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TREATMENT OF CASES OF SHELL SHOCK
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It would be out of place here to enter into any general consideration of the significance to be attached to a positive Wassermann reaction obtained where all other evidence of congenital syphilis is lacking, but at the risk of being held guilty of heresy I will venture to say that in my opinion a positive Wassermann reaction, extremely valuable as it is as confirmatory evidence, is not by itself proof of congenital syphilis.

These cases of cœliac disease in which the test was positive were none of them in any way suggestive of congenital syphilis so far as family history or the child's own past history was concerned, nor on examination was there any symptom of congenital syphilis, unless the cœliac disease itself was to be regarded as such. Treatment with mercury in one case, so far from doing any good, seemed rather to hinder improvement; at any rate, the child began to improve directly it was stopped; in another it seemed to have no effect at all.

There seemed, in fact, so little to warrant accepting the reaction as proof of congenital syphilis that we sought corroboration in testing the parents and other members of the family.

In one severe case of cœliac disease, a girl aged 3 years and 5 months, the Wassermann reaction was done twice and was on each occasion strongly positive; the mother and the father were both definitely negative.

Another case, a girl aged 5½ years with moderately severe cœliac disease and showing, as did the previous case, very marked arrest of growth, gave a definite positive Wassermann reaction; her mother gave a negative; the father was abroad and could not be tested.

The third case, a girl aged 5 years and 4 months, a milder case of cœliac disease but showing arrested growth, gave a positive reaction; the father was not available for testing, but the mother and also the sister, 15 months younger than the patient, were both entirely negative.

In none of these cases had the relatives who were tested had any treatment for syphilis so far as was known.

I leave those who consider a positive Wassermann reaction to be *per se* proof of congenital syphilis to explain such cases with what ingenuity they may.

These findings seem to me rather to emphasise the uncertainty of significance attaching to this reaction where there is no other reason to suspect syphilis, and to support the view that there are other conditions beside syphilis which may give a positive result with this test.

TOXIC ABSORPTION.

I shall turn now from the unanswered, and at present unanswerable, questions of pathogeny which I have raised to a consideration from the pathological standpoint of some of the clinical phenomena of cœliac disease. One of the noticeable symptoms, especially in severe cases, is dropsy occurring chiefly in the distal parts of the limbs and also the face, and sometimes associated with ascites.

This is common enough in any prolonged diarrhœa in infancy and, indeed, with any condition of extreme wasting. As it occurs most commonly in advanced stages of disease it is apt to be regarded as chiefly, if not altogether, an evidence of failing circulation, with, perhaps, some hydræmia as a contributory cause.

Without denying that these may play some part, as also may the diminished urinary secretion in diarrhœa with the resulting retention of chlorides, I would point out that there may be another factor, and that the œdema which occurs in cœliac disease may well be determined by it—namely, toxic absorption from the intestine.

Apart from the fact that no particular substance having this effect has been isolated from the intestine or its contents, it may be objected that in cœliac disease wasting and feebleness are usually pronounced, and that therefore there is no reason for seeking further for a cause; at any rate, such a view would require support from cases in which these recognised causes are absent.

There are at least two groups of cases which seem to me to corroborate this suggestion.

First, a very common one in which there is puffiness of the eyelids, especially of the lower eyelid, accompanying chronic constipation or chronic indigestion; this surely is œdema at its vanishing point, or rather its first appearing point. I have thought that among the children brought to one for minor ailments one could often pick out those with chronic intestinal disorder, especially affecting the lower bowel, by this puffiness of the lower eyelids.

The second group of cases, in which clinical association and sequence seem to point to a similar connexion, is a rare condition of general œdema with no albumin in the urine and no evidence of organic disease. Puzzling cases are these, and various are the explanations which have been offered. I am not referring to those in which such a condition follows scarlet fever and is associated with diminution in the secretion of urine without albuminuria, but to children in whom there has been no recent illness, and the general œdema apparently stands by itself, an unexplained phenomenon. In some of these the stools are found to be extremely fœtid and offensive although there is no diarrhœa, rather indeed the reverse; therewith the urine may or may not be diminished in quantity.

An instance of this was a boy, aged 11, who a week before coming under observation was noticed to be puffy in the face, and three days later his legs were swollen; the bowels were open three or four times a day, and the stools were extremely offensive. There was some fluid in the abdomen and general dropsy. The urine contained no albumin, it was high coloured, and rather scanty. The boy was kept under observation, and the outstanding feature, apart from the œdema, was the foul fœtor of the stools.

I can offer no proof that the œdema in such a case is due to toxic absorption from the bowel, but the association suggests it.

How far the œdema which accompanies such a condition as ileocolitis and prolonged failure of digestion is due to such absorption and how far to the wasting and exhaustion is, I think, open to question, but there is much to suggest that in some of these also the œdema is determined largely by the unwholesome character of the stools. I have particularly noticed that in some wasted infants the appearance and disappearance of œdema corresponded closely with variations in the degree of unwholesomeness of the stools, and seemed to be much more influenced by this than by the number of the stools.

This observation certainly applies to cases of cœliac disease, in which, therefore, I would suggest that œdema is to be regarded as due, in part perhaps to feeble circulation and hydræmia, but in part at least to toxic absorption from the bowel.

Certainly the œdema is not necessarily terminal. I have known it to occur in several cases quite early in the disease, and in those that recovered as well as in those that died.

Another symptom which is worthy of consideration in this connexion is purpura.

This, like œdema, is common enough as a late symptom of many diseases which end with emaciation and exhaustion, but it is particularly noticeable that in cœliac disease purpura is sometimes a recurring symptom for years.

For instance, in a girl who died at the age of 9½ years after suffering with cœliac disease from the age of 20 months, bruise-like patches of purpura appeared on many occasions from the age of 2½ years up to the time of her death, although during the greater part of this time she was well enough to be up and about.

In another case, a boy, aged 7½, in whom the disease had been present for five years, but who was still well enough to get up and walk about, patches of purpura occurred from time to time, and noticeably in relation to unhealthiness of the stool; when the stools improved and became more normal the purpura would cease to appear, then a particularly unhealthy stool, without diarrhœa, would be associated with a fresh patch or two of purpura.

