From these experiments it is evident that this alkaline solution of hypochlorite is not much more stable than bleaching powder, and is of little use as a stable sterilizer in tropical countries. The amount of chlorine liberated depends on the acidity of the water, and in no case of the water samples examined was the amount necessary to liberate all the available chlorine found to be present.

I am deeply indebted to Major W. W. Browne, R.A.M.C., Officer Commanding Enteric Convalescent Depot, Wellington, and to Major G. S. Wallace, R.A.M.C., D.A.D.M.S. (Sanitary), Secunderabad Division, for permission to do the above work in the Divisional Laboratory, Wellington, India, during my convalescence.

Lecture.

CLINICAL AND PATHOLOGICAL CO-OPERATION.1

By Captain P. H. Bahr,
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The subject for discussion to-night is undoubtedly a controversial one and, though there is a natural tendency for laboratory workers to be dogmatic on subjects to which they have devoted particular attention, yet I will endeavour to be as little didactic as is possible. The title I have chosen for this paper is one, which interpreted to its widest sense, is of vital interest to us all; by that I mean the collaboration between the clinician and the pathologist so as to arrive at a sure and as speedy a diagnosis as possible, one which will not only be of benefit to the patient, but also to the Army and, ultimately of course, to the country which we serve. I think one can say that there has been on the whole too great a tendency in the past for the clinician and the pathologist to work in watertight compartments and, in certain quarters, a tendency on the part of the clinician to rely too much upon a pathological diagnosis which is perhaps unsupported by clinical data and which a short consultation in the wards or in the laboratory would soon have adjusted.

I would like to see the clinician much more often in the laboratory and the pathologist in the wards, both acting in a consultative capacity, and I would like to see it established as a rule that a pathological diagnosis which does not agree with the clinical signs should be regarded every bit as unsatisfactory as a clinical diagnosis which does not tally with the pathological report.

To quote Emerson in the preface to his well-known “Clinical Diagnosis,” he says: “The function of the clinical laboratory worker is to aid the ward worker. The findings of the former are seldom conclusive and must be interpreted in the light of the ward findings; especially is this true now that functional diagnosis is the goal. . . . The clinical chemist must first be a good clinician and secondly a chemist; he should remember that, even from a laboratory point of view, his

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1 An address delivered before the Cairo and Delta District Medical Society on December 15, 1916.
stethoscope is of more importance than the microscope, his percussion finger than the whole outfit of chemical apparatus."

The modern pathologist cannot afford to disregard clinical phenomena. I will now give you a few details of what I mean based upon my experience as pathologist and clinician during the last two years of military practice as well as upon a varied tropical experience during the last seven years.

I have long been of the conviction that an intimate knowledge of Tropical Medicine is almost essential to every clinician and pathologist practising in the East, and I look forward to the day when a knowledge of what is now a speciality may become part of the ordinary medical curriculum. Now the essence of Tropical Medicine is a study of the blood, and if possible, it is a still higher factor here in Egypt, situated as this country is in a zone common to diseases of temperate and those peculiar to tropical climates.

I shall deal first with the aid which can be derived from a simple blood examination of the pyrexias of this country. Firstly, it is advisable to examine the blood for the malarial parasite in every case of fever. The discovery of this parasite in an ordinary Leishman-stained film is an easy matter, and such a film should be examined in every case. This is of vital importance, because it is the only fever for which we possess an absolutely specific drug in quinine.

There is an idea abroad that the malaria parasite can only be found in the peripheral blood during a short period immediately preceding and following the malarial rigor. This I might say is a complete fallacy as regards the benign forms of malaria—the benign tertian, which is extremely common, and the quartan, which seems to be an extremely rare parasite in this country.

