derives from assimilation of his food, which gives him a chance of building up his natural resistance to infections generally, whether malarial or other. It should be remembered that the malarial patient has been robbed of a good deal of his haemoglobin by the parasite, and this has to be replaced by his own metabolism; therefore his *primae viae*—liver and other organs—should be given a chance of doing their duty, and his digestive system should not be upset by heroic doses of quinine by the mouth. It serves no useful purpose, but only intoxicates the patient and every tissue of his body, and does not produce the full parasitocidal action of quinine, weight for weight, against the malaria protozoon.

For the information of general practitioners and accoucheurs I may state that quinine given by this hypodermic method can be safely administered to pregnant women—at least that is my personal experience of many cases, and I have never heard of any untoward event in such cases in the hands of any medical man who has used it in India.

I have never advocated the intramuscular injection of quinine, for the big doses used produce local necroses (so-called cold abscesses) in the muscle; this statement has recently been verified by Major MacGilchrist, I.M.S.,² by a *post-mortem* examination of a case fatal thirty-six hours after such an injection (11 grains of quinine bihydrochloride) into the gluteal muscle. This case serves as an object lesson to those who advocate the administration of quinine by intramuscular injection.

I saw many cases of cerebral malaria in the German East African campaign in the field far remote from the base hospitals (some of them mistaken for cerebro-spinal meningitis), and most of these cases recovered when treated on the lines recommended by me, by hypodermic injections. I have no doubt the intravenous method, as recommended by Sir Leonard Rogers, would produce just as good results, but it is not practicable for sections of field ambulances to carry about the more elaborate equipment for intravenous saline injections, in preference to the simpler and safer equipment for hypodermic administration, especially in the bush of Africa and similar terrain.

Prophylaxis.

As regards the prophylaxis of malaria, I hold, as do many other medical men, that the daily dose of quinine is not a prophylaxis against malaria; recent investigations and experiments on this question by Lieut.-Colonel Anderson, I.M.S.,³ on gael populations, showed that quinine as a prophylactic was useless and a distinct waste of public money, for the price now of quinine is enormous.

From a scientific point of view I ask this question: "If you state you are sterilizing a medium (that is, cinchonizing the patient's blood) in order to prohibit the growth of a parasite by daily dosing with quinine, and then find that, after all, this parasite does develop in this medium (through some specially enlisted resistance on the part of the parasite to its environments or any other explanation you like), how are you now going to kill (sterilize) by medication this parasite that has learnt to grow in this cinchonized medium? Are you going to increase the cinchonization of the medium by adding more quinine, and thereby teach the parasite to resist further and remain dormant in this medium till such time as he can regenerate?" Obviously the answer is in the negative. Then why do it? For this is the cause of chronic and relapsing malaria, in my opinion; so the only option apparently left to the physician to cure the case is to poison both patient and parasite by heroic doses of quinine and develop in him some other chronic ailment the result of quinine poisoning, the commonest of which is permanent deafness.

Of course it is understood that occasionally cases of malaria gravidarum occur which have to be tackled heroically to save life, but such cases are not now being considered.

The views here stated will, I know, be criticized by those who advocate doses of gr. xv to xx once weekly on Saturdays, or bi-weekly, on the grounds that they have not thereby "cinchonized the media," but have by this means caught the young parasites already there which would ultimately fulminate into a recognized attack if they were not killed. If this theory is right, and really represents what has actually occurred, then of course the answer is that the

dose of quinine given has been not a prophylactic but a curative dose, and if no malarial outburst occurs an actual infection of the parasite in the medium (blood) has been inadvertently sterilized. But prohibition of the infection, which is prophylaxis, has not been accomplished. Moreover this method (gr. xx once a week) has not kept people immune against malaria, and has therefore failed as a prophylactic.