I would lay some stress upon the fact that often, so far from there being any increase of frequency of stools, there has often been a noticeable decrease in their frequency, perhaps no stools at all for 24 hours, just before the purpura appears. The non-frequency of stools may be more than a coincidence; delay in the evacuations of the unhealthy fæces may be a determining cause of the absorption which results in purpura.

But, even if this view be correct, the particular substance which causes the purpura is still to seek.

Mere constipation, even when most chronic and severe, does not cause purpura, although there may be various symptoms suggestive of toxic absorption, headache, lassitude, sallow skin, and so forth; it is probable, therefore, that the cause is to be sought either in the products of particular bacteria or in the chemical products of intestinal putrefaction, and only when these hypothetical substances are present does absorption from the intestine produce purpura.

It has been suggested that indol may be the poison in question; and when indol is being formed in large quantities in the bowel this is indicated by a marked indican reaction in the urine. It has been specially noticed that in many cases of purpura a marked indicanuria is present; in one of my cases of cœliac disease I found a very definite indican reaction during the eruption of purpura, and this reaction became much less, in fact, almost entirely absent, when the purpura ceased to appear.

Apart from œdema and purpura, the occurrence of tetany in cœliac disease probably points to toxic absorption.

Here, at any rate, the question of cause is not complicated by any likelihood of production by feebleness or failing circulation; the tetany may, as in one of my cases of cœliac disease, recur for years, and that when the patient is well enough to be up and about.

It is true that tetany has been attributed to deficiency of calcium salts in the blood; and in cœliac disease there is an obvious reason for excessive loss of calcium. Quite apart from the question of tetany, this drainage of calcium was emphasised by Dr. Herter in connexion with the amount of split fat in the form of soap passed in the fæces. In one case he found that in ten days a boy aged 8 years lost 1·07 g. of calcium oxide in 198 g. of soap in the fæces, and such a loss, though it may sound very small compared with the intake of calcium in the food, is really large in relation to the very small proportion of the food-calcium which can be absorbed in health.

But does this satisfactorily explain the tetany?

I think not. If it were so, one would certainly expect that the occurrence of tetany should correspond more or less closely with the periods when the fatty character of the stools is most in evidence. I have no analytical evidence that the early pale, rancid, obviously fatty stools contain more fat than the later brown slimy or shreddy stools in which excess of fat, if present at all, is certainly not obvious; but if one may assume this, as I think one fairly may, then there is no such correspondence between the tetany and the fatty stools; indeed, the reverse is the case, for tetany is not an early symptom in cœliac disease, it occurs after the disease has been present for a long time and usually when the stools, though extremely offensive, have lost the characteristic fatty appearance of the early stage.

Absorption of some toxic material from the bowel seems much more in harmony with the clinical fact.

ARREST OF GROWTH.

From these questions of toxic absorption I pass to the consideration of one of the most striking phenomena of cœliac disease, the arrest of growth.

Why do these children fail to grow? It is easy to reply vaguely that the cause is the general failure of nutrition, but such an answer is inadequate. It is unfortunate that much that has been written on the problems of growth entirely ignores the fact that increase of weight and length do not necessarily go *pari passu*, and that experimental evidence as to the one is not necessarily applicable to the other. A physiological illustration of this must be recognised by any one who measures many children at the growing age; a rapid increase in height is not associated usually with increase in weight, often, indeed,

with a marked decrease, and the child may be visibly thinner; this is very noticeable in infants and gives rise to much needless anxiety, though I have not seen it mentioned in a single text-book that an infant in perfect health will often cease to gain or will actually lose weight when it is growing rapidly in length.

In disease, also, there is no necessary correlation between the growth in length and the state of general nutrition as shown by the weight. As I have mentioned already a marasmic infant may grow steadily in length, and a child may become tall whilst wasted with chronic tubercular disease.

It would seem, therefore, on grounds of analogy that there is no justification for assuming that the remarkable arrest of growth in length in cœliac disease is a necessary corollary of the arrest of growth in weight, and, while it seems only reasonable to assume that both may be related in some degree—though not necessarily in the same degree—to the general failure of nutrition, the failure of growth in length is so much more pronounced in cœliac disease than in most other conditions of disturbed nutrition that we may well seek for some special cause.

Amongst the various possibilities which present themselves there is one which, I think, ought to be considered—namely, that deficiency of fat absorption may be sufficient cause for arrest of growth.

This suggestion has the support of experimental evidence; young rats fed on a diet of lean horseflesh, so that there was hardly any fat given, became stunted in growth, even though apparently in good health; with a diet exclusively of oxflesh, which, though still affording them too little fat, contains more than does horseflesh, the rats throve better, but their growth was still retarded to some extent. (Watson and Hunter.[13])

If the failure of fat assimilation be the cause of the failure of growth this would bring cœliac disease into line with Byrom Bramwell's pancreatic infantilism, where arrest of growth was associated with the passage of fatty stools and where improvement of fat absorption by administration of pancreatic extract, with consequent disappearance of the fatty character of the stools, was followed by rapid growth.

It is not apparent, however, how deficiency of fat in the diet has any special influence in arresting growth.

If the absorption of calcium is dependent to any considerable extent upon the splitting of fat and combination with the fatty acids thus set free, and if deficient absorption of calcium inhibits growth then the order of events is easy to understand. As I have already pointed out, Herter has already shown that there is an appreciable loss of calcium with the soaped fat passed in the stools of cœliac disease. Clearly the same result would come also from any considerable failure of fat splitting or saponification.

I have mentioned a case in which remarkable bending of the bones began at the age of 8 years, after the cœliac disease had been present nearly five years, and the child had been kept for four years on a diet containing a minimum of fat; an occurrence strongly suggestive of calcium deficiency.

I have shown, also, that there is very striking delay of ossification of epiphyses, which may indicate a deficiency of calcium, but, granted this deficiency, is it adequate cause for the striking arrest of growth in cœliac disease? I have shown in my previous lecture that growth in height shows no necessary relation to the date of commencement, nor to the stage of advancement of ossification in epiphyses.