As regards the malignant or sub-tertian parasite the case assumes an entirely different and much more serious complexion. I would like to remind you that only the youngest and the most mature (i.e., the ring and the crescent) forms of this parasite are found normally in the peripheral blood. The rest of the cycle, of which specimens have been exhibited for your inspection to-night, takes place in the internal organs—such as the brain, spleen, bone-marrow, pancreas and intestinal capillaries. From the blood stasis thus produced in a non-immune subject, the most serious symptoms, dangerous to life itself, may be suddenly produced. The symptoms elicited vary according to the organ attacked—thus we may get hyperpyrexia, this is often named heat-stroke; coma; the so-called sun-stroke of West Africa and other tropical countries; conditions resembling appendicitis, cholera, intestinal obstruction, rheumatism, acute nephritis, pernicious anaemia, malignant jaundice (often misnamed yellow fever, the bilious remittent fever of the tropics) or it may even produce blackwater fever itself. All these conditions can be promptly cured with an early and efficient dose of quinine. It is therefore of the utmost importance to examine the blood at the earliest possible moment. But may I beg you not to do as often as has been done in the past, when treating with such suddenly developed and calamitous symptoms, to await a pathological diagnosis, which cannot be rapidly obtained, before exhibiting quinine, but rather to take a blood-smear and forward it to the nearest laboratory, immediately after examining the patient, and then to administer quinine before awaiting the result, which might take days to arrive. If this practice were invariably conformed to, I should be unable to exhibit some of
the microscopical post-mortem specimens you will see to-night. An intelligent appreciation of the life-history of this parasite will soon convince you that in the majority of cases of fresh infections the parasite may not be present in the peripheral blood for thirty-six hours or more from the commencement of the attack, and before that period has elapsed some patients may be dead or in too serious a condition for the quinine to act. It is therefore important to give quinine immediately in all cases exhibiting serious symptoms of this sort, in whom either a pathological diagnosis cannot be obtained or in whom such diagnosis is unsatisfactory from a clinical point of view. You may take it as a general rule from experience gained on the canal zone, as well as elsewhere, that a pyrexia followed by a rigor occurring in a malarious district during the forenoon or early afternoon is almost invariably of malarial origin, and it is necessary to bear in mind, as a corollary of this statement, that cases of malignant malaria may and do occur without any pronounced pyrexia at all—the so-called algide forms. I would once more remind you that, though the benign forms may be trivial and do not warrant the invaliding of an individual, the malignant form is a different matter altogether.

The diagnosis of relapsing fever is generally an easy matter, as the temperature chart is so typical and the spirochete is so easily found in a properly stained film (carbol fuchsin being the best stain), but it is necessary to keep in mind that the spirochete very frequently disappears from the blood-stream about forty-eight hours before the crisis, and, unless this fact is recognised, many cases which would otherwise be diagnosed on clinical grounds as relapsing fever will be missed for want of a pathological confirmation.

As regards the procedure to secure a diagnosis in cases of pyrexia in which the malarial parasite or the spirochete cannot be found, we can obtain a great deal of information, if not a probable diagnosis, from the correlation of the temperature chart together with a total and a differential leucocyte count. I should like to impress upon you that one without the other is useless, and that, in order to obtain the necessary accurate information, the films for the differential count should be made in a particular way, and that they should be stained with hematoxylin and eosin. I find that any other stain gives, owing to specific reactions for glycogenic granules, etc., a totally erroneous idea of the due proportions of the different varieties of white cells present in the film. At least a total of 300 cells should be counted and a considerable practice in this procedure is necessary. A leucopenia of 2,000 to 3,000 whites with a high mononuclear count, i.e., from fifteen per cent upwards (by the large mononuclear cell I mean the large hyaline cell with kidney-shaped or elongated nucleus of text-books) strongly indicates a persistent protozoal infection, such as malaria, leishmaniasis or trypanosomiasis. The latter two diseases of course are of infrequent or of extremely rare occurrence in this country, but may presumably occur from time to time in troops hailing either from the Sudan or East Africa. Now, a continued pyrexia which does not respond to quinine together with a reduction in the number of red cells with a differential leucocyte count such as I have described ought to lead the clinician to puncture the spleen or liver for the Leishman-Donovan body and the pathologist to make a long-continued search of the blood for the trypanosome. A leucocytosis, on the other hand, with an actual increase of the polymorphonuclears over seventy-five per cent in a patient with a continuous nocturnal
pyrexia of a septic type together with a peculiar pallor of the facies and, it may be, with symptoms of a right pleurisy, strongly suggests the probable diagnosis of liver abscess. Such a patient may or may not have given a history of a previous dysentery with the presence of the Entamoeba histolytica or its cysts in the stools. In cases of this nature the therapeutic test alone—i.e., the strenuous exhibition of emetine both orally and intramuscularly administered—will alone decide the diagnosis. And I am glad to say many favourable results have latterly been recorded as a result of this procedure. At all events, in amoebiasis of the liver or of the gut it is absolutely necessary in the interests of the patient to act energetically and promptly without a moment's hesitation.