A further criticism I have heard is contained in the theory that although quinine given prophylactically does not actually immunize the patient against an attack, yet it modifies the toxic action of the parasitic infection, so that only a mild attack is produced—as an anti-typhoid injection acts as a prophylactic. Against this theory I advance my practical experience in the East African campaign. I have never seen so many cases of cerebral malaria, haemoglobinuria (blackwater fever), in all my twenty-eight years' experience in the tropics as I saw in East Africa, yet every one who could was taking quinine prophylactically.

Finally, the only prophylactic medication against malaria for armies in the field, in my opinion and experience, on the ground of practical politics, is arsenious acid $\frac{1}{2}$ grain daily, thereby keeping up the natural resistance of the body generally against many ills, not only malaria. There need be no fear of the cumulative action of arsenic, for the troops very often do not get access to the drug, and a break from the drug can always be arranged for one week every month if necessary. It is easier to stop issuing a drug than to supply it regularly.

A golden rule for tackling the question of malaria for the armies in the field is "arsenic as a prophylactic and quinine as a curative only." Yet this is no new precept, for the Italians have done this years ago.

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EPIDEMIC POLIOENCEPHALITIS.

(SO-CALLED EPIDEMIC BOTULISM.)

BY

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PART II.

IN considering the nature and cause of the present epidemic of cases, many of them presenting paralysis of cranial nerves, which in a recent contribution to this JOURNAL I have designated as polioencephalitis, it is well to bear in mind—in view of the fact that they have been so widely referred to as cases of botulism—that somewhat similar symptoms may occasionally be the result of diverse causes. In both botulism and polioencephalitis paralysees of cranial nerves are features in common, and Van Ermengem, in his classical paper on the outbreak of botulism in Ellezelles, lays stress upon this point. He states that "in the absence of the previous history" (of poisoning having occurred), "it is impossible to distinguish with certainty cases of botulism from bulbar paralysis (Erb's syndrome), subacute polioencephalo-myelitis, and certain ophthalmoplegias."

Polioencephalitis.

Everybody who has paid attention to the subject now recognizes that there exists an acute primary specific disease affecting the grey matter of the brain, of which sporadic cases are not uncommon. This malady, known as cephalitis or polioencephalitis, was first described by Strümpell in 1884, and, in the form which not uncommonly leads to the development of spastic hemiplegia in young children, is fairly familiar. It is comparable with acute anterior poliomyelitis (infantile paralysis), and, like that disease, often occurs in children, is of acute onset and tends towards recovery. It may or may not be associated with such general symptoms as fever, convulsions, headache, stupor or loss of consciousness, or pronounced irritability. The special symptoms produced by the disease depend, of course, upon the particular region of the grey

matter of the brain involved. For example, polioencephalitis *superior*, which affects the nuclei of the cerebrum and cerebellum, may give rise to marked mental changes, coma, hemiplegia, diplegia, and ataxia, while polioencephalitis *inferior*, which affects the nuclei of the pons and medulla, may cause paralysis of one or other or several of the cranial nerves, and combinations of the two may be met with.

The evidence, both clinical and pathological, goes to show that these affections of the grey matter, whether of the brain or spinal cord, are in reality one and the same disease, and are due to a single factor. Recent researches have demonstrated that this factor is a living contagium, since it has been shown that the contagion can be conveyed indefinitely from monkey to monkey by inoculation, though the organism, which is so minute as to pass through the Berkefeld filter, has not yet been for certain isolated. The two affections may occur in epidemics, in which some members of the same family may suffer from the spinal and others from the cerebral variety. Indeed cases are known in which such eye symptoms as ptosis, ophthalmoplegia, and pupillary changes have been associated with affection of the spinal cord, and the combined condition has been termed polioencephalomyelitis.

Stephenson¹ relates a series of sporadic cases over a considerable period in which affection of the ocular muscles was the most prominent feature, and to his excellent introductory remarks on the nature of the disease I am much indebted. The majority of his cases were in children, and consisted mainly of simple oculomotor paralysis, most commonly of the external rectus alone, but in others of various branches of the third nerve. There were associated symptoms in eight cases out of twenty-eight, such as fits, unconsciousness, sleepiness, and loss of energy.