That relative deficiency of calcium and rapid growth are by no means inconsistent is shown by what happens in cretins when thyroid treatment is begun at the age when walking has recently been acquired, the child begins to grow rapidly and at the same time if the child is allowed to run about much, the previously straight legs become bowed and bandy, presumably from deferred calcification of the rapidly growing bone.

The disease which might have been expected to throw most light upon this point is rickets.

There is strong evidence that fat deficiency in the diet is a large factor, perhaps the determining factor, in the causation of rickets; moreover, there is clear evidence of deficient calcium deposit in the rachitic bone. Here, then, we seem at first sight to have the required link bringing the fat absorption into direct relation with formation of bone.

But if this view of the pathogeny of rickets is correct we are faced by another difficulty in regard to cœliac disease—it should be associated closely with rickets; whereas one of the very striking facts which I have already mentioned in connexion with its symptomatology is the entire absence of rickets in most cases. Conversely also, if the failure of fat absorption be the cause of the stunting in cœliac disease, arrest of growth might be expected far more commonly than it occurs in rickets.

The problem is evidently a complicated one, and it is not rendered less so by a comparison with such a condition as the congenital steatorrhœa described by Garrod and Hurley,[14] in which the passage of 80 per cent. of fat in the fæces, amounting to a loss of 25 per cent. of the fat intake, was associated neither with arrest of growth nor with rickets.

THE LATE JOHN ST. CLAIR BOYD, M.D. R.U.I.—Dr. John St. Clair Boyd, the well-known Belfast surgeon, who died suddenly of cardiac disease, was a graduate of Queen's College, Belfast. After further study at Edinburgh and Paris he became an assistant to Mr. Lawson Tait in Birmingham when that well-known surgeon was at the height of his career. Returning to Belfast, he was appointed surgeon to the Samaritan Hospital, but ill-health obliged him to retire about ten years ago. He was also consulting gynæcologist to the Ulster Hospital for Women. Dr. Boyd was a man of literary taste and well versed in the study of the Irish language. He married a daughter of the late Dr. Stevenson Macadam, lecturer on chemistry in the Edinburgh Extra-Mural School.

THE TREATMENT OF CASES OF SHELL SHOCK IN AN ADVANCED NEUROLOGICAL CENTRE.

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WHILE in charge of an advanced neurological centre in France during the period November, 1916—February, 1918, I have had to deal with between two and three thousand cases of psychoneurosis (neurasthenia, hysteria, and psychasthenia), and in the following paragraphs I will endeavour to give, in broad outline, a general view of the methods of treatment which have seemed to be most useful, and a statement of certain statistical results. The great majority of cases came under my care within 48 hours of their breakdown, and I was able to return 70 per cent. of them to the line after an average of a fortnight's rest and treatment in hospital; of course, certain individual cases needed a longer rest in hospital, up to two or three months. I feel sure that this success was due more often to *prevention* than to cure. By seeing so many of these light cases so early I was able to *prevent* the development of further hysterical or pithiatic symptoms which would otherwise have taken place.

Essential Factors in Causation and Treatment.

It is important that the patient should have the benefit of a thorough examination of his nervous system at the earliest possible moment after the shock of the shell explosion. The incipient functional symptoms from which he is suffering make him fear, vaguely or definitely, that he has sustained some organic injury of the nervous system. This fear encourages the further development of such symptoms. It and its effects can be brought to an end by the reassuring remarks of the neurologist, after he has completed his examination. But those remarks must be repeated and forced upon the patient's notice. Especially is it the note of certainty in the doctor's voice which carries conviction. By one means or another the patient must be completely convinced of the truth of the doctor's explanation of his symptoms, and of the promise that they will quickly disappear. This conviction must pass beyond the stage of mere intellectual awareness and acceptance, and acquire the dynamism of strong emotion. Enthusiastic expectation of a rapid recovery is another essential condition of success, just as it was an earlier apprehension which was responsible for the development of the symptoms.

This is more than mere suggestion and counter-suggestion. The mechanical processes of auto- and hetero-suggestion do certainly come into play in the *fixation* of the symptoms and in their later development and multiplication. But the *origin* of the symptoms (tremors, difficulties of speech and locomotion, profuse sweating, headaches, diminution or loss of various forms of sensation, &c.) is to be found in the intense emotion of fear caused by the shell explosion, of which they are the objective physical manifestations. In like manner, although counter-suggestion plays its part in the cure of these functional symptoms, the factors which are of very much greater efficacy and importance in bringing about a *permanent* cure are: (1) persuasion, whereby the patient is rationally convinced of the true nature of his symptoms; and (2) the sthenic emotions of confidence, conviction, and expectation, which have a unifying effect upon the mind, and counteract the disintegrating effect of mechanical suggestion.

It will thus be seen that I adhere to Déjerine's theory of the pathogeny of the psychoneuroses in preference to that of Babinski. As regards the "period of meditation" which often elapses between the occurence of the emotional shock and the onset of the earliest symptoms, I would also follow Déjerine in holding that it is a period of subconscious emotional development. Babinski and his followers believe that it is a period during which auto- and hetero-suggestion coöperate to bring about the ultimate outbreak of the symptoms. The original shock, no doubt, produces some weakening of mental synthesis, although outwardly the patient does not appear greatly disturbed. The appropriate

emotional reaction, with its innervations and external manifestations, develops gradually during the following few hours or days, as the patient's intellectual awareness of the accident arouses by association earlier emotional memories. The symptoms that eventually arise are such emotional manifestations in a state of relative dissociation and permanence. As Déjerine says:—

"Everything that an emotion may create in an accidental and transient way hysteria may accomplish in a lasting way."[1]

Case of Delayed Onset of Symptoms.

I have several examples of this delayed onset of symptoms in the case of hysterical mutism and hysterical hemiplegia which I hope to analyse and describe on another occasion. But I have obtained the following unbiased introspective description from one of my officer patients who suffered from hysterical spasmodic contractions of the abdominal and leg muscles and profuse sweating and tachycardia, which came on some days after the time when he was exposed to heavy shelling. His account is as follows:—

"My feelings during the shelling are hard to define, as I was too fully occupied to allow for much thought on the subject. Owing to the small area to which we were confined, there was no opportunity of being able to give vent to the pent-up feelings that were in me, and in consequence my nerves were strung up to such a pitch that I felt that something in me would snap. Every shell fired seemed to be nearer the mark than the last, and the ground all around was covered with shell holes. The general feeling was that 'the next one' would land right in the post. Part of the trench had already been blown in. The back blast from each explosion flattened us up against the wall of the trench.