Now a slight rise in the mononuclear count, together with a decided leucopenia occurring on the third or fourth day of the pyrexia, if associated with typical clinical phenomena such as a bradycardia, is characteristic of three tropical fevers—yellow fever, dengue and phlebotomus fever. The latter two of course are commonly met with in this country, and the subsequent course of the fever will naturally decide the diagnosis. As far as I know there, is no blood change peculiar to seven-day fever. The saddle-backed character of the chart alone can render the diagnosis in this case.

The season of the year at which these fevers occur is of importance, phlebotomus fever occurring either in the early summer—i.e., June or in the autumn (the latter part of August and September), when the insect transmitter is most abundant, while dengue generally only occurs during the hottest part of the year—i.e., July and early August, though epidemics have been exceptionally recorded in October. High atmospheric temperature appears to be one of the conditions it demands, and when perhaps the breeding season of Culex fatigans, the transmitting mosquito, is at its height. Unfortunately for diagnosis, the blood-picture of undulant fever or Malta fever is in no way characteristic. But a continued diurnal and persistent lymphocytosis (i.e., a rise above thirty per cent) may be taken as indicating that the pyrexia is of tubercular origin. An eosinophilia of over five per cent without a leucocytosis in this country is almost diagnostic of some helminthic infection such as the ascaris or the ankylostome or the urinary bilharzia. But a high eosinophilia of twenty to thirty per cent or over, together with a total leucocytosis of 10,000 to 15,000 white cells, in a patient with anaemia, emaciation, enlarged liver, tenderness over the gall-bladder and often urticaria as well, should lead the pathologist to search long and on several occasions with a low-powered lens for the ova of the rectal bilharzia. There is no doubt that these cases—a chart of one of which has been exhibited this afternoon—suggest at first sight enterica. Rectal bilharzia (B. mansonii) then, which is occurring among the troops in Egypt, produces a systemic disease identical in its clinical features with "Katayama disease," due to the allied Schistosomum japonicum in Japan. This is an entirely new clinical fact, and the credit of the discovery belongs to the staff of the Third Australian General Hospital. On the other hand the urinary bilharzia appears to produce no such marked systemic disturbance and only a slight eosinophilia.

I next propose to deal shortly with the serum diagnosis of enterica and Malta or undulant fever. At the outset I might say that my statements are based entirely on the readings obtained by Captain Garrow's agglutinometer, from which
I venture to say results of very great military importance have been obtained. With reference to the diagnosis of enterica we learn that:

1. Any reading of a titre under 1 in 80 in a person who has been inoculated within the past year with a vaccine of enterica—"T." or "B."—must be disregarded as of any diagnostic importance.

2. The residual agglutination titre remains at this figure for a year or more.

3. The "A." agglutinins do not persist to anything like the same extent as the other two, and it is rare to find a case with an agglutination titre to "A" of over 1 in 40 six months after a "T.A.B." inoculation.

The value of a serum diagnosis is still unimpaired in cases in whom a blood culture cannot be obtained or in which it is too late to obtain it. Supposing then the titre either to "T." "A." or "B." be found to rise after repeated Widal tests from the ninth to the sixteenth day of the disease the case may be justly regarded as of being that particular infection.

Blood culture still remains the most certain means of diagnosis in enterica, but apparently this can only be expected in not more than 30 per cent of cases in hospital practice. Personally I place considerable reliance on a positive result of urine culture as indicating the particular infection from which the patient suffered. But it is otherwise I think in the case of stool cultures, for instances have occurred in which a certain bacillus, say para. B, has been obtained from the stool in cases which certainly suffered originally from para. "A" septicemia, the former bacillus having presumably previously existed in the intestinal canal as a saprophyte.

The serum diagnosis of Malta and para.-Malta fever presents certain characteristics which recent experience has verified. Firstly, the agglutinins appear in the blood only after a considerable period of pyrexia, say three weeks or more, and then a positive agglutination may occur only in a low dilution of 1 in 10 or 1 in 40. Secondly, in cases with a high agglutinin serum-content a marked inhibition zone occurs: that is, the serum, owing apparently to the presence of anti-agglutinins, may not agglutinate the specific organism in lower dilutions than 1 in 160, but may strongly agglutinate the organism from that figure upwards. As regards the clinical aspect of Malta fever in this country, the temperature charts are by no means always typical, and I have recently seen a series of cases of para.-Malta fever which resembled closely those of miliary tuberculosis, but it has been pointed out that a persistent maximum rise of temperature daily at 2 p.m. is nearly always in itself diagnostic of Malta fever. I think we should remember then that para.-Malta fever is not of infrequent occurrence in this country and that in its serum reactions the paramelitensis coccus is quite distinct from the original melitensis strain.