The cases that have been recently described accord in general with this description, and with that of other writers, of sporadic cases of polioencephalitis, and the case that I saw in 1913, which was generally recognized to be of this nature, would unhesitatingly be classed among the epidemic cases had it occurred in the past few months. One feature in that case, which at first made me distrust the diagnosis of polioencephalitis and favour that of cerebral tumour, was the presence of optic neuritis, but I subsequently found that this, though not a common feature, yet was occasionally met with in polioencephalitis, and it is paralleled by two, at any rate, of my present series. Most of the cases are subacute, the paralysis mostly develops suddenly, but in several certain phases of the paralysis were delayed for days or even weeks. The pupils have not been dilated, nor has there been paralysis of accommodation in any of my cases.

Botulism.

Now with this picture of polioencephalitis let me compare that of botulism, mainly relying on Van Ermengem's description.² In his cases, which occurred after eating an infected ham (raw after the foreign fashion), there was a sudden onset of symptoms after a period of twelve to twenty-four or at most thirty-six hours. These consisted of thirst and a feeling of constriction of the throat, soon followed by paralysis of accommodation and other ocular paralyses. The paralysis for near vision and the dilatation of the pupils were constant symptoms, and double vision, squint, and ptosis, were usually met with. In the more severe cases difficulty of swallowing, or inability, supervened, with loss of voice, constipation, and retention of urine. Fever and disturbance of sensation or consciousness were absent, only in the very last stage of fatal cases did coma or a mild delirium develop. The fatal cases were characterized by gradually advancing respiratory and cardiac failure due to progressive bulbar paralysis. About one-third of those who had eaten the poisonous food died in a few days; another third, after more or less severe symptoms, got well; the rest, who had only taken very small amounts of the infected flesh, remained free from symptoms. In those that recovered the ocular paralysis began to improve after two to three weeks, though in some it was several months before the paralysis of accommodation got completely well. The general weakness, which was a characteristic feature, also took several weeks to recover.

Differential Diagnosis.

The likeness between the two conditions rests upon the occurrence of ocular paralysis in each, particularly the double vision, squint, and ptosis. Apart from this striking feature difference outweighs resemblance. In botulism the onset is sudden, with urgently acute symptoms coming on after partaking of some often definitely recognizable poisonous article of diet. There is dryness and a sense of constriction in the throat, with dilatation of the pupil and loss of accommodation; there is great bodily prostration but no psychical change, and there is early death or early cessation of the acute symptoms with slow convalescence. In the cases in this epidemic and in other cases of polioencephalitis these features are in the main wanting. The onset is either subacute, or the development of the paralysis may be spread over several days, or even weeks. There is no feeling of constriction of the throat, no dilatation of the pupil, no loss of accommodation. There has been little bodily prostration in the cases I have seen, except in those cases of polioencephalitis *superior* in which the grey matter of the cortex is involved, when it is associated with psychical changes—sleepiness, lethargy, coma, and delirium—which are absent in botulism. The majority of cases recover; if not, the fatal ending comes after a course of two or three weeks or more, though occasionally earlier.

But the most striking difference lies in the mode of occurrence of the outbreaks of the two conditions. In botulism, almost without exception, the disease occurs in groups, varying in their numbers but including most of those who have partaken of the infected food. Since sometimes the infection of the food is limited to certain parts, those who eat of the non-infected parts may escape; but though the groups may thus be diminished in size it remains true that botulism, as hitherto recorded, is a "group infection." On the other hand, it is notable that in the present epidemic in no instance, so far as cases have yet been published, has any group infection occurred. The cases have been widely scattered, sometimes two in the same neighbourhood, but no two even in any instance in the same house. This might be thought to prove too much, since it might be supposed to disprove the infectious, and hence infective, character of the disease. But it is well recognized that epidemics of other diseases commonly show similar features. It is often very difficult in the case of diphtheria, enteric fever, or cerebro-spinal meningitis to trace the mode of infection in the numerous scattered cases that constitute an epidemic, but it is now proved that the symptom-free carrier or the individual with minimal and unrecognized symptoms plays an important part in such diffuse epidemics. Poliomyelitis in its epidemic form is known to fall into the same category; and such, I take it, is the true explanation of the apparently unrelated cases in the present epidemic. It is a truism of epidemiology that if the movements of contacts are only worked out with sufficient care and thoroughness, indirect connexions between apparently isolated cases in epidemics of the above diseases can almost always be established.