The days following I was always thinking of this episode, and nights I could never sleep, but would just doze and then wake up with a start, with my heart palpitating furiously and with great difficulty in breathing. I would also find myself in a profuse cold sweat, especially of the scalp, forehead, and hands. Then my legs began to be affected and would shake as though I had the ague. This would come on in the day at times, but invariably happened at night when I was lying down. About 10 days later I was troubled with my stomach [he means abdomen]; at night something—I suppose my nerves—started pulling me up forward as I lay down and my body shook all over. This would happen every two or three minutes. This action affected my back, and I had a belt of pain around me. During this time I was always troubled with my heart, and I found it hard to lie down at all. The cold sweats also continued."

Emotional Origin of the Symptoms.

This description, written by an officer unversed in psychology and quite ignorant of the question at issue, is in favour of the emotional origin of the symptoms, although, of course, factors of suggestion are not entirely absent. The emotion of fear in this patient during civil life always produced cold sweats, palpitations, and tremors of the limbs—just as in the case of a patient subsequently suffering from functional paraplegia it used to "take him in the legs" and he would feel weak at the knees. The period of incubation in some cases seems to correspond to the time during which the patient is struggling, with a certain degree of success, to repress the painful emotional memories and to remain "captain of his soul." The final outbreak of the symptoms represents the partial failure of this effort of repression, or rather is a condition of the success of the *psychological* repression. The symptoms are "convertion" symptoms, in Freud's sense of the term—i.e., they represent painful emotion converted into physical innervations.

With a certain class of patients, a more detailed form of mental analysis is necessary. In these cases the nervous breakdown is partly due to earlier mental worry, and the "active service" element is simply the last straw. The patient's mind is a battlefield of conflicting emotional tendencies, many of which he himself fails to recognise. By long talks with him, following up thread after thread in the association of his ideas, one can recall repressed memories of earlier epochs in his life, showing him the true emotional significance of them and their connexion with his present disability. In this way the patient learns to understand himself, and therefore I would call the method "*autognosis*." Self-knowledge brings with it self-control in the psychic domain. An analysis of the patient's dreams is often of the greatest help in this method. Indeed, the dreams of some of these patients are so terrifying that they have to be dealt with, and the quickest way to exorcise them is to analyse them—i.e., to trace the different parts of the dream back to their source in the patient's earlier life (by the method of "free" association), and so to discover the underlying unconscious dream thoughts which have been enjoying a disguised or distorted fulfilment in the dream itself.

[1] Psychoneuroses and Psychotherapy, pp. 269, 270.

Treatment.

I now come to a therapeutic method which I have found to be especially valuable in dealing with *early* cases of shell shock exhibiting pronounced symptoms of functional nervous disorder, such as functional mutism, deafness, paralysis, tremulousness, &c., accompanied by more or less extensive amnesia. It is a form of hypnosis, but free from the defects attaching to the ordinary use of that method. To explain by an example:—

Here is a patient who was blown up and buried by a shell explosion two days ago and lost consciousness. On regaining consciousness some hours later he found that he was quite dumb, and also had lost all recollection of the shell explosion and of the events immediately following thereon. In other words, he is functionally mute and has retrograde amnesia. His memory for other recent experiences is also vague, but he is in full command of gesture-language and can write down on paper all that he wishes to say.

I interview him alone in my office and tell him in a tone of conviction that I shall restore his speech to him within a few minutes if he will do exactly what I say. I then tell him to lie down on a couch, close his eyes, and think of sleep. I urge him *to give himself up to sleep*, to let sleep come to him, as it assuredly will. I tell him that he is getting drowsy, his limbs are getting heavy with sleep, all his muscles are relaxed, he is breathing more and more slowly, more and more deeply. Above all, that his eyelids are getting heavy, as heavy as lead, that he feels disinclined to open them, that he cannot open them however hard he tries. At this stage, which generally supervenes within two or three minutes, he really cannot open his eyes. This is a stage of very light hypnosis quite sufficient for my purpose.

I now tell him that the moment I put my hand upon his forehead he will seem to be back again in the trenches, in the firing line, in the fighting, as the case may be, and will live again through the experiences that he had when the shock occurred. This I say in a tone of absolute conviction, as if there is not the slightest shadow of possibility of my words not coming true. I then place my hand on his forehead. He immediately begins to twist and turn on the couch and shouts out in a terror-stricken voice. He talks as he talked at the time when the shock occurred to him. He really does live again through the experiences of that awful time. Sometimes he speaks as if in dialogue, punctuated with intervals of silence corresponding to the remarks of his interlocutor, like a person speaking at the telephone. At other times he indulges in imprecations and soliloquy. In some cases he is able to reply to my questions and give an account of his experiences. In others he cannot do so, but continues to writhe and talk as if he were still in the throes of the actual experience. *In every case he speaks and acts as if he were again under the influence of the terrifying emotion.* It is as if this emotion had been originally repressed, and the power of speech with it, and is now being worked off and worked out.

Abreaction.

This process of "working off" repressed emotion is what Freud calls "abreaction." In my view it is the essential therapeutic process in dealing with the majority of war psychoneuroses. The original shock caused a *dissociation* of consciousness—i.e., an apparent suppression of a certain series of memories, and of a certain motor function (speech). Now, without accepting Freud's sexual theory of the origin of the psychoneuroses, for which I have never been able to find any convincing evidence (i.e., as a universal pathogenic factor), I *do* accept his theory of the cause of mental dissociation—viz., that it is the result of mental conflict and involves repression of emotional states. The repression of any emotion at its inception involves the danger of dissociation, the ideas accompanying the emotion being then more ready to split off from the rest of the mind and pursue a subconscious life of their own.

