This conception of the central nervous system is an unusual one, but provides a working hypothesis which appears of value from a clinical point of view. It exalts the importance of the reflex centres, the really vital portions of the central nervous system. The cerebrum, and the same is true of the cerebellum, is looked upon as a distinct organ which can receive and initiate impulses, but these impulses are sorted and to a large extent co-ordinated in the lower reflex centres. The thesis, pushed to its logical conclusions, involves one in seeming absurdity. Voluntary muscular action becomes in this sense a reflex. Initiated in the cortex cerebri, it passes as an afferent impulse to some part of the reflex centre, and is there translated into efferent impulses which result in co-ordinated movement.

Voluntary muscular movement cannot be classified as a reflex in the restricted physiological sense of the term, and it might be better to employ some other term to denote the conception the wider view requires. It is difficult to find a term which expresses the general meaning so well as that ordinarily implied by "reflex," and it is at least open to question whether one cannot justifiably extend its scope to include the greater part of the mechanism whereby voluntary muscular contraction is produced. Mental processes can initiate visceral reflexes. Recollections of various kinds, mental excitement, anger, shame, and other conditions provide impulses which provoke widespread activity of the reflex centres. The resulting cardiac, vasomotor, and other visceral phenomena are undoubtedly reflex, and why should not the simultaneous contraction of voluntary muscle be included in the same category? It is true that the contractions of striped muscle can be inhibited by the will, while there is no such control over the visceral responses; but this power of inhibition is largely acquired by education and is not always complete. Many voluntary actions have much in common with the reflex. The proper swing of a golf club, and in fact skilled movements generally, show many of the characteristics of reflex action, and it is possible that they actually are reflexes. It is, indeed, in many cases difficult to determine when an action ceases to be voluntary and becomes a reflex.

There is ample reason to believe that voluntary movement and the reflex action of voluntary muscles do in many instances make use of essentially the same mechanism, and this is all that the St. Andrews view of the reflex requires. Whether all voluntary action is to be regarded as reflex involves serious problems with which we are not concerned.

The cerebrum and cerebellum occupy a very special position. While capable of acting as effectors and as receptors in the reflex arc they have their own distinctive functions, and, moreover, they exercise a control over the lower reflex centres. The control exercised by the cerebrum is mainly that of inhibition, that of the cerebellum is a reinforcement of the activity of the reflex centres. The response of the body to a given stimulus is greatly complicated by the controlling influence of these organs and the same influences complicate the altered reflexes brought about by the action of injurious agents in disease.

Reflexes are frequently grouped in pairs, the one being antagonistic to the other. Examples are seen in the voluntary muscles which are grouped to form opposing forces: the heart has accelerator and inhibitor nerve fibres, the blood vessels vaso-constrictor and vaso-dilator fibres, and so on. Opposing reflexes are nicely balanced one against the other, but the balance is in some cases influenced by this third factor acting upon the centre—namely, a control by the cerebrum and cerebellum. The influence of the cerebellum is mainly concerned with the postural reflexes, and the importance of the reflex in the maintenance of posture is well known. In a recent article Blundell Bankart² has given an excellent description of the postural deformities which arise from alterations in the normal reflexes. Slight postural changes may be early indications of disease, and their production is the result of a disturbance of balance of certain reflexes. Temporary alterations may be physiological, as in fatigue, but they may be pathological, and if the conditions persist actual structural deformity may ensue.

Head,³ in a Croonian lecture upon diseases of the nervous system, points out that the manifestations of disease comprise both loss of function and outbursts of

excessive activity. He ascribes these phenomena to a "release of function," and divides them into categories entitled "disintegration" and "escape from control." It may be true that, as Hughlings Jackson taught, "destructive lesions never cause positive effects, but induce a negative condition which permits positive symptoms to appear." By the impairment or destruction of one reflex arc a negative condition is produced, the normal balance of the reflexes is disturbed, and positive symptoms arise from over-action of other reflexes which are no longer held in check. The same results are shown even more strikingly outside the central nervous system. If the activity of the vagus nerve is impaired by the use of atropine the heart beats more rapidly. The heart is released from the function of vagus control, but the real interpretation of the condition lies in a disturbance of the reflexes controlling the organ, impairment of vagus activity resulting in over-action of the accelerator mechanism. Many examples of a similar nature might be given. The term "release of function" may be more appropriate to some of the conditions described by Dr. Head, but the principle underlying them is the same, and Sir James Mackenzie's theory of disturbed reflexes provides a working hypothesis of wider application.