The diagnosis of dysentery exhibits in the closest possible manner the point I wish to emphasize to-night, for it demands, more than any other, the closest co-operation between the physician and the pathologist. It is necessary to bear in mind that the specific cause of the commonest forms of dysentery in this country—the entameoba and the dysentery bacillus are both delicate organisms and die out in a very short time after the stool has been passed; therefore a microscopic examination of a stool specimen which has stood for four hours or more can be of no value whatever; the amœba will be dead and therefore non-motile; and I hold that this is impossible for the most practised microscopist to differentiate in this state between the dead amœba, and any other dead and disintegrating cell, such as
Clinical and Pathological Co-operation

are found in such numbers in any dysentery stool. In freshly passed stools living and active amoebae containing ingested red cells should be passed in 90 per cent of all amoebic cases, and the diagnosis thus becomes an easy matter, and I think that Lieutenant-Colonel Wenyon's rule; that any active amoeba with ingested red cells in a dysentery stool should be regarded as the Entaméba histolytica, is an extremely sound one to follow.

It is highly necessary that the clinician himself, in order to bring about this utopian state of affairs, should inspect all dysentery stools himself, but more especially those he sends to the laboratory for diagnosis. In any case—whether amoebic or not—it is useless to send feculent stools when the patient may be from time to time passing ones consisting almost entirely of blood and mucus, for in both diseases the specific cause is found only in the pathological exudate and not in the faces themselves. One ought to bear in mind that a class of diarrheas of amoebic origin does undoubtedly exist in which the amoebae are only intermittently excreted and these account for the remaining cases in which the specific cause cannot be found during the first microscopic examination. Therefore should the clinician be satisfied that from his own point of view the pathological report is unsatisfactory, he should not rest content with the result of a single microscopic examination of his patient's stool.

I think from what I have told you, that you will bear me out that by itself the diagnosis of dysentery constitutes the strongest plea for the decentralization of simple laboratory work such as has been attempted latterly here in Cairo, and that everyone in the tropics should have its own laboratory, however small, for the diagnosis of these conditions on the spot.

The question arises whether it is possible to differentiate between the dysenteries on the macroscopic appearances of the stool. I think it is by no means always possible to do so, but as a general rule the amoebic stool contains small clots of mucus, together with small clots of blood intimately mingled with the fecal contents, thus giving the appearance of "anchovy sauce." I must apologize for the very disagreeable habit, peculiar to our profession, of comparing pathological exudates to articles of diet, but naturally their exact appearance depends, as indeed also in the bacillary form, on the situation, condition, and extent of the lesions; on the other hand, the stools passed in the early stages of bacillary dysentery resemble nothing more than red-currant jelly, or sometimes frog's spawn, while in the more advanced stages they have been compared to meat washings, due to the hemolysis of the red cells and the consequent diffusion of the haemoglobin and its decomposition products into the surrounding mucus.

In the microscopical examination I lay great stress on the cell exudate, as not only differentiating the two forms, but as also indicating the stage of the disease and the ultimate progress of the patient. Briefly then the presence of a number of macrophage cells—that is a large hyaline cell twenty to thirty microns in diameter, containing chromatid bodies and often red cells as well, but with no very definite structure—together with a large portion of pus cells, relatively few red cells, and few visible bacilli, indicates a bacillary infection and suggests a suitable stool for culture. These macrophage cells I have just mentioned are very important, as they have often been mistaken by the uninitiated in the past for amoebae, and being derived, as I have good reason to believe, from the submucosa, their continued presence in the stool is of favourable import as indicating that the...
repair of the tissues is taking place. On the other hand, bile-stained disintegrating pus cells and the intestinal epithelium, together with numerous bacilli in the microscopic field, denote that an extensive necrosis of the mucosa has taken place. Such a stool is quite unsuitable for culture and in many such cases I have been able to obtain a culture of the dysentery bacillus from the mucus membrane post mortem when I was quite unable to do so from the stool during life. The failure, then, to obtain the specific bacillus from the stool during life does not invariably mean that the case is not one of bacillary origin. Under the circumstances it seems to me a waste of time and material to demand, as has been done, two negative examinations of the stools of a patient convalescent from bacillary dysentery before his discharge from hospital. This is a most tedious practice and endeavours to find the specific dysentery bacillus in a feculent stool are rarely, if ever, successful.