In most cases of nervous shock caused by shell explosion a state of intense fear is aroused in the patient's mind, which, from its very magnitude, produces loss of self-control and apparent loss of consciousness. There is no real loss of consciousness, but the attempted repression and control of the fearful emotion at its inception brings about a splitting of the mind, which appears later as an amnesia of greater or less extent, often involving other losses of function also, such as dumbness, deafness, tremulousness, or paralysis. The fact that under light hypnosis, and with the appropriate suggestions, these memories return, together with the lost voice, hearing, &c., is evidence that they were not abolished at the time, but were simply split off from the main personality. It is also evidence that the shock worked mentally rather than by its accompanying *physical* concussion (which is often absent).

In my method, then, the patient goes through his original terrifying experiences again, his memories recurring with hallucinatory vividness. It is this which brings about the return of his powers of speech, and not direct suggestion, as in the ordinary method of hypnosis.

My second modification of the ordinary method is in my manner of awakening the patient. Remembering that his disability is due to a form of dissociation and that in some cases hypnotism accentuates this dissociation, I always suggest at the end of the hypnotic sleep that he will

remember clearly all that has happened to him in this sleep. More than this, I wake him very gradually, talking to him all the time and getting him to answer, passing backwards and forwards from the events of his sleep to the events in the ward, the personalities of the sister, orderly, doctor, and patients—i.e., all the time re-associating or re-synthetising the train of his memories and interests.

By the process of *abreaction* I remove the underlying *cause* of the patient's original dissociation, and in attacking this cause I cannot be accused of merely treating symptoms.

I agree entirely with Pierre Janet that only hysterical patients can be hypnotised—in fact that, as Charcot said many years ago, "hypnosis is an artificial hysteria." It follows from this that as the patient improves under treatment he should become less and less hypnotisable. This is the case with my form of the method. Hypnotism has been brought into disrepute by those seekers after the marvellous who have intentionally split up the personality of their subjects more and more in order to get ever more wonderful and abnormal manifestations from them.

Using this modified form of the hypnotic method, one can abolish that tendency towards "*equivalents*" which Janet mentions as one of the three stigmata of hysteria (the other two being *suggestivity* and *distractivity*). By this is meant the tendency which the patient shows to suffer from another functional symptom as soon as his original symptom is removed by suggestion under hypnosis. For example, a mute may develop headaches or gastric symptoms as soon as he has been made to speak by suggestion under hypnosis. By using "abreaction" this tendency is completely eliminated. The use of suggestion *without* hypnosis quite fails to remove this tendency to develop equivalent symptoms. The equivalent symptoms are often so vague that they are not noticed at the time, although they are none the less disabling.

Results of Treatment.

The hypnotic method is needed only for the very small minority of cases that arrive at an advanced neurological centre—only for those showing so-called major hysterical symptoms. In two series of 1000 cases each I treated 173, or 17·3 per cent., in the first, and 132, or 13·2 per cent., in the second by means of hypnosis. In these two series there were altogether 121 cases of loss of speech, or hysterical mutism. I succeeded in curing *every single one of these cases*. Many of them were deaf as well as dumb. These were given a paper of instructions to read, in which they were told to lie down, close their eyes, and give themselves up to sleep. When such a patient showed by the calm look on his face that he had reached a quiet frame of mind, I would suddenly and unexpectedly bang two books together near him, and have the pleasure of seeing his eyes flicker, and then find that he could hear. I could then continue the treatment by word of mouth in the ordinary way. Curiously enough, these deaf-mutes were the easiest of all my cases to treat.

Of course, there are some patients who are ready to malinger with major hysterical symptoms. I caught out 28 such patients in my first series of 1000 cases, and induced them to confess. But this is, happily, a very small proportion—less than 3 per cent.

If, finally, the modified hypnotic method is limited in application, wherever possible, to *one* treatment only, all the drawbacks with which the ordinary hypnotic method is beset are evaded. I only applied the method to the more severe cases, those that had later to be evacuated to the base.

Nevertheless, in such apparently severe cases as those of functional paraplegia this method was not found to be necessary. Vigorous persuasiveness was sufficient to produce a cure. In some of the lighter cases of mutism also this was found sufficient.

Stammering is much more difficult to cure than mutism. The patient can more easily cling to the first symptom than to the second, and every hysteric clings to his symptoms. But in several cases I have produced a complete cure, and a decided improvement in others. In several cases where the patient had stammered in civil life I was able to trace the stammer back to a shock in early life, in the first few years of childhood, and under hypnosis I was able to call up the experiences with hallucinatory vividness before the patient's mind. The stammering was not completely cured thereby, because it had become the habit of a lifetime, but this recall of the shock was the first necessary step towards a cure.

Another Illustrative Case.

As a further illustration of my method I will describe the case of an officer, aged 25 years, whom I treated in France in December, 1916, and whom I happen to have again under my care at Craiglockhart in May, 1918.

He had been sent into my neurological wards on account of his complete inability to stand shell fire, having the irresistible impulse to rush to his dugout and crouch down there the moment shells began to fall in his neighbourhood. Earlier in the year (1916) he had been wounded during an advance and had been left out in No Man's Land under continuous shell fire for several hours. He gave a history of great timorousness and extreme weakness of memory during his youth. I learnt from him that he had been nearly drowned at the age of 3 years, according to what his mother had told him. He himself, of course, could remember nothing of the incident. I should also add that he exhibited many psychasthenic symptoms—e.g., he would begin to count the squares round the top of the stove in his ward, and find that he could not stop counting them. He would be impelled to count them hundreds of times.

I hypnotised this officer and then directed his attention back to the drowning incident at the age of 3 years. He at once began to live again through this incident, gasping with terror as he again, in memory, fell into the water. He described the whole event with such a wealth of detail that it was difficult not to believe that he had been taken straight back to this early period in his life and was living again through the terrifying experience. Later on, he re-experienced with hallucinatory vividness the incident of his baby brother's funeral, seeing again the coffin being lowered into the grave and feeling the desperation of grief *as a present emotion*. He was 18 years old when this funeral took place.

I concluded from these and other results of the hypnosis that the patient's memory was intact, but that certain factors were interfering with the power of recall—viz., the very strong emotional tone of many of these memories, combined with repression of them following upon mental conflict. The "abreaction" of these painful memories in the hypnotic state—i.e., the working off of their emotional accompaniments—should therefore prove beneficial to the patient's powers of memory and to his general mental condition. This was found to be the case, and the patient appeared quite a different, and certainly a much more normal, person after the treatment.