It is probably true that during the last few months, during which the supply of fresh meat has been so greatly restricted, more tinned food has been eaten in this country than ever before. That tinned food may occasionally become accidentally infected with the *Bacillus botulinus* is a well-known fact, but, as I have argued, the incidence of this epidemic is quite different from the incidence of such attacks of botulism as would thereby be produced. The only conceivable way in which such a distribution could occur would be on the supposition that there was a widespread infection of tinned food with a bacillus of attenuated virulence. In such a case there might be extensive ingestion of small doses of its toxin so small that only those with special susceptibility to its action, or those who ate specially freely of tinned food, showed symptoms of poisoning. This is exactly what is met with in ergotism, in which widespread epidemics may occur, due to poisoning of the bread with the ergot of rye, and in the slighter epidemics, when the degree of contamination is low, the disease may pick out sporadic victims in the midst of general immunity. However, though I mention this possibility, there is no reason to suppose that it represents the facts of the present epidemic, since, so far as investigation has gone, there is no evidence at present of any widespread or even occasional

infection of tinned food with *B. botulinus*. Moreover, certainly one of my patients has disclaimed ever having taken tinned food at all; and its relative infrequency among soldiers from abroad who have lived very largely on tinned food is significant.

Prognosis and Treatment.

It remains now to speak of the prognosis and treatment. In the majority of cases recovery occurs, though there is more likelihood of a fatal termination in those cases of the *superior* type; even here, however, recovery may quite unexpectedly take place. But what is of importance is that we are able to hold out to the patient's friends the possibility of recovery from a condition which, but for our knowledge of its nature, we should otherwise regard either as absolutely fatal or as the commencement of a chronic incurable disease, since different cases may simulate cerebral tumour, tuberculous meningitis, or the onset of disseminate sclerosis. The recovery from paralysis may be incomplete, as in poliomyelitis; it is too soon to speak for certain of this in the present epidemic, but some, at any rate, have made a full recovery.

Treatment seems to have little influence on the course of the disease, but, as in poliomyelitis, we should resort to complete and absolute rest in the dark, with or without the use of bromides or other nerve sedatives, during the stage of onset. Urotropin has been given, from a belief in its antiseptic effect on the central nervous system, and salol for a similar action on the nasopharynx, by which route infection is supposed to reach the brain. Lumbar puncture should be performed and, if necessary, repeated in cases which show the more marked signs of intracranial pressure, such as severe pain in the head, vomiting, and unconsciousness. Later, tonic treatment, such as iron, arsenic, strychnine, and cod-liver oil, with fresh air, are likeliest to restore the vitality of the damaged nerve centres.

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THE "THICK DROP" METHOD FOR THE DETECTION OF SCANTY SPIROCHAETES IN THE BLOOD.

BY

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In the majority of cases of Palestine relapsing fever (among British troops) which have come under my notice the spirochaetes have been throughout present in the blood in very scanty numbers. This has entailed a prolonged search before the diagnosis could be established; frequently a negative finding has had to be returned, as films cannot be carefully scrutinized in their entirety when a large number have to be examined. On several occasions the spirochaetes have first been demonstrated, after considerable search, at the second or even third time of examination of the particular case.

I have therefore made use of the "thick drop" method, which has proved of much service, not only saving time, but also frequently enabling me to report as relapsing fever, cases in which the examination of an ordinary blood film yielded no result.