I sincerely think that this practice ought to be discontinued as tending to keep patients in hospital longer than necessary and as being a quite unpractical procedure. It is like looking for a pin in a haystack.

The culture of the dysentery stool presents certain peculiarities and it is impossible even with a pure blood and mucus stool and the most improved technique to render a definite report before forty-eight hours have elapsed from the time of the dispatch of the specimen to the laboratory.

I think it is quite permissible for the pathologist under these circumstances, in order to facilitate early specific treatment in the interests of the patient, which is really all that is required in practice, to suggest a diagnosis on his microscopical findings of the stool alone, and he will, according to my experience, be right in nearly every case; he then can render a finished report at his leisure.

The bilharzia dysentery stool had characteristics of its own—that is, the mucus is of a gelatinous nature and of a yellow colour, containing clots of dark blood in which the characteristic ova are present in large numbers. The pathologist ought to be warned of the clinician's suspicions as to the diagnosis of this condition, as bilharzia ova are searched for under a low-power lens and would certainly be missed with a higher magnification.

I think one must admit that there exists a distinct clinical entity, "lamblia dysentery"—due to the presence, adhering to the intestinal epithelium of the small gut, of large numbers of lamblia.

The stools in the acute stage of this disease consist of green bile-stained tenacious mucus and may sometimes contain a little blood as well. Such patients even in the acute stage are not really seriously ill. None of them appear to be very emaciated, and as the lamblia is an extremely common protozoal commensal parasite among tropical natives, and as these people by long association appear to become tolerant of it, I do not see why the troops in this country should not do the same, and personally I do think that a fetish is now being made of the presence of a few lamblia cysts in an otherwise perfectly normal healthy stool. Why should these entitle a strong, lusty, and apparently healthy man to be permanently invalided when directly he becomes tolerant of the parasite he may be of real service in the field?

Nearly all the vital diagnostic pathological work which I have recounted to you can be done with a very simple apparatus, the use of a few stains, slides and
a travelling microscope. In fact such a portable laboratory can be contained in a small case, such as one which has been my constant companion for years past.

Finally, gentlemen, I hope you will see that in order to satisfy all claims in Egypt one requires to be a clinician, to have a knowledge of tropical medicine, and I am not quite sure, in order to fully appreciate the needs of the Service and one's combatant fellow-officers, to have been a regimental M.O. as well.

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**Reviews.**


The management of venereal diseases amongst the troops of a huge army such as one of those now operating in the present War is a problem which is complicated by many novel factors. Considerations of man-power wastage, for example, make it necessary to strike a right balance between the claims of treatment and military necessities. How far the former must be subordinated to the latter is largely a matter of individual opinion guided by experience, and the book under review is interesting as an exposition of the views of an eminent French syphilologist with regard to the syphilis problem in the French Army. It will be particularly interesting to those who are acquainted with the manner in which the same problem is treated in our own Army.

In common with all who have studied the subject, the author is alarmed at the outlook for his country if syphilis is to continue uncombed by energetic measures. Calculating that the infection of each mobilized soldier or munition worker will mean the loss of one mother of a family and one soldier in the decennial period 1936-45, he estimates that the infections during the War will cost France 400,000 soldiers in that period, or the equivalent of two military classes. His analysis of the sources of infection reveals, as in this country, the important part which is being played by "amateurs" of all classes. He is greatly struck by the number of women infected by their husbands and of lads between 16 and 18 who have contracted syphilis. The prospect outlined by the author is anything but cheerful, but will be useful if it serves its purpose of rousing the country to a true idea of the problem as one of urgent national importance.

Dealing with the diagnosis and treatment of syphilis, the author is mainly concerned with the earlier stages of syphilis, since the tertiary affect the Army very little. For the diagnosis of primary syphilis he lays stress on the importance of microscopic examination and describes the dark-ground illumination as well as a number of staining methods of demonstrating the *Treponema pallidum*. In some instances the instructions are not very precise; one could wish, for instance, for more details regarding the preparation of the ammoniacal solution of silver nitrate used in Fontana's stain and as to the method of dark-ground illumination, particularly as the former is such a popular method of staining and the latter the method of choice for the demonstration of the *Spirocheta pallida*. In describing methods of obtaining the material no mention is made of the very simple technique of gland puncture when the sore has healed or has been treated with antiseptics, though the author mentions the taking of material for sections from the chancre in such difficult cases. Apparently in France, as in this country, the