A minor point of interest arising out of this case is that the patient was readily hypnotised in spite of his psychasthenic symptoms. Some authorities have held that psychasthenics are not hypnotisable.

In May of this year I learn from this patient that his mother vouches for the accuracy of his revived memories of the drowning incident in every particular. He is again in hospital suffering from certain neurasthenic symptoms, but he has completely recovered from his previous hysteria. He has done a year's duty since he was with me in France.

Other Treatment.

General hygienic measures are, of course, necessary in the after-care of severe cases, and suffice for the entire treatment of the largely preponderating light cases. Regularity is insisted upon in all habits—alimentation, excretion, sleep, exercise. The patients are put on physical drill and sent for short route marches. Many do light duty in the wards or in the grounds of the hospital. But to the "gospel of work" is added the gospel of cheerfulness and hope.

For insomnia suggestion treatment at night is often very efficacious. The patient can often be soothed to sleep and incidentally taught how to employ auto-suggestion in this case.

Only very rarely have I found it needful to *isolate* patients. But this device succeeded in the case of three patients showing persistent tremors of the head, where every other method had failed.

Summary.

The essential therapeutic agent in the case of *hysteria* caused by shell shock is "abreaction," or the working off of the repressed emotion caused by the shock. Mental analysis is a means to this end, and light hypnosis, applied under proper safeguards, is the quickest and most effective method of effecting this analysis, where amnesia is present and the case is seen early.

For cases of *neurasthenia*, which are the more numerous and involve emotional preoccupation often dating back many years, mental analysis and re-education, without hypnosis in any form, are the needful agents, although the abreaction of the original emotional disturbance or disturbances is again essential.

In both classes of cases the arousing of *sthenic emotion* in the patient's mind is an important adjunct in the cure, both in the form of enthusiastic confidence in his doctor and expectation of a complete recovery, and also in the form of

vivid interest in some form of occupation in the stage of convalescence, during which the mind becomes more unified and consolidated.

I have hitherto not mentioned *rest*, which is, of course, also fundamental. But no complete rest is possible while the mind is obsessed with bottled-up emotion. This emotion must be completely worked off, and then true rest will come. The preoccupations of the neurasthenic also must first be dealt with if any form of rest cure is to produce good results.

After three and a half years of work with nerve patients in military hospitals in Egypt, England, and France, during which over 4000 cases have passed through my hands, I feel no hesitation in saying that mental analysis is the ideal method of treatment, provided that it is carried far enough to produce true abreaction of emotional states. As an aid, and as a sort of short cut, the special form of hypnosis which I have described is very useful in early cases showing major hysterical symptoms.

In conclusion, I should like to thank Lieutenant-Colonel C. S. Myers, F.R.S., R.A.M.C., for his unfailing encouragement and most helpful advice as consultant psychologist in his frequent visits to my wards in France.

May 29th, 1918.

ON THE DIFFERENTIAL DIAGNOSIS OF THE DYSENTERIES:

THE DIAGNOSTIC VALUE OF THE CELL-EXUDATE IN THE STOOLS OF ACUTE AMŒBIC AND BACILLARY DYSENTERY.

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IN a paper on Dysentery published in 1913 by Savage and one of us,[1] it was pointed out that dysentery, from its first authentic record in the days of the Athenian pestilence during the Peloponnesian War up to the present time, has been *the* scourge of armies in the field, and that it has been responsible for more sickness, invaliding, and military waste than any other factor.

Scope of Investigation.

The greater part of the present paper was written by us in the spring and summer of 1916, and the results to be set forth in the bacillary section were based upon the microscopic and cultural examination of a series of 1165 cases (43 fatal) admitted to a general hospital in Egypt, from April 30th, 1915, to April 30th, 1916. As many stools as possible were plated out on a specially modified MacConkey's medium, and all the fatal cases were submitted to autopsy.

Unfortunately, our collaboration was interrupted by the recall of one of us (C. H. S.) to Government service in Australia, in the late summer of 1916, before we were able, owing to lack of amœbic material, to complete our paper together for publication.*

Since then a further very large series—though we have not the exact figures to hand—has come under our observation. This series included many cases of acute amœbic dysentery, and has been examined by one of us, who had had in addition the experience of 14 years' residence in Egypt and at El Tor upon which to draw. Any criticism, therefore, which may be directed against the amœbic part of this paper must be held to refer only to that one of us (J. G. W.).

Infantile diarrhœa, due, practically speaking, in Alexandria to the *B. dysenteriæ*, levies an appalling toll amongst Egyptian babies at the present day.

Amœbiasis, on the other hand, is a disease of protozoal origin, by reason of which it is much less infectious than bacillary disease, but immunity to it is less easily acquired and less complete.

It thus follows that the lower class Egyptian with his dirtier habits constantly exposes himself to both infections, but succumbs, as a rule, to the one only, unless his constitution has been previously strained by excessive hardship or disease. The better class Egyptian, on the other hand, thanks to his cleanlier habits, is usually exposed only to the bacillary infection, against which, owing to its far greater infectivity, precautionary measures are more difficult to apply.

The conclusion at which we arrived together—namely, that a diagnosis between the two great types of dysentery, amœbic and bacillary, can be made not only more rapidly but even with greater accuracy by simple direct microscopic examination of the stools than it can by cultural methods—has been strengthened by this further study and by a consideration of the results published by independent workers, particularly Bahr, Wenyon and O'Connor, Ledingham and Mackie, to mention only a few with whose work we are most familiar.

The results in particular of the investigations conducted by or under the ægis of the Medical Advisory Commission have established beyond all manner of doubt the contention which we, in 1915, were almost alone in upholding—namely, that bacillary dysentery has been, and is, the predominating and the more severe type of the disease in the whole of the Near Eastern area of operations.† To this conclusion we were led by investigations the object of which was to determine whether or not two diseases so distinct in their etiology and morbid anatomy would give rise in the dejecta to different types of cellular exudate, each peculiar to its own class—in other words, to determine whether a diagnosis could be made without identifying either the one or the other causal organism.

Importance of Early Diagnosis.