The theory is being tested at the St. Andrews Clinical Institute, and is found to be of great value in the elucidation of the early symptoms of disease. It enables the drawing up of a rational classification of symptoms, and gives a clue to their relative importance.

Incidentally it has revealed to members of the institute, and to others who have attended the clinics, the necessity of a closer correlation between the teaching of anatomy and physiology. Both subjects form the basis of any rational conception of the problems which have to be solved in medicine, and a thorough grounding in the anatomy and physiology of the mechanism of the living body is the first essential of medical education.

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- ¹ Mackenzie, *BRITISH MEDICAL JOURNAL*, January 23rd, 1921, p. 147.
² Blundell Bankart, *BRITISH MEDICAL JOURNAL*, April 25rd, 1921, p. 587,
and April 30th, 1921, p. 636. ³ Head, *Proc. Roy. Soc., B*, vol. 92, p. 184,
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A British Medical Association Lecture

ON

SOME POINTS IN ABDOMINAL DIAGNOSIS.*

BY

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WE all know the admirable lists of physical signs and symptoms carefully compiled in textbooks, the perusal of which makes differential diagnosis appear so straightforward and almost infallible, but we know, and to our cost, that in actual practice we find ourselves at our wits' end to make a diagnosis, and that the difficulty arises from overlapping of the signs and symptoms of widely different lesions and of what are called atypical cases. So instead of adding to the lists compiled in this manner I will discuss some of the difficulties we encounter, and try to give some hints whereby we may learn to avoid in future those failures we have all perpetrated. In brief, were we able to ascertain and elucidate in our minds those small warnings which precede nearly all acute lesions we would be led naturally and spontaneously to the climax they inevitably end in, and the crisis ceases to be a matter of doubt and is revealed as a clear inevitable event; it becomes a logical deduction. Much as is the bursting of an explosive charge inevitable when the lighted fuse reaches the detonating charge, so a gastric ulcer or chronic appendicitis or a gall stone inevitably flares up into a crisis. The exceptions to an inevitable flare-up are few and far between, and our proper mode of conduct is to advise an early operation and not to rely upon the very doubtful chance of a "flare-up" not occurring. Where we find our real difficulty is in unravelling the warnings, since so often various acute "crises" in their early stages have

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many signs and symptoms in common. This leads to the next point I want to emphasize, namely, the taking of the history.

THE CLINICAL HISTORY.

My personal experience has impressed me with the fact that a history of a case may be acquired most laboriously and painstakingly, and yet be of comparatively small value in diagnosis. All the facts of the case may be recorded, but they bear no relation to one another; they have no sequence; they fall over one another and confuse us. Yet the taking of a history is the actual unravelling and elucidation of those warnings which, as I have indicated, nearly always precede the crisis or climax, and is the key to its logical diagnosis. It is in the taking of a history that one person may ascertain new facts, or correlate and elucidate already acquired facts in their true bearings, and so be enabled to stick to the real course of events and avoid the many by-paths amongst which a mere jumble of events is so apt to lead the unwary.

The taking of a history does not mean asking questions merely; it entails the guiding of the answers, since in all cases there are salient facts and others which are immaterial, and one salient fact leads up to the succeeding ones, and if we miss the hint conveyed by one salient fact and do not follow it up, we lose our way and fog our minds. For example, gastro-intestinal symptoms may be set up by genito-urinary disease, or a nervous lesion, or by certain poisons; and unless we are put on the right track we are apt to treat the symptom as the disease. This is where the personal factor enters. The taking of a good history—one, that is, which will inevitably lead to what I have called a logical deduction, in other words, a diagnosis—necessitates a good working knowledge of all the abdominal viscera in health and disease.

CAUSES OF ABDOMINAL CRISES.

Diseases, or crises, such as we are now discussing may have common origins. The essential cause of acute cholecystitis and appendicitis are usually the same—namely, a septic condition of the gastro-intestinal tract. Why should one individual's appendix be unable to resist the infection while his gall bladder is proof and his neighbour's appendix resist while his gall bladder succumbs? Here we have the personal factor of the patient to consider, his personal habits, diet, leanness or corpulence, constipation, and a thousand and one other things in general, while in particular there is the organ involved. I believe a person to be born with a vast inequality in the initial capacity of his various organs to sustain the work thrust upon them, and, above all, in their ability to resist bacterial invasion or the effect of toxæmia. In fact, a man may be a millionaire in liver and a pauper in pancreas. This is what used to be called "constitution," and if the particular characteristics are familial and not individual a family history assumes a new aspect towards other diseases than tuberculosis and syphilis. I believe such a familial characteristic can frequently be established with regard to arterio-sclerosis and renal disease, and insurance companies charge a higher premium to insure the child against appendicitis if its parents have suffered from that disease.

If we consider the common causes of abdominal disease, we shall find that if we look for primary causes alone to elucidate the enigma of a crisis we are often disappointed. Many times, indeed, the primary cause will have disappeared. We all recognize the disappearance of the primary causal rheumatism and the unhappy persistence of the cardiac disease, or of the primary septic urethritis and the persisting ankylosed joints, or of scarlet fever and the persistence of the chronic nephritis. We do not equally recognize this in cases of acute abdominal crises, such as is best illustrated in a case of gastric ulcer where all the septic teeth have been extracted and a clean mouth now exists, although the very fact that many teeth are absent is in itself a proof that much sepsis must have existed at one time or another in the mouth, and have contaminated the stomach mucosa.

The factor of time in producing a crisis is not sufficiently appreciated. If a patient has acquired a gall stone, however long it may remain quiescent, sooner or later with the lapse of time other factors arise which cause a disturbance

of the equilibrium hitherto maintained between the stone and the gall bladder. Other stones form, and one is propelled into the orifice of the duct by some trivial accident, and is just small enough to be firmly grasped by the peristaltic action its presence excites, and thus produces gall-stone colic, while if it is carried far enough it may obstruct the main duct and so cause jaundice; or duodenitis is set up by an indiscretion in diet or an increase of sepsis, and a stream of virulent germs gains access to the ducts and gall bladder and sets up acute cholecystitis in the already damaged organ. At any rate, given an initial lesion, time is more than likely to witness an increase in its virulence or superimpose a complication. It is this complication which is the crisis we are called upon to diagnose and treat.

An appreciation of the nature of the sequelae which must, according to my opinion, sooner or later arise in every initial lesion suspected or known to exist, narrows considerably the difficulties of diagnosis of a crisis in being, and leads me again to emphasize the need to hark back far enough in the past history for strong presumptive suggestions of an initial lesion, and in the light of that presumption to assess the existing crisis as the inevitable termination to that particular lesion. It would save many lives and much pain and danger were we to advise our patients to accept early operation once an initial lesion is recognized, instead of deferring it till an inevitable crisis arises.

THE PATHOLOGICAL ELEMENT IN DIAGNOSIS.

In diagnosis there are two chief considerations: one is the pathological, the other the anatomical. An accurate appreciation of the predominating signs or symptoms will help very greatly in narrowing down the possible lesions from the pathological standpoint. If a case is seen moderately early one should then be able to locate the lesion as in one or at most two possible organs, so making an anatomical diagnosis. It must be in the experience of all of us that at a late stage of an abdominal crisis the whole aspect of a case may alter by the addition of a complication, so that neither a pathological diagnosis—that is to say, of the primary lesion—nor an anatomical is possible.

Some acute lesions result in the predominance of one feature such as: (1) Gastro-intestinal disturbance (vomiting, diarrhoea, intestinal obstruction, meteorism); (2) profound toxæmia; (3) pain and its concomitants; (4) the signs of hæmorrhage; and (5) peritonitis.

One of the most important of the gastro-intestinal disturbances is intestinal obstruction. What is intestinal obstruction? Academically this is easily answered, as its name implies mechanical interference with the onward flow of the intestinal contents. Actually it is not so easily answered. Its manifestations are confused with a totally different condition—namely, physiological obstruction. The first difficulty arises because so many lesions which are said to produce intestinal obstruction do not in the least obstruct the lumen of the bowel. What they do is to inhibit the peristaltic action, or, in other words, paralyse the gut. Take the case of a Littre's hernia, in which a by-pass—Meckel's diverticulum—is strangulated; this has no effect upon the lumen of the bowel, which is entirely patent throughout. Or take the case of a mass of great omentum strangulated in an external hernia: this has still less connexion with the lumen of the bowel, yet the effect is to stop the onward flow of the intestinal contents and death may result.

These lesions act first by inducing irritation, then inflammation and necrosis of the peritoneum, and at a later stage by systemic poisoning due to absorption of virulent poisons from the proximal side of the constriction. Peristaltic inhibition may be set up by the proximity of an abscess or an acutely inflamed organ, as is seen in pyosalpinx with pelvic abscess, or an acutely inflamed gall bladder. Then, again, torsion of an ovarian cyst or a pedunculated fibroid will produce all the symptoms of intestinal obstruction and be treated as such. In all these cases the interference with the normal action of the bowel is only a symptom. It is not the crisis, and thus we may be misled. Physiological obstruction is a passive obstruction generally induced by an inflammatory lesion or severe twisting of the peritoneum. Mechanical obstruction is an active lesion.

THE ANATOMICAL ELEMENT IN DIAGNOSIS.

Then we have the converse state of affairs, in which, after the onset of an actual gross obstruction from a band, one or more faecal evacuations occur, either spontaneously or as the result of treatment by an enema or saline purgative, and such an occurrence is accepted as a proof that, whatever the lesion, it is not intestinal obstruction. The fact is that if the big bowel is loaded with faeces and actual obstruction is set up in the small intestine or proximal part of the large bowel, the bowel can and often will spontaneously empty its contents, especially those lying in the rectum. Further efforts to relieve the patient, by enemas especially, or by saline purgatives, will then empty the contents of the pelvic colon and other portions of the big gut, but a time will arrive when all efforts to effect further relief fail. The sooner this state of affairs is reached and recognized, the better for all concerned.

If we make a routine examination of the rectum in every abdominal crisis, we can in some degree safeguard ourselves against making false deductions, for, assuming we find a loaded rectum and a descending colon with obvious contents (felt by the abdomen), we reason: "Now we must get rid of all the contents of the rectum and colon, and when that is effected then we must ascertain whether food from the small bowel (or from a part of the colon proximal to the assumed lesion) comes along and is evacuated." If this happens we may fairly make the deduction that the lesion is non-obstructive, and twelve hours should be the outside limit to set. If the rectum is empty and all our efforts to produce an evacuation are failures, we may take it that there is a mechanical obstruction, since there is always some residue in the colon and a considerable quantity in the small intestine.

The passing of flatus in small quantity at infrequent intervals, unaccompanied by any sense of relief to the patient or improvement in physical signs, must not be taken as evidence that intestinal obstruction does not exist; flatus in small quantity is readily formed in the rectum and colon distal to an obstruction. Thus the less we rely upon the complete cessation of the passing of flatus and faeces as proof of intestinal obstruction, and the more we rely upon the collateral signs and symptoms, especially increasing distension, outlining of the intestine and coiling, the absence of early profound changes in pulse and temperature but their steady deterioration, the better for the patient. We must always bear in mind that sudden change in the usual regular habits of a person's bowel associated with other gastro-intestinal symptoms, for instance, nausea or vomiting and colicky pains, is a highly suspicious event.

Mechanical obstruction varies greatly in its manifestations according to the site of the lesion. The nearer to the stomach the interruption the earlier does persistent vomiting arise, the less marked is the general distension, there are fewer coils, and as they more completely empty they are not so prominent, collapse and grave general symptoms supervene quickly, while the "classical" signs of complete absence of flatus and faeces are long delayed; if waited for, the patient will probably succumb. When the obstruction occurs at the pelvic colon, and especially if it be due to volvulus, we get a typical acute intestinal obstruction, with complete cessation of flatus and faeces from the onset, rapid formation of outlines and coiling, marked and increasing distension, steady deterioration of pulse, slight rise of temperature, foul tongue, and late onset of vomiting, but once established, persistent, distressing and of the gravest prognosis. Even in this instance a slight evacuation of faeces may occur at the onset. Meteorism, or great distension by gas in the intestine, is characteristic of a volvulus; a twisted sigmoid may fill the whole abdomen, pushing up and obliterating the liver dullness everywhere except in the mid-axillary line. A twisted caecum or small intestine may form coils of great dimensions.

INTUSSUSCEPTION.

In infants suffering from an acute crisis we have to distinguish between intussusception and enterocolitis. There are many signs in common—for instance, collapse, vomiting, diarrhoea, pain, pulse and temperature—but two very characteristic signs are present in intussusception: (1) the absence of the caecum, and (2) the presence of a tumour. The vast number of intussusceptions sooner or later carry on the caecum.

(1) In the commonest form, the ileo-caecal, the caecum is quickly carried away from the right iliac fossa; but if we are fortunate enough to be able to examine that region very soon after the onset of the crisis, we may detect a swelling there which is the beginning of the tumour formation. Usually, however, we see the case at a later stage; then we fail to feel the caecum, and a peculiar emptiness and absence of the normal elasticity is observed; there is obviously something wanting, and our fingers sink deeply into the region and may actually palpate the back of the hollow cavity of the iliac fossa. It is a very characteristic and early sign.

(2) The absence of the caecum proves that it must be elsewhere, and at an early stage we cannot find where it is, for the good reason that it is tucked up under the liver. We may be fortunate enough to catch it before it entirely disappears, as a definite vertical tumour just below the costal margin. When it has passed beyond the hepatic flexure it reappears as a horizontal (sausage-shaped) swelling about the level of the umbilicus, which is best felt during a quiescent period—that is, during the intervals between colicky pains.

If we examine the rectum at this stage we cannot feel the tumour, but we learn a definite fact—namely, that the rectum is empty of faeces; the finger returns covered by some amount of blood-stained mucus, but devoid of faeces. In acute enterocolitis a rectal examination will show, when the finger is removed, *there is always some amount of faeces adherent*, whether there be also blood-stained mucus or not, and very often the withdrawal of the finger will be followed by a slight escape of faeces or an explosive discharge of flatus, which does not occur in intussusception. The explanation is simple. In intussusception the big bowel very quickly empties itself, and nothing additional in the way of food residue can pass the obstruction to replace it, while the blood and mucus formed by the strangulation is passed along by the colicky contraction of the colon, and is found in the rectum quite early. In enterocolitis every spasm carries on some small quantity of faecal residue which suffices to stain the finger. It may contain blood and mucus, but the quantity of either is small in comparison with the amount found in intussusception. At a later stage the soft extremity of the intussusceptum may be felt from the rectum and the finger actually passed into the lumen.

PULSE AND TEMPERATURE.

Pulse and temperature records are the best methods of recording progress in every lesion, but in abdominal crises such records are invaluable, especially if graphically recorded on charts; without them we are placed at a distinct disadvantage. The frequency and quality of the pulse afford the most constant and best general guides to increasing severity or improvement in a case. The nearer the diaphragm, the larger the area of peritoneum involved, the quicker is vomiting induced by the lesion, the more frequent is the pulse and the worse its quality. Similarly with internal haemorrhage, the more rapid the loss of blood or the more persistent the loss, the worse the pulse. Indeed, the long continuance of a state of collapse may be the only indication of internal haemorrhage, whether into the lumen of a hollow viscus or into the peritoneal cavity. If it is being extravasated into the retroperitoneal tissue it is invariably associated with severe pain from stripping up of the tissues. Grave lesions may start without a pulse alarming in frequency or quality, but steady deterioration will be noticed as the case proceeds; especially is this the case in non-inflammatory lesions. In an acute pelvic lesion, or one in which the organ or structure is retroperitoneal or excluded by adhesions from the general peritoneum, or in the case of the gastro-intestinal tract the more distant the lesion, the less is the relative frequency and quality of the pulse disturbed. A consideration of a pulse record is a valuable guide in prognosis.

The curve of the temperature is more erratic and appears less amenable to rules, as may be observed in gall-stone colic when a high temperature, often accompanied by a rigor, initiates a mild attack. Also in acute renal infection, such as an ascending infection by *Bacillus coli*, alarming temperatures may be recorded, yet a few days of appropriate treatment clears up the condition. It is notorious how slight a lesion suffices to produce a high temperature in a child or a nervous adult. On the contrary, in the aged and infirm we may find either an absence of or a low or

subnormal temperature associated with grave abdominal lesions.

In internal haemorrhage we regard a continued absence of a rise of temperature or a fall as one of our leading clinical signs, and if associated with a frequent small and weak pulse, sighing respiration, and restlessness, it is diagnostic. A continued low temperature, one only slightly raised, after the stage of collapse has passed, indicates a non-inflammatory lesion as distinct from a perforative or other peritoneal lesion; if continuously subnormal it indicates the supervention of a peritoneal lesion of a mild virulence. A sudden fall of a previously raised temperature, unless accompanied by equivalent signs of improvement, is generally a very bad sign.

Thus the associated condition of the pulse and temperature will often enable us to decide between a mucosal infection and a peritoneal lesion, and in conjunction with the absence of muscular resistance (rigidity) a good pulse is of good augury even though the temperature be high. A bad pulse with even a moderate temperature makes one anxious, since it gives ground to fear either that the cardio-vascular system is not of good quality or that there is profound toxæmia. For instance, in intestinal obstruction from bands, twists, or kinks—that is to say, non-inflammatory obstruction—the initial rise of pulse frequency and temperature is slight; but the steady deterioration of the pulse and rise of temperature, with the continuance of abdominal discomfort, increasing distension, intermittent pain, the onset of nausea and vomiting, the absence of flatus and faecal evacuation—these clearly indicate the diagnosis.

Often symptoms may appear to be better as a result of treatment. For example, in an acute case an enema or a laxative may produce a fair evacuation of flatus and faeces and the patient experiences relief, and a small drop in the temperature perhaps occurs; but if a careful estimate of the pulse frequency and quality fails to correspond and confirm the apparent improvement, we must discount the good impression and recognize that the good effect is solely due to removing a certain amount of the contents of the abdomen, so relieving tension, and not to any improvement of the actual crisis.

As distinguishing between thoracic and abdominal crises, the pulse, temperature, and respiration ratio is a valuable guide. If the respiration increases in frequency by 50 per cent. or over, the temperature rises rapidly to a high level (anything over 101°), and the pulse also increases in frequency, say to 100 and over, but is of good volume, big and bounding, a thoracic condition is almost certain. Each symptom may continue to get worse, but a rapid development of such a complex within a few hours, whatever the reflex pain in the abdomen and the apparent muscular rigidity, points conclusively to pneumonia. The diagnosis of a basal pleurisy is not so readily made by the pulse-temperature-respiration ratio alone, but the catchy character of the increased respiration, the stitch-like pain relieved by pressure, the early development of a "rub," associated with the others, is strong evidence that the lesion is in the thorax.

THE late Dr. Charles Albert Hingston of Plymouth, who died on April 5th, leaving net personalty of £86,269, has bequeathed over £25,000 to charitable institutions, including £200 to the Plymouth Public Dispensary.

Mr. Alfred Poynton, solicitor of Birmingham, who died on July 28th, has bequeathed £1,250 to the Children's Hospital, Birmingham, to endow a bed in memory of his daughter, £2,500 each to the General Hospital and the Queen's Hospital, Birmingham, for like purposes, £1,750 each to the Women's Hospital, the Orthopaedic and Spinal Hospital, the Blind Institution, the Eye Hospital, the Ear and Throat Hospital, and the Skin and Urinary Hospital, Birmingham, and £500 to the Birmingham Dispensary.

THE following are among the bequests made under the will of the late Mr. William Sutcliffe of Manchester, who died on May 25th: £1,000 each to the Manchester Royal Infirmary, St. Mary's Hospital for Women and Children, Salford Royal Hospital, and the Ancoats Hospital; £500 to the Northern Counties Hospital for Incurables; £300 each to the Northern Hospital for Women and Children, Cheetham Hospital, and the Manchester Victoria Memorial Jewish Hospital; £250 to the Manchester Children's Hospital; £200 each to the Manchester and Salford Hospital for Skin Diseases, the Royal Albert Institution, Lancaster, and the Christie Hospital (Cancer Pavilion and Home).

EIGHTY-NINTH ANNUAL MEETING

OF THE

British Medical Association.

Held at Newcastle-on-Tyne, July, 1921.

SECTION OF VENEREAL DISEASES.

Colonel L. W. HARRISON, D.S.O., R.A.M.C., President.

PRESIDENT'S INTRODUCTORY REMARKS.

In opening the first meeting of the Section, Colonel HARRISON remarked that this was only the second year in which the diseases known as venereal had been grouped for discussion by a distinct section. The bond uniting their interest in these diseases, which were similar only in the manner of their contraction, was forged by their interest in the public health. He hoped that the members of the Section would keep before them in these discussions and afterwards the public health ideal in the treatment of venereal disease. He feared that it was an ideal which was too often forgotten, and that the medical attendant was too prone to cease his efforts at a lower plane—the mere relief of the patient. It was only by aiming always at rendering the patient permanently non-infective, and being content with nothing less, that we should achieve any results of value to the public health.

DISCUSSION ON THE TREATMENT OF SYPHILIS.

TREATMENT OF SYPHILIS IN THE MALE.

BY

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A PAPER which opens a discussion ought to be provocative rather than constructive, interrogatory rather than didactic, and I propose to raise problems rather than aim at laying before you a cut-and-dried scheme for the treatment of syphilis. A little more than a decade has elapsed since the introduction of salvarsan, and the time seems ripe for a free discussion of some of the many points that have arisen since the arsenobenzol group of remedies came upon the field. I trust that one result of this meeting will be to clear up some matters on which there is still considerable divergence of opinion.

If I may for a moment be allowed to indulge in a personal note, I should like to say that my experience in the treatment of syphilis extends to exactly twenty-one years, so that for ten and a half years before salvarsan was introduced I had experience of the old remedies; and I will take this opportunity of saying that, although the Ehrlich-Hata discovery has wellnigh revolutionized the treatment of syphilis, it was quite possible in the old days to obtain excellent results from the careful and prolonged use of mercury and potassium iodide. I mention this in passing because it is sometimes forgotten. In those days mercury was our sheet-anchor, and, if properly administered, rarely failed us. But the introduction of the arsenobenzol group of remedies supplied us with new and invaluable allies. Their wellnigh dramatic effect in causing the disappearance of the outward and contagious lesions of the disease, especially those affecting the mucous membranes, was something that we had never been privileged to see before. But this was and is a qualified advantage, for it is capable of luring both patient and physician into a condition of ill-grounded security, since the disappearance of visible lesions does not indicate that the disease has been eradicated.

Modern research has contributed enormously to the efficient treatment of syphilis, but I am of opinion that the chief contribution has not been a therapeutic but a diagnostic one. By the recognition of the *Treponema pallidum* in a small, possibly innocent-looking, sore we are able to make a diagnosis at a very early date, and the earlier syphilis can be diagnosed the more efficiently can it be treated. If there is any ground for the recent statement that the special neurotropic strain of spirochaete has