increase the loading of the disordered bowel. The quantity of material which can be introduced into the intestine by steady and persistent administration of repeated small quantities is astonishing. If vomiting be present the mistake is even greater, for the patient becomes exhausted by it and by the failure to assimilate even the small quantity of nourishment he otherwise could. Of all food, that which I most dread is milk. The popular ignorance of the fact that it curdles when introduced into the stomach, and that it is not easily digested by persons suffering from intestinal obstruction, is productive of infinite harm. It is not only dangerous because its good reputation induces its administration in larger quantities, but also because the curds are quite indigestible in these cases, and the use of milk adds to the solid load in the bowel. I have over and over again seen acute typhlitis stopped by a judicious process of starvation. This is particularly true in the case of children, and singularly difficult to carry out in them owing to the anxious prejudices of mothers.

4. The value of temperatures as a diagnostic aid in all disturbances involving the peritoneum is slight. They are very deceptive, and any undue reliance on them will lead to trouble. They may range high in insignificant cases, and are even more likely to be very low in grave examples of disease.

So much for things to be avoided. What of more positive treatment? This may be arranged under the heads of (1) diet. (2) local applications, (3) purgatives, (4) enemas.

1. The food should be fluid, and of as small a quantity as will sustain the patient. If vomiting be present it ought to be reduced to the lowest possible point. Rectal alimentation is impracticable owing to the necessity of inducing the bowel to act. The choice lies between meat juice, beef-tea, whey, water, and some form of alcohol. The distressing thirst which exists in these cases is most difficult to overcome. If it be sought to relieve it by introducing much fluid into the stomach this provokes vomiting. Ice increases the thirst, and by insidiously introducing water into the stomach tends to induce vomiting. Frequent rinsing of the mouth with hot water, and swallowing occasionally a teaspoonful of warm water, are the best means at our command to allay thirst.

2. The only local application of much value is a soft linseed meal poultice, not so hot as to redden the skin. It should not, as has lately been pointed out, be covered with waterproof material, which by preventing evaporation lessens its cooling qualities. I have never seen any other local measure of use.

3. Purgatives, if cautiously used, are not to be dreaded, except in cases of great acuteness and intensity. If there be vomiting they must be administered by the rectum, but in that event they are of less value. The sulphate of soda is perhaps the best, and if a purgative by the mouth be permissible, I usually order it in 2-drachm doses, repeated every hour until an effect has been produced or four doses taken. But purgatives should be avoided unless the enema has failed, or until it has commenced to be effective, or except in cases where complete obstruction does not exist in the beginning of the attack.

4. Of all means at our command, the enema, if properly employed, is the safest and best agent for the relief of a loaded large intestine. The method of employing it which I now invariably use is that described in the *BRITISH MEDICAL JOURNAL* of January 26th in the present year. To the enterprising operating surgeon it has one objection—it is extremely slow. I have often spent two hours at the bedside of a patient suffering from impacted large intestine, with or without typhlitis, before fecal matter began to come away. But when it does begin to come away the corner is generally turned. If the process is tedious to the surgeon, it is, at all events, not exhausting to the patient. There is nothing of which I have become more thoroughly convinced than that nearly all cases of acute typhlitis have their origin in a loaded colon, except the fact that if that colon can be unloaded in time, recovery will take place. The process of fecal impaction of the large intestine begins in the sigmoid flexure; it gradually extends in a proximal direction, first involving the descending colon, then the transverse colon, and, lastly, the ascending bowel and cæcum. The time at which the proximal accumulation may set up inflammation varies greatly. The hostile effects of the bacillus may show themselves in some patients who have accumulation not nearly reaching to the cæcum; in others it is not evident until the caput coli itself is involved. But this in no way upsets the theory of fecal accumulation as an exciting cause of typhlitis. It is merely an assertion of what every pathologist knows—that some people and some bowels are more susceptible to morbid influences than others. If it once be conceded (and can it be denied?) that the colon bacillus is rendered hostile by constipation, it seems evident that purgation is, if achieved early enough, likely to be a remedy for its ill effects. If, as I believe, the bowel can best be emptied by a process of washing—a process perhaps laborious, but almost certain in its result—then it has a claim for careful employment. Beyond the theory of the matter is the practical fact, which I trust will obtain acknowledgment, that purgation, if obtained before perforation has occurred or an abscess formed, is generally the end of immediate danger in typhlitis.

I do not produce a list of cases in illustration of my views, although my observations have been numerous and extended over many years, because conclusions, and not isolated instances, are what we want in matters of debate. My conclusions, very anxiously arrived at, are:

1. That in this, as in some other acute peritoneal inflammations, operation is most unfavourable, should be seldom resorted to, and has been too freely adopted; and

2. That purgation, if it can be induced, is the best remedy

² *Trans. Acad. Med. Ireland*, 1889.

³ *BRITISH MEDICAL JOURNAL*, January 26th, 1895.

at our disposal; and that the most likely and safest way to effect it is by hydrostatic washing with warm water and a soft tube.

MORPHEA HERPETIFORMIS:

A NEUROLOGICAL STUDY.

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(WITH A COLOURED PLATE.)

THE coloured plate which forms the principal illustration of my paper represents the condition of the foot in Mrs. B., a woman, aged 63, who was the subject of morphea. On the outer side of her foot and ankle, it will be seen that a large, irregularly-shaped patch of skin has become hardened, and assumed a tint of yellow ivory or smooth washleather. To the finger the affected skin felt board-like, and it was quite impossible to pinch it or lift it from the subjacent parts. A certain amount of contraction was present by which the three outer toes were drawn upwards, and the movements of the ankle were much impeded. At the margin of the affected part the skin was a little congested, and on the side of the leg, about 4 inches above the outer malleolus, there was an ulcer as large as a half-crown. These conditions had been present about nine months when the portrait was taken on July 11th, 1893. The woman has remained under my observation since, and I am now, nearly two years later, able to report that the ulcer has healed, and that the whiteness and induration of the skin have to a very large extent disappeared. There remains, however, a tight, almost scar-like, condition of the affected skin, which is of a dusky colour, and adherent to the parts beneath.

We are in a position in this case to trace the progress of an attack of herpetiform morphea from beginning to end. The patient in October, 1892, first noticed "a reddish ring" on the dorsum of the foot, just above the base of the little toe. Others near to it soon followed, and in the course of about two months the patch was well mapped out. After that there was no lateral extension, but the ulceration did not take place until three or four months later. The morbid processes had attained their height in about eight months, and during the two years which have subsequently elapsed they have been slowly subsiding. The patient has been in fair health throughout, and has had no similar patches elsewhere.

I have now to ask attention to the interesting fact that the portion of skin involved is exactly that supplied by the terminal twigs of the short saphena nerve. This nerve, made up of the two communicating branches from the popliteal and peroneal, passes down the lower half of the back of the calf subcutaneously, and is distributed to the skin on the outer side of the ankle and foot, as far as the roots of the toes. That the outlines of the patch are not much suggestive of nerve distribution is to be freely admitted. They are irregular, and in some parts run almost in straight lines, and the upper boundary is almost square. In explanation of these peculiarities something may be said in a further part of the paper another time.

I have next to ask attention to particulars of the case which the woodcuts on p. 1196 concern. This case was one in some respects exactly similar to that just related, but it went beyond it in several most important features. Both feet were affected, and with almost exact symmetry, and although on the outer sides of the feet the distribution was precisely like that in Mrs. B., that, namely, of the lesser saphena nerve, there were patches on the inner side of the feet as well. These latter could only be explained by supposing that certain twigs from the popliteal, in addition to that which it supplies as its communicans branch, were involved. There is nothing improbable in this supposition, and we may note that the affected parts were as definitely limited as in Mrs. B.'s case, and that in neither was there the slightest tendency to extension of the patches after their limits had been once well declared. The subject of this second case was

a drayman, aged 32, residing in Liverpool. He was under the care of Dr. Foulston and Dr. Hugh R. Jones, the latter of whom had drawings made which he was kind enough to allow me to copy. He also procured me an opportunity of seeing the patient, for myself, during a visit to Liverpool. The disease had existed eighteen months at the time the drawing was taken, and the patient described it as having commenced by "a speckling of scaly white spots on the skin," which gradually coalesced into white, hard patches. The patches had been present fifteen months before the ulceration occurred. As in the preceding case, the affected portions of skin were somewhat depressed by contraction. Excepting "a little springing and tickling," no abnormal sensations were complained of, and there was no appreciable loss of sensibility. The man was in good health and had no skin disease elsewhere. He remained under observation for some considerable time, but Dr. Jones tells me he has now been lost sight of. It is believed that his disease was undergoing the same retrogressive changes which were noted in the preceding case.

The above two cases illustrate in brief the most important facts which have been established respecting what has hitherto been known as the ivory-patch morphea. They both show that it has slow stages, alike of development and decline; that it is abruptly and definitely limited to the parts affected and has no tendency to serpiginous spreading; that, after lasting for a certain time, the affected parts are restored to a more or less healthy condition. The first case shows us the disease in its unsymmetrical or definitely unilateral form, whilst in the second changes of a precisely similar character were arranged with almost accurate symmetry.¹

It is more than twenty years since I gave a special lecture² at the Blackfriars Hospital on some of the points of resemblance between herpes and morphea. At that time the connection of morphea and the nervous system had been scarcely if at all recognised. I pointed out that the distribution of the patches was in many cases exactly like that of herpes zoster, and ventured the theory that, as herpes zoster is an example of an inflammation of the skin located by sensory nerves, so morphea is an instance of changes of a more chronic kind and of a very different character, located either by the same nerves, or perhaps more probably by the vasomotor filaments which accompany them. My friend the late Dr. Hilton Fagge, one of the best observers who ever lived, amongst others, expressed some dissent from this view, and I remember that he was good enough to send me a case in which the ivory patches were arranged almost as a circle below the knees, and appeared to be definitely influenced by the pressure of the woman's garters. One of the arguments which I strongly urged in reference to the parallelism between zoster and morphea was that the latter, like the former, was rarely arranged with bilateral symmetry, and next that the patches of neither the one nor the other, after having once well declared themselves, show any tendency to enlarge their borders or to spread serpiginously. To these two statements many of those who have written on morphea recently have more or less demurred. I have been shown repeatedly by my friends cases which were considered to be symmetrical, and others in which the patches were alleged to have gone on increasing in size. In spite, however, of these apparent or supposed exceptions, I believe that the majority of derma-

¹ I am obliged to speak with a little doubt on this point, for, although in the Liverpool case both feet were affected, I possess drawings and detailed descriptions of only one. The other is stated to have been like it. The late Professor Rasmussen, of Copenhagen, was I believe the first to describe the microscopic appearances in morphea. He was in England in 1862, and I had the pleasure of his company for a day in the country. We spoke much of morphea, but for some time, because I asserted that I had seen six cases, I could not convince him that we meant the same malady, for morphea was, he alleged, so rare that "no physician had seen more than one example of it." Since then I have no doubt that I have seen forty cases and, thanks to the organisation of special departments at our hospitals and the multiplication of societies for clinical observation, there are now others whose familiarity with the disease may equal my own.

² I cannot find that this lecture was ever published even in abstract, nor can I assign its exact date. It had for its title "On the Parallelism between Herpes Zoster and Morphea." A letter from Dr. Hilton Fagge on the subject bears date July 13th, 1867. In 1879 I published in *Lectures on Rare Skin Diseases*, a summary on the same subject, and at page 347 the reader will find a reference to what had previously been written as regards the connection between morphea and the nervous system.