It is our purpose to show in this paper that—

1. Pure amœbic dysentery—i.e., uncomplicated by coexistent bacillary infection—gives rise to a characteristic exudate in the stools.
2. Bacillary dysentery, whether complicated by amœbic infection or not, also gives rise to a cellular exudate of specific character in the stools.
3. The finding of *Entamœba histolytica* in the midst of such a "bacillary" exudate indicates, not that the case is one of simple amœbic dysentery, but that a double infection is present, although all attempts to isolate dysentery bacilli may fail—as they do in many instances even when the case is one of "simple" bacillary dysentery.
4. In such cases of double infection, which are of more frequent occurrence than has been supposed, the diagnosis of the amœbic moiety can be made only by finding *E. histolytica*, for the reason that the exudate associated with the latter is comparatively so scanty and insignificant that it is masked by the overwhelming bacillary exudate. While the consequences of neglected amœbic infection may be disastrous, it is of still more immediate importance that bacillary dysentery should receive prompt recognition and appropriate serum treatment, for the following reasons:

(*a*) In fulminating cases, the time limit during which treatment is of any avail is much shorter in bacillary than it is in amœbic cases.

(*b*) In severe cases the patient rapidly becomes soaked with bacillary toxin, which results in severe degenerative changes occurring in myocardium, liver, and kidneys.

(*c*) The earlier a bacillary case is treated with *massive* doses of antiserum the more prompt and complete is recovery. While a delay of a day or two in instituting emetine treatment usually makes little difference to an amœbic case, a like delay in administering antitoxin to a bacillary patient may make all the difference between the abortion of the illness and months of invaliding and abdominal misery.

(*d*) Antidysentery serum is harmless to an amœbic patient; emetine, on the other hand, seems to us, as a result of our experience, to be a highly risky drug to administer to anyone whose myocardium is degenerated. As this does not occur in simple amœbic dysentery there is little risk in administering it in large doses to such cases, and even less to healthy carriers.‡

Pathology.

In order that the principle on which this cyto-diagnosis is based may be more fully understood it is necessary to give the briefest possible account of the pathology of the two diseases.

* From September, 1915, up to July, 1917, I (J. G. W.) was unable to obtain amœbic material in quantity sufficient for my purpose from European troops. All my cases of amœbic infection occurred in Egyptians of low class, among whom alone does the amœbic type of dysentery predominate. The explanation of this would appear to lie in their relative immunity to infection by the dysentery bacilli—a fact which is paralleled by their well-known immunity to organisms of the enterica group, which they have acquired through generations of existence in defiance of the most elementary rules of personal hygiene.

† We do not deny that there was a great deal of amœbic dysentery also in the summer of 1915, but not later.

‡ It must not be forgotten that *Entamœba coli* may coexist, as a harmless commensal, with *E. histolytica* or any of the dysentery bacilli.

By the term "dysentery" we understand a clinical condition the essential characteristic of which is the destruction of any part of the mucosa of the large intestine by one of the specific infections, amœbæ or dysentery bacilli; only those cases which showed evidence microscopically in the stools of such destruction in the form of mucosal sloughs and cellular exudate are included in the tables of our cases. Thus it is likely that many cases of early infection by dysentery bacilli which were recognised as such by the clinicians and treated immediately by them with massive doses of antiserum—with consequent abortion of the illness—figure in our returns simply as cases of enteritis. This was due to the fact that during the rush of the epidemic the pressure of work prevented the stools of all cases being examined until some days after the admission of the patient to hospital.

Pathology of Amœbic Dysentery.

Amœbic dysentery is due to infection by the *Entamœba histolytica*, and by it alone. No other amœba is capable of causing this disease, and it requires no help from secondarily infecting organisms of the bowel in the production of its lesions.

The life-history of this parasite is briefly as follows:—

The encysted form is swallowed with the food. Arrived in the small intestine, the digested juices there dissolve the capsule and set free the small daughter amœbæ, which make their way to the large intestine and thence down the lumina of the mucosal glands into the submucosa. There they grow into the adult or vegetative form, multiply by binary fission, and, in our opinion, give rise to a direct colliquative necrosis of the submucosa and muscularis mucosæ by means of a proteolytic ferment which they are able to secrete.

Direct communication with the lumen of the gut—i.e., ulceration—is only attained when either the solvent attacks the intercellular cement of the mucosa or the necrosis extends far enough to interfere with its blood-supply. According to which occurs first the mucosal cells are shed singly or *en bloc*, and an irregular ulcer with ragged, undermined edges is formed. The loss of substance is far greater in the submucosa than in the mucosa, as large vegetative amœbæ are found in the greatest numbers there; often sinuses lead from one ulcer to another under bridges of perfectly healthy mucosa.

As the amœbæ approach the surface of the mucous membrane again they divide more and more rapidly, and consequently become smaller and smaller until on the surface of the gut they assume the "*minuta*" form. Many of them now encyst, and they are passed out of the body in the stools as cysts or *E. minutæ*. It is worthy of note that the *E. minutæ* § produce no destruction of the tissues, and consequently no symptoms of dysentery; a fact which is distinctly in favour of the lager, or older, amœbæ producing a proteolytic ferment.

Adami was the first to point out that the reproductive and the higher functional activities of cells are mutually antagonistic; the more embryonic the cell the more readily does it multiply, and thus the more rapid is the growth of a neoplasm composed in greater part of cells of embryonic type. The smaller size of these amœbæ is due to their more rapid division, which has used up all their reserve of energy; consequently none is left for the development of that particular function which differentiates *E. histolytica* from all other amœbæ.

It has never been shown that the entamœbæ produce a diffusible toxin, or that they are capable of exerting any poisonous effect other than the proteolysis of cells in their immediate neighbourhood.

In consequence, the onset of the disease as a rule is insidious—it may be even unnoticed—and it is not until a comparatively large area of the gut is involved that the patient becomes at all acutely ill. It will thus be noted that the mucosal cells are in general healthy, apart from the areas of actual infection, while at autopsy the typical picture seen is one of areas of circumscribed necrosis, dotted like islands in the midst of healthy pink mucosa.

Now-a-days, when the emetine treatment is so universally adopted, cases where the bowel has undergone "gangrene en masse" are, fortunately, so rare as to warrant only passing notice. This condition is brought about by such an extensive amœbic invasion of the submucosa—and even the muscularis—with consequent thrombosis of blood-vessels, that the blood-supply of the bowel is cut off as effectually as by multiple thrombosis arising from any other cause.

The result is that the entire thickness of the bowel over a greater or lesser extent becomes gangrenous, and is passed as stringy black sloughs having a horribly fœtid odour. On very rare occasions it has even been impossible to remove the colon at autopsy, as all that remained to represent it was a slimy, stringy, black mass lying in a bath of corruption. Unless one had actually seen such a case it would be inconceivable that a man could survive long enough for his intestines and his peritoneum to attain such a state.

Sellards and Baetjer have shown that fatal cases of amœbic dysentery often terminate with a blood infection by members of the *Streptococcus fæcalis* group.

There is nothing astonishing in this if one considers how often the intestinal bacteria invade the body before death in different chronic and subacute diseases. The astonishing part is that it does not occur more frequently in amœbic dysentery—a disease in which there is such an extensive solution of continuity of the mucosal barrier. The point which we wish to emphasise is that in the vast majority of cases of non-fatal amœbic dysentery no such invasion of the blood stream can be demonstrated by hæmoculture.

This may be due to one or two causes acting together or separately.

In the first place, the liver may act as an efficient bacterial filter; in the second, the bowel itself is probably endowed, even when diseased, with a high immunity against just such organisms. Wright has shown that different parts of the body possess different degrees of resisting-power against pyococci. It seems reasonable to suppose that this local immunity acquired by countless minute inoculations with the bacteria-containing fluid in which the mucosa has been bathed since birth is sufficient to protect not only the body against invasion by these bacteria, but even the mucosa itself when the latter is partially destroyed by amœbæ.

In amœbic dysentery only when extensive areas are killed by interference with their blood supply does any secondary infection of the mucosa take place, just as a limb may become affected by wet gangrene after the blocking of its blood-supply. When, however, a pathogenic organism, such as a dysentery bacillus, which is a stranger to the mucosal barrier, and against which the mucosa is in consequence not immunised, arrives on the scene in sufficient numbers and virulence immediately its destructive properties come into play.

When sections are made through the edges of early amœbic ulcers, and more particularly when the unbroken amœbic nests in submucosa or liver are examined histologically, the salient point to strike the observer is the absence of the usual cellular infiltration characteristic of inflammatory reaction.

This is all the more striking when a comparison is made with the corresponding sections of early bacillary dysentery which is an inflammatory disease.

Again, the so-called amœbic liver "abscess," when sterile, is not an abscess at all. It is a primary colliquative necrosis of hepatic tissue, which is broken down and changed by a proteolytic enzyme from protein to albumoses and peptones; the patient's symptoms are due to the absorption of these poisons directly into the blood stream and can be reproduced experimentally by the intravenous injection of peptone.

To sum up: the pathology of histolytica amœbiasis is a primary degeneration due to the chemical digestion of the cells in the *immediate* neighbourhood only of the amœbic enzyme. It is not a primary inflammation with consequent degeneration; any inflammation which occurs is due to secondary microbic infection, is generally not severe, and takes place late in the disease.

The nature of the cellular exudate in the stools and in the uncontaminated liver "pus" is in entire accord with this basal fact of its pathology.

Pathology of Bacillary Dysentery.

Very different from the amœbic lesions are the changes which occur in bacillary dysentery. The infecting agents belong to a different kingdom.

The two diseases are so totally distinct that the only excuse for grouping them both under the heading of "Dysentery" is that of historical usage. In logic it were as reasonable to group cancer and tuberculosis of the intestine under the same heading because they may both give rise to what used to be called the "bloody flux."

Whatever may be the route of infection the essence of the pathology of bacillary dysentery is the intense diffuse catarrhal inflammation of the mucosa, which according to the severity of the attack may resolve or may go on to degenerative changes, such as coagulation-necrosis and sloughing. It is a primary inflammation of bacterial toxic origin, with all the cardinal signs of this condition—viz., "*rubor, tumor, calor, dolor et functio læsa.*"

The process is essentially one of diffuse destruction of the superficial layers of the mucosa, accompanied by œdema, large immigration of leucocytes, diapedesis of blood cells, and hæmorrhagic extravasation—such as is seen in all cases of acute bacterial inflammation of tissue. The immigration of polymorphs is an especially noteworthy feature. At autopsy the appearance of the intestine in any case of bacillary dysentery will vary with the intensity of the infection and the length of time the patient has survived it.

In the so-called fulminating ileo-colic dysentery, the entire surface of the lower part of the ileum and large intestine (*always excepting Peyer's patches and the appendix*) is seen to be the site of intense inflammatory change. No part escapes, as it does in amœbic dysentery; the bowel wall is œdematous, enormously thickened, deep plum-red in colour, and the mucosa may resemble the surface of a large granulating wound. A case of this intensity is more likely to be met with in patients who happen to be affected with renal disease of long standing, and death takes place from poisoning before the changes later to be described have had time to occur.

If the patient survives this stage, the superficial layers of the entire mucosa undergo coagulation-necrosis, become bile-stained, and are replaced by a green diphtheritic false membrane which may entirely hide the red granular layer underneath it. Here or there smaller or larger areas of this membrane may have sloughed, disclosing buds of granulation tissue of greater or lesser extent. Coincidently, small but numerous intramucosal hæmorrhagic extravasations take place.

If the patient again survives this stage small superficial ulcers, affecting particularly the summits of the valvulæ conniventes and mucosal folds, are formed. The minutely eroded, pinkish-grey "coraline" appearance of these ulcers, which run transversely across the bowel along the ridges of the mucosal folds of the colon, and are often present in large numbers, confers a very characteristic appearance. The sloughed mucosa is gradually replaced by well-formed and vascularised granulation tissue, and later by fibrous hyperplasia; if the patient recovers entirely the large intestine becomes shrunken into a thick-walled firm tube, with narrowing of its lumen and permanent damage to its functions.

Again, the ulcerative process may continue and spread, with the result that the gut wall may be honeycombed with myriads of small ulcers and so finally resemble a piece of red porous rubber.

§ They never contained red blood cells.