To prepare a thick drop for examination, a small drop of blood is placed upon a clean slide. The drop should be about as large as is required for making a good-sized smear and should be spread out a little (with the needle) so as to cover an area about $\frac{3}{8}$ in. in diameter. Too thick a drop is not an advantage, as it is difficult to lake (dehaemoglobinize) completely, and more fibrin is present. The drop must be allowed to dry thoroughly, and before proceeding to lake it, may be gently heated by passing it over a flame two or three times.

There are several well-known methods of laking the thick drop. I have made use of two: (a) Distilled water, or (b) acidulated methyl alcohol.

(a) A few drops of distilled water are poured upon the thick drop, and the slide is gently agitated to and fro. The water may be renewed until the blood has completely lost its colour.

The process takes four or five minutes, depending on the thickness of the drop.

(b) Methyl alcohol acidulated is used in the proportion of 10 or 12 drops of HCl to 50 c.cm. of methyl alcohol; the procedure is the same as in the former case, but the laking takes rather longer. After the colour has been extracted, the drop is well rinsed with distilled water and dried.

Dehaemoglobinization is more complete and rapid by the former method, and there is less formation of fibrin. But unless the greatest care is exercised, the drop, or portions of it, are apt to be removed from the slide, in spite of complete drying (as there has been no proper fixation previously). If distilled water is used, and the preparation is to be stained by gentian-violet, it is advisable, after carefully draining off the water when the laking is complete, to pour on a few drops of methyl alcohol, for a minute or two, to fix the preparation to the slide.

The thick drop may be stained by the Giemsa or Leishman method, or with aniline gentian-violet; the last gives excellent results with spirochaetes, the intensely blue-stained parasites standing out well from the surrounding elements. Aniline gentian-violet is used in the proportion of 9 parts of aniline water to 1 part of alcoholic gentian-violet, the stain being left on for twenty seconds or so. The preparation is then well washed with distilled water, by which means the requisite degree of extraction and differentiation can be obtained, and finally dried.

Where examination is desired for suspected malaria or relapsing fever, it is a useful plan to have the slide sent in with both a fair-sized film (extending, say, over two-thirds of the slide) and a thick drop (on the other third) upon it. For staining, the smear can be separated from the drop by drawing a line with a grease pencil across the slide, between the two. The smear is stained by the Giemsa or Leishman method, and if no malarial parasites are found, nor spirochaetes quickly seen, the thick drop is used. If it is desired to examine the drop also for scarce malarial parasites (for example, crescents) as well as spirochaetes, the second method of laking is to be preferred, as distilled water is very liable to disintegrate malarial parasites; and in this case the Giemsa or Leishman method must be used for staining.

INTERNAL DERANGEMENTS OF THE KNEE-JOINT.

BY

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DURING the past eighteen months several patients have been sent to the Military Hospital, Tidworth, on account of painful cicatrices on the inner side of the joint. In all cases the cicatrices were the result of linear incisions down the inner side of the joint in line with the patella and ligamentum patellae, over the prominent part of the internal condyle. The history was of an operation performed for some internal derangement of the joint. This has been especially noticeable in cavalymen.

Since September, 1916, it has been found advisable to segregate the knee cases, owing to the large number, principally the result of football accidents, but at one time a number of injured knees followed trench training. There has been on an average at least twenty cases in hospital daily suffering from derangements of the knee-joint the result of injury.

They practically all suffered from traumatic synovitis, some simple, the others with complications.

The most frequent complications were:

Firstly: Rupture of some fibres of the internal lateral ligament, indicated by acute pain on pressure over the inner side of the internal condyle.

Secondly: Looseness with partial or complete dislocation of the internal semilunar cartilage, indicated by pain on pressure over the site of the anterior end of the cartilage, inability to completely extend the joint, and a history of "locking."

Thirdly: A general stretching of the ligaments was the only objective symptom, besides the synovial effusion. This applied more especially to the posterior crucial ligament, which is the one ligament of the joint